Hyperpyrexia due to meningococcal septicaemia treated with cold peritoneal lavage

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Summary: An 18 year old male presented with fulminant meningococcal septicaemia and renal failure. He subsequently developed hyperpyrexia which failed to respond to conventional methods of treatment. Cold peritoneal lavage was employed which resulted in rapid lowering of his core temperature and helped manage his renal failure. The possible causes of the late onset of hyperpyrexia and the use of cold peritoneal lavage to treat refractory hyperpyrexia are discussed.

Introduction

Sustained hyperpyrexia can be life-threatening unless treated rapidly and effectively.

A variety of clinical conditions are associated with hyperpyrexia. We describe a case of meningococcal septicaemia complicated by hyperpyrexia which was successfully treated with peritoneal lavage using unwarmed dialysate.

Case report

An 18 year old apprentice bricklayer was admitted with a one day history of pyrexia, myalgia and a generalized purpuric rash. On examination he was unwell with a systolic blood pressure of 70 mmHg and a pulse of 140/min. His core temperature (rectal) was 38°C. He had a widespread haemorrhagic rash involving the trunk and limbs. He had no evidence of meningism.

Investigations revealed a neutrophil leucocytosis of $21 \times 10^9/l$, a haemoglobin of 174 g/l and a platelet count of $54 \times 10^9/l$. He had evidence of disseminated intravascular coagulation with a prothrombin time of 26 s (control 15 s), and an activated partial thromboplastin time of 106 s (control 48 s). The fibrinogen titre was 1/16 with a control of 1/128. His blood urea was 22 mmol/l and serum creatinine 422 $\mu\text{mol/l}$.

A provisional diagnosis of meningococcal septicaemia was made and the patient was commenced on intravenous benzylpenicillin. Circulatory sup-

port was provided with plasma expanders and inotropes and was monitored by measuring pulmonary artery wedge pressure, peripheral vascular resistance and cardiac output. The diagnosis was confirmed by the isolation of *Neisseria meningitidis* Group C sensitive to penicillin from blood cultures.

During the next 24 h oliguric renal failure and respiratory distress ensued necessitating haemodialysis and intermittent positive pressure ventilation. His core temperature on the second day of admission was 38°C. On day 3 of his admission his core temperature rose to 42.6°C. A computed tomographic scan of the brain excluded an abscess, haemorrhage or cerebral oedema. Attempts to reduce his core temperature with conventional methods such as fanning and tepid sponging with ice-cold water for 2 h did not succeed.

A semi-rigid peritoneal dialysis catheter was introduced in the midline infraumbilically and intermittent peritoneal dialysis was commenced using dialysate fluid at room temperature (27°C). Each exchange comprised 1.5 litres of 1.36% dialysate and lasted 45 min. This resulted in rapid reduction of his core temperature and at the end of 6 h his core temperature was 37°C. At the end of 12 h his temperature had fallen to 36.5°C. Thereafter dialysate fluid warmed to 37°C was used (Figure 1).

During subsequent peritoneal dialysis his temperature remained between 37.5 and 38°C. Peritoneal dialysis was continued for 3 days following which the catheter was removed. In addition to peritoneal dialysis the patient had a total of 14 haemodialysis sessions. His renal function recovered and his general condition improved. He was extubated on day 12 of admission. Low grade pyrexia continued for 2 weeks and he has made an uneventful recovery.

Figure 1 Temperature chart on day 3 of admission. Antibiotics were commenced on day of admission and fever recurred after cessation of peritoneal dialysis.

Discussion

Hyperpyrexia occurs when the body core temperature is greater than 40°C. It can be seen in patients with severe infections, exertional and non-exertional heat stroke, and rarely as an idiosyncratic response to anaesthetic agents and neuroleptic drugs.¹ Parenchymal cell damage due to protein denaturation may occur in brain, skeletal muscle, liver and kidneys if the core temperature exceeds 41°C and is not reduced rapidly.

The appearance of hyperthermia 3 days after the initial presentation of our patient is unusual. Hyperthermia is regarded as an adverse prognostic feature of meningococcal septicaemia;² when it does occur, however, it is usually a presenting rather than late feature of the illness. Two possible mechanisms can be considered to explain the late onset of hyperthermia.

Firstly, in view of the evidence of consumptive coagulopathy the question of a brainstem haemorrhage was considered. The computed tomographic scan of the brain, which was reported as normal, may not have been sensitive enough to detect a small lesion in the hypothalamus. Such a lesion may have resulted in alteration of the hypothalamic thermoregulatory function.

Secondly, recent studies in animals have shown biphasic fever response following high dose intravenous administration of interleukin I beta which is a known endogenous pyrogen. It is postulated

that release of this cytokine from macrophages may cause a higher rise in temperature if this release occurs in the intraoptic anterior hypothalamic area of the brain.³ In a recent case report very high levels of Interleukin 6, another endogenous pyrogen were observed in the course of fatal meningococcal septicaemia.⁴ None of the drugs administered to the patient has been known to cause malignant hyperthermia and there was no evidence of viral or secondary bacterial infection on blood cultures and serology. The cause of the late appearance of hyperthermia in our patient is therefore not certain.

The conventional methods employed to achieve rapid body cooling include evaporation using cold water sponging, conduction using whole body immersion in cold water or covering with refrigerated rubber blankets, and convection using cool air.⁵ The rapidity of cooling rather than the exact method used is an important consideration in managing these patients. More invasive measures may be considered if the above mentioned methods fail to achieve rapid cooling. Iced gastric lavage, pleural lavage and cold peritoneal lavage have been described in experimental canine models.⁶ In a recent report the successful use of cold peritoneal lavage has been described in a patient with heat stroke.⁷

Thermodynamic studies were not undertaken in our patient and the reduction in temperature cannot be attributed solely to cold peritoneal lavage. The control of the infective process and improvement or reversal of the factor or factors causing hyperthermia may also have helped in reduction of temperature. In a controlled situation, and taking into account the subject's metabolic rate and energy expenditure, it may be possible by measuring heat transfer from the body to the dialysate to predict the temperature, volume and dwell time of dialysate which would be required to decrease body temperature at a desired rate.

In our patient's case peritoneal dialysis served the dual purpose of helping manage uraemia and life-threatening hyperthermia. Peritoneal dialysis has been indicated for rewarming the body core in patients suffering from hypothermia⁸ but the potential use of this method in treating hyperthermia is less well recognized. Acute peritoneal dialysis is a widely available procedure which should be considered in cases of hyperthermia refractory to treatment with conventional methods.

References

1. Guze, B.H. & Baxter, L.R. Neuroleptic malignant syndrome. *N Engl J Med* 1985, **313**: 163–166.
2. Gedde-Dahl, T.W., Bjark, P., Hoiby, E.A. *et al.* Severity of meningococcal disease: assessment by factors and scores and implications for patient management. *Rev Infect Dis* 1990, **12**: 973–992.
3. Murakami, N., Sakata, Y. & Watanabe, T. Central action sites of Interleukin 1 beta for inducing fever in rabbits. *J Physiol Lond* 1990, **428**: 299–312.
4. Romijn, J.A., Godfried, M.H., Wortel, C. *et al.* Hypoglycaemia, hormones and cytokines in fatal meningococcal septicaemia. *J Endocrinol Invest* 1990, **13**: 743–747.

5. Kielbock, A.J., Van Rensburg, J.P. & Franz, R.M. Body cooling as a method for reducing hyperthermia: an evaluation of techniques. *South Afr Med J* 1986, **69**: 378–380.
6. Bynnum, G., Patton, J., Bowers, W. *et al.* Peritoneal lavage cooling in an anaesthetised dog heat stroke model. *Aviat Space Environ Med* 1978, **49**: 779–784.
7. Horowitz, B.Z. The golden hour in heat stroke; use of iced peritoneal lavage. *Am J Emerg Med* 1989, **7**: 616–619.
8. Reuler, J.B. & Parker, R.A. Peritoneal dialysis in the management of hypothermia. *JAMA* 1978, **240**: 2289–2290.