CASE REPORT

Fulminant liver failure: an indicator of silent myocardial rupture

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A 56 year old man presented with an atypical chest infection. Remote inferoposterior myocardial infarction was noted on electrocardiography and transthoracic echocardiography. Hepatic failure developed with sudden gross elevation of liver aminotransferases and coagulopathy. No primary hepatic cause could be identified. Subsequent right heart failure led to transoesophageal echocardiography that revealed a large inoperable ventricular septal defect. Histopathological data showed ischaemic hepatitis and reinfarction of the inferoposterior myocardial wall. Acute cardiac events may be silent and precipitate misleading severe hepatic dysfunction.

A 56 year old man presented with cough productive of purulent sputum and breathlessness. His previous medical history was unremarkable. In particular there was no history suggestive of ischaemic heart disease, excessive alcohol ingestion, or recreational drug use. He was a smoker of 80 pack years. Established medication was allopurinol, tramadol, and amitriptyline.

On initial examination, he was orientated but dyspnoeic at rest with a low grade pyrexia of 37.5°C, sinus tachycardia of 128 beats/min, hypotension (88/58 mm Hg), and tachypnoea (respiratory rate 20 breaths/min). Jugular venous pressure was slightly raised and a soft pansystolic murmur was heard at the left lower sternal edge. Coarse crepitations were present in both lung fields.

Abnormal investigations included raised white cell count of 21.7×10^9 and C-reactive protein of 149 mg/l. Biochemistry was mildly deranged with sodium 128 mmol/l, potassium 4.9 mmol/l, urea 15.1 mmol/l, and creatinine 129 μ mol/l. Liver function tests were also abnormal with alanine transaminase 197 IU/l, alkaline phosphatase 116 IU/l, bilirubin 39 μ mol/l, and lactate dehydrogenase of 490 IU/l. Blood clotting parameters and serial cardiac enzymes were normal. Inferior Q waves were noted on the 12-lead electrocardiogram. Chest radiography showed no focal abnormality. Transthoracic echocardiography demonstrated moderately

impaired right ventricular function with grade II tricuspid regurgitation. Left ventricular function was preserved apart from posterior septal dyskinesia.

A diagnosis of an atypical respiratory tract infection, in association with smoking related lung disease, was made and antibiotics (benzylpenicillin 1.2 g four times a day and erythromycin 500 mg four times a day) were started. A clinically silent remote inferoposterior myocardial infarction was suspected.

On day 3 the patient became icteric and developed marked peripheral oedema and ascites. Coagulopathy and gross elevation of liver aminotransferases (fig 1A) were found. Abdominal ultrasound confirmed abnormal liver texture. A comprehensive assessment of serum markers, viral and autoimmune autoantibodies was negative including A, B, C, E hepatitis viruses, cytomegalovirus, Epstein-Barr virus, influenza viruses, toxoplasma, mycoplasma, legionella, coxiella, brucella, and leptospira. Angiotensin converting enzyme, α_1 -antitrypsin, and caeruloplasmin were also within normal ranges. Ferritin was raised (2386 μ g/l) but since no other clinical features of haemochromatosis were evident this was attributed to an acute phase response.

Urgent transoesophageal echocardiography requested to further evaluate the right heart transthoracic echocardiogram findings was delayed by an upper gastrointestinal bleed and initial patient refusal of further investigation. Liver function improved but the patient's condition rapidly deteriorated with progressive peripheral oedema, hypoalbuminaemia, and renal impairment. With patient consent, transoesophageal echocardiography was performed on day 29 of admission, 24 hours before death. A very dilated right heart was seen communicating with the left ventricle via a large, inoperable ventricular septal defect (fig 2). The inferoposterior myocardial wall appeared aneurysmal with evidence of rupture.

Postmortem examination provided evidence of an old posterior septal myocardial infarction that had very recently reinfarcted with myocardial rupture and septal defect formation. Liver histology demonstrated florid centrilobular liver necrosis suggestive of ischaemic hepatitis (fig 1B).

Figure 1 (A) Graph depicting liver function test results during the admission. Note the sudden sharp transient elevation of alanine aminotransferase (ALT); ALP, alkaline phosphatase; BR, bilirubin. (B) Liver histology preparation (magnification $\times 250$) depicting centrilobular necrosis (arrow).

Figure 2 (A) Transoesophageal image showing a large ventricular septal defect (arrow). (B) A large left to right shunt is demonstrated with colour Doppler (arrow); LA, left atrium; LV, left ventricle; RV, right ventricle.

Learning points

- Acute right heart failure should be considered as a cause of multisystem disorder.
- Transoesophageal echocardiography is a useful investigation in deteriorating right heart failure without obvious cause.
- The clinical triad of a new left lower sternal edge pansystolic murmur, oedema, and hypotension without left ventricular failure are strongly suggestive of an acute ventricular septal defect.
- Ischaemic hepatitis can occur in patients with severe hepatic venous congestion arising from cardiac dysfunction and is characterised by a dramatic but reversible rise in transaminases and coagulopathy.

DISCUSSION

To our knowledge, this case report is the first to describe ischaemic hepatitis in association with an acute ventricular septal defect.

Ischaemic hepatitis is a condition produced from hypotensive liver anoxia. It is characterised by a pronounced, 10–20-fold reversible increase in serum transaminase concentrations in the absence of any other cause. Histological examination of liver biopsy characteristically shows centrilobular necrosis. Further serological features include raised lactate dehydrogenase, coagulopathy, and a degree of coexisting renal impairment.¹ Other terms used to describe this disorder include “shock liver” and “hypoxic hepatitis”. Prognosis depends very much on that of the underlying condition.

Literature review suggests that ischaemic hepatitis may be the result of impaired cardiac output, systemic hypotension, respiratory embarrassment, and hepatic venous congestion. Impaired cardiac output has been estimated to cause an incidence of ischaemic hepatitis of 21.9% in such patients admitted to coronary care units.² Hypoxia resulting from acute exacerbation of chronic respiratory failure has been implicated in the aetiology of up to 12% episodes of ischaemic hepatitis.³ This mechanism is likely to be complemented by concurrent venous congestion from associated cor pulmonale. Seeto *et al* studied cardiac function in a group of 31 patients with ischaemic hepatic presentation. They found a 94% incidence of biventricular or lone right ventricular failure thus corroborating the likely contribution of hepatic venous congestion to the aetiology.⁴ Treatment requires correction of the precipitating cause.

In the patient presented here, right ventricular impairment and an ineffective exacerbation of smoking related lung disease produced hepatic venous congestion and hypoxia, respectively. However reinfarction of the left ventricular septum and myocardial rupture were undoubtedly the triggering factor for the development of ischaemic hepatitis in this case. This catastrophic event produced systemic hypotension, left to right

shunting, acute deterioration in right heart function, and consequent severe exacerbation of hepatic venous congestion.

Since the myocardial ischaemia was clinically silent the profound derangement in liver function tests and clinical deterioration was felt initially to be a primary hepatic pathology or hepatic venous congestion secondary to right heart failure. The murmur across the defect could not be appreciated clinically because of its size and previously noted tricuspid regurgitation. The inferoposterior site of septal rupture could not be imaged by transthoracic echocardiography. This investigative technique was further limited since the patient's deteriorating condition gave rise to a reduction in the quality of available acoustic windows. Transoesophageal echocardiography was crucial in making the diagnosis but was unfortunately and unavoidably delayed. Surgical repair of the defect was not an option because of its size and because of the magnitude of operative risk in a patient who had sustained a recent myocardial infarction and who exhibited severe multiorgan failure at the time of diagnosis.

CONCLUSION

Acute ischaemic ventricular septal defects can be difficult to diagnose, particularly within the contexts of posterior coronary circulation interruption or silent ischaemic episodes. Transoesophageal echocardiography is an essential investigation especially if deteriorating right heart function is noted. It should be remembered that acute ventricular septal defects may present with multiorgan dysfunction with renal failure, severe hepatic venous congestion, and ischaemia.

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