Hepatorenal syndrome

What is hepatorenal syndrome?

Hepatorenal syndrome is a complication of end-stage liver disease which occurs in patients who have chronic liver dysfunction with cirrhosis and ascites and also in acute liver failure. In hepatorenal syndrome (HRS) there is impaired renal function which is often precipitated by events lowering blood pressure.[1]

A number of factors can precipitate hepatorenal syndrome, including infections, alcoholic hepatitis and bleeding.[2]

How common is hepatorenal syndrome? (Epidemiology)[3]

HRS is a common complication of end-stage liver disease. The incidence of HRS is unknown. However, it is estimated that 35-40% of patients with end-stage liver disease and ascites eventually develop HRS.

Diagnostic criteria for hepatorenal syndrome[4]

HRS is essentially a diagnosis of exclusion - ie there is an absence of other identifiable causes of renal failure. The diagnostic criteria have been defined as follows:

Important information
Criteria for diagnosis of hepatorenal syndrome in cirrhosis [5] Diagnosis of cirrhosis with ascites. Diagnosis of acute kidney injury. No response after two consecutive days of diuretic withdrawal and plasma volume expansion with albumin. Absence of shock. No current or recent use of nephrotoxic drugs. No macroscopic signs of structural kidney injury - ie an absence of proteinuria, absence of microhaematuria and normal findings on renal ultrasound.

HRS is probably the result of a combination of the following: splanchnic vasodilatation leading to systemic circulatory dysfunction, activation of the sympathetic nervous system and renin-angiotensin-aldosterone system, and changes in cardiac output (usually low but is always less than the patient's requirements). In addition there is enhanced release of vasoactive mediators - eg, thromboxane A2 and endothelin-1. The end result is intrarenal arteriolar vasoconstriction.[6]

Diagnosis of hepatorenal syndrome

This is made after excluding other causes of renal failure in patients with liver failure:[7]

- Pre-renal causes (eg, whether there is history of dehydration, over-diuresis, GI fluid loss).
- Any history of nephrotoxic drugs.
- Whether there is history of shock before renal failure (which would suggest acute tubular necrosis).
- Whether there is any proteinuria ± haematuria, suggesting a parenchymal renal disease (renal ultrasound scan may be helpful) - particularly, glomerulonephritis (associated with hepatitis B/hepatitis C or chronic alcoholism).

Management of hepatorenal syndrome[4]

General measures

- Admit to hospital - ideally to the high-dependency unit.
- Monitor fluid status closely - eg, urine output and CVP monitoring which will help guide fluid replacement.
- Restrict fluids if necessary.
- Treat any precipitating infections aggressively. If there is no clear focus of infection, patients should still be started on broad-spectrum antibiotics - and a full course completed.[4]
- Avoid nephrotoxic drugs and stop diuretics.

Splanchnic vasoconstrictors

- Start splanchnic vasoconstrictors - eg, terlipressin in combination with albumin replacement.
- Terlipressin leads to an increased blood pressure and GFR, through constriction of splanchnic blood vessels.
- Monitor closely for: ischaemic heart disease, arrhythmias, fluid overload and digital ischaemia.
- Alternatives include noradrenaline (norepinephrine) or midodrine (unlicensed in the UK) and octreotide, although at present there is little experience of using these drugs.

Transjugular intrahepatic portosystemic shunt (TIPS)

- TIPS is used to reduce ascites in patients with portal hypertension, in those who do not respond to medical treatment.[4]
- It is contra-indicated in severe liver failure, which has limited its use.
- It may be used as a 'bridging' procedure whilst awaiting liver transplant, but can be complicated by liver failure and encepalopathy and should only be used in the context of a careful multidisciplinary assessment.[8]

Other methods

- The best chance of long-term survival would seem to be liver transplantation.

- Renal replacement therapy (RRT) may be necessary - eg, pulmonary oedema, severe hyperkalaemia or metabolic acidosis not responding to other treatment. Yet there are no data suggesting improvements in survival in HRS with RRT.

Liver transplantation [4]

- Liver transplantation is often the best option for HRS (whether or not they respond to vasoconstrictors).
- Combined kidney and liver transplant may be needed in patients with HRS who require prolonged renal support (ie >3 months).

Complications of hepatorenal syndrome

Life-threatening bacterial infections (septicaemia, spontaneous bacterial peritonitis, pneumonia)

- Gastrointestinal (GI) tract bleed.
- Superimposed infections - eg, pneumonia.

Prognosis [4]

HRS is associated with low survival, which varies with type and treatment and is significantly improved by liver transplantation. Some studies show a median ssurvival of untreated HRT to be less than two weeks, rising to a five-year survival of 60% for those treated with a liver transplant. [9]

Prevention of hepatorenal syndrome

- It may be possible to reduce the incidence of HRS in patients by early administration of albumin (especially in patients with bacterial peritonitis). [6]
- Pentoxifylline and norfloxacin may decrease the incidence of HRS in selected patients but further studies are needed. [4]

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