Hepatorenal syndrome precipitated by infective endocarditis

Introduction

Hepatorenal syndrome is defined as progressive kidney failure in a patient with liver cirrhosis (Ng et al, 2007), and is classified into two distinct types. The pathophysiology of both is similar, associated with alterations in blood flow and vessel tone in kidneys but without direct damage (Ginès and Arroyo, 1999). However, they differ in their speed of progression and prognosis. Hepatorenal syndrome is commonly precipitated by infections and gastrointestinal bleeding. Iatrogenic causes include excessive use of diuretics and drainage of large volumes of ascites by paracentesis. Spontaneous bacterial peritonitis is the commonest infectious cause (Ginès et al, 1993). This case report describes an unique presentation of hepatorenal syndrome secondary to infective endocarditis.

Discussion

A literature search has not identified any previous reports of a direct correlation between infective endocarditis and hepatorenal syndrome. Two case reports, one by Strizhakov et al (2010) and the other by Togashi et al (1996), illustrate a connection between the two entities. In both instances, however, the end outcome was part of the spectrum of multi-organ failure rather than a direct sequelae. In a study by Kobayashi et al (1984) 90 post mortems were conducted on patients with severe valvular heart disease, including but not exclusively those with infective endocarditis. Again, abnormal hepatorenal function was cited as a clinical feature in some cases but not related to hepatorenal syndrome specifically.

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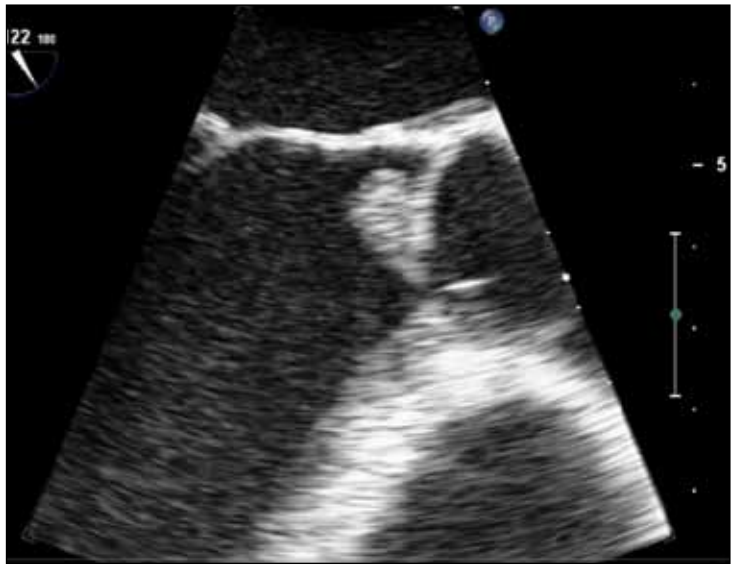
Infective endocarditis can cause a diverse range of systemic complications (Habib, 2006), including renal involvement. Several types of glomerulonephritis have been described in relation to infective endocarditis, as outlined by Neugarten and Baldwin (1983). The commonest are focal or diffuse proliferative forms, which are

Figure 1. Transoesophageal echocardiogram showing a mobile mass attached to the aortic valve.



Case Report

A 67-year-old man presented to the emergency department with a 3-week history of intermittent pyrexia, rigors and weight loss. Past medical history was relevant for previous sepsis, liver cirrhosis secondary to alcohol excess, hypertension and malignant melanoma. There was no history of foreign travel or recent contact with individuals with similar symptoms. On examination, he was pyrexial at 38.3°C with a sinus tachycardia of 100 beats per minute. He had auscultatory signs of mixed aortic valve disease. There were no cutaneous signs of infective endocarditis and the remainder of the examination was normal.

Initial blood investigations showed a normal full blood count (haemoglobin 13.5 g/litre, white cell count 9.97×10^9/litre, platelets 156×10^9/litre), creatinine of 106 μmol/litre and mildly abnormal liver function tests (bilirubin 28 μmol/litre, alanine aminotransferase 32 U/litre, aspartate aminotransferase 28 U/litre, alkaline phosphatase 198 U/litre, gamma glutamyl transferase 84 U/litre). C-reactive protein was notably elevated at 81 mg/litre. Four sets of blood cultures taken 5 days apart were consistently positive for coagulase-negative *Staphylococcus lugdunensis*, and he was commenced on intravenous vancomycin and oral rifampicin. A transthoracic echo showed a mobile echogenic mass attached to the aortic valve consistent with probable valve endocarditis (*Figure 1*). An abdominal ultrasound excluded hydronephrosis but confirmed moderate ascites and splenomegaly. An ascitic tap excluded spontaneous bacterial peritonitis. A subsequent computed tomography scan of the head revealed new low attenuation lesions in the left frontal lobe, consistent with infarction from septic emboli.

His renal function began to deteriorate on day 8 and he became oliguric, which was unresponsive to intravenous hydration. Simultaneously, he developed progressive hepatic encephalopathy. Tests for vasculitis and coagulopathy were consistently negative. His urinary protein:creatinine ratio of 33.3 excluded glomerular pathology and tests for urosepsis were negative. Hepatorenal syndrome was diagnosed and as other possible precipitants had been excluded, it was concluded that this had been initiated by infective endocarditis. He was commenced on terlipressin, human albumin solution and lactulose. His antibiotic regimen was changed to daptomycin (2 mg/kg/day) to remove the potentially nephrotoxic effects of vancomycin, although levels of the latter had consistently remained within normal range. He was not deemed to be a suitable candidate for liver transplantation and died 21 days after admission.

both immune complex-mediated. However, a more recent study by Majumdar et al (2000) suggested that vasculitis-related glomerulonephritis may be the most frequent form. In the current case report, a negative vasculitic screen and normal complement levels makes this diagnosis unlikely. With the presence of systemic emboli in the cerebral circulation, it was important to consider renal infarction, either local or advanced, as a cause of acute renal failure (Nonnast-Daniel et al, 1987). In the present case, the normal urinalysis, urinary protein:creatinine ratio and renal tract ultrasound makes such a diagnosis unlikely. Computed tomography of the abdomen is the investigation of choice for renal infarction but this was contraindicated in view of his abnormal renal function.

Conclusions

As hepatorenal syndrome is a diagnosis of exclusion, it is imperative to be aware of likely potential causes of renal pathology so that these can be rigorously excluded. However, in patients with a past medical history of hepatic dysfunction or features suggestive of decompensated liver disease, there should always be a high index of suspicion for hepatorenal syndrome. This will allow prompt initiation of supportive treatment with the hope of reducing patient morbidity and mortality. BJHM

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LEARNING POINTS

- Hepatorenal syndrome is a diagnosis of exclusion and it is therefore imperative to maintain an awareness of and eliminate other causes of renal dysfunction.
- It is usually precipitated by infections in susceptible individuals, particularly spontaneous bacterial peritonitis.
- To the authors' best knowledge, this case highlights the first documented presentation of hepatorenal syndrome secondary to infective endocarditis. Physicians likely to encounter such pathology, including cardiologists, nephrologists, hepatologists and microbiologists, should be aware of this association.

Dissecting thoracic aneurysm: an unusual presentation

An 80-year-old man presented to the emergency department having collapsed. On arrival his Glasgow Coma Scale was 9, his airway was patent with oxygen saturations of 100% and a respiratory rate of 30 breaths per minute. His heart rate was 69 beats per minute and irregular, and his blood pressure was 152/108 mmHg. On examination the patient had profound right-sided weakness with upgoing plantars bilaterally.

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A clinical diagnosis of cerebrovascular accident was made. An urgent computed tomography scan of the brain did not reveal any acute abnormality.

The patient subsequently became hypotensive. A computed tomography scan of the chest, abdomen and pelvis revealed a dissecting thoracic aneurysm (*Figure 1*). The patient was stabilized and transferred to the regional cardiothoracic unit, where he underwent successful emergency surgery.

This highlights the importance of adequate initial assessment and regular reassessment to detect any deterioration, allowing an appropriate response to a potentially fatal diagnosis. BJHM

Figure 1. Sagittal section of computed tomography scan showing a type A thoracic aortic dissection.