Final diagnosis**Keywords:** eosinophilic fasciitis; soft tissue tumour

Eosinophilic fasciitis.

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Acute anxiety – not always a psychiatrist's problem

S P L Meghjee, R J T Wilson

A previously fit 37-year-old man was a psychiatric admission, with acute onset of lethargy, hyperventilating, vomiting, and paraesthesia in his hands. He had a stressful job, with difficult financial and family circumstances. He was a strict Mormon Christian, and on direct questioning, denied taking any drugs. Except for hyperventilating, his initial examination was normal. An initial diagnosis of acute anxiety state was made, and he was treated with diazepam and chlorpromazine. Fourteen hours after admission, he developed fever of 38.7°C and became pale, clammy, sweaty and drowsy. He had no rash. His pulse was 150 beats/min with blood pressure of 180/90 mmHg. Pulse oximetry showed an oxygen saturation of 89% on 60% inspired oxygen. His Glasgow Coma Scale was 14/15 with no focal neurological signs. The rest of the examination was normal.

Despite being on maximal inspired oxygen via face mask, he deteriorated further with increasing tachycardia, labile blood pressure and falling saturation levels, and hence was formally anaesthetised, paralysed, and ventilated. Chest X-ray and electrocardiogram (ECG) rhythm strip are shown in figures 1 and 2. His initial blood tests, and invasive monitoring results were as follows: haemoglobin 17.8 g/dl, white cell count $22 \times 10^9/l$, platelets $407 \times 10^9/l$, sodium 141 mmol/l, potassium 4.6 mmol/l, urea 11.4 mmol/l, creatinine 227 mmol/l, blood glucose 6.9 mmol/l, adjusted calcium 2.40, INR 1.9, and activated partial thromboplastin time 86.2 s (control 27–38.0). Pre-ventilation arterial blood gases showed pH 7.378, pCO_2 2.73 kPa, pO_2 11.7 kPa, bicarbonate 12.2 mmol/l and base excess of –9.9 mmol/l. Anion gap was 25. Invasive monitoring revealed a central venous pressure of 7 cmH₂O, pulmonary artery wedge pressure of 7 cmH₂O (14–18), cardiac index of 4.8 l/min/m² (2.5–4.2) and systemic vascular resistance of 346 dynes/cm⁵ (800–1400).

Figure 1 Chest X-ray **Figure 2** ECG rhythm strip

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Questions

- 1 What abnormalities do the ECG rhythm strip and chest X-ray show?
- 2 What do the arterial blood gases show?
- 3 How would you interpret the invasive monitoring data?
- 4 What is the differential diagnosis?
- 5 What is the treatment?

Answers

QUESTION 1

The ECG (rhythm strip) shows sinus tachycardia at a rate of 150 beats/min. The chest X-ray shows pulmonary oedema with upper lobe diversion and fluid in the horizontal fissure.

QUESTION 2

The arterial blood gases show mixed metabolic acidosis and respiratory alkalosis.

QUESTION 3

These data are consistent with septic shock syndrome or SIRS (Systemic Inflammatory Response Syndrome), ie, high cardiac index with low pulmonary wedge pressure and decreased systemic resistance.

QUESTION 4

The differential diagnosis is as follows:

- septicæmia secondary to meningitis
- poisoning due to, eg, salicylate, methanol, ethylene glycol, or paracetamol
- neuroleptic malignant syndrome
- acute liver failure.

The diagnosis in our patient was severe salicylate poisoning (levels of 858 mg/l).

QUESTION 5

It is strongly advised to contact the nearby Poisons Unit for specialist advice. Gastric lavage should be considered within 4 hours if more than 120 mg/kg body weight salicylate has been ingested, followed by multiple oral dose of activated charcoal. Metabolic acidosis should be corrected with intravenous sodium bicarbonate (1.5 litres 1.26% over 3 hours if the plasma salicylate concentration is greater than 500 mg/l). Fluid should be given according to clinical examination and invasive monitoring parameters.

Haemodialysis is the treatment of choice in severe poisoning with plasma salicylate levels greater than 700 mg/l. Plasma salicylate levels should be repeated to ensure that treatment has been effective (personal communication, National Poisons Information Service).

Discussion

Over 22 000 cases of aspirin overdose were reported to the American Association of Poison Control Centre in 1989.¹ Of these cases, 65% of patients were treated in healthcare facilities, 5% had a 'moderate' reaction, 0.6% had a 'severe' reaction, and approximately 0.1% died due to the overdose.

Most cases of salicylate intoxication in adults and teenagers are due to attempted suicide. In toddlers it is due to accidental overdoses, and is therapeutically acquired in elderly patients.² The side-effects of aspirin can occur at any dose, and include dyspepsia, nausea, gastrointestinal ulceration, hypersensitivity, haemolysis and nephrotoxicity. Common features of mild aspirin intoxication are vomiting, tinnitus, deafness, sweating, warm extremities with bounding pulses, increased respiratory rate and

Table Calculation of probable toxicity

Ingested dose (mg/kg)	Likely toxicity
150	mild
250	moderate
500	severe, possibly fatal

Causes of metabolic acidosis with high anion gap

- ketoacidosis: diabetes, alcohol poisoning
- drug poisoning: salicylate, methanol, paraldehyde, ethylene glycol
- uraemia
- lactic acidosis: type A (eg, shock, severe hypoxia), or type B (eg, metformin, acute liver failure, poisoning with paracetamol or ethanol)

Box 1

Poor prognostic factors

- late presentation
- age >70 years
- hyperpyrexia
- pulmonary oedema

Box 2

hyperventilation. In moderately severe reaction, the patient will also have hyperpyrexia, dehydration, restlessness, ecchymoses and loss of coordination. In severe poisoning, disorientation, coma, oliguria, pulmonary oedema, cyanosis and hypoglycaemia are also found.³

A mixed respiratory alkalosis and metabolic acidosis is present in every case, the severity depending on degree of intoxication. Respiratory alkalosis is due to direct stimulation of the respiratory centre, and the metabolic acidosis is due to uncoupling of oxidative phosphorylation and lactic acidosis (box 1). Rarely, patients can have gastrointestinal obstruction due to concretions of the tablets.³

The risk to a patient with intoxication can be estimated if one knows the ingested dose (table), and the serum concentrations of salicylic acid.² Deaths occur in patients whose concentrations exceed 700 mg/l (5.1 mmol/l). Poor prognostic factors are shown in box 2.

Salicylate poisoning represents an acute medical emergency. Its treatment is discussed above, but it must be stressed that advice from a Regional Poisons Unit is paramount. Attempting to force diuresis probably does not enhance salicylate excretion and may cause pulmonary oedema (personal communication, National Poisons Information Service.)

Final diagnosis

Severe salicylate poisoning

Keywords: anxiety; aspirin intoxication; metabolic acidosis; respiratory alkalosis; salicylate poisoning

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Hepatomegaly and multiple liver lesions

Ho-Choong Chang, Ba Nguyen, Fintan Regan

A 33-year-old man was referred for possible liver transplantation. The patient was initially diagnosed at birth when he presented with an enlarged liver and episodes of hypoglycaemia. A liver biopsy at the time showed pale hepatic cells by virtue of cytoplasmic granularity and periportal nuclear ballooning (figure 1). He was treated initially with dietary modifications but subsequently required night time dextrose and corn starch. Failed medical therapy prompted referral for liver transplant evaluation. Physical examination showed massive hepatomegaly. Liver function tests were abnormal with a significantly raised alkaline phosphatase and transaminase. Sonography showed hepatomegaly with multiple focal lesions unchanged in size since ultrasound 3 years earlier. Computed tomography (CT) showed multiple well-defined low-attenuation lesions throughout the liver. The largest of these measured $8 \times 8 \times 4$ cm and contained foci of coarse calcification (figure 2).

Figure 1 Liver biopsy showing pale hepatic cells

Figure 2 Axial CT scan showed multiple well-defined low-attenuation lesions throughout the liver

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Questions

- 1 What is the probable diagnosis, and what enzyme deficiency and type of inheritance characterise this disease?
- 2 What substance accumulates in the liver and kidney?
- 3 What liver complications are associated with the disease?