


References

Fulminating streptococcal sepsicaemia

Sir,

Fulminating streptococcal sepsicaemia in previously healthy young people is rare.1–3 We report two recent cases at our hospital that serve to remind of the lethal properties of some Lancefield Group A streptococci.

A 35 year old woman, known to suffer from bulimia nervosa, and to abuse both laxatives and diuretics, was admitted with a 6 day history of headaches and fever, treated with simple angesis by her general practitioner. Clinically, she appeared alert and orientated, but somewhat dehydrated. There was generalized muscle weakness but no focal neurological deficit. Blood pressure was 80/40 mmHg. Blood investigations revealed potassium 3.3 mmol/l, sodium 131 mmol/l, urea 19.2 mmol/l, glucose 5.4 mmol/l, haemoglobin 10.6 g/dl, white cells 3.9 x 10^9/l and platelets 104 x 10^9/l. The initial impression was of deranged biochemistry and symptoms secondary to her bulimia and a recent viral illness.

Ten hours later, she collapsed with peripheral circulatory failure, unrecordable blood pressure, erratic respirations and a mottled skin rash. Further tests revealed disseminated intravascular coagulation and metabolic acidosis (arterial pH 7.2). Electrocardiogram showed sinus tachycardia and chest X-ray showed a small opacity in the right mid-zone. Septic shock with multi-organ involvement was now suspected. Despite intravenous broad-spectrum antibiotics, fluids, dobutamine and dopamine infusions cardio-respiratory arrest ensued from which resuscitation was unsuccessful. Autopsy revealed bilateral bronchopneumonia. Blood cultures grew Streptococcus pyogenes. Lancefield Group A, type T12 M12.

Case 2

A previously healthy 35 year old man presented as an emergency. The history obtained from his wife was of a 4 day flu-like illness with myalgia and sore throat, again treated with simple analgesia. Clinically he was now semi-conscious, hypotensive, cyanosed and had a striking mottled purpuric rash on his trunk and lower limbs. He was apyreal, the abdomen was soft and he had no menisngism. Blood tests revealed metabolic acidosis (arterial pH 6.9), urea 18.1 mmol/l, white cells 15 x 10^9/l (90% neutrophils), platelets 80 x 10^9/l. There was evidence of disseminated intravascular coagulation and anylase was moderately elevated at 416 IU/l (normal laboratory range up to 110 IU/l). The differential diagnosis was of septic shock or acute pancreatitis. He was ventilated and given intravenous piperacillin, metronidazole and gentamicin. Plasma volume expanders and inotropic support with dopamine and dobutamine infusions were also initiated. However, his cardiovascular status deteriorated culminating in a cardio-respiratory arrest with electromechanical dissociation. Resuscitation was unsuccessful. Blood cultures grew Streptococcus pyogenes, Lancefield Group A, type T1 M1 and autopsy revealed no source of infection.

Group A streptococcal infections account for only 2% of bacteriamaemias.1 Fulminant streptococcal sepsicaemia is rare and may occur in previously healthy adults without any recognized primary focus of infection.2 The clinical scenario of hypotension and multi-organ failure is reminiscent of the staphyloccocal toxic shock syndrome and the term ‘toxic strep syndrome’ has been suggested.4 The pathogenesis of this toxic shock remains inconclusive. Group A streptococci do not possess endotoxin.
but a number of pyrogenic exotoxins which are potential culprits.

Stevens et al. studied ten strains of Group A streptococci isolated from patients with toxic-shock-like syndrome and found that eight produced toxin A. It is noted for increased virulence and the rash of scarlet fever. Its protein sequences resemble that of the enterotoxin B of Staphylococcus aureus that is associated with toxic shock syndrome. However, the role of toxin A is incompletely understood. Gram-positive bacteraemia itself may lead to shock and fulminating infections have been reported with B-haemolytic streptococci of other groups that do not produce toxins.

Fulminating infection with severe hypotension may be secondary to Gram-negative endotoxaemia. However, one should consider toxin-producing Group A streptococci as alternative causative agents especially in the presence of skin rashes.

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References


Hospital patients – are they active enough?

Sir,

Prolonged bed rest is anatomically, physiologically, and psychologically unsound. It can lead to local or systemic complications. In 1954 Arnott wrote: excess prescribing of rest, and excess prescribing of drugs, are millstones around the health service and the nation – extravagance we cannot afford. This is an important point on which medical and nursing staff perhaps do not give high enough priority, and this may account for the high prevalence (5.7%) of pressure sores in the medical unit in the Leeds western health authority compared to 0.9% in the community.

The activities of 335 patients (in medical/gериatric wards) including bed rest, sitting, walking, or receiving therapy, were recorded between 11.00 and 13.30 a.m. when patients are unlikely to be restricted to beds because of ward rounds or investigations. A total of 137 patients (41%) were resting in bed, eight of whom were noticed asleap, 29 (9%) patients were out of the ward, 169 (50.4%) were sitting by the bed, 18 (5%) patients were receiving various forms of therapy including intravenous drip, oxygen, or nebulization, but interestingly, fewer (7/22 (14%)) patients on a geriatric ward were in bed in comparison to 130/268 (46%) on medical wards.

This survey demonstrates that a significant number of patients with acute medical problems stay in bed for long hours. The fact that only 5% of patients were receiving oxygen, nebulization, or intravenous drips cannot justify why so many patients were resting in bed until 11.30 a.m. Moreover, maintaining patients’ mobility, particularly the elderly, promotes a sense of well-being and plays an important role in their continuing physical and mental health.

The result of this limited survey highlights the need for further investigations of hospital patients’ activities in relation to age, diagnosis, complications, discharge, and re-admission. It is important therefore to have a strategy on patients’ mobilization which is likely to speed up recovery and early discharge to the community. There is also a need to know more about the types of exercise and activities that may be suitable for hospital patients.

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References

3. Lindsay, M. Equipment Survey and Pressure Sore Prevalence Study. Leeds Western Health Authority, 1989.