Confusion and lethargy in a 48 year old man

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Answers on p 244.

A 48 year old retired fireman was admitted with a one week history of confusion, lethargy, and generalised weakness. This was associated with a non-productive cough for which he had been prescribed oral amoxycillin. He had a history of hypertension and was on lisinopril 20 mg daily. He was a smoker of 30 pack years and consumed 30 units of alcohol per week.

On admission his temperature was 37.7˚C, pulse rate 100 beats/min, and blood pressure 80/40 mm Hg. He was clinically dehydrated, but otherwise clinical examination did not reveal any abnormalities. His mini-mental score was 8/10.

On admission, initial investigations revealed a haemoglobin concentration of 142 g/l and white cell count 5.5 x 10^9/l with a normal differential white cell count. Prothrombin time was 16.2 seconds (control 12 seconds). Biochemical tests revealed evidence of renal impairment with urea 43.4 mmol/l, creatinine 416 µmol/l, sodium 128 mmol/l, potassium 4.3 mmol/l, and corrected calcium 2.3 mmol/l. Urinalysis was normal and urinary sodium excretion was 11 mmol/24 hours in keeping with pre-renal renal failure.

Liver tests revealed bilirubin 18 µmol/l, alkaline phosphatase 88 U/l, γ-glutamyl transferase 195 U/l, aspartate aminotransferase 88 U/l, albumin 18 g/l. Blood glucose concentration was 5.9 mmol/l and creatine kinase was normal. Paracetamol and salicylate levels were normal. Multiple blood cultures revealed no growth.

Initial electrocardiography revealed a sinus tachycardia, and chest radiography demonstrated right basal atelectasis.

Subsequent investigations including antistreptolysin titre, autoimmune screen, iron studies, serum angiotensin converting enzyme level, leptospirosis and brucella serology were normal. Viral hepatitis screen and HIV test were performed, after informed consent. Initially hepatitis C and HIV antibodies were detected and reported as reactive, however these were later confirmed to be negative.

An electroencephalogram demonstrated features consistent with hepatic encephalopathy.

Ultrasound of the abdomen demonstrated slightly enlarged kidneys with no hydronephrosis, a bulky spleen and moderate hepatic enlargement. Computed tomography was performed which is shown below (fig 1); a specimen was also taken for histology (fig 2).

Initial management included fluid resuscitation, careful fluid balance, and broad spectrum antibiotics. Over the next few days his renal function tests returned to normal. However his liver tests and prothrombin time deteriorated, his confusion increased, and he continued to have daily pyrexia.

Three weeks after admission the patient made a spontaneous clinical and biochemical recovery without any further specific treatment. He was subsequently treated appropriately when further results became available. He remained well at six month follow up.

QUESTIONS
(1) What does the computed tomogram show and in light of this, what is the most appropriate investigation?
(2) What is the histological diagnosis? List the possible causes for this.
(3) Can you link the histological diagnosis with the clinical history described and suggest the management of this condition?

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Figure 1 Computed tomogram of abdomen.

Figure 2 Histology specimen (haematoxylin and eosin stain).