

SHORT REPORTS

Toxic myocarditis in paracetamol poisoning

We describe the case of a young girl who died in cardiac failure 80 hours after a paracetamol overdose. Necropsy showed congested lungs, a dilated left ventricle, and myocardial muscle necrosis.

Case report

A 15 year old girl was admitted 55 hours after taking an unspecified quantity of paracetamol. She was fully conscious and oriented. Her pulse rate was 88 beats/min regular and blood pressure 110/70 mm Hg. The liver edge was palpable and tender. Plasma paracetamol concentration was 41 mg/l, serum sodium concentration 138 mmol/l, serum potassium concentration 4.2 mmol/l, plasma urea concentration 3.0 mmol/l, plasma bicarbonate concentration 10.0 mmol/l, prothrombin time 65 seconds, serum bilirubin concentration 56 µmol/l, serum alanine aminotransferase activity 790 IU/l, and serum aspartate aminotransferase activity 1075 IU/l.

Over the next 12 hours her plasma bicarbonate concentration improved to 16.6 mmol/l but serum potassium concentration decreased to 3.9 mmol/l. Serum creatinine concentration was 115 µmol/l at 15 hours, and urinary output exceeded 2 l in the first 21 hours. She received an intravenous infusion of dextrose, oral neomycin, vitamin K, cimetidine, and lactulose. Her blood pressure remained stable at 100/70 mm Hg, but persistent sinus tachycardia was recorded up to 21 hours after admission, when there was a sudden onset of cardiac arrhythmia and hypotension. Various atrial and ventricular arrhythmias were recorded. Electrocardiography showed gross ST segment depression and T wave inversion in leads 2, 3, aVF, V1-4. She became unconscious, with dilated pupils and absent brain stem reflexes. Spontaneous breathing ceased, and intermittent positive pressure ventilation was started. She went into ventricular tachycardia, followed by cardiac asystole, 25 hours after admission.

Necropsy showed a dilated left ventricle, pale myocardium (particularly subendocardially), which was soft in consistency; patent coronary arteries; and normal valves. Myocardial histology showed focal infiltrates of neutrophils and occasional mast cells among necrotic myocardial muscle fibres. Both lungs were diffusely oedematous. The liver was soft and showed punctate areas of centrilobular necrosis. Liver histology showed almost complete necrosis of hepatocytes, most pronounced in the centrilobular zones, with vacuolation of necrotic cells around the lobules. Pigment laden macrophages were present, but no inflammatory infiltration was found. The kidneys were slightly swollen with pale cortices. The brain was diffusely oedematous, but there was no coning of the brain stem and no evidence of necrotising myopathy.

Comment

The combination of hypotension, cardiac dilatation, and pulmonary congestion, in the absence of brain stem compression, made acute cardiac failure the immediate cause of death in this patient. Left ventricular dilatation is not a feature of fulminant hepatic failure, and this finding thus suggests toxic myocarditis. The absence of focal necrotising myopathy, a normal serum potassium concentration, and the improved plasma bicarbonate concentration¹ also indicate that cardiac failure was not related to the general metabolic disturbance.

Two previous reports of myocardial necrosis in paracetamol poisoning were questioned because other factors might have contributed to the outcome.^{2,3} Dixon evaluated 20 unselected fatal cases of paracetamol poisoning confirmed by toxicology and claimed to have found no obvious cardiotoxicity, yet more than a quarter of these patients died from unexplained cardiac arrest.⁴ He did not define his criteria for cardiotoxicity, and his conclusion must also be questioned. More importantly, he found no correlation between hepatic necrosis and death from paracetamol poisoning. Indeed, five of the patients he studied showed no hepatic necrosis at necropsy. Three others died from cardiac arrest, and a further three died from cerebral anoxia, which was itself a result of earlier cardiac arrest or inhalation of vomit. Only three patients died in unequivocal hepatic failure. He concluded that hepatic failure was not necessarily the cause of death in paracetamol poisoning and suggested the "acute toxicity" effect.

The number of patients who died from cardiac arrest in this small sample is large enough to suggest an alternative mechanism. The earliest histological change in the cardiac muscle after coronary artery occlusion may be detected five hours later,⁵ or at 30 minutes with a special stain. Consequently, the absence of morphological evidence of cardiotoxicity in the six patients dying from cardiac arrest in Dixon's series does not rule out cardiotoxicity, assuming that standard histological techniques were used. In our case the finding of cardiac dilatation at necropsy and polymorph infiltration of the myocardium provides a helpful lead in this direction.

We thank Professor R A Risdon, professor of pathology at the Institute of Child Health, London University, for providing us with a copy of his postmortem report.

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- 2 Sanerkin NG. Acute myocardial necrosis in paracetamol poisoning. *Br Med J* 1971;iii:478.
- 3 Pimstone BL, Uys CJ. Liver necrosis and myocardiopathy following paracetamol overdose. *S Afr Med J* 1968;42(11):259-62.
- 4 Dixon MF. Paracetamol hepatotoxicity. *Lancet* 1976;ii:35.
- 5 Olsen EGJ. Myocardial infarction. In: Silverman MD, ed. *Cardiovascular pathology*. Vol 1. Edinburgh: Churchill Livingstone, 1983:406-19.

(Accepted 10 July 1987)

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Hyperthyroidism and de Clérambault's syndrome in a young woman

The features of de Clérambault's syndrome,¹ or erotomania, are a delusional belief of being loved by another person, usually of a higher status, who has been the first to express love and make advances. The onset is sudden, and the object of the delusion of love remains unchanged. In addition, the patient gives an explanation for the paradoxical behaviour of the loved one.

This chronic syndrome may be a primary disorder or secondary to other illnesses, such as schizophrenia, mania, alcoholism, or epilepsy. Ellis and Mellsop, reviewing reports of the syndrome, noted that the primary disorder was rare and that most of the cases were of secondary erotomania.² There are no previous reports of erotomania in patients with hyperthyroidism.

Case report

The patient, a 36 year old, single woman, worked as a clerk in a government office. She complained that her colleagues had laughed when she confided in them that the director was in love with her. Feeling hurt, she had started to cry and had accused them of being jealous.

She had a five year history of hyperthyroid Graves' disease but was non-compliant with treatment, and her attendance at the follow up clinic was irregular.

On admission she was restless and irritable. She had lost 5 kg in four months, although her appetite was good. There was a fine tremor of the hands, and her pulse was 96 beats/min. The thyroid gland was diffusely enlarged and soft, and a bruit was heard.

Thyroid function tests indicated thyrotoxicosis, thyroxine concentration being 156 nmol/l (normal range 55-145), thyroid stimulating hormone <0.5 mU/l (normal range 0.5-4.0), thyroxine binding globulin 186 nmol/l (normal range 168-324), and the ratio of thyroxine to thyroxine binding globulin 8.4 (normal range 2.8-4.8).

During the interview she mentioned that about two months previously she had noticed that whenever the director entered the office he would look at her. She also said that every morning he followed the bus that she took to work in his car. They had never spoken to each other, but she thought that this was "because he was a married man and it could be very embarrassing." She had sent 14 letters to him, and in one letter she wrote: "You loved me first and I have responded." When he did not reply she wrote again saying, "I hate you, but I know you are in love with me."

She came from a family of four children and was the only daughter. She had no steady boyfriend and worried that she might not get married. Except for the hyperthyroidism there was no important medical or psychiatric history.

The director said that he had not known her previously; there were about 300 workers in the department and he could not remember every face or name.

She was treated three times daily with carbimazole 10 mg, propranolol 20 mg, and chlorpromazine 50 mg. She responded well and was euthyroid after four weeks. The delusion of love persisted until the fifth week after admission, when she began to realise that "the whole thing is quite absurd and very embarrassing to him and me." After she was discharged she agreed to be transferred to another office "to avoid meeting him or my colleagues."