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Case Report

Internal Medicine Section

Acute Ischaemic Stroke as a Manifestation of Pituitary Apoplexy in a Young Lady

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ABSTRACT

Pituitary Apoplexy (PA) is defined as a clinical syndrome comprising headache, visual deficits and altered sensorium, which can result from haemorrhage or infarction of the pituitary gland. Acute ischaemic stroke following PA is very rare. We are presenting a 35-year-old young otherwise healthy lady who presented with neuro ophthalmological and vascular symptoms on a background of PA. Imaging revealed a pituitary macro adenoma with parasellar extension with internal bleed. Cerebral angiography revealed that the mass compressed the bilateral cavernous sinuses (left more than right), resulting in obliteration of the cavernous portion of the left Internal Carotid Artery (ICA). She was treated with steroids and surgical debulking of the tumour through trans-sphenoidal approach and postoperative imaging showed recanalization of the ICA with reduction of the tumour size. The histopathological diagnosis was consistent with pituitary macro adenoma. Patient improved in level of sensorium, eye movement and the patient showed almost full recovery after the operation. PA resulting in ICA occlusion is very rare. Early intervention is required for reducing mortality and morbidity and to improve quality of life.

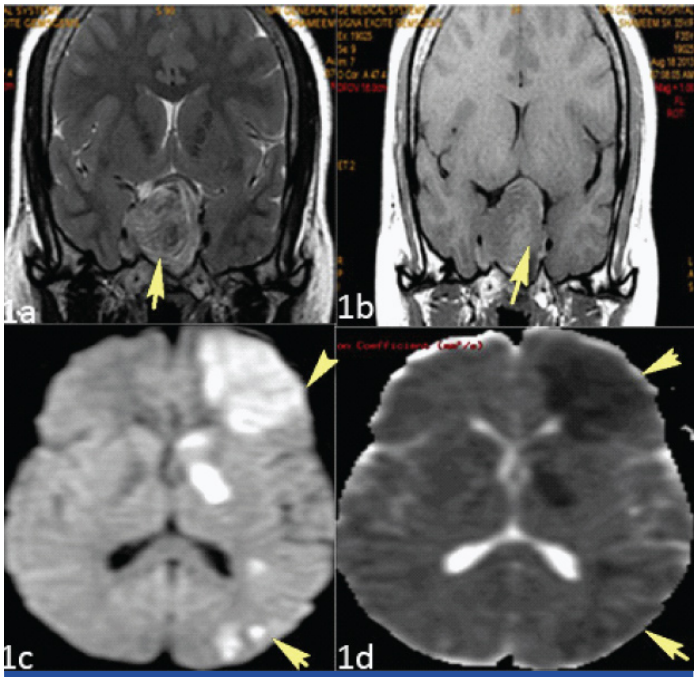
Keywords: Haemiplegia, Internal carotid artery, Pituitary haemorrhage, Visual failure

CASE REPORT

A 35-year-old young healthy lady presented with one day history of fever, right frontal headache, vomiting, drooping of right eyelid, visual failure in both eyes all concurring on same day evening. Next day fever, headache and vomiting subsided, but the patient had persistent visual failure and lid ptosis, however she could walk, verbalize and feed herself for initial three days of illness. She was imaged with MRI brain [Table/Fig-1a,b], which showed large suprasellar mass with left parasellar extension into the left cavernous sinus with T1 hyperintensity suggesting internal bleed probably pituitary macroadenoma with apoplexy. On the fifth day of illness, she had developed sudden onset difficulty in walking with right lower limb weakness which progressed to involve the right upper limb with inability to speak but able to comprehend minimally.

On examination she was conscious with Broca`s aphasia, right eyelid ptosis, bilateral visual failure to the extent of no perception of light with right eye, total ophthalmoplegia with bilateral mydriatic non reactive pupil (right more than left) and right haemiplegia with right seventh UMN (Upper Motor Neuron) facial palsy.

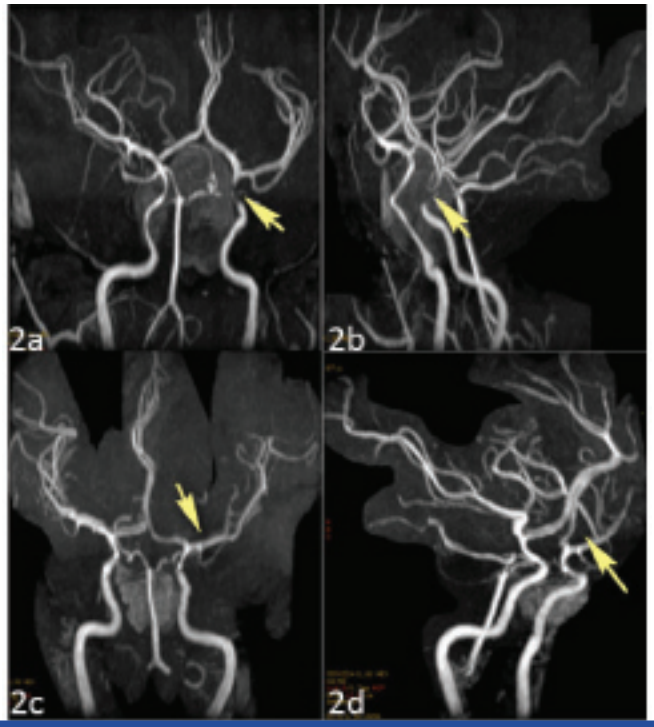
She was reimaged with MRI brain [Table/Fig-1c,d], revealing acute left ICA infarct with pituitary apoplexy in T2 and DWI sequences. Cerebral angiogram [Table/Fig-2a,b] revealed narrowing of bilateral ICA left side more than the right side. She had history of amenorrhea of one year without galactorrhea. She was the mother of four children (two neonatal deaths) with age of the last child being four years. She was non-diabetic and normotensive and she denied use of oral contraceptive pills. She was treated with steroids and subjected to definitive trans-sphenoidal resection of pituitary tumour. Postoperatively she was alert and eye movements were completely improved with slightly reactive pupils and vision improved to counting fingers to two meters with persistent lid ptosis. Two weeks later she could regain ability to speak and was ambulant with one-person support a week later. Two months



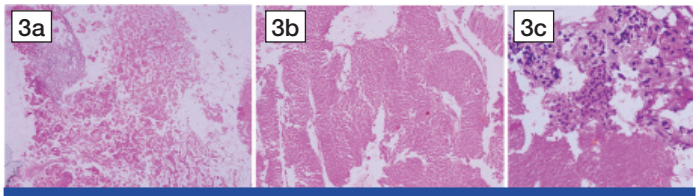
[Table/Fig-1]: a-b MRI Brain, T2 and T1 coronals of pituitary showing a large lobulated macroadenoma in sella with changes of bleed (arrow); **c-d:** DWI-MRI Brain, hyperintensity on Diffusion Weighted Imaging (DWI) (arrow head) and corresponding Apparent Diffusion Coefficient (ADC) map showing hypointensity (arrow head) suggestive of acute infarcts in left apsuloganglionic, frontal and parietal lobes (left ICA acute infarct).

postoperative imaging revealed complete resolution of tumour with complete recanalisation of ICA [Table/Fig-2c-d].

Evaluation revealed normocytic normochromic blood picture with negative viral parameters like HIV, HBsAg and HCV. Cardiac work up was normal through 2D Echocardiogram. We consider this tumour as non-secretory as levels of hormones (PRL, TSH, IGF-1) were within normal limits. Being a non-functioning (null-cell) tumour, post-



[Table/Fig-2]: (a-b) Pre op MR 3D TOF angiogram show critical stenosis of the left cavernous ICA (arrow) with splaying of bilateral ICA. (c-d) Post op 3D TOF angiogram showing opening of the left cavernous ICA.



[Table/Fig-3]: (a) Section showing pituitary adenomatous tissue with inflammatory cells (lymphocytes) and extensive liquifactive necrosis (H&E X400); (b) Showing pituitary adenomatous tissue with haemosiderin pigment and ghost outlines of necrotic monomorphous tumour cells arranged in broad bands (H&E X400); (c) Showing pituitary adenomatous tissue with infarcted necrotic tumour with monomorphic cells (with roud cells, scant cytoplasm, dense nucleus) (H&E X400).

operative medications like steroids or hormones were not advised. Despite the obvious aetiology of apoplexy, we made an attempt to rule out all causes of stroke in young like hypercoagulable states (normal protein C, S, ATIII, APLA, Homocysteine), vasculitidis (ANA, dsDNA) which were negative.

Histopathological examination [Table/Fig-3a-c] revealed large areas of infarcted pituitary adenoma with ghost outlines of relatively monomorphous cells in lobulous and sinusoidal pattern with adjoining areas showing sheets of foamy macrophages and a few lymphocytes suggestive of apoplexy.

DISCUSSION

Acute ischaemic stroke following PA is an extremely rare event and a few cases have been reported in the literature. Apoplexy occurs in 10% of intracranial tumours and 2-7% of pituitary adenomas [1].

PA results in a rapid increase in intrasellar contents with consequent sudden rise in Intrasellar Pressure (ISP) up to a median value of 47 mmHg [2]. Normal ISP is not known, but is believed to be similar or less than the normal intracranial pressure of 7–15 mmHg [3]. The sharp increase in ISP leads to disturbance of anterior pituitary cell viability, which could increase the risk of ischaemic necrosis and limit the potential for recovery of pituitary function.

Usually carotid artery strokes results in ipsilateral visual failure and contra lateral haemiplegias [4]. In our case bilateral visual failure was due to the tumour compressing the optic chiasm. Though bilateral cavernous sinuses are involved, it is the left sided ICA (carotid siphon), which produced contralateral right haemiplegia. Opthalmic manifestations in pituitary macroadenoma results from lateral expansion of the tumour compressing the III, IV and VI cranial nerves (causing ophthalmoplegia) and superior extension of the tumour compressing the optic nerve or optic chiasm (causing visual loss) [5,6]. Ptosis at the level of the cavernous sinus can be either due to palsy of parasympathetic third nerve, sympathetic plexus around ICA (Horner`s) or can be parasympathetico sympathetic palsy (Dilatation lag) [5].

The probable two most important mechanisms of cerebral ischaemic in patients with PA are mechanical obstruction of the circle of willis by the enlarging mass [7] and cerebral arterial vasospasm [8]. The ICA was occluded in the cavernous sinus or supraclinoid portion by the enlarged tumour in most cases [9]. The pathophysiology of vasospasm could be the release of vasoactive substances from the necrotic haemorrhagic tumour itself [8,10], hypothalamic dysfunction, intra-operative manipulation, direct arterial wall injury and the subarachnoid blood [11]. In our case the compression of cavernous portion of left ICA is the mechanism of ischaemic stroke.

There are only a few cases reporting mechanical compression of circle of willis as the chief event of cerebral ischaemic as illustrated by Rosenbaum TJ et al., Lath R and Rajshekar V et al., [12,13], and few other studies [7,9,14], while a few reporting cerebral vasospasm as the primary event [10,11,15-18]. Following [Table/

Author	Age/ Sex	Symptoms	Days after onset	Vascular territory	Angiographical findings	Mechanism of Stroke
Rosenbaum TJ et al., [12]	77/M	Stupor, Left Haemiparesis	0	Rt MCA	Rt ICA occlusion, Lt ICA stenosis	Compression
Cordoso ER and Peterson EW [10]	34/F	Altered consciousness	21	Diffuse	Bilateral ICA, MCA, ACA stenosis	Vasospasm
Haens D et al., [15]	43/M	Aphasia, Rt Haemiparesis	0	Bil ACAs	Lt ICA stenosis	Vasospasm
Pozzati E et al.,[8]	15/M	Drowsiness Rt facial palsy	0	Rt MCA	Bilateral ICA stenosis	Vasospasm
Clark JD et al., [14]	40/M	Dysphagia, Rt haemiplegia, Rt facial palsy	0	Lt ACA	Rt ICA stenosis, Lt ICA occlusion	Compression
Itoyamma Y et al., [17]	45/M	Rt Haemiparesis	14	_	Lt ACA and MCA stenosis	Vasospasm
Lath R and Rajshekar V [13]	40/M	Lt Haemiplegia	1	Rt ACA	-	Compression
Akutsu H et al., [16]	29/M	Drowsiness Lt Haemiplegia	5	Lt Heubner's and medial lenticulo striate arteries	Lt ACA mild stenosis	Vasospasm
Rebeiz T et al., [18]	81/F	Stupor, blindness, quadriparesis	0	Bil ACA/MCA MCA/PCA	Anterior displacement of ACA	Compression
Present case	35/F	Headache, right Haemiplegia, blindness	3	Lt MCA	Left ICA Stenosis	Compression

[Table/Fig-4]: Reviews the published studies on association of acute ischaemic stroke following pituitary apoplexy.

Fig-4] reviews the published studies on association of acute ischaemic stroke following PA. Bilateral caudate nuclear infarcts secondary to PA was also described recently by Rebeiz T et al., [18].

CONCLUSION

Acute ischaemic stroke following PA is very rare. High index of suspicion is required in every case of sellar lesions to identify the signs of neurovascular compromise to prevent lethal complications. Surgical decompression through the trans-sphenoidal approach is appropriate. Early intervention is required to reduce the mortality and morbidity and to increase health related quality of life.

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