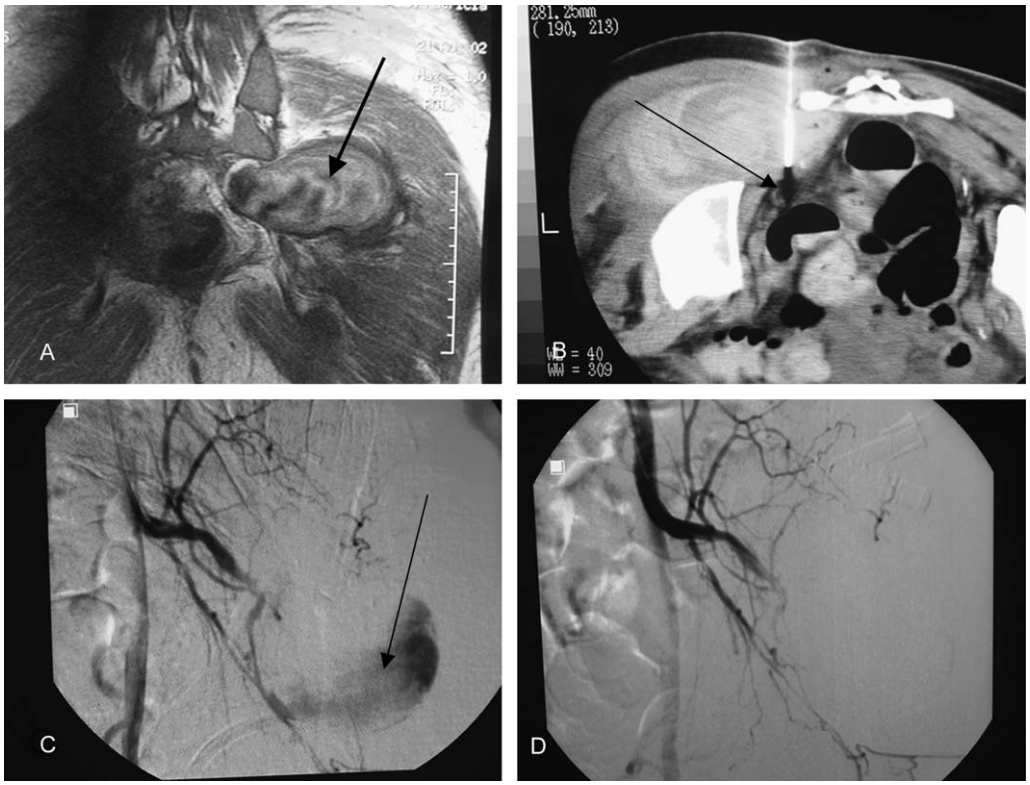

Fig. 1. (A) Coronal STIR MR image showing haematoma in the left hemipelvis (arrow). (B) Axial prone computed tomography scan with needle placement (arrow) within the haematoma prior to attempted biopsy. (C) Angiogram showing filling of the pseudoaneurysm (arrow). (D) Postembolization angiogram showing no filling of the pseudoaneurysm.

given the potential severity of the symptoms and the relief that can be afforded by percutaneous embolization.

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doi:10.1016/j.jocn.2005.03.027

Ophthalmoplegia in tiger snake envenomation

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Received 22 March 2005; accepted 8 April 2005

Abstract

Herein, we present the case of a 67-year-old grazier who was bitten by a tiger snake and developed coagulopathy and respiratory distress. The patient required intubation and ventilation in intensive care. There was delayed detection of snake envenomation and

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administration of antivenom. On extubation several days later, gross external ocular paresis was noted. Clinical testing indicated that the ocular pathology was secondary to neurotoxin-mediated presynaptic blockade. The paresis was partially resolved by the time of discharge one week later. The present case report discusses the possible mechanisms for the delayed development of ophthalmoplegia.

Keywords: Envenomation; Gaze paresis; Ophthalmoplegia; Presynaptic blockade; Snake bite; Tiger snake

1. Case report

A 67-year-old man was referred for a neurological opinion from a peripheral country hospital. He had been previously active and independent, with a past history of hypercholesterolaemia, hypertension and diabetes mellitus that was diet-controlled.

The patient was found by his wife in a paddock, face down and unconscious, after failing to return home. On rousing a short time later, he had retrograde amnesia; his only recollection was of working next to an irrigation channel. He had been loosely clothed in shorts and uncovered shoes.

At presentation to the local hospital emergency department, the patient was noted to have torrential epistaxis and significant mid-facial injuries. In addition, he was hypoxic with poor baseline saturations. Routine bloods revealed a coagulopathy with an INR > 10, activated partial thromboplastin time > 200 and D-Dimer $> 13\,000$. A creatinine kinase level of 12 000 indicated severe myolysis. The patient received four units of fresh frozen plasma and was intubated in the intensive care unit for respiratory support.

No focal neurology was evident at this stage. On review 12 h later, two discrete puncture marks on the right thigh were noted. An emergency venom detection kit revealed tiger snake toxin to be present. Seven vials of antivenom were administered, which totalled 21 000 neutralising units.

On the third day of admission, the patient was extubated successfully, but complained of binocular diplopia and blurred vision. He was transferred to the Royal Melbourne Hospital for further neurological evaluation.

Clinical examination revealed intact visual fields and visual acuity of 6/6 bilaterally. Fundoscopy was normal. There were hypometric saccades and broken smooth pursuit. There was a significant limitation of horizontal and vertical gaze, more pronounced with left and downwards gaze. The left gaze was limited to 5°, the upward gaze was limited to 15° and the downward gaze was limited to 10° (Fig. 1). There was no nystagmus and upper and lower limb examinations were normal. Magnetic resonance imaging (T_1, T_2, DWI) was within normal limits.

The admission was complicated by persistent hypoxia, determined on nuclear scanning to be secondary to multiple pulmonary embolism. The patient was anticoagulated with warfarin.

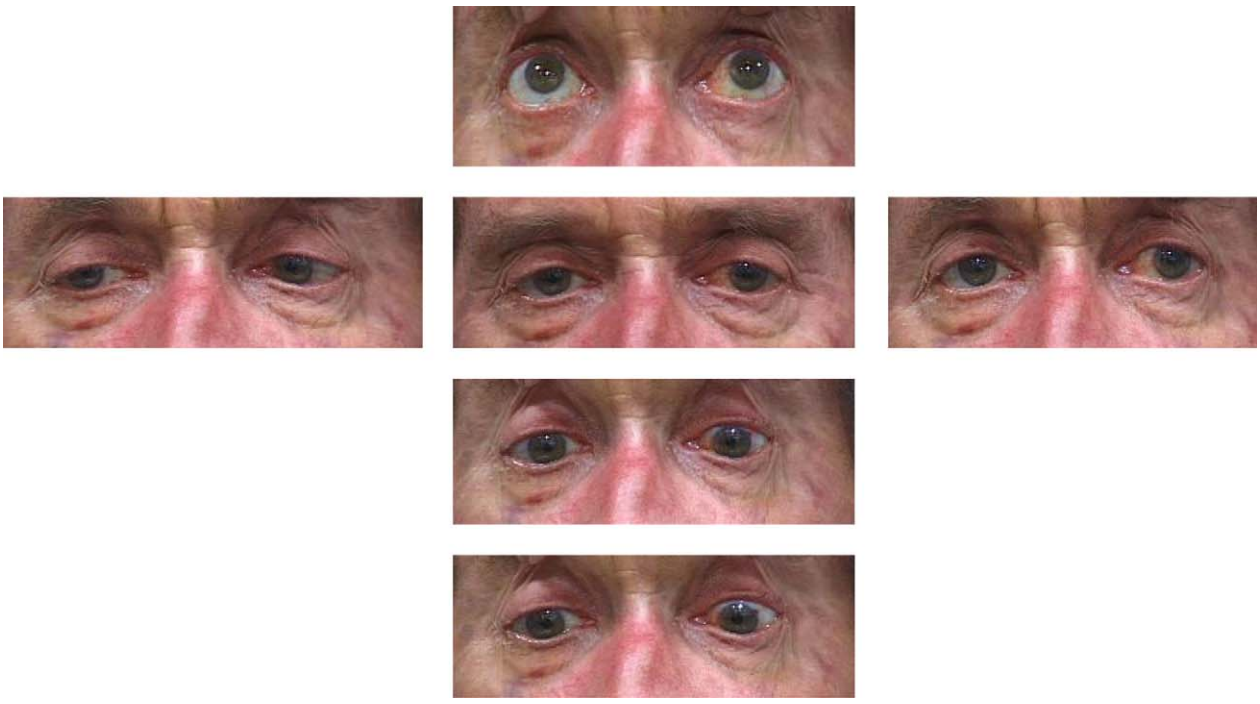

Fig. 1. Gaze positions demonstrating ocular paresis. Limitations in vertical and horizontal gaze were consistent with gross external ocular paresis. Dextroversion and levoversion were limited to 5°. Supraversion was reduced to 15° and infraversion was limited to 10°.