addressed, although King suggested that anatomical asymmetry in the neck, particularly of the large vessels, may be a contributory factor.⁴

About 11% of the population is left handed, and 10% of our patients were left handed. Left handed people therefore seem as likely to develop pharyngeal pouches as right handed people. Symptoms are more likely than signs to occur predominantly on one side of the neck; the close correlation between our patients' signs and symptoms and the available radiographs suggests that symptoms are an excellent indication of the site of the pouch anatomically. Our results also show a striking association between handedness and the occurrence of pharyngeal pouches on the opposite side. Limb and girdle musculature is greater on the dominant side,⁵ and we suggest that the enlarging pouch follows a path of least resistance to the contralateral side.

We conclude that symptoms that are predominantly on one side of the neck accurately predict lateral extension of a pharyngeal pouch, and that the side on which the pouch occurs is determined by the handedness of the patient.

We are indebted to the surgeons of the Royal National Throat, Nose and Ear Hospital for permission to study patients under their care, and to Mr A E Thompson and Mr H R Grant for allowing us to study their patients at St Thomas's and University College hospitals respectively.

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Myocardial depression in streptococcal cellulitis

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Abstract

A previously healthy woman developed streptococcal cellulitis of the leg after falling and lacerating her knee. On admission her blood pressure was unrecordable and her respiratory rate greater than 60 beats/minute. Mechanical ventilation was necessary, and plasma volume expansion was started, with little effect. Infusion of dopamine restored the blood pressure to 150/90 mm Hg, but interrupting the infusion for as little as 30 seconds resulted in profound hypotension. Insertion of a second central venous catheter allowed the dopamine to be given continuously, and the infusion was stopped successfully four days after her admission. She eventually made a complete recovery.

This case highlights the need for intensive supportive treatment in many streptococcal infections and, in particular, the need for inotropic support.

Introduction

Life threatening and fatal infections with *Streptococcus pyogenes* (β haemolytic streptococcus, Lancefield's group A) continue to occur despite the use of antibiotics.^{1,2} We describe a case of streptococcal cellulitis affecting the leg in a previously healthy patient.

Case report

A 32 year old woman in her third pregnancy was admitted as an emergency at 32 weeks' gestation with colicky lower abdominal pain, thought to be due to labour. On admission she was moribund and unable to give a history because of severe dyspnoea. On examination she had central and peripheral cyanosis; the blood pressure was unrecordable, and the respiratory rate was greater than 60 beats/min. The left leg was swollen to the thigh. Because of suspected fetal distress an emergency caesarean section was performed. A dead fetus was delivered. She was transferred to the intensive care unit, and deep venous thrombosis and pulmonary embolism were provisionally diagnosed.

Ventilation with 70% oxygen was started, and blood gas analysis showed pH 7.24, oxygen tension 28.3 kPa (212 mm Hg), and carbon dioxide tension 4 kPa (30 mm Hg). Ventilation was continued and a Swan-Ganz catheter inserted. The mean pulmonary artery pressure was 15 mm Hg and the pulmonary capillary wedge pressure 2 mm Hg. A chest x ray film showed soft shadowing throughout both lung fields. Plasma volume expansion was started and the diagnosis reviewed. A small laceration was noted overlying the left patella. Cellulitis was suspected, and appropriate bacteriological specimens were sent. Benzylpenicillin 4 MU and flucloxacillin 1 g were administered intravenously.

After she had received 3.5 l of intravenous fluid the pulmonary capillary wedge pressure was 18 mm Hg and the blood pressure 60 mm Hg systolic; she remained anuric. A dopamine infusion was started: 13 μ g/kg/min produced a blood pressure of 150/90 mm Hg with a pulmonary capillary wedge pressure of 14 mm Hg and a pulmonary artery pressure of 32 mm Hg, indicating that the circulatory failure was due to vasodilatation or myocardial depression or a combination of both.

She began to pass urine. Five hours after her admission to the intensive care unit her husband was available to provide a history. Thirty six hours previously she had fallen and lacerated her left knee while gardening, and subsequently the whole leg had become swollen and painful. Culture of fluid aspirated from beneath the lacerated area yielded a growth of haemolytic streptococcus (Lancefield's group A). Electrocardiography showed a normal tracing, apart from the rhythm, throughout her stay in hospital.

Mechanical ventilation had to be continued for 24 hours. An alarming feature was that whenever the infusion of dopamine was stopped for as little as 30 seconds—for instance, for injection of antibiotics—she developed circulatory collapse with a pulse rate of 15-20 beats/min

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and an unrecordable blood pressure. A second central venous catheter was therefore inserted exclusively for infusion of dopamine. Four days after her admission the infusion was successfully stopped. She was eventually discharged from hospital having made a complete recovery.

Discussion

This patient had streptococcal cellulitis that caused circulatory and respiratory failure masquerading as venous thrombosis and pulmonary embolism. Streptococcal infection may be rapidly progressive and fatal despite treatment with antibiotics, and seven out of eight patients reported on recently died.^{1,2} In our patient antibiotics would not have influenced the outcome without early intensive supportive measures, including full haemodynamic monitoring. In particular, we were impressed by the extreme dependence of her condition on dopamine.

The existence of a factor that acts as a myocardial depressant

and contributes to shock in cases of severe sepsis has previously been postulated.^{3,4} Although thermodilution studies to measure cardiac output were not performed in this patient, we believe that the rapidity of onset and degree of hypotension together with the bradycardia that occurred when the dopamine infusion was interrupted strongly suggest the presence of such a humoral factor in our case.

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Effect of prenalterol on orthostatic hypotension in the Shy-Drager syndrome

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Abstract

Treatment of idiopathic orthostatic hypotension is often unsatisfactory. A patient with the Shy-Drager syndrome, in which the most important symptom is orthostatic hypotension, was treated with prenalterol, initially 30 mg six times daily. The dosage was reduced to 30 mg four times daily because of the development of complex ventricular premature beats. Orthostatic symptoms were reduced and standing blood pressure increased. Fludrocortisone 0.5 mg a day was added to treatment with further improvement. This clinical effect was maintained throughout 12 months of follow up, during which the treatment was continued unchanged.

Prenalterol was effective in reducing orthostatic symptoms in this patient. Further studies in patients with a similar haemodynamic pattern are indicated.

Introduction

Idiopathic orthostatic hypotension may occur either as an isolated disorder or in association with multiple defects of the central nervous system when it is known as the Shy-Drager syndrome. Treatment of this disabling condition is often unsatisfactory.¹ We report the effects of prenalterol, a selective β_1 adrenoceptor agonist,² in a patient with the Shy-Drager syndrome.

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Case report

A 50 year old man was admitted to hospital because of an attack of syncope. He had suffered from dizziness, fainting, and stiffness of the legs for several months. For two years he had complained of urinary frequency, nocturia, urge incontinence, loss of potency, diarrhoea, faecal incontinence, and diminished sweating. On admission his supine blood pressure was 115/69 mm Hg and heart rate 64 beats/min. When he stood up his blood pressure immediately fell to 66/24 mm Hg and his heart rate increased to 92 beats/minute. Despite a partial recovery of blood pressure after one minute (94/52 mm Hg) he could not walk because of dizziness and blurring of vision.

A thorough investigation excluded secondary forms of orthostatic hypotension. The Shy-Drager syndrome was diagnosed because of the association of features of autonomic failure with clinical signs that the central nervous system was affected (pyramidal, cerebellar, vestibular, and sensory signs); an abnormal blood pressure response to the Valsalva manoeuvre; a normal baseline plasma noradrenaline concentration (0.384 $\mu\text{g/l}$) with an inadequate increase on standing (+46%); and mild supersensitivity to noradrenaline and tyramine. According to Bannister *et al* supersensitivity to noradrenaline is defined as a rise in systolic blood pressure of >15 mm Hg in response to an infusion of noradrenaline <4 $\mu\text{g/minute}$.³ An equal response to a tyramine infusion of <300 $\mu\text{g/minute}$ indicates supersensitivity to tyramine. In our patient systolic blood pressure increased by 16 mm Hg during infusion of 1 μg noradrenaline/minute and by 19 mm Hg during infusion of 250 μg tyramine/minute. Brachial artery catheterisation (Vygon, 115.09) and flow directed right heart catheterisation with a Swan-Ganz catheter were performed. Cardiac output was calculated according to the Fick principle (cardiac output = oxygen consumption: arteriovenous oxygen difference). Systemic vascular resistance was calculated from the mean brachial intra-arterial pressure, obtained by electrical damping, and cardiac output. Stroke volume was calculated from cardiac output and heart rate. Heart rate was recorded from the electrocardiogram. Haemodynamic data obtained before treatment indicated that orthostatic hypotension was related to a dramatic fall in stroke volume with inadequate cardiac output despite the increase in heart rate (table).

Treatment with a 150 mmol(mEq) sodium diet and fludrocortisone in increasing dosage up to 1.0 mg/day resulted in symptomatic im-