A PUZZLING CASE OF CARDIAC ARREST

BY

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SUMMARY

A case of cardiac arrest during anaesthesia for appendicectomy is presented. The diagnosis was uncertain and seemed to lie between malignant, hypertonic hyperpyrexia and endotoxic shock, but the features were not typical of either syndrome.

This case presented a problem in diagnosis, which was not resolved by postmortem examination.

CASE REPORT

A 33-year-old Malay soldier was admitted complaining of vague abdominal pain of 2–3 days' duration. He had had one previous admission 8 months previously for a similar pain, which resolved rapidly. Otherwise he had had no serious illnesses or operations and his family history was unremarkable.

On examination he was a fit-looking man of normal physique, weighing 120 lb. His temperature was 37.2 °C and diffuse abdominal tenderness was noted. Chest X-ray was normal.

After observation for 2 days, appendicectomy was proposed. He was premedicated with papaveretum 20 mg and hyoscine 0.4 mg, 1 h before induction. Anaesthesia was induced at 14.00 hours with thiopental 400 mg and suxamethonium bromide (Brevidil) 20 mg. A cuffed, oral, endotracheal tube was passed. Anaesthesia was maintained with nitrous oxide, 33 per cent oxygen and 0.5 per cent halothane, ventilation being controlled using a Manley ventilator, at a tidal volume of 500 ml, and a minute volume of 10 L/min. Relaxation was provided by repeated doses of suxamethonium bromide to a total of 160 mg.

As soon as anaesthesia was established, a mass was palpable in the right iliac fossa and the operation proceeded better than anticipated. The appendix being inflamed, friable and adherent, and required considerable dissection. Accordingly an intra-abdominal drain was left. About 2 litres of blood were lost and he was transfused with 5 packs of blood and given fibrinogen and aprotinin, because fibrinopenaemia and a degree of fibrinolysis were demonstrated. The electrocardiogram showed a pattern of acute myocardial injury which gradually improved over the next 6 hours. Clinically he was unconscious, cyanosed and rigid. Further bicarbonate was given as acid-base measurements showed the blood pH to be 6.82. Rheomacrodex 1 litre was given i.v. No abnormal haemoglobins or malarial parasites were demonstrated in the blood.

Cooling was stopped at 18.10 hours but the temperature continued to fall and remained at 35 °C. The systolic pressure remained persistently at 50 mm Hg and a noradrenaline drip was started in an attempt to raise mean arterial pressure and improve coronary filling. Warming with blankets was commenced at 18.55. Further treatment consisted of hydrocortisone 100 mg, carbenicillin and sodium bicarbonate up to a total of 150 m.equiv. By midnight his pupils were fixed and the rigidity was unchanged.

The patient then started to bleed from venepuncture sites and from the wound drain. About 2 litres of blood was lost and he was transfused with 5 packs of blood and given fibrinogen and aprotinin, because fibrinopenaemia and a degree of fibrinolysis were demonstrated. Central venous pressure was at this time 25 cm H₂O, but this fell to normal over the next few hours.

The patient remained in a similar clinical state, being ventilated by an East-Radcliffe ventilator, with added oxygen, with no recordable pulse or blood pressure but with relatively normal cardiac electrical activity. This gradually lessened and disappeared and at 14.00 hours the next day he was pronounced dead.
Postmortem examination was unrevealing. There was moderate atheroma but no coronary occlusion. His lungs were congested and oedematous. There was considerable inflammation around the appendicectomy site and blood-stained fluid was present in the peritoneal cavity. The central nervous system was macro- and microscopically normal. He had a very small spleen (90 g) and his adrenals were small but not haemorrhagic. There was no evidence of disseminated intravascular coagulation.

**DISCUSSION**

Malignant, hypertonic hyperpyrexia has been recognized with increasing frequency in recent years. This syndrome has not to the author's knowledge been reported in a Malay though Tobias and Miller (1970) described this condition in a Chinese boy. Most cases have occurred in patients of European or African race.

This case is not typical, however, as the serum potassium was only marginally raised, there was no overt myopathy or family history, no myoglobinemia (tests for myoglobinuria were not performed) and the hyperpyrexia was moderate and responded to cooling. There was an unusual difficulty in rewarming, but Hawthorne, Richardson and Whitfield (1968) and Britt and Gordon (1969) reported cases in which hypothermia was noted before death. It would have been interesting to know the blood sugar level in this patient because presumably if the brain is deprived of substrate, glucose metabolism must stop and the temperature fall. However, hyperkalaemia may be transient, myopathy absent and other members of the family may not have been subjected to general anaesthesia. The clotting defect occurred late and may have been due to undemonstrated disseminated intravascular coagulation, secondary to the hypotensive and hypothermic state. Succinylcholine has been shown to cause hyperkalaemia, cardiac arrest and cerebral damage (Tolomie, Joyce and Mitchell, 1967), but the rigidity preceded arrest and there was no cerebral abnormality at postmortem examination. There was nothing in the anaesthetic course to suggest cerebral anoxia before arrest took place.

Endotoxic shock was a distinct possibility in view of the operative procedure, the pyrexia and the hypotension. Blood cultures were not performed, although a negative result does not exclude toxaemia. In retrospect it might have been useful to try the effects of vasodilators, such as phenoxybenzamine, instead of noradrenaline (Hardaway et al., 1967).

While a syndrome of septicaemia due to pneumococcal infection and disseminated intravascular coagulation associated with splenectomy or a small fibrous spleen has been described by Bisno and Freeman (1970), it is possible that this man might have been more susceptible to auto-infection than one with a normal spleen.

Cerebrovascular accident and coronary thrombosis were ruled out by the postmortem examination and the return of the initially ischaemic electrocardiogram to normal.

Finally it is worth recording that it was later discovered that the patient had been consulting the bomoh (local religious medicine man) for some time and was told that if he went into hospital he would die. Most patients who die after this intelligence just waste away and die of inanition. The author could not discover a report of a previous death under anaesthesia following suggestion of this type.

In view of the lack of certain diagnosis in this case, the cause of the cardiac arrest must remain speculative. It would appear to underline the necessity, restressed by Drury and Gilbertson (1970), for the establishment of a central investigating committee for malignant, hypertonic hyperpyrexia.

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**REFERENCES**


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UN MYSTERIEUX CAS D'ARRET CARDIAQUE

SOMMAIRE

L'auteur rapporte un cas d'arrêt cardiaque durant l'anesthésie pour une appendectomie. Le diagnostic fut incertain et on hésita entre une hyperpyrexie hypertonique maligne et un choc endotoxique, mais la symptomatologie ne fut typique pour aucun de ces syndromes.

UN CASO INTRIGANTE DE PARO CARDIACO

RESUMEN

Se presenta un caso de paro cardíaco durante anestesia para appendicectomía. El diagnóstico fue dudoso, quedando entre hipertrofia hipertónica maligna y shock endotóxico, pero los síntomas no fueron típicos para ninguno de estos síndromes.

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