Recurrent acute heart failure caused by sliding hiatus hernia

C-W Siu, M-H Jim, H-H Ho, F Chu, H-W Chan, C-P Lau and H-F Tse

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CASE REPORTS

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The case is reported of a 75 year old woman who presented with recurrent nocturnal episodes of acute pulmonary oedema. The cause was uncertain as she had normal cardiothoracic ratio on chest radiography and normal left ventricular systolic and diastolic function by transthoracic echocardiogram. Another transthoracic echocardiogram was repeated when she was recumbent for an hour and had a full stomach. It showed a striking finding of severe left atrial compression by an external structure. Computed tomography of the thorax showed an intrathoracic mass behind the left atrium causing external compression of the left atrium suggestive of a sliding hiatus hernia. Cardiac catheterisation confirmed the diagnosis by showing a pronounced rise of pulmonary capillary wedge pressure in the recumbent position compared with the sitting up position.

A 75 year old woman presented with recurrent episodes of shortness of breath and chest pain in the previous three months requiring multiple admissions. The diagnosis of acute pulmonary oedema was made but no cause could be found on previous admissions. Her cardiothoracic ratio was normal on chest radiography, her left ventricular function, both systolic and diastolic, were normal by transthoracic echocardiogram. Her symptoms occurred typically at bedtime, especially after a heavy dinner, and were associated with orthopnea, paroxysmal nocturnal dyspnea, and ankle oedema. Physical examination showed regular pulses with a normal blood pressure finding of 124/61 mm Hg. The jugular venous pressure was raised, the heard sounds were normal, and no murmur could be heard. There was bilateral ankle oedema as well as basal crackles heard over both lungs. An electrocardiogram showed normal sinus rhythm without any ischaemic or hypertensive changes. Careful examination of the chest radiograph showed congested lung field with mild bilateral pleural effusion compatible with acute pulmonary oedema. There was also a round shadow behind the heart with an air-fluid level within it. Blood tests including complete blood counts, renal and liver function test, and creatinine kinase activity were within normal limits. Transthoracic echocardiography was repeated when the patient was in the supine position for an hour and had a full stomach. It showed normal left ventricular function but the left atrium was severely compressed by an extrinsic structure confirmed by multiple views (fig 1). Spiral computed tomography of the thorax showed a large hiatus hernia with intrathoracic extension. The hernia was located behind the left atrium causing anterior shift of the heart (fig 2). Subsequently coronary angiography showed normal coronary anatomy. Right heart catheterisation showed that baseline right atrial pressure and pulmonary capillary wedge pressure during prolonged supine positioning were 8 mm Hg and 18 mm Hg respectively. However, after sitting upright for 30 minutes, the right atrial pressure and pulmonary capillary wedge pressure decreased to 5 mm Hg and 6 mm Hg respectively, confirming the diagnosis of significant left atrial compression by the sliding hiatus hernia. She was successfully treated with conservative measures including frequent small meals, avoidance of a late dinner, and sleeping in slanting position using several pillows. She had no further recurrence of acute pulmonary oedema in the subsequent 12 months.

DISCUSSION

Hiatus hernia is a common condition and its incidence increases with age.[1] It does not produce symptoms itself in most patients, but may contribute to the pathogenesis of reflux oesophagitis. Infrequently, sliding hiatus hernia may become incarcerated and strangulated, which may subsequently lead to acute chest pain, dysphagia, and a mediastinal mass.[2] Furthermore, cardiac compression with haemodynamic collapse has been reported in patients with complicated or large hiatus

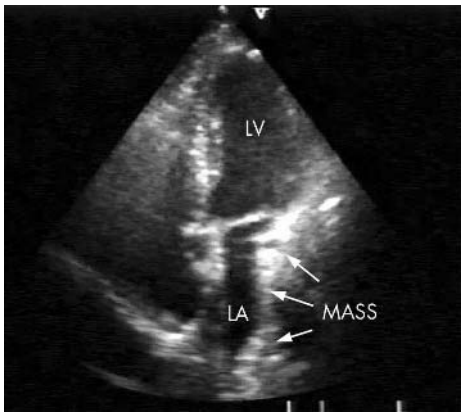

Figure 1 Echocardiogram in apical four chamber view showing extrinsic compression of the posterior wall (arrows) of the left atrium by a large mass. LV, left ventricle; LA, left atrium.

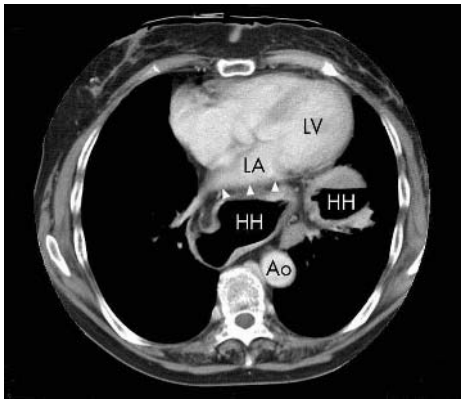

Figure 2 Computed tomogram of the thorax showing a large, mixed type hiatus hernia compressing the left atrium from posterior aspect (arrow). LV, left ventricle; LA, left atrium; HH, hiatus hernia; Ao, descending aorta.

hernia.[3 4] To our knowledge, this is the first reported case of recurrent acute heart failure caused by sliding hiatus hernia. As reported previously,[5 6] hiatus hernia may mimic a left atrial mass on transthoracic echocardiography, and is usually shown by spiral computed tomography as shown in this case. However, the clinical significance of these findings remains unclear. In this case, we performed detail cardiac haemodynamic measurements during supine and upright posture, and clearly showed the direct compressive effect of the hiatus hernia on the left atrium. This resulted in an increase in pulmonary capillary wedge pressure and subsequently contributed to the development of acute pulmonary oedema in this patient. This case shows that hiatus hernia is a potentially reversible cause of recurrent acute heart failure; accurate diagnosis and successful treatment of hiatus hernia can prevent further recurrence of acute heart failure.

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Acute liver failure: a message found under the skin

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Acute liver failure is a rare syndrome with rapid progression and high mortality. It is characterised by the onset of coma and coagulopathy usually within six weeks but can occur up to six months after the onset of illness. Viral hepatitis, idiosyncratic drug induced liver injury, and acetaminophen ingestion are common causes. This report describes the case of a 35 year old man who presented with acute liver failure shortly after binge drinking. Repeated history taking disclosed a gluteal disulfiram implant that the patient had received to treat his alcohol dependence. The patient recovered with maximum supportive care after surgical removal but without liver transplantation. This case illustrates that only meticulous history taking will disclose the sometimes bewildering causes of acute liver failure.

Acute liver failure is characterised by liver cell dysfunction leading to coagulopathy and hepatic encephalopathy, mainly attributable to viral, acetaminophen, or drug induced liver injury. Fulminant hepatitis is a rare but potentially fatal adverse reaction that may occur after the use of disulfiram, a drug used to treat alcoholism. We report a case of a 35 year old man who experienced acute liver failure associated with a gluteal disulfiram implant and alcohol misuse.

CASE REPORT

A 35 year old man first presented to a primary hospital in April 2003 with fatigue, vomiting, and vague abdominal complaints. His medical history included ongoing alcohol misuse despite various attempts of treatment. An alcohol binge had occurred three days before admission. On examination by the admitting physicians, he was jaundiced and drowsy. Initial laboratory studies showed increased aspartate aminotransferase (24012 U/l), total bilirubin (150 μmol/l), and blood alcohol (7.7 mmol/l). Transfer to our medical intensive care unit was arranged with a tentative diagnosis of alcohol induced liver failure.

On admission, the patient appeared acutely ill with pronounced jaundice, hepatic foetor, and hepatomegaly. Auscultation and percussion of heart and lungs were normal and the patient had no clinical signs of liver cirrhosis or portal hypertension. A 2 cm scar in his left lateral gluteal region was noted. Laboratory studies in our hospital on admission confirmed a massive increase in aspartate aminotransferase (60 620 U/l), alanine aminotransferase (16726 U/l), lactate dehydrogenase (38180 U/l), glutamate dehydrogenase (12211 U/l) total bilirubin (179 μmol/l), and lactate (5.2 mmol/l). Severe coagulopathy with thromocytopenia was present (INR 8.29; factor V 12%; 16 000/μl platelets), which precluded liver biopsy. Abdominal ultrasound showed hepatic oedema and excluded cirrhosis. The portal vein, hepatic artery, and hepatic veins were all patent. In view of progressive encephalopathy the patient was sedated and intubated. Cerebral oedema and haemorrhage were excluded by cranial computed tomography. Fluid refractory hypotension ensued, vasopressor support was begun, and anuric renal failure prompted continuous veno-venous haemodialysis. Fresh frozen plasma, platelets, packed red cells, factor XIII, and fibrinogen were given. Further laboratory tests excluded common causes of acute live failure like viral hepatitis A-C, Wilson's, and liver autoimmune diseases as