

## **POST TRAUMATIC PARKINSON'S SYNDROME A Case Report**

Mohinder Singh MS, FRCS, FRCOphth.\*

**A rare case of Parkinson's syndrome following head injury is described. The ocular abnormalities did not show any recovery. Early recognition of ocular changes helps to limit the imaging investigations to the cavernous sinus area. Aneurysm of the internal carotid artery, metastasis and trauma are the common aetiological factors in the few reported cases.**

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Anatomical and microsurgical observations of a small sympathetic twig joining the abducent nerve in the posterior cavernous sinus led Parkinson<sup>1</sup> to predict the occurrence of ipsilateral Horner's syndrome and sixth nerve palsy as a distinct clinical entity which is now popularly known as Parkinson's syndrome<sup>2</sup>. It is an extremely rare occurrence and the few reported cases of this syndrome have shown the presence of a space occupying lesion in the posterior cavernous sinus. Its recognition has a significant localising value and it alerts the clinician to focus the investigations on the cavernous sinus region. The present case report is rather unusual in the sense that it occurred following head injury causing trauma to the base of skull.

### **The CASE**

A 19 years old horse rider was involved in a car accident in February 1997 and was hospitalised with severe head injuries. He was unconscious on admission and had bleeding from the nose and left ear canal. There was periorbital oedema of the right eye. He had multiple fractures involving the base of skull. Opacification of the ethmoid, frontal and sphenoid sinuses was seen. There was fracture of the left petromastoid complex and wide spread subarachnoid haemorrhage in the basal and sylvian cisterns were noted. Scattered haemorrhages in the temporal and parietal lobes were present. Intraventricular bleed was also noted. He regained his consciousness after 17 days and slowly made a full recovery.

He presented to the eye clinic in July 1997 with complaint of left gaze horizontal diplopia and small size of the left eye. His ocular examination revealed visual acuity of 6/6 each

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\* Consultant

Department of Ophthalmology

Salmaniya Medical Complex

State of Bahrain

Currently working as Consultant Eye Surgeon in Specialized Eye Centre, Bahrain.

eye unaided. There was 3 mm left eye ptosis. The right pupil measured 4 mm and the left 2.5 mm (Fig1). There was no exophthalmos . Ocular motility testing showed no deviation in the primary gaze but gross limitation of abduction of the left eye. (Fig 2). Elevation and depression were normal in each eye. The posterior segments were normal. General and neurological examination was unremarkable. There was no history of any previous eye disease and family history was negative.. CT scan revealed the presence of fracture of the skull base involving the left sphenoid bone with evidence of air bubble in the posterior cavernous sinus. (Fig 3 & 4).

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*Figure 1. Photograph showing mild ptosis and smaller pupil of the left eye*

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*Figure 2. Photograph showing defective left eye abduction*

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*Figure 3. CT scan showing defect in the left sphenoid bone*

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*Figure 4. CT scan showing air bubble in the left cavernous sinus area*

He has not shown any improvement in the ocular findings during the follow up to date but continues to manage himself well.

## **DISCUSSION**

One to three sympathetic branches leave the superior cervical ganglion and accompany the internal carotid artery to the parasellar region. In this course they branch and rejoin to form the sympathetic plexus. After reaching the cavernous sinus with the internal carotid artery, some of the sympathetic fibres part company and join the abducent nerve for a short distance of about 2 mm and then leave it to accompany the ophthalmic division of

the fifth cranial nerve<sup>3-5</sup>. On the basis of this strange and short “get together” of sympathetic and abducent nerve, Parkinson predicted the occurrence of ipsilateral Horner’s syndrome and sixth nerve palsy on the side of cavernous sinus lesion. Only few cases of this rare clinical manifestation have appeared in the literature from time to time. In the two cases reported by Abad<sup>6,7</sup>, one had a traumatic aneurysm. Total obstruction of the proximal left internal carotid artery was the aetiology in a five year old child reported by Saur and Levinsohn<sup>8</sup>. In another case of Gelber and Sundt<sup>9</sup>, an aneurysm of the intrapetrous portion of the internal carotid artery had extended into the cavernous sinus and caused the Parkinson’s syndrome. Involvement of the ophthalmic division of the trigeminal nerve has also been noted in two cases of Parkinson’s syndrome<sup>10,11</sup>. Intracavernous carotid artery aneurysm was discovered in one case who was initially treated as a Tolosa-Hunt syndrome because of acute painful sixth nerve palsy at presentation<sup>12</sup>. Breast cancer metastasis in the posterior cavernous sinus was considered the most likely cause in another case<sup>12</sup>.

In the present patient, the initial head trauma producing fractures at the base of skull appeared to have damaged the structures within the cavernous sinus on the left side. The presence of air bubble in the left cavernous sinus area is suggestive of such an occurrence which caused permanent injury to the sixth cranial nerve and the accompanying sympathetic branches as this patient did not show any recovery during follow up. Possibility of a space occupying lesion in the posterior cavernous sinus should always be kept in mind when a patient presents with Parkinson’s syndrome. Patients presenting with acute sixth nerve palsy should be kept under close observation because they might develop Horner’s syndrome during follow up and then require investigation of the cavernous sinus area<sup>12</sup>.

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