Acute Hepatic Failure Caused by an Acute Aortic Dissection with Cardiac Tamponade: A Case Report

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ABSTRACT

An acute cardiac tamponade from any cause may result in rapid deterioration of hepatic function in a previously healthy patient. We describe a case of an acute ascending aortic dissection that presented as acute hepatic failure, due to an acute cardiac tamponade and severe right heart failure. The differential diagnosis of the aetiology of acute liver failure is extensive and includes poisonings, vascular obstruction and sepsis, particularly on the background of decompensated liver disease. Many of these conditions are associated with hypotension. The acute presentation in our patient, combined with the lack of a characteristic history delayed the diagnosis of a proximal (type A) dissection with tamponade and subsequent hepatic failure. Severe right-sided heart failure as a result of conditions such as cardiac tamponade should be excluded in patients presenting with acute hepatic failure of unknown aetiology.
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Key words: Cardiac tamponade, acute hepatic failure, acute aortic dissection

The classical presentation of an acute aortic dissection is generally well recognised and leads to confirmatory investigations and definitive management. The combination of chest pain radiating to the back and asymmetric upper limb blood pressure readings, associated with neurological deficit or loss of peripheral pulses necessitates the consideration of a thoracic aortic dissection. There are a few cases in the literature of acute aortic dissection that have resulted in hepatic failure. Most of the patients that pursued a chronic course made a full recovery. All recorded cases were due to loss of arterial blood supply.[1-4]

Cardiac tamponade is not an uncommon sequela of aortic dissection and, uncorrected, results in poor cardiac filling, elevated venous pressures, diminished cardiac output and death. However, the development of acute hepatic failure secondary to cardiac tamponade is not well reported. We present a case in which the primary presenting feature was an acute hepatic failure due to an undiagnosed acute ascending aortic dissection with cardiac tamponade.

CASE REPORT

A 74 year-old female, who had a mild episode of diarrhoea and vomiting associated with a small amount of blood in her stool a few days previously, was found with a Glasgow Coma Score (GCS) of 3, two hours after being last seen fit and well. Apart from a small skin tear above the right eye, there were no signs of trauma. As the blood sugar testing at the scene demonstrated a blood sugar level of 0.7mmol/L, 1 mg of glucagon was given intramuscularly.

Her past medical history included a mild transient ischaemic attack 2 years ago, a hysterectomy some years

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previously and two laparotomies for adhesive bowel obstructions. There was no history of cardiac disease or hypertension.

On presentation to the emergency department at 1955 hours, the patient's conscious state had improved. She had a GCS of 14 and no focal neurological features. However, she had poor peripheral perfusion and was hypotensive with a mean arterial pressure (MAP) of 50 mmHg despite the administration of two litres of 0.9% saline intravenously. Clinical examination revealed abdominal distension with abdominal tenderness in the right upper quadrant and mild splenomegaly. Bowel sounds were present, femoral pulses were normal and there was no clinical evidence of an abdominal aortic aneurysm. There were no clinical signs of heart failure and no cardiac bruits.

A computerised tomography (CT) scan of the head was within normal limits. A chest X-ray showed clear lung fields, an unfolded aorta and a normal mediastinum. The initial arterial blood gas (ABG) revealed a PaO_2 216 mmHg, $PaCO_2$ 23 mmHg, HCO_3^- 14 mmol/L and a pH 7.39. The blood glucose was 17.9 mmol/L and the troponin I level was < 0.4 (Table 1).

She was admitted to the intensive care unit where a further 1.5 L of intravenous fluid was administered (0.5 L 0.9% saline and 1 litre of Haemaccel®). However, she remained hypotensive and anuric. The ABG at this stage revealed a PaO_2 139 mmHg, $PaCO_2$ 26 mmHg, HCO_3^- 12 mmol/L and pH 7.28 (Table 1). The plasma biochemistry showed a blood sugar level of 23.8 mmol/L, venous lactate 12 mmol/L, urea 5.2 mmol/L and creatinine 0.13 mmol/L. The plasma hepatic enzyme levels were elevated with an ALP of 118 U/L, GGT 116 U/L, ALT 537 U/L, AST 728 U/L, LDH 963 U/L and total bilirubin of 6 µmol/L. The haemoglobin was 118 g/L and white cell count was 12.5 x 10^9/L. The coagulation profile showed an INR of 1.3 and an APTT of 36 seconds. The paracetamol level was < 10 mg/L.

A central venous catheter was inserted at 0130 hours which recorded a central venous pressure of 8 mmHg. A non-contrast CT scan of the abdomen revealed an enlarged liver, thickened gall bladder wall and a small amount of pelvic free fluid. A provisional diagnosis of an intra abdominal vascular event with sepsis was made and broad-spectrum antibiotics (ceftriaxone, metronidazole, ampicillin, gentamicin, and flucloxacillin) were administered and an adrenaline infusion commenced. Seven hours after admission the patient became

Table 1. Plasma biochemical and arterial blood gas values on admission and during the illness

Variable	*Time(hours)*					*Reference range*
	2030	2300	0300	0600	1000	
Urea	5.2	6.0	7.9	8.7	7.4	3.0 - 8.0 mmol/L
Creatinine	0.13	0.15	0.16	0.15	0.16	0.07 - 0.10 mmol/L
Glucose	17.9	23.8	15.3	15.1	14.9	3.0 - 8.0 mmol/L
AST	310	728	773	816	544	< 35 U/L
ALT	209	537	501	518	464	< 35 U/L
LDH	565	963	816	837	966	100 - 250 U/L
ALP	120	118	107	108	40	35 - 140 U/L
GGT	101	116	115	126	34	< 50 U/L
Bilirubin		6				< 20 µmol/L
Ammonia	76	101		54		< 50 µmol/L
PaO_2	216	139	165	157	120	80 - 110 mmHg
$PaCO_2$	23	26	29.3	56	46	35 - 45 mmHg
HCO_3^-	14	12	11.6	7.8	12	22 - 31 mmol/L
pH	7.39	7.28	7.22	6.9	6.95	7.35 - 7.45
Base Excess	-8.8	-13.3	-14.7	-24.8	-19.4	-5 - 5 mmol/L
Lactate		12.0	7.9	9.0	14.8	< 2.5 mmol/L
Haemoglobin	130	118	118	118	100	110 - 165 g/L
White cell count		12.5				4.0 - 11.0 x10^9/L
Platelets	269		105	68	20	150 - 400 x10^9/L
INR		1.3	2.0	2.4	4.3	0.8 - 1.2
APTT		36	57	60	>150	24 - 39 seconds
Troponin I	< 0.4		3.6	6.8	3.9	< 0.4 ug/L

INR = International normalised ratio, APTT = activated partial thromboplastin time, AST = aspartate amino transferase ALT = alanine amino transferase, LDH = lactic acid dehydrogenase

confused and encephalopathic. She was afebrile, hypotensive and remained refractory to resuscitation. Repeated plasma biochemical tests showed worsening hepatocellular function and an elevated troponin I level (Table 1).

A diagnostic laparotomy was performed which revealed torrential haemorrhage from the whole of the liver surface with no other major intra-abdominal abnormalities. The hepatic artery was pulsating normally and the portal vein was patent. In an attempt to reduce the hepatic surface bleeding the liver was packed and the abdomen closed. The patient was returned to the intensive care unit where a review of her condition led to a trans-oesophageal echocardiogram (TOE) being performed. This revealed a proximal (type A) aortic dissection with a probable entry point 2 - 3 cm above the left posterior cusp of the aortic valve and extending distally to the arch and proximally to the aortic root (Figure 1). There was an associated pericardial effusion with biatrial and right ventricular compression indicating cardiac tamponade (Figure 2). The left ventricular size was normal and demonstrated vigorous systolic function.

Due to the clinical condition of the patient, which was believed to be terminal, further intervention was considered unwarranted by the cardiothoracic unit and comfort care was initiated. The patient died 21 hours after admission.

DISCUSSION

Acute hepatic failure has been described infrequently as the presenting feature of acute pericardial tamponade. In addition, there have been only a few cases reported where an acute aortic dissection has presented with minor or absent localising signs and progressive hepatic dysfunction as the dominant feature. In a study of 109 patients with acute ascending aortic dissection, Ehrlich *et al*,[5] identified pre and postoperative factors that predicted mortality. An element of liver dysfunction was found in 6.4% of patients in the postoperative period and was identified as a statistically significant predictor of mortality. However, liver dysfunction was not identified as a preoperative predictor of mortality. Turner and Pussey[1] described three patients who had a prolonged illness accompanied by fever, weight loss, high erythrocyte sedimentation rate, neutrophilia and abnormal liver function tests. Aortic dissection was diagnosed 3 - 12 weeks after the onset of the illness and all patients survived with resolution of their organ dysfunction following antihypertensive treatment.[1]

We have identified only 3 cases in the literature of acute aortic dissection presenting with acute hepatic dysfunction as the dominant feature. All of these cases exhibited dissection to the bifurcation of the aorta with the hepatic dysfunction caused by obstruction of hepatic arterial flow and loss of portal flow due to extraluminal obstruction of the celiac and mesenteric arteries. All of these patients died.[2-4] In contrast, in the case we describe the acute hepatic dysfunction was due predominantly to right heart failure secondary to cardiac tamponade.

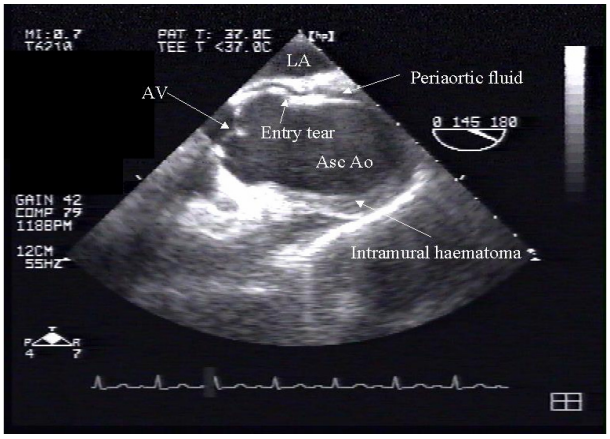

Figure 1. Long axis transoesophageal echocardiographic image of the proximal ascending aorta demonstrating the intramural haematoma involving the anterior aortic wall and periaortic fluid posteriorly. The site of an entry tear is shown (AV = aortic valve, Asc Ao = ascending aorta, LA = left atrium).

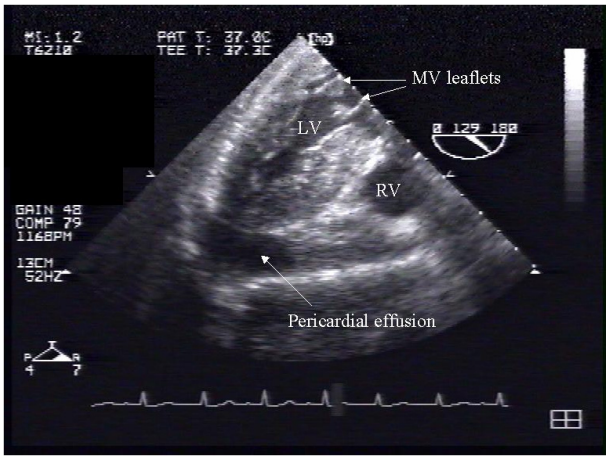

Figure 2. A transoesophageal echocardiographic image demonstrating the pericardial effusion. (LV = left ventricle, MV = mitral valve, RV = right ventricle).

The development of cardiac tamponade has been recognised to be a preoperative complication in 15.5% of ascending aortic dissections that do not have a pulse deficit.[6] This results in venous hypertension and eventually poor left ventricular filling and systemic arterial hypotension. These factors combine to decrease the transhepatic pressure gradient and produce a congested liver with inadequate oxygen delivery. The patient we describe had no obstruction to the portal vein or hepatic

artery, and there was no evidence of distal aortic dissection beyond the aortic arch. Therefore, the cause of hepatic dysfunction was most likely due to the cardiac tamponade, rather than the more commonly described dissection of arterial vessels supplying the liver.

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