to cover all the potential pathogens following severe animal bites, co-amoxiclav 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.

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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/min 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%.

Our patient's dramatic presentation with false localizing diencephalic signs mislead us to believe that he had acute brainstem compression. Wallis et al. 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. Retinal haemorrhages and papilloedema have occasionally been observed in WE 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 lifesaving treatment in this situation.

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