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A Case Report on Pituitary Apoplexy.

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ABSTRACT:
Objective: To report a case of pituitary apoplexy presenting with unilateral ptosis and unilateral third nerve palsy in diabetic and hypertensive patient. **Clinical Presentation and Intervention:** A 50 years old man developed unilateral third nerve palsy. A pituitary macro adenoma with hemorrhagic and necrotic changes on MRI scan was resected by endoscopic transsphenoidal procedure. Third nerve palsy improved promptly and completely few hours postoperatively. **Conclusion:** This is a rare case of pituitary apoplexy that presented with unilateral third cranial nerve palsy recovered promptly after surgical excision of mass.

Key words: pituitary adenoma, apoplexy, magnetic resonance imaging

Introduction:

There are two general type of pituitary tumor based on size, microadenoma (with tumor size <1cm) and macro adenoma.[1] Pituitary tumors are divided functionally as functional or non-functional.[1] Non-functional pituitary macroademonas are called pituitary apoplexy. Pituitary apoplexy is defined as hemorrhage into pituitary tumor causing sudden onset headache, visual disturbance or decreased mental status and endocrine dysfunction.[1]

Case Summary:

A 50 years old gentleman presented in the emergency department with a chief complaint of headache.

Patient had acute onset right fronto-parietal headache, dull in nature, 5/10 in pain scale partially relieved by oral analgesics for 10 days. He had progressive dropping of right eyelids which started with the beginning of headache and complete dropping was noted 5 days prior to admission. Patient also complained of associated multiple episodes of vomiting of previously ingested food particles. During this course patient also had blurring of vision on bilateral eyes since 5 days prior to admission

No other accompanying symptoms such as loss of consciousness, dizziness, abnormal body movements or decreased sensorium were noted. Patient had no history of head injury. He had normal bowel and bladder habit.

Patient is known case of hypertension for 6 years under medication (Tablet Hydrochlorothiazide 12.5mg + Telmisartan 40mg once daily, Tablet Metoprolol 25mg once daily and Tablet Ecosprin 75mg once daily). Patient

was diagnosed with Diabetes mellitus type -2 6 years ago and was under medication (Tablet Metformin 500 mg twice daily). He is also diagnosed to have Dyslipidemia and was under Tablet Rosuvastatin 10 mg once daily for 6 years. He had hemorrhoidectomy done 20 years ago. His father had history of asthma otherwise unremarkable family history. Patient is non-smoker, non-alcoholic beverage drinker. He is follower of Islam.

Upon arrival to emergency department his vital signs were within normal range. GCS was 15/15. Pupils were bilaterally equal and reactive. His right eye was fixed on outward and downward position. Other neurological and physical examinations were normal.

MRI scan of brain was done revealing pituitary gland measuring 2.3 x 1.8 x 2.9 cm (CC x AP x TR) with expansion and enlargement of sella. It showed heterogeneous high signal intensity on T2W and high signal on T1W suggestive of hemorrhage with evidence of cystic/necrotic changes. It was resulting in expansion of the sella with suprasellar extension of the mass and narrowing at the waist giving a snowman appearance. The suprasellar component measured 0.8 x 1.1 x 1.1 cm. The mass was invading the bilateral cavernous sinuses and abutting the right intracavernous ICA by approx. 180 degrees. It was displacing the infundibulum to the left as well as chaism superiorly. Optic nerves are unremarkable.

Patient had growth hormone of 0.714 ng/ml, prolactin of 69 µlU/ml and hyponatremia with sodium of 125 mEq/L was noted. Other laboratory examinations were within normal range.

Endoscopic Trans-Sphenoid Pituitary mass excision was performed. Tense sella floor dura and solid tumor with intracavity blood was noted. The mass was excised and sent for histopathology.

Post operatively patient was shifted to surgical ICU and started with antibiotics, hydrocortisone and other supportive medications.

Patient was noted to have prompt improvement of ptosis few hours post-operatively. He was discharged on 6th post-operative day after full recovery of symptoms.

Discussion:

Pituitary tumor-associated hemorrhage was first described in 1898 by Bailey but Pituitary apoplexy was first described as a fatal case of pituitary tumor associated with hemorrhage or infarction in 1950 by Brougham et al.[1] Headache, visual deterioration and visual field defect were 3 of most common symptoms in patient with pituitary apoplexy.[2] Patient complains of retroorbital or frontal headache and not relieved by analgesics.[3] Our patient presented with 2 out of 3 (fronto-parietal headache and visual field defect) most common symptoms of pituitary apoplexy . Sudden onset of headache with neurologic and/or endocrinologic symptoms is result of abrupt expansion of mass within the sella turcica because of hemorrhage, necrosis or infarction in pituitary tumor and adjacent pituitary gland.[4]

Patients present with visual impairment in more than 80% of cases, hemianopsia is reported as the most frequent finding. Acute onset of visual acuity loss or even complete blindness is also recorded in some cases. Impairment of third, sixth and sometimes fourth cranial nerves lead to diplopia and ocular paresis in 50% of the cases. Ptosis, mydriasis and limited eye movement are noted most commonly due to involvement of third cranial nerve.[5] Visual field examination with the ophthalmologist is advised in this condition.

Magnetic Resonance Imaging (MRI) or Computed Tomography (CT) shows haemorrhage and/or necrosis within pituitary tumor. CT/MRI shows typical

heterogeneous intrasellar or suprasellar lesions with coexisting solid and haemorrhagic areas. Although CT scan can present alterations in pituitary parenchyma in acute phase of apoplexy and also detect sub-arachnoid hemorrhage or cerebral ischemia, MRI is first-line imaging technique for diagnosis of pituitary apoplexy.[6]

Risk factors of pituitary apoplexy can be reduced blood flow for large sized tumor, hypertension, diabetes, trauma, and increased intracranial pressure resulting in acute increase in hypophyseal blood flow, hormonal stimulation of pituitary gland and its tumor and anticoagulation therapy. Most of the patient with pituitary apoplexy were found to have hypertension and diabetes mellitus.[2] Use of dopamine agonists and estrogen administration is also considered as the risk factor for pituitary apoplexy, some studies suggest that cerebral angiography and cardiac or orthopedic surgeries are also associated with pituitary apoplexy and sometimes noted within hours to days after traumatic brain injury or pituitary radiosurgery.[5] Pituitary apoplexy is more evident in male in their 50s and 60s similar to that of our patient in the case.[7,8]

Patients sometimes present with hyponatremia as our patient presented.[5] Hyponatremia in cases of pituitary apoplexy can be due to severe hypocortisolism or sometimes inappropriate antidiuretic hormone secretion because of hypothalamus irritiation.[9] Decrease in Thyrotropin (TSH) noted in 50% of cases and gonadotorpins (LH and FSH) deficiencies are noted in 75% of cases but they are clinically relevant after several months.[5]

Rapid decompression of optic pathway is associated with favorable outcome in patient presenting with progressively worsening visual impairment.[10] Surgery within 7-8 days from diagnosis is indicated if patient have progressive visual field defect.[5] Surgical decompression results in improvement in visual acuity in 6-36% of cases and normalizes in 50% of cases.[11, 12] Other studies suggest that impairment of third, fourth and sixth cranial nerves can be permanent and no significant improvements are noted post operatively.[13] Turgut et al reviewed literature of pituitary apoplexy resulting in monocular or binocular blindness and concluded that timely surgery i.e. within 7-8 days from the emergence of symptoms can have positive impact on visual recovery.[14]

Conclusion:

Pituitary apoplexy is common among men in their 50s and 60s. Most of the cases are asymptomatic, those who are symptomatic commonly present with severe frontal or retroorbital headache associated with visual symptoms like ptosis, monocular or binocular blindness and mydriasis, and endocrinological signs like hpocortisolism or inappropriate antidiuretic hormone secretion. Early surgical decompression of the optic pathway results in prompt and complete resolution of visual field defects. Regular endocrinologic and ophthalmological evaluation is advised.

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