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.

DISCUSSION

Hiatus hernia is a common condition and its incidence increases with age. 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. Furthermore, cardiac compression with haemodynamic collapse has been reported in patients with complicated or large hiatus hernia.
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 the patient had no clinical signs of liver cirrhosis or portal hypertension. A 2 cm scar in his left lateral gluteal region was present (INR 8.29; factor V 12%; 16 000/µl platelets). 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 haemodiafiltration. Fresh frozen plasma, platelets, packed red cells, factor XIII, and fibrinogen were given. Further laboratory tests excluded common causes of acute liver failure like viral hepatitis A-C, Wilson’s, and liver autoimmune diseases as 

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judged by the absence of autoantibodies (ANA, SMA, LKM, SLA). A comprehensive drug screen was negative. High urgency orthotopic cadaveric liver transplantation was considered but declined on the basis of ongoing alcohol misuse in accordance with policies of German organ transplant legislation and Eurotransplant.

The medical history was scrutinised again to shed light on the aetiology of the liver failure. The patient was not receiving any medication and denied recreational or occasional exposure to drugs or toxic substances. It transpired that our patient had received a subcutaneous implantation of the oral drug disulfiram (Esperal) in his left buttock in Poland three months previously to “get rid of the drinking”. The implant was then immediately excised (fig 1). After surgical removal and under further supportive treatment the patient made an uneventful recovery after six days in the intensive care unit during which hepatic synthesis and detoxification normalised. He was then discharged to the referring hospital without neurological sequelae.

**DISCUSSION**

The main differential diagnosis in a 35 year old patient with acute liver failure would include alcohol induced liver disease, acetaminophen intoxication, viral hepatitis (predominantly HBV) as well as drug reactions and other rare diagnoses such as autoimmune hepatitis, Wilson’s disease, and Budd-Chiari syndrome.1

Disulfiram has been in use for adjunctive treatment of severe alcoholism since 1948.2 A thiuram derivative, it inhibits the second step of ethanol metabolism by inhibition of acetaldehyde dehydrogenase.3 This leads to immediate accumulation of acetaldehyde and results in nausea, flushing, and vertigo. By virtue of this action it exerts a penalising effect on alcohol consumption.2,3 However, disulfiram has been widely abandoned because of its unfavourable safety profile. Inadvertent ingestion of alcohol may cause severe acetic aldehyde reaction requiring medical assistance.3 Fulminant hepatitis after the use of disulfiram usually occurs within the first two months after disulfiram treatment, with symptoms suggestive of acute hepatitis including fatigue, malaise, anorexia, nausea, vomiting, abdominal pain, jaundice, fever, rash, and pruritus.3 The pathophysiology, however, has not been elucidated.3 Both accumulation of toxic metabolites such as carbon disulfide, an end product of the disulfiram metabolism, and immunological mechanisms have been suggested.4,5 Forns et al concluded that disulfiram hepatotoxicity is mainly produced by the accumulation of toxic metabolites,6 whereas many case reports are consistent with a hypersensitivity reaction and include clinical findings such as eosinophilic infiltrates, arthralgia, fever, rash, and pruritus.6 Depot preparations of disulfiram have been described in the literature albeit without proper evaluation of their benefit-hazard ratio.3 Notably, concomitant alcohol misuse opens the possibility of aggravated reactions to drugs.3 Based on the literature, we believe that an idiosyncratic adverse drug reaction of disulfiram is the most probable pathophysiological mechanism, which is compatible with the course of the disease.

**CONCLUSION**

Our patient experienced liver failure associated with a gluteal disulfiram implant and alcohol misuse. This case illustrates that acute liver failure can have a bewildering aetiology while concomitant alcohol misuse opens the possibility of aggravated reactions to drugs such as disulfiram induced toxic hepatitis. Maximum supportive care was started only after the implant had been discovered and appreciated as a potentially reversible cause of hepatotoxicity.

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Submitted 23 August 2004

Accepted 30 August 2004

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Acute liver failure: a message found under the skin

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Postgrad Med J 2005 81: 269-270
doi: 10.1136/pgmj.2004.023382

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