Protocol of the initial management of decompensated alcohol-related liver disease

Stewart Campbell, April 2008

This guideline is intended to be very pragmatic. It is designed to offer guidance to junior and middle grade medical staff, particularly in the context of acute receiving, for optimal management within 24 hours of admission. It is not designed to be comprehensive guide to managing jaundice or liver disease, or to replace a formal gastroenterological opinion (which should be sought, for example, prior to considering corticosteroid therapy).

Identification of ALD patients and risk stratification

Alcohol abuse will usually lead to abnormal LFT’s, although only 20-30% of alcoholics will develop significant liver disease (i.e. alcoholic hepatitis or cirrhosis).

There are various scoring systems for grading severity of liver disease and mortality during an admission with ALD. For pragmatic reasons, these can be simplified:

- Bilirubin > 100 and INR > 1.5 = may have severe ALD or high mortality
- Bilirubin < 100 and INR < 1.5 = severe ALD unlikely, mortality from ALD likely to be low in the short term

These parameters can change rapidly, and patients should be scored on admission and also after 3-5 days if they remain as inpatients.

Remember, ALD usually causes a mild rise in AST and ALT. If AST or ALT are > 300, then you should suspect (strongly suspect if > 500):
- Concomitant paracetamol toxicity
- Hypotension causing shock liver (esp secondary to sepsis)
- Biliary obstruction/ cholangitis
- Viral hepatitis

Seek and Treat Infection

Most patients admitted with ALD will have a chronic history of liver dysfunction +/- jaundice. You should ask yourself “What has caused them to deteriorate suddenly or to seek help now”. Although not clinically obvious, the answer will often be sepsis.

- 1 in 8 cirrhotic patients admitted to hospital has spontaneous bacterial peritonitis (SBP). It is usually “silent”.
- 1 in 2 cirrhotic patients with GI bleeding has SBP during the course of their admission
Therefore: **Do a diagnostic ascitic tap on all decompensated ALD patients with ascites at presentation, or if there is deterioration in their condition**

This is simple:
- Take 20-30mls of ascitic fluid, using a small gauge (blue) needle if possible, and inoculate 5mls into an EDTA bottle, and the rest into blood culture bottles.
- An EDTA bottle is needed for the cell count. **It is the single most important test.**
- Ascitic fluid WCC>500, or PMNL>250 is diagnostic of SBP
- The culture yield rises from 40-70% if you use blood culture bottles.
- Don’t bother with measuring LDH, albumin, etc in the acute setting.
- Coagulopathy is not a contraindication (unless there is very severe thrombocytopenia or fibrinolysis).
- Remember to phone the lab- you need an instant result on the cell count (just as you would with CSF)
- Treat with at least 5 days of IV cephalosporin, and albumin (see below)

Don’t just diagnose & treat empirically, because:
- If SBP confirmed there is a requirement for lifelong antibiotics
- SBP is an indication to consider transplant referral
- If SBP is present, you should strongly consider giving IV albumin, although it would be dangerous to do this empirically

Don’t perform total paracentesis if infection is present (renal function will worsen)- but up to 3 litres could be removed to ease discomfort and lower abdominal pressure.
Treating 5 patients with SBP with IV albumin will save 1 life. (1.5g/kg over 24hrs on day 1, then 1g/kg on day 3, nil on day 2). This is because it prevents death from renal failure. (Sort, NEJM 1999)

**GI Bleeding**

GI bleeding is currently managed by the surgical team at Hairmyres hospital. There are some important general points about medical management:

*Glypressin* (1-2mg IV 4-6 times daily for 72 hours) is the only drug which has been shown to reduce mortality from variceal haemorrhage. It is contra-indicated in ischaemic heart disease. It can be given prior to endoscopic confirmation of varices, if there is a strong clinical likelihood of varies, although it does not replace the need for endoscopy (Levacher, Lancet 1996).

**Broad spectrum antibiotics** (IV then oral)- improve survival in cirrhotic variceal haemorrhage. Give them for 7 days post bleed, even if no signs of sepsis, and absolute mortality is improved by 10%. Seek SBP in those with bleeding.

If considering a *Sengstaken tube*, this should be done in a high dependency setting with intensivist support, by an experienced operator. These tubes can give
excellent control of haemorrhage, but have a high tube-related mortality (from aspiration, rupture, etc.) and require close monitoring.

**Fluid balance and renal failure**

This is the most challenging problem in ALD patients. However, there are some simple principles which can guide management.

Cirrhosis leads to vasodilatation, and consequently systemic hypotension. Most ALD patients therefore tend to have a “low-normal” blood pressure at best (e.g. 110-130mmHg systolic). Decompensated cirrhotics are very prone to the effects of dehydration, further vasodilatation, and hypotension. This is what causes *hepatorenal syndrome* (HRS). HRS was previously considered irreversible. We now know it can be reversed in 30-40% of cases with meticulous treatment of fluid balance problems and sepsis.

Therefore in decompensated ALD:

- Stop diuretics unless the patient is well
- Do not be afraid to give IV fluids including saline, especially in septic patients, (a total body saline overload can be treated later with paracentesis once the patient has stabilised, but restricting fluid may lead to worsening renal function)
- Minor degrees of HRS can be subtle- remember that alcoholics are often malnourished, and as such may have a “normal” urea of 1.5 and Creatinine of 30.
- Review drugs- stop NSAIDs, be very cautious with paracetamol in the acute withdrawal phase (certainly < 2g/day), avoid gentamicin unless essential.
- Hyponatraemia is not an indication for fluid restriction in ALD. It is a sign of intravascular volume depletion with free water retention, and will be exacerbated by fluid restriction.
- Dietary sodium restriction is appropriate.
- Oral *pentoxifylline* 400mg tds is relatively harmless and may prevent development of HRS. It can be started in ALD patients on admission, even if renal function is normal. Treating 5 patients may save 1 life (*Akriviadis, Gastroenterology* 2000)
- IV albumin +/- low dose glypressin may be indicated, but this should be discussed with a gastroenterologist.

**Encephalopathy**

- Rehydrate
- Review medication (esp sedatives)
- Assess for bleeding
- Assess for electrolyte imbalance, check BM
- Treat with lactulose and enemas
- No need to restrict protein intake