American Journal of Case Reports

ISSN 1941-5923
© Am J Case Rep, 2016; 17: 707-711
DOI: 10.12659/AJCR.900647

Received: 2016.07.17
Accepted: 2016.09.06
Published: 2016.10.06

Unusual Complication of Pituitary Macroadenoma: A Case Report and Review

Authors' Contribution:
Study Design A
Data Collection B
Statistical Analysis C
Data Interpretation D
Manuscript Preparation E
Literature Search F
Funds Collection G

ABCDEF 1 Mohamed Said Abbas
BDE 1 Mohamad Najm AlBerawi
E 2 Issam Al Bozom
ACDEF 3 Nissar F. Shaikh
ACDE 1 Khalid Yacout Salem

1 Department of Clinical Imaging, Hamad General Hospital, Hamad Medical Corporation, Doha, Qatar
2 Department of Pathology, Hamad General Hospital, Hamad Medical Corporation, Doha, Qatar
3 Department of Anesthesiology/SICU and Perioperative Medicine, Hamad General Hospital, Hamad Medical Corporation, Doha, Qatar

Corresponding Author: Mohamed Said Abbas, e-mail: m.abbas89@outlook.com
Conflict of interest: None declared

Patient: Male, 48
Final Diagnosis: Pituitary apoplexy complicated by cerebral infarction
Symptoms: Disturbed conscious level • loss of vision
Medication: —
Clinical Procedure: —
Specialty: Radiology

Objective: Unusual clinical course

Background: Pituitary macroadenoma is a common benign tumor that usually presents with visual field defects or hormonal abnormalities. Cerebral infarction can be a complication of a large pituitary adenoma. We report a rare case of bilateral anterior cerebral arteries infarcts by a large pituitary macroadenoma with apoplexy.

Case Report: A 48-year-old male patient presented with altered conscious level and sudden loss of vision for one-day duration. Magnetic resonance imaging of the brain showed a large seller and suprasellar hemorrhagic mass of pituitary origin, with associated bilateral areas of diffusion restriction in the frontal parasagittal regions, consistent with infarctions. Magnetic resonance angiography showed elevation and compression of A1 segment of both anterior cerebral arteries by the hemorrhagic pituitary macroadenoma. The patient underwent trans-sphenoidal resection of the pituitary adenoma, but unfortunately, ischemia was irreversible. Computed tomography (CT) done post-operatively showed hypodensity in the frontal and parietal parasagittal areas, which was also persistent in the follow up CT scans. The patient's neurological function remained poor, with GCS of 8/15, in vegetative state.

Conclusions: Vascular complications of the pituitary apoplexy, although uncommon, can be very severe and life threatening. Early detection of vascular compromise caused by hemorrhagic pituitary macroadenoma can prevent delay in intervention. Clinicians should also consider pituitary adenoma as a possible cause of stroke.

MeSH Keywords: Cerebral Infarction • Pituitary Apoplexy • Pituitary Neoplasms

Full-text PDF: http://www.amjcaserep.com/abstract/index/idArt/900647

Background

Pituitary apoplexy is a life threatening clinical condition with reported incidence of 0.6–22% [1]. Pituitary apoplexy complicated by cerebral infarction is a rarely reported condition, currently only 25 cases have been reported in the literature [2]. This case report shows an uncommon complication of a large macroadenoma of the pituitary gland.

Case Report

A 48-year-old South-Asian male patient, with unknown medical history, presented to the emergency department of an outside hospital with sudden loss of vision and confusion that began more than 24 hours before the presentation.

Plain and contrast-enhanced computed tomography (CT) scan of the brain done at the same hospital showed a large intrasellar and suprasellar mass lesion measuring 4.2×3.4×2.9 centimeters, with surrounding edema with suprasellar extension with downward sagging of the sellar floor, significantly encroaching upon the sphenoid sinus (Figure 1). The mass was seen effacing the suprasellar cistern, abutting the under surface of the optic chiasm, and splaying the circle of Willis. The lesion showed mild heterogeneous enhancement in post-contrast images. CT scan findings suggested the possibility of large pituitary macroadenoma. His hormonal profile taken at that time showed normal TSH, FSH, and prolactin but low free thyroxin, cortisol, Luteinizing hormone, and insulin growth factor.

The patient was then urgently transferred to our tertiary hospital for further evaluation and management. On arrival, the patient's GCS was found to be 8/15, so a follow-up CT scan of the head was done, and it showed a mild increase in the pituitary fossa lesion size 4.9×3.7×3.7 centimeters with perilesional edema.

Brain magnetic resonance imaging (MRI) with multiplanar and multisequences obtained specifically for the pituitary gland pre- and post-IV gadolinium contrast as well as magnetic resonance angiography (MRA) showed an enlarged sella turcica with large sellar and suprasellar heterogeneous mass lesion showing figure-of-eight configuration in the coronal images (Figure 2). The mass measured about 4.9×4×3.5 centimeters, with mixed signal intensity and heterogeneous post-contrast enhancement, as well as multiple intra-lesional areas of blood products at different ages showing T1 bright and SWI sequences dark signal (Figure 3). The lesion could not be separately visualized from the pituitary gland.

The mass had suprasellar extension and seen obliterating the suprasellar cistern with poorly defined optic chiasm, which

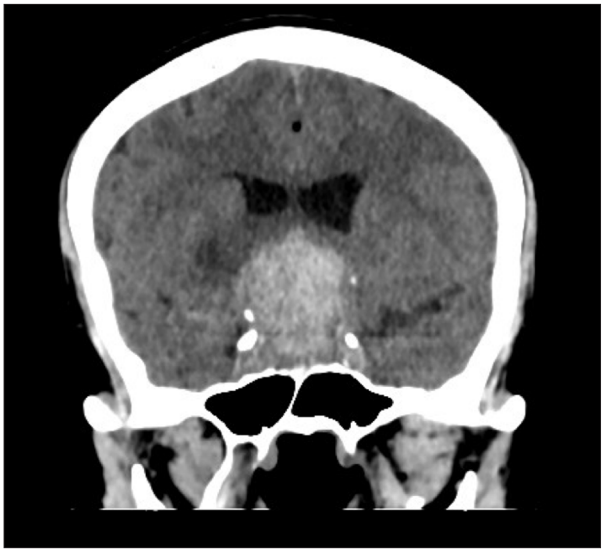

Figure 1. Coronal NECT showing fairly large sellar/suprasellar hyperdense hemorrhagic mass.

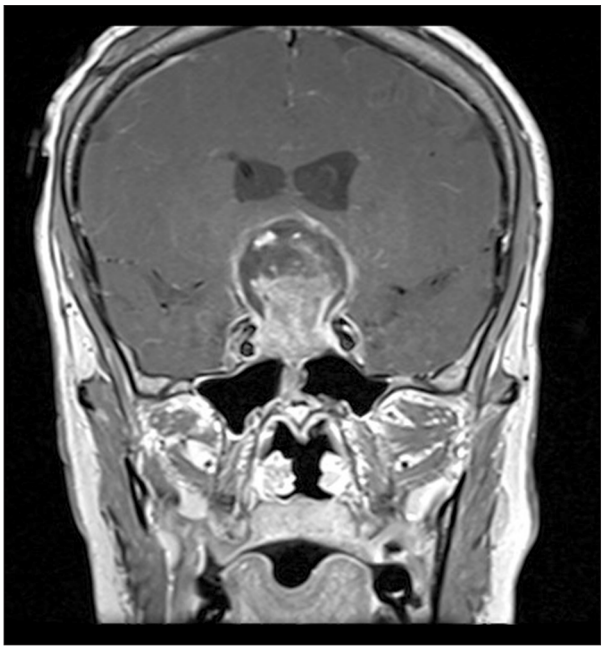

Figure 2. MR Coronal T1 WI showing heterogeneous enhancing sellar/suprasellar mass with supratentorial obstructive hydrocephalus.

is likely severely compressed by the mass. Cavernous sinus was intact with patent intra-cavernous internal carotid arteries segments.

MRA showed significantly elevated, stretched A1 segment of the anterior cerebral artery (ACA) as well as outer displacement and splaying of the supraclinoid portions of the internal carotid arteries (Figure 4). Diffusion weighted images (DWI)

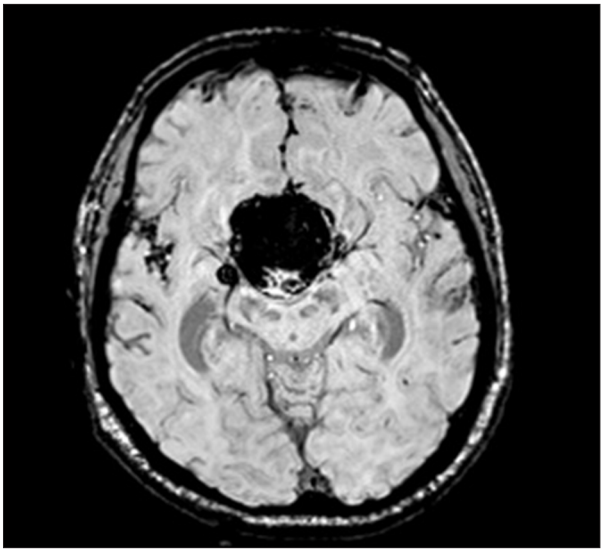

Figure 3. MRI axial SWI showing dark blooming within the mass, indicating underlying hemorrhage.

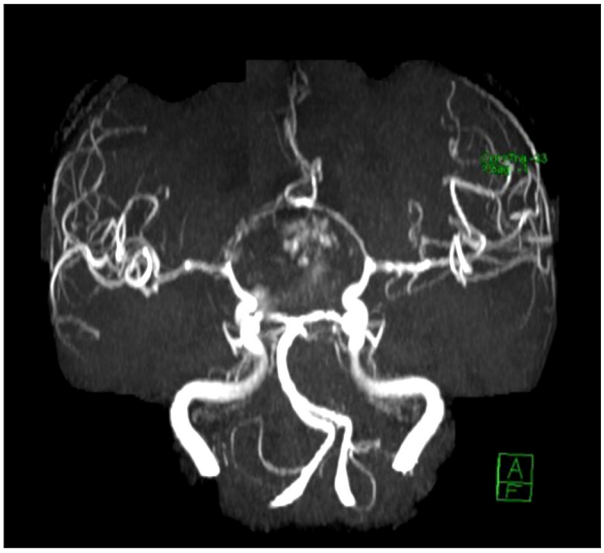

Figure 4. Cranial MRA TOF showing wide splaying, stretching, and upward anterior displacement of the A1 and proximal A2 segments of the ACA by the mass.

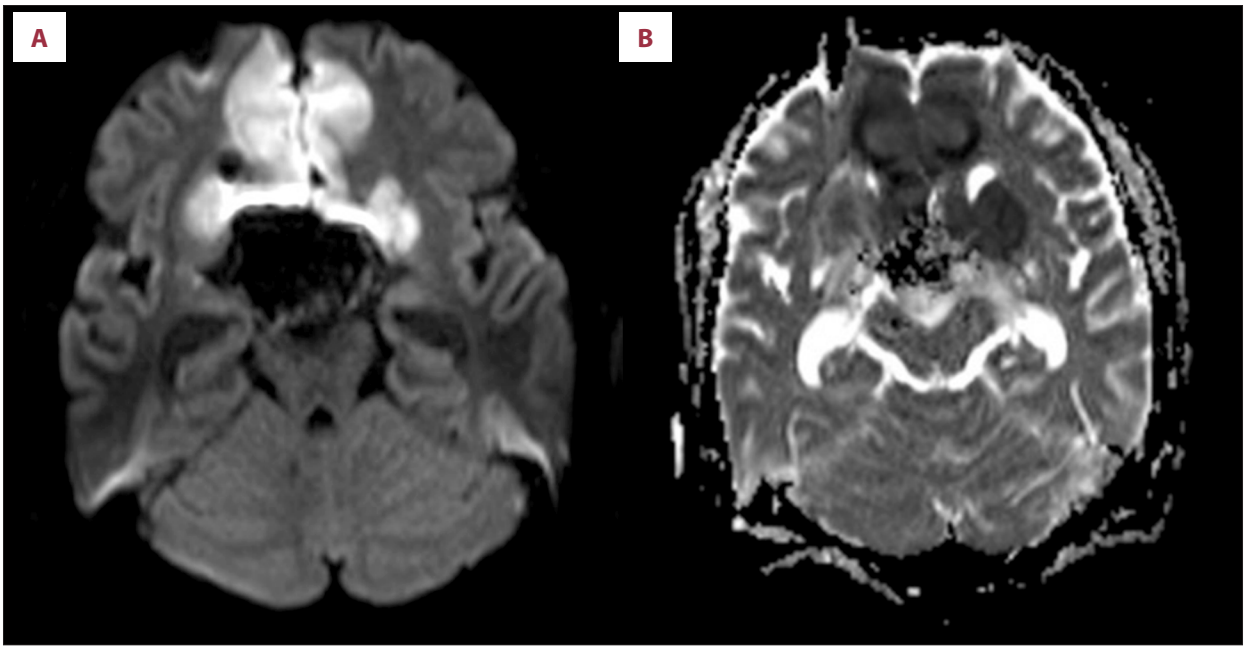

Figure 5. MRI DWI (**A**) and ADC map (**B**) images showing bilateral frontal parasagittal restricted diffusion bright signal in DWI and dark signal in ADC indicating underlying acute infarctions.

showed areas of diffusion restriction along the anterior frontal para-sagittal region bilaterally denoting infarction along the ACA tributaries (Figure 5A, 5B).

The patient then underwent endoscopic transnasal hypophysectomy. The pituitary mass and hematoma were removed. The histopathology report showed pituitary adenoma with apoplexy, expressing FSH, which was invading the bone focally (Figure 6A, 6B).

Post-operative non-contrast CT scan of the head showed ill-defined hyperdense area in intrasellar and suprasellar area suggestive of residual hemorrhage as well as bilateral hypodensities involving the frontal and parietal parasagittal regions and both caudate nuclei suggestive of bilateral ACA infarctions (Figure 7A, 7B).

A later follow-up CT scan showed irreversible ischemia of the ACA territories, with no improvement in the patient's

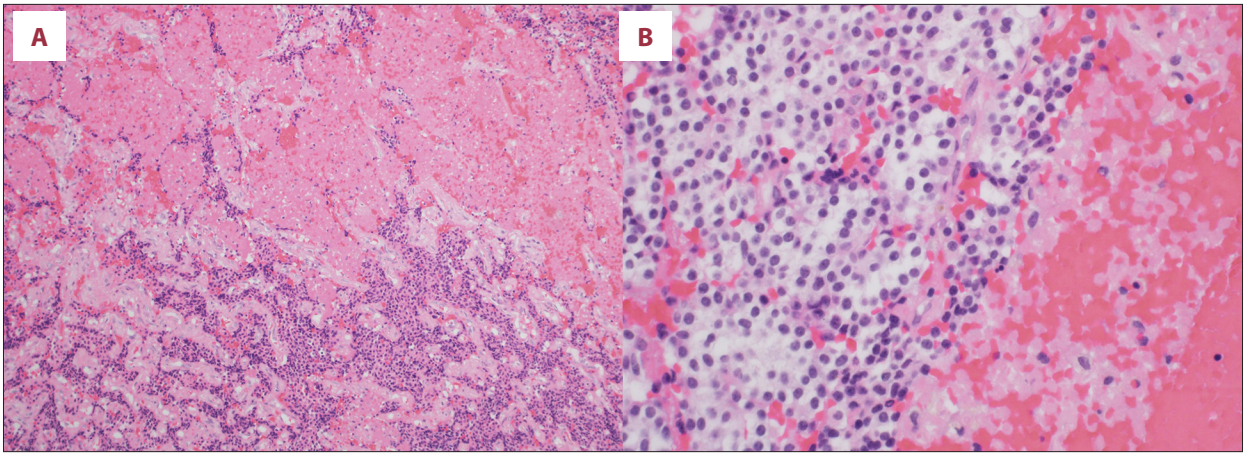

Figure 6. (**A**) Low power microscopic (H & E ×100) appearance of the adenoma with extensive areas of necrosis and hemorrhage seen in the upper half of the field. (**B**) High power microscopic view (H & E ×400) showing the pituitary adenoma on the left side of the view, and the details of the hemorrhagic necrosis (apoplexy) on the right.

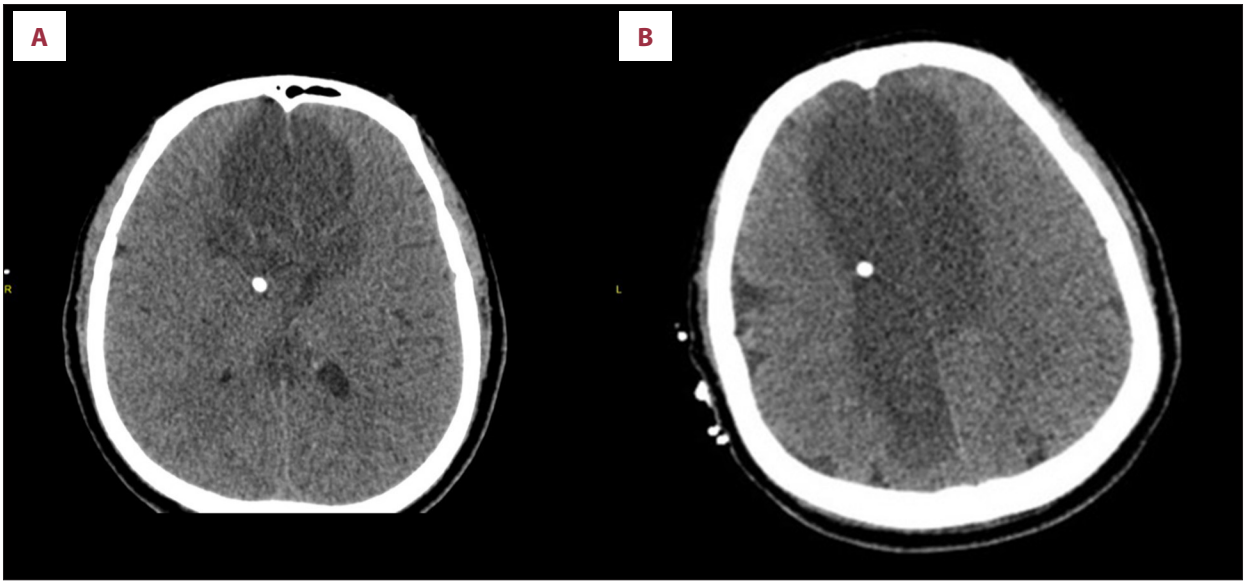

Figure 7. (**A, B**) Two days post-operative NECT of the head showing established large bilateral medial cerebral parasagittal hypodense recent infarcts along the territory of ACA.

neurological function. The patient remained in a vegetative state.

Discussion

Pituitary adenomas are one of the common neoplasms of the central nervous system. It accounts for 12.7% of all primary CNS tumors, with annual incidence of 2.63 per 100,000 person-years and prevalence between 25 per 100,00 to 77.6 per 100,000 population (CBTRUS 2010) [4]. They are further classified by size into micro-adenoma (≤1 cm) and macro-adenoma (≥1 cm), and according to hormonal activity as functional and non-functional adenomas. Macroadenomas usually present with mass effects as visual distrubances, while functional adenomas usually present with hormonal syndromes [5].

Pituitary apoplexy is the process where hemorrhage occurs within a pituitary adenoma due to excessive tumor infarction. It can be symptomatic causing headache, acute visual disturbances or disturbed conscious level, or asymptomatic [6]. It occurs subclinically in approximately 10–22% of patients with pitutiary adenoma, and symptomatically in 0.6–9% [7].

Reversible and irreversible cerebral ischemia are two of the reported complications following pituitary apoplexy. It can occur due to compression of vascular strucutres, or vasospasm following apoplexy, or due to tumor invasion of the nearby vascular structures.

In this case report we show another rare and unreported mechanism of injury by a pituitary apoplexy which is the bilateral streching and compression of the ACA, causing irreversible cerebral ischemia of the ACA territories. This case emphasizes the importance of considering screening for vascular complications caused by pituitary adenoma or apoplexy early by using cerebral angiography for better timely management and surgical planning.

ACA infarction has been previously reported in five out of all 25 reported cases of cerebral infarction complicated pituitary apoplexy (n=25) (20%) [2], unilaterally in two [8,9], while bilateral ACA was reported in three out of the five cases [10–12]. The mechanism of injury in ACA infarction cases was mentioned as vasospasm in 2 two cases [8,11], mechanical compression in two cases [10,12], and one was not known [9]. The outcome of these cases was mild to complete resolution in three cases and death in two cases — both of mechanical compression was the mechanism of injury [2].

Conclusions

Vascular complications of pituitary adenoma are uncommon, however, it can be very severe and life threatening. Early detection of vascular compromise caused by pituitary adenoma can prevent delay in intervention. Pituitary adenoma or apoplexy can be an unusual cause of cerebral ischemia, and should be considered.

References:

1. Kim JP, Park BJ, Kim SB et al: Pituitary apoplexy due to pituitary adenoma infarction. J Korean Neurosurg Soc, 2008; 43(5): 246–49
2. Banerjee C, Snelling B, Hanft S et al: Bilateral cerebral infarction in the setting of pituitary apoplexy: a case presentation and literature review. Pituitary, 2015; 18: 352–58
3. CBTRUS (2010). CBTRUS Statistical Report: Primary Brain and Central Nervous System Tumors Diagnosed in the United States in 2004–2006. Source: Central Brain TumorRegistry of the United States, Hinsdale, IL
4. Fernandez A, Karavitaki N, Wass JAH: Prevalence of pituitary adenomas: A community-based, cross-sectional study in Banbury (Oxfordshire, UK). Clin Endocrinol (Oxf), 2010;72: 377–82
5. Asa SL, Ezzat S: The pathogenesis of pituitary tumours. Nat Rev Cancer, 2002; 2: 836–49
6. Randeva HS, Schoebel J, Byrne J et al: Classical pituitary apoplexy: Clinical features, management and outcome. Clin Endocrinol (Oxf), 1999; 51: 181–88
7. Bi WL, Dunn IF, Laws ER: Pituitary apoplexy. Endocrine, 2015; 48: 69–75
8. Bhansali A, Dutta P, Khandelwal N et al: Pituitary apoplexy: An unusual cause of frontal lobe syndrome. Australas Radiol, 2005; 49: 127–31
9. Rodier G, Mootien Y, Battaglia F et al: Bilateral stroke secondary to pituitary apoplexy. J Neurol, 2003; 250: 494–95
10. Kurschel S, Leber KA, Scarpatetti M et al. Rare fatal vascular complication of transsphenoidal surgery. Acta Neurochir (Wien), 2005; 147: 321–25; discussion 325
11. Ahmed SK, Semple PL: Cerebral ischaemia in pituitary apoplexy. Acta Neurochir, 2008; 150: 1193–96; discussion 1196
12. Lill CM, Hoch H, Dieste FJ et al: Bilateral stroke following pituitary apoplexy. J Clin Neurosci, 2009; 16: 1670–73