Alcohol Related Liver Disease and Alcoholic Hepatitis - Full Clinical Guideline

Reference no.: CG-T/2012/190

The spectrum of ALD includes simple steatosis, alcoholic steatohepatitis (ASH), progressive fibrosis and cirrhosis. Liver histology is required to confirm the diagnosis and this should be considered in patients with suspected cirrhosis, cofactors such as obesity or iron overload and in those patients in whom a period of abstinence does not lead to an improvement in liver enzymes. The majority of individuals drinking in excess of 60g (7.5 units) of alcohol/day develop steatosis, but only 10% of those with steatosis have cirrhosis after approximately 10 years. On the other hand 40% of those with steatohepatitis progress to cirrhosis.

Alcoholic hepatitis

Consider in patients who have developed jaundice within the last three months with alcohol misuse that is ongoing or stopped less than 4 weeks prior to presentation with jaundice. Patients often have fever, weight loss and tender hepatomegaly. In this situation, fever is not synonymous with the presence of infection although a high index of suspicion is appropriate. Liver Histology demonstrates the co-existence of steatosis, hepatocyte ballooning and an inflammatory infiltrate incorporating neutrophils BUT is not essential for diagnosis.

Management

ALL PATIENTS MUST HAVE THE "CIRRHOSIS CARE BUNDLE" COMPLETED WITHIN 12 HOURS OF ADMISSION- (additional specific requirements are outlined below)

- Pabrinex - 1 pair od for 3/7 or treatment doses if indicated (see prevention/ management of Wernicke's encephalopathy)
- Vitamin K 10mg daily iv for 3 days
- Dietician assessment
  - Prescribe Carbohydrate 50g load at night (ICM – "Carbohydrate shot")
  - Consider need for enteral feeding but balance with risk of aspiration (Consultant level decision)
- Screen for HBV, HCV and HIV at each new presentation and other elements of non-invasive screen if not previously performed
- Ensure infection screen including blood cultures in all patients, MSU, CXR and diagnostic ascitic tap if ascites present irrespective of clotting parameters
- Consider measures to prevent the development of an AKI i.e volume expansion
  - are the urea/ creatinine "normal" for the patient - compare with previous results
- Only start intravenous antibiotics in patients with positive microbiology or where physiology is deteriorating and then after discussion with Consultant or SpR

Calculate prognostic scores on Day 0 and Day 3:

- Maddrey's (modified) discriminate function (DF) - [4.6 x (PT -control)] + Bilirubin(umol/l)/17
  - Patients with DF < 32 have 28 day survival 94% and do not benefit from steroid therapy – STOPAH study figures[1]
- Glasgow score:

	Score given		
	1	2	3
Age	<50	⩾50	–
WCC (10^9/l)	<15	⩾15	–
Urea (mmol/l)	<5	⩾5	–
PT ratio	<1.5	1.5–2.0	>2.0
Bilirubin (μmol/l)	<125	125–250	>250

Management - HIGH RISK PATIENTS (DF ≥ 32 and/or Glasgow score ≥ 9) on Day 0

- Ask Consultant if patient is candidate for a clinical trial
- Review infection screen and re-calculate prognostic scores post iv vitamin K
- Patients who are stable after treatment for GI bleeding and/or infection (usually at least 48hrs) should be also be considered for steroids
  - ***If Glasgow score is* ≥ 9 and no signs of spontaneous improvement on Day 3 then start**:
- Prednisolone 40mg od (+ omeprazole 20mg) for 4 weeks (28d mortality treated 21% vs untreated 29.3% excluding those presenting with gi bleeding or sepsis). Serious infections occurred in 13% steroid treated patients versus 7% controls[2].
- Consider co-trimoxazole 960mg and fluconazole 100mg once daily for patients not receiving antimicrobials when steroids are commenced
- Calculate Lille score at day 7 - stop steroids if ≥ 0.56 (null responder), consider stopping if ≥ 0.45 (poor responder) **LilleModel** (http://www.lillemodel.com/score.asp)
- For day 7 non-responders arrange transjugular liver biopsy, repeat sepsis screen and consider empirical broad spectrum antibiotics and fluconazole**.**

N-acetylcysteine

- Can be added to steroids by giving:
- Intravenous N-acetylcysteine 150mg/kg per 24hrs in 1000 ml of 5% glucose solution over 24hrs

References

1 Thursz et al NEJM 2015 Apr 23;372(17):1619-28

2 Forrest E J Hepatol. 2017 Nov 21. pii: S0168-8278(17)32440-6

1. Documentation Controls

Development of Guideline:	Dr Andrew Austin
Consultation with:	Hepatology consultant and specialist nurse team
Approved By:	Hepatology – November 2018 Medical Division 15/11/18
Review Date:	December 2021
Key Contact:	Dr Adam Lawson