therefore very important in neurological localisation. A lesion above the decussation would cause ipsilateral CED, while lesions below the decussation and in the PGC would cause contralateral CED. Lesions at the decussation would produce bilateral gaze palsy.

We present here a patient with a lesion in the left striatal-subthalamic region and a CED to the left. Contralateral CED (similar to seizure activity involving the frontal lobe) has been observed during electrical stimulation of the globus pallidus internus for the treatment of Parkinson's disease. This lends credence to the theory that subcortical structures may be implicated in the generation of CED.⁷ Anagnostou et al. in their study of stimulation location and parameters, concluded that CED was a result of activation (through volume conduction) of fibres in the internal capsule originating from centres in the frontal lobe (FEF or supplementary eye field) and projecting to the brainstem. An alternative explanation would be involvement of the diencephalic grey matter structures, such as the striatum and subthalamic nucleus. Clinically, striatocapsular haemorrhage may be associated with transient ipsilateral CED.^{8,9} Animal studies have demonstrated the complex nature of the genesis and control of CED with respect to the interplay between deep ganglionic structures such as the caudate nucleus¹⁰ and subthalamic region¹¹ with the well known cortico-mesencephalic pathway. Positron emission tomography (PET) studies in humans are also consistent with these animal experiments, demonstrating the role of the basal ganglia.¹²

In our patient, we believe that the ipsilateral CED was a result of a destructive lesion at the striato-subthalamic level, before the decussation of the supranuclear fibres subserving lateral gaze (functionally similar to lesions of the ipsilateral FEF). To the best of our knowledge, this is the first report of a traumatic brain injury presenting with gaze palsy due to a lesion in the striato-subthalamic region.

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Pituitary apoplexy in a child presenting with massive subarachnoid and intraventricular hemorrhage

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Summary A case of pituitary apoplexy in a child with massive subarachnoid and intraventricular hemorrhage is reported. The patient presented with a sudden onset of headache, diminution of visual acuity and a bitemporal visual field defect. Computerized tomography revealed a massive subarachnoid hemorrhage with extension of blood into the third ventricle. Angiography did not show an aneurysm. The patient was operated on and altered blood within a necrotic pituitary adenoma was decompressed. His vision and field defect improved along with resolution of headache in the postoperative period.

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INTRODUCTION

Pituitary apoplexy is a well known clinical entity caused by acute enlargement of a pituitary adenoma as a result of hemorrhage or massive infarction.^{1,4,8} Blood from the pituitary tumor may extravasate from the sella turcica into the adjoining basal subarachnoid space and produce a clinical picture of subarachnoid hemorrhage.^{1-3,6,7} Rarely, a pituitary hemorrhage may even extend into the ventricles, leading to an intraventricular hemorrhage.4,5 We report a rare case of pituitary adenoma that presented with sudden onset of severe headache and visual disturbance. Computerized tomography (CT) revealed massive subarachnoid hemorrhage in the basal cisterns and sulci along the cerebral hemisphere. Blood was also noted in the third ventricle. These findings simulated an aneurysmal hemorrhage, so cerebral angiography was performed, which revealed no aneurysm, arteriovenous malformation or spasm of vessels. However, the A1 segments of both anterior cerebral arteries were displaced upward, supporting the diagnosis of a suprasellar mass. A magnetic resonance imaging (MRI) study revealed a mass in the sella with suprasellar extension and evidence of hemorrhage.

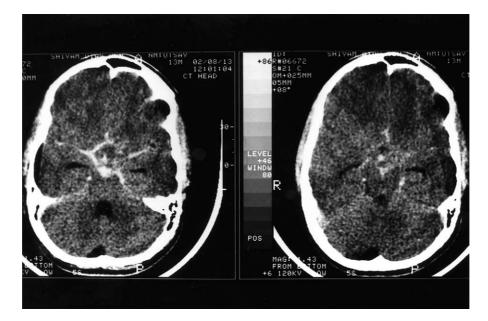


Fig. 1 The cranial CT scan revealed massive subarachnoid hemorrhage with extension of blood in to the third ventricle.

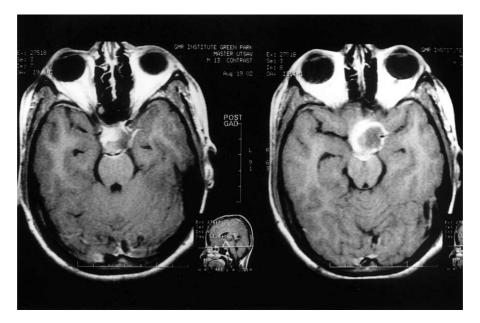


Fig. 2 MRI axial view showed a large suprasellar mass, with a small sellar component, with heterogeneous enhancement after contrast administration.

CASE REPORT

A 13-year-old school student reported to our emergency service with a sudden onset of headache and visual disturbance for 6 h. He had a similar headache with mild visual disturbance four weeks prior which had responded to simple analgesic. His parents ignored the event as a mild medical illness. On admission, he was conscious and orientated. He was thinly built with an average height. Secondary sexual characteristics were well developed. His visual acuity was 6/9 on the right and 6/36 on the left. He had a bitemporal field defect on bed side examination. The pupils were normal in size and briskly reactive to light. The optic fundi were normal. Signs of meningeal irritability were present. The rest of the neurological examination was unremarkable. The hematological and biochemical profiles were normal, and the endocrine profile showed normal hormonal status. The X-ray cone view of the sella revealed a normal size sella. The cranial CT scan revealed massive subarachnoid hemorrhage with extension of blood into the third ventricle (Fig. 1). MRI showed a large suprasellar mass with a small sellar component, with heterogeneous hyperintensity on T1 weighted images, suggestive of recent hemorrhage in pituitary adenoma. Heterogeneous enhancement was demonstrated with contrast administration (Fig. 2). The mass was causing elevation of the optic chiasm. Cerebral angiography revealed no aneurysm or arteriovenous malformation. The A1 segments of both anterior cerebral arteries were displaced upward. He underwent decompression of the pituitary adenoma using a transcranial approach. At surgery there was a soft gray mass with altered blood in the suprasellar region with a postfixed optic chiasma. Evidence of old subarachnoid hemorrhage was also noted by xanthochromic pigmentation of the arachnoid matter in the chiasmatic region. Histopathology revealed a chromophobe pituitary adenoma with evidence of necrosis and hemorrhage. Immunohistochemistry was negative for growth hormone and prolactin. He showed improvement in his vision in the immediate postoperative period with almost complete recovery; however, he was continued on a small dose of cortisol and thyroxin for maintenance therapy.

DISCUSSION

Pituitary apoplexy is a well-known clinical syndrome.^{1-4,6-8} In pituitary tumors, bleeding frequency is 4-5 times higher than that of other primary intracranial neoplasms.¹³ The incidence of apoplectic presentation of pituitary adenoma varies in the literature between 15% and 27.7%.¹⁶⁻¹⁸ It is a cascade of complex clinical events, occurring following fulminate expansion of a pituitary tumor by hemorrhage, infarction or a combination of the two, involving the tumor and the neighboring pituitary gland.¹⁸ Because of the anatomical proximity of the sella turcica to the basal cisterns, blood from pituitary tumors may enter the subarachnoid space.^{1–3,8} When the pressure gradient within the sella exceeds the resistance of the surrounding structures, blood is expelled into the subarachnoid space, producing a clinical picture that may be indistinguishable from the typical aneurysmal subarachnoid hemorrhage. Furthermore, many patients are unaware they harbor an asymptomatic pituitary adenoma.

Subarachnoid hemorrhage may be a presentation of pituitary apoplexy.^{1,9–11} Mohanty et al¹ reported evidence of xanthochromic CSF in lumbar punctures in two out of three cases who had pituitary adenomas presented with apoplexy. Further, in one case, xanthochromic pigmentation of the arachnoid matter in the chi-asmatic region was also noted during surgery. Rarely, pituitary tumor blood may spread into the ventricle. Patel et al⁴ and Challa et al⁵ also reported extension into the ventricle. Our case presented with apoplexy, and a CT revealed subarachnoid hemorrhage and seepage of blood into the third ventricle. Our patient did not have intracerebral bleeding.

Our case is unique as the patient had two episodes of headache associated with massive subarachnoid and intraventricular hemorrhage with blood in the intrasellar and suprasellar regions. In this patient, a diagnosis of ruptured aneurysm was suspected because of the abrupt onset of headache and vomiting together with the CT findings. The previous episode of headache could be considered a warning headache. But visual deterioration in aneurysmal bleeding due to compression of the anterior visual pathway is rare.¹³ On the other hand, visual deterioration is a cardinal sign of pituitary apoplexy which occurred in 80% of 38 cases of major pituitary apoplexy, analyzed by Wakai et al¹⁴ As our case also had visual symptoms, we suspected the possibility of pituitary apoplexy. The cone view X-ray of the sella is simple but helpful, as an enlarged sella will support a diagnosis of pituitary apoplexy. But our patient had a normal sella. Bills et al¹² recommended that pituitary apoplexy be kept in the differential diagnosis of "angiographically negative SAH". The cranial CT scan is the investigation of choice for the initial investigation of

SAH. It shows the spread and severity of hemorrhage in the subarachnoid space, as well as its extension into the ventricles or cerebral parenchyma.⁷ When subarachnoid hemorrhage is caused by pituitary apoplexy, CT often shows the pituitary tumor as well as the hemorrhage. In our case it was not clearly evident, so cerebral angiography was required to exclude an aneurysm. Finally, MRI is required to delineate the tumor as well as the associated hemorrhage or infarction. Wohaibi et al⁷ reported that contrast enhanced MRI will also reveal the defect in the tumor capsule, which represents the site of rupture of the tumor capsule.

The present case highlights the importance of angiography in differentiating SAH of aneurysmal origin and that caused by pituitary apoplexy. Pituitary adenoma has been reported to co-exist with aneurysm in up to 7.4%,¹⁵ therefore a patient with a pituitary adenoma may also bleed from an associated aneurysm. Cerebral angiography may exclude an aneurysm but MRI is advisable in patients with angiographically negative subarachnoid hemorrhage with visual symptoms.

CONCLUSION

The possibility of pituitary apoplexy must be considered in every patient with a history of intense headache and worsening visual acuity. "Angiographically negative SAH" must be followed by cerebral MRI with special emphasis of the sellar region. Surgical decompression should be carried out as early as possible for preservation of vision. Surgery not only ameliorates the visual deficit but prevents further morbidity associated with pituitary apoplexy.

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