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Traumatic neuropathy of second cervical spinal nerves

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Abstract

The second cervical spinal nerves are unduly vulnerable to forcible approximation of the arches of the atlas and axis and to excessive rotation of the atlas on the axis. Sequelae of such injury include sensory aberrations ranging from loss of feeling to severe neuralgia and disorders of balance.

Diagnosis of second cervical neuropathy may be difficult when there are multiple injuries to the cervical spine, but most cases clear up spontaneously within one to three years.

Introduction

Occipital neuralgia was recognised in the nineteenth century and descriptions of the condition may be found in contemporary textbooks. It was attributed to an "affection" of the great occipital nerve until Charles Toogood Downing observed that "the Earl of . . . , a nobleman of strong constitution, was not relieved of cervico-occipital neuralgia when the sub-occipital nerve was divided by Mr Syme." This led Downing to suggest that occipital neuralgia was due to damage to the second cervical nerve.¹ A century later Hunter and Mayfield provided the anatomical explanation why the second cervical nerves are particularly susceptible to trauma.² Unlike the other spinal nerves, they are not protected by bony pedicles and facets. Their ganglia lie on the vertebral arch of the axis and the nerves pass between the arches of the atlas and the axis to the soft tissues in the neck. Hence they are clearly vulnerable and may be crushed when the two bony surfaces are approximated by forced hyperextension of the head. They may also be injured when excessive rotation occurs in one direction and the arches of the atlas and axis exert a scissor like action on the ipsilateral nerve. Thus both

hyperextension and excessive rotation may result in injury to one or both of the second cervical nerves. Wrenching movements of the head are particularly dangerous since they combine both movements.

Case report

A young waitress was a front seat passenger when her car was hit at high speed by another car coming from the opposite direction. She was not wearing a seat belt and was thrown forward and upward and then violently backwards. She was taken to hospital complaining of severe headache and dizziness and her neck was immobilised in a collar. She returned to work three months later. She was still having symptoms, however, which were in the form of sharp, intermittent headaches originating in the nape of the neck and radiating to the right temple and dizzy spells associated with nausea and loss of balance. (On one occasion when she was spooning cream on to a customer's pudding she suddenly lurched to one side and spilt cream all over his tie.) Examination showed diminished sensation to pin prick in the territory of the right second cervical spinal nerve and tenderness over the right great occipital nerve.

Review of 17 cases of post-traumatic neuropathy of the second cervical spinal nerve disclosed 12 that were caused by similar such acceleration-deceleration road traffic accidents. In the remainder the initiating trauma was less severe, and in two patients the injury occurred when the head was violently turned to one side to avoid a threat to the eyes.

Comment

Injury to the second cervical spinal nerve is associated with sensory aberrations usually associated with disturbances of equilibrium. The sensory aberrations range from loss of sensation to severe neuralgia, and the pain is often burning or stabbing. It tends to be intermittent, and associated with superficial tenderness of the scalp, which may be noticed on brushing the hair. The sensory changes may be hemispherical or localised to fixed areas within the distribution of the second cervical nerve—for example, periorbital, temporal, suboccipital, or, in rare cases, around the ear or at the angle of the jaw. Tenderness on palpation of the greater occipital nerve is common. The condition is usually unilateral, and in the rare cases of bilateral nerve damage the sensory abnormalities are usually asymmetrical.

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Occasionally the onset of the neuralgia is delayed because of temporary nerve block.

Disturbance of equilibrium was first noted by Magnus in 1924,³ and it is now thought that this is due to dysfunction of nerve fibres conveying afferent impulses from the muscle receptors in the neck to the vestibular nuclei. This is supported by Jongkees, who found that intradural section of the second and third cervical nerves led to immediate and severe vertigo.⁴

The diagnosis of second cervical nerve neuropathy should be straightforward if the variable nature of the sensory aberrations is borne in mind. In practice the diagnosis is often missed and indeed may be difficult in the presence of multiple injuries to the cervical spine. The differential diagnosis includes post-traumatic migraine and cluster headaches.⁵ In patients who have cervical spondylosis it is possible that less severe trauma may precipitate a neuropathy. Diagnosis and management may well be difficult when a patient seeks legal compensation for persistent symptoms.

In practice the diagnosis of second cervical neuropathy should be considered in any patient complaining of post-traumatic headache. A meticulous sensory examination of the scalp should be carried out in all such patients. The natural history of the condition is for most patients to recover spontaneously within one to three years. Surgery is seldom, if ever, indicated.

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SHORT REPORTS

Lymphomatoid granulomatosis in a 10 year old boy

Lymphomatoid granulomatosis was first described in 1972.¹ It is a form of angitis and granulomatosis, which predominantly affects lungs, skin, nervous system, and kidney. The condition has been reported with increasing frequency in the United States and Australia but has not been reported in Britain. It most commonly presents as a diffuse pulmonary infiltrate in middle age. We describe a case in a young boy.

Case report

A 10 year old boy presented in July 1981 with a productive cough, fever, night sweats, and lethargy. He had had asthma since the age of 4, which was well controlled with disodium cromoglycate and salbutamol. His condition deteriorated despite treatment with cephalothin and he developed respiratory failure with cyanosis, dyspnoea, intercostal and subcostal recession, and crepitations throughout both lungs. Liver and spleen were enlarged and palpable. A radiograph of the chest showed bilateral dense shadows (see figure). Results of investigations were: haemoglobin concentration 13.4 g/dl, white cell count $4.8 \times 10^6/l$ (neutrophils 87%, lymphocytes 10%), erythrocyte sedimentation rate 9 mm in first hour, and serum IgM concentration 0.2 g/l (normal range 0.54-1.98). All other tests were normal. *Haemophilus influenzae* was isolated on one occasion from the sputum but no other bacteria, fungi, or viruses were detected. Result of a tine test was negative. There was an initial titre of 1/320 to adenovirus and this fell to 1/80.

Despite intensive treatment with antibiotics the patient continued to deteriorate, and 14 days after admission he began mechanical ventilation and an open lung biopsy was performed. No bacteria, viruses, or fungi were grown and stains for *Pneumocystis* were negative. Macroscopically the lung was nodular, and histologically there was a dense interstitial infiltrate of lymphocytes, polymorphs, and histiocytes, these last showing pleomorphism and many mitoses. The cells invaded the walls of many small arteries (figure) with consequent obliteration and areas of coagulative necrosis and peripheral fibrosis. The appearances were suggestive of lymphomatoid granulomatosis as described by Leibow *et al.*¹ There was no bronchial or bronchiolar abnormality as seen in most infective conditions, and little intra-alveolar desquamation as seen in desquamative interstitial pneumonia, alveolar proteinosis, or lipoid interstitial pneumonia. Treatment was started with 60 mg prednisolone daily. He made a very rapid recovery clinically, and the chest x ray picture returned to normal. His pulmonary function was persistently abnormal, with forced expiratory volume in one second (FEV₁) 0.8 l and forced vital capacity (FVC) 1.2 l (predicted values 2.0 and 2.2 l, respectively). Two attempts at steroid withdrawal over the subsequent months resulted in a return of respiratory insufficiency and pulmonary infiltrates. When taking high doses of prednisone he became hypertensive. An intravenous pyelogram was normal.

In May 1982 he developed bilateral, huge parotid swellings and right facial palsy. There was no evidence of mumps and because of the persistence of the swellings a biopsy specimen was taken. This showed an infiltrate of lymphocytes and plasma cells with a disordered arrangement of salivary gland ducts. Treatment with high dose prednisolone resulted in almost complete disappearance of the swellings in 10 days. He had also had a skin infiltrate behind both ears, which responded to steroids. In November 1982

there was no evidence of active disease and the chest x ray picture was normal. Nevertheless, he was left with severe obstructive and restrictive airways disease, being dyspnoeic on exercise and showing an FEV₁ of 0.55 l, FVC 0.98 l, and a single breath carbon monoxide transfer factor (T_lCO) of 3.6 mmol/min/kPa (10.7 ml/min/mm Hg). These values again improved with 60 mg prednisolone daily to FEV₁ 0.95 l, FVC 1.35 l, and T_lCO 4.3 mmol/min/kPa (12.8 ml/min/mm Hg). Serum IgM concentration was normal on three occasions after diagnosis.

Comment

The clinical and histological features in this patient were compatible with a diagnosis of lymphomatoid granulomatosis, although parotid

Chest radiograph on admission, and histological appearances of open lung biopsy specimen two weeks later showing small pulmonary artery with segmental infiltration by atypical lymphoid cells (haematoxylin and eosin $\times 160$, original magnification).