Toxic shock syndrome complicated by laryngeal oedema

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Summary

A 50-year-old woman was admitted as an acute medical emergency and was diagnosed as having toxic shock syndrome. Thirty-six hours after admission she went into hepato-renal failure and had disseminated intravascular coagulation. She developed severe laryngeal oedema, a complication which has not been reported previously, and was intubated with great difficulty. She recovered from this, but died 5 weeks after the presentation of a pulmonary embolus.

KEY WORDS: toxic shock syndrome, laryngeal oedema.

Introduction

The term 'toxic shock syndrome' was first coined by Todd *et al.* (1978) who described 7 cases in children. The association between the toxic shock syndrome (TSS) and the use of tampons was first reported in 1980 in the United States by Scherock. Most of the patients with TSS have been seen in the United States, but there is now an increasing number of cases being reported in this country (Holt, 1980; Anonymous, 1980; de Saxe *et al.*, 1982).

This case demonstrated the typical features, but was also complicated by severe laryngeal oedema necessitating artificial ventilation.

Case report

A 50-year-old woman was admitted with a 24 hr history of fever, pleuritic chest pain, breathlessness, diarrhoea and vomiting. Four weeks before this, she was treated with antibiotics for a boil on her left hand. She menstruated regularly and always used tampons. Her last menstrual period commenced 6 days before the presentation. On examination she looked very ill and generalized erythroderma and conjunctival hyperaemia were prominent. Her tem-

perature was 38.4°C, pulse and blood pressure were unrecordable. Bilateral coarse crepitations were audible in her chest and the lower abdomen was tender. Vaginal examination revealed no tampon or discharge. Chest X-ray showed patchy shadowing at both bases. Arterial blood studies revealed a marked metabolic acidosis. A diagnosis of toxic shock syndrome was suspected and, after appropriate cultures were taken, she was treated with intravenous flucloxacillin, benzylpenicillin, steroids and sodium bicarbonate. After initial fluid replacement, the central venous pressure rose to +11 cm; a dopamine infusion was commenced and gradually increased over the next 6 hr to 7.5 µg/kg min.

Twelve hours after admission, her blood pressure rose to 90/60 mmHg, but she developed generalized oedema, the profuse watery diarrhoea continued and despite adequate fluid replacement, she became oliguric.

Thirty-six hours after admission, she was in established renal failure. Other investigations showed markedly disturbed liver function tests, total bilirubin was 103 µmol/litre, aspartate transaminase 280 IU/litre (normal < 18 IU/litre), lactate dehydrogenase 384 IU/litre (normal < 90 IU/litre), alkaline phosphatase 153 IU/litre (normal < 90 IU/litre) and the serum albumin was 27 g/litre. Serum creatine kinase and amylase levels were markedly elevated at 4750 IU/litre (normal < 70 IU/litre and 1500 IU/litre (normal < 300 IU/litre) respectively. Her platelet count, which was normal on admission, dropped to 64×10^9 /litre, and the peripheral blood film showed red cell fragments. The coagulation studies were markedly abnormal and consistent with the diagnosis of disseminated intravascular coagulation. Blood cultures grew no organisms, but *Staphylococcus aureus* type 29 (Group 1) was isolated from a high vaginal swab. *In vitro* studies by double gel diffusion confirmed the production of enterotoxin F by the staphylococcus isolated from the high vaginal

swab. Platelet concentrates, fresh frozen plasma and heparin were added to the treatment regime.

Five days after admission, she still remained very ill with generalized oedema, hypotension and acute renal failure. Conjunctival oedema was prominent preventing effective eye closure. While a shunt was being sited for haemodialysis, she had a haematemesis and went into circulatory and respiratory collapse. She was intubated with great difficulty because of marked laryngeal oedema. However, ventilation was effective in rapidly correcting her hypercapnia. Over the next 8 days, she was haemodialyzed intermittently and was ventilated throughout this period.

Twelve days after admission she developed generalized desquamation of the skin. However, her general condition, liver function tests, coagulation studies and urine output began to improve and she was extubated. Her course was complicated by a pseudomonas pneumonia. Her renal function slowly improved and at the peak of diuretic phase, she was passing 10 litres of urine per 24 hr.

On the thirty-third day, she was much improved and, for the first time, stood up without assistance. However, the next day she had a pulmonary embolus of sufficient severity to produce breathlessness, hypotension and poor urine output. Despite adequate anticoagulation she died 4 days later of congestive cardiac failure. At post mortem, the kidneys showed acute cortical necrosis, the stomach contained blood, but no bleeding site was found, the lungs showed pulmonary oedema and a small pulmonary embolus was present in one of the pulmonary arteries.

Discussion

This case provides a classic example of TSS with complications of acute renal failure, disseminated intravascular coagulation and respiratory failure (Scherock, 1980). It is important to recognize this illness for several reasons: (i) to ensure the use of penicillinase-resistant antibiotics; (ii) to search for the source of staphylococcal infection and (iii) to be

aware of the risk of respiratory obstruction from laryngeal oedema.

Laryngeal oedema has not been previously reported in this syndrome and represents a further life-threatening complication to a critically-ill patient. Mucosal oedema is a characteristic feature of TSS normally limited to conjunctivae, but in severe cases can obviously be extensive. In the patient, it produced respiratory failure, but was probably instrumental in preventing significant inhalation of blood during the haematemesis.

The characteristic features of this staphylococcal infection are thought to be due to circulating enterotoxin F. Bergdoll *et al.* (1981) have shown that patients suffering from TSS less readily develop antibodies to enterotoxin F, even after serious infection, than the general population. Infection or colonisation with these strains of staphylococcus appears to be essential for the development of TSS and an increasing proportion of cases not associated with menstruation are being reported with a variety of *S. aureus* infections (Reingold *et al.*, 1982). Future management of this condition may depend upon the development of a specific antitoxin.

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