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Review

Pituitary macroadenoma causing symptomatic internal carotid artery compression: Surgical treatment through transsphenoidal tumor resection

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ABSTRACT

Pituitary macroadenomas can invade the cavernous sinus and rarely cause occlusion of the internal carotid artery (ICA). Most patients with symptomatic obstruction of the ICA by a pituitary tumor have been reported as a result of apoplexy. The authors review the literature about this condition and report a 48-year-old man who presented with transient ischemic attacks leading to a stroke. Imaging studies demonstrated complete occlusion of the left ICA and critical narrowing of the right ICA at the level of the clinoid processes, most likely due to macroadenoma mass effect. There was no radiologic evidence of apoplexy. Surgical resection of the tumor and ICA decompression via the transsphenoidal route resulted in prevention of further symptoms. Histopathologic analysis confirmed a nonfunctioning pituitary adenoma without evidence of hemorrhage or intratumoral infarction. This patient, to the authors' knowledge, is the first documented patient with symptomatic carotid compression by a pituitary adenoma without evidence of apoplexy.

1. Introduction

Pituitary adenomas usually become clinically symptomatic as a consequence of hypersecretion, hypopituitarism, or mass effect on the adjacent neural structures. Common symptoms related to mass effect are headaches, presumably caused by stretching of the diaphragma sellae, visual field defects, and decreased visual acuity secondary to optic apparatus compression. Further suprasellar extension can cause hypothalamic dysfunction and obstructive hydrocephalus. Lateral extension toward the temporal lobes can rarely cause seizures [1]. Involvement of the cavernous sinus is common and infiltration can occur in up to 10% of patients [2,3]. Symptomatic cavernous sinus involvement usually presents with dysfunction of cranial nerves, most often in the context of an apoplectic event [4]. Compression or occlusion of the cavernous carotid artery by pituitary adenomas is rare and usually asymptomatic. Most patients with symptomatic compression reported in the literature were secondary to pituitary apoplexy [5–24].

In this article, we review the literature and report a patient with a pituitary macroadenoma who presented with symptoms of transient ischemic attacks (TIA) that eventually evolved into a complete stroke without radiologic evidence of pituitary apoplexy. We considered these ischemic events to be related to internal carotid artery (ICA) compression due to the tumor. Further events were avoided through ICA decompression by tumor resection. To our knowledge, this is the first report of such a presentation.

2. Illustrative patient

2.1. History and examination

A 48-year-old man presented with episodes of right-hand weakness and speech difficulty lasting for a few minutes. For 6 months prior to presentation, he had noticed a decrease in his visual acuity. MRI subsequently demonstrated a large pituitary adenoma encasing and narrowing both ICA (Fig. 1). He was started on aspirin therapy and continued to experience more frequent episodes of imbalance, slurred speech, and new left-sided weakness. After several episodes, the left-sided weakness became persistent.

Further diagnostic tests before neurosurgical consultation included an MRI that demonstrated restricted flow within the right middle cerebral artery watershed distribution (Fig. 2). The size of the tumor remained stable without any evidence of hemorrhage or acute infarction within it (Fig. 3). CT angiogram revealed complete occlusion of the left ICA and severe stenosis of the right ICA at the level of the clinoid process (Fig. 4). A complete stroke

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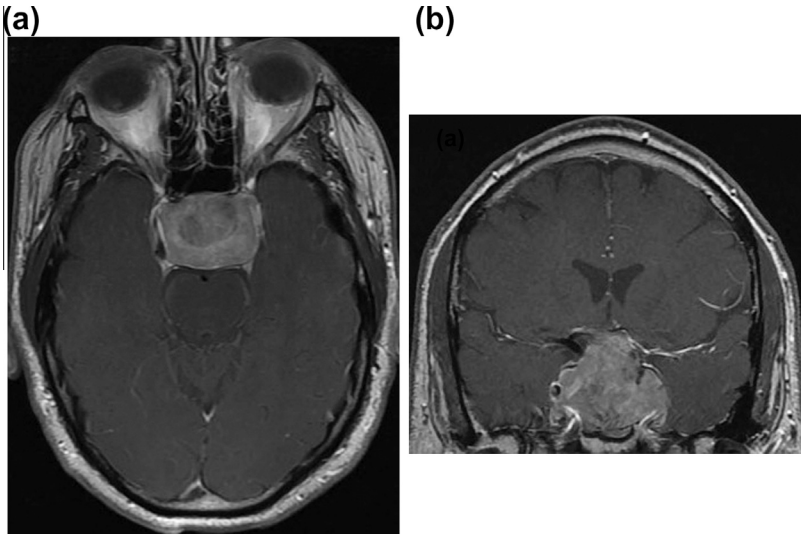

Fig. 1. Axial contrast enhanced T1-weighted MRI (a) showing a large homogeneously enhancing sellar mass. Coronal image (b) demonstrates a significant suprasellar component of this tumor and absence of a flow void within the left supraclinoid internal carotid artery.

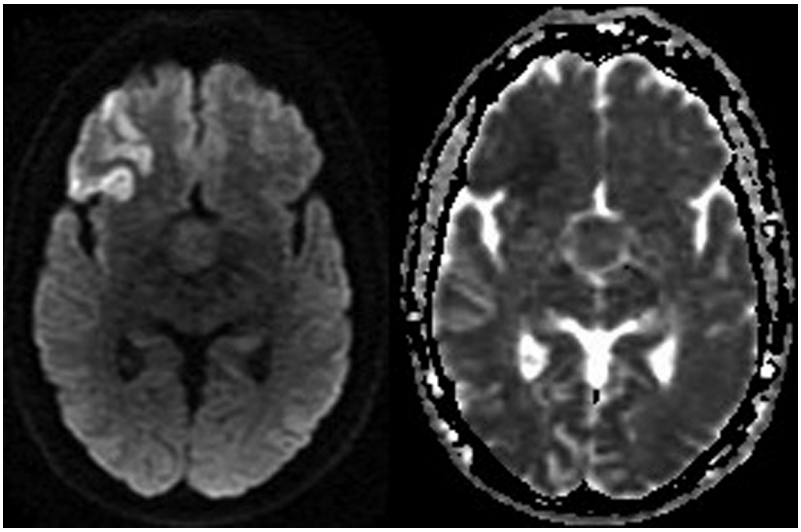

Fig. 2. Axial diffusion-weighted imaging (left) and apparent diffusion coefficient map (right) MRI sequences demonstrating ischemic changes in the right frontal lobe.

workup, including transesophageal echocardiogram, hypercoagulability, and autoimmune-inflammatory profiles, were within normal limits. Neurosurgical evaluation disclosed a mild left hemiparesis 4/5, left central facial paresis, and left tongue deviation. Visual field testing revealed bitemporal hemianopsia, worse on the left than the right side.

2.2. Surgery

The patient underwent an endonasal transsphenoidal resection of the tumor. Tumor decompression was achieved and diaphragma sellae descended into the sellar defect.

2.3. Histopathologic findings

Histopathologic examination was consistent with nonsecretory pituitary adenoma. No evidence of hemorrhage or necrotic tissue was found in the specimen.

2.4. Postoperative course

The patient had an uneventful postoperative course and his vision improved immediately after surgery. He did not suffer from any TIA during an 8 month follow-up period. A CT angiogram and MRI obtained 3 months postoperatively demonstrated adequate tumor decompression (Fig. 5). CT angiogram demonstrated restoration of normal flow through the right ICA; the left ICA remained occluded (Fig. 6).

3. Discussion

Among patients presenting with TIA or stroke, pituitary tumors are generally not considered on the differential. Although large pituitary tumors can cause compression or occlusion of the ICA, carotid compromise by pituitary adenomas is rarely symptomatic [25–28]. A number of mechanisms of ICA obstruction by pituitary adenomas have been proposed, including (1) direct compression of the vessel, most often secondary to rapid expansion of the tumor