


A Unilateral Nasal Mass With Generalized Seizures: Potential Diagnostic Pitfalls in Giant Pituitary Adenoma

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Abstract

Giant pituitary adenomas are clinically nonfunctioning adenomas, and the clinical presentation is usually secondary to compression of the neighboring structures. Visual impairment and visual field defect are the most common preoperative symptoms, followed by headache. Generalized seizures may occur in giant pituitary adenomas when there is involvement of frontal lobes or medial temporal lobes. We present a case of a unilateral nasal mass with generalized seizures in a 55-year-old woman without prior episode of seizure and any predisposing factors. Imaging showed a sinonasal tumor with intracranial extension and histopathological examination confirmed a corticotroph adenoma. On seeing a patient with a unilateral nasal mass extending down from the roof of nasal cavity, olfactory neuroblastoma, or meningo-encephalocele readily comes to mind. To avoid misdiagnosis and delay in treatment, imaging and, if possible, a biopsy should be considered. Giant pituitary adenoma although not common should be thought of as one of the differential diagnosis.

Keywords

giant pituitary adenoma, unilateral nasal mass, generalized seizures

Introduction

Pituitary adenomas are one of the commonest pituitary neoplasm, with prevalence rates of 100 per 100 000 individuals reported.¹ Most of pituitary adenomas are confined to the sella turcica. Initial complaints leading to diagnosis vary from symptoms of mass effect such as headaches, visual field defects, and ophthalmoplegias to symptoms related to hormonal hypersecretion. In the case of a giant and invasive pituitary adenoma, the lesion may extend down to the nasal cavity.¹ Giant adenomas are classified as tumors with extension of 4 cm or more in its diameter while invasive macroadenomas are defined as large tumors which grossly invade dura and/or bone and may have suprasellar or parasellar extensions.² When such a mass extends to the nasal cavity or nasopharynx, it may pose a possible diagnostic pitfall, delaying proper treatment from being administered.

Case Report

A 55-year-old woman presented with a generalized tonic clonic seizures which lasted for approximately

40 minutes and aborted spontaneously. She had history of an intermittent unilateral right nasal blockage, rhinorrhea, hyposmia, and epiphora for the past 3 months. There were no epistaxis, visual impairment, facial pain, headache, and no change of behavior. There was also no prior episode of seizure or any family history of seizure. She did not give history of exposure to radiation material, cancerogenic food or medication, head trauma, or family history of malignancy. Ocular examination was normal with no sign of proptosis or ophthalmoplegia. A thorough eye examination by ophthalmologist

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showed early sign of cataract over bilateral eyes without sign of peripheral visual loss. Cranial nerves examination was unremarkable. Nasal endoscopy revealed a right lobulated sinonasal mass with pale mucosa and prominent blood vessels (Figure 1). The mass extended from the right sphenoidal recess down to the right inferior meatus and the nasal floor. A computed tomography (CT) scan showed a heterogenous enhancing soft tissue mass within sphenoid sinus, ethmoidal air cell, extending down to the nasal cavity, superiorly extending to the right temporal lobe, and laterally involving the right cavernous sinus. There was erosion at the sphenoid sinus, ethmoidal air cells, right base of skull, and petrous part of right temporal bone (Modified Hardy classification type E). Magnetic resonance imaging (MRI) showed similar findings (Figure 2). Whole body CT scan (contrasted CT scan from brain to pelvic) did not reveal any suspicious changes suggestive of metastasis. The pituitary stalk was displaced to the left. Based on

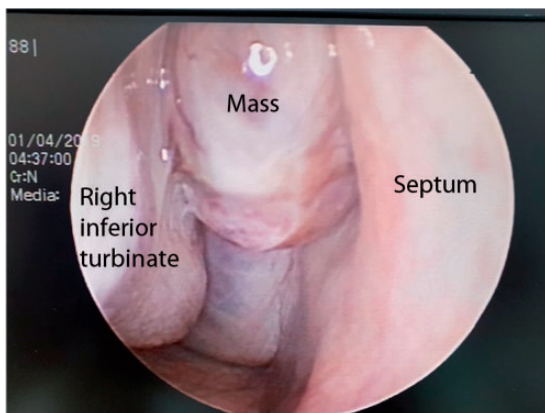


Figure 1. Endoscopic view of right nasal cavity showing mass extending down from sphenoidal recess to the nasal floor.

the imaging, an aggressive sinonasal tumor with intracranial extension was suspected.

Histological examination showed that the tumor cells were arranged in sheets. It exhibited medium-sized cells, round to oval hyperchromatic nuclei with bubbly ill-defined cell border cytoplasm. There was no atypical mitosis, lymphovascular, and perineural invasion seen. The tumor cells are positive for pancytokeratin, synaptophysin, and chromogranin A and negative for calretinin, epithelial membrane antigen, and leukocyte common antigen. Ki67 (proliferative index) is less than 2% which suggestive tumor of benign origin. Further immunohistochemical analysis for pituitary hormones confirmed a corticotroph adenoma (Figure 3). Blood hormonal evaluation showed an elevated cortisol level of 811 nmol/L (normal: 118.6–618 nmol/L). The serum

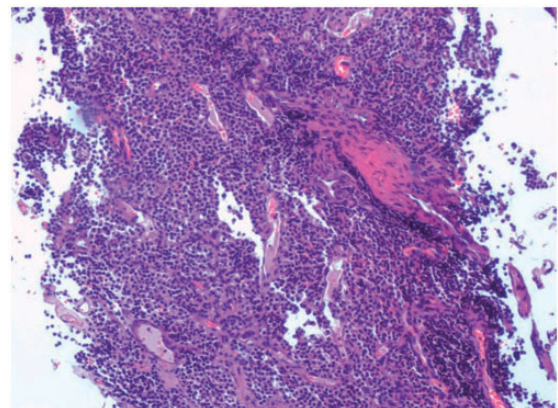


Figure 3. The tumor cells mainly arranged in sheets, consisting of monomorphic medium-sized cells with oval to round hyperchromatic nuclei (seen under H&E $\times 100$ magnification). Similar group cells with higher magnification (H&E, $\times 400$ magnification) showing hyperchromatic nuclei with moderate bubbly cytoplasm and some cells appear plasmacytoid.

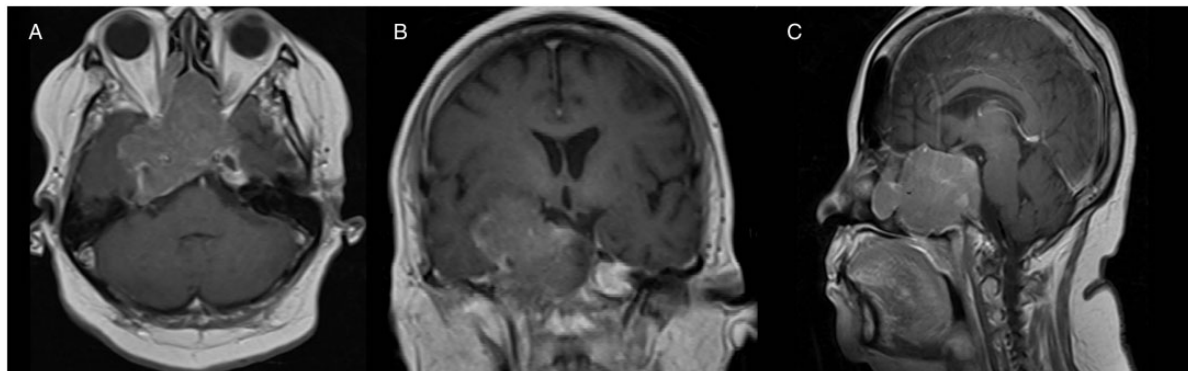


Figure 2. Magnetic resonance imaging of brain showed a large lobulated enhancing soft tissue mass at the sellar region extending anteriorly to the sphenoid and ethmoidal sinus, laterally to the cavernous sinus and the right carotid artery (A and B) and the extension of mass into the nasal cavity (C).

cortisol level was not suppressed with low-dose dexamethasone suppression test. Serum insulin-like growth factor-1, thyroid function test, prolactin, follicular stimulating hormone, and luteinizing hormone levels were within normal limit.

In view of the extensive tumor, she was managed by a multidisciplinary team consisting of neurosurgery, otorhinolaryngology, and endocrinology specialties. She was counseled for an endoscopic-assisted transcranial approach for tumor removal, but she declined the procedure. She was treated by an anticonvulsant, initially sodium valproate and later changed to phenytoin. There was no more seizures attack after the treatment. She was routinely reviewed once in every 4 weeks in a combined multidisciplinary clinic. Eleven months after her first presentation, she came to emergency room with shortness of breath and chesty cough. Clinical examination at that time showed bilateral pedal edema and bibasal crepitations on chest auscultation. Electrocardiography showed a left ventricular hypertrophy, while echocardiography demonstrated a left ventricular hypokinesia with left ventricular ejection fraction of 44%. While waiting to receive treatment, she suddenly became unresponsive and did not respond to resuscitation.

Discussion

On seeing patients with a unilateral nasal mass extending down from the roof of nasal cavity, olfactory neuroblastoma readily comes to mind. To avoid misdiagnosis and delay in treatment, giant pituitary adenoma should be considered alongside olfactory neuroblastoma as one of the differential diagnosis in such patients. Even though histological diagnosis of pituitary adenoma may not be difficult, when a biopsy is taken outside pituitary fossa, the diagnosis of pituitary adenoma may be unexpected. Moreover, a pituitary tissue from the nasal cavity might represent an ectopic pituitary adenoma. Other conditions such as neuroendocrine tumor, olfactory neuroblastoma, paraganglioma, plasmacytoma, and lymphoma have to be considered and should be differentiated from pituitary adenoma. Imaging study such as CT and MRI scan is required to determine the origin and extension of the mass in an extrasellar pituitary adenoma.

Most of giant pituitary adenomas are clinically non-functioning adenomas (68.8%), and the clinical presentation is usually secondary to compression of the neighboring structures.³ Visual impairment and visual field defect (72%) are the most common preoperative symptoms followed by headache (13%). Symptom at initial diagnosis is usually visual field defect (51%). As for functioning giant pituitary adenoma, prolactinoma (54.2%) is the most reported cases followed by somatotropinoma (29.2%), gonadotropinoma (8.3%),

corticotropinoma (4.2%), and thyrotropinoma (4.2%).³ Majority of corticotroph adenomas are microadenomas, slow growing, and not visible on MRI. Corticotroph macroadenomas are rare, but behave as an aggressive tumor. Generalized seizures may occur in giant pituitary adenomas when there is involvement of frontal lobes or medial temporal lobes.³ The generalized seizures in our patient are due to the tumor extension to medial temporal lobes intracranially as seen on the MRI (Figure 2(A)).

The reason why certain adenomas behave in a locally aggressive fashion is yet unknown. Based on a cohort study,⁴ giant pituitary somatotroph adenomas have poor response to surgery, radiotherapy, or medical therapy and required aggressive multimodal management strategy to achieve disease control. Thus, a multidisciplinary approach consisting of neurosurgery, otorhinolaryngology, ophthalmology, and endocrinology specialties is required to achieve better outcome. Surgery is considered as the first line of treatment. Complete tumor excision, either through endoscopic transsphenoidal or transcranial surgery (reserved for supra and parasellar extension), is the treatment of choice in the majority of patients with giant pituitary adenomas.³ Medical treatment has limited roles and mainly employed in patients who have not achieved remission after pituitary surgery, in patients who are unable to undergo surgery and in patients who undergo pituitary radiation.⁵

Author Contributions

R. A., I. S. S., L. C. C., and B. A. were involved in the clinical care of the patient, literature review, and manuscript preparation. All authors read and approved the final manuscript.

Declaration of Conflicting Interests

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Statement of Human and Animal Rights

This article does not contain any studies with human or animal subjects.

Statement of Informed Consent

Patient consent has been obtained.

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