Investigation vignette

An 80 Year old Man with a Five Day History of Vomiting, Renal Failure and Jaundice

CASE REPORT

An 80 year old man was admitted to hospital with a one month history of progressive malaise and a five day history of vomiting, renal failure and jaundice. He had a past history of a right hip replacement but otherwise had been in reasonable health. A provisional diagnosis of hepatitis had been made by his local medical officer and he was admitted to hospital for further management.

On admission he had mild peripheral oedema. His pulse was 94 beats per minute, respiratory rate 18 per minute, blood pressure 145/70 mmHg and temperature 36.4°C. His liver function tests revealed a bilirubin of 64 μmol/L, ALT 1240 U/L and ALP 40 U/L. His plasma creatinine was 0.22 mmol/L. He had no history of recent exposure to hepatotoxins, drank alcoholic cider occasionally and during his 5 day stay in hospital, serology tests for HAV, HBV, HCV, HEV, CMV and EBV were all negative. A diagnosis of 'sepsis' was made, although there was no clinical evidence of infection and his temperature, white cell count and C-reactive protein estimations remained within normal limits. However, as his hepatic and renal functions progressively deteriorated, admission to the intensive care unit (ICU) was requested.

At this stage he had clinical features of peripheral cyanosis (e.g. cyanosis of ears, fingers and knees) his blood pressure was 160/70 mmHg, pulse 88 beats per minute and pulse oximetry revealed a saturation of 98% on nasal oxygen at 4 L/min. The arterial blood gases revealed a PO_2 of 76 mmHg, PCO_2 of 18 mmHg and a pH of 7.45. A biochemical profile performed on admission to the ICU (Figure 1) led to the diagnosis.

Name	Age	Sex	Time of Collection	Time of Analysis	Date
Mr. R. D.	80	M	1945	2030	16.3.01

Sodium	134	mmol/L	(137 - 145)
Potassium	4.7	mmol/L	(3.1 - 4.2)
Chloride	101	mmol/L	(101 - 109)
Bicarbonate	12	mmol/L	(22 - 32)
Anion Gap	16.7	mEq/L	(8 - 16)
Calc Osmolarity	302	mmol/L	(280 - 300)
Glucose	7.0	mmol/L	(3.0 - 6.0)
Urea	27.2	mmol/L	(3.0 - 8.0)
Creatinine	0.33	mmol/L	(0.05 - 0.12)
Albumin	30	g/L	(39 - 50)
Globulins	24	g/L	(22 - 35)
LD	1183	U/L	(110 - 200)
ALT	1104	U/L	(10 - 45)
AST	967	U/L	(10 - 45)
GGT	50	U/L	(0 - 50)
ALP	103	U/L	(30 - 100)
Total bilirubin	82	μmol/L	(4 - 20)

Figure 1. Plasma biochemical profile performed on a venous blood specimen taken from the patient on admission to the intensive care unit.

Diagnosis: Ischaemic (hypoxic) hepatitis

Ischaemic hepatitis was suspected in this patient as his ALT/LD ratio was 0.93 with clinical evidence of poor peripheral perfusion. A pulmonary artery floatation catheter was inserted which revealed a right atrial pressure of 25 mmHg, pulmonary artery pressure of 70/35 mmHg, pulmonary artery occlusion pressure (PaOP) 31 mmHg and cardiac index of 0.76 L/m^2/min. A transthoracic echocardiograph was perf-ormed which showed a markedly dilated left ventricle with global dysfunction (estimated ejection fraction 10%), dilated left atrium and right ventricle, moderate mitral regurgitation and mild tricuspid regurgitation.

The patient was treated with captopril (2 mg 4-hourly increasing over the next three days to 50 mg 8-hourly) and digoxin (0.75 mg i.v. followed by 0.25 mg at 2 hr and 0.25 mg at 12 hr). One litre of 5% dextrose and 1 litre of 0.9% saline were also administ-ered in the first 24 hours. Over the next three days the treatment was associated with a reduction in blood pressure from 160/75 to 110/50 mmHg, a decrease in PaOP from 31 mmHg to 18 mmHg and an increase in cardiac index from 0.76 L/m^2/min to 2.35 L/m^2/min. However, the pulmonary artery pressure remained elevated at 70/35 mmHg, although the right atrial pressure decreased from 25 mmHg to 9 mmHg. A progressive reduction in the plasma ALT, LDH and creatinine also occurred and were all within normal limits 10 days after his admission. He was discharged from hospital 2 days later.

Ischaemic hepatitis is not an uncommon complica-tion of cardiac failure and is believed to be due to elevated systemic venous pressure causing hepatic congestion associated with a reduction in hepatic arterial blood flow. The hepatic injury is characterised by centrilobular necrosis in the absence of inflamm-ation and is often associated with acute renal impairment.[1,2] The diagnosis is usually suspected in a patient with chronic cardiac failure who has a sudden reduction in systemic blood flow or increase in right heart failure due to an acute myocardial infarct, pulmonary embolism, pneumonia or arrhythmia,[3] although cardiac failure may have been previously unrecognised.[4] It also occurs in cirrhotic patients following haemorrhagic shock[5,6] and has been described in patients with sleep apnoea syndrome.[7,8]

The diagnosis is often made in a patient with cardiac or circulatory failure who has a mild elevation of plasma bilirubin, prothrombin time and ALP, and a characteristically greater increase in plasma LD compared with plasma ALT or AST.[2,9,10] One study found that the mean serum ALT/LD ratio in patients with ischaemic hepatitis was 0.87, compared with 1.46 in paracetamol hepatitis and 4.65 in viral hepatitis. They concluded that a serum ALT/LD ratio of < 1.5 differentiated ischaemic hepatitis from paracetamol hepatitis and viral hepatitis with a sensitivity of 94% and a specificity of 84%.[9] With the correction of cardiac failure, the enzyme changes usually resolve rapidly (i.e. > 50% decrease within 72 hr).[1,3]

The patient in this report had an unsuspected cardiac failure. The ischaemic hepatitis and renal failure may have been provoked by the associated fluid loss due to vomiting.

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