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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TPJ Meaney, M Musadiq and PGJ Corridan

Wolverhampton and Midlands Counties Eye Infirmary, Compton Road, Wolverhampton WV39QR West Midlands, UK

Correspondence: TPJ Meaney
Tel:+44 01902 645 023
Fax: +44 01902 645 018
E-mail: tpjm@dz25.fsnet.co.uk

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

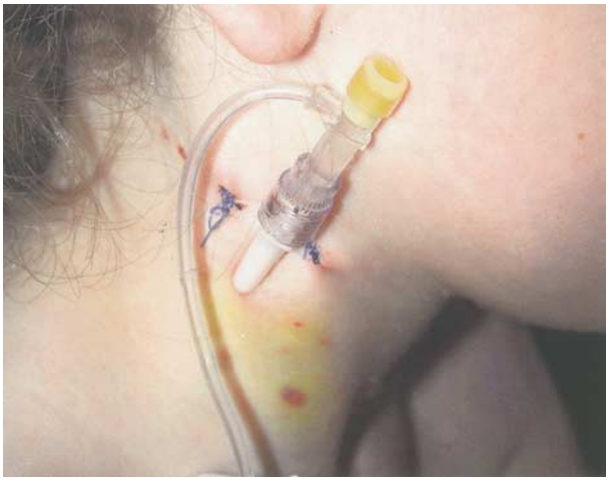

Figure 1 External jugular venous line *in situ*, day 3 post-operately.

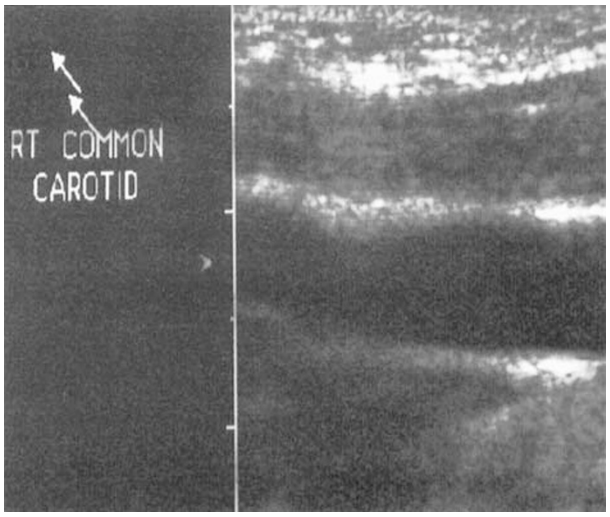

Figure 2 Ultrasound scan of right neck vessels; no arterial dissection or haematoma evident.

dissection or haematoma. After 18 days, the anisocoria and ptosis had improved.

Comment

Horner's syndrome results from interruption of the normal sympathetic nervous supply to the eye. The sympathetic nervous supply to the eye originates in the hypothalamus, and during its course passes up the cervical part of the sympathetic trunk. The sympathetic trunk is embedded in the carotid sheath, a condensation of deep cervical fascia. The internal jugular vein, common and internal carotid arteries, and the vagus nerve are also found in the carotid sheath. Normally, the internal jugular vein lies lateral to the internal carotid artery. The external jugular vein descends obliquely across the superficial aspect of the sternocleidomastoid muscle and pierces the deep cervical fascia just above the clavicle to enter the subclavian vein.[3] The cervical sympathetic trunk normally lies medial to the internal jugular vein, and therefore a needle should not normally encounter the sympathetic trunk *en route* to the vein. It has been suggested that damage to the sympathetic trunk can occur during internal jugular venous cannulation caused by excess rotation of the head and neck, which disturbs the normal anatomical relationships.[4] The ansa subclavia is the most anterior nerve bundle of those connecting the middle and inferior cervical ganglia of the sympathetic chain and is said to be a direct posterior relation of the internal jugular vein around the level of the cricoid cartilage. Haematoma can damage the ansa subclavia.[5] Haematoma could also compromise the blood supply to the superior cervical ganglion.[6] However, ultrasound in this case provided no evidence for haematoma formation. The needle may, therefore, have directly traumatised the sympathetic trunk. In the present case, internal jugular venous cannulation was unsuccessful. This may suggest that either abnormal anatomical relationships were present or that there was a greater than normal degree of exploration of the region with the tip of the cannula during attempted cannulation. In previous reports on Horner's syndrome following internal jugular venous cannulation in adults, there were several attempts to place the line incorrectly.[7,8]

Conclusion

If difficulties are encountered during internal jugular venous cannulation in children, the possibility of abnormal anatomical relationships should be borne in mind, and the degree of excursion of the tip of the cannula minimised. Horner's syndrome resulting from direct trauma to the sympathetic chain, as seems the likely aetiology in the present case, may recover spontaneously. Conversely, acute ipsilateral Horner's syndrome, particularly in postoperative or intensive care patients in whom pupils are observed to assess neurological function, may be due to recent attempts at internal jugular venous cannulation, even if an internal jugular venous line is not present.

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MA Williams, C McAvoy and JA Sharkey

Department of Ophthalmology
Royal Victoria Hospital
Belfast BT12 6BA, UK

Correspondence: MA Williams
Tel: +44 28 9 0240 503x 2346
E-mail: mikewilliams99@hotmail.com

Sir,

Primary position upbeat nystagmus associated with amitriptyline use

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This case illustrates that amitriptyline withdrawal may be responsible for the onset of primary position upbeat nystagmus. This is not a recorded side effect of the drug and it has not been previously reported in the literature.

Case

A 42-year-old man was seen in the West Suffolk Eye Department complaining of blurred vision for 5 days. His Snellen visual acuity was 6/60 in the primary position, and on down gaze it improved to 6/6. He had no medical history of eye problems; however, he did suffer from asthma and had had a basal skull fracture in 1996. A computerized tomography scan from 1996 showed right posterior temporal and left subfrontal region gliosis.

Eye examination revealed a primary position vertical nystagmus, and the fast phase was up. The nystagmus reduced on down gaze. He had no relative afferent pupillary defect; his eye movements were full; his colour vision, measured with Ishihara plates, was intact; and fundal examination showed no abnormalities. An examination of his central nervous system was normal. A magnetic resonance scan of his brain showed no new changes.

He had been taking salbutamol and flixotide inhalers for a number of years to control his asthma. At 6 months before the onset of nystagmus, he had started taking amitriptyline 150 mg once daily, for depression. At 2 weeks before the nystagmus commenced, he reduced his amitriptyline dose down to 100 mg. At 2 weeks after the start of the nystagmus he cut the dose to 50 mg, under the guidance of his GP. The dose was reduced to 25 mg a week later, and stopped after a further week. With the reduction of the drug, he found that his nystagmus gradually resolved.

The medicines control agency was informed of a possible association between amitriptyline and the induction of nystagmus; the agency reported two previously documented cases.

Comment

Upbeat nystagmus is a type of central vestibular nystagmus. It can be caused by lesions from the medulla to the midbrain. It usually increases in up gaze, but not on lateral gaze, and fixation does not dampen it. Causes of upbeat nystagmus include: cerebellar degeneration, multiple sclerosis, infarction of the medulla, tumours, Wernicke's encephalopathy, brain stem encephalitis, Behcet's syndrome, meningitis, congenital, middle ear disease, and drugs. This condition is often seen with medullary lesions; these involve the perihypoglossal nuclei, medial vestibular nuclei, nucleus intercalatus, and the ventral tegmentum. It is also reported in lesions of the anterior vermis of the cerebellum, the brachium conjunctivum, and midbrain.

Primary position upbeat nystagmus has been reported secondary to organophosphate toxicity[1] and tobacco.[2]

We propose that amitriptyline withdrawal may cause upbeat nystagmus by the following mechanism. Acetylcholine and histamine are found in the vestibular system; acetylcholine is a central vestibular agonist. Amitriptyline blocks histamine and muscarinic acetylcholine receptors and is therefore a vestibular suppressant. We suggest that withdrawal of amitriptyline removes this inhibition and may result in nystagmus owing to receptor hypersensitivity; this is a temporary symptom. If this is the case, there may be a role for amitriptyline in the control of nystagmus.

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SF Osborne and AJ Vivian

Department of Ophthalmology
West Suffolk Hospital
Bury St Edmunds IP33 2QZ, UK

Correspondence: SF Osborne
E-mail: sarahfosborne@hotmail.com