A case of Pourfour Du Petit syndrome following parotidectomy

Paralysis of the cervical sympathetic nerves causes an ipsilateral Horner’s syndrome and this is a well recognised phenomenon in clinical neurology. However, the opposite hypersympathetic state following damage to the cervical sympathetic nerves is seldom recognised. We present a case report of such a condition which has been termed the Pourfour Du Petit syndrome.

A 41 year old man had a left parotidectomy for mixed parotid tumour. After the operation he noticed left facial weakness which was of lower motor neuron type. This resolved within three months. He also noticed immediately postoperatively that his left pupil was larger than the right. Initially he had some blurring of vision related to this pupillary dilatation but this completely resolved. However, the pupillary abnormality continued until the time of neurological examination six months later. Examination revealed lid retraction on the left side and a left pupil which when measured in a shaded room was two millimetres larger than the right (see fig). Both pupils responded briskly to light and accommodation; there was full eye movement and no double vision. There was no abnormal sweating response. The rest of the neurological examination was normal.

In conclusion, his signs were of autonomic overactivity to the left eye and this was thought to be due to damage to the sympathetic plexus which ascends around the carotid artery. This could have occurred during the time of surgery. An alternative explanation could be that there was a right-sided abnormality with relative pupils and miosis of the pupil as in Horner’s syndrome. This was thought to be untenable as clearly the surgery was on the left side. Pourfour Du Petit, a French physician, first reported this condition in 1820. The occurrence of the sympathetic plexus in the eyes and upper limb and relate these to cervical sympathetic chain injuries. These were largely caused by sword wounds on the neck inflicted during the Napoleonic war. Further cases have been reported after direct non-penetrating injury to the cervical sympathetic chain and brachial plexus.

We think our case is the first to have been reported following an operation. We suspect that this syndrome is often unrecognised but should be considered as a possible cause of pupillary dilation and lid retraction.

PAUL BYRNE

CHRISTOPHER CLOUGH

The Brook General Hospital, London, United Kingdom

Figure

Right mesodiencephalic haematomata.

Hypothermia in a mesodiencephalic haematomata

Hypothermia is defined by rectal temperatures of 35°C or less.¹ The most frequent causes are prolonged exposure to cold especially in the elderly, immersion in cold water, prolonged immobilisation, mountain-eering, intoxication (barbiturate, pheno-thiazine, carbon monoxide, alcohol), hypothroidism and hypopituitarism, sepsis, ketaocidosis and hypoglycaemia, and uraemia.²

This symptom is rarely reported in patients with well defined diseases of the central nervous system. Hypothermia may be continuous, as in various hypothalamic disorders (neoplasms, inflammatory or degenerative lesions) or, unusually, episodic as in “diencephalic autonomic epilepsy”, or agenesis of the corpus callosum (Shapiro’s syndrome).² Several recent reports emphasise the relative frequency of hypothermia during Wernicke’s encephalopathy and this can be rapidly reversed by thiamine treatment.³

In the case we report, moderate hypothermia with oculomotor disturbances were associated with a haematoma of the mesodiencephalic junction.

A 50 year old man was admitted with a sudden onset of apathy, general malaise and nausea which he experienced when taking a walk. His past history included five years of hypertension, the treatment of which had been interrupted one week earlier. On admission, he was alert and well oriented. Blood pressure was 240/120 mm Hg and pulse rate 75. Respiration was regular. Rectal temperature was 35°C, at repeated intervals. There was moderate motor neglect and hyposthesia of the left upper limb. Visual fields and pupils were normal. Vertical eye movements on command were not possible, both upwards and downwards. During forced elevation, nystagmus retractorius was observed. Vertical oculocerephalic reflexes were normal, as were all horizontal eye movements. No other abnormalities were detected.

Despite the absence of evidence of infectious disease, blood cultures were made, but no bacterial growth was observed. Serum sodium was 142 mmol/l, glucose 7.3 mmol/l, and creatinine 92 mol/l. Full blood count was normal.

Computed tomography of the brain without contrast revealed a right haematoma in the posterior mesodiencephalon, with a discrete mass effect on median structures, and traces of blood in the adjacent lateral ventricle. Magnetic resonance imaging (MRI) gave more precise localisation of the lesion (fig). The upper edge of the haematoma reached the pulvinar, while its lower limits only skimmed the red nucleus. Anteriorly, it did not cross the rostro-caudal axis of the brainstem, and was clearly posterior to the hypothalamus.

Hypothermia remained at 35°C for three days. Body temperature then rose slowly to 37°C, 10 days after admission. At that time, recording of eye movements showed full recovery of downward saccades and smooth pursuit, whereas upward saccade velocity remained extremely slow. The patient, when seen at follow up two months later, had completely recovered.

The physiology of thermal regulation by the central nervous system is not well understood. Experimental data from animals and pathological observations of human brain show that the hypothalamus plays a crucial role.¹,² Two types of neurons are found in the anterior hypothalamic nuclei and in the preoptic area: some respond to increases in blood temperature by releasing noradrenalin, whereas others respond to decreases in temperature by releasing serotonin.¹¹,¹² The former are more numerous, indicating that the anterior hypothalamus is particularly involved in heat dissipation. Anterior nuclei are connected by efferent pathways to the caudal part of the hypothalamus that is insensitive to temperature variations but essential for heat maintenance by regulating thermogenesis.³ The hypothalamus is connected to the thermosensitive areas of the brainstem, especially the lateral parts of the medulla oblongata, by several pathways. Fibres sensitive to cold would seem to be distinct from those sensitive to heat, the latter being redundant.¹³

Anatomical correlations are difficult to establish in human pathology since the lesions are usually large compared to the hypothalamic nuclei. Furthermore, one cannot exclude a remote mass effect. In our patient, the posterior hypothalamic region seemed intact on the MRI. Indeed, the mammillary bodies, located under the posterior
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P Byrne and C Clough

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