been using an 'abaya' (black veil) and had very little sunlight exposure. Physical examination was normal except for severe proximal muscle weakness and tenderness over the pelvic girdle and thoracic cage. Biochemical evaluation revealed a mild hyperchloremic metabolic acidosis (pH 7.3, Cl 107 mmol/l), severe hypophosphataemia (0.6 mmol/l), markedly elevated alkaline phosphatase (655 IU/l) and normal serum calcium (2.4 mmol/l). X-rays of the pelvis showed multiple pseudo- fractures, X-ray skull/lateral view showed a salt and pepper appearance, and hand X-rays showed subperiosteal bone resorption. A urine amino-acid study showed marked generalized aminoaciduria.

Serum parathyroid hormone (PTH) was 237 pg/ml (normal range: 10–55), 25 (OH) vitamin D3 16 nmol/l (25–104), and 1,25 (OH)2D3 42 pmol/l (16–42). A Thallium-technetium subtraction scan showed a left inferior parathyroid adenoma and an ultrasound of the neck revealed that the adenoma was about 2.5 cm in diameter. A diagnosis of tertiary hyperparathyroidism due to long-standing nutritional osteomalacia was made. Since therapy with long-acting vitamin D3 in this setting carries a significant risk of hypercalcemia, the patient was treated with 1-alpha (OH) vitamin D3, calcium carbonate and neutral phosphate. Five months later, the patient showed a 50% improvement in bone pains and proximal weakness, the alkaline phosphatase decreased to 406 IU/l and PTH decreased to 105 pg/ml, but her serum calcium rose to 2.8 mmol/l and serum phosphate was still low at 0.7 mmol/l. She underwent a neck exploration and a 2.5 cm x 3 cm left inferior parathyroid adenoma was removed. Postoperatively serum calcium declined to 1.8 mmol/l and phosphate increased to 0.98 mmol/l. The dose of 1-alpha (OH) vitamin D3 was increased to 2 μg/day and 4 months after surgery, the patient is asymptomatic with normal serum calcium and phosphate values, and undetectable serum PTH.

Tertiary hyperparathyroidism is a rare complication of nutritional osteomalacia and may be due to an increase in parathyroid cell mass beyond a critical level as in the experimental model of Gittes and Radde. When this complication occurs, it may be prudent to use short-acting vitamin D metabolites, such as 1-alpha (OH) vitamin D3 or 1,25 (OH)2D3 rather than native vitamin D3 in order to avert protracted hypercalcemia.

M.S. Seshadri
Mohammed A.F. Qurttom
R. Sivanandan
Shihab-Al-Mohannadi
Samiaman
Mubarak Al-Kabeer Hospital,
Department of Medicine,
Faculty of Medicine, PO Box 24923, Safat, Kuwait 13110.

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Purpura fulminans following a dog bite
Sir,
Prevention of infection post-splenectomy has focused on the risks of infection due to the encapsulated organisms, Neisseria meningitidis, S. pneumoniae and H. influenzae. Currently, advice includes recommending lifelong penicillin prophylaxis, appropriate vaccinations, and the need to seek urgent medical attention, at the first signs of respiratory infections or other febrile conditions. There are, however, other less common infections that may be equally devastating and to which splenectomized patients are peculiarly susceptible.

I would like to report a case of a fulminating septicaemia following a trivial dog bite in an asplenic patient. He had undergone a splenectomy for Hodgkin's disease 10 years previously, and had never taken penicillin prophylaxis.

A 37 year old engineer presented with shock, purpura fulminans and disseminated intravascular coagulation, 3 days after a playful nip on the cheek by his pet dog. A striking malar purpura and incipient gangrene of the tip of his nose was noted. He was treated initially with benzyl penicillin, cefotaxime, fluocxacillin and metronidazole, and was transferred to our intensive care unit for haemofiltration and ventilation. Gram-negative rods were seen within the neutrophils on the peripheral blood film, and a provisional diagnosis of Capnocytophaga canimorsus (Dysgonic Fermenter type 2) septicaemia was made. Ciprofloxacin was substituted for the cefotaxime and fluocxacillin, the first reported use of ciprofloxacin in this condition.

There followed a stormy clinical course, complicated by multisystem organ failure. Epileptiform seizures due to accumulation of penicillin resulted in the continuation of ciprofloxacin alone. Blood cultures yielded C. canimorsus 11 days after admission.

Despite no microbiological confirmation of meningitis at any time, there was a relentless and unexplained decrease in cerebral function, and after cessation of all sedation for 10 days he still remained comatose. Thirty-five days after admission, the consensus of neurological opinion was that at best a 'severe, near vegetative brain dysfunction' was likely. Withdrawal of all active measures was considered, but not implemented.

On day 37, a slight movement of his hand was noted, and over the next 48 hours he unexpectedly regained consciousness. He was finally discharged home cerebrally intact 2 months after admission. He was given Pneumovax, and advised to take prophylactic antibiotics for life.

Clinicians and patients should be aware of the potential severity of seemingly trivial animal bites in the asplenic patient, particularly if not on antimicrobial prophylaxis. Of the 60 C. canimorsus septicaemias reported to date, 86% followed animal bites or contact. One third involved asplenic patients, none of whom were taking prophylactic antibiotics on admission. The overall mortality in asplenic patients was 41%.3

Capnocytophaga canimorsus (DF2) is an oral commensal of many domestic animals, present in 24% of dogs.4 The organism is sensitive to penicillin, ciprofloxacin and erythromycin, but is resistant to gentamicin – an important point, since the antibiotic treatment of Gram-negative septic shock often includes gentamicin. In order
to cover all the potential pathogens following severe animal bites, co-amoxyclav as a single agent would be effective. It is important to ensure that all patients post-splenectomy are aware of their increased susceptibility to infection following animal bites. Finally, the laboratory should be informed of any history of animal bites preceding any severe infection, since prolonged culture may be necessary to isolate the organism.

M.S. Morgan

Department of Medical Microbiology, Pathology Laboratory, Church Lane, Heavitree, Exeter EX5 2AD, UK.

References


Coma in Wernicke’s encephalopathy

Sir,

We recently managed a patient who presented in coma and developed signs suggestive of acute brainstem compression; after resuscitation the diagnosis of Wernicke’s encephalopathy (WE) was established.

A 46 year old unconscious man was brought into the emergency department and within minutes had a witnessed respiratory arrest. He underwent prompt endotracheal intubation and was ventilated with an ‘ambu-bag’ under no sedation. Shortly after this he became asystolic. Atropine and adrenaline were administered by vein and sinus rhythm at a rate of 70/minute was quickly restored. His left pupil was 8 mm in diameter and the right pupil 3 mm. Doll’s eye movements were absent, but there was no papilloedema or retinal haemorrhage. We suspected that he had brainstem compression due to an intracranial mass lesion and he immediately underwent a computed tomographic brain scan. This showed only mild cerebral atrophy and a 10 × 15 mm hypodense area in the right temporal lobe. He was transferred to a mechanical ventilator. He was cachectic, tattooed, and had unkempt hair and finger nails. The rectal temperature was 29°C, pulse rate 70/minute, blood pressure 55/40 mmHg. Both pupils were now 8 mm in diameter and unreactive to light, doll’s eye movements remained absent and there was no caloric response to 50 ml of ice-cold water instilled into each ear.

The limbs were flaccid, areflexic and there was no response to pain. The blood glucose was normal and a serum poison screen was negative.

He was warmed and a multivitamin preparation containing 50 mg of thiamine hydrochloride was administered by vein. Over the next 2 hours his temperature rose to 32°C and the blood pressure rose to 140/70 mmHg. His pupils constricted to 4 mm in diameter and became reactive to light. Doll’s eye movements were restored and he started to flex his limbs in response to pain. The tendon reflexes were now present and symmetrical except the ankle jerks which were absent. Further parenteral thiamine was administered. Over the next 6 hours he regained spontaneous eye movements and required sedation to maintain adequate ventilation. The next day he was extubated. His brother was able to confirm that he drank alcohol to excess and had a poor diet. Over the next month he remained confused and disorientated, and at discharge he had residual nystagmus and ataxic gait.

Coma is an unusual and life-threatening manifestation of WE, with a mortality of over 50%.1,2 Our patient’s dramatic presentation with false localizing diencephalic signs mislead us to believe that he had acute brainstem compression. Wallis et al.3 have reported four patients with coma due to WE who presented with similar signs to our patient including hypothermia, hypotension and absent caloric responses. Pupillary reflexes were present in their patients, but sluggish light reactions and anisocoria have been noted in up to a third of larger series.1,2 Retinal haemorrhages and papilloedema have occasionally been observed in WE1,4 and may cause further confusion in establishing the correct diagnosis. Physicians should maintain a high degree of suspicion that WE is the underlying diagnosis in any patient presenting with unexplained neurological signs and impaired consciousness, as prompt administration of parenteral thiamine (50–100 mg) is a potentially life-saving treatment in this situation.

S.H.S. Pearce
C.J. Rees
Department of Medicine, North Tees General Hospital, Stockton on Tees, Cleveland TS19 8PE, UK.

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M. S. Morgan

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