Department of Internal Medicine

TREATMENT PROTOCOLS

K.C.M.C

Second edition

Edited by:
DR. KAJIRU KILONZO
DR. HUDA AL AKRABI

FACULTY OF MEDICINE- KCMUCo

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PREFACE

Authors

Professor Venance Maro,
Physician, KCMUCo

Dr. Kajiru Kilonzo
Physician, KCMC

Dr. Sarah Urasa
Neurologist, KCMC

Dr. Huda Al Akrabi
Physician, KCMC

Dr. Nyasatu Chamba
Physician, KCMC

Dr. Furaha Lyamuya
Physician, KCMC

Dr. Saleh Mbarak
Resident Internal Medicine, KCMUCo

DR. Lucas Kieji
Resident Internal Medicine, KCMUCo

Dr. Said Kanenda
Resident Internal Medicine, KCMUCo

Dr. Flora Ruhangisa
Resident Internal Medicine, KCMUCo

Dr Titus Msoka
Registrar, Internal Medicine, KCMUCo

Dr. Bhavana
Resident Internal Medicine, DUKE

Dr. Grace
Resident Internal Medicine, DUKE

Prof. William Howlett
Neurologist, KCMC

Dr. Isaac Lyaruu
Physician, KCMUCo

Dr. Gloria Temu
Physician, KCMC

Dr. Mike Maze
Pulmonologist, KCMC

Dr. Amos Mwasamwaja
Physician, KCMC

Dr. Gissela Nyakunga
Physician, KCMC

Dr. Josephine Rogath
Resident Internal Medicin, KCMUCo

Dr. Pantaleo Joseph
Resident Internal Medicine, KCMUCo

Dr. Elichilia Shao
Resident Internal Medicine, KCMUCo

Dr. Neema Minja
Registrar Internal Medicine, KCMC

Dr. Sharma
Resident, Internal Medicine DUKE

Dr. Monica
Resident Internal Medicine, DUKE

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CLERKING GUIDELINE

Introduction
These guidelines are not intended to replace the standard textbooks on history taking and physical examination. Neither are these guidelines intended to remove creativity in getting information used to arrive to a diagnosis. These aim to standardize clerkship in the department of internal medicine and thus give guide to the minimal required data.

The following subheadings will be required

1.0 HISTORY
2.0 PHYSICAL EXAMINATION
3.0 SUMMARY
4.0 PROBLEM LIST/ DIAGNOSIS
5.0 INVESTIGATIONS
6.0 TREATMENT
    6.1 METHOD OF FEEDING
    6.2 MEDICATION
    6.3 FLUIDS
    6.4 PHYSICAL THERAPY
    6.5 SPECIAL NURSING CARE

1.0 HISTORY the general scheme would be as follows:
Basic data, presenting complaint, History of presenting complaint, Review of other systems, past medical history including, gynecological and obstetric history, Drugs and allergy history, Personal, Family and Social history
Basic data. Name, address including contact telephone, Age, gender, ethnicity, Occupation, date and time of admission and name of admitting officer
Presenting complaint (c/o) Major symptoms (up to 3) and duration
History of presenting complaint Amplify the details of the presenting complaint. Onset, Duration, frequency, Site, Radiation, Severity, Character, Periodicity, Precipitating and relieving factors
Review of other systems General, Cardiovascular, Respiratory, Gastrointestinal, Genito-urinary, Central nervous system, locomotor system, Endocrine and psychiatric/mental health.
Past medical, gynecological and obstetric history Past serious medical illness, Surgeries, accidents, gynecological hx and pregnancies.
Drugs (duration and reasons). Allergies and if HIV tested before.
Family and social history (PH and SH) Marital status, number of children, Alcohol consumption, Tobacco consumption, present and past occupations, circumstance and conditions at home and recent travel. Family history: number and health of siblings, cause of death of parents, diseases in family: diabetes, thyroid, hypertension, heart attack, stroke or asthma.

2.0 PHYSICAL EXAMINATION

General examination followed by systemic examination

2.1 General examination Appearance, pallor, cyanosis, jaundice, clubbing, lymphadenopathy, peripheral edema, oral exam. Vital signs BP, PR, TEMP, RR. So2. Local examination if indicated.

2.2 Cardiovascular Pulse, JVP, carotid pulses, apex beat (position and character), precordium (impulses- thrills, heaves), HS I &II, murmurs and added sounds, peripheral pulses and fundi.

2.3 Respiratory shape of chest, trachea, chest movement, expansion, TVF, percussion note, breath sounds, vocal resonance and added sounds.

2.4 Abdomen shape, Scars, movements, Hernia orifices, tenderness/guarding, Liver/Kidneys/spleen, Masses, Bowel sounds, external genitalia and DRE.

2.5 Central nervous system conscious level, orientation, memory, speech, gait, handedness R/L, signs of meningeal irritation, cranial nerves, upper limbs (Tone, Power, Reflexes, Coordination, Sensation). Lower limbs (Tone, Power, Reflexes, Coordination, Sensation and Babinski).

2.6 Locomotor joint swelling, erythema, deformity and muscle wasting. warmth, tenderness, range of movement, instability and crepitus.

3.0 SUMMARY not more than 5 sentences including the important negatives and positives.

4.0 PROBLEM LIST diagnoses and differential diagnoses

In case a symptom/sign has no diagnosis yet, it may be placed as a symptom/sign. *E.g. Ascites cause undetermined*

5.0 PLAN

- 5.1 INVESTIGATIONS
- 5.2 TREATMENT
  - 5.2.1 FEEDING METHOD
  - 5.2.2 MEDICATION
  - 5.2.3 FLUIDS
  - 5.2.4 PHYSICAL THERAPY
  - 5.2.5 SPECIAL NURSING CARE

CARDIOPULMONARY RESUSCITATION

Table 1: BASIC LIFE SUPPORT (BLS) AND ADVANCED LIFE SUPPORT

STEP	ASSESSMENT	ACTION	
Patient changed condition?			
1.	Is the patient able to answer you?	Call him and tap Observe for absent breathing or gasping ➔ If patient not responding ➔ Call for help ➔ Call for defibrillator	
2.	Check for pulse	➔ if present provide AMBU 1 breaths every 5 sec ○ Check pulse every 2 min ➔ If absent go to 3	
3.		PR 30:2 for two minutes rhythm after two minutes of CPR er advance airway as soon as possible.	
4.	Rhythm by defibrillator/monitor	**Asystole/PEA** ue with CPR drenalin 1mg iv. every 3-5min pulse and rhythm every two minutes	**VT/VF** ne shock ue CPR for two minutes 30:2 pulse rhythm every two minutes ➔ If shockable, shock again (max 3 shocks) ➔ After the 2nd shock give epinephrine ➔ After 3rd shock amiodarone 300mg bolus
5	For reversible causes	**5H-5Ts** Hypovolemia Hypoxia Hydrogen ion (acidosis) Hype/Hypokalemia Hypothermia Tension pneumothorax Tamponade Toxins Thrombosis pulmonary Thrombosis coronary	
6	Signs of return of spontaneous circulation	**YES** CPR for one minute Post cardiac arrest care	**NO** Continue CPR for 30 minutes

nce: Advanced Cardiovascular Life Support, American Heart Association. 2011

CARDIOVASCULAR SYSTEM

SHOCK
HEART FAILURE
ATRIAL FIBRILLATION
MYOCARDIAL INFARCTION
HYPERTENSIVE EMERGENCIES

SHOCK

Definitions: Cell and tissue hypoxia due to reduced oxygen delivery, increased oxygen consumption, and/or inadequate oxygen utilization.

Differential Diagnosis:
Chronic hypotension
Drug-induced hypotension
Autonomic dysfunction
Vasovagal syncope
Peripheral vascular disease

Clinically defined as
Tachycardia HR >100
Tachycardia HR >100
Hypotension BP < 90/60
Altered mental status
Decreased urine and cold extremities
Altered mental status
Decreased urine output

Goals of Management:

- Rapid assessment for signs of shock
- Identify different types of shock
- Provide optimal therapy for patients in shock

Approach flowchart
- See chart below

Further Management Details

Hemodynamic support:
- Intravenous fluids
    - Repeat normal saline boluses until blood pressure MAP > 60 and tissue perfusion adequate. Should monitor BP and volume status (listen to their lungs) before and after each bolus. If hemorrhagic, prefer blood.
        - MAP = (SBP + 2 x DBP)/3
    - Obstructive usually require small volumes (250 ml at a time). This is not definite treatment; the cause must also be treated.
    - Distributive and hypovolemic usually require large volumes (1L initially, then 500 mL each bolus).

Vasopressors: Second line if not responsive to fluids, or in cardiogenic shock
- See table below

Diagnostic Approach.

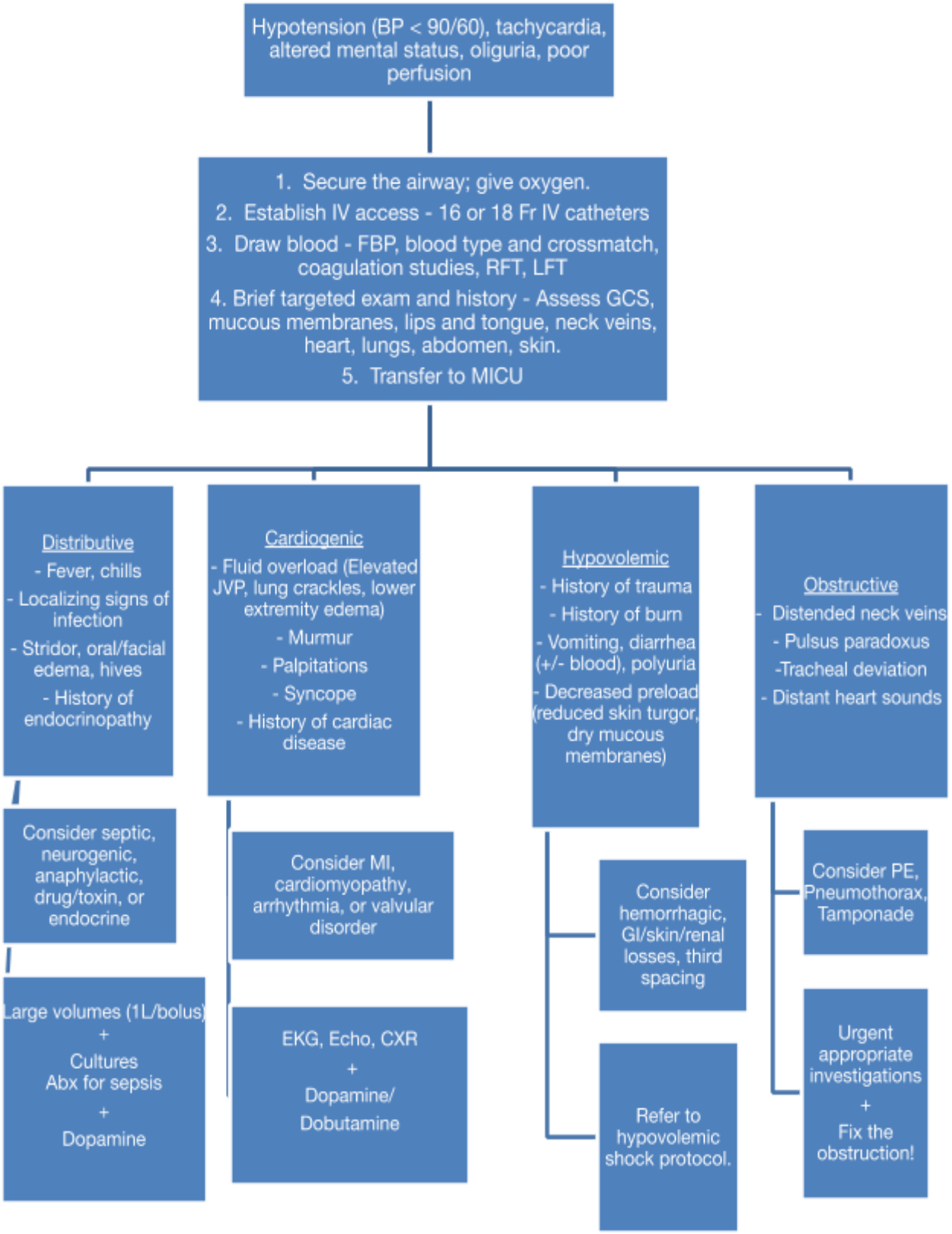


Table 2: Vasopressors

Drug	Dose	Best Use
Dobutamine	Start at 2.5 mcg/kg/min, Max 20 mcg/kg/min Add 200 mg/250 mL of NS For 70 kg person, Start at 5 drops/minute Adjust by 5 drops q 20 min to achieve MAP >60 Takes 16.5 hours per bolus	Cardiogenic Shock
Dopamine	Start at 5 mcg/kg/min, Max 20 mcg/kg/min Add 200 mg/250 mL of NS For 70 kg person, 9 drops/minute Adjust by 2 drops q 20 min to achieve MAP >60 Takes 9 hours per bolus	Cardiogenic Shock Septic Shock Obstructive Shock
Epinephrine	Start at 0.05 mcg/kg/min Max 2 mcg/kg/min Add 1 mg in 250 ml of NS For 70 kg person, 18 drops/minute Adjust by 10 drops q 20 min to achieve MAP > 60 Takes 5 hours per bolus	Septic Shock Anaphylactic Shock

* For calculations, 1 drop = 0.05 mL

Consider urgent appropriate therapies:

- Needle thoracostomy/Chest tube for tension pneumothorax
- Pericardiocentesis for cardiac tamponade
- Epinephrine for anaphylaxis (0.3 mg IM every 5 minutes as needed)
- IV steroid for adrenal crisis (Hydrocortisone 100 mg IV)
- Surgical consultation for hemorrhage

Continued management:

- Continue hemodynamic support and monitor response (BP, HR, urine output) in ICU
- Reverse the etiology.

Septic Shock:

- Rapid fluid resuscitation: Mean Infusion Volumes of 3-5L. Well-defined rapidly infused boluses. Assess blood pressure and volume status before each bolus. Repeat until blood pressure and tissue perfusion are acceptable. Monitor for ARDS.
- Vasopressors: Second-line agents. Indicated if hypotensive despite adequate fluid resuscitation or those who develop pulmonary edema.
- Blood transfusion for patients with a Hb < 5
- Consider IV steroids (IV Hydrocortisone 100 mg q6h) if not responding to pressor
- Infection Control:
    - Identify focus (Urinalysis, Urine culture, Sputum Culture, Blood Culture, MRDT, Stool culture, HIV serology)
    - IV Antibiotics: Ceftriaxone 1 g IV BD.

Cardiogenic Shock

- Treat arrhythmia if present
- Inotropes: Refer to table above.
- Diuretics: Once blood pressure is adequate.
    - Furosemide 40-120 mg IV. Re-dose based on response – double dose until diuresis ensues.
    - Monitor electrolytes, renal function.
- Consider dialysis if unable to diuresis successfully.
- If in respiratory distress, consider mechanical ventilation
- Sodium restriction (<2 g/day) and Fluid restriction (1.5-2L/day)
- Monitor ins and outs, daily weights. Consider urinary catheterization for reliable output.

HEART FAILURE

Definition

Heart failure is a clinical syndrome that results from impaired structure or function of ventricular filling or ejection of blood, which in turn leads to the cardinal clinical symptoms and signs of dyspnea, edema and crepitation.

Classification

Heart failure was once thought to arise primarily in the setting of reduced left ventricular ejection fraction (EF) but studies have shown that almost half who develop HF have preserved EF (EF ≥50%). HF patients are now broadly categorized into:

- HF with a reduced EF (HFrEF; formerly *systolic failure*)
- HF with a preserved EF (HFpEF; formerly *diastolic failure*).

Causes

Reduced Ejection Fraction (EF <40%)	Coronary artery disease Hypertension Regurgitant valvular disease Intracardiac (left-to-right) shunting Chronic lung disease (Cor pulmonale) Nonischemic dilated cardiomyopathy Toxic/drug-induced damage Chronic brady-/tachy-arrhythmias
Preserved Ejection Fraction (EF >50%)	Hypertrophic/ Restrictive cardiomyopathy Hypertension Aging Fibrosis Endomyocardial disorders
High Output States	Thyrotoxicosis Beriberi Chronic anemia

Clinical Presentation

Patients with HF generally can present with symptoms of difficulty in breathing, cough, chest pain, difficulty in laying flat, air hunger at night and lower limb swelling.
Upon physical examination, the patients can present with the following signs:

Features of Right Heart Failure:	Features of Left Heart Failure:
Raised JVP	Raised JVP
Hepatomegaly	Pulmonary edema
Ascites	Cardiomegaly
Peripheral edema	Pleural effusion
	Peripheral edema

Diagnosis

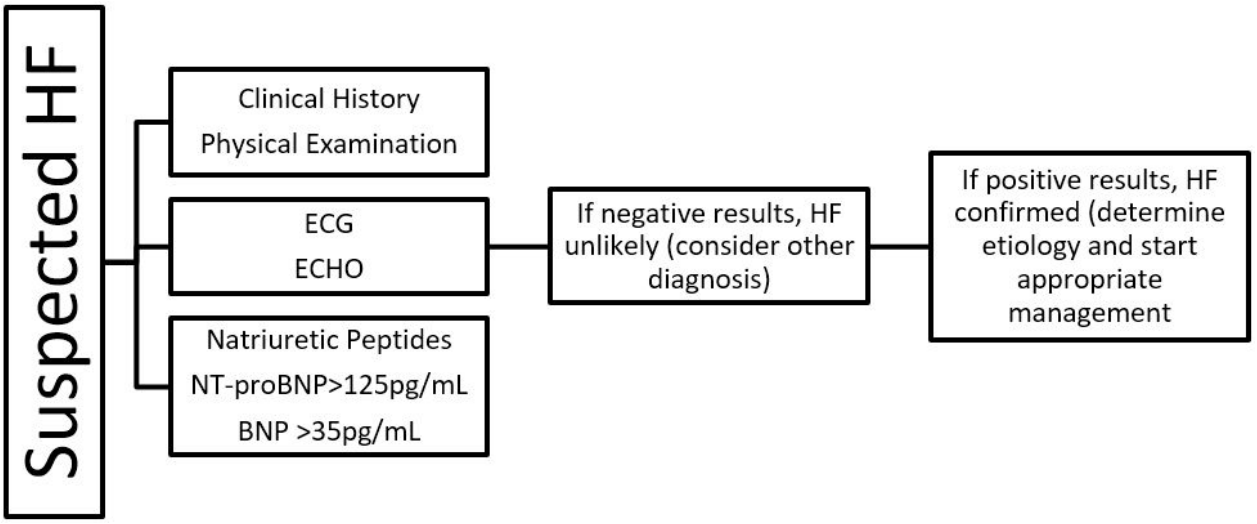
The diagnosis is HF is made clinically. By using the Framingham Heart Failure Diagnostic Criteria, selecting 2 major criteria or 1 major and 2 minor criteria, which will help make that diagnosis.

Major Criteria:	Minor Criteria:
Acute Pulmonary Edema	Ankle Edema
Cardiomegaly	Dyspnea on Exertion
Distended Neck Veins	Hepatomegaly
Hepatojugular Reflux	Nocturnal Cough
Paroxysmal Nocturnal Dyspnea/Orthopnea	Pleural Effusion
Pulmonary Rales	Tachycardia (>120bpm)
Third Heard Sound (S3 Gallop)	

Investigations

Further to the clinical diagnosis, all heart failure patients should be investigated further by ordering the following investigations:

- Full blood picture
- Serum electrolytes (sodium and potassium)
- Serum urea
- Serum creatinine
- Serum lipid profile
- Chest x-ray
- Electrocardiogram
- Echocardiogram



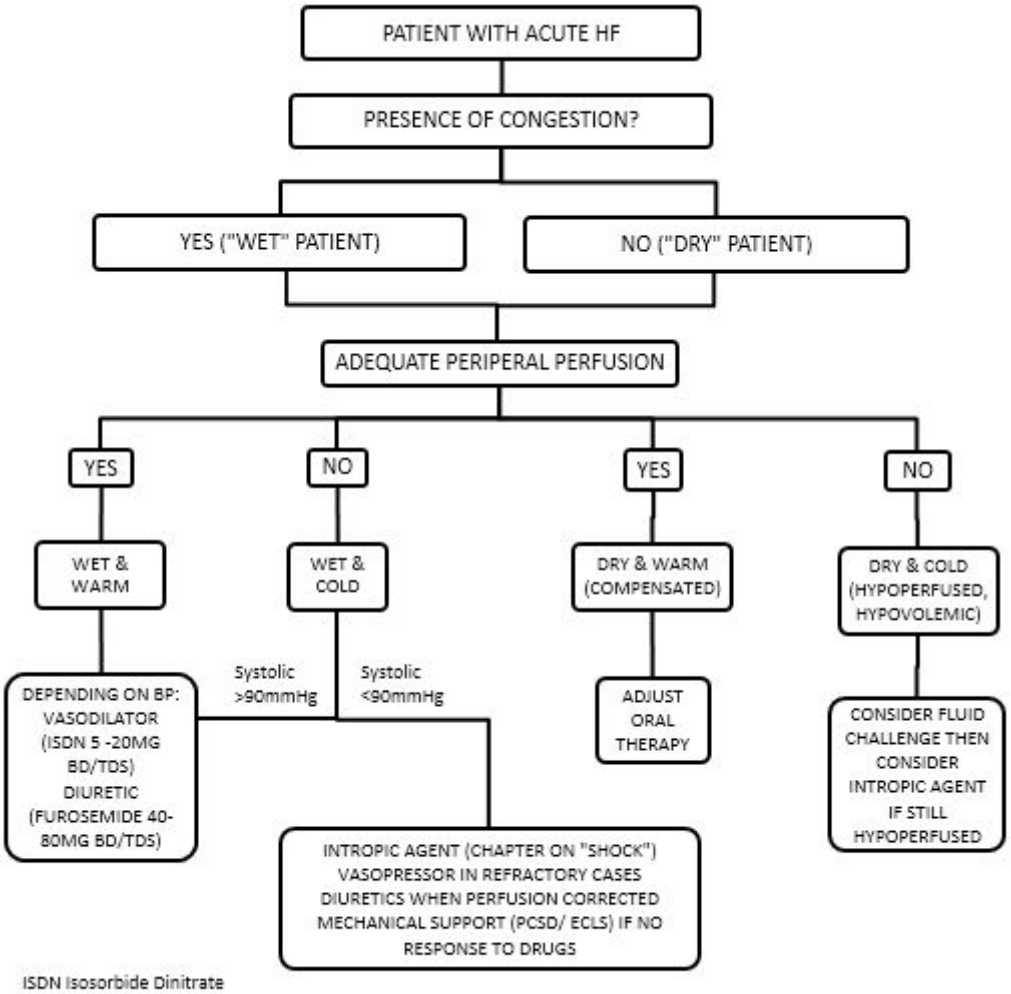
Goals of Therapy

- To treat the underlying cause
- To decrease the likelihood of disease progression
- To lessen symptoms
- To improve the quality of life

Initial Management

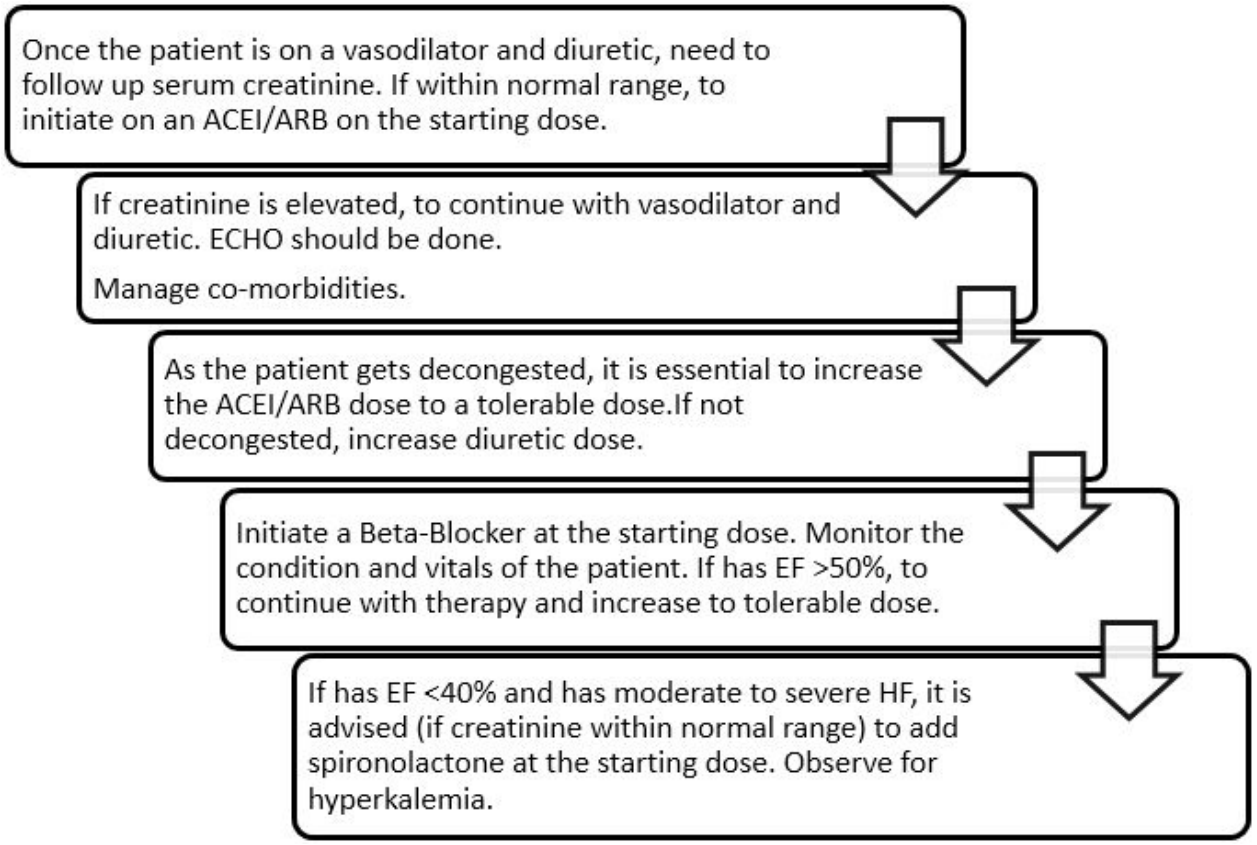
Firstly, to triage the patient and identify if patient is need of oxygen and put the patient on oxygen via nasal prongs or face mask if mouth-breathing, to achieve an oxygen saturation of 94% and higher.

Put the patient in a comfortable position between 45° and 90°



ISDN Isosorbide Dinitrate
PCSD Percutaneous Cardiac Support Devices
ECLS Extracorporeal Life Support

Management



Drug Therapy

Drug Therapy	Starting Dose	Target Dose (mg)
ACEI		
· Captopril	6.2mg TDS	50mg TDS
· Lisinopril	2.5mg OD	20mg OD
· Enalapril	2.5mg BD	20mg BD
ARB		
· Losartan	50mg OD	150mg OD
· Telmisartan	20mg OD	160mg OD
· Candesartan	4mg OD	32mg OD

Beta-Blocker		
· Carvedilol	3.125mg BD	25mg BD
· Bisoprolol/Nebivolol	1.25mg OD	10mg OD
· Metoprolol	12.5mg OD	200mg OD
MRAs		
· Spironolactone	25mg OD	50mg OD
Diuretics		
· Furosemide	20mg OD	80mg TDS
· Torsemide	5mg OD	10mg BD
· Bendroflumethiazide	2.5mg OD	10mg OD
· Hydrochlorothiazide	25mg OD	100mg OD
· Metolazone	2.5mg OD	10mg OD

Note: Increase the dose that is tolerable for the patient. When increasing drug doses to target/tolerable doses, it should be done one drug at a time with increments done 2 weeks apart.

<u>Lifestyle Modification</u>

- Fluid restriction of 1.5–2L/day may be considered with severe HF to relieve congestion
- Eat healthily, avoid excessive salt intake (>6 g/day) and maintain a healthy body weight
- Regular moderate exercise of 150 minutes/week
- Regular body weight monitoring at home and at the hospital
- Stop smoking
- Avoid excessive alcohol intake

Note: Drugs that should be avoided or used with caution in patients with heart failure

- NSAIDS (COX-1 & -2)
  - Prostaglandin inhibition leading to sodium and water retention, increased systemic vascular resistance, blunted response to diuretics, renal dysfunction and hyperkalemia (except low dose aspirin/clopidogrel which is indicated in HF only if cause is CAD)
- Calcium Channel Blockers (nifedipine, verapamil & diltiazem)
  - Negative inotrope effect (except for amlodipine)
- Trimethoprim-sulfamethoxazole

Increased risk of hyperkalemia and AKI in patients taking ACEI/ARB/MRA

- Antifungals (itraconazole & amphotericin B)
  Negative inotrope effect
- Thiazolidinediones (pioglitazone)
  Cause fluid retention and possible calcium channel blockade
- Metformin
  Increased anaerobic metabolism and elevated lactic acidosis
- Dipeptidyl peptidase-4 inhibitors (sitagliptin)
  Exacerbate cardiac and renal dysfunction
- Salbutamol (oral)
  Decreased β -receptor responsiveness with increased exposure
- Antidepressants (amitriptyline & fluoxetine)
  Negative inotrope and proarrhythmic properties
- Carbamazepine
  Negative inotrope & chronotrope effect
- Pregabalin
  Calcium channel blockade
- α 1-blockers (Tamsulosin)
  β 1 receptor stimulation with increase in renin and aldosterone
- Phosphodiesterase-5 inhibitors (sildenafil & tadalafil)
  Has vasodilator effect and contraindicated in use with other vasodilators because can lower pulmonary and systemic arterial pressure
- Chemotherapy agents (doxorubicin, cyclophosphamide and bevacizumab)
  Prolonged oxidative stress
- Sodium bicarbonate
  Increased risk of acute phosphate nephropathy in patients using ACEI

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ATRIAL FIBRILLATION

Definition: irregular atrial electrical stimulation causing mechanical atrial dysfunction, irregular and rapid ventricular stimulation and reduced cardiac output.

Treatment goals:

1. Relieve symptoms (rate and rhythm control)
2. Prevent complications (thrombo-embolism)

Clinical presentation:

Conditions associated with AF	Increased age, Hypertension, Diabetes mellitus, Valvular heart disease, Heart failure, Myocardial infarction, Alcohol use, Cardiomyopathy and Hyperthyroidism.
Symptoms	Asymptomatic (many patients), Breathlessness, Palpitations, Dizziness/syncope, Stroke/TIA
Physical examination findings	Irregularly irregular pulse, Pulse deficit
Investigations	ECG (absence of P waves and irregularly irregular rhythm), CXR, ECHO, baseline blood tests as needed

Management

1. Relieve symptoms- Rate and rhythm control:

Unstable patient: (hypotension, confusion, chest pain, acute pulmonary edema)
Supportive care- Support airway, give oxygen, provide cardiac bed and admit in MICU.

Rate control-consider when unknown duration of AF or >48 hours since onset	
1st line Beta blocker	IV Metoprolol loading dose of 5mg (over 3-5min) 3 doses, then maintenance dose of 1.25-5mg q6h, then maintenance oral dose of 25-100mg BD or 50-400mg OD.
Non-dihydropyridine calcium channel blocker	-IV Verapamil loading dose of 5-10mg (over 3-5min) then maintenance dose of 2.5-10mg/h.
2nd line Digoxin	Give 0.25mg IV start, then repeat 8hourly for 24hours, then maintain with oral 0.125-0.25mg OD.
Rhythm control (Cardioversion (electrical or pharmacological) Consider especially if new onset AF < 48 hours duration, or if >48hours duration has been on therapeutic anticoagulation for at least 3weeks, or life threatening hemodynamic instability. Pharmacological cardioversion	
Structural heart disease	Amiodarone 360mg IV (over 6hrs) or 150mg over 10minutes, then 0.5-1M g/min. Oral 100-200mg OD.
Electrical cardioversion: - 120-200 joules biphasic or 200joules monophasic	

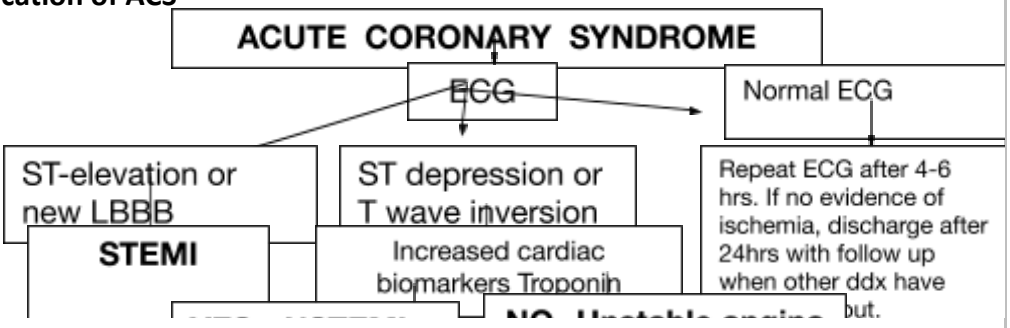
Stable patient/ long term management:

Rate control First line strategy, except if: Reversible cause of AF or rhythm control strategy found to be more suitable based on clinical judgement	1st line beta blocker (Carvedilol 3.125-25mg BD, Atenolol 25-100mg OD, Propranolol 10-40mg TID or QID, Bisoprolol 2.5-10mg OD, and Metoprolol 25-100mg BID) Non dihydropyridine calcium channel blocker (Verapamil 180-480mg OD and Diltiazem 120-360mg OD) 2nd line -digoxin (0.125-0.25mg OD) If monotherapy insufficient, consider combination of either two of beta blocker, digoxin or diltiazem
Rhythm control Considered if symptoms persist despite rate control, or rate control strategy unsuccessful	Cardioversion (electrical or pharmacological) -If AF duration ≥48hours, do TOE prior to cardioversion or anti-coagulate for 21 days before cardioversion (meanwhile, maintain rate control)
Pharmacological	**No structural heart disease** (class 1 anti-arrhythmic drugs) Flecainide - 50-200mg BD Propafenone- 150-300mg 8hr Dysopiramide-100-300mg 6-8hr **Structural heart disease** (class 3 anti-arrhythmic drugs) Sotalol-80-160mg BD Dofetilide oral 0.125-0.5mg BD, use for prevention (do ECG monitoring) Amiodarone (400-600mg daily in divided dose for 2-4wks, maintenance dose of 100-200md OD).
Electrical	120-200 joules biphasic or 200joules monophasic.

2. Prevent complications- Intervention to prevent stroke:

Assess risk of stroke	-AF in valvar heart disease- offer anticoagulation -CHA2DS2VASc score for risk assessment in non-valvar AF. If score ≥2- offer anticoagulation
Assess risk of bleeding	-HAS-BLED score to assess risk of bleeding. If score ≥3, high risk of bleeding.
Preparation for anticoagulation	-Ensure BP is controlled if hypertensive -Avoid concurrent NSAID use -Counsel patient to avoid harmful consumption of alcohol -Baseline INR check
Anticoagulation	-Before anticoagulation, counsel the patient first. **Warfarin**- starting dose 5mg p/o od, therapeutic goal INR 2-3 -Check INR after 3-5 days, then two weekly until INR therapeutic and stable. Then INR monthly. -Regular review of need for anticoagulation and bleeding risk

ACUTE CORONARY SYNDROME

Definition	Spectrum of diseases consisting of STEMI, NSTEMI and unstable angina.
Treatment goals	• Pain relief • Rapid identification of patients who are candidates for urgent reperfusion therapy • Early antiplatelet therapy • Institute appropriate long-term management for primary and secondary prevention. • Maintain oxygenation
Clinical presentation • Chest Pain typically *heavy*, *squeezing*, or *crushing*. Retrosternal or left sided, radiating to the left shoulder, left arms, jaw or neck. Usually lasting >20minutes. • May have prior history of stable angina. • Often have background of coronary risk factors – smoking, obesity, sedentary life, diabetes, hypertension and dyslipidemia etc. • Atypical presentation is common for diabetics, elderly and women**.**	
Classification of ACS 	
Other investigations. CXR, ECHO, Lipid profile, serum creatinine, S. potassium, Sodium. Coronary angiography	
Management	
General measures	• Nurse in intensive care unit • Oxygen therapy (so2>94%)

Pharmacotherapy	
Emergency management Aspirin-300 mg tablet chewable, followed by 75mg daily and Clopidogrel 300mg followed by 75 mg/day.	
Control discomfort	
Nitrates	If hypotensive withhold nitrates. Sublingual glyceryl trinitrate (GTN) 0.4 mg stat, can be repeated at 5-min intervals maximum 3 doses/24hrs, or oral isosorbide dinitrate 20mg, 2–3 times daily
Morphine	If patient in pain despite nitrates Loading dose 5-10mg iv. STAT Maintenance dose 5mg 6hourly
Statins	Atorvastatin 80mg stat, then 40mg po daily.
ACE inhibitor	
Beta blockers	Should be considered within 24hrs. Carvedilol 12.5mg twice daily. Metoprolol 50 mg every 6 h for 48 h, followed by 100 mg every 12 h. ***Avoid*:-** AV block, HR <60 bpm, SBP <90 mmHg, Shock, Left ventricular failure, Severe reactive airway disease.
Antithrombotic	
Anticoagulant	Prophylaxis for all patient unless contraindicated 1) Heparin (UFH) - Bolus 60–70 U/kg (maximum 5000 U) sc 12hrly. 2) LMWH (Enoxaparin) 40mg sc 12hrly.
Definitive management	
STEMI	Within 12 hrs. of symptoms consider reperfusion therapy
NSTEMI	Refer for coronary angiography when stable.
UA	

Long-Term Management

- Risk factor modification - smoking cessation, achieving optimal weight, daily exercise following an appropriate diet, blood-pressure control, tight control of hyperglycemia (for diabetic patients), and lipid management.

- Benefit with long-term therapy with five classes of drugs (Beta blockers, statins (at a high dose, e.g., atorvastatin 80 mg/d), and ACE inhibitors or angiotensin receptor blockers and Antiplatelet therapy (aspirin and clopidogrel or prasugrel).

HYPERTENSIVE EMERGENCIES

ITION e hypertension (>180/120mmHg) associated with acute end-organ damage which could include one of the following: ▪ Malignant hypertension ▪ Hypertensive encephalopathy ▪ Subarachnoid or intra-cerebral hemorrhage ▪ Acute pulmonary edema ▪ Aortic dissection ▪ Acute renal Failure ▪ Pre-eclampsia/eclampsia (BP may be below above cut off) AL PRESENTATION toms and signs will depend on which end organ is involved. Fundoscopy **MUST** be done	GOAL OF THERAPY ● To lower the DBP by 25% ● Note: maximum initial fall in BP not exceeding 25%of the presenting value INVESTIGATIONS ● Creatinine/BUN ● Electrolytes ● Urine dipstick ● ECG ● CXR ● CT-scan if indicated Others: ● FBP ● Blood sugar ● Lipid profile (Total cholesterol, HDL/LDL)

TREATMENT

A. Supportive care
   - Maintain Airway, Breathing, and Circulation
   - Give oxygen therapy where indicated
   - Get an Iv access
   - Monitor vitals and Input/output closely
   - Admit in ICU or Sub-ICU

B. DRUGS
   1. Labetalol - an alpha-and β -adrenergic blocker, iv. Bolus or infusion.
      - Bolus: 20mg initially, followed by 20 to 80 mg every 10minutes to a total dose of 300 mg.
      - Infusion: 0.5to 2 mg/min
      - Safe in patients with active coronary disease
   2. Nicardipine-calcium channel Blocker, given as an intravenous infusion.
      - Initial dose:5 mg/h; maximum dose 15mg/h.
      - Reduce both cardiac and cerebral ischemia.
   3. Hydralazine- an arteriolar dilator
      - 20mg/in 5% dextrose or DNS over 8hours
      - ***To be administered cautiously***

4. Propranolol- a β -adrenergic blocker,
    - Given as an intravenous and then followed by oral therapy
    - Dose: 1 to 10 mg load, followed by 3mg/h.
5. Enalapril- an ACE-inhibitor, given as an intravenous bolus
    - Dose:1.25mg every 6 hours
    - Contraindicated in pregnancy
6. Esmolol is an ultra-short-acting, cardio selective B-adrenergic blocking agent
    - Onset of action within 60s, with a duration of action of 10-20min
    - Useful in severe postoperative hypertension, acute myocardial infarction.
    - Dose 0.5-1mg/mg loading dose over 1min, followed by an infusion starting at 50µg/kg per min and increasing up to 300µg/kg per min. as necessary.

HYPERTENSIVE URGENCIES

- Severe hypertension (BP>180-120mmHg), with no acute sign of end –organ damage
- Initial goal, reducing the blood pressure to <160/100mmHg over several hours to days with conventional oral therapy.
- Previously treated patients
    - Adjusting medication regimen, or
    - Reinstituting medications (if non adherent).
    - Previously untreated patients

  Initiate ant-hypertensive
    - The choice of agent should take into consideration patient characteristics and co-morbid conditions
    - Initial dose should be taken immediately and observe an initial dro of BP over the next few days

References
1. 2013 ESH/ESC guidelines for hypertension
2. http://jama.jamanetwork.com/article.aspx?articleid=1791497

RESPIRATORY SYSTEM

RESPIRATORY FAILURE
ACUTE ASTHMA
COPD EXACERBATION
TUBERCULOSIS DIAGNOSIS
PNEUMONIA
PLEURAL EFFUSION

RESPIRATORY FAILURE

Definition/ Rationale: Respiratory failure can be either:
Type 1: Hypoxia with normal carbon dioxide: most patients
Type 2: Hypoxia with elevated carbon dioxide: those with neuromuscular disease, severe COPD and the morbidly obese. Those with purely hypoventilation (such as neuromuscular disease) need help to ventilate – rather than extra oxygen.

Goals of therapy:

1. Treat the underlying disease - respiratory failure is an emergency. Rapidly attempt to make a diagnosis and begin specific therapy
2. Support oxygenation and ventilation in the patient during recovery.

Oxygen
Oxygen is a drug that should be prescribed to maximise benefit and minimise unnecessary use and harm. It is a scarce resource, so prescribe to only those that will benefit. Oxygen should be given to treat hypoxaemia. Conversely oxygen does not relieve breathlessness in the absence of hypoxaemia.

Oxygen saturations: Pulse oximeters give an indication of arterial oxygen saturation. It is a vital sign and should be recorded on the observations chart.
In critically ill patients, pulse oximetry can be inaccurate, especially when there is poor peripheral circulation. Low oximtery should make you evaluate both the cardiovascular and respiratory systems.

Target saturations from pulse oximetry:
Chronic Respiratory Failure: Oxygen saturations of 88-92%
Acute Medical Conditions: 92-96%
Patients with oxygen saturations above these levels generally do not benefit from oxygen.

Oxygen toxicity:
Patients who are chronically hypercapnic rely on a hypoxic respiratory drive. Too much oxygen can cause them to under breathe. You need to have caution in those with type 2 respiratory failure, but giving oxygen to the targets above should prevent problems in most situations.

Oxygen delivery:

Method of Delivery	Flow Rates	FiO_2
Room Air	N/A	21%
Nasal Cannulae	1-5 L	24-35%
Simple face mask	6-10 L	36-60%
Non-rebreather mask	10-15L	60-80%

Indications for Intubation and Ventilation
Patients requiring intubation generally require ventilation and vice versa
Indications for Intubation: An unprotected airway
Indications for ventilation: Hypoxia, hypoventilation, unacceptably high work of breathing
Contra-indications for intubation/ ventilation: Non-reversible endstage pathology to either the pulmonary system or any other vital organ system (eg massive stroke, end-stage renal failure without approval for dialysis)

Remember: Mechanical ventilation carries a high mortality from the underlying disease and complications of ventilation. Consult a senior before ventilating if at all possible.

Assess the airway: Is the patient talking are there signs of airway obstruction such as snoring gurgling or stridor?

If airway obstructed, tilt the head and lift the chin as shown in the diagram. Remove any visible foreign body and clear secretions.
Then maintain the airway by inserting an oropharyngeal airway as shown

Figure 1. How to perform head tilt and chin lift

Figure 2. How to use an OP airway

Assess breathing: If breathing and measure SpO_2. If breathing and SpO_2<88% in those with chronic lung disease or 92% in those without, give oxygen

Figure 3. Use of nasal prongs

Give oxygen at 4-5 L/minute via nasal prongs. REASSESS the

If SpO_2<90% then increase the flow rate to 6-10L/minute via a face mask

Figure 4. Use of a face mask

If SpO_2<90% then increase flow rate to 10-15L/minute via non-rebreather face mask

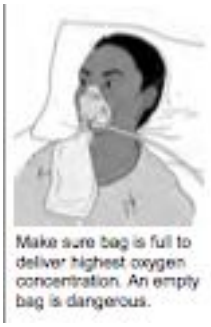


Figure 5. Use of a rebreather mask

If SpO_2 <90% and there is a reversible condition – call a senior to consider ventilation

ACUTE ASTHMA

Diagnosis: Look for evidence of airflow obstruction – hyperexpansion, prolonged expiration and wheeze on auscultation.

Assess Severity:

Look for evidence of severe or life-threatening asthma

Severe	PEF 33–50% best or predicted Respiratory rate ≥25/min Heart rate ≥110/min Inability to complete sentences in one breath
Life-Threatening	Altered consciousness Exhaustion Hypotension (SBP<90 or DBP<60mmHg) Cyanosis Silent Chest Poor Respiratory Effort

Investigations

Measure Peak expiratory flow rate on admission, and then 2-3 times/ day
For mild-moderate asthma when the diagnosis is clear-cut no blood tests or x-rays are needed.
Full blood count, Creatinine, Urea, Na and K+ for severe or life threatening asthma
Arterial blood gas – for severe or life threatening asthma only
CXR – any patient with suspected pneumonia, pneumothorax or severe/ life threatening asthma. It is not required for patients with less unwell patients with clear cut asthma

Management:

Make an initial assessment of severity and **then REASSESS after beginning therapy**

Mild-Moderate	Salbutamol 100mcg inh 2-4 puffs via a spacer PRN Prednisone 40mg PO od for 3 days then 20mg od for 3 days
Severe	Obtain IV access Salbutamol 5mg nebulised Hydrocortisone 100mg IV Q6H, with switch to Prednisone (regimen as above) when improving Oxygen , ideally to keep SpO_2>94%
Life-Threatening	Transfer to the ICU and consult specialist Salbutamol 5mg nebulised P5mg Q15min (can be continuous) + Ipratropium bromide 0.5mg Q6H Obtain IV access Hydrocortisone 100mg Q6H IV Oxygen (ideally to keep SpO_2>94%) If not improving: Magnesium Sulphate 2g IV stat IV Salbutamol Ventilation – if impending respiratory failure, see separate guideline and discuss with your senior

Antibiotics are only needed if there is clinical or X-ray evidence of pneumonia

If no Salbutamol: Aminophylline is a less good alternative to Salbutamol but can be used if salbutamol is unavailable. Dose: 250mg (6mg/kg) IV over 20min (MUST be given slowly), then 0.3-0.6mg/kg/hour (eg 250mg in 500ml fluid over 8-12 hours)
Do NOT give bolus if already on aminophylline/ theophylline

Ongoing Care:

Monitor Symptoms, Peak expiratory flow rate and vital signs
In those with mild/ moderate disease this can be 3 times per day, but must be more frequent (2-4 hourly) in those with severe or life threatening asthma
As improving change nebulised medication to inhaled via a spacer and all IV medication to oral.

Begin Outpatient therapy
Begin inhaled corticosteroid inhaler. Currently available medications are:
Beclomethasone 50mcg/100mcg – Use 200mcg bid
Budesonide 100/200mcg – Use 200mcg bid
Use Salbutamol 100mcg inhaler 2 puffs bd to relieve symptoms

If patients are unable to afford inhaled medications, use the (less good) alternative: Salbutamol 4mg PO prn for dyspnoea

Teach inhaler technique.
Develop an action plan (See below) – Every asthma patient MUST leave with an action plan.
Follow up in MOPD in 1 month with spirometry

Spacers
These are essential for all asthmatics to ensure the inhaler reaches to the medium and small airways of the lung. Teach your patients how to use and care for them.
Spacers can be made out of old 500ml bottles
YOU CAN make one for your patients by using the following instructions:
www.ajol.info/index.php/samj/article/download/13678/15737

Using a spacer
1. **Shake the inhaler whilst holding upright**
2. **Fit the inhaler into the opening fashioned at the bottom of the bottle spacer**
3. **Seal lips around top of the bottle spacer**
4. **Press the inhaler once**
5. **Take 2—4 slow breaths**
6. **Repeat for further doses**

Washing a spacer
1. **This should be done weekly**
2. **Wash with warm water and dishwashing liquid.**
3. **Do NOT rinse**
4. **Leave to drip dry**

Asthma Action Plan

Symptoms	Plan
Your asthma is under control when ● you don't have asthma symptoms most days (wheeze, tight chest, breathlessness, or a cough) ● you don't wake at night with asthma symptoms ● you can continue with all your usual activities ● you use a reliever **less than** 3 times per week	Use ICS every day and salbutamol PRN
Caution – your asthma is getting worse when ● you are waking at night with asthma symptoms; or ● you are very breathless or wheezy; or ● exercise or daily activities are becoming difficult because of asthma symptoms; or ● you are using more reliever than usual; or ● your reliever lasts a much shorter time	Double the dose of inhaled corticosteroid Continue to use salbutamol as needed If worsening or not improving: visit nearest clinic or KCMC for assessment
EMERGENCY ● you have severe breathlessness; or ● you are finding it hard to speak; or ● you feel faint or are frightened; or ● your reliever is not working	Come immediately to KCMC or nearest health centre for treatment.

References

1. BTS-SIGN British guideline on the management of asthma, Thorax 2014;69:Suppl 1 i1-i192
2. National Asthma Council Australia. Australian Asthma Handbook, Version 1.1. National Asthma Council Australia, Melbourne, 2015. Website. Available from: http://www.asthmahandbook.org.au

COPD EXACERBATION

Diagnosis:

The keys to diagnosis are evidence of airflow obstruction and a compatible history. Smoking tobacco is the main risk factor but indoor biomass cooking and long term poorly controlled asthma can result in fixed airflow obstruction as well.

Severity assessment:

This relates to both their underlying disease and the current exacerbation.

Baseline COPD Severity

Severity	Mild	Moderate	Severe
Symptoms	Few symptoms but breathless on moderate exertion	Breathless walking on level ground	Breathless on minimal exertion with activities severely curtailed
Lung Function	FEV_1 60-80%	FEV_1 40-59%	FEV_1 <40%

Adapted from COPD-X Guidelines :
http://copdx.org.au/copd-x-plan/confirm-diagnosis/c3-assessing-the-severity-of-copd/

Look for warning signs of a life-threatening exacerbation:
Use of accessory respiratory muscles
Paradoxical chest wall movements
Cyanosis
Development of peripheral oedema
Haemodynamic instability
Drowsiness
Asterixis

Signs of hypercapnic respiratory failure

Patients with these features need ICU admission – if uncertain discuss with a senior.

Acute Investigations:

CXR – look for evidence of pneumonia, TB or CHF. These require specific management.
Sputum for AFB (if productive cough)
Full blood count
Creatinine, sodium and potassium
Pulse oximetry

Management:

Oxygen: Aim to keep the SpO2 between 88-92%: Excessive oxygen can precipitate hypercapnic respiratory failure but insufficient oxygen can also be fatal. Ideally use nasal cannulae and have extra care if oxygen is given with a face mask.

Bronchodilators: Nebulise Salbutamol 5mg Q1-4 hourly
Ipratropium 0.5mg Q4H can be added in severe exacerbations
Aminophylline can be used if salbutamol unavailable.

Steroids: Prednisone 40mg od for 5 days or Hydrocortisone 100mg IV tds if cannot take oral therapy/ life threatening exacerbation

Antibiotics: Use if ≥2 of the following: increased dyspnoea, increased sputum volume, or increased sputum purulence. Unless pneumonia use amoxicillin 500mg PO tds or doxycycline 200mg PO stat then 100mg PO bd.

Ongoing Management:

De-esculate care: Change IV to oral therapy and nebulised to inhaled therapy when stable

Educate: Educate about quitting smoking cigarettes, avoid indoor cooking with wood or charcoal, undertake regular exercise. Educate about inhalers and spacer technique (see inhaler technique SOP)

Vaccinate: Patients with moderate-severe COPD should get the pneumococcal vaccine.
Outpatient Medication: Discharge with a Salbutamol inhaler 100mcg 2 puffs prn for dyspnoea.
If >1 exacerbation this year consider adding inhaled Budesonide 100mcg 2 puffs BD via spacer – this may help reduce exacerbations but probably won't make them feel better.
Follow up: Organise review in clinic in MOPD in 1/12 **with spirometry** either prior to discharge or prior to clinic.

MANAGEMENT OF ACUTE PNEUMONIA

Diagnosis: Suspect in those with a short history of fevers, cough with sputum dyspnoea and chest pain. Common pathogens in our setting are S. pneumoniae, S. aureus and Q fever. In severe bacterial pneumonia, S. pneumonia, S. aureus and gram negatives such as K. pneumoniae are common.

Assess Severity

Confusion	1 point	CRB65 Severity
Respiratory Rate>30 /min	1 point	0 = Mild (Mortality <1%)
Systolic BP<90mmHg or	1 point	1-2 = Moderate (Mort 3-10%)
Diastolic BP<60mmHg		3-4 = Severe (Mortality>15%)
Age>65	1 point	

CRB65 is NOT perfect – USE YOUR CLINICAL JUDGEMENT

Investigations

CXR – useful for diagnosis and identification of complications.
Sputum for AFB – TB is a mimic of acute pneumonia
Blood cultures – Bacteraemia carries a worse prognosis and identification of the bacteria will influence antibiotic choice
HIV test – Pneumonia is an important first presentation of HIV related illness.

Obtain a Chest X-Ray

Chest XR shows discrete focal consolidation

- Treat for bacterial pneumonia
- If HIV positive, there is a broader microbiological differential diagnosis, especially consider the possibility of TB

Chest X-ray shows diffuse bilateral infiltrate or miliary shadowing

If HIV positive with CD4 <200 or unknown, or clinical suspicion of HIV, treat for PCP in addition to bacterial pneumonia and consider TB.

Initial Management

Admit if CRB65>2 or if they otherwise appear unwell (use your clinical judgement)
Consider ICU admission if hypotensive, tachypnoeic or hypoxic
Use oxygen if SpO2<92% (see oxygen protocol)
Give IV fluids (0.9% saline) in 500ml boluses if hypotensive. Use IVF with caution if hypoxic as IVF will often make this worse.

Initial Antimicrobials:

DO NOT USE Fluoroquinolones (eg Ciprofloxacin) due to the possibility of undiagnosed TB and generation of drug resistant TB.

Aspiration pneumonia : Amoxicillin 1g IV Q8H + Metronidazole 400mg tds PO (or 500mg Q8H IV if unable to take oral medications)

Ongoing Care

Switch to oral antimicrobials after 48 hours clinical stability (afebrile, respiratory rate and pulse rate normalised). These are: amoxicillin 500mg tds and erythromycin 500mg QID/ doxycycline 100mg bd. Ceftriaxone is not available orally, so in severe pneumonia switch to amoxicillin-clavulanate 625mg tds.

Duration of antibiotics is 5-7 days unless there is a complication such as pleural effusion or lung abscess.

Repeat chest X-ray only if clinical findings suggest development of a pleural effusion or failure to improve

References:

1. Ministry for Health and Social Welfare. Standard Treatment Guidelines (Stg) And The National Essential Medicines List (NEMLIT) For Mainland Tanzania (3rd Ed.) Dar es Salaam, Tanzania; 2007
2. Peck R, Smart L, Lie A (eds). Bugando Medical Center Inpatient Ward Guidelines For Patient Management. Department Of Internal Medicine (2nd Ed.) Mwanza, Tanzania; 2014
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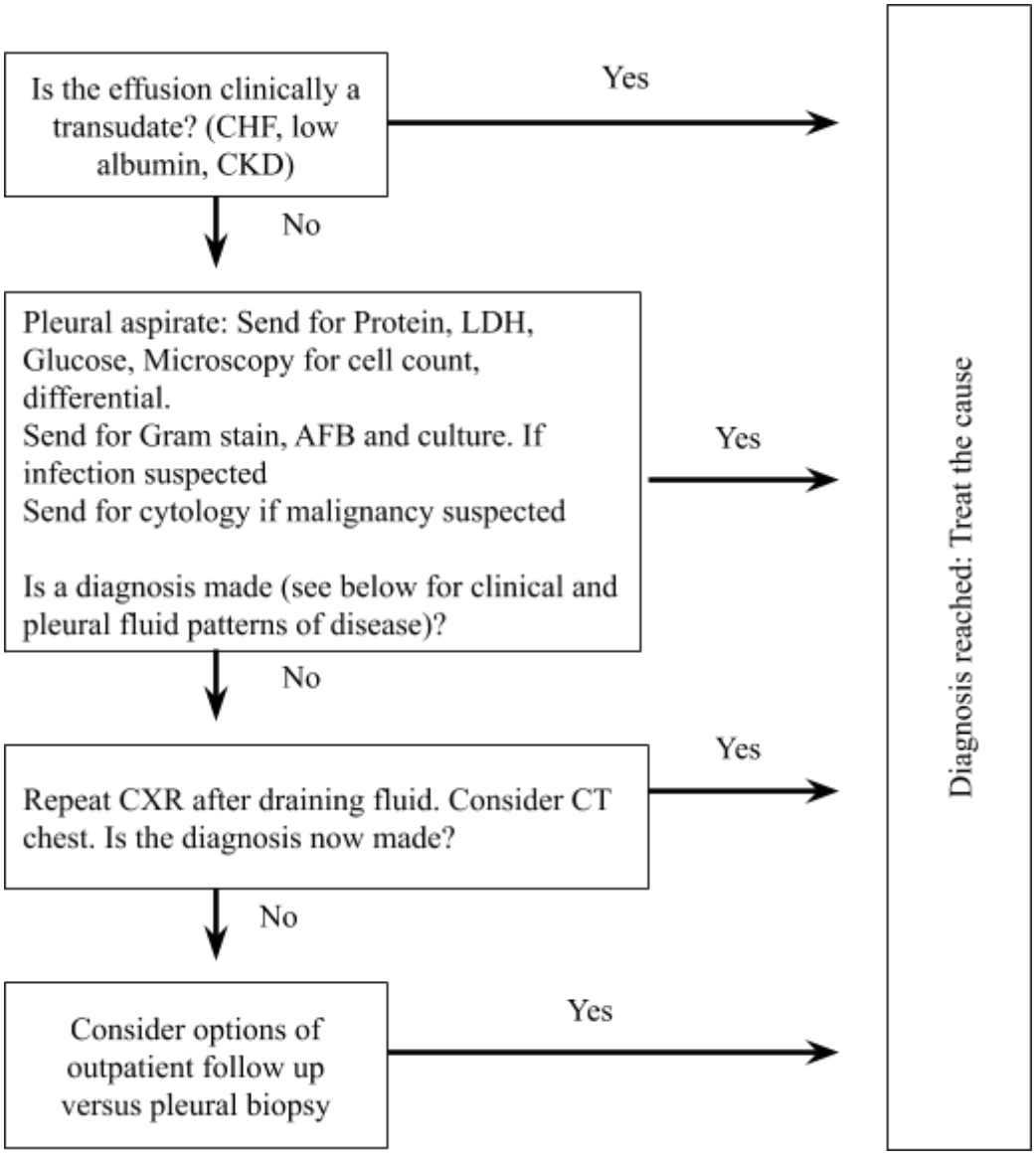
7. Regasa B. Aetiology of bacterial pathogens from adult patients community-acquired pneumonia in arba minch hospital, south Ethiopia. Science Journal of Clinical Medicine 2014, May 30;3(3):33-6.
8. Blomqvist J. Pneumococcal carriage in Tanzanian children with respiratory tract infections. University of Gothemburg, Sweden; 2014.

PLEURAL EFFUSION

Definition: Fluid in the pleural space

Goal of therapy: Improve symptoms associated with the effusion, identify the underlying aetiology and help recovery from the underlying cause



Specific Causes of Pleural Effusion

Transudate: Pleural fluid protein/ serum protein <0.5 & pleural fluid LDH/ serum PDH <0.6 & pleural fluid LDH is <2/3 normal serum LDH

Potential causes*:* Heart failure, low protein states (eg CKD, nephrotic syndrome, chronic liver disease)

Exudate: Pleural fluid protein/ serum protein >0.5 or pleural fluid LDH/ serum PDH >0.6 & pleural fluid LDH or >2/3 normal serum LDH.

Potential Causes:

Empyema: Short history with fever (and often pain) and rapid accumulation of fluid. Fluid may be frank pus, or be thick turbid fluid. Bacteria may (but not always) be seen on Gram stain or be isolated on culture

Parapneumonic effusion: Similar history to empyema, but with straw coloured fluid. On microscopy, the leucocytes are predominantly neutrophils.

Tuberculosis effusion: Several weeks- months symptoms, often with fevers/sweats. Straw coloured fluid which usually will have a predominance of lymphocytes. Look for evidence of pulmonary TB or other extra-pulmonary TB (eg lymph nodes). Adenine deaminase performed on the pleural fluid can be very helpful (although not currently performed at KCMC laboratory)

Therapeutic Protocol

Principles:

1. Perform the fewest number of pleural procedures needed
2. When performing a diagnostic thoracentesis, remove 1-1.5L (or until dry if smaller effusion) as this will improve dyspnoea, and may reduce the need for further procedures
3. Indications for chest drains: Empyema, haemothorax, planned pleurodesis. In other situations, chest drains often slow recovery: check with a senior if you think it is needed.
4. Not all effusions must be drained completely. Some will improve with appropriate medications (eg tuberculosis, heart failure)

Thoracentesis Standard Operating Procedure

PREPARATION

1. Obtain informed consent from the patient, explaining the potential benefits (diagnosis, relief of symptoms, faster recovery) and hazards (bleeding, introduction of infection and symptomatic pneumothorax – all rare)
2. Obtain the equipment: 21G needle x2, 10ml syringe x1, 20ml or 50ml syringe x1, 16-18G cannula x1, 10ml 1% lignocaine, sterile gloves x1, gauze x3-5 small sheets, iodine/ chlorhexidine solution, small sterile dressing pack
3. Obtain the chest radiograph and ensure you have correct location for the fluid
4. Obtain a colleague, medical student or nurse to assist.
5. Recheck clinical signs to ensure they match effusion: dull to percussion, with reduced breath sounds and reduced vocal resonance. Identify and mark the optimal location for thoracentesis. Remember it should be as lateral as possible from the spine and in the intercostal space, just above the rib

6. If in doubt: STOP. Ask a resident/ senior/ radiologist to perform bedside pleural ultrasound to confirm the presence of an effusion and optimal site for aspiration

PROCEDURE

1. Wash hands
2. Put on sterile gloves
3. With assistant, make a sterile area from the dressing pack
4. Clean skin with iodine working from the site of thoracentesis, outwards to cover at least a 10cm diameter circle
5. Infiltrate 10ml 1% lignocaine. Approximately half should be just under the skin. Then aspirating gently as you go, insert the needle until you obtain the first pleural fluid. Then withdraw 1-2mm and infiltrate the remaining lignocaine.
6. Wait 2-3 minutes.
7. Attach the cannula to the 50ml syringe. Insert the cannula in the same site as the needle and advance carefully, gently aspirating. When pleural fluid is obtained, advance the needle an additional 0.5 cm
8. Obtain 50ml of pleural fluid for diagnostic tests. Ask your assistant, to fill the following tubes:

a. Red top tube x2 (1 for biochemistry at KCMC, and another if send-away tests such as adenine deaminase are needed)
b. Container for microbiology stains (AFB and Gram stain and culture
c. Container for cytology if malignancy suspected

9. STOP: If you think the fluid is an empyema (frank pus or turbid fluid in the context of likely infection) do not drain fluid now, but organise an urgent chest tube with under water seal drainage.

If not empyema, continue to drain 1-1.5L to relieve dyspnoea as described below

10. Advance the plastic cannula over the front of the needle and then remove the needle.
11. Attach the IV giving set to the cannula, and the urinary catheter bag to the IV giving set
12. Hold the giving set while flow is established. It may then be taped to the patient in a way that allows flow to continue. However, you must remain near to the patient during the entire process of draining the fluid. After 1-1.5L has been drained. Reassess the patient. If you think there is further fluid, and they are tolerating the procedure you may continue to take another 1L. In some patients the rapid removal of several litres of fluid can transiently worsen their breathlessness through a re-expansion pulmonary oedema.
13. When finished you MUST write a note in the file explaining: the indication for the procedure, what you did and any complications encountered.

ENDOCRINE SYSTEM

DIABETES MELLITUS
DIABETES KETOACIDOSIS
HYPERGLYCEMIC HYPEROSMOLAR STATE
HYPOGLYCEMIA

MANAGEMENT OF DIABETES

Diabetes Diagnosis

Criteria for Diabetes Diagnosis: 3 options
FPG ≥126 mg/dL (7.0 mmol/L) Fasting defined as no caloric intake for ≥8 hrs
2-hr PG ≥200 mg/dL (11.1 mmol/L) during OGTT (75-g) using glucose load containing the equivalent of 75g anhydrous glucose dissolved in water
Random PG ≥200 mg/dL (11.1 mmol/L) In persons with symptoms of hyperglycemia (Polyuria, Thirst, Hunger, Weight loss, Malaise, Tiredness, Blurred vision Poor wound healing)

Frequency of A1C Testing (used to measure glycemic control)	
At least **2 times each year** in patients who are meeting treatment targets and have stable glycemic control	**Quarterly** in patients whose therapy has changed or who are not meeting glycemic targets

Investigations for diabetic patients

- Urine for protein; to be done in every visit
- Serum creatinine; biannually or annually
- lipid profile (Serum cholesterol, HDL, LDL, Triglyceride); at least once a year
- CXR; base line on Diagnosis and later when need arise
- ECG; at least twice a year

TREATMENT OF DIABETES

Non pharmacologic therapy

Life style changes

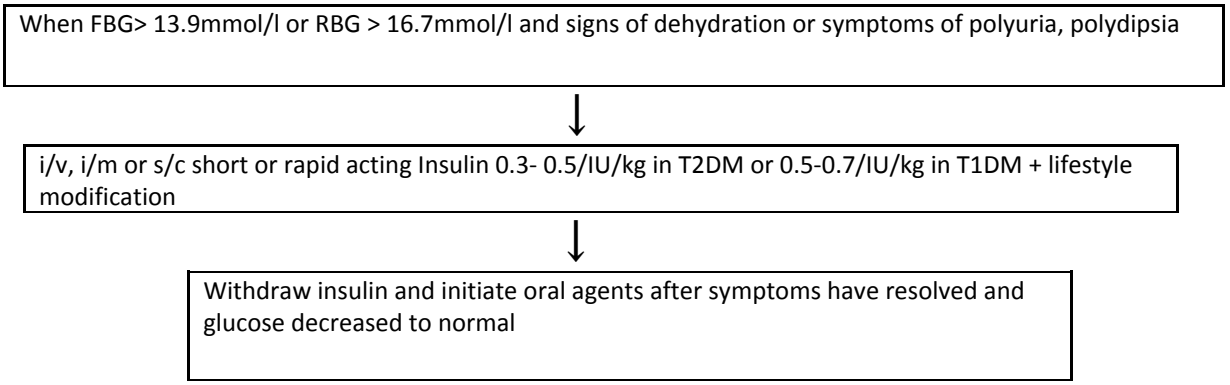
- Physical exercise
- Dietary counselling
- Weight reduction
- Moderate alcohol intake
- Smoking cessation

Indications for use of Insulin in T2DM

- Initial presentation with severe hyperglycemia
- Peri-operative period especially major or emergency surgery
- chronic complication of diabetes
- Pregnancy

- Contraindications to OHAs
- Failure to meet glycemic targets with OHAs
- Severe infections which require hospitalization

Inpatient management of severe hyperglycemia



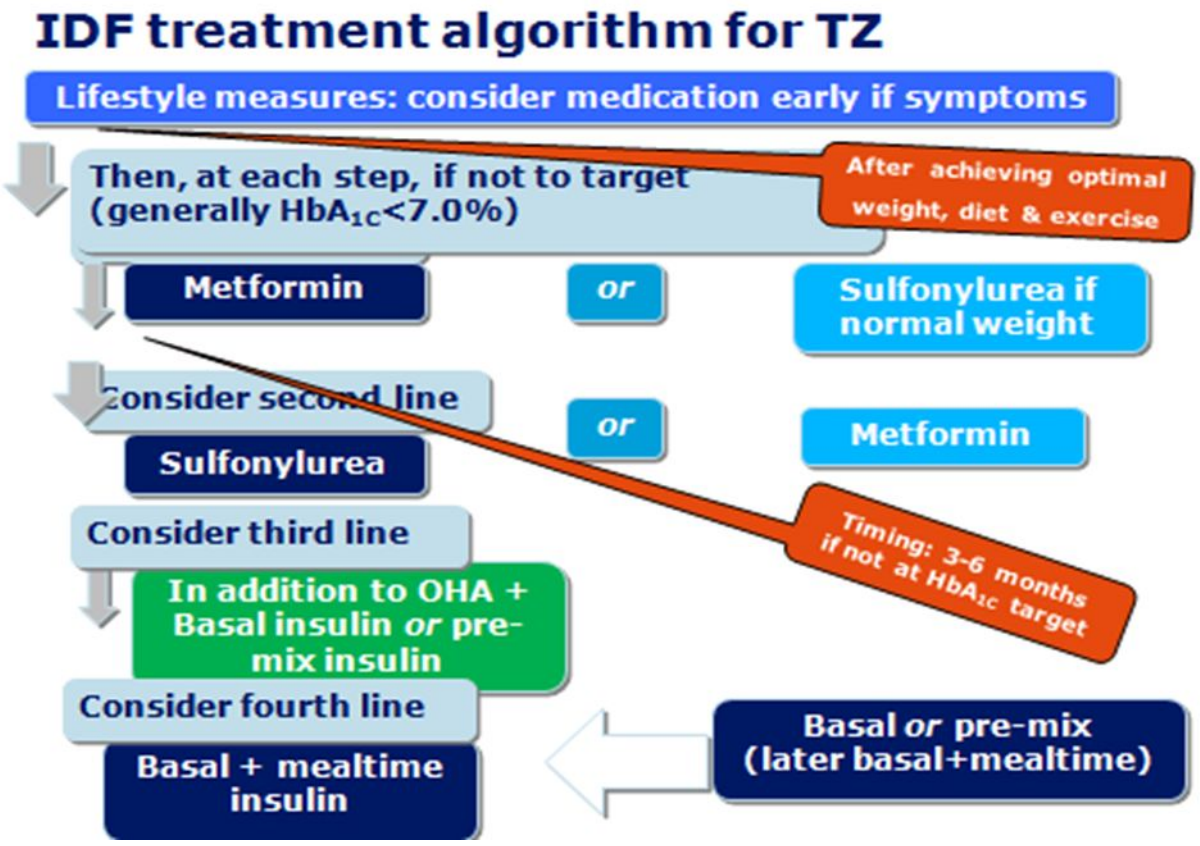
Note: Insulin Doses >25units/day (intermediate acting insulin) divide into 2 doses (2/3 and 1/3).

- T2DM Patients should be discharged with insulin if
  - ≥30IU has been used to control hyperglycemia during inpatient care
  - Patient has chronic complication.

Glargine (long acting insulin)

T2DM, 0.2unit/kg once daily; T1DM, glargine 1/3 + actrapid 2/3 (divided into 3 premeal doses) of total insulin dose per day.

Pharmacologic therapy for type 2 diabetes



TITRATION OF METFORMIN

Name of drug	Starting dose	Maximum dose
metformin	**500mg BD/TDS**	**2000mg**

TITRATION OF SULFONYUREA

Name of drug	Starting dose	Maximum dose
glibenclamide	2.5mg or 5mg OD	10mg BD
gliclazide	40 to 80 OD	160mg BD
glimepiride	1mg OD	6mg OD
glipizide	5mg OD	15mg BD
Chlorpropramide (diabenese)	250mg OD	500mg

* Other available OHAs which can be used are sitagliptin 100mg OD, pioglitazone 15-30mg OD

Pharmacologic Therapy for Type 1 Diabetes

Insulin Therapy Is Recommended for Individuals with Type 1 Diabetes

DOSE: 0.5 — 0.7IU/kg

Assessments during clinic visits

- Body weight
- Blood pressure target of <130/80 mmHg (Refer management of HTN)
- 0Blood glucose levels
- Foot exam to identify risk factors predictive of ulcers and amputations once a year
    - Assessment of foot pulses
    - loss of protective sensation (LOPS) testing
        - best accomplished using monofilament
    - Provide foot self-care education
        - Educating patients about proper foot care and periodic foot examinations
- Fundoscopy
    - If no retinopathy: consider exams every 2 years
    - If retinopathy: annual exam
    - Retinopathy progressing or sight threatening: more frequently

References

1. American Diabetes Association. Standards of medical care in diabetes—2015. Diabetes Care.2015; 38(suppl 1):S1-S93.
2. RSSDI Clinical Practice Recommendations for Management of Type 2 diabetes Mellitus- 2015.
3. Type 1 Diabetes Treatment Guideline - 1996–2015 Group Health Cooperative.

DIABETIC KETOACIDOSIS

Definition
DKA is a state of severe metabolic derangement that occurs in patients with insulin dependent diabetes mellitus secondary to insulin deficiency, excess stress hormone and volume depletion. It is an acute complication of diabetes mellitus. The patient will present with key features of **hyperglycemia, ketonemia, ketouria, acidosis and altered mental status.**

Precipitating factors

- Previously undiagnosed DM / Newly diagnosed diabetics
- inadequate insulin administration
- Interrupted of Insulin therapy
- intercurrent infection (pneumonia, UTI, sepsis, candidiasis, gastroenteritis etc)
- infarctions (cerebral, coronary, mesentery, periphery etc)
- Drugs: cocaine, steroids
- Pregnancy
- Acromegaly
- Idiopathic

Clinically, a patient may be

- very weak with normal mental state or altered level of consciousness to coma
- Hyperglycaemia > 250mg/dl (>14 mmol/l)
- dehydration: tachycardia, and possibly hypotension and low or no urine output
- Metabolic acidosis: Kussmaul breathing and acetone smell

Investigations: Diagnostic criteria

Blood glucose, serum electrolytes and Urine analysis–dipstick

- **Blood glucose** :- Hyperglycemia **>250mg/dl (>14mmol)**
- **Serum Electrolytes**
  - Serum K : mildly elevated at presentation (acidosis, fat and muscle destruction), despite of total body K deficit (diuresis and vomiting)
  - Serum sodium : usually 1.6mmol reduction with 5.6 mmol rise in serum glucose
  - Total Cl, phosphorus and magnesium are also reduced
- **Serum bicarbonate**:- **≤18mEq/L**
- **Arterial pH** : **<7.0**
- **Ketone bodies:- Urine ketones–positive +4**(detected by nitropusside stick (dipstick)

Serum ketones – positive

- **Anion gap:** (Na^+)-[$(Cl^-$ + $HCO3^-$ (mEq/L)] :- **>12**
- **Serum osmolality** = 2[serum Na^+ (mEq/L)] + plasma glucose (mg/dl)/18
  - Mild to moderately elevated (300-320mOsm/L)

- **Other tests: BUN and serum creatinine, Full blood count, Lipid profile, Blood and urine cultures, CXR, ECG**

Management:

6.0 Transfer to Medical Intensive Care Unit (MICU)
7.0 Correct Dehydration – Fluid replacement
8.0 Correct Hyperglycemia – Short acting insulin

9.0 Correct Electrolyte imbalance – Replacement of Potassium
10.0 Identify and manage precipitating factors – administration of antibiotic if infection is present
11.0 Chart fluid intake and urine output, put catheter if unconscious and NGT for feeding and to prevent aspiration.

Use flow chart to record:
Fluid intake and output and laboratory values as a function of **insulin administered**

KCMC Medical Department	Management of Diabetic Ketoacidosis
Name of Attending Doctor	Hospital Reg. No.
Duration of Diabetes	Surname
Precipitating Factor	Other names
	Residential Address
	Date of Birth Sex
	Religion Ward
	Date of treatment

Hour	Time Start	B/sugar mMol/L	Urine Multi stick		Acid-base balance		Electrolyte		IV Fluids	Urine output	Actrapid insulin	Remarks
			Glucose	Ketone	CO_2 an acidic gas	HCO_3 an alkaline	K^+	Na^+				
0		check	check	check	check	check	check	check	1L N/S -30 mins 1L N/S.90 mins		5u IM 5u IV	
2nd		check									5u IM	
3rd		check							1L N/S + KCl 20mmol		5u IM	
4th		check					check	check	500 ml N/S		5u IM	
8hrs		check							500 ml N/S + 20 mmol KCL		5u IM	
12hrs		check					check	check	500 ml N/S		5u IM	
16hrs		check							500 ml N/S + 20 mmol KCl		5u IM	
20hrs		check							500 ml N/S		5u IM	
24hrs		check	check	check	check	check	check	check	500 ml N/S + 20 mmol KCL		5u IM	
		9	**2**				**4**	**4**	**6 L**		**50 u**	

1. Check Blood glucose hourly for first 4 hours, then 4 hourly

2. Give 2 liters IV fluids in 1st 2 hours, 1 L for 30 Mins and 2nd L for 1 and 30 Mins then continue as per chart

3. Add 20meq of KCL in 3rd Litre N/Saline. Do not give KCL if no urine output.

4. After each 2L fluid check for fluid overload.

5. When Blood Glucose falls to ≤ 14 mmol/L change insulin to 8 units sc – 6 hourly.

    a. Start 5% Dextrose 500mls 6 hourly, and Check Blood glucose before insulin injection.

6. When B, sugar is controlled in 2nd or – 3rd day calculate insulin dose 0.7 u/kg/day ÷ bd or tds
If patient is stable and B sugar is controlled – change to insulatard.

7. With severe Acidosis pH < 7.0 give $NaHCO_3$ 100 -300mls of 1.26 %.

8. Treat inter-current infection based on suspected organism and change per laboratory finding

9. Do Baseline – serum creatinine, cholesterol, Blood count, fundoscopy in first day

10. continue with regimen until fluid deficit replaced, ketonuria diminished and adequate oral take is feasible

11. Start diabetic education and self-insulin injection as soon as patient can able to do so

Complications

- Cerebral edema
    - May be caused by rapid reduction of blood glucose, use of hypotonic fluid and / or bicarbonate
    - High mortality
    - Treat with mannitol and oxygen (intubation with hyperventilation)
- Hypokalemia
- Hypophosphatemia
- Pulmonary Oedema – result of aggressive fluid resuscitation
- Thromboembolism – severe dehydration, Cerebral vessels, occurs hrs to days after DKA
- Acute circulatory failure – Shock r/o MI

Follow-up

- Diabetic education and self-insulin injection method should be taught before discharge
- Should be given appointment to diabetic clinic for follow up in regular basis

HYPERGLYCEMIC HYPEROSMOLAR STATE

Definition:
An acute metabolic complication of diabetes mellitus characterized by impaired mental status and elevated plasma osmolality in a patient with hyperglycemia.
(Extreme hyperglycemia, hyperosmolarity and dehydration)

At risk

- Occurs predominately in Type 2 diabetes

Presentation:	**Diagnostic Criteria**
Extreme dehydration (deficit 10L) Supine or orthostatic hypotension Hyperosmolar state Confusion→coma 1.0 Neurological findings Seizures 2.0 Transient hemiparesis 3.0 Hyperreflexia 4.0 Generalized areflexia	Elevated RBG >600mg/dl (>33.3mmol/L) Serum bicarbonate >18mEq/L Serum osmolality >320mOsm/kg Urine ketone +/- Serum ketone +/-

Management: Fluid replacement

Fluid repletion 2L 0.9%NS rapidly in the 1-2 hrs Normal saline 2-3 litres rapidly 2L NS over 2-4 hrs: Total deficit = 10litres 1L NS over 4-6hrs* Replete half of it in the first 6hrs 1L NS over 6-8hrs* 1L NS over 8-10hrs*
*according to response, elderly, pregnant, heart and renal failure.
Note: Require more than 6L

Insulin: Should be started only once aggressive hydration has taken place (after the 3rd litre)

- continuous infusion of low-dose insulin IV (~ 0.1 U/kg/hr) is effective
- goal is to *slowly* decrease serum glucose 5.5mmol/L (≤ 100 mg/dL/hr

Give 5% Dextrose when blood glucose is ~ 250 mg/dL or when glucose level falling faster than 100 mg/dL/hr

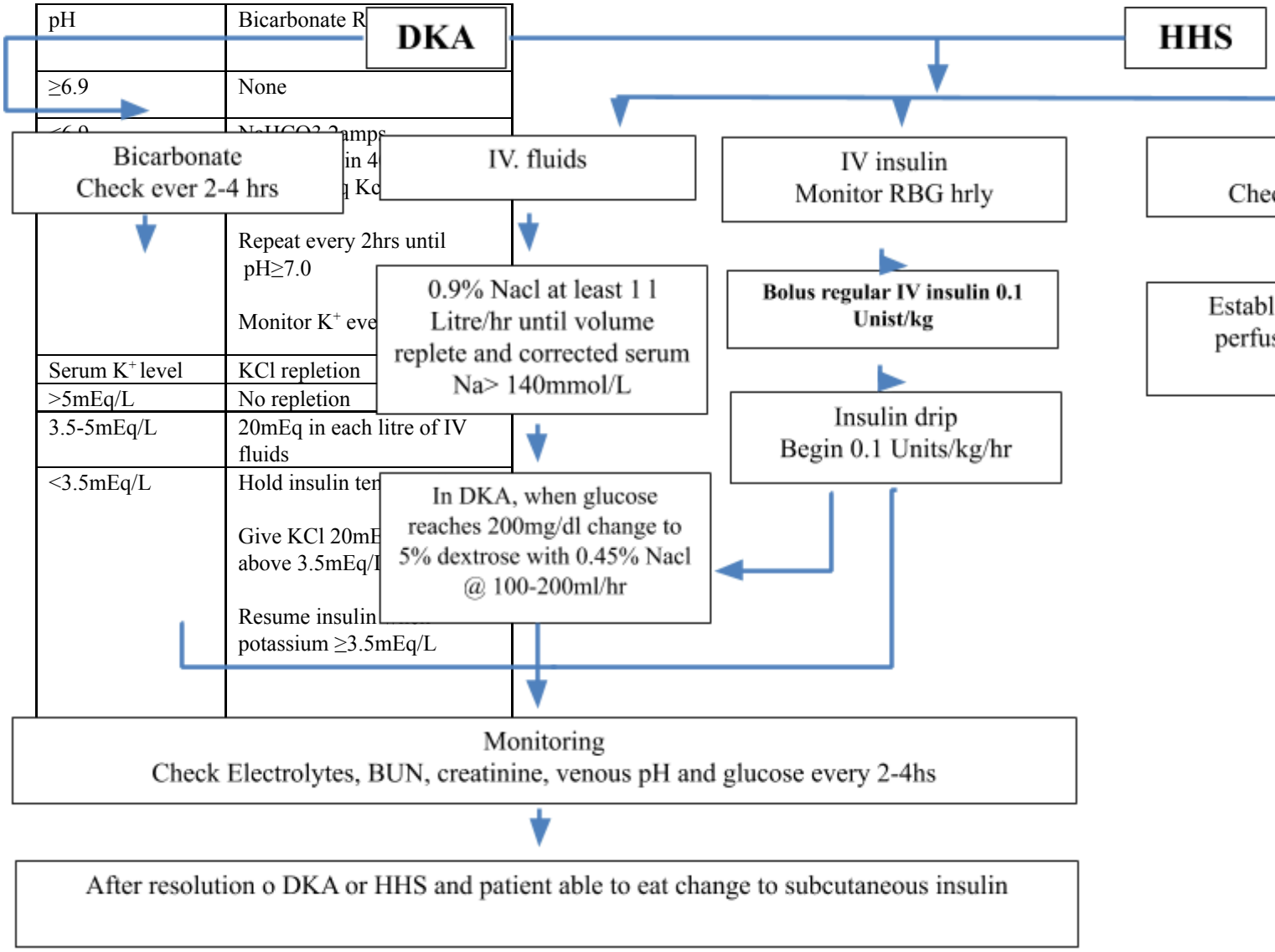
Alkali: BICARBONATE IS ALMOST NEVER ADMINISTERED (Bicarbonate administration can lead to increased cerebral acidosis)

Treat underlying precipitating illness

Follow-up:

- According to the precipitating factors
- Re-start usual medications and consider insulin if not on it previously.
- Counseling on Diet, compliance to meds and general diabetes education.
- Should be given an appointment to diabetic clinic for follow up in regular basis

MANAGEMENT PROTOCOL IN SUMMARY



Adapted from Kitabchi et al., Diabetes Care, 2001

REFERENCES

1. Kitabchi AE, Umpierrez GE, Miles JM, Fisher JN. Hyperglycemic crises in adult patients with diabetes. Diabetes Care.2009;32(7):1335-1343
2. American diabetes Association Clinical Practice Guideline

HYPOGLYCEMIA

Definition:

In diabetes; Low glucose level ≤ 3.9 mmol/l

In non-diabetic patients (Whipple's triad)

- symptoms consistent with hypoglycemia
- low plasma glucose of ˂3.0mmol/l with symptoms of hypoglycemia
- relief of symptoms after plasma level is raised

Causes of hypoglycemia in adults

a. Drugs
a. Insulin
b. insulin secretagogue (e.g. sulphonyurea), Other drugs (e.g. quinine, quinolones)
b. Critical illnesses
Hepatic, renal, or cardiac failure, Sepsis (including malaria), Prolonged starvation.
c. Hormone deficiency
Cortisol, Glucagon and epinephrine (in insulin-deficient diabetes mellitus)
d. others
a. Alcohol
b. Insulinoma (check creatinine if insulinoma suspected)
c. Functional $_{\beta}$-cell disorders (nesidioblastosis)
d. Noninsulinoma pancreatogenous hypoglycemia
e. Insulin autoimmune hypoglycemia (Antibody to insulin , Antibody to insulin receptor)

Risk factors precipitating hypoglycemia

a. Skipping meals
b. over dosage (multiple insulin injections)
c. Unplanned exercise
d. long acting hypoglycemic drugs

SYMPTOMS OF HYPOGLYCAEMIA

Autonomic response tends to occur with BG <3.6 mmol/L	Neurologic (neuroglycopenic symptoms) tends to occur with BG <2 mmol/L
• Sweating (common) • tachycardia • Tremors • palpitation • Hunger • Nervousness • paresthesia	• irritability • Confusion • weakness • Drowsiness • Difficult in speaking • Difficulty in concentration • Incoordination • Seizure, Occasionally, transient focal neurologic deficits • Loss-of-consciousness • Visual disturbance

NOTE: Any patient with acute change in mental status or coma should undergo rapid assessment of blood glucose as a possible cause

DIAGNOSTIC EVALUATION

Obtain blood glucose concentration as soon as possible ● usually with Glucometer
For symptomatic patient known to have diabetes and with a low glucose value<3.9 mmol/L ● Start treatment. If a glucose test cannot be performed, do not delay. **Treat as if** hypoglycemia has been confirmed
If the glucose is <3.0 mmol/L and the patient is not diabetic ● **Do not delay** treatment if symptomatic hypoglycemia is suspected but rapid blood glucose tests or blood for diagnostic studies are not available.

TREATMENT

*Injection glucagon is less useful in T2DM

* *Hypoglycemia may be recurrent due to the prolonged duration of these drugs**.**

If the patient is conscious and able to drink and swallow safely ● administer a rapidly-absorbed carbohydrate (fruit juice or non-diet soda, or Buccal smear with honey or table sugar)
If the patient has altered mental status, is unable to swallow, or does not respond to oral glucose ● Give an IV bolus of 25 to 50 mL of 50% dextrose within 15 minutes followed by continuous glucose infusion to maintain the blood glucose > 4.4 mmol/L ● Measure blood glucose 10 to 15 minutes after the IV bolus. ● Re administer 25 to 50 mL of 50% dextrose if still hypoglycemic. ● Monitor blood sugar every 30 to 60 minutes thereafter until stable (minimum of 4 hours)
If glucose cannot be given by parenteral or oral routes ● Give glucagon 1 mg IM or SC.*(when available) ● Response may be transient and should be followed by careful glucose monitoring and oral or intravenous glucose administration
Admit patients with ● Ingestion of a long-acting hypoglycemic agents or long acting insulin** ● Recurrent hypoglycemia during observation, and those unable to eat

References

1.Cryer PE. Hypoglycemia in diabetes: Pathophysiology, prevalence, and prevention, American Diabetes Association, Alexandria, VA 2009

2. Harrison's Principles of Internal Medicine, 18th Edition

GASTRO-INTESTINAL SYSTEM

ACUTE UPPER GIT BLEEDING
LOWER GIT BLEEDING
ACUTE LIVER FAILURE
ACUTE PANCREATITIS
HEPATITIS B

UPPER GASTROINTESTINAL BLEEDING

DEFINITION: Upper gastrointestinal bleeding (or haemorrhage) is that originating **proximal to the ligament of Treitz**; in practice from the oesophagus, stomach and duodenum.

PRESENTATION:

- Hematemesis (bright red blood suggest ongoing moderate to severe bleeding)
- Coffee-ground emesis
- Melena
- Maroon/ black currant like or bright blood per rectum
- Signs of Anemia and Shock is massive bleeding

CAUSES:

- **Esophageal causes**

Esophageal varices, Esophagitis, Esophageal cancer, Esophageal ulcers, Mallory-weiss tear

- **Gastric causes**

Gastric ulcer, Gastric cancer, Gastritis, Gastric varices

- **Duodenal causes**

Duodenal ulcer, vascular malformation, including aortoenteric fistulae, Hematobilia, Hemosuccus pancreaticus

GOAL OF MANAGEMENT:

- Resuscitation and Hemodynamic stabilization
- Identify the source of bleeding
- Definitive treatment of the cause to stop the bleeding

Check Physical Exams on following:

- ✓ Epigastric tenderness on exam?
- ✓ Exam findings of portal hypertension (splenomegaly, ascites etc)?
- ✓ Check vitals /orthostatics

-Tachycardia (10% volume loss)
-Orthostasis (20% volume loss)
-Shock (>30% volume loss) loss)

INITIAL EVALUATION: Incudes history, physical examination and triage.

Triage:

- Admit to MICU all patients with **hemodynamic instability**
  - i.e. Shock, orthostatic hypotension.
  - active bleeding manifested by hematemesis, bright red blood per nasogastric tube, or Hematochezia

Consider history of following:

- ✓ Dyspepsia/PUD, GERD?
- ✓ Prior bleeding?
- ✓ Liver disease?
- ✓ NSAID use, warfarin use?
- ✓ EtOH, smoking
- ✓ Anti-platelets, Anticoagulants
- ✓ Schistosomiasis, working in irrigation scheme areas

RESUSCITATION AND HEMODYNAMIC STABILIZATION

IV access:

- ✓ 2 large bore cannula 16 0r 18 gauge
- ✓ Hb, grouping and Cross-matching
- ✓ Take FBP before transfusion
- ✓ Supplemental oxygen by nasal cannula
- ✓ Consider elective endotracheal intubation (ETT) in patients with ongoing hematemesis or altered respiratory or mental status may facilitate endoscopy and decrease the risk of aspiration

Restore the circulating volume

- Give crystalloids to obtain/maintain circulatory volume;

  -Titrate to a **SPB > 100mmHg & PR≤100 bpm** (Refer to Fluid management section)

  -Insert Foley Cather to monitor UOP > 0.5mls/kg/min

- Urgent Blood Transfusion;
  -Estimated blood loss from Hx/PE
  -Patient current Hemoglobin/Hematocrit
- Insert NGT, if variceal source known, call senior to Insert Sangstaken-Blakemore or Mennisota tube
- Keep the patient nil by mouth for endoscopy
- Give prokinetics to improve gastric visualization at the time of endoscopy by clearing the stomach of blood, clots, and food residue
  - Erythromycin 500mg IV STAT
  - Metoclopramide 10mg IV STAT

DEFINITIVE TREATMENT POST ENDOSCOPY

Etiology	**Treatment**
Varices	**Pharamcology** ● *Octreotride 50µg IV bolus, then 50µg/hr iv infusion 48hrs (5x/d)s* ● *Give Ciprofloxacillin 500mg po q12h or Ceftriaxone 1g iv q12h for Cirrhotics with ascites to prevent SPB ? consider also for {PHTN 2°HSS}* **Non Pharmacology** ● *Early band ligation* ● *Sclerotherapy* ● *Baloon temponade (sangstaken blakemore tube/Mennisota tube* ● *Consult surgeon for surgical options if endoscopy treatment fail*
PUD	***If Active bleed or non bleeding visible vessel (Forest Ia/Ib/IIa) on OGD*** ● *IV PPI e.g Rebebazole 20mg iv OD, Pantoprazole 40mg iv OD, or Esomeprazole 40mg iv OD for 48hrs.* ● *?Octreotide if no acess to OGD* ● *Endoscopic therapy (ET)* ***If adherent clot (Forest Iib)*** ● *IV PPI (see above)* ● *Endoscopic removal of clot to r/o NBVV* ***If flat,pigmentedspot or clean base*** ● *No endo treatment indicated* ● *Oral PPI bd* ● *Consider early hospital discharge*
Mallory weiss tear	*Ussually self limiting, endoscopic Rx if activee.g hemoclip*

Before discharge:

Varices with PHTN

- *Start propranolol 20mg po BD Max 160mg /24hrs (titrate so HR decreases by 25%)*
- *Start aldactone 100mg OD if ascites present*
- *Consider nitrates to further reduce PHTN*
- *Iron supplementation*
- *Praziquantel 4omg/kg STAt or divided in 6hrs apart if schistosoma periportal fibrosis*
- *Schedule for Band ligation/scleroRx 4-6 session every after 2/52*
- *Iron supplementation*

Non Variceal - PUD

- *Start oral PPI for 6/52 if NSAID- induced.*
- *Empirical Rx for H. pylori with triple therapy:*
- *Oral PPI either*
  - *Omeprazole 40mg BD or*
  - *Pantoprazole 40mg OD or*
  - *Rabeprazole 20mg OD*
- *Clarithromycin 500mg BD and*
- *Amoxicillin 1g BD OR*

Tinidazole/metronidazole 500mg BD for 10-14days

- *Iron supplementation*

ACUTE LOWER GIT BLEEDING (LGIB)

Definition: Bleeding that emanates from a source distal to the ligaments of Treitz.
Causes: Neoplasm (Cancer & Polyps), Hemorrhoids, Infective colitis, IBD, Diverticulitis, Vascular malformations and miscellaneous causes i.e. radiation, CMV and KS.
Goal of Management

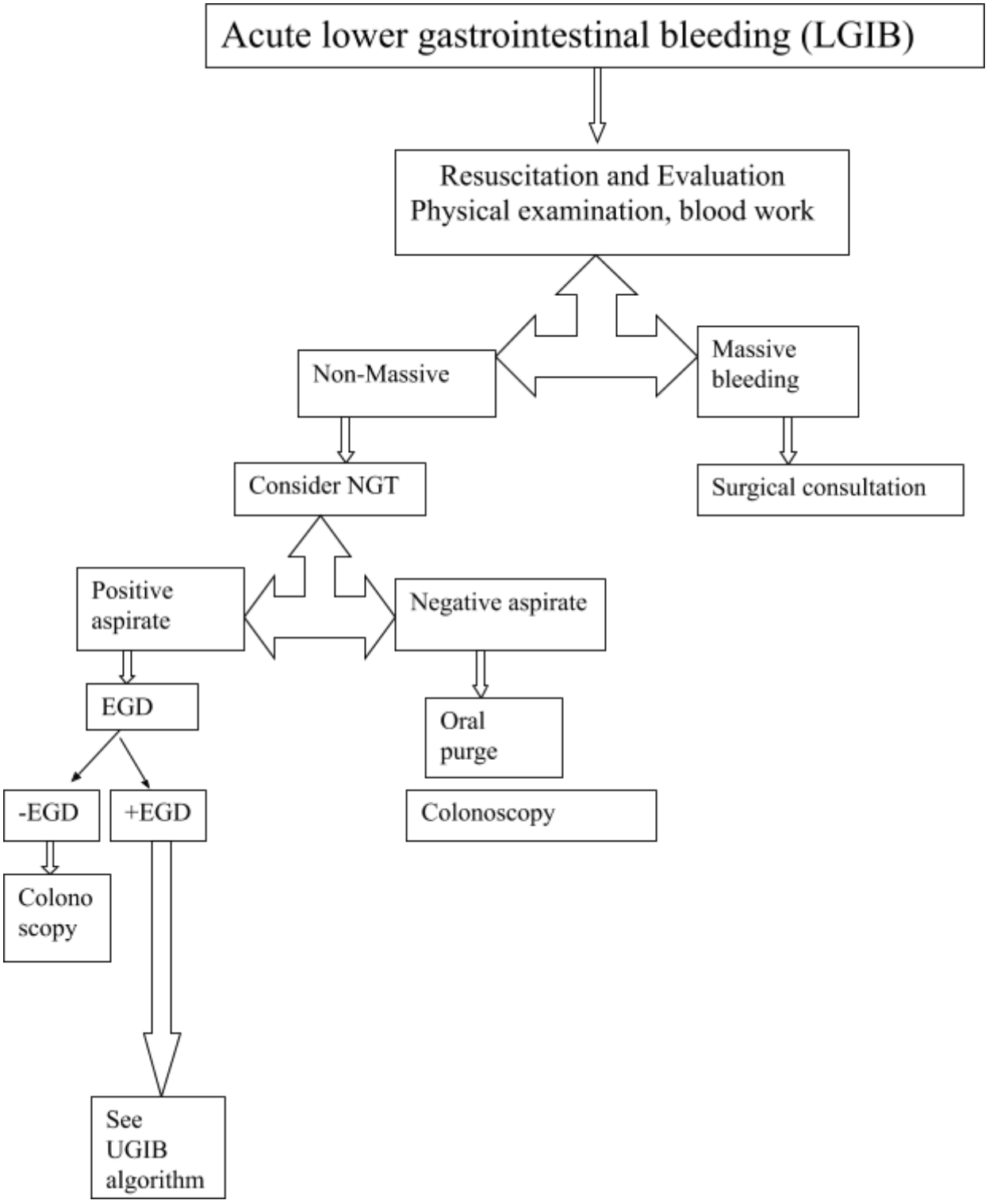
1. Stabilize patient
2. Identify the cause
3. Definitive treatment

Initial Evaluation and resuscitation
History: A history of abdominal pain, anal pain, weight, bleeding per rectal, and changes in bowel habits, high frequency chronic diarrhoea, prior dx IBD, Hemorrhoids, use of NSAIDS, pruritus, fresh red blood on toilet paper.
Physical Examination: Check vitals:

- Tachycardia (HR>100 bpm), 10% volume loss,
- Orthostasis (HR ↑ by >10bpm, SBP ↓ by >20 mmHg, OR DBP ↓ by >10mmHg (Equivalent to 1liter blood loss), 20% volume loss
- Shock (MAP <65), 30% volume loss. Careful digital rectal examination should be performed.
- Rectal tumors are usually palpable digitally

Triage: If patient is Orthostatic or Shock needs ICU care

Resuscitation & Stabilization of the patient:
Fluid resuscitation (refers other protocol-type, amount)
HB X-Matching and grouping
Blood transfusion (should be the first to be considered)
Oxygen
Medications: **Vasopressin**-0.2U/M, if bleeding persists, 0.6U/M.
Epinephrine-start 1:10,000units


Acute lower gastrointestinal bleeding (LGIB)
Resuscitation and Evaluation
Physical examination, blood work
Non-Massive
Massive bleeding
Consider NGT
Surgical consultation
Positive aspirate
Negative aspirate
EGD
Oral purge
-EGD
+EGD
Colonoscopy
Colono scopy
See UGIB algorithm

NOTE:

Massive bleeding- include 50% blood volume loss within 3 h or a rate of loss of 150 ml min^{-1}.

References:

1. American Society of Gastrointestinal Endoscope (ASGE) guideline: The role of endoscopy in patient with lowers GI-bleeding.Vol 79: no6, 2014.
2. Management of massive blood loss: a template guideline. Hamilton et al, 2010. British Journal of anesthesia.
3. World organization of digestive endoscopy 'approach to lower GI bleeding' OMED 2010.
4. http://emedicine.medscape.com/article/188478-treatment#d15- accessed on 16th February 2016.

ACUTE LIVER FAILURE

Definition:

Acute onset of severe impairment of liver function characterized with jaundice, slowing of mentation, sleep disruption, confusion, coma and coagulopathy in an individual without preexisting liver disease

Subcategories of ALF:

- Hyperacute liver failure (e.g., <7 days from illness onset to ALF);
- Acute liver failure (e.g., ALF onset within 7–21 days); and
- Subacute liver failure (e.g., ALF developing 3–26 weeks after symptom onset

Causes:

- Viral
  - HAV, HBV, HCV
- Drugs:
  - INH, Rifampicin, Isoniazid .Pyrazinamide ARTs e.g. Niverapine, Abacavir, Efavirenz
- Vascular
  - Ischemic Hepatitis, Budd-Chiari syndrome
- Autoimmune Hepatitis
- Herbal products and dietary suppliments
- Miscellaneous
  - HELLP syndrome

Clinical presentation:

- Initial presentation is nonspecific with nausea, vomiting, malaise followed by jaundice
- Neurologic:
  - **Encephalopathy:**

-*Stage I Change in Mental status, Stage II: Lethargy and confusion,*
-Stage III Stupor, Stage IV: Coma

  - **Asterixis**: *In encephalopathy Stage I/II/III*
  - **Cerebral Edema:**

↑ ICP ↓ CPP →→→cerebral hypoxia, uncal herniation, Cushing's reflex, (hypertension bradycardia), pupillary dilatation, decebrate posturing, apnea.

- Symptoms of complication.

Goal of therapy:

1. Supportive care to address the underlying pathophysiological abnormalities is recommended as the standard of care for ALF patients.
2. Rapid evaluation for treatable causes
3. Manage the complication

Investigations:

1.	**PT INR APTT**	
2.	**CHEMISTRIES:**	Potassium, chloride, bicarbonate, AST, ALT, alkaline phosphatase, GGT, total bilirubin and indirect bilirubin, albumin Creatinine, blood urea nitrogen
		Urea , Sodium and Glucose for Osmolality
3.		
4.	***VIRAL HEPATITIS TESTS***	HBSAg, anti-HBc IgM
5.	***FBC***	
6.	***HIV serology***	*HIV 1 &2*
7.	***ARTERIAL BLOOD GAS***	
8.	***AMMONIA LEVELS***	*Arterial if possible*
9.	***AUTOIMMUNE MARKERS***	*ANA, ASMA, Immunoglobulin Levels*
10	***AMYLASE AND LIPASE***	
11.	**CHEST X-RAY**	
12	**ULTRASOUND OF LIVER AND PANCREAS.**	Hepatic vein Doppler studies if Budd–Chiari syndrome is suspected
13	**BLOOD CULTURES**	—even if afebrile
14	**BS FOR MALARIA/MRDT**	

Management

- ICU care
- Protect airway : ETT and +/- Mechanical Ventilation in Comatose
- Nasogastric tube if comatose
- Hemodynamic support:
    - Adequate intravenous access;
    - Infuse isotonic crystalloid.
    - Add dextrose to intravenous fluids to avoid hypoglycemia
    - Consider pressor support (dopamine, epinephrine, norepinephrine) as needed to maintain adequate mean arterial pressure
    - Avoid nephrotoxic agents
- Give intravenous Rabeprazole 20mg iv. bd. or pantoprazole 40mg iv od

Treatment of complications

Complication	Intervention
Hepatic encephalopathy	**Lactulose**. ● Oral: 20-30 g (30-45 mL) every 1-2 hours to induce rapid laxation; adjust dosage daily to produce 2-3 soft stools; doses of 30-45 mL may be given hourly to cause rapid laxation, then reduce to recommended dose; usual daily dose: 60-100 g (90-150 mL) daily. ● Avoid Diarrhea & over distension (It means overdose). ● If Encephalopathy stage III or IV intubate & mechanically ventilate ●
Cerebral edema	● 20% Mannitol (0.5 to 1 g/kg) (for severe ↑ ICP or impending herniation) ○ 100mls IV For 30min should be administered as an intravenous bolus and then on an as-needed basis to ● Maintain the plasma osmolality between 310 and 325 mosmol/kg. It is essential to monitor urinary output closely while using mannitol. ○ Hypertonic saline to raise serum sodium to 145-155 mmol/L ● Nurse at 30^{O} to prevent exacerbation of Cerebral edema ● Hyperventilation: effects short-lived; may be of use for impending herniation NB: Dexamethasone has not proven to be effective in the treatment of cerebral edema caused by FHF and should not be administered
Acute renal failure	● Maintain arterial pressure ● Avoid nephrotoxic drugs
Infection and Sepsis	Empirical antibiotic treatment (No proven mortality benefit to empiric Abx j ○ Ceftriaxone 1g iv BD x 5/7 ○ Metronidazole 500mg iv 8hrly x 5/7
Hypoglycemia	Dextrose 5% or 50% to keep the blood glucose above 3.5mmol/L
Coagulopathy	● Vitamin k 10mg IM STAT ● FFP/Platelets/Cryoprecipitate if active bleeding or invasive procedures ● PPI prophylaxis (see dose above)

ACUTE PANCREATITIS

Introduction:

Clinical features (abdominal pain and vomiting) together with elevation of plasma concentrations of pancreatic enzymes (amylase or lipase) levels greater than 3 times the upper limit of normal in the absence of renal failure.

Goal of Management:

- To determine the cause
- To relieve clinical symptoms.
- To treat the cause
- To prevent complications.

Evaluation to determine the cause:

1. **History**- Pain radiates directly through the abdomen to the back, Nausea, Vomiting, Previous gall stones, Alcohol intake, FHx, Drug hx, Infections (H BV, CMV, VZV, HSV, Cryptosporidium and Salmonella).
2. **Physical examination**- Fever, tachycardia, abdominal tenderness, muscle guarding, abdominal distension, Jaundice
3. **Initial Investigation**-Amylase, Lipase, CBC, Liver function tests, U/S of gall bladder, BUN, electrolytes, lactic dehydrogenase and bicarbonate.
4. **Follow up Investigation**- Fasting plasma lipids, Fasting plasma calcium, CT and CRP

Consider: C-X-ray and ABG if there is any respiratory compromise.

DIAGNOSTIC CRITERIA:

Diagnosis of Acute pancreatitis should be established within 48hrs of admission.

Clinical features (abdominal pain and vomiting) together with elevation of plasma concentrations of pancreatic enzymes (Amylase or LipaseX3) and radiological support (Ultra sonography or CT Scan).

Relieve signs and symptoms

1. Supportive care: NPO, pain control
2. Hydration (Crystalloid or colloid) to maintain urine output of 0.5 ml/kg body weight.
3. Patient may require Patient Controlled Analgesia, morphine-first line.
4. Antibiotics- (Cipro and Metro) for 14/7 controversial (only in severe pancreatitis)
5. Treat metabolic complications (hyperglycemia, hypocalcemia).
6. Nutritional support

Treat the cause:- Cholecystectomy, treat infect ion, stop alcohol and stop offending drugs

Follow up to reduce chances of complications

-Subsequent image studies- pseudo cysts,

-Recurrent of pancreatitis

- Sterile necrosis doesn't need treatment

Reference

1. UK guidelines for the management of acute pancreatitis, 2015 by CD Johnson.
2. http://emedicine.medscape.com Accessed on 23rd May 2016.
3. American Gastroenterology Association Institute Medical Position Statement on Acute Pancreatitis. Gastroenterology 2007:132:2019-2021.

HEPATITIS B VIRUS

Evaluation and treatment of patients who are infected by Hepatitis B Virus

Introduction:

1. **Hepatitis B-** is a liver infection caused by the Hepatitis B virus (HBV)
2. **Acute HBV infection**- is characterized by the presence of HBsAg and immunoglobulin M (IgM) antibody to the core antigen, HBcAg.
3. **Chronic infection** -is characterized by the persistence of HBsAg for at least 6 months (with or without concurrent HBeAg).
4. **Inactive HBsAg carrier state** -means a persistent **HBV** infection of the liver without continual significant necro-inflammatory disease. (Undetectable **HBV** DNA levels and normal serum aminotrasnferase).

Goal of Management:

- To determine if it is chronic or acute
- To decrease clinical symptoms.
- To decrease complications.
- To delay/prevent hepatocellular carcinoma.

Initial evaluation:

1. History and physical examination and vaccination history
2. Family history of liver disease, HCC
3. Laboratory tests to assessments- FBP with platelets, hepatic panel and prothrombin time
4. Tests for HBV replication- HBeAg/anti-HBe, HBV DNA
5. Tests to rule out viral coinfections- anti-HCV, ant-HDV, ant- HAV and HIV
6. Tests to screen for HCC-AFP at baseline and in high risks patients, ultrasound
7. Consider liver biopsy to grade liver disease- for patients who meet criteria for chronic hepatitis.

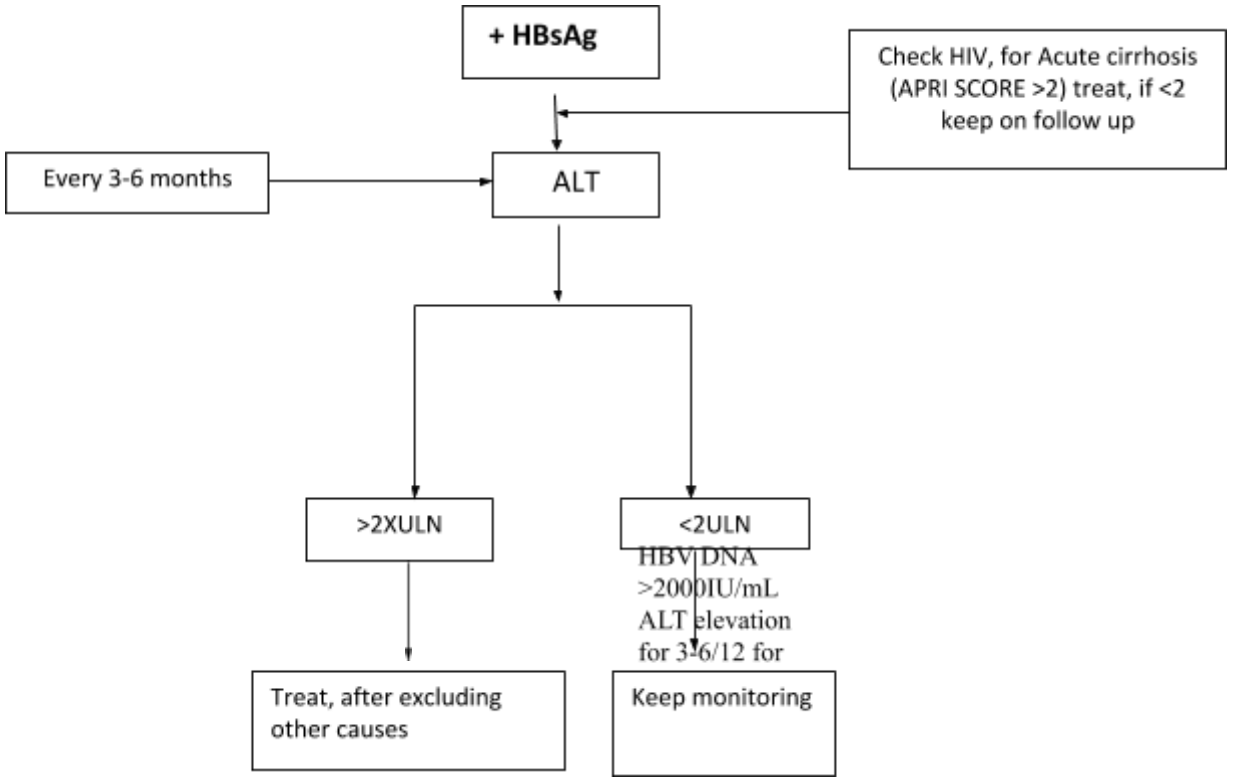
Suggested follow-up for patients not considered for treatment.
(HBeAg+, BHV DNA >20,000 IU/mL and normal ALT)

1. ALT every 3-6months, more often if ALT becomes elevated
2. If ALT levels are between 1-2xULN, recheck ALT every 1-3 months
3. Consider liver biopsy if age >40yrs
4. ALT borderline or mildly elevated on serial tests,
5. Consider screening for HCC in relevant population.

Inactive HBsAg carrier state

1. ALT every 3 months for 1 year, if persistently normal, ALT every 6-12 months.
2. If ALT>1-2XULN, check serum HBV DNA level and exclude other causes of liver disease.
3. Consider liver biopsy if ALT borderline or mildly elevated on serial tests or if HBV DNA persistently ≥2000 IU/mL.
4. Consider treatment if biopsy shows moderate/severe inflammation or significant fibrosis.

Treatment recommendations for chronic hepatitis B



APRI = * (AST/ULN) x 100) / platelet count (10^9/L Where; Upper limit o normal (ULN) =40um/L

RX: Adults: Tenofovir dosage: 300mg PO OD

Because of unavailability of Tenofovir monotherapy, we use truvada (Tenofovir+ Emtricitabine)

Reference:

1. WHO 2015, guidelines for prevention, care and treatment of Hepatitis B infection.
2. SHEA Guidelines (management of healthcare workers with Hepatitis B infection, David K et al, 2010.

INFECTIOUS DISEASES

MALARIA
FEVER OF UNKNOWN ORIGIN
TETANUS
MENINGITIS

MALARIA

The majority of infection in Africa is caused by P.falciparum. Other species include P. malariae, P. ovale, P. vivax and P.knowlesi.

Diagnosis
ALL cases suspected of malaria should have a parasitological test, with either Rapid Diagnostic Test (RDT) or Microscopy to confirm the diagnosis.

In severe malaria RDT shouldn't be used alone because microscopy is necessary for parasite quantification, speciation and treatment follow up.

In patients suspected with severe malaria and in other high risk groups (see below) absence or delay of parasitological diagnosis should **NOT** delay an immediate start of antimalarial treatment.

Other investigations
RBG, Urine analysis, FBP, RFTs

Uncomplicated malaria

Symptoms:
Headache, Lassitude, fatigue, abdominal discomfort, muscle and joint pain usually followed by fever, chills, perspiration, anorexia, vomiting and worsening of malaise.
Treatment: Uncomplicated Malaria
Treatment goal: To cure infection as rapid as possible and to prevent progression to severe disease. "Cure" is defined as elimination of all parasites from the body.
Artemisinin based Combination Therapy (ACT) Artemether(20mg) + Lumefantrine (120mg) (e.g ALu): 4 tabs start then 4 tabs after 8hrs on the 1st day and 4 tabs BD for the next 2days (a total of 3 days, this dose is for those with ≥ 35kg) Dihydroartemisinin (D) + Piperaquine (PQ): FDC tablets (e.g Duo-Cotecxin): 40mg (D) + 320 mg (PQ) ,2 tabs OD for 20 – 40 kg, 3 tabs OD for > 40kg (3 days) Others: Artesunate + Mefloquine: (e.g Artequin) Artemisinin + Piperaquine (e.g Artequick) Artesunate + sulfadoxine-pyrimethamine (SP)

A Patient who presents with symptoms of malaria and a positive parasitological test but with no features of severe malaria (see below) is defined as having uncomplicated malaria.

Treatment: Special risk groups with Uncomplicated P. falciparum Malaria	
Pregnant women in the 1st trimester	7 days with quinine + clindamycin. The alternative is Artesunate + clindamycin or quinine or Artesunate alone if clindamycin is not available
Patients co-infected with HIV	Avoid Artesunate + SP if they are being treated with cotrimoxazole. Avoid Artesunate + Amodiaquine if they are being treated with either Efavirenz or Zidovudine
Non-immune travellers	Treat travellers returning to non-endemic setting with ACT
Hyper-parasitemia$^{\mu}$	Are at increased risk of treatment failure, severe malaria and death and should be closely monitored in addition to receiving ACT
Excessive vomiting without features of severe malaria	Parenteral antimalarial treatment may be required until the patient can take oral medications. Then a full 3 day course of ACT should be given

$^{\mu}$ - Hyper-parasitemia: parasite density of ≥ 4% (~200,000/µL)

SEVERE MALARIA

It is a medical emergency defined as one or more of the following, occurring in the presence of P. falciparum asexual parasitemia and in the absence of an identified alternative cause.

Convulsions	
Impaired level of consciousness	
Prostration:	Inability to sit, stand or walk without support
Acidosis	Base deficit of > 8 mEq/L OR Plasma bicarbonate levels < 15 mmol/L OR Venous plasma lactate ≥ 5 mmol/L Clinically - severe acidosis manifests as respiratory distress (rapid, deep, labored breathing)
Hypoglycemia	Blood or plasma glucose < 2.5 mmol/L (45mg/dl)
Renal impairment	Serum Creatinine > 265 µmol/L or BUN > 20 mmol/L
Jaundice	Plasma or serum bilirubin > 50 µmol/L (3mg/dl) with a parasite count of > 100,000µL
Pulmonary edema	Radiological confirmed or O_2 saturation < 92% on room air with respiratory rate > 30/min, often with chest indrawing and crepitations on auscultation.
Severe malarial anemia	Hb ≤ 7 g/dl with parasite count > 100,000µL
Significant bleeding	Including recurrent or prolonged bleeding
Shock	Decompensated shock SBP < 80 with evidence of impaired perfusion (cool peripheries, or prolonged capillary refill > 3 seconds)

Hyper-parasitemia	P.falciparum malaria of > 10%
Treatment: Severe Malaria	
1st line: Artesunate 2.4mg/kg IV or IM given on admission, then at 12 hours, then at 24 hours. After 24hrs change to ACT if the patient can take oral, if not parenteral Artesunate should continue OD for a maximum of 7 days. (Given in 5% dextrose IV/IM concentration 10 mg/ml and 20mg/ml rounded up to the next whole number respectively, IV at a rate of 3-4ml/min)	
If Artesunate is not available, use Artemether in preference to quinine: 3.2 mg/kg as a loading dose by intramuscular injection, followed by 1.6 mg/kg daily until the patient is able to tolerate oral medication or for a maximum of 7 days.	
IV Quinine 10mg/kg up to 600mg IV every 8 hours, given in 500mls of 5% dextrose Must infuse very slowly (over 4 hours), can cause hypotension ● May cause hyper-Insulinemic hypoglycemia, especially during pregnancy, QT prolongation, arrhythmias, and ototoxicity	

● Reduce dose by 1/3 after 48 hours in cases of renal or hepatic impairment ● Quinine is safe to use in pregnancy, during all trimesters ● Increased risk of hypoglycemia during pregnancy; monitor the RBG ● Once PO administration is appropriate, continue Quinine TDS dosing for 7-10 days
Control Microscopy: Should be done at 24, 48 and 72hrs

Manifestations/ complications:

Manifestation or complication	Immediate management
Coma (cerebral malaria)	Maintain airway, place patient on his or her side, exclude other treatable causes of coma, intubate if necessary
Hyperpyrexia	Administer tepid sponging, fanning, a cooling blanket and paracetamol
Convulsions	Maintain airways; treat promptly with I.V or rectal diazepam/ Lorazepam. Check blood glucose
Hypoglycemia	See management on page……..
Severe anemia	Blood transfusion – whole blood
Acute pulmonary edema	Prop patient up at an angle of 45^{o}, give oxygen, diuretics, stop I.V fluids, intubate and add positive end-expiratory pressure or continuous positive airway pressure in life-threatening hypoxemia.
Acute kidney Injury	See management on page……..
Spontaneous bleeding and coagulopathy	Transfuse with fresh whole blood (cryoprecipitate, fresh frozen plasma and platelets, if available) give vitamin K injection.
Shock	See management on page……..

FEVER OF UNKNOWN ORIGIN

Definition: A temperature higher than 38.3ºC on several occasions lasting for at least 3 weeks without an established etiology despite intensive evaluation and diagnostic testing. It includes:

***Nosocomial FUO**: fever occurring on several occasions in a patient who has been hospitalized for at least 24hrs and has not manifested an obvious source of infection that could have been present before admission.*

***Immunodeficient (neutropenic) FUO:** Recurrent fever in a patient whose neutrophil count is ≤500 per mm^3 and who has been assessed for 3 days without establishing an etiology for the fever.*

***HIV-associated FUO**: Recurrent fever over a 4-week period in an outpatient or a 3-day period in a hospitalized patient with HIV infection.*

Diagnosis:

The following evaluation should have been performed and should have been unrevealing:

- FBP & ESR or CRP, differential and platelet count
- Blood cultures (3 sets from different sites over several hours without administering antibiotics)
- Routine blood chemistries, including liver enzymes and bilirubin
- Hepatitis serology (if liver tests abnormal)
- Urinalysis, microscopy and culture
- CXR
- BS for malaria parasites

If any signs or symptoms point to a particular organ system, further testing, imaging, and/or biopsy should be pursued.
Since antibiotic use is the main reason for culture negativity, recent or current antibiotic use should be documented.
Fever of unknown origin is more often caused by an atypical presentation of a common entity than by a rare disorder.

Etiology:

Infections:

- Tuberculosis
- Abscesses :
- Typhoid fever
- *Zoonosis: Brucellosis, Leptospirosis, Q-fever, Rickettsioses*
- Sub-acute infective endocarditis
- Malaria
- Viral infections: EBV, CMV,

Chikungunya

- Fungal: Cryptococcus neoformans

Malignancies:

- Lymphoma
- Leukemia

- Renal cell carcinoma
- Hepatocellular carcinoma
- Other tumors metastatic to the liver.

Noninfectious inflammatory diseases:

- SLE
- Vasculitis
- Rheumatoid arthritis

Drug fever: as an adverse reaction to drugs e.g. antibiotics, sulfa drugs, antidepressants and antipsychotics

Undiagnosed causes:

Despite intensive efforts, some FUO cases remain undiagnosed. In some cases, the diagnosis can be established months to years later

Evaluation:

- A detailed **history and meticulous physical examination** which will guide the choice of investigations
- **Frequent patient reassessment**
- Blood cultures
- ESR or CRP
- Serum lactate dehydrogenase
- HIV antibody test and viral load
- Rheumatoid factor
- Creatine phosphokinase
- Antinuclear antibodies
- Serum protein electrophoresis
- CT scan of abdomen and chest

The primary evaluation and diagnostic workup can suggest a directed biopsy

Treatment:

- Depends on the etiology.
- The diagnostic evaluation may fail to identify an etiology in as many as 30-50% of patients.
- Most adults who remain undiagnosed have a good prognosis.
- For Drug fever: discontinue the offending drug
- Neutropenic fever: use empiric broad spectrum antibiotics

TETANUS

Definition: a disease of the nervous system characterized by muscle spasms, caused by toxins produced by the bacterium *Clostridium tetani*

Symptoms: increased muscle tone at rest and continuous spasms (generalized, localized or cephalic) with relatively preserved consciousness. Trismus or risus sardonicus (facial muscle spasms) or opisthotonus (spinal extensors spasm) may be observed. Autonomic instability is a later finding, characterized by restlessness, tachycardia, sweating, labile blood pressures, fever and/or cardiac arrhythmias.

Differential diagnosis: dystonia, rabies, meningitis, strychnine poisoning, cerebral malaria, seizure disorder, head/neck infection (including dental)

Diagnosis: Tetanus is a clinical diagnosis. The following investigations should not delay treatment:
1. Blood glucose
2. HIV test
3. Complete blood count
4. Creatinine
5. Malaria test

Goals of care: airway management, halt toxin production, neutralize unbound toxin, control muscle spasms, manage autonomic dysfunction, supportive measures, ensure immunity

Care: Admit to ICU.

1. Airway management

Intubate if airway is compromised. Consider early tracheostomy if laryngospasm or heavy secretions; often patients will need this eventually given prolonged mechanical ventilation

2. Halt toxin production:

a. Anti-tetanus antibiotic: metronidazole 500 mg IV q8h for 7-10 days
*Alternative: penicillin 4 million units q4h for 7-10 days
*Note: if there is surrounding cellulitis, suspicion of polymicrobial or evidence of systemic infection, consider broadening coverage

b. Early debridement of wound and exploration for foreign bodies. Keep debrided wound open to the air and clean. If wound bed appears infected, consider further debridement

3. Neutralize unbound toxin:

Human tetanus immunoglobulin (HTIG): 500 IU IM/IV
*Alternative: Equine antitoxin 1500-3000 IU IM/IV, after testing for hypersensitivity

4. Control muscle spasms

a. Put patient in a low-nose and low-light environment and minimize unnecessary interactions to avoid provoking spasms

b. Sedatives:
i. Diazepam 10-30 mg IV/PO (via NGT) q8h, though may be given in higher doses for uncontrolled spasms, monitoring carefully for respiratory depression and caution with higher doses given IV (lactic acidosis)
ii. If still uncontrolled: Chlorpromazine 25-50 mg PO/IM q6h
iii. If still uncontrolled and severe: Phenobarbitone 50-120 mg IV BID

c. Magnesium sulfate – primarily for autonomic dysfunction (dosage below) but also reduces need for other drugs to control spasms

d. Neuromuscular blocking agents – consider only for intubated patients with severe, uncontrolled spasms despite escalated therapy:
i. Pancuronium:
Initial: 0.06-0.1 mg/kg IV
Maintenance: 0.8-1.7 mcg/kg/min IV infusion

ii. Vecuronium:
Initial: 0.08-0.1 mg/kg IV
Maintenance: 0.8 – 1.2 mcg/kg/min IV infusion

5. Control of autonomic dysfunction:
a. Magnesium sulfate 40 mg/kg IV initially (push slowly over 30 min), followed by MgSO4 infusion at 2 g/hr IV for patients $\geq$ 45 kg, or 1.5 g/hr for patients < 45 kg if spasms still uncontrolled (monitor patellar reflexes q6-8h for signs of Mag toxicity)
b. Labetalol 0.25 to 1.0 mg/min (avoid selective beta-blockers due to reports of sudden death)
c. Morphine 0.5 to 1.0 mg/kg/hr IV infusion (watch for respiratory depression if using benzodiazepine and not intubated)
d. Atropine 0.5 mg q3-5 min IV, not to exceed a total of 3 mg or 0.04 mg/kg for treatment of bradycardia

6. Supportive care:
a. Hypotensive or low urinary output, NS bolus (consider 30 cc/kg) and possibly further blousing and/or maintenance given high insensible losses
b. Fever: Paracetamol 1 gm q8h (but do not forget this may be sign of autonomic dysfunction or of systemic infection and thus may need further management)
c. Pain: Diclofenac 50 mg PO q8h. May use morphine but be cautious in combination with benzodiazepines (respiratory depression)
d. Nutritional support: begin early, via NGT, given high energy demands
e. Decubitus ulcers: Frequent turning given high risk
f. Deep vein thrombosis prophylaxis: start early and continue throughout immobility
g. Physical therapy: begin as soon as spasms cease

7. Immunize:
a. Tetanus toxoid 0.5 mL IM given during initial hospitalization (but not together at same site as tetanus immunoglobulin)
b. Boost with same dose 1-2 months after first dose

c. Boost with same dose 6-12 months after second dose

MENINGITIS

Definition: Inflammation of the leptomeninges, the tissues surrounding the brain and spinal cord.

Clinical classification	
Acute: (days/ hours)	**Chronic: (weeks)**
● Aseptic/Viral meningitis (VM) ● Bacterial meningitis (ABM)	● Cryptococcus meningitis (CM) ● Tuberculous meningitis (TBM)

Symptoms:

ABM	Acute onset headache, fever, and meningism. Others: Altered mental status, seizures, photophobia, nausea, vomiting, backache and lethargy. Hemorrhagic skin rash: suggestive of meningococcal meningitis.
VM	As above, with possible addition of extra-CNS signs and symptoms (i.e. herpetic lesions in HSV)
CM	As above, presenting over 1-2 weeks with relative absence of meningism.
TBM	Usually more chronic (> 4 weeks), with prodrome of headache, fever, vomiting, anorexia, followed by meningism progressing to focal neurological deficits, seizures and coma. M or may not have signs/symptoms of extra-CNS TB.

Differential diagnosis:

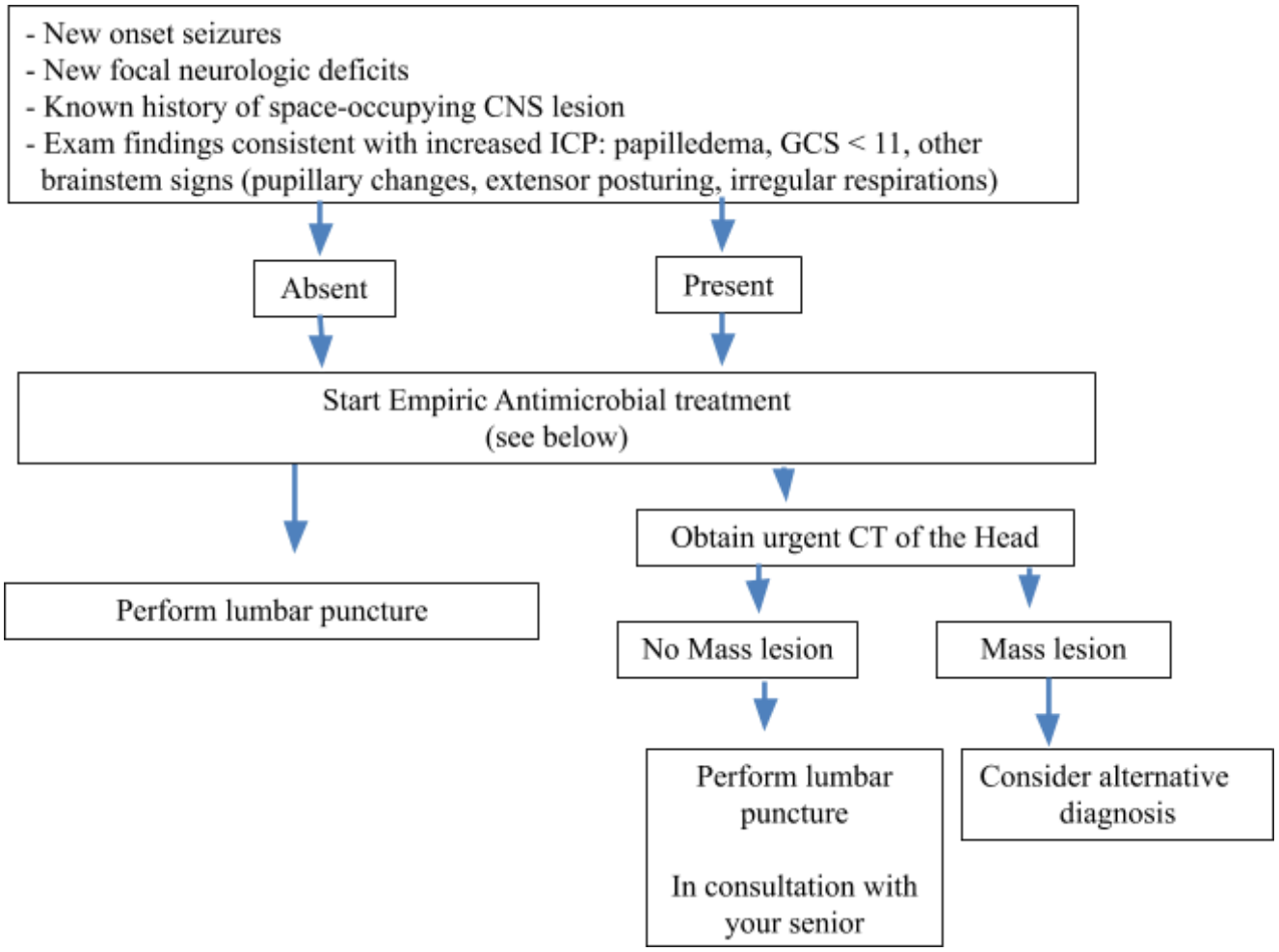
- Encephalitis, cerebral malaria, brain abscess, tetanus, rabies, CNS hemorrhage, other CNS lesions/infections such as toxoplasmosis or tuberculoma.

Investigations:

- RBG, FBP, HIV serology, +/- Lumbar puncture, Blood culture, MRDT +/- BS-MPS, Other tests (liver, kidney) per clinician's discretion
- For Lumbar puncture: Check opening pressure. Examine color/quality of CSF. Send for: Gram stain, WBC count (total and differential), Cryptococcus antigen/ India ink, Glucose, Protein, Smear and culture for AFB (to maximize sensitivity, send at least 6 mL),

	ABM	VM	CM	TBM
Opening pressure (n = <20cm in adults)	**++**	**normal or ++**	**+++**	**++**
Appearance (n= clear)	**Cloudy/ Purulent**	**Clear**	**Clear/ cloudy**	**Yellow/ cloudy**
Cells/mm^3 (n=<5mm^3) Main type	**++ Neutrophils**	**normal or ++ lymphocytes**	**normal or ++ lymphocytes**	**++ Lymphocytes**
Glucose(n=> 50% plasma)	**++**	**normal**	**normal or -**	**normal**
Protein (n=<0.5gm/L)	**++**	**normal or ++**	**normal or ++**	**normal or ++**
Diagnosis confirmed	**Gm stain & culture**	**PCR/ Culture**	**India ink stain, CRAg/ Culture**	**PCR/ Culture**

Approach to the patient: If there is a clinical suspicion of meningitis, assess for the following:



1. Suggestive of Bacterial meningitis (see above) or in empiric treatment:

Drug	Dose/Route	Frequency	Duration
Ceftriaxone OR Cefotaxime	2gm IV 2gm IV	12 hourly 4 hourly	10-14 days 10-14 days
Or			
Penicillin & Chloramphenicol	2.4gm or 4 MU IV 1 gm IV	4 hourly 6 hourly	10 – 14 days 10 – 14 days

2. Suggestive of Cryptococcus Meningitis* or empiric treatment in HIV seropotive patients:

Drug	Dose/Route	Duration
Fluconazole Induction phase Consolidation phase Prophylaxis	 1200mg po/iv/OD 800mg po/OD 200mg po/OD	 2 weeks 8 weeks Until CD4 is above 200 cells/mm^3

*In patients confirmed with serum/CSF CrAg or India ink +ve the 1st line treatment should be: **Amphotericin B 0.7 – 1.0 mg/kg IV QD + fluconazole 800 mg PO QD** in the 1st 2weeks then the consolidation phase.

To minimize risk of renal damage, give NS infusion before and after. To palliate infusion reaction, pre-medicate with acetaminophen and/or diphenhydramine.

Management of increased ICP:
All patients will need LP, regardless of the opening pressure on day 0,3,7 and 14 if the opening pressure is elevated in any of these readings, daily therapeutic LPs need to be done until the opening pressure is < 20 cm.

c. Antiretroviral therapy – if HIV positive and already on ART, continue treatment. If treatment-naïve, do not start ART right away

2. Viral meningitis – consider if symptoms do not improve with antibiotics, CSF is consistent with viral etiology and/or other etiologies are ruled out by tests.

a. HSV meningitis: **Acyclovir – 10 mg/kg IV q8h**

b. Other viruses: The ones to keep in mind that warrant different approaches are primary HIV infection, CMV and VZV.

3. Tuberculous meningitis – if suspected, do not delay therapy for diagnostic work-up!

a. Anti-tuberculous therapy – Intensive phase: **Isoniazid 300 mg PO QD, Rifampicin 450-600 mg PO QD, Pyrazinamide 1.5 – 2 g PO QD, and either Ethambutol 15 mg/kg PO QD or Streptomycin 20 mg/kg IM QD.**

b. Glucocorticoids – **Dexamethasone 0.4 mg/kg IV QD or prednisone 60 mg PO QD**. 2 weeks of initial dose then taper for total duration of 8 weeks.

c. Antiretroviral therapy – if HIV positive and already on ART, continue treatment. If treatment-naïve, do not start ART right until after 2 weeks

BODY FLUIDS AND KIDNEY DISEASES

ACUTE KIDNEY DISEASE

Definition: AKI is defined as an increase in SCr by ≥26.5 µmol/l within 48 hours; ***OR*** an increase in SCr to ≥1.5 times baseline ***OR*** a urine volume of <0.5 ml/kg/h for 6 hours.

Goal of therapy:

1. To reverse and prevent further damage by treating reversible causes promptly
2. To maintain fluid and electrolyte and acid base balance
3. To prevent and treat associated complications

Approach to management:

Every suspected AKI patient should get a urine dipstick, IV line access, urine catheter and a input/output chart for daily monitoring, FBP, serum creatinine, BUN, Electrolyte, RBG, Abd USS and urine microscopy.

CATEGORY	RECOGNITION	MANAGEMENT
Pre-renal - Decreased perfusion (vomiting, diarrhoea, burns, haemorrhage) - Drugs (diuretics, ACE inhibitors, ARBs, NSAIDs, iodinated contrast) -Liver disease - Cardiac failure	History suggestive of prerenal etiology Look for signs and source of infection – fever Volume assessment- BP, PR, RR, CRT, JVP Ix: FeNa < 1% Urine microscopy: Bland sediment, SG >1.020 Cardiac workup for CCF patients blood and urine C/S.	Hypovolaemia: Fluid challenge: IV fluid 500 mls NS bolus over 15 mins, reassess if still hypovolemic give 250-500mls in 15 mins. (Monitor JVP, RR, and measure urine output) Sepsis -early antibiotics (see sepsis protocol). CCF- cardiac output should be maintained. Consider vasopressors (Dopamine/Dobutamine) Review patients' current medications, hold BP lowering agents.
Intrinsic -Acute tubular injury ● Prolonged prerenal AKI ● Sepsis ● Toxic ATN : Drugs (aminoglycosides, cisplatin, tenofovir,methotrexate, iodinated contrast) ● Other (rhabdomyolysis, snake bites) -Tubulo-interstitial ● interstitial nephritis: drugs (PPIs, penicillins, NSAIDs) ● sarcoidosis ● tubular obstruction (myeloma, tubular crystal nephropathy) -Glomerulonephritis -Vascular ● Hypertensive emergency ● Pre-eclampsia ● Vasculitis ● renal vein thrombosis ● renal infarction	Hx. Suggestive of intrinsic renal cause Ix CK, uric acid, kidney biopsy (where indicated) Inflammatory markers: ESR, ANA, ANCA if indicated Urine dipstick: Protein, RBCs Urine microscopy: Muddy brown casts for ATN; WBC casts for AIN; RBC casts for glomerulonephritis; +/- RBC or protein. SG =1.010 – 1.020	Avoid Nephrotoxic agents, Dose adjustment for the renally excreted drugs. Some drugs must be avoided e.g: NSAIDS Iodine Contrast Aminoglycosides ACEIs/ARBs
Post-renal (Obstructive Uropathy) Kidney stones Prostatic enlargement (benign/malignant) Urethral stricture Bladder cancer & other pelvic Tumours	Hx of Haematuria , flank pain, LUTS Ix: U/S Kidney, ureter, bladder KUB X-ray (if suspect stones)	Target to relieve obstruction Consult urology to assess for surgery/ further evaluation

ATN -acute tubular necrosis, ACE- angiotensin-converting enzyme, ANA- antinuclear antibody, ANCA- antineutrophil cytoplasmic antibody, ARBs - angiotensin receptor blockers, CRT - capillary refill time, CCF - Congestive cardiac failure, LUTS - lower urinary tract symptoms, PR - pulse rate, RR - respiratory rate, Scr - serum creatinine

*Baseline SCr is defined as the lowest within the past 7 days and if not known, the lowest in the previous 3 months

Complications:

COMPLICATION	RECOGNITION	RESPONSE
Hyperkalaemia Mild (5.5-5.9 mmol/L) Moderate (6.0 – 6.4 mmol/L) Severe (≥6.5 mmol/L)	Do an ECG (findings in hyperkalemia: 'tented' T-waves; PR prolongation; Flattened P waves; Widening of the QRS complex) Bradycardia Management guided ECG findings together with clinical scenario and rate of rise of K. Serial measurements of electrolytes indicated for monitoring based on individual case.	**Mild** Use K binding resin (e.g. Sodium Polystyrene Sulfonate 15 g P.O in 60mls NS TID or 30-60g in 100mls Rectally BID in 24 hours. Alternatively lactulose 30mls tds **Moderate to severe** or rapidly rising K, give ● 50mls of Dextrose 50% + Actrapid 10 IU IV over 5mins ● **OR** Nebulized salbutamol 10mg **ADD** 10 ml Calcium gluconate 10% over 10 mins repeated as necessary (for severe cases or ECG abnormalities to stabilize myocardium) Please see indications for RRT
Volume overload	↑ JVP, peripheral oedema, pulmonary oedema. Inx : CXR, ABD US to assess IVC	Nurse in propped up position Oxygen via nasal prongs or face mask Give Furosemide 120 mg stat then asses after 2 hours. If Urine output >200mls continue with furosemide 120 mg Bid If <200 mls consider Dialysis Restrict fluid & Salt intake Daily weights indicated
Metabolic Acidosis	Altered mentation with increased RR, kussmaul's breathing Ix: ABG	Sodium bicarbonate Non life threatening:2-5 mEq/kg IV infusion over 4-8 hrs Severe: 90-180 mEq/L in the 1st hr. Monitor serum potassium
Hypertension	High BPs +/- cardiovascular complications Ix: CXR and cardiovascular workup	Treat with CCB eg Nifedipine 20 mg po bd increase to max 120 mg/day in divided doses Add on drugs: – B-blocker,Hydralazine Monitor BP
Coma/encephalopathy	Patient confused, disoriented, comatose with no focal neurology fundoscopy All baseline investigations	Nursing support, Oxygen, NGT See indications for RRT
Uremia	-Uremic Pericarditis: Chest pain, DIB, Pericardial rub. -Uremic gastritis: Abdominal pain, vomiting, UGIB -Bleeding	Gastritis: Consider PPI Bleeding: Resuscitation if required according to UGIB protocol +/- blood transfusion Plan the patient for dialysis

Indications for dialysis:

Acidosis (Intractable); **E**lectrolyte disturbances (Intractable hyperkalemia); **I**ngestion of toxins; **O**verload of fluid (mainly pulmonary oedema that cannot be corrected with diuresis) and **U**remia (encephalopathy, pericarditis, bleeding)

References:

1. Acute Kidney Injury Work Group. Kidney Disease: Improving Global Outcomes (KDIGO). KDIGO Clinical practice guideline for acute kidney injury. Kidney Int Suppl 2012;2:1-138. www.kdigo.org/clinical_practice_guidelines/pdf/KDIGO%20aki%Guideline.pdf
2. Acute Care Toolkit 12. Acute Kidney Injury and intravenous fluids September 2015.Royal College of Physicians (RCP)

CHRONIC KIDNEY DISEASE

Definition: Diagnosed when there is irreversible and progressive impairment of kidney function (GFR < 60ml/min) for more than 3 months

Duration of symptoms (> 3 months), kidney size (small less than 9cm each) and increased echogenicity point towards chronic kidney disease rather than Acute kidney injury.

Criteria for CKD either of the following present for ≥ 3 months

Markers of kidney damage	Albuminuria >30mg per day Urine sediment abnormalities (e.g., hematuria,red cell casts etc) Electrolytes and other abnormalities due to tubular disorders Abnormalities detected by histology Structural abnormalities detected by imaging History of kidney transplantation
Decreased GFR	GFR $^{<}$60mL/min/1.73m^2

GFR <60 mL/min/1.73m2 is associated with a higher risk of complications of CKD:
• Drug toxicity
• Metabolic and endocrine complications
• CVD and death

The eGFR is primarily determined by serum creatinine (SCr), and the current gold standard for estimating GFR is the body surface area-normalized, 4-variable, Modification of Diet in Renal Disease Study (MDRD) Equation based on SCr, age, gender, and ethnicity.

eGFR (mL/min/1.73 m2) = 186 × (SCr)–1.154 × (Age)–0.203× (0.742, if female).

Replace the constant 186 with 175, if the laboratory uses a standardized SCr (IDMS method). This reduces eGFR by 6%.

The goal of Management.

1. To treat/manage possible causes
2. Retard progression to ESRD
3. Reduce cardiovascular risk
4. To maintain fluid and electrolyte balance
5. Provide nutritional support
6. Prevent or treat associated complications

Initial Assessment.

- **o** *Find out the cause, if possible.*

- *o* *Baseline Investigations*

  - *Urinalysis*- microscopy
  - *Urine Multistick (Bedside)*
    - Protein, pH, Specific gravity, Glucose, Leucocytes, Red blood cells
  - If Proteinuria present - 24hr urine estimation or UPCR
  - *Biochemistry*-Serum creatinine, BUN, Serum Electrolytes (K^+, Na^+, Ca^{2+}), RBG, Uric acid, Total protein, Fasting lipid profile (LDL, HDL, total cholesterol).
  - *Full Blood Picture (Haemoglobin and RBC indices, Platelets and WBC including differentials)*.
  - *ECG if indicated (obvious cardiovascular disease and CKD-3 and above)*
  - *U/S Abdomen and Pelvis (Kidney, ureters, prostate and Bladder)*
  - *X-ray of Kidney, Ureter and Bladder (KUB) if indicated*

- ***o*** *Estimate the GFR and stage using formula CKD-EPI Cockcroft-Gault or Modification of Diet and Renal disease* (MDRD)

GFR CATEGORIES IN CKD

Stage	GFR(mL/min/1.73m^2	Terms	Intervention
1	≥90	Risk factors: age >60 years, obesity, autoimmune disorders, DM, HTN, kidney stones, ADPKD, prior AKI/ARF, UTIs, toxic drug exposures, and FH of CKD	Screen for general and specific conditions Screen for CKD w/ eGFR Initiate CKD risk reduction /intervention strategies
2	60-89	Kidney damage with mild GFR decrease (urinary, imaging or histologic abnormalities) Most lower GFRs in this range are due to age-related GFR decline and do not require evaluation, if no proteinuria is present	Estimate CKD progression rate Diagnose and treat CVD risk factors and comorbid conditions
3a	45-59	Moderate decline of GFR Complications more frequent at CKD Stage 3B as GFR to <45 mL/min/1.73 m2 Protein is a serious CV risk factor and has prognostic importance for progression of CKD	Estimate CKD progression rate Diagnose and treat CVD risk factors and comorbid conditions Kidney imaging study, eg, US or CT Consider Nephrology Consultation
3b	30-44	Moderately to severely decreased	Get a nephrologist review
4	15-29	Severe decline of GFR Major increase in CVD risk, ie, CKD Stage 4 should be considered equivalent to a major CVD clinical event	Nephrology consultation with transition of management and care Initiate decisions regarding kidney replacement therapy, vascular access, and kidney transplant Diagnose and treat CVD risk factors and comorbid conditions Adjust drug dosing for CKD stage
5	<15	Kidney failure	Consider RRT Urgent indications for dialysis ● Uncontrolled severe hyperkalemia ● Uncontrolled fluid overload ● Uncontrolled metabolic acidosis ● Uremic symptoms (intractable vomiting, pericarditis and encephalopathy)

Management.

Presentation/Complication	Management
1. Fluid overload 3. raised JVP 4. Pulmonary edema 5. Generalized edema	-Oxygen therapy if tachypnoeic -Limit fluid intake(less than 1 Litre/24hrs) if there is fluid overload. Intake (oral+IV) based on: urine output plus 500ml /24hrs. -Restrict salt intake -Avoid nephrotoxic medications -Diuretics. The use of furosemide at higher doses (eg. 120mg iv. 12hrly) may be necessary

2. Hyperkalemia > 6.5 mmol/L with or without ECG changes ● Tall "tented" T-waves, ● flattened P-waves, ● increase P-R interval ● widening of the QRS complex	<u>Cardio-protection</u> - Give 10ml of calcium gluconate 10% iv. Over 2 minutes repeated as necessary if severe ECG changes <u>Shift potassium intracellularly</u> - Measure RBG before actrapid. Repeat after one hour if symptomatic. -10-20 IU of actrapid iv. + 50mls of glucose 50%iv over 20min. (this effect lasts for only 2-3hrs only) - If RBG above 14mmol don't give 50% glucose - Salbutamol inhalation - Sodium Bicarbonate <u>Remove potassium from the body</u> - Loop diuretics - Polystyrene sulphonate 15-30g po with 30-60mls of water/NS 8hrly for 24hrs. (caution – may cause intestinal perforation) Low potassium diet (avoid bananas, tomatoes, citrus and grape fruits) **Hyperkalemia protocol** **Causes of Hyperkalaemia** ● Pseudohyperkalaemia ● Test tube haemolysis - ensure samples arrive at the laboratory within 5 hours and NEVER refrigerate samples ● EDTA contamination (from FBC sample tube) ● Prolonged tourniquet time ● Sample taken from drip arm ● Acute kidney injury ● Chronic kidney disease ● Drugs (potassium supplements, potassium-sparing diuretics such as amiloride, aldosterone antagonists such as spironolactone and eplerenone, ACE inhibitors, angiotensin II antagonists, NSAIDs, heparin, -blockers, digoxin poisoning) ● Acidosis, including diabetic ketoacidosis ● Mineralocorticoid deficiency (e.g. Addison's disease) ● Endogenous (tumour-lysis syndrome, rhabdomyolysis, trauma, burns) ● Please note that this list is not comprehensive and that other causes may need to be considered. **Clinical Assessment** ● Urine output – very important. If oliguric, medical treatment much less likely to work. ● Review potassium intake e.g. IV fluids, potassium supplements, diet. ● Review drugs: ACE inhibitors, Angiotensin II Antagonists, potassium-sparing diuretics (e.g. amiloride, spironolactone, eplerenone), potassium supplements, β -blockers (small effect). ● Review history for possible causes of renal disease or major tissue destruction. ● Review recent biochemistry results, in particular renal function and recent potassium levels. ● Fluid status – signs of dehydration or fluid overload. ● Potassium levels may be assessed on a venous blood sample using a point of care blood gas analyser in emergencies (results correlate well). This must be followed up with a formal laboratory measurement. **Investigations**

- 12-lead ECG
- U&Es, venous bicarbonate, glucose, FBC
- If unwell, consider venous blood gases and lactate.

Treatment of Hyperkalaemia

- Exclude pseudohyperkalaemia.
- Stop all potassium supplements (IV and oral).
- Review patient's medication for possible contributors to hyperkalaemia and or acute kidney injury.
- Reduce dietary K+ intake.
- Ensure adequate hydration and urine output.
- If potassium ≥6.5mmol/l or ECG changes monitor patients cardiac rhythm until it is stable and potassium level is in range.

After the above, there are three steps in managing hyperkalaemia. If serum K+ <6.5 mmol/L and there are no ECG changes/symptoms of hyperkalaemia

Then omit Step 1 and 2 and move on to Step 3.

Step 1: Reduce cardiac cell membrane excitability

CALCIUM GLUCONATE 10% 10 mL IV over 5 mins

- This does not lower the serum potassium but protects the cardiac membrane.
- ECG changes should improve within 1 to 3 minutes and its effect lasts for approximately 30 minutes.
- If necessary doses may be repeated after 5 minutes up to maximum 3 doses.
- If the patient is taking digoxin, the calcium gluconate should be given slowly (mixed with 100mL 5% glucose and given over 20 minutes) as rapid calcium administration may precipitate myocardial digoxin toxicity.
- Never give at the same time as sodium bicarbonate via the same access site due to the risk of precipitation.

Step 2: Shift potassium from extracellular to intracellular space

Shifting potassium intracellularly is a useful holding measure in life-threatening hyperkalaemia. However, it does not reduce total body potassium, and after two to six hours, there is an efflux of potassium back out into the extracellular space resulting in serum levels as high or sometimes even higher than at the outset. Therefore, any of the steps in section 2 must be combined with those in section 3, and serum potassium must be regularly rechecked.

INSULIN ACTRAPID.

- 10 units in 50 mL of Glucose 50% IV over 30 minutes via syringe pump
- Always give into a large vein as it is irritant.
- Reduces serum K+ by 0.65 - 1.0mmol/L.
- Monitor blood glucose before and after infusion, every 15-30 minutes and hourly for up to 6 hours as there is a risk of late hypoglycaemia.

Then consider:
SALBUTAMOL

- 10mg nebulised
- Can be given for an additive effect to insulin/glucose.
- Reduces serum K+ by 0.62 - 0.8mmol/L but response has been shown to be inconsistent – this step is optional and must not be used as single agent.
- Caution in patients with ischaemic heart disease and history of cardiac arrhythmias (avoid/use lower dose).
- Response reduced in patients on -blockers and digoxin.

SODIUM BICARBONATE.

- 1.4% 500 mL IV over 2 hours – ONLY CONSIDER IF pH < 7.2 and on advice of Renal registrar or Critical Care.
- The use of sodium bicarbonate is controversial in patients with acidosis. There is insufficient evidence to justify routine use and use of sodium bicarbonate is associated with significant risk of sodium and fluid overload (e.g. pulmonary oedema)
- **It should therefore only be used after discussion with a Renal Registrar/Consultant or Critical Care**.
- Risk of tetany in patients with chronic renal failure and underlying hypocalcaemia.
- Never give at the same time as IV calcium via the same access site (risk of precipitation).

After any of the above steps: **Recheck potassium 2 hours, and 4-6 hours (risk of rebound hyperkalaemia), after treatment. Check kidney function at 4-6 hours post-treatment and then daily.**

- If K+ remains ≥6.5mmol/L or ECG changes persist, contact on call Renal Registrar/Consultant urgently.
- If potassium has improved but the patient is oligo/anuric or developing acute kidney injury contact the Renal Registrar/Consultant on-call urgently as the potassium will almost certainly rebound.

Step 3: Reduce total body potassium
3a) REDUCE POTASSIUM INTAKE

- Low potassium diet (consider dietetic review and order appropriate diet, remember food from home).
- Avoid drugs which raise potassium.

3b) PROMOTE URINARY POTASSIUM LOSS

- Monitor fluid balance and encourage good urine output by ensuring adequate hydration with oral or IV fluids. Normal saline 0.9% is preferable as long as the patient is not significantly overloaded. Avoid fluids containing potassium e.g. Hartmann's.
- Treat hypotension – remember to review the drug chart e.g. antihypertensives.
- If well hydrated, consider starting or increasing the dose of a loop diuretic.

3c) REMOVE EXCESS POTASSIUM

- Calcium Resonium has a slow onset of action (at least 2-6 hours)
- Prescribe Calcium Resonium 15g PO TDS.
- Removes K+ from gut by ion exchange thus lowers total potassium load.
- Each gram of Calcium Resonium removes approximately 1mmol potassium from the gut.
- **Caution**: contraindicated in patients with pre-existing hypercalcaemia;
- May cause constipation – co-prescribe Senna 2 tablets twice daily.
- May not be necessary if the obvious cause of hyperkalaemia has been identified and corrected.
- Oral Calcium Resonium is unpalatable and poorly tolerated.
- Monitor U&Es daily and consider stopping when K^+ <6.0mmol/L. Once K^+ ≤5.5mmol/L discontinue treatment.
- If oral route not available consider Calcium Resonium enema 30g per rectum(PR) daily; however this is poorly tolerated by patients (please contact pharmacist or on call pharmacist for guidance on administration).

3d) DIALYSIS

If the patient does not respond to the above measures dialysis will be required.
DIALYSIS IS LIKELY TO BE NEEDED IF POTASSIUM VERY HIGH, PATIENT IS OLIGO/ANURIC, PATIENT IS ALREADY ON LONGTERM DIALYSIS OR HAS ADVANCED CKD. In these situations contact the Renal Registrar/Consultant on-call urgently to discuss management.

Notes on Treatment

Treatment	Mechanism of Action	Time to onset of action	Duration of action	Achievable reduction of serum K^+
Calcium Resonium	Ion exchange resin that exchanges sodium for potassium as it passes through intestine	2-6 hours or longer	4-6 hours	Unknown
Calcium gluconate	Antagonizes cardiac membrane excitability	Immediate	5minute	N/A
Insulin Actrapid with glucose	Increases intracellular uptake of K^+ via Na-K ATP pump	Within 15 minutes	60 minutes	0.65-1mmol/L

Nebulized sulbutamol	Increases intracellular uptake of K^+ via Na-K ATP pump; response reduced by beta blockers and digoxin.	Acts in 30mins, up to 90mins	1-3 hours	0.62-0.98mmol/L
Sodium bircarbonate	Corrects acidosis and thus promotes intracellular uptake of K^+	After 60mins, effect variable	Unknown	Unknown

Low potassium diet

Food group	**High potassium food to limit**	**Lower potassium choices**
Fruit	Bananas, avocado, currants (black, red or white), dried fruit(raisins, sultanas, dates, dried apricots).	Apple, pear, satsuma, clementine, 10 grapes, tinned fruit.
Starchy Foods	Jacket or baked potatoes; oven, microwave or retail chips; manufactured potato products such as hash browns, potato waffles, frozen roast potatoes or potato wedges. Fried cassava, yam or sweet potato. Taro, plantain and parsnip. Breakfast cereals containing lots of dried fruit, nuts or chocolate for example, muesli, granola, fruit and fibre	Boiled potatoes or potatoes which have been par-boiled before roasting or frying. Pasta, rice, noodles, couscous, and breads – these are all much lower in potassium than potato. Boiled cassava, yam or sweet potato. Suitable breakfast cereals include rice or corn based cereals, wheat biscuits, Shredded wheat®, Special K® and porridge oats

Snacks	Potato crisps, chocolate, fudge, nuts. Biscuits and cakes containing lots of dried fruit, nuts or chocolate.	Corn, rice, wheat or maize based snacks, popcorn, boiled or jelly sweets, marshmallows, mints. Plain biscuits and cakes such as rich tea, digestives, shortbread, custard creams, sponge cake, madeira cake, angel cake.
Drinks	Coffee (limit to 1 cup a day), malted milk drinks for example Ovaltine® or Horlicks®, hot chocolate, fruit and vegetable juices, smoothies.	Tea, herbal tea, squash or cordial, water, fizzy drinks

Milk and Dairy Products	Limit milk to 1⁄2 pint per day (300ml). Limit yogurt to 3 small pots per week. Condensed milk, evaporated milk and milk powders	Limit milk to 1⁄2 pint per day (300ml). Cheese, crème fraiche or cream. Rice or oat milk.
Salt Substitutes	Lo-Salt, So-Low, reduced sodium salt.	Pepper, fresh or dried herbs, spices, chilli, garlic.

(The information above has been adapted from the "First Line Potassium Lowering Dietary Advice"diet sheet developed by the Renal Nutrition Group of the British Dietetic Association 2018.)

3. Anaemia ● Normocytic ● Might have Iron deficiency ● Folate/B12 deficiencies	- Start Iron sulphate/ folate orally if not on dialysis - Start EPO 50 Units/kg 2-3x/week sc. Titrate to maintain Hb 10-11g/dL - Might individualize target HB level according to activity level. **Introduction** Anemia of CKD is defined as a Hb (hemoglobin) <12 g/dL (female) or <13.5 g/dL (male), with adequate iron availability by parameters: TSAT (transferrin saturation) >20% and ferritin >100 ng/mL. In CKD Stage 5, the ferritin target is >200 ng/mL. Anemia of CKD usually begins during CKD Stage 3, ie, GFRs <60 mL/min/1.73 m2 **Rule out other causes.** The primary reason for anemia in CKD is an absolute or relative deficiency of renal erythropoietin (EPO) synthesis. Occult causes of blood loss and iron deficiency must be ruled out in all patients as a cause of hyporesponsivenss to ESA treatment. , vitamin deficiencies, eg, B12 and folate, and inflammatory causes of ESA resistance should be ruled out secondarily. Inflammation upregulates hepcidin, a liver-synthesized protein that reduces gut iron absorption and impedes iron release from the reticuloendothelial system to the developing erythron. Finally, effective iron delivery is required for optimal ESA-stimulated production of fully hemoglobinized red blood cells. **Iron Deficiency** Iron deficiency is common in CKD. CKD patients should be iron replete before initiating ESAs. To correct iron deficiency, oral iron should always be tried initially, and multiple iron salt preparations are available. However, to achieve iron repletion, parenteral iron may be required in non-dialysis CKD patients. **Safety** During anemia treatment, Hb elevations of 1–2 g/dL per month are generally well tolerated. More rapid increments are not advised. Recent clinical trials describe an increased risk of blood clots, strokes, and heart attacks in CKD and dialysis patients, in association with treatment to Hb levels of ~13 g/dL, particularly at high doses of ESA. **Therapeutic Targets** Hb 10–12 g/dL (do not exceed Hb 13 g/dL) TSAT >20% but <50% Ferritin >100 ng/mL . **Evaluation** CBC, absolute reticulocyte ct, TSAT, ferritin, vitamin B12, and folate levels. Always rule out other causes of anemia, eg, malignancy, inflammatory conditions, vitamin D deficiency, and iron deficiency before starting an ESA. Monitor iron parameters and CBC twice monthly after initiating therapy or until Hb stabilizes within the target range, then monthly. Use the absolute reticulocyte count to assess efficacy. **Treatment**

	Iron Ferrous sulfate: 200 mg elemental iron/24-h (alternative, ferrous fumarate) Iron dextran : 500–1000 mg iv infusions of low molecular wt iron dextran Iron sucrose : 100–200 mg iv infusions in non-dialysis-dependent CKD Ferumoxytol : 500–1000 mg iv in non-dialysis-dependent CKD **Erythropoiesis-Stimulating Agents (ESAs)** Epoetin alfa: 10–40,000 Units, subcutaneously, q1–4 wk; begin therapy at Hb <10 g/dL at starting dose, 100 Units/kg/wk. Darbepoetin alfa: 40–300 mcg, subcutaneously, q2–4 wk or q1 mo; begin therapy at Hb <10 g/dL at starting dose, 0.9 mcg/kg/q2 wk (equivalent to package insert dose, 0.45 mcg/kg/wk)
4. Hypertension	- target <130/80mmHg if Proteinuric or <140/90mmHg if not proteinuria - For Early disease (stage I and II) a) ACE inhibitors and b) ARBs have been recommended in combination with loop diuretics. For Late disease (stage III-V) a. Calcium Channel blockers. If possible Verapamil or Diltiazem. **b.** Loop diuretics **Beta-blockers (adjust dose according to GFR)** Hydralazine and centrally acting like methyldopa or clonidine might be required. **Therapeutic Targets** BP <130/80 mmHg CKD without proteinuria BP 120–129/75–79 mmHg CKD with proteinuria **First-line Agents** GFR >20mL/min/1.73 m2 ACEI or ARB Most CKD patients with HTN require 2 or more antihypertensive medications **Second and Third Line Agents** GFR 40 mL/min/1.73 m2 Add thiazide and/or CCB, if anti-RAAS agent is first-line GFR <40mL/min/1.73 m2 Add loop agent, eg, bumetanide or furosemide (twice-daily dosing) or torsemide (once-daily dosing) and/or CCB, if anti-RAAS agent started as first-line therapy **Fourth-line Agents** HR >80 bpm Beta blocker or alpha/beta blocker HR 80 bpm Consider adding ARA (spironolactone or eplerenone), if proteinuria present **Specific Clinical Situations** Diabetes ACEI or ARB for type 1 diabetes ARB or ACEI for type 2 diabetes

	CAD Beta blocker, CCB, alpha/beta blocker, e.g., labetalol BPH Alpha-1 blocker, e.g.,prazosin, terazosin, doxazosin Thiazide-resistant HTN Amiloride or ARA Primary aldosteronism ARA Orthostatic hypotension Target 2-min standing SBP (>120 mmHg) **Stage 2 Hypertension (uncontrolled)** SBP ≥150 mmHg on ≥2 occasions, separated by ≥2 days DBP ≥90 mmHg on ≥2 occasions, separated by ≥2 days
5. Metabolic Bone Disease	Start dietary restriction in stage 3, phosphate binders if no response to hyperphosphatemia (calcium carbonate 2 tablets three times a day with food). Vitamin D (calcitriol) should be the last resort. **Management and Therapeutic Options** Nutritional vitamin D, ergocalciferol (plant sources) or cholecalciferol (animal sources), can be used as treatment for hypovitaminosis D at any CKD stage. 25(OH)D levels <30 ng/mL represent vitamin D insufficiency. Treatment (with active vitamin D sterols) is indicated when 25(OH)D levels are >30 ng/mL; Corrected calcium (Corr Ca) is <9.5 mg/dL; P <4.6 mg/dL; and PTH levels are elevated and continue to rise with time. . **Corrected Ca (mg/dL)=Total serum Ca-[0.8 times(4-Albumin)]** Doxercalciferol, a vitamin D2 prohormone, requires hepatic hydroxylation for activation. Paricalcitol, a calcitriol analog, is active upon administration and does not require in vivo activation. Doxercalciferol and paricalcitol exert vitamin D-like actions and are less prone to induce hypercalcemia than calcitriol. **Phosphorous Binders (always taken with meals)** Calcium acetate (PhosLo) 1.0–1.5 g elemental Ca daily for P >4.6 mg/dL and Ca 8.8–10.2 mg/dL; 667 mg of Ca acetate contains 167 mg elemental Ca (25%) Sevelamer HCl (Renagel) 800–2400 mg 3 times daily for P >4.6 mg/dL and Ca >10.2 mg/dL (Ca-based P-binder contraindicated) FDA-approved for CKD Stage 5 Sevelamer Carbonate (Renvela) 800–2400 mg 3 times daily for P >4.6 mg/dL and Ca >10.2 mg/dL (Ca-based P-binder contraindicated) FDA-approved for CKD Stage 5 Lanthanum Carbonate (Fosrenol) 500–1000 mg 3 times daily for P >4.6 mg/dL and Ca >10.2 mg/dL (Ca-based P-binder contraindicated) FDA-approved for ESRD

They are recommended in CKD Stages 3 and 4 for P >4.6 mg/dL when the corrected Ca is <10.2 mg/dL and there is no evidence of coronary, peripheral vascular/cardiac valvular calcification.

With Ca-based P-binder therapy, the total daily elemental Ca intake (dietary + prescribed) should not exceed 2000 mg daily.

So as to limit excessive Ca loading and extraskeletal calcification and dystrophic medial arterial calcification that occur earlier in diabetes and CKD.

Sevelamer HCL and lanthanum carbonate are non-Ca-based P-binders. These agents may be used as initial P-binder therapy, if arterial/cardiac vascular calcification is present or, if the corrected Ca is >10.2 mg/dL.

These drugs do not alter Ca or PTH levels and do not affect treatment by vitamin D or its analogs. Sevelamer typically reduces LDL-C by 30% and raises HDL-C.

KDIGO targets for CKD-mineral and bone disorder

CKD (Stage)	Ca (mg/dL)	P (mg/dL)	iPTH (pg/mL)	HCO_3 (mEq/L)
3	NL range	NL range	2-9 times ULN	22-26
4	NL range	NL range	2-9 times ULN	22-26
5	Lower toward NL	Lower towards NL	2-9 times ULN	22-26

Evaluation

Ca, P, iPTH — Every 2 wk initially in CKD Stages 3–4 until normalized, then every 3–12 mo depending on stage and trends

Serum HCO3 — Every 1–4 mo, depending on degree of metabolic acidosis

25(OH)D — <30 ng/mL at initial evaluation; begin therapy, then repeat level every 3 mo until 30 ng/mL; subsequent levels are evaluated depending on CKD stage and levels.

Vitamin D and Active Vitamin D Sterols

Vitamin D

Ergocalciferol (D2) 25(OH)D <15 ng/mL: 50,000 IU q1 wk × 4, then every 1 mo × 8, unless corrected Ca >9.5 g/dL and/or P >4.6 mg/dL (new)

25(OH)D 15–30 ng/mL: 50,000 IU q1 mo × 6, unless Corr Ca >9.5 g/dL and/or P >4.6 mg/dL

weekly therapy may be required.

Monitor levels every 3 mo and continue weekly or monthly accordingly.

Cholecalciferol (D3) 25(OH)D <30 ng/mL: 1,750 IU once daily (new)

Active Vitamin D Sterols

	Calcitriol Initial dose for CKD Stages 3–4: 0.25–0.50 mcg once daily Doxercalciferol Initial dose for CKD Stages 3–4: 1.0 mcg once daily Paricalcitol Initial dose for CKD Stages 3–4: 1.0 mcg once daily or 2.0 mcg, 3 times weekly (see COMMENTS) **COMMENTS** Active vitamin D sterols: therapeutic choices at CKD Stages 3–5 include calcitriol, doxercalciferol or paricalcitol. Treatment plan includes periodic monitoring of Ca, P, albumin and PTH and rarely induces Ca or P elevations that warrant their discontinuation. Active vitamin D sterols: may co-administer with ergocalciferol, if SHPT is present because ergocalciferol rarely suppresses PTH to target levels. Active vitamin D sterols: initiate during CKD Stages 3–4, if Ca <9.5 mg/dL, P <4.6 mg/dL, and PTH greater than target range. Ergocalciferol: consider in CKD Stages 3 and 4, if corrected Ca <9.5 mg/dL, P <4.6 mg/dL, PTH elevated, and 25(OH)D <30 ng/mL **Metabolic Acidosis** NaHCO3 0.5–2.0 mEq/kg daily; target HCO3 22–26 mEq/L
7. Treat underlying/co-morbidities or precipitating cause	

RENAL REPLACEMENT THERAPY

Advanced CKD Stage 4 patients should be referred to a vascular access surgeon at eGFR <20mL/min/1.73 m2 for timely planning of appropriate HD vascular access or peritoneal dialysis (PD) catheter placement.

Timing of Kidney Replacement Therapy

- The decision to treat patients with CKD by RRT as kidney transplantation, peritoneal dialysis, or hemodialysis should be made collaboratively by patients and their healthcare team.
- Recent data reveal that starting RRT early does not increase patient survival. Although CKD Stage 5 begins at eGFR <15 mL/min/1.73 m2 KRT does not have to begin then, if the patient is well.
- The number of comorbidities that a patient has must be taken into consideration before initiating RRT.
- Patients >75 y.o., particularly, if male, with multiple comorbidities managed conservatively fare just as well as those on dialysis because death versus dialysis is a competing survival risk.
- Therefore, conservative management, when chosen, focuses the shift from simply attempting to prolong life to providing quality of life and alleviation of symptoms.

Education

- CKD education entails information of various kidney replacement therapies: transplantation, PD, in-center HD, and home HD.
- Physical conditions such as vision and manual dexterity, motivational level to actively participate in care, and family/social circumstances all play roles in the decision-making process.

Peritoneal Dialysis

- Peritoneal dialysis (PD) is a viable option for most ESRD patients;
- Instillation of a hypertonic glucose-containing solution via a trans-abdominal PD catheter provides uremic solute (diffusion) and excess sodium and water removal (convection).
- PD catheters should be implanted at least 4 weeks prior to the date of their anticipated use.
- BP and P control are superior with PD compared to conventional HD.
- PD involves diffusion of uremic solutes and electrolytes from capillaries lining the peritoneal membranes into the externally infused dialysate.
- The continual nature of PD is suited for heart failure patients and/or volume-dependent HTN.

Contraindications

- To PD include extreme obesity, multiple abdominal surgeries, and recurrent peritonitis.
- Complications of PD catheters include peritonitis, catheter malfunction, and failure of PD due to membrane loss/fibrosis.
- Peritonitis can be treated with intra-peritoneal or iv antibiotics and may require catheter exchange.
- Catheter removal is absolutely indicated when pseudomonal, MRSA, or fungal peritonitis occurs.

Hemodialysis

- Treatment times of 3–4 h. by slow, low-efficiency dialysis.
- Home HD is conducted in the home environment, 5–6 sessions weekly for 2.5–3 h.
- Control of BP and phosphorus are superior with PD, nocturnal PD, and home HD compared to conventional thrice-weekly HD.
- Optimal HD requires a well-functioning vascular access and this can be provided via an autogenous AVF (arteriovenous fistulas), bioprosthetic AVG (arteriovenous grafts), or HD catheters.
- The AVF is the best HD vascular access and most closely satisfies the requirement for adequate blood flow delivery to the dialysis machine, and has the lowest maintenance cost among all vascular access types.

Vascular Access Planning and Construction

- Key issues include timely nephrology referral; vein preservation; vascular access creation planning; timely referral to a surgeon specialized in access construction; post-construction follow- up; and appropriate intervention(s).
- Protection of superficial hand and forearm veins, particularly of the non-dominant arm, is critical in CKD patients.
- The dorsum of the hand should be used for peripheral lines and blood draws.
- The patient should be evaluated by venous mapping, preferably by ultrasound duplex scanning of the non-dominant arm (non-handwriting); if unsuitable, the dominant arm may be used for access creation.
- The patient and healthcare workers must know the intended surgery site.
- Therefore, vein preservation during hospitalizations and outpatient care must occur.
- Subclavian vein catheter placement(s) and PICC lines are discouraged and are associated with high central vein stenosis rates, and their use may preclude access creation(s).
- Cardiac AICD and pacemaker placements should be contralateral to the planned vascular access arm.
- Educational programs reinforcing the above should be provided to patients, their families and healthcare providers.
- Preparation of the HD patient includes surgical vascular access construction
- An AVF, typically created from a native artery and vein in the distal non-dominant upper extremity, is the optimal HD vascular access.
- Placement of an AVF should precede the time of anticipated HD by 6 months, to ensure sufficient fistula maturation before HD needle cannulation.
- When AVF creation is not feasible, AVG construction should proceed 3–6 weeks before anticipated HD.

HYPOKALEMIA

Definition

Hypokalemia is defined as a serum potassium level of **< 3.5 mEq/L. (Severe if K^+ < 2.5 mEq/l).** Commonly asymptomatic, however, severe hypokalemia may be symptomatic

Signs & Symptoms

Cardiovascular: bradycardia, tachycardia, hypotension, arrhythmia, arrest, palpitations
Respiratory: Respiratory distress & failure, hypoventilation
M/S-cramps, tetany, reduced deep reflexes, reduced muscle strength, rhabdomyolysis
General-lethargy, constipation, nausea, vomiting, abdominal cramps and parasthesia

Goals of management

1. Reduction of potassium losses
2. Replenishment of potassium stores
3. evaluation for potential toxicities
4. Identification of the underlying cause(s) so as to prevent future episodes(1)

Evaluation

- History should focus on possible underlying factors, medications and dietary and bowel habits, chronic laxatives abuse,
- Exclude history of diuretics and high dose penicillin, use of β -agonists, exclusive use of IV fluids without K, metabolic alkalosis from other causes, insulin administration and diabetic ketoacidosis
- Evaluate for hypomagnesemia (it aggravates hypokalemia, also hypomagnesemia with hypokalemia is associated with refractory to treatment with K+)
- Order an ECG [changes in severe hypokalemia - broad flat T waves, U waves, ST depression, and QT prolongation]

Lab

1. Serum electrolytes: Na^+, K^+, urea, glucose: calculate serum osmolality [$2Na^+ + 2K^+$ + glucose + urea]
2. Creatinine
3. Urinalysis
4. Urine [K^+] if available
5. ABG
6. FBP
7. Other investigations depending on underlying illness

Approach to the causes of Hypokalemia

Redistribution into cells	Insulin treatment e.g. in correction of DKA Metabolic alkalosis B_2-adrenergic stimulation/ B-agonists Acute Myocardial infarction Vitamin B_{12} or folic acid administration
Reduced intake	Dietary deficiencies: Fruits/vegetables IV fluids without K^+
Gastrointestinal losses	Chronic severe diarrhea laxatives abuse Vomiting Ileostomy Fistulae clay (bentonite) ingestion glycogenesis during enteral hyperalimentation or total parenteral nutrition
Increased aldosterone (increases secretion)	Heart failure Liver failure Nephrotic syndrome Cushings' syndrome Conn's syndrome
Renal disease	Renal tubular damage Renal tubular acidosis Adrenal steroid excess (Cushing's syndrome) Primary hyperaldosteronism Rare renin-secreting tumors Glucocorticoid-remediable congenital adrenal hyperplasia. Ingestion of substances such as glycyrrhizin Bartter syndrome Gitelman syndrome Liddle syndrome Fanconi syndrome Hypomagnesemia
Drugs	Corticosteroids Thiazides Loop diuretics Osmotic diuretics Laxatives Amphotericin B Antipseudomonal penicillins (carbenicillin) Penicillin in high doses Theophylline (both acute and chronic intoxication) Insulin B agonists Aminoglycosides

Approach to Management

Determine if it is an emergency; the urgency and duration of therapy depends on:

- The severity of hypokalemia
- Associated clinical symptoms and clinical factors (cardiac disease, digoxin therapy)
- The rate of decline in serum K^+
- Approximate total deficit: total body K^+ deficit correlates to serum K^+ (approximately 0.3 mmol/L drop in serum K^+ for every 100 mmol/L reduction in total body stores, in the absence of abnormal K^+ redistribution)

(Simplified : for every 1 mmol/l drop of serum potassium, the total body deficit is 200 – 400 mmol/l)

NB:

- In the presence of poor renal perfusion/renal impairment, treatment should be in consultation with a senior.
- Failure to correct hypokalaemia may be due to concurrent hypomagnesaemia, hence serum Magnesium should be measured and any deficiency corrected.
- The patient may also be on other sources of K e.g. IV fluids containing K, include these in your calculation
- Intravenous K^+-Cl^- should always be administered in saline solutions rather than dextrose since the dextrose-induced increase in insulin can acutely exacerbate hypokalaemia.
- Check infusion sites for signs of inflammation/phlebitis
- Consider strategies to minimize K^+ losses:
    - Minimizing the dose of non-K^+-sparing diuretics
    - Restricting Na^+ intake
    - Minimizing other medications that exacerbate hypokalaemia.

Recommendations

Hypokalemia	Treatment	Comments
MILD 3.0 – 3.4 mEq/l	**Oral replacement** *Slow K [3 tabs t.i.d.] or i.v. potassium infusion 25mL Aim at 70-75 mmol/day	Usually asymptomatic Monitor K^+ daily and adjust treatment accordingly
MODERATE 2.5 – 2.9 mEq/l	**Oral replacement** Slow K [4 tabs t.i.d.] or i.v. potassium infusion 25mL Aim at 100 mmol/day	Monitor K^+ daily and adjust treatment accordingly Consider IV if patient cannot tolerate PO or symptomatic
SEVERE < 2.5 mEq/l OR symptomatic	Intravenous replacement 40 mmol KCl in 1L 0.9% NaCl** BD or t.i.d (20 mmol per 500 ml) Standard infusion rate 10 mmol/hr (maximum 20 mmol/hr) Recommended not to exceed 2-3 mmol of K^+/kg in 24 hrs. Check Mg levels **(ask lab for Mg^+ levels automatically if K^+ is < 2.5 mmol/L)** If hypomagnesaemic, initially give 4ml $MgSO_4$ 50% (8 mmol) diluted in 10ml 0.9 % NaCl over 20 minutes before starting KCl administration	**DO NOT GIVE CONCENTRATED KCl DIRECTLY INTO A VEIN- THIS IS LETHAL!** Monitor K^+ levels after each IV infusion and adjust accordingly. Monitor for ECG changes In fluid overload, a higher K^+ concentration warranted (e.g. 40 mmol KCl in 500 ml) [NB: Higher concentrations are painful and cause phlebitis. Use a large peripheral vein possibly using an infusion pump & seek senior guidance first] Must monitor patients fluid status

* Slow K contains 600mg per tablet equivalent to 8mmol/L. (1 mmol equals about 75 mg of potassium chloride)
**Regarding i.v. therapy, 0.9% sodium chloride is the preferred infusion fluid, as 5% glucose may cause transcellular shift of potassium into cells.

References

Longo DL, Fauci AS, Kasper DL, Hauser SL, Jameson J, Loscalzo J. eds. *Harrison's Principles of Internal Medicine, 18e.* New York, NY: McGraw-Hill; 2012.
NHS Guidelines for the management of Hypokalaemia in adults. Medicines Information, CGH. August 2010David Garth, MD; Romesh Khardori, MD, PhD, FACP. Hypokalaemia in emergency medicine: Medscape emedicine [extracted from http://emedicine.medscape.com/article/767448-overview]

HYPERNATRAEMIA

Definition

Hypernatremia is defined as a serum sodium level of >145mmol/L. (Severe if >160mmol/L.) It is termed acute hypernatremia if it occurs in < 48hours and chronic if > 48hrs. Presentation is mostly neurological with symptoms of irritability, lethargy, agitation, dizziness, confusion, and coma.

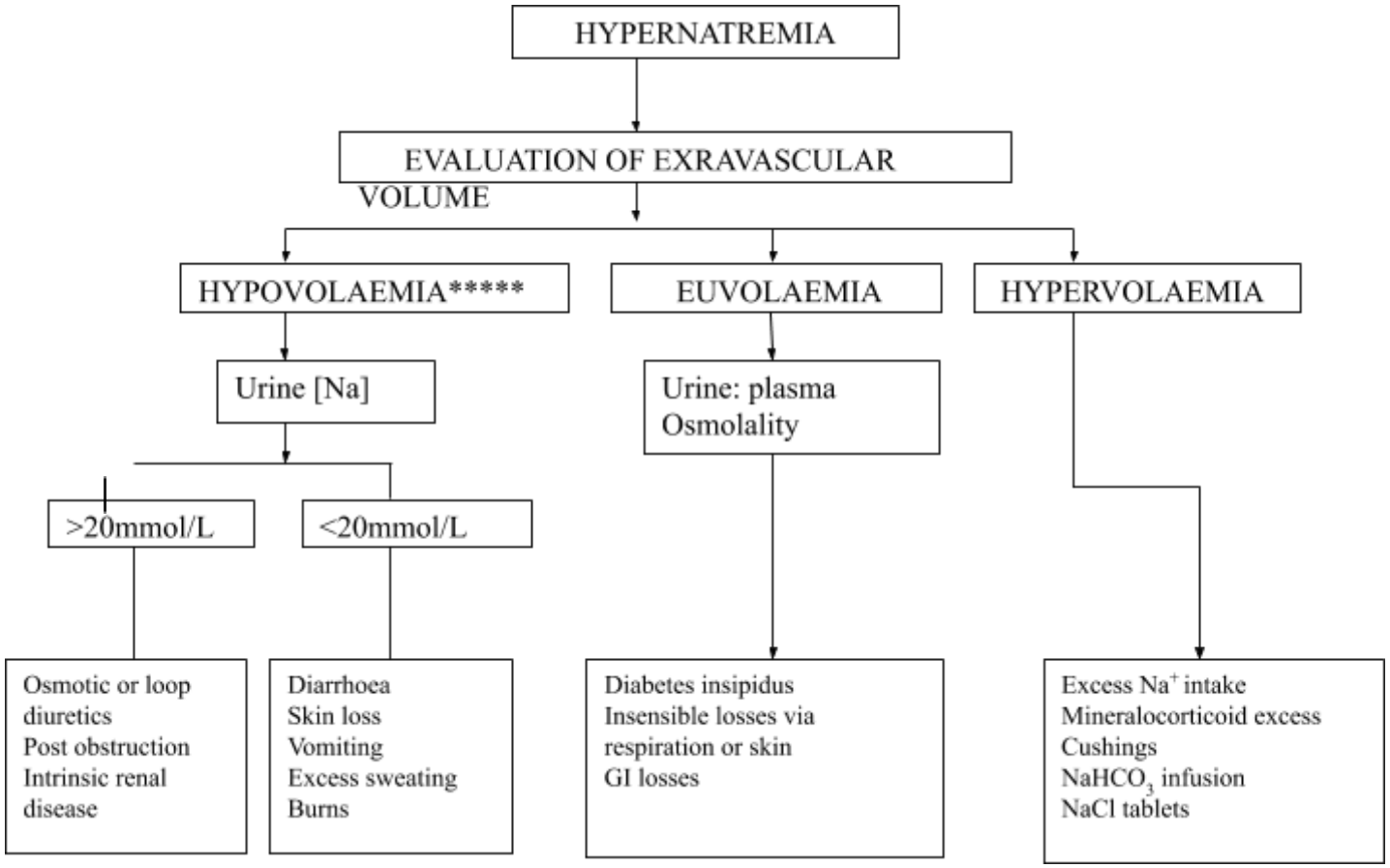
Goals of management

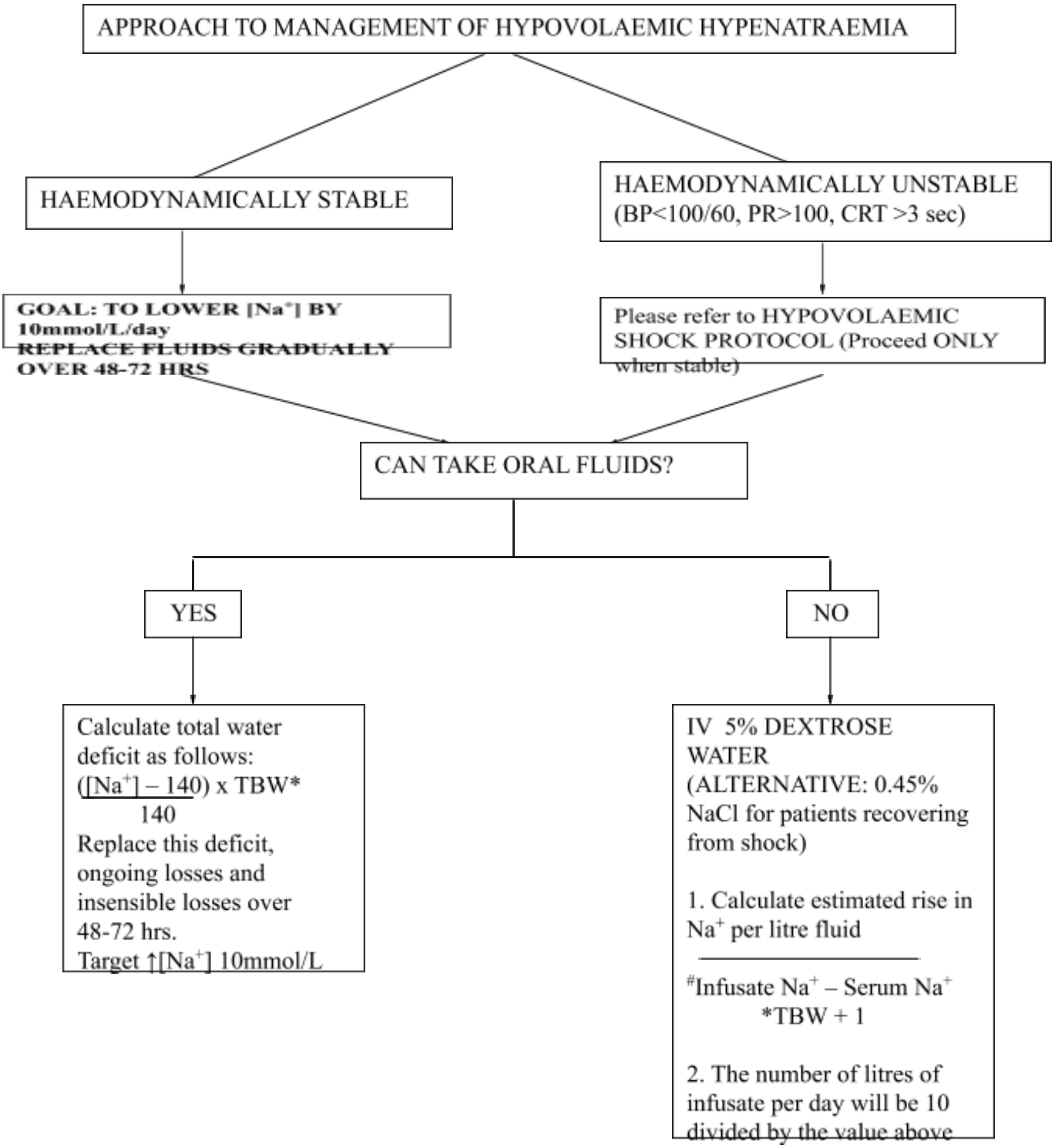
1. Correct hypernatremia
2. Prevent complications of cerebral edema during correction
3. Correction of volume disturbances
4. Identification of the underlying cause(s)

Lab evaluation

1. Serum electrolytes: Na^+, K^+, urea, glucose (calculate serum osmolality [$2Na^+ + 2K^+$ + glucose + urea] mmol/L)
2. Creatinine
3. Measured serum osmolarity
4. Urine [Na^+]
5. Specific gravity (most urine dipsticks measure SG)
6. Other investigations depending on underlying illness

The diagnostic approach to hypernatremia





IMPORTANT

- Treat underlying causes (diarrhoea, hyperglycaemia, electrolyte imbalances etc)
- 4 hourly monitoring of serum [Na^+], other electrolytes and neurological status, if symptoms of deterioration, STOP, reassess and consult senior.
- Blood glucose should be monitored to avoid hyperglycaemia
- If previous Na^+ is known taken < 48 hrs prior to treatment, and this is deemed true hypernatraemia, a higher rate of lowering up to 1mmol/L/hr is indicated with more frequent monitoring to reach normal serum sodium within 24 hours. This should be done ideally in a high care setting in collaboration with a senior doctor.

*Total body water (TBW) = 50% of Body weight [use 60% for males]

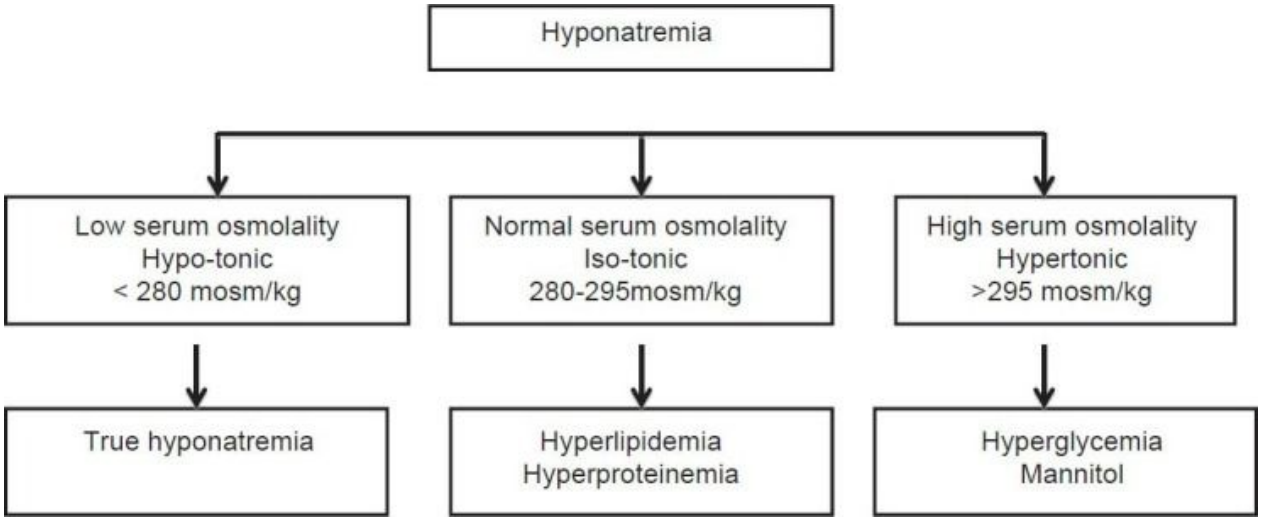
#Infusate Na^+ is the amount of [Na^+] is the IV fluid [5% Dextrose Water =0; 0.45% NaCl = 77; 0.9% NaCl= 154; Ringer's Lactate= 130; 5% Dextrose NaCl=154]

Hyponatremia Protocol

Hyponatremia is defined as plasma sodium concentration less than 135 mmol/l.

Classification of hyponatremia

Hyponatremia is classified as pseudo hyponatremia, true and translocational hyponatremia.



Pseudo (normo-osmolal) or isotonic hyponatremia occurs when seemingly low sodium levels are actually normal. Causes include hyperglycemia, hyperproteinemia, mannitol use, laboratory errors or increase in plasma proteins. Osmolality remains unchanged, and patients are usually euvolemic.

Translocational (hyper-osmolal) or hypertonic or redistributive hyponatremia is due to presence of osmotically active solutes in the serum e.g., mannitol or glucose.

True (hypo-osmolal) hyponatremia is associated with reduction in serum osmolality and is further classified as euvolemic, hypervolemic and hypovolemic hyponatremia

(i) Hypovolemic hyponatremia

This results from loss of body sodium or potassium with secondary water retention. Sodium and free water are lost and replaced by hypotonic fluids such as tap water, half sodium saline or dextrose in water.

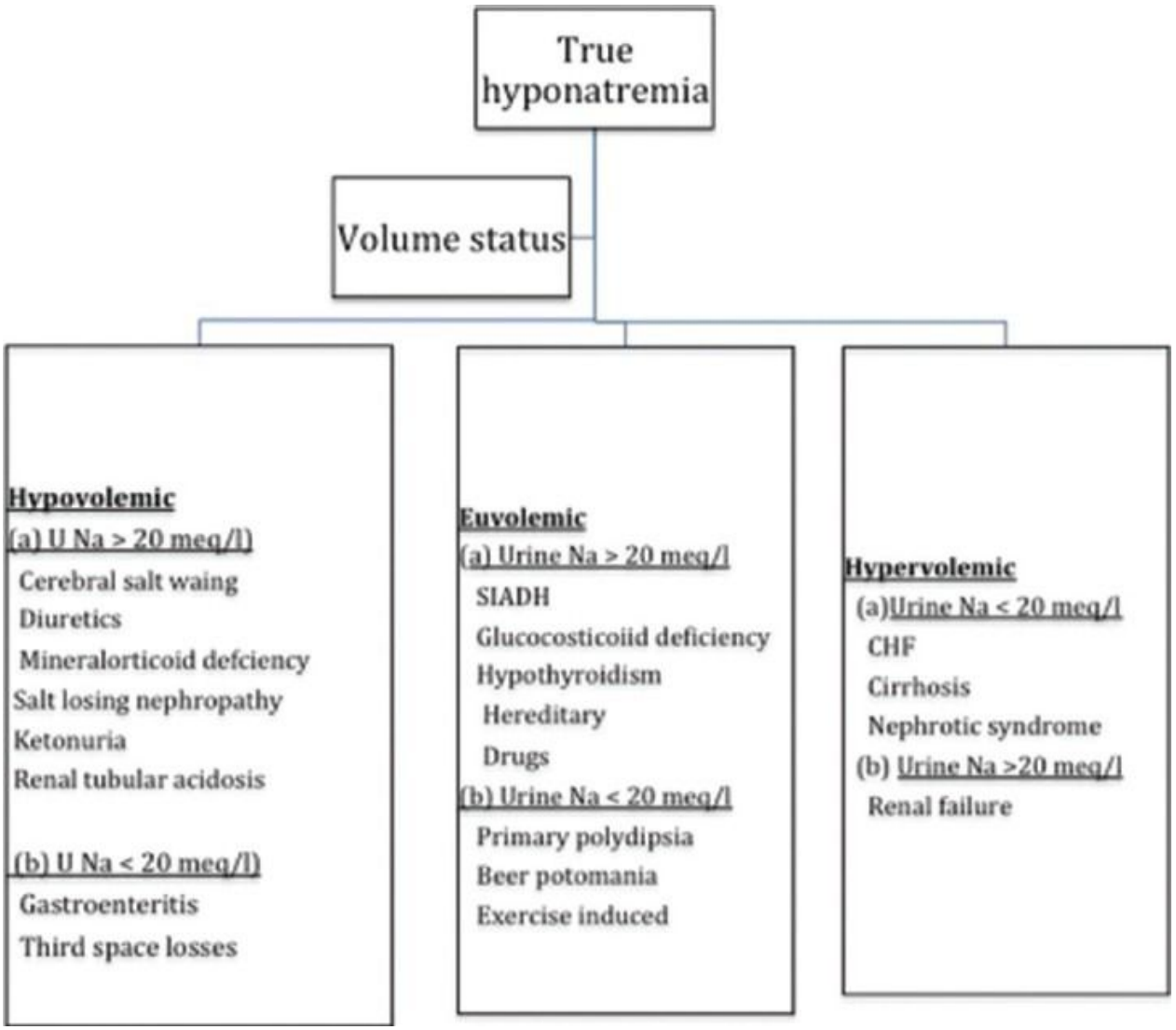
(ii)Euvolemic hyponatremia

This results from relative or absolute excess of body water. Total body sodium remains normal.

(iii)Hypervolemic hyponatremia

This results from excess renal sodium and water retention. Total body sodium increases and total body water increases to a greater extent.

Possible causes

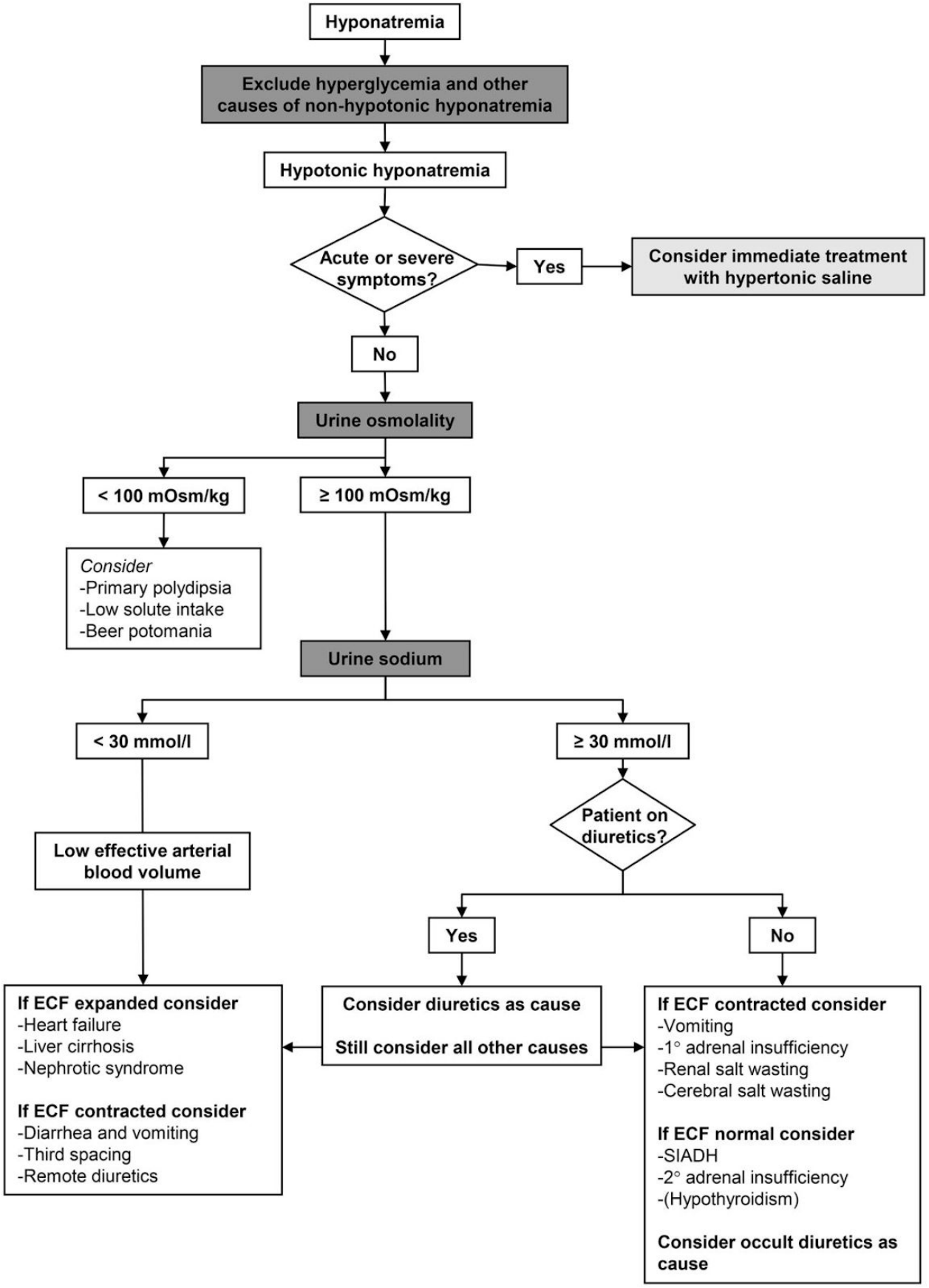


Clinical presentation

- Asymptomatic
- Headache
- Lethargy
- Dizziness and ataxia
- Mild confusion
- Psychosis
- Seizures
- Coma

Goal of Management

1. Correct hyponatremia (Prevent brain edema/ herniation)
2. Avoid complications during correction of chronic hyponatremia
3. Diagnose and treat underlying cause


Hyponatremia
Exclude hyperglycemia and other causes of non-hypotonic hyponatremia
Hypotonic hyponatremia
Acute or severe symptoms?
Yes
Consider immediate treatment with hypertonic saline
No
Urine osmolality
< 100 mOsm/kg
≥ 100 mOsm/kg
Consider
-Primary polydipsia
-Low solute intake
-Beer potomania
Urine sodium
< 30 mmol/l
≥ 30 mmol/l
Patient on diuretics?
Low effective arterial blood volume
Yes
No
If ECF expanded consider
-Heart failure
-Liver cirrhosis
-Nephrotic syndrome
If ECF contracted consider
-Diarrhea and vomiting
-Third spacing
-Remote diuretics
Consider diuretics as cause
Still consider all other causes
If ECF contracted consider
-Vomiting
-1° adrenal insufficiency
-Renal salt wasting
-Cerebral salt wasting
If ECF normal consider
-SIADH
-2° adrenal insufficiency
-(Hypothyroidism)
Consider occult diuretics as cause

Approach to management of hyponatremia

Step 1: Diagnose Hypotonic Hyponatremia
2Na+2K+Urea+Glucose should be <275 mmol/L
Exclude high lipids and protein in blood which bring about lab errors in Na measurement

Step2: Assess need for urgent therapy and respond
Are there severe symptoms? (Regardless of acuteness)
Are there severe symptoms?

YES (Target of 5 mmol/L increase in serum sodium in the first hour)

1. Initiate treatment with iv. Infusion of 2 ml/kg or 150ml 3% hypertonic saline over 30 min
2. Check serum sodium after 30 min while repeating an infusion of (1) above
3. If target not reached you may repeat (1) & (2) above ONCE

IF THE PATIENT HAS IMPROVED

1. Stop hypertonic saline, keep iv. line open with minimal 0.9% saline
2. Start diagnosis specific treatment
3. Limit increase in serum Na+ concentration to 8 mmol/l in 24 hrs until Na> 129
4. Check serum sodium 6hrly in the first 24hrs, then daily until Na >129

IF THE PATIENT HAS NOT IMPROVED (after 5 mmol/L target increase)

1. Seek senior/Expert advice, transfer patient to HDU/ICU
2. Exclude other explanations
3. Consider **iv. In**fusion of 3% saline titrated to rise Na by 1mmol/L/hr (4 mmol/L/4 hrs) and monitor serum Na 4hrly. Use Androgue-Madias formula to calculate the expected increase after 1L of 3% (change Na= [513-serum Na]/[Total body water + 1] STOP when a total rise of 8 mmol/L is achieved OR symptomatic improvement

NO severe symptoms but moderate severe symptoms present OR asymptomatic acute hyponatremia more than 10 mmol/L decrease

1. Start diagnosis specific assessment and treatment
2. Immediate treatment with a <u>single</u> i.v. 3% hypertonic saline over 30 min
3. Check serum Na 6hrly for the first 24hrs and daily until Na>129

Target of 5 mmol/L daily increase and limit to 8 mmol/L daily

INTRAVENOUS FLUID THERAPY

Hypovolemia is defined as the abnormally decreased volume of circulating fluid (plasma) in the body (loss of > 20% of body fluid).

The role of fluid therapy:

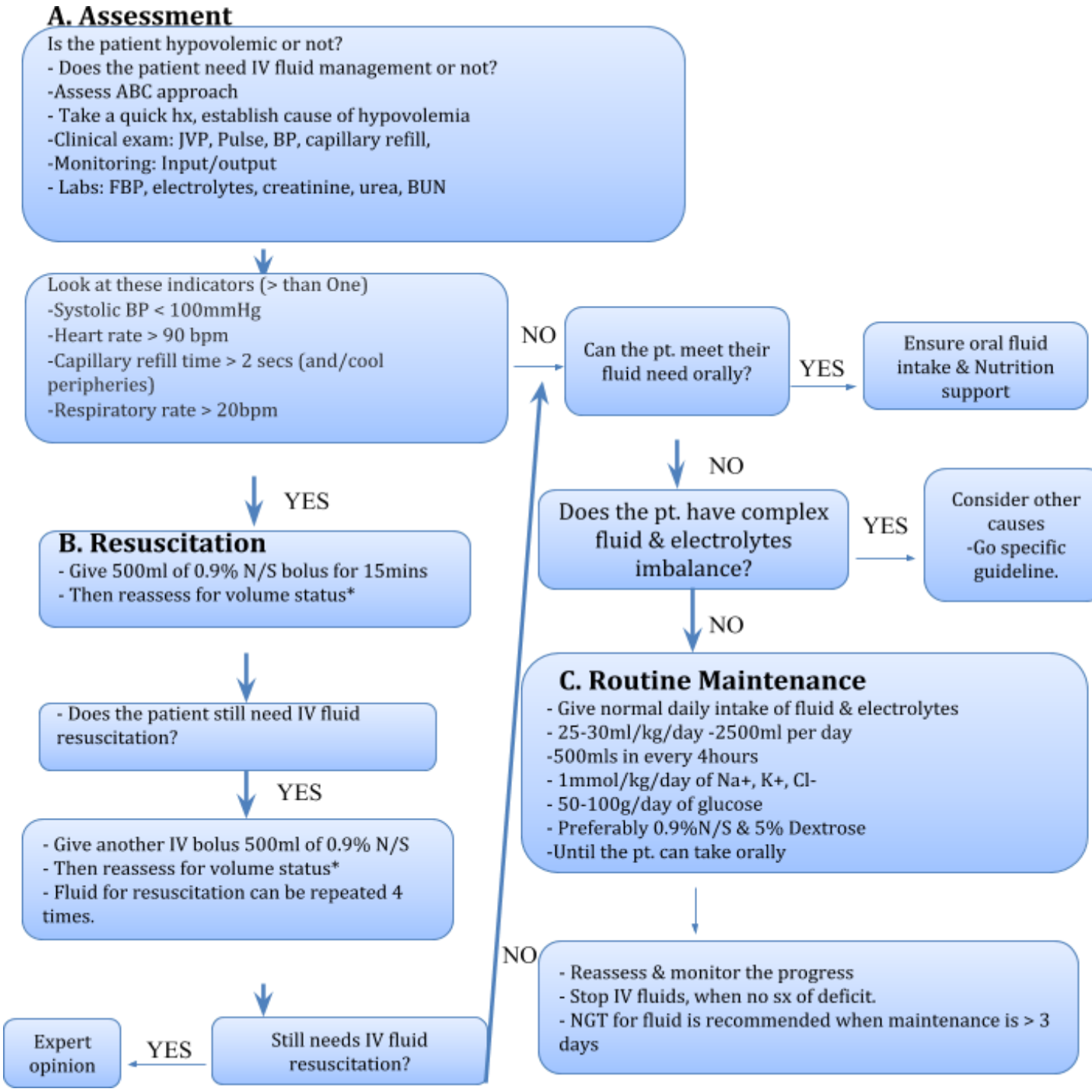
- ✓ Is to correct the hypovolemia, Replace the ongoing loss and to maintain circulation.

The following steps have to be taken into consideration when managing the hypovolemia.

Note: -For every step assess the patient for ABC as well as the sign for fluid overload and make appropriate decision.

-Resuscitate for Maximum 1hr, if no responses call for expert's opinions.

* Volume status: JVP, oedema, crackles, DIB



Types of fluid and their uses

Types of fluid	
A: Isotonic	When to use
0.9% NaCl (Normal Saline)	Shock, Hemorrhage, Vomiting & Diarrhoea, Mild Hyponatremia, Initial mnx DKA
Ringer Lactate or Hartmann Solution	Replace GI loss: D &V Burns & Trauma and Acute blood loss
B: Hypotonic	
0.45% NaCl,	Used in intracellular dehydration

	E.g.: Hypernatremia, DKA, HHS
C: Hypertonic	
3% NaCl and 5% NaCl	Severe hyponatremia Cerebral oedema
5%Dextrose Normal Saline (DNS)	Replaces Na+, Cl- & calories, Hyprenatremia

- Calculate the IV flow rate
  - ✓ (Volume (ml) *Drops per ml)/ Time in mins: where 10drops/ml (in blood set) and 15 drops /ml (in regular set).
  - ✓ E.g.: 500ml of IV N/S ordered in 4hrs

  (500mls * 15drops/ml) /4hrs. * 60 min = **31drops/minute**
- Fluid must be prescribed in fluid chart.
- Date/Time/Fluid type/Volume/Rate
- End point of IV fluid therapy
  - Normalization of vital signs, Urine output>0.5mls/kg/hr., restoration of normal mental status, lack of clinical signs of deficit and pt. can take orally.

CLINICAL APPROACH TO PROTEINURIA

Definition: Proteinuria refers to the presence of large amounts of protein in urine of more than 2.5g per day (TSTG 2017).

Clinical presentations:

1. In transient proteinuria, usually there are no symptoms and is often picked by urinalysis. Rule out with PCR or ACR.
2. Persistent proteinuria, there may be urine with frothy appearance and oedema.

Possible causes

1. *Transient proteinuria*
    a. After vigorous exercise
    b. During high fever
    c. Severe dehydration
    d. In heart failure
    e. In people with UTIs/cystitis

1. *Persistent proteinuria*
    a. Disease of the glomeruli eg. glomerulonephritis
    b. Uncontrolled diabetes mellitus
    c. Eclampsia in pregnancy

Pathophysiology

1. The abnormal trans-glomerular passage of proteins due to increased permeability of glomerular capillary wall (D'Amico 2003)
2. Subsequent impaired reabsorption by the epithelial cells of the proximal tubule.

Clinical Diagnosis

History: Hypertension, oliguria, polyuria, weight loss, recent infections, recent intake of medications (NSAIDs, ACEIs, Penicillamine). Family history of hypertension, renal diseases, and autoimmune diseases.

Physical Examination: Oedema, flank pain, fluid overload, organomegaly, rashes, anemia, joint swelling.

Goals of Management of Proteinuria

1. To delay the decline of kidney functions (Thilly et al, 2009)
2. To achieve maximal renal protection

Investigations

Proteinuria can be evaluated best using the following methods:

1. Urinary dipstick test (Routinely used in clinical practice)
2. Random or first morning urine (FMU) for spot urine protein-to-creatinine ratio (UPCR)
3. The 24 hours urinary protein and creatinine
4. Urinary microalbumin

Quantifying proteinuria in random urine samples (Davidson 23ed)

ACR	PCR	Typical dipstick results	Significance
<3.5 (female) <2.5 (male)	<25	-	Normal
3.5 - 30	25 - 50	-	Moderately elevated albuminuria
30 - 70	50 - 100	+ to ++	Dipstick positive
70 - 300	100 - 350	++ to +++	Glomerular disease more likely; equivalent to >1g/24 hrs
>300	>350	+++ to ++++	Nephrotic range: almost always glomerular disease, equivalent to >3.5g/24 hrs

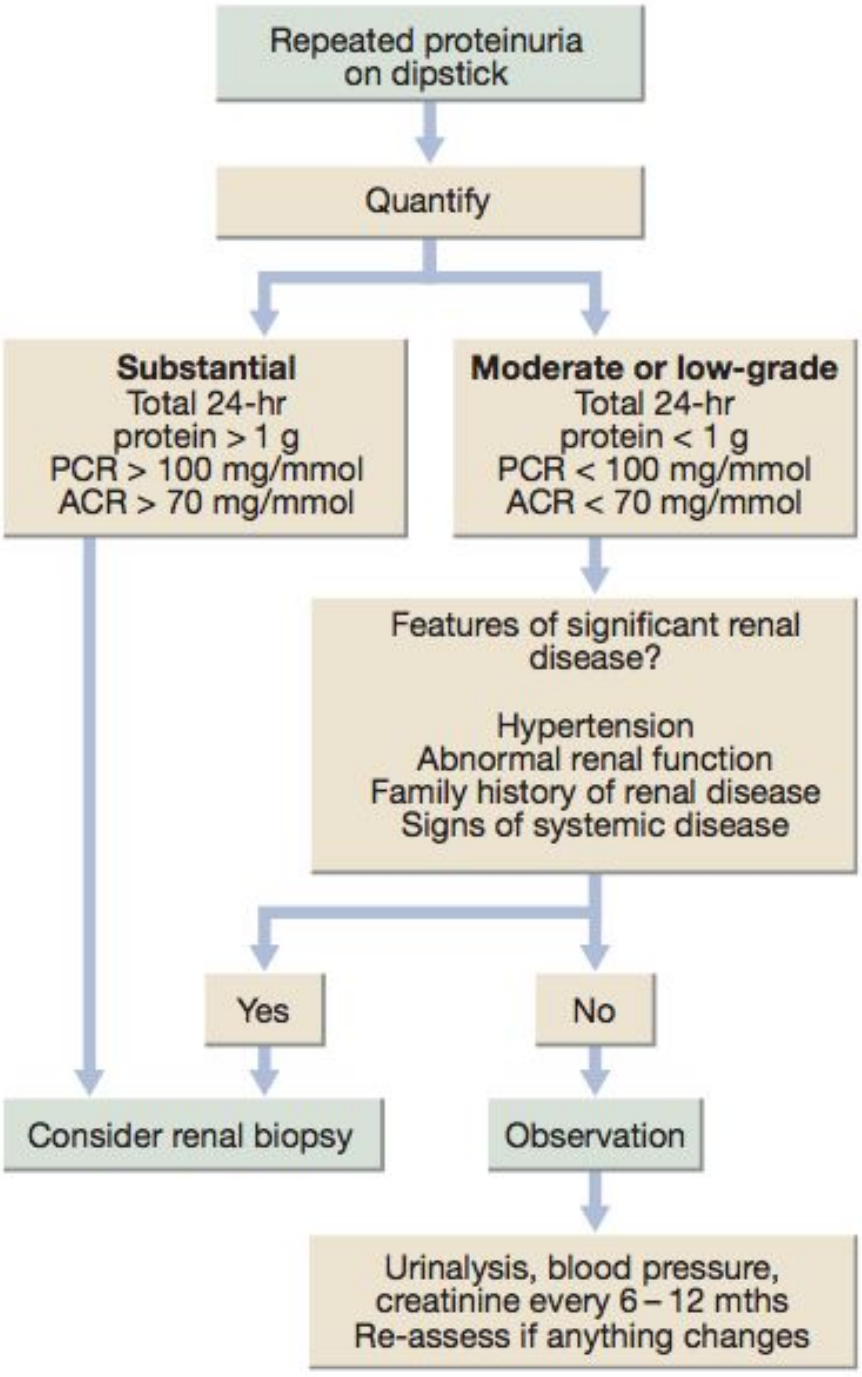


Figure: **Investigation of proteinuria.** (*ACR = albumin:creatinine ratio; PCR = protein:creatinine ratio.*) [Davidson 23ed]

NEUROLOGICAL DISEASES

STATUS EPILIPTICUS
MYSTHENIA GRAVIS
DELIRIUM
COMA

STATUS EPILEPTICUS

Defined as continuous seizures or repetitive, discrete seizures with impaired consciousness in the interictal period, duration traditionally 15-30min. Any situation requiring the acute use of anticonvulsants in GCSE: typically when seizures last > 5min

Clinical features

Subtypes

i. Generalized convulsive status Epilepticus (GCSF)

ii. Non convulsive status Epilepticus

Etiology

Withdrawal of antiepileptic drug especially phenobarbitone, Non-compliance, Metabolic disturbance, Drug toxicity, GNS infections, CNS tumors, Refractory epilepsy or Head trauma.

General measures: ABC, check bld glucose, if h/o alcohol. Rx:Vit B1 (thiamine) iv 100-250mg

Drug treatment of status Epilepticus.

Stage	Drug/route/rate	Comment
Early status (0-30 mins)	**Diazepam** 10mg/iv bolus over 2 minutes *or* **Lorazepam** 4mg (0.1 mg/kg) iv bolus over 2 minutes.	If seizures continue 5 mins either one can be repeated just once but may cause respiratory depression.
Established status (30-60 mins) (if seizures continue & patient is not on phenytoin or phenobarbitone)	**Phenytoin** iv n. saline infusion of 15 mg/kg (900 – 1,000 mg total dose) @ 50 mg/min/iv (in N/S over 20 mins) *or/and* **Phenobarbiton**e infusion iv 10 mg/kg/@100 mg/min/over 7-10 minutes *(adult total dose is 600mg*)	If iv is unavailable, give phenytoin via nasogastric tube, *(absorption is excellent)* If patient is already on phenytoin or phenobarbitone then use half the usual full loading dose If seizures stop continue with maintenance oral phenytoin or phenobarbitone
Refractory status (>60 mins) (if seizures still continue)	Involve anaesthesia & proceed to ventilation, Rx **thiopentone or midazolam or propofol**	If seizure are stopped for >24 hours, stop ventilation & continue daily maintenance dose of phenytoin or phenobarbitone & reinstate regular AEDs

Adopted W.P. Howlett, Neurology in Africa

MYASTHENIA GRAVIS

Definition: Myasthenia gravis **(MG)** is a chronic autoimmune neuromuscular disease characterized by varying degrees of fatigable weakness of the skeletal (voluntary) muscles of the body. ***Latin and Greek in origin****-"grave muscle weakness.*

A progressive inability to sustain a maintained or repeated contraction of striated (voluntary) muscle (fatigability) caused by an autoimmune defect in neuro-muscular transmission.

Main types of Myasthenia Gravis: Generalized myasthenia 85-90%, Ocular myasthenia 10-15%
Presenting symptoms: fluctuating & fatigable muscle weakness that improves with rest (remissions and exacerbations)

Main clinical features	
eyes	drooping eye lids & double vision >50%
face, mouth	weakness smiling (myasthenic snarl)
speech	voice nasal & weak & easily tires
bulbar	weakness jaw closure, chewing & swallowing
limbs	proximal > distal weakness combing hair weak hand grip difficulty arising from chairs or climbing stairs
central muscles	head drop weakness sitting up
respiratory	weak cough shortness of breath (if severe leads to respiratory failure i.e. myasthenic crisis)

*NOTE: Aminoglycosides, Macrolides, Flouroquinolones, Quinine, b-blockers, ca-channel blockers & should be avoided in myasthenics as may exacerbate the block

Diagnosis:

1. **Clinical signs:** Fatigue on repeated muscle contraction e.g. increased ptosis on up gaze or weakness or repeated (20 times) arm elevation, speed of counting

Ice pack test (place bag ice on closed eyelid for 2-5 mins: Sensitivity = 80% e.g. improvement in ptosis

- **Edrophonium test:** Inject dose of short acting anti cholinesterase: this will reverse symptoms for few minutes. **Edrophonium (Tensilon)* has been developed for this test, (10mg IV slowly; starts with 1-2mg and waits for the response give additional 5-6 mgs to max 10 mgs**)**
  ***not readily available** *Neostigmine 2mg,* under supervision may be used
- Atropine 0.5 to 1.0 mg should always be available for emergency IV use because of potential bradycardia.

2. **Electrophysiological:** Repetitive nerve stimulation, single fibre EMG
3. **Serum:** acetylcholine receptor antibody (AChR) 85% positive.
4. **Chest X-ray and CT scan:** thymus gland enlargement or cysts and calcification (Thymoma in 10-15%).

Differential Diagnosis: AIDP, Dermatomyositis, Lambert-Eaton Myasthenic Syndrome, Botulism

General Approach

Always consider managing in ICU, Secure ABC but don't delay
Treat any exacerbating factors: Infections, medication, endocrine disease

Treatment: Summary of management of myasthenia gravis

Treatment	Drugs	Indication
Cholinesterase Inhibitors	**Pyridostigmine** The starting dose is **30 mg/po/qds** and this is doubled in 2 days until the patient is taking **60 mg/po/qds.** *May be increased to 60 mgs 4 hourly* **Propanthaline 15-30 mg** given 30 minutes before dose of pyridostigmine if cholinergic symptoms	Any muscle weakness Maximum total daily dose is **360 mg** Given for cholinergic crisis, and can be prophylaxis
Immunosuppressant	**Prednisolone 10 mg/po/ loading dose** increasing slowly by **10 mg/alt days** until **1.5 mg/kg** or **100 mg** is reached whichever is the lower dose.	Inadequate control on Pyridostigmine alone
	Azatioprine 25 mg/po/bd and increased by 50 MG weekly until the patient is on 2.5 mg/kg/po or 150 mg/po/daily**. S/Es** leukopenia, hepatotoxicity	Inadequate control on Prednisolone & Pyridostigmine
IVIG/plasma exchange	However, these treatment options are unavailable at most centres in Africa	*Myasthenic crisis, bulbar & respiratory weakness
Ventilation	*Respiratory failure (FVC <1.5 litres) is an urgent indication for consideration for mechanical ventilation.	Bulbar/respiratory failure
Thymectomy		All patients <45 years and suspected Thymoma at any age

***Myasthenic Crises:** A myasthenic crisis occurs when the muscles that control breathing weaken to the point that ventilation is inadequate VC < 1.5Lts, creating a medical emergency and requiring elective intubation & assisted ventilation**.**

Prognosis:

Ocular myasthenia will develop into generalised MG in 80-85%
Course is variable but for most is progressively disabling
Exertion, infection or childbirth can worsen the disease
Untreated MG carries a mortality rate of 25-31%
Treated MG with modern critical care & immunotherapy has a <4% mortality rate

Refs: The Lancet. Handbook of Treatment in neurology. Ed Charles Warlow 2006
Howlett WP. Neurology in Africa 2015

DELIRIUM

Step 1. Assess all patients admitted for the presence of risk factors

Age > 60	**Severe illness**
Cognitive impairment	Acute stroke

- Severe illness should be defined by an agreed set of physiological parameters. The NEWS (National Early Warning Score) has an excellent ability to predict unanticipated ICU admission, cardiac arrest and death within 24 hours (Appendix 1).
- If cognitive impairment is suspected use the IDEA cognitive screening tool (Appendix 4).

Step 2. Follow do's and don'ts to prevent delirium in at risk patients and to shorten already established delirium

Do's multicomponent intervention package (**NICE recommendation in bold**)			Avoid
Optimise sensory impairment e.g spectacles / hearing aid	**Rationalise medication**	**Hydrate (oral >S/C >IV)**	Unnecessary procedures
Frequently reorientate and reassure patient	**Monitor bowels / treat constipation**	**Optimise O_2 sats**	Use of restraints
Enable sleep in calming environment	**Look for and treat pain**	**Optimise nutrition**	**Unnecessary catheterisation**
Encourage early supervised mobilisation	**Look for and treat infection**	**Familiar nursing staff Involve family / friends**	**Moving the patient between and in the ward**

Step 3. Assess for indicators of delirium on admission and daily thereafter

Clinical Indicators - a new or change in:	
Cognition/concentration	Hallucinations
Appetite, sleep, mood	Social behaviour
Physical function	Consider delirium in all falls

Step 4. If an indicator is present, then use CAM to diagnose delirium (Appendix 3)
Take collateral history.

Step 5. Management of symptoms; first consider and address issues of patient safety

(1) For maintaining patient safety;
   i) Protect the airway and preventing aspiration- ensure patients are not supine for meals or NGT feeds or immediately afterwards.
   ii) Maintain hydration and nutrition (encourage relatives/carers to assist patient)
   iii) Prevent skin breakdown with regular checks of pressure points and regular turning;
   iv) Provide safe mobility while preventing falls (encourage mobile patients to walk with relatives' assistance and walking aids as needed), consider physiotherapy.
   v) Avoid restraints and bed alarms which have been shown to increase risk and persistence of delirium, and of injury.

Secondly, identify and treat all causes of delirium immediately **(usually >1)**

History

In addition to standard questions in the history, the following information should be specifically sought:

- Full drug history including non-prescribed drugs e.g. over the counter or herbal/traditional

medicines
- Alcohol history
- Previous intellectual function (eg ability to manage household affairs, pay bills etc.)
- Functional status (eg activities of daily living)
- Onset and course of confusion
- Previous episodes of acute or chronic confusion
- Symptoms suggestive of underlying cause (eg infection)
- Sensory deficits
- Aids used (eg hearing aid, glasses etc.)
- Pre-admission social circumstances and care needs
- Comorbid illness

Examination:
- Neurological examination (including assessment of speech)
- Consciousness level
- Nutritional status
- Evidence of pyrexia
- Evidence of alcohol abuse or withdrawal (e.g. tremor)
- Cognitive function using a standardised screening tool e.g. IDEA cognitive screening tool
- Attention (e.g. weekdays or months of year backwards)

Investigations:
- Full blood count and ESR
- Urea and electrolytes
- Liver function tests
- Calcium
- Glucose
- Chest X-ray
- ECG
- Urinalysis

Other useful investigations include TFTs, Vitamin B12, Folate, blood cultures, LP, CT, EEG however these should be done as per clinical need.
Document diagnosis of delirium in file. Actively seek and treat all common causes.

Common causes of delirium	
Respiratory failure	Surgery
Pain	Electrolyte disturbance
Infection	Urine retention
Dehydration	Metabolic disorder
Drugs (esp. CNS acting)	Constipation

Step 6. Drugs: Indications for pharmacological sedation

Verbal and non-verbal techniques above have failed
Prevent danger to self or others
Carry out essential investigations
Relieve patient distress

1st line: Haloperidol 0.5mg-1.5mg PO BD, in the elderly. (Younger adults >18yrs only: Orally 1mg-2mg BD, peak effect 4-6 hours, usually for 5-7 days or less)

CAUTION: Haloperidol should be avoided in the following groups and a benzodiazepine prescribed for the following groups;
- Patients with a QTc of >470ms,
- Lewy Body dementia,
- Parkinson's disease or Parkinsonism

- Patients with seizures,
- Recreational drug intoxication/withdrawal
- Alcohol withdrawal should be prescribed benzodiazepines as first line.

Principles of prescribing: Use one drug at a time, start at the lowest clinically appropriate dose and titrate cautiously according to symptoms. If repeated doses required, consider regular prescription for a short period. Review requirement daily. Do **not** continue at discharge.

NICE clinical guideline 103 Issued July 2010.

COMA

Definition of Coma: "Unarousable unresponsiveness". Clinically, GCS ≤ 8.

Goals:

- To identify treatable conditions
- Early treatment to optimize neurologic recovery

History:

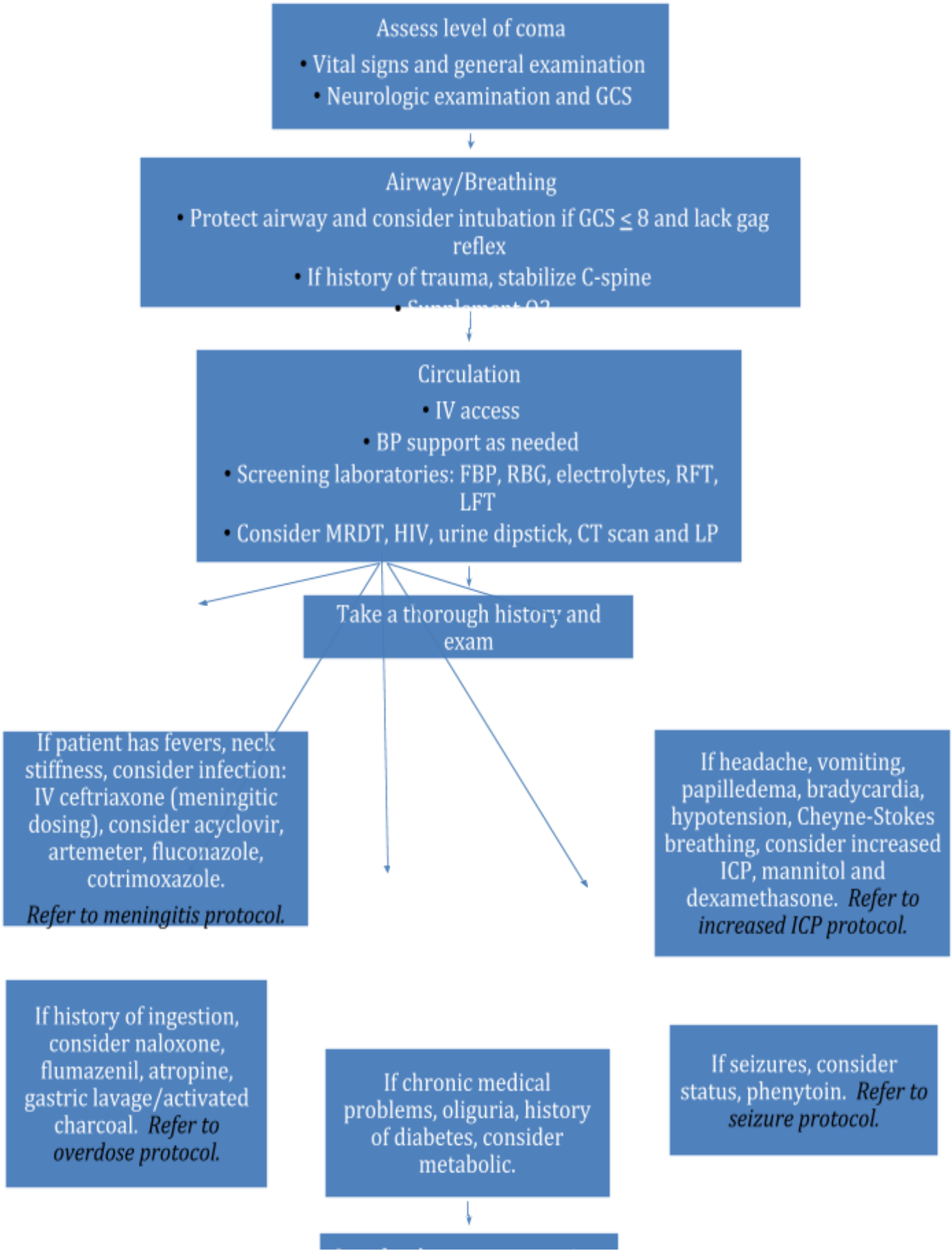
- Collateral from witnesses, family.
- Any recent illnesses, change in behavior, history of trauma?
- Time course (Abrupt, gradual, or fluctuating). Has this ever happened before?
- Is there any focality?
- Medications, past medical history, drug use

Examination:

- Vital signs (blood pressure, pulse, temperature)
- Cardiovascular
- Respiratory, including ventilatory pattern
- Abdominal, including organomegaly
- Skin and mucosal abnormalities, including bruising, jaundice, tongue laceration, rash
- Full neurologic exam including level of consciousness, motor responses, brainstem reflexes, neck stiffness, kernig's sign, and fundoscopy

Consider:

- **Neurologic:** *Trauma, Ischemia/Hemorrhage, Epilepsy, Space occupying lesion*
- **Metabolic:** *DKA, Uremic encephalopathy, Hyponatremia*
- **Infections:** *Meningitis, Brain Abscess, Encephalitis*
- **Toxins/Drugs:** *Overdose, Organophosphate poisoning*
- **Psychiatric:** *Schizophrenia, Catatonia*


Assess level of coma
• Vital signs and general examination
• Neurologic examination and GCS
Airway/Breathing
• Protect airway and consider intubation if GCS ≤ 8 and lack gag reflex
• If history of trauma, stabilize C-spine
Circulation
• IV access
• BP support as needed
• Screening laboratories: FBP, RBG, electrolytes, RFT, LFT
• Consider MRDT, HIV, urine dipstick, CT scan and LP
Take a thorough history and exam
If patient has fevers, neck stiffness, consider infection: IV ceftriaxone (meningitic dosing), consider acyclovir, artemeter, fluconazole, cotrimoxazole.
Refer to meningitis protocol.
If headache, vomiting, papilledema, bradycardia, hypotension, Cheyne-Stokes breathing, consider increased ICP, mannitol and dexamethasone. Refer to increased ICP protocol.
If history of ingestion, consider naloxone, flumazenil, atropine, gastric lavage/activated charcoal. Refer to overdose protocol.
If chronic medical problems, oliguria, history of diabetes, consider metabolic.
If seizures, consider status, phenytoin. Refer to seizure protocol.

Appendix 1
The National Early Warning Score

Physiological parameters	3	2	1	0	1	2	3
Respiratory rate (breaths per minute)	≤8		9-11	12-20		21-24	≥25
SpO2 level %	≤91	92-93	94-95	≥96			
Any supplemental Oxygen?		YES		NO			
Temperature	≤35		35.1-36.0	36.1-38.0	38.1-39.0	≥39.1	
Systolic BP (mmHg)	≤90	91-100	101-110	111-219			≥220
Heart rate/pulse rate (beats per minute)	≤40		41-50	51-90	91-110	111-130	≥131
Level of consciousness using AVPU system				A			V,P or U

Patients scoring 5 or more or three in one parameter should be considered acutely ill and **at risk** of delirium.
A score of 5 or more should trigger the nursing staff to request an urgent medical review of the patient, and should increase the frequency of monitoring of the patient's physiological observations (1-6 hourly).

Appendix 2

Delirium indicators

At presentation to the medical ward, assess patients at risk for recent (within hours or days) changes or fluctuations in behaviour. These may be reported by the person at risk, or a carer or relative.

Be particularly vigilant for behaviour indicating hypoactive delirium (marked*).Behaviour changes (within hours or days)		**Is indicator present?**[1]
Fluctuations in **cognitive function**	Worsened concentration*	
	Slow responses*	
	Confusion	
	Other cognitive function changes	
Perception	Visual hallucinations	
	Auditory hallucinations	
Physical function	Reduced mobility*	
	Reduced movement*	
	Restlessness	
	Agitation	
	Changes in appetite*	
	Sleep disturbance	
Social function	Lack of cooperation with reasonable requests	
	Withdrawal*	
	Alterations in communication	
	Alterations in mood	
	Alterations in attitude	
Other behaviour changes		
[1] Please record 'yes' or 'no' for each behaviour change.		

If any of these behaviour changes are present, a healthcare professional, trained and competent in diagnosing delirium should carry out a clinical assessment to confirm the diagnosis.

Appendix 3

WARD BED KCMC FILE NUMBER .....NAME ..... Date :

The Confusion Assessment Method

Delirium should be suspected with the presence of Features 1 and 2 and either 3 or 4

Feature		Present?
Feature 1: **Acute Onset and Fluctuating Course**	This feature is usually obtained from a family member or nurse and is shown by positive responses to the following questions: **Is there evidence of an acute change in mental status from the patient's baseline? Does the (abnormal) behaviour fluctuate during the day; that is, does it tend to come and go, or increase and decrease in severity?**	
Feature 2: **Inattention**	This feature is shown by a positive response to the following question: **Does the patient have difficulty focusing attention; for example, is the patient easily distractible, or having difficulty keeping track of what's being said?**	
Feature 3: **Disorganized Thinking**	This feature is shown by a positive response to the following question: **Is the patient's thinking disorganized or incoherent, as evidenced by rambling or irrelevant conversation, unclear or illogical flow of ideas, or unpredictable switching from subject to subject?**	
Feature 4: **Altered Level of Consciousness**	This feature is shown by any answer other than "alert" to the following question: **Overall, how would you rate this patient's level of consciousness? (alert [normal], vigilant [hyperalert], lethargic [drowsy, easily aroused], stuporous [difficult to arouse], or comatose [unarousable])?**	

Is delirium present? Yes or No	

Appendix 4

WARD CODE NUMBER

.....NAME.....Date.....

IDEA study six item screen

Preparation for ten-word list; "I am going to read out a list of words. Please listen carefully and I will ask you to repeat them back to me once I have finished" (read out the words slowly).

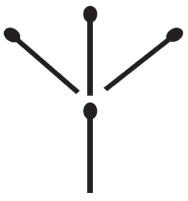
10 word list	First attempt	Second attempt	Third attempt
Butter (siagi)			
Arm (mkono)			
Letter (barua)			
King (mfalme)			
Ticket (tikiti)			
Grass (nyasi)			
Corner (kona)			
Stone (jiwe)			
Book (kitabu)			
Stick (fimbo)			

First attempt: "Now tell me all the words you can remember" (tick on the grid the words remembered).

Second attempt: "Now I will read out the words again, listen carefully and I will ask you to repeat as many as you can. Now tell me all the words you can remember" (tick on the grid the words remembered).

Third attempt: "Now I will read out the words one last time, listen carefully and I will ask you to repeat as many as you can. Now tell me all the words you can remember" (tick on the grid the words remembered).

1	I will tell you the name of something and I want you to describe what it is. **What is a bridge?**	**0** - incorrect **2** - correct (*correct answer: something that goes across a river, canyon or road*)	Score:____/2
2	I want you to name as many **different** animals as you can in one minute. Number of animals named: ____	**0** - 0-3 animals named **1** - 4-7 animals named **2** - 8 or more animals named.	**Score: ____/2**

3	Who is the chairman of your village? (Or street leader))	**0** –incorrect **1** - correct	Score:____/1
4	What day of the week is it?	**0** - incorrect **2** - correct	Score:____/2
5	Can you tell me the ten words we learned earlier? Try to remember as many as you can.	**0** no words remembered **1** 1 word **2** 2 words **3** 3 words **4** 4 words **5** 5 or more words	Score:____/5
6	Can you make the design shown below using these four matchsticks. I will show you once and then you have to copy exactly) (*The examiner should make the design first using the matchsticks and specifically point out to the person that the heads of the matchsticks all need to point the same way. Once the examiner has made the shape, collect up the matchsticks in a bunch and place them in front of the person being*  *interviewed)*	Score **1** for each part of the design that is performed correctly **1-**Middle two matchstick heads pointing same way **1**-Outside two matchsticks pointing at an angle **1-** Matchstick heads are orientated correctly	Score :____/3
7.	Allow the person to see the design below as they make the shape. ***(only if failed previously at item 6)***	**Score as ABOVE**	**Score :____/3**
	Best score from 6 or 7	**Use best score of 6 or 7**	**Total Score:____ /15**

MISCELLANEOUS

HIGH ALITTUDE ILLNESS
ORGANOPHOSPHATE POISIONING
APPROACH TO ARTHRITIS

HIGH ALTITUDE ILLNESS (HAI)

Definition: HAI is an illness that can affect unacclimatized climbers at altitudes over 2400m/ 8000ft above sea level.

Questions to ask: How high reached, how quickly is the ascent? Any symptoms prior climbing, past hx of HAI and pre-existing cardiac or pulmonary conditions.

Prophylaxis for HAI:

- Gradual ascent with a rate of 500 meters increase in sleeping altitude per day above 3000 meters with a rest day every 1200m ascent.

-Pharmacological prophylaxis generally not recommended but shall be considered in special cases.

- **Acetazolamide** 125mg twice daily should be **started the day before ascent** and may be stopped during descent or after 2-3days at the target altitude.
- **Dexamethasone** 4mg 12 hourly **starting the day of ascent** in those with a history of intolerance or allergic to acetazolamide.
- **Nifedipine** 30mg 12 hourly only in patients with **prior hx** HAPE started the day prior to ascent and discontinued at descent or after 5days at target elevation.

Clinical features	AMS	HAPE	HACE
Symptoms	**Headache** pluse atleast 1 of below symptoms • Loss of appetite • Nausea &Vomiting • Fatique • Dizzyness • Insomnia	• Dyspnoea • Reduced exercise tolerance • Dry cough • Blood stained sputum **Refer to HAPE severity classification**	• Behavioural change • Hallucinations • Disorientation & confusion • Seizures (rare) • Altered consciousness • Coma
Signs		• Resting tachycardia • Tachypnea • Crackles • Cyanosis • Signs of pulm. HTN • Low SaO2 • Fever **#ECG** -Sinus tachycardia -Right axis deviation,RBBB **#CXR** -Normal heart size, -features of pulmonary oedema **# FBP to R/0 Infection**	• Ataxia • Cranial nerves palsy • Focal neurological signs (rare) **#Fundoscopy** Papilledema ± retinal haemorrhages.

HAPE Severity Classification (Modified from: Hackett 1995)

Grade	Symptoms	Signs	Chest X-ray
Mild	Dry cough Dyspnea on exertion Fatigue while moving uphill	HR (rest) < 90-100 RR (rest) <20 Exertional desaturation Localized crackles, if any	Minor exudate involving less than 25% of one lung field
Moderate	Dyspnea at rest Weakness Fatigue on level walking Raspy cough	HR 90-110 RR 16-30 Cyanotic nail beds Crackles present	Some infiltrate involving 50% of one lung or smaller area of both lungs
Severe	Dyspnea at rest Extreme weakness Orthopnea Productive cough	HR > 110 RR > 30 Facial & nail cyanosis Bilateral crackles Blood-tinged sputum Stupor Coma	Bilateral infiltrates > 50% of each lung

Criteria for discharge:

SaO_2 > 90% on room air, Clinical + radiological improvement and no desaturation on exercise.

Points to keep in mind:

-Crackles may be absent in up to 30% of cases of HAPE
-It is common for some ataxia to persist for days or weeks incase of HACE
-Consider non-altitude causes of coma in patients with focal neurologic deficits, or who don't get better with the above treatment.
-Acetazolamide is a sulfonamide derivative, and should not be used in Sulfa-allergic patients.
- Nifedipine used in HAI in a hospital setting is extended release (Nifedipine-SR)
-Medication can be stopped after clinical recovery.

References:

5.0 Bartsch P, Swenson ER. Acute high-altitude illnesses. N Engl J Med. 2013 Oct 24;369(17):1666–7.

6.0 Hackett PH, Roach RC. High-altitude medicine and physiology. In: Auerbach PS, editor. Wilderness Medicine. 6th ed. Philadelphia: Mosby Elsevier; 2012.p. 2–32.

7.0 Luks AM, McIntosh SE, Grissom CK, Auerbach PS, Rodway GW, Schoene RB, et al. Wilderness Medical Society consensus guidelines for the prevention and treatment of acute altitude illness. Wilderness Environ Med. 2010 Jun;21(2):146–55.

ORGANOPHOSPHATE POISONING

Organophosphates are potent cholinesterase inhibitors capable of causing severe cholinergic toxicity, this leads to accumulation of acetylcholine at muscarinic receptors, nicotinic receptors and in CNS.

-**Organophosphorus compounds** are well absorbed by ingestion, inhalation and through skin. (Parathion, fenthion, malathion, diazinon, and dursban).

Goal of treatment

- Reversal of hyper cholinergic state
- Supportive management

Clinical features

-Patients may have an organophosphates smell

-Muscarinic signs can be remembered by use of the following mnemonic: **SLUDGE/BBBM** - Salivation, Lacrimation, Urination, Diarrhea, Gastric Emesis (vomiting)/ Bronchorrhea, Bronchospasm, Bradycardia (PR <60 bpm) & Miosis

-Nicotinic effects include fasciculation, muscle weakness, and paralysis via acetylcholine stimulation of receptors at the neuromuscular junction.

-CNS effects include respiratory depression, seizures, lethargy, excitability and coma.

Investigations

Check blood glucose
FBP & ESR
Serum electrolytes
Chest x-ray

Management

- Decontamination - removal of the patient's clothes and vigorous irrigation of the affected areas by water and soap.
- **Activated charcoal**
- Nasogastric lavage if an OP agent is ingested in less than 6 hours after.
- Transfer poisoned patient to Medical Intensive Care Unit.
- Oxygen supplements 4-6 l/min for patients with markedly depressed mental status. If no improvement consults a senior doctor for possible intubation.

. IV fluids N/S or RL solution should be administered at least 3 liters per 24 hours

Reversal of hypercholinergic state

. Atropine 1 - 2 mg IV every after 10-15 minutes until atropinization. (Atropinisation: dry of respiratory secretions and cessation of bronchoconstriction)

-If no effect is noted, the dose should be doubled

- seizures should be treated with a benzodiazepine , give diazepam10 mg IV stat then PRN.

NB
Note:

- Hospitalizing all symptomatic patients for at least 48 hours following resolution of symptoms is recommended Because of the risk of respiratory depression or recurrent symptoms after resolution of an acute cholinergic crisis,
- **There may be delayed neuorological signs/symptoms.**
- Patients need to be counseled and consultation to the Psychiatrist should be made before discharge in case of intentional/suicidal attempt.

ARTHRITIS

Goals of Therapy:

1. To identify different types of arthritis
2. To manage symptoms
3. Prevent complications and preserve joint function

Definitions:

Rheumatoid arthritis – An autoimmune disorder affecting the joints. Usually a symmetrical polyarthritis of unknown etiology

Osteoarthritis – Articular cartilage failure due to genetic, metabolic and biomechanical factors with components of inflammation

Septic arthritis - Infection in a joint, usually caused by bacteria but can be caused by fungi or mycobacteria. Often a destructive form of acute arthritis that is a medical emergency.

Gout – Recurrent attacks of acute inflammatory arthritis usually due to monosodium urate crystal deposition

Key points to elucidate in history:

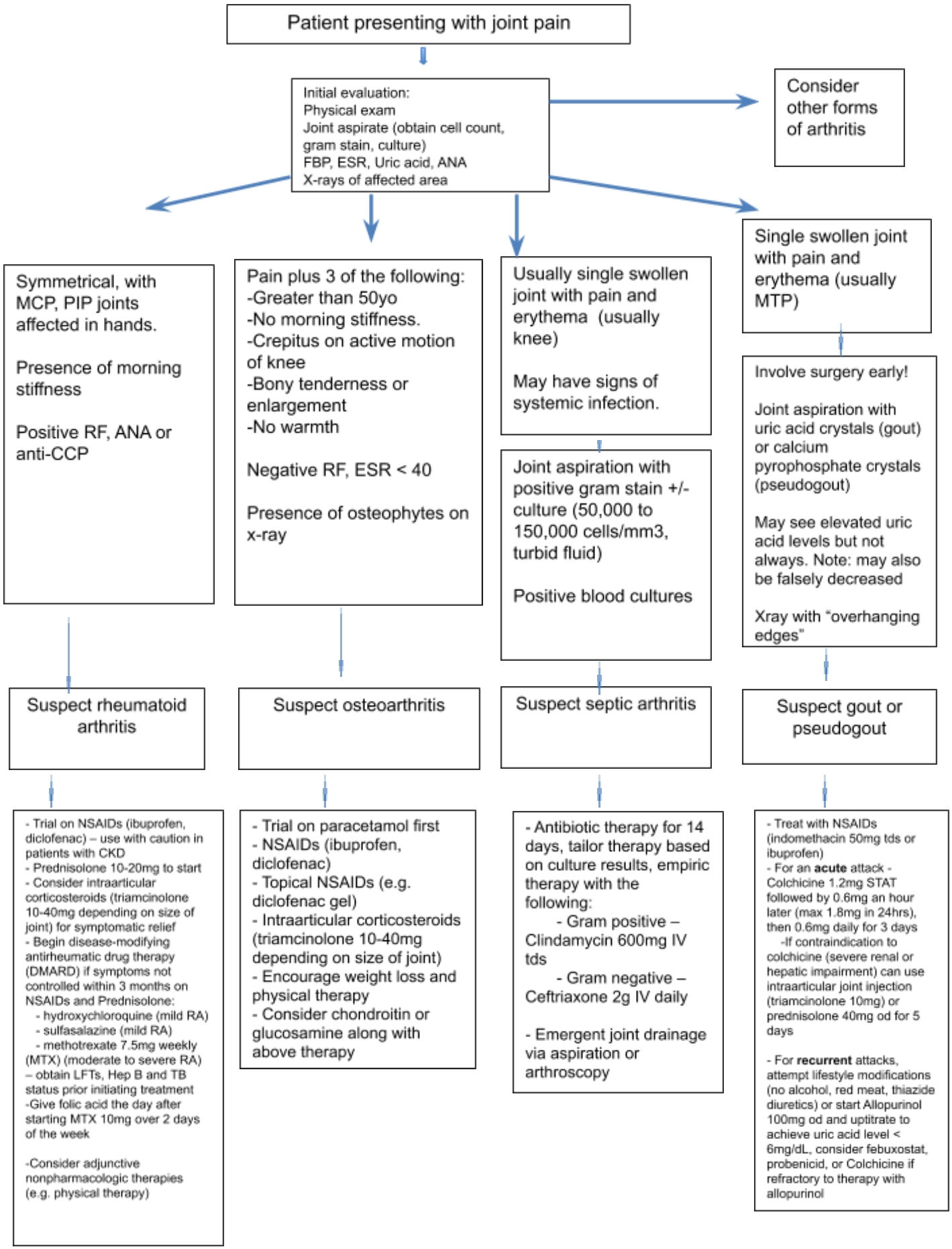
- Duration of symptoms
- Location of pain and swelling
- Presence of fevers
- Presence of morning stiffness
- Prior episodes
- Family history

Differential diagnosis:

Bursitis/ cellulitis (can be confused with septic arthritis)
Psoriatic arthritis (can be confused with rheumatoid arthritis)
Systemic lupus erythematous (can be confused with rheumatoid arthritis)
Dermatomyositis/Polymyositis (can be confused with rheumatoid arthritis)
Fibromyalgia (can be confused with osteoarthritis, rheumatoid arthritis)
Neoplasms (can be confused with rheumatoid arthritis, osteoarthritis)

Clinical findings:

- Perform a thorough musculoskeletal exam, focused on:
  - Swelling
  - Presence of erythema
  - Location
  - Range of motion
  - Warmth
- Look for extra-articular findings, such as: rheumatoid nodules, tophi, erythema nodosum


Patient presenting with joint pain
Initial evaluation:
Physical exam
Joint aspirate (obtain cell count, gram stain, culture)
FBP, ESR, Uric acid, ANA
X-rays of affected area
Consider other forms of arthritis
Symmetrical, with MCP, PIP joints affected in hands.
Presence of morning stiffness
Positive RF, ANA or anti-CCP
Pain plus 3 of the following:
-Greater than 50yo
-No morning stiffness.
-Crepitus on active motion of knee
-Bony tenderness or enlargement
-No warmth
Negative RF, ESR < 40
Presence of osteophytes on x-ray
Usually single swollen joint with pain and erythema (usually knee)
May have signs of systemic infection.
Joint aspiration with positive gram stain +/- culture (50,000 to 150,000 cells/mm3, turbid fluid)
Positive blood cultures
Single swollen joint with pain and erythema (usually MTP)
Involve surgery early!
Joint aspiration with uric acid crystals (gout) or calcium pyrophosphate crystals (pseudogout)
May see elevated uric acid levels but not always. Note: may also be falsely decreased
Xray with "overhanging edges"
Suspect rheumatoid arthritis
Suspect osteoarthritis
Suspect septic arthritis
Suspect gout or pseudogout
- Trial on NSAIDs (ibuprofen, diclofenac) – use with caution in patients with CKD
- Prednisolone 10-20mg to start
- Consider intraarticular corticosteroids (triamcinolone 10-40mg depending on size of joint) for symptomatic relief
- Begin disease-modifying antirheumatic drug therapy (DMARD) if symptoms not controlled within 3 months on NSAIDs and Prednisolone:
- hydroxychloroquine (mild RA)
- sulfasalazine (mild RA)
- methotrexate 7.5mg weekly (MTX) (moderate to severe RA) – obtain LFTs, Hep B and TB status prior initiating treatment
-Give folic acid the day after starting MTX 10mg over 2 days of the week
-Consider adjunctive nonpharmacologic therapies (e.g. physical therapy)
- Trial on paracetamol first
- NSAIDs (ibuprofen, diclofenac)
- Topical NSAIDs (e.g. diclofenac gel)
- Intraarticular corticosteroids (triamcinolone 10-40mg depending on size of joint)
- Encourage weight loss and physical therapy
- Consider chondroitin or glucosamine along with above therapy
- Antibiotic therapy for 14 days, tailor therapy based on culture results, empiric therapy with the following:
- Gram positive – Clindamycin 600mg IV tds
- Gram negative – Ceftriaxone 2g IV daily
- Emergent joint drainage via aspiration or arthroscopy
- Treat with NSAIDs (indomethacin 50mg tds or ibuprofen)
- For an **acute** attack - Colchicine 1.2mg STAT followed by 0.6mg an hour later (max 1.8mg in 24hrs), then 0.6mg daily for 3 days
-If contraindication to colchicine (severe renal or hepatic impairment) can use intraarticular joint injection (triamcinolone 10mg) or prednisolone 40mg od for 5 days
- For **recurrent** attacks, attempt lifestyle modifications (no alcohol, red meat, thiazide diuretics) or start Allopurinol 100mg od and uptitrate to achieve uric acid level < 6mg/dL, consider febuxostat, probenicid, or Colchicine if refractory to therapy with allopurinol