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Sir,

Diplopia following intravenous administration of pamidronate

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We report the case of a 77-year-old man who developed lid oedema, chemosis, and diplopia on two occasions following intravenous administration of disodium pamidronate.

Pamidronate,[1] or aminobiphosphonate, is a potent inhibitor of osteoclastic bone resorption but does not inhibit bone formation. It is used in the control of hypercalcaemia of malignancy, and in the treatment of osteolytic lesions and bone pain associated with metastatic breast carcinoma and multiple myeloma. It is sometimes indicated in the management of Paget's disease of bone.[2] Ocular side effects, including conjunctivitis, episcleritis, scleritis, and uveitis have been described in the literature.[3] Although the manufacturer has confirmed rare reports of transient diplopia, we found no other cases in the published literature.

Our patient was given intravenous disodium pamidronate for treatment of osteolytic lesions secondary to multiple myeloma. On each occasion, he became symptomatic 2 days after receiving the treatment. He developed erythema and swelling of both upper lids, chemosis, and vertical diplopia, worse on upgaze. On the first occasion, he was assessed in the eye clinic several days after the onset of symptoms. Chemosis and conjunctival injection had begun to subside by the time of examination. He had a 1 mm left ptosis and bilateral limitation of upgaze, which affected the right eye more than the left. There was some limitation of abduction bilaterally. After 4 months, he received intravenous disodium pamidronate again. Eyelid oedema, chemosis, conjunctival injection, and diplopia developed within 48 h of administration, and again resolved spontaneously. The time course of onset and resolution were similar to the previously observed pattern. On review, 2 months later he was free of symptoms and ocular motility testing was normal. Vertical diplopia in the primary position recovered over the course of 2 weeks on each occasion, diplopia on elevation persisting for a further 2 weeks. Chemosis and lid oedema had resolved within 5 days of onset of each episode. No signs of uveitis were observed on either occasion.

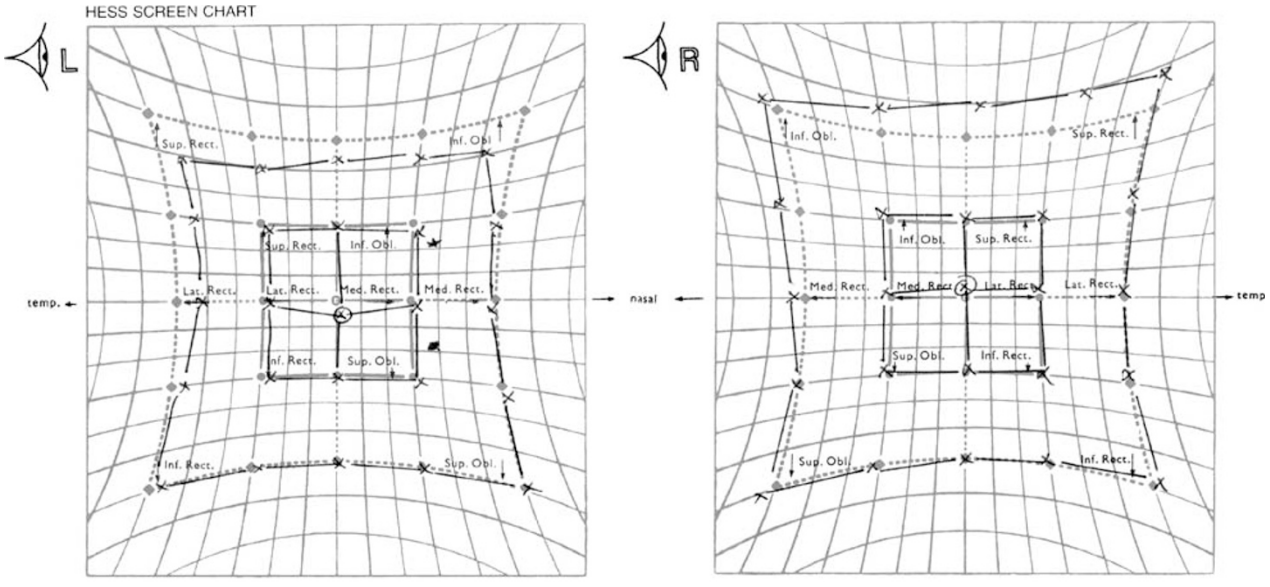

Figure 1 Hess chart recorded following re-challenge with intravenous disodium pamidronate.

Discussion

It is unclear why pamidronate should have a selective effect in inducing an inflammatory reaction in the orbital tissues, with no apparent effect on muscle function elsewhere in the body. It is probable that the intraorbital inflammation and consequent oedema involved the recti muscles during both the episodes. If the effects of intraorbital oedema on muscle function were not equal bilaterally, as is evident from Hess chart (Figure 1) recorded during the second episode, then diplopia during conjugate eye movements would be expected. Diplopia in the primary position may have been due to breakdown of a small pre-existing vertical phoria, although none was clinically evident when he was assessed after resolution of the second episode.

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Sir,

Horner's syndrome following attempted internal jugular venous cannulation

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Intraoperative central venous cannulation permits rapid administration of intravenous fluids and assessment of changes in intravascular volume. The right internal jugular vein has several advantages as a cannulation site, having a predictable anatomy, a high success rate in adults and children, and being easily accessible.[1] Complications of cannulation at this site include air embolism, pneumothorax, and brachial plexus damage. The most common complication is haematoma formation, which can result from puncture of the carotid artery.[2] Horner's syndrome has rarely been described as a complication. We present a case of Horner's syndrome following unsuccessful internal jugular cannulation and successful external jugular venous cannulation.

Case report

A 6-year-old girl was admitted for debridement of wounds under general anaesthesia. Sites for venous access were limited as she previously had bilateral below-knee and below-elbow amputations following meningococcal septicaemia. Intraoperatively right internal jugular vein cannulation was not successful. Access was gained to the right external jugular vein. In the recovery ward, the patient's mother immediately noticed a right ptosis and a 'large left pupil'. On returning to the ward, the patient was reported to have a 'large left pupil' by medical staff. The child had not complained of headache or severe neck discomfort. Questioning and the patient records revealed that the patient had a history of strabismic ambylopia. No signs suggestive of Horner's syndrome had been noticed previously at the ophthalmic clinic or by the patient's mother. On ophthalmic examination, a right-sided ptosis and miosis were noted. Pupils were both reactive to light and there was a full range of eye movements. The anisocoria was noted to be greater in the dark. No heterochromia iridis was present. A diagnosis of right Horner's syndrome was made. There were no localising signs other than the presence of the external jugular venous cannula. Ultrasound scan 1 day later of right-sided neck vessels revealed no evidence of carotid

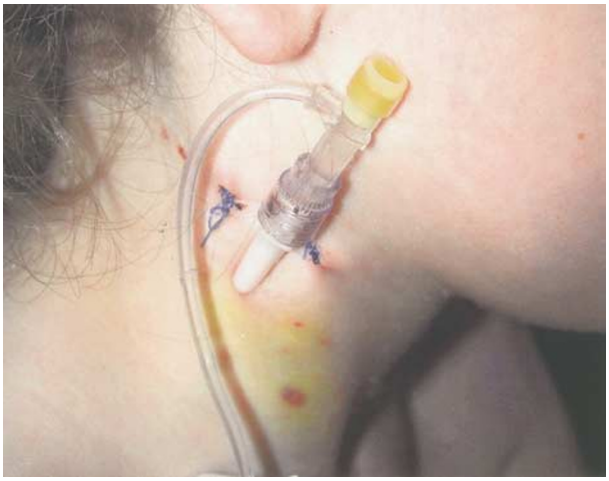

Figure 1 External jugular venous line *in situ*, day 3 post-operately.