Group B streptococcal meningitis in an adult

Sir,

Group B streptococcal infection causing acute sepsis and meningitis is rare and accounts for less than 1% of cases. Group B streptococcus is a very invasive organism which seldom causes acute inflammation at the site of entry to the body and the approach is usually unheralded.

A 45-year-old woman was admitted for varicose vein surgery. She had a history of varicose eczema and left deep vein thrombosis. She was allergic to penicillin. Spinal anaesthesia was performed aseptically and lignocaine was injected through a bacterial filter. Stripping of varicose veins was performed satisfactorily. Prophylactic subcutaneous heparin was used.

Six hours post-operatively she developed a sore throat and 18 hours later she became confused, restless, had a temperature of 38°C, neck stiffness but no focal neurological signs. A clinical diagnosis of meningitis was made and she was given intravenous ceftriaxone and chloramphenicol after blood cultures had been taken. Lumbar puncture was performed aseptically under sedation. Cloudy cerebrospinal fluid (CSF) was obtained with 2000 white blood cells (95% polymorphs and 0.007 red blood cells per ml, 6.1 g/l protein, glucose 4.0 mmol/l; Gram-positive cocci were seen. Venous blood glucose was 10.1 mmol/l and white blood count 13.2 x 10⁹/g. Urea and electrolytes, liver function tests and arterial blood gases were normal. She was commenced on vancomycin and ceftriaxone and transferred to the intensive care unit.

At this point she was protecting her own airway and was haemodynamically stable; an erythematous rash developed on her right leg. A retained tampon was removed. She remained restless and was sedated with a propofol infusion. Computed tomography (CT) of the head showed slight cerebral oedema. On day 2 she was making spontaneous purposeful movements with her left side but only localising pain with her right side. She remained restless. Her respiration was extensor. Repeat head scan showed no focal lesion. She was commenced on dexamethasone, 4 mg intravenously qid, for suspected cortical thrombophlebitis.

The blood culture on day 1 group B streptococci were isolated from the two sets of blood cultures, high vaginal swab and the CSF. On day four she had three grand mal fits and was commenced on phenytoin. Magnetic resonance imaging (MRI) showed a small lacunar infarct. Her antibiotic regime was changed from vancomycin to clindamycin.

Over the following few days she improved with resolution of her confusion and hemiparesis. On day 15 she developed left-sided pleuritic chest pain and shortness of breath, strongly suggestive of pulmonary embolus. This was confirmed by a perfusion scan which showed a large perfusion defect at the left base. Treatment was commenced with heparin and warfarin. On day 23 she was discharged home with no evidence of focal neurological deficit. Intravenous antibiotics had been continued for a total of 14 days; dexamethasone was reduced and stopped over this period and she remained on phenytoin and warfarin on discharge.

The reported mortality rate in adults with group B streptococcal meningitis is 27%, 45% in patients with a comorbid condition, and 0% in patients without underlying disease; 30% of those with bacteriaemia die. The presentation is usually similar to that of other bacterial meningitides. It is usually very sensitive to penicillin but has a relatively high mean inhibitory concentration. More than 50% of reported cases have a comorbid condition such as diabetes, or malignancy. Although infections associated with the female reproductive system form a considerable proportion of the cases in young adults, overall, a greater number of males and patients over 45 years are affected.

Meningitis after lumbar puncture and spinal anaesthesia is very rare. In two reported cases1,2 no organisms were grown in the CSF and the patients made a full recovery. The postulated causes are shown in box 1.

It has been suggested that lumbar puncture creates a site of low resistance to infection across the blood – brain barrier. In this case we postulate that the retained tampon was responsible for the entry of group B streptococci into the bloodstream from the female reproductive system. The spread of infection into the CSF was then facilitated by the spinal anaesthetic having created a site of low resistance. Although she developed a skin rash the skin swabs were negative, making this very unlikely to be the source of infection.

AG HATFIELD
AH AL-HILLAWI
Department of Medicine,
Stoke Mandeville Hospital,
Aylesbury, Bucks HP21 8AL, UK

Box 1

Causes of meningitis after lumbar puncture or spinal anaesthesia

• aseptic meningitis from disinfectants and detergents
• introduction of bacteria from the bloodstream of a septicemic patient
• introduction of foreign material due to corticosteroids
• use of certain local anaesthetic solutions
• incidental viral infection

Box 2

Causes of meningitis in adults with group B streptococcal meningitis

• in patients with a comorbid condition, and 0% in patients without underlying disease; 30% of those with bacteriaemia die.
• The presentation is usually similar to that of other bacterial meningitides. It is usually very sensitive to penicillin but has a relatively high mean inhibitory concentration.
• More than 50% of reported cases have a comorbid condition such as diabetes, or malignancy.
• Although infections associated with the female reproductive system form a considerable proportion of the cases in young adults, overall, a greater number of males and patients over 45 years are affected.

Genetic predisposition of ischaemic heart disease

Sir,

Wat et al reported a 29-year-old man who developed acute myocardial infarction.3 Another recent publication reported a 28-year-old Olympic champion, without any conventional risk factors, for the absent of a family history of acute myocardial infarction associated with coronary heart disease.2 Wat et al reported that their patient had no coronary heart disease risk factors other than hypercholesterolaemia.1 Patients with hypercholesterolaemia of 7.1 mmol/l are commonly observed in out-patient clinics. However, it is quite rare that they develop such severe coronary heart disease, or fatal acute myocardial infarction, at this age. Genetic predisposition for the development of coronary heart disease is largely unknown, but recent observations have revealed that some genetic variants are associated with coronary heart disease in Caucasians.2-4 We propose that there is a marked ethnic difference in a coronary heart disease susceptibility gene variant.5 Whether such genetic alterations are associated with coronary heart disease in Chinese is currently unknown. However, an involvement of some genetic factors other than hypercholesterolaemia in the pathogenesis of his acute myocardial infarction is probable in Wat et al’s patient judging from the absence of a family history of acute myocardial infarction, he may have inherited genetic abnormalities from each of his parents. Compound heterozygosity of more than two mutations or homozgyosity of a genetic mutation may have led to his acute myocardial infarction of juvenile onset.

It is obvious that conventional risk factors for coronary heart disease, such as hypercholesterolaemia should be controlled, but investigation of genetic predisposition is likely to contribute to decreasing fatal coronary heart events. Other than conventional risk factors, genetic analysis of patients for coronary heart disease susceptibility genes will be important for the prevention of fatal coronary heart events.

Learning points

• rare in adults but high mortality
• often comorbid conditions
• high index of suspicion
• look for upper respiratory tract symptoms and ensure tampons are removed pre-operatively
• strict asepsis for spinal anaesthesia
• treat early with intravenous antibiotics and fluids on the intensive care unit; anticoagulants may be required

unlikely that a properly informed patient or his delegate would give his consent to participate in such a trial. For these reasons, two recent prospective randomised controlled trials comparing HBO with NBO have excluded patients with severe poisoning (those with loss of consciousness or myocardial instabili-
ty) and concentrated on moderate intoxica-
tion. Both these studies have confirmed the
utility of HBO, compared to NBO, in accel-
erating recovery and preventing delayed neu-
rological sequelae.6

However, does it make sense in clinical practice to treat moderately intoxicated cases with HBO and not severe cases for the simple reason that a trial cannot be performed as treatment is considered potentially life-saving? Would such a position be tenable in a court of law? Is it logical to treat moderately intoxicated
women, except when they carry a foetus very vulnerable to hypoxia, when it has been shown that HBO is safe in pregnancy? As regards the transport of critically ill patients, it is accepted that supportive care must never be compromised in transport and that the logistics of every case must be considered and carefully evalu-
ated in a local context. However I feel strongly that, in line with guidelines in the literature, comatose patients, or patients with prolonged loss of consciousness should, whenever possible, be offered HBO. HBO treatment in patients presenting late is still debatable. Whether the cost of an HBO unit or the transport expenses are justified or not by the benefit of therapy depends on the logistics and in the ranking priorities of different healthcare setups.

RD HARDERN
AJ GRAY

Department of Medicine,
St Luke’s Hospital,
Glandomangia, Malta

2 Thom SR, Taber RL, Mignedurens II, Clark JM, Hardy KR, Fisher AB. Delayed neuropsychologic-
4 Nortool DM, Kirkpatrick JN. Treatment of acute carbon monoxide poisoning with hyperbaric

Heart failure in the elderly

Sir,

Although I enjoyed reading the well-re-
searched review of the management of heart failure in the elderly,1 I disagree with the emphatic restatement of the conventional view that adjunctive treatment with spirono-
lactone is absolutely contraindicated in pa-
tients already co-prescribed angiotensin-
converting enzyme (ACE) inhibitors and
loop diuretics. The reality is that amongst patients already co-prescribed ACE-inhi-
bitors and loop diuretics, there will always be a few with a refractory, and potentially life-
threatening fluid overload, in some instances, to hyperaldosteronism,2 for which correc-
tive treatment could either be resection of an adenocortical adenoma or co-prescrip-
tion of spironolactone and ACE-inhibitors.2

In my own register, dating back to 1984, comprising 349 patients co-prescribed ACE-in-
hibitors and loop diuretics for heart failure, three women, now aged 81, 81 and 79, respective-
tly, were converting hypertension and hypokalaemia, the latter refractory to ACE-
inhibitors. In the first patient, with a nadir potassium of 2.7 mmol/l, following co-

prescription of spironolactone 25 mg/day, frusemide 40–80 mg/day and enalapril 10 mg/day followed by lisinopril 20 mg/day, the plasma potassium was maintained in the range 3.9–4.4 mmol/l during the last six months of a 30-month period of ‘triple’ therapy. Prior to commencement of ‘triple’ therapy, her 24-hour urinary aldosterone was 7 pmol (reference range 10–50) in 1050 ml urine, and her 09.00 h serum cortisol was 516 nmol/l.

In the second patient, the co-prescription of spironolactone was precipitated by a fall in serum potassium to 2.2 mmol/l whilst receiving enalapril 20 mg/day and frusemide 80 mg/
day. Her 24-hour urinary aldosterone output was 25 pmol (in 1620 ml urine), but neither receiving contraceptive hormones nor serum cortisol were requested during this (and a subsequent) admission. The second reason was absolute hypokalaemia, which started 11 years previously, rendered the diagnosis of Cushing’s syndrome unlikely in the absence of the development of central stigmata over that period. Ultrasonography had also not identified any adrenal abnormality. This patient was subsequently prescribed spironolactone 25 mg/day, in addition to frusemide 80 mg/day, enalapril 20 mg/day, whilst serum potassium remained at 16.5 mmol/l, creatinine of 139 mmol/l and body weight of 47 kg. Unfortunately, her 24-hour urinary aldosterone and cortisol levels were quantified when she was already taking spironolactone 25 mg/day, frusemide 120 mg/day, ramipril 10 mg/day, and laci-
pine 4 mg/day, yielding values of 2.0 mmol and 71 nmol, respectively, in 800 ml urine. Computed tomography showed that she had right-sided hydrocephalus, but no adrenal adenoma. During the subsequent three months, spironolactone was progressively increased to 200 mg/day, frusemide reduced to 80 mg/day, whilst ramipril was main-
tained at 10 mg/day and lacidipine increased to 6 mg/day. Consequently, her blood pressure fell to 200/90 mmHg, but her plasma potassium remained at 2.9 mmol/l, with urea 17.1 mmol/l and creatinine 130 mmol/l. Due to the subse-
quent development of a pruritic maculo-
papular rash, losartan 50 mg bid was substi-
tuted for ramipril, and she is now undergoing titration of red spironolactone dose, due to a 75% reduction in frusemide requirements (based on transient develop-
ment of reversible prerenal uraemia).

OMP JOLLOI
Tameside General Hospital,
Ashon-under-Lyne, Lancashire OPE 9RW, UK

2 Geist M, Dorian P, Davies T, et al. Hyperaldoso-

This letter was shown to the author, who responded as follows:

Sir,

I make no apology for stating that the use of spironolactone (and other potassium-sparing diuretics), in conjunction with ACE-inhibitors should be discouraged in the elderly. This combination in older people
Genetic predisposition of ischaemic heart disease.

M. Odawara, A. Matsunuma and K. Yamashita

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