

Appendix 1. Hyperkalaemia ¹

Hyperkalaemia
Potassium >6.0 mmol/L

Life threatening?

Potassium > 6.5 mmol/L	<i>and/or</i>	ECG changes Bradycardia Tall and narrow T waves Prolonged PR interval Loss of P waves Broadening of QRS complexes Ventricular fibrillation	<i>and/or</i>	Symptoms Muscle weakness Paraesthesia Irregular heart beat Syncope
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Risk factors:

- Rapid onset
- Ischaemic heart disease
- Heart failure

Consider spurious hyperkalaemia
Delayed centrifugation or cold storage
High platelet or white cell count
Haemolysis
Potassium EDTA contamination
Familial pseudohyperkalaemia

Life threatening

Not life threatening

Urgent inpatient referral

Cardiac monitor

+

Immediate treatment

Intravenous calcium gluconate

and

Insulin and dextrose infusion

and/or

Salbutamol nebuliser

and/or

Dialysis

Exclude common causes

Acute kidney injury	Chronic kidney disease
Drugs	Excessive potassium intake
Potassium shift (for example, metabolic acidosis)	

Basic Investigations

Full blood count	ECG
Serum creatinine, urea and bicarbonate	Blood glucose

Cause determined

No obvious cause

Specialist referral

Endocrine	Renal
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Treat underlying cause
For example omit drug, remove high potassium food from diet

Retest

Less common cases

Mineralocorticoid deficiency (including Addison's disease)	Tumour lysis
Renal tubular acidosis (RTA type 4)	Rhabdomyolysis
Hyperkalaemic periodic paralysis	

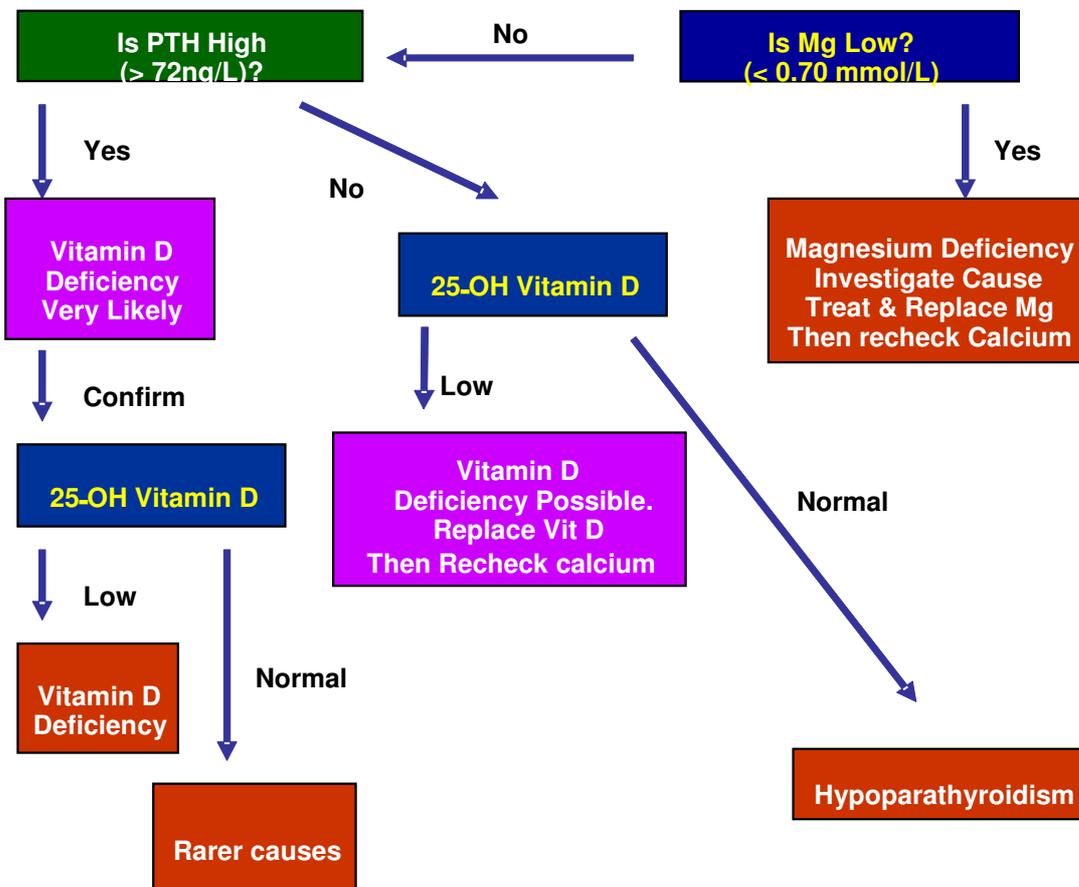
Special investigations

Short synacthen test	Creatine kinase
Plasma aldosterone and renin	

Investigating New Hypocalcaemia Summary

Adjusted Calcium < 2.20 mmol/L

Laboratory measures (usually within 24 hours):
 Creatinine If eGFR < 60 follow renal guidelines
 Phosphate Low value suggest vitamin D deficiency
 ALP High value suggests vitamin D deficiency
 Magnesium See below
 PTH See below

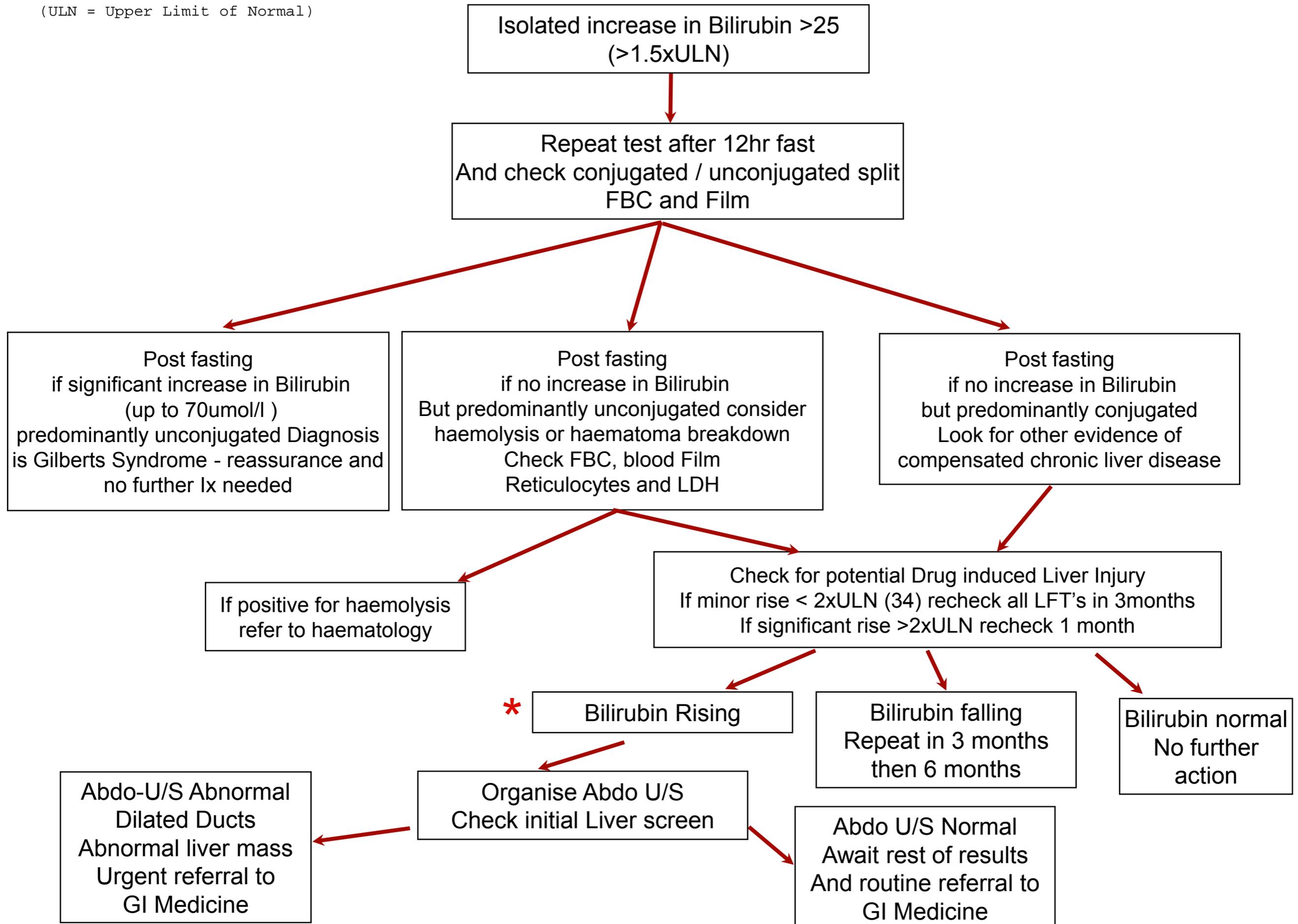


Hypoparathyroidism is not common and can arise from autoimmune disease, infiltration, post-surgery and congenital causes. All newly diagnosed hypoparathyroidism should be referred to a Consultant Endocrinologist for full assessment and management plan.

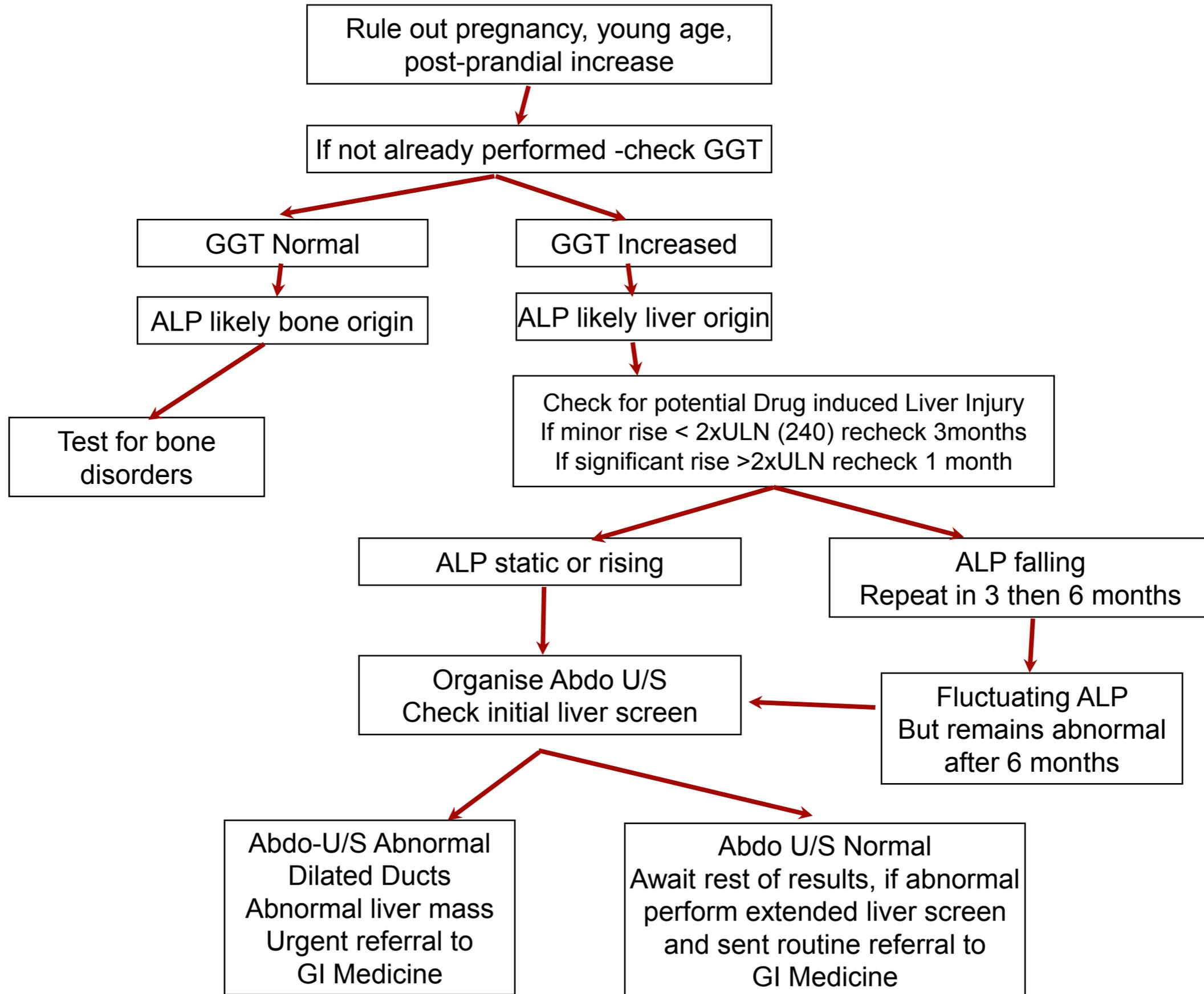
Investigation of an isolated rise in Bilirubin 1

APPENDIX 3 a

(ULN = Upper Limit of Normal)



Investigation of Isolated Raised Alkaline Phosphatase 2



Investigation of Isolated Raised Transaminases 3

APPENDIX 3 c
(ULN = upper limit
of normal)

Isolated increase in AST/ALT

Advise stop alcohol

Check for and stop any recently initiated hepatotoxic drugs
Advise **stop all** over the counter herbal /alternative products
If BMI > 25 or recent increase in wt - advise loss of 2-5kg
Discuss if high risk - current / previous drug misuse /partner of drug
misuser / from high prevalence area - check BBV
If <2xULN (<80) - recheck in 6 months
If >2xULN (>80) - recheck in 3 months
If >3xULN (>120) - recheck in 1month

If persisting degree of abnormality
or rising values at repeat testing
Reinforce above advice

If falling values at repeat test
Repeat in 6 -12 months

Abdo U/S - Abnormal
Dilated Ducts
Abnormal liver mass
Urgent referral to
GI Medicine

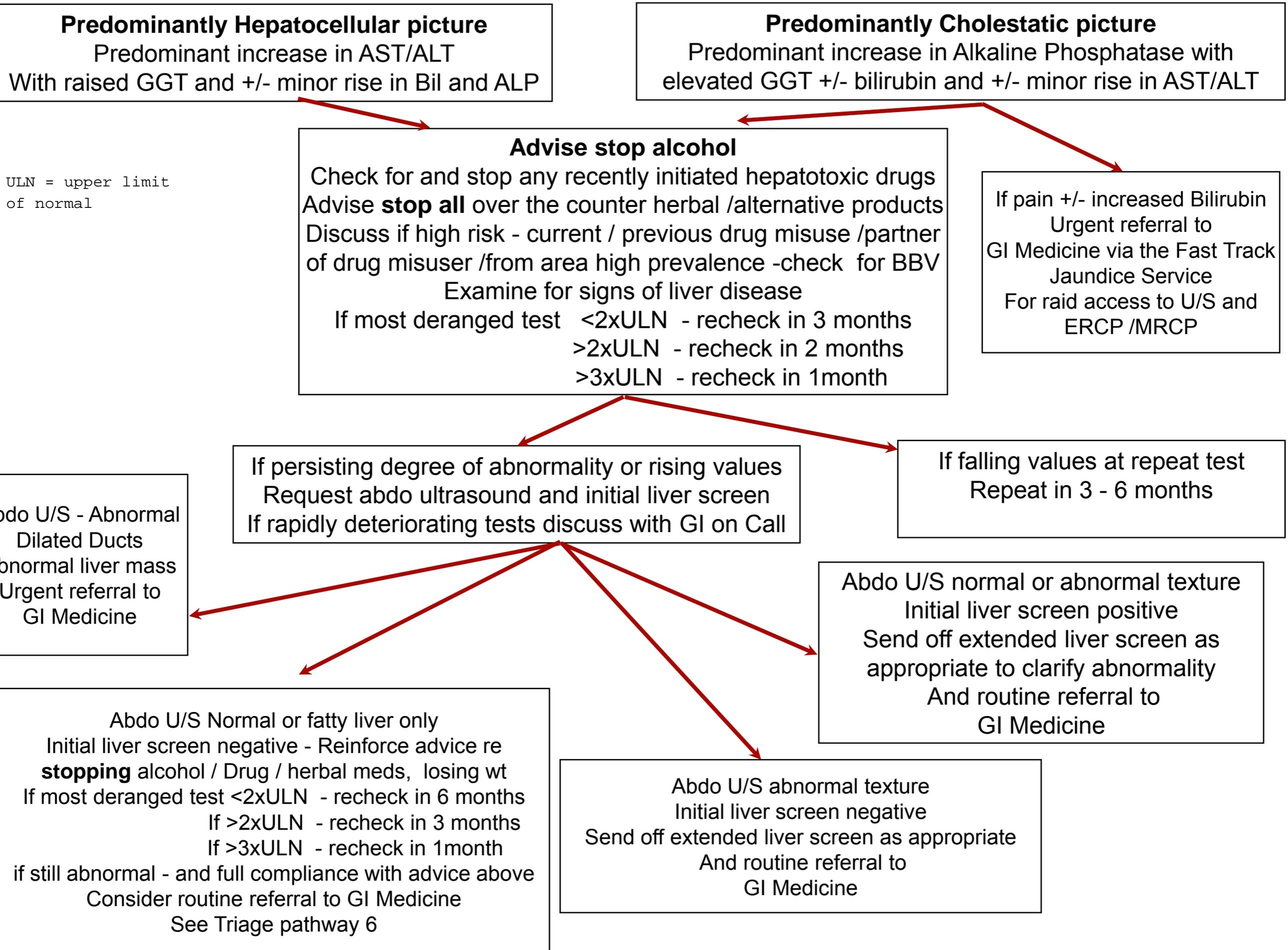
Organise Abdo U/S
Examine for signs of liver disease
Check Initial Liver screen

Abdo U/S normal or abnormal texture
Initial liver screen positive
Send off extended liver screen as
appropriate to clarify abnormality
And routine referral to
GI Medicine

Abdo U/S Normal or fatty liver only
Initial liver screen negative - Reinforce advice re
stopping alcohol / Drug / herbal meds, losing wt
If <2xULN (<80) - recheck in 6 months
If >2xULN (>80) - recheck in 3 months
If >3xULN (>120) - recheck in 1month
if still abnormal - and full compliance with advice above
Consider routine referral to GI Medicine
See Triage pathway 6

Abdo U/S abnormal texture
Initial liver screen negative
Send off extended liver screen as appropriate
And routine referral to
GI Medicine

Investigation of cluster of Abnormal LFT's 4



should include

Hepatitis Screen (hepatitis B surface antigen, hepatitis C antibody), HIV
liver autoantibody screen (anti-mitochondrial antibody, anti-smooth muscle
antibody), anti-nuclear antibody (ANF), serum immunoglobulins,
serum ferritin, Thyroid function tests (TFT's),
full clotting screen.

Also request Abdomineal U/S

Extended 'liver screen'

should include Tissue Transglutaminase (TTG) for coeliac disease

Anti-Neutrophil Cytotoxic Antibody (ANCA)

ceruloplasmin, *

α 1 antitrypsin level *

Alpha Fetoprotein

- ◆ if HBV sAg positive - labs should check other HBV markers and Hep B DNA load
- ◆ If Ferritin raised above ULN range check Serum Iron / Total Iron Binding Capacity (Fe/TIBC) = transferrin Sat% If Transferrin Sat% above 55% male or 50% female - check HFE (haemochromatosis) gene assay
- ◆ If Ceruloplasmin below 0.15g/l - on referral Hepatology will organise 24hr urinary copper excretion and ophthalmology review for Kayser Fleischer rings

Appendix 4. Investigation of low magnesium (Derbyshire Mg, emedicine)

[Editor's note: although there is no case on hypomagnesaemia in this module, the expert reviewer of this module receives frequent requests for advice on correcting low magnesium, so a brief summary is provided here.]

This condition can present with other biochemical abnormalities, as mentioned already in the Information Section. Magnesium is crucial in the renal re-absorption of potassium and calcium. Therefore, in some cases of hypocalcaemia and hypokalaemia, it is essential to replace magnesium to enable the correction of the other abnormalities.

As in other parts of this module, the degree of hypomagnesaemia can be important:

- magnesium 0.5 – 0.7mmol/L = not a medical emergency
- magnesium 0.3 – 0.5 mmol/L = possible medical emergency
- magnesium less than 0.3 = likely medical emergency.

Abnormal magnesium levels can result in disturbances in nearly every organ system, and can be fatal (ventricular arrhythmia or coronary artery vasospasm leading to sudden death).

Causes

Hypomagnesaemia is more common than people think, with probably 90% of cases not identified clinically. It is present in 25% of poorly controlled diabetics, 80% of alcoholics, and 90% of haematological malignancies. The commonest causes are GI and renal abnormalities.

Renal causes include:

- Drugs
 - proton pump Inhibitors (common cause)
 - diuretics
 - cytotoxic drugs
 - aminoglycosides
 - immunosuppressants
 - theophylline
- Osmotic diuresis (poorly controlled diabetes).

GI causes include:

- Reduced intake – i.e. dietary deficiency (rare, but occurs in alcoholics)
- Reduced absorption - coeliac disease, chronic diarrhoea, laxative abuse, fistulas, short bowel syndrome.

Treatment

Treat the causes above (e.g. stop the relevant drugs), and replace magnesium, which may need to be IV in severe cases. Intramuscular magnesium injections are very painful and are not recommended. Oral administration requires up to 50 mmol/L day. Suggested initial treatment is Maalox 10-20 ml qds (10ml Maalox = 6.8mmol Mg), rechecking magnesium 1-2 weekly initially depending on clinical context – this may take 6-8 weeks. Long term maintenance replacement may be needed if a reversible cause is not found and removed.

Although dietary causes are rare, the foods which are high in magnesium include

- green vegetables such as spinach (magnesium is contained in the chlorophyll molecule)
- some legumes (beans and peas)
- nuts and seeds
- whole, unrefined grains.

Magnesium glycerophosphate (1 tablet = 4mmol Mg, 1-2 tablets three to four times daily (12-32 mmol/day) is an unlicensed medication, available on a named patient basis, for cases of Maalox intolerance. Patients with renal Impairment should be treated with caution - obtain renal advice before commencing treatment if your patient has CKD stage 3 to 5.