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Internal Carotid Artery Aneurysm Masquerading as a Pituitary Macroadenoma, Presenting as Pituitary Apoplexy-a Case Report

KEYWORDS: Internal carotid artery aneurysm, Pituitary apoplexy, Pituitary macroadenoma

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ABSTRACT *We report here a case of 30 Years old male, presented to us,with headache, right sided second and third nerve palsy , diagnosed as pituitary macroadenoma initially, latter turned out to be a case of thrombosed Internal carotid artery aneurysm.Subsequently he developed complete right middle cerebral artery infarct and pituitary apoplexy.*

Introduction
It is known that intrasellar aneurysm (ISA) represents only 1-2% of all cerebral aneurysm.[1] They commonly grow in the intracavernous or supraclinoid portions of the internal carotid artery although the anterior communicating artery and the basilar tip may sometimes be involved. Especially when larger than 25 mm(giant aneurysms), such aneurysms may mimic pituitary adenomas clinically, neuro-opthalmologically and endocrinologically.[1] Brian in his study found that 70% of ICA Aneurysm became symptomatic due to mass effect on surrounding structures,the most common presenting symptoms were decreased visual acuity, headache and endocrinopathy.[2]The first published report of an aneurysm occurring within the sella turcica dates back to 1887,when Sir Byroom Bramwell reported on a 31 year old man with bitemporal hemianopia which evolved to blindness and progressive hebetude[2].

CASE REPORT:
A 30 years old male presented with history of severe headache ,nausea and diminished vision in the right eye since two days .He also complained of drooping of right eyelid since one month. He had no history of fever, seizures or head injury. On admission, his pulse was 90/min and blood pressure was 130/90 mmHg. On initial neurological examination, the vision in his right eye was 6/36 and in the left eye was 6/6. There was ptosis in the right eye . All the movements in the right eye were affected except abduction and intorsion. Pupils were unequal , right eye 4 mm minimally reactive and left eye was 2mm and strongly reactive . There was a relative afferent pupillary deficit on the right side. There was no proptosis or signs of ocular inflammation and other cranial nerves were normal. There was no other neurodeficit or neck rigidity. Clinically there was no evidence of hyper or hypoactivity of the pituitary gland. Other systems revealed no abnormality.

On laboratory examination, his haemoglobin was 12gm %, total leucocyte count was 7200/cumm, blood sugar random was 180mg%. His electrocardiogram (ECG), X-ray chest and Ultrasonography (USG) Abdomen was normal . Magnetic resonance imaging of the brain showed a spherical T1 isointense sellar mass with patchy hypointensities (Fig 1)which was hypointense on T2 (Fig 2). On the next day he developed left sided hemiparesis acutely followed by fall in blood pressure to 80/50mmHg which further dropped to 60/40mmHg . Hormonal assay for pituitary studies were sent in view of MRI Brain (first day) showing a probability of pituitary macroadenoma.

The hormonal assay was as follows:

Table No 1 Hormonal assay for Pituitary function:-

HORMONES	LEVELS	NORMAL VALUE
S.ACTH	2.0 pg/ml	6.0-76 pg/ml
S.Cortisol(AM)	<1.0 ug/dl	3.70ug/dl-19.40ug/dl
S.Prolactin	1.47ng/ml	3.46ng/ml-19.4ng/ml
S.TSH	0.20uIU/ml	0.40 - 4.00 uIU/mL
S.IGF-1(Somatomedin C)	322.0 ng/ml	26 - 30 Yrs : 117 - 329 ng/mL
S.LH	0.64mIU/ml	1.14-8.75mIU/ml
S.FSH	3.59mIU/ml	0.95-11.95mIU/ml

(S.ACTH:-Serum Adrenocorticotropic hormone, S.TSH:-Thyroid Stimulating hormone, S.IGF-1:-Insulin like growth factor-1,S.LH:-Serum Luteinizing hormone, S.FSH:-Serum Follicular stimulating hormone)

To our surprise hormonal assay revealed that our patient was suffering from pituitary apoplexy. His repeat blood sugar was 50mg% , serum sodium was 101mEq/l . In view of left hemiparesis, repeat Magnetic Resonance Imaging (MRI) with Magnetic Resonance Angiography (MRA) was done. Gradient reversal echo showed a focus of blooming in the lesion (Fig-2) and MRA showed obstruction of flow in the supraclinoid part of the right internal carotid artery(Fig-3), leading to a diagnosis of a thrombosed internal carotid artery aneurysm. There was diffusion restriction in the entire left MCA territory with hypointensity on ADC maps, suggestive of acute MCA territory infarction. (Fig-4).

Patient was started on Inj. Hydrocortisone 100mg three times a day in view of pituitary apoplexy. Gradual replacement of fluids and electrolytes were done. Patient's relatives refused any further management of the aneurysm in view of the low general condition of the patient.

DISCUSSION:
Our patient initially presented as a sellar mass which mim-

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icked a pituitary macroadenoma on MRI. Clinically however it appeared to be a non functional adenoma as there were no signs of any hormonal overactivity. There were symptoms of mass effect in the form of right sided second and the third nerve compression. The differential diagnosis of sellar lesions is broad and includes a variety of neoplasms, cystic pathology, inflammatory and infectious lesions and uncommon entities of vascular origin.[2] Later it was diagnosed that the so-called pituitary adenoma was in fact a thrombosed internal carotid artery aneurysm which caused an MCA territory infarction and pituitary apoplexy due to pituitary ischaemia.

Pituitary apoplexy is a rare endocrine disorder which can occur due to infarction or haemorrhage of the pituitary gland. This disorder most often involves a pituitary adenoma .However pituitary apoplexy may also occur in non adenomatous or even the normal pituitary gland as in our case. Although Pituitary apoplexy can occur without any precipitating factor in most cases , there are some well recognizable risk factors. The multiple factor reported as precipitants of pituitary apoplexy can be reduced to four categories:1) Reduced blood flow in the pituitary gland may result from fluctuations in blood pressure.Indeed,hypotension in the setting of cardiac surgery,lumbar laminectomy,or haemodialysis,have been associated with pituitary apoplexy of both normal and adenomatous gland. 2)Acute increase in blood flow in the pituitary gland is considered as a classic triggering factor for pituitary apoplexy.3) Stimulation of the pituitary gland through increased estrogen states such as exogenous estrogen administration and pregnancy,dynamic testing of the pituitary using gonadotropin releasing hormone,thyrotropin releasing hormone or other secretologues,as well as other hormone treatment such as bromocriptine have also been reported to cause apoplexy. 4)Another identified predisposing factor is the anticoagulant state,whether from

administration of anticoagulant drugs,thrombolytic agent or thrombocytopenia which is usually associated with haemorragic pituitary apoplexy.[3] The etiology of pituitary apoplexy in our case was a thromboembolic episode which jeopardised the vascular supply of the pituitary gland.In our case, the hypophyseal and distal cerebral circulation was compromised by the thrombosed supraclinoid ICA aneurysm.

As Endocrine dysfunction is concerned the majority of Pituitary apoplexy patients (nearly 80%) will have deficiency of one or more anterior pituitary hormones at presentation. Clinically the most important endocrine dysfunction is adrenocorticotrophic hormone (ACTH) deficiency. Reveiwing a series of patients that had pituitary apoplexy Veldius and Hammond found multiple pituitary deficiencies such as Growth hormone (GH) deficit(88%),ACTH deficit(42%) hypogonadotrophic hypogonadism (85%).Hyperprolactenimia may be due to prolactinoma or due to the impairment of the inhibitory influence from the hypothalamus[4] . Our patient had ACTH,TSH,LH deficiency and hypoprolactinemia. Hypoprolactinemia is explained by the fact that our patient did not have any pituitary tumour and ischaemia of pituitary gland caused death of pituitary tissue leading to reduced production of prolactin.

For patients with unruptured large or giant carotid artery aneurysms presenting with cranial nerve dysfunction, therapeutic carotid artey occlusion, when tolerated is the treatment of choice. Clinical results are excellent and complications are exceptional. In patients who cannot tolerate carotid artery occlusion or who have bilateral aneurysm, selective coiling,with or without balloon or stent assistance is the best alternative with comparable clinical results. 0nyx treatment is not justified in view of high complication rate and high rate of delayed carotid artery occlusion . Bypass surgery should only be considered in patients with intradurally located aneurysm with a high chance of rupture.[5]

Conclusion

We should be aware that while diagnosing pituitary macroadenoma in any patient differential diagnosis of other sellar lesions including internal carotid artey aneurysm should also be kept in mind and visa versa. Apart from rupture , mass effects and thrombosis, it can also present as pituitary apoplexy.

Fig-1a MRI Brain:-T1 weighted sagittal section showing a spherical mass in the sellar region possibility of pituitary macroadenoma with haemorrhage

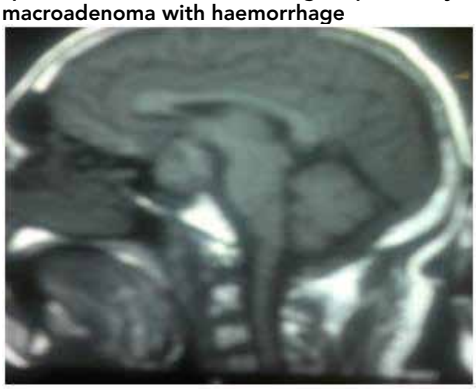

Fig-1b MRI Brain T2 weighted sagittal section showing hyperintensity with speckled hypointensities suggestive of a pituitary macroadenoma with haemorrhage.

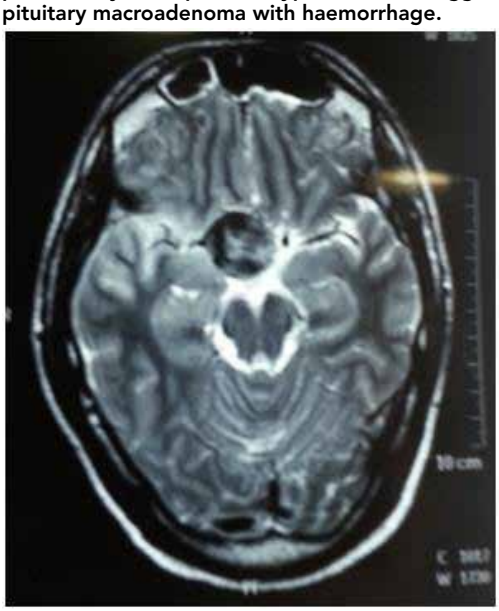

Fig-2 Gradient reversal echo showing focus of blooming in the lesion; possibility of Internal Carotid artery aneurysm

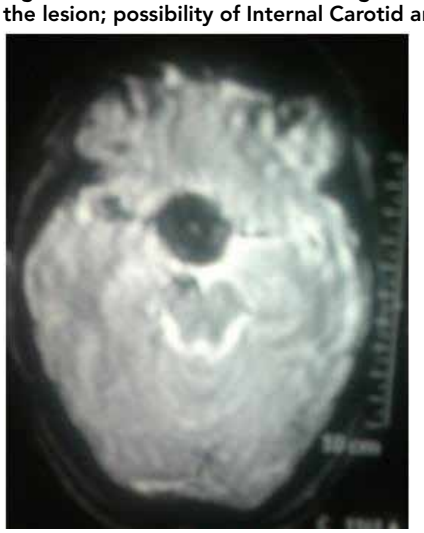

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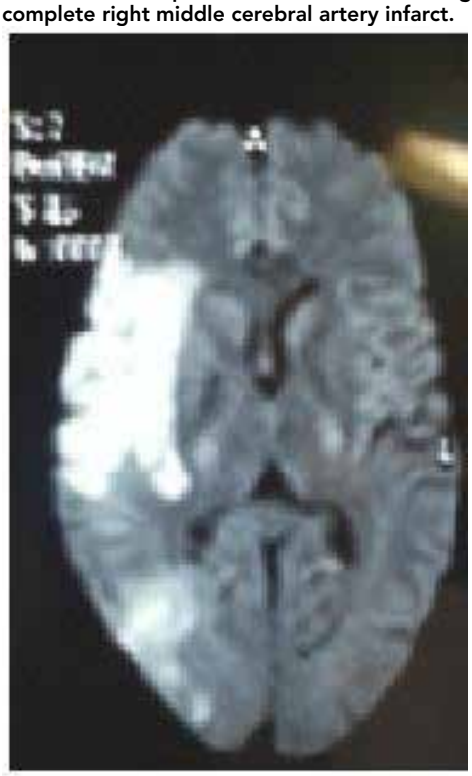
Fig-3.MR Angio: Obstruction of flow in supraclinoid internal carotid artery

Fig 4:Diffusion image showing involvement of subcortical area, internal capsule, lentiform nucleus, suggestive of complete right middle cerebral artery infarct.



Legend:

Fig-1a MRI Brain:-T1 weighted sagittal section showing a spherical mass in the sellar region possibility of pituitary macroadenoma with haemorrhage.

-1b MRI Brain T2 weighted sagittal section showing hyperintensity with speckled. hypointensities suggestive of a pituitary macroadenoma with haemorrhage.

Fig-2 Gradient reversal echo showing focus of blooming in the lesion; possibility of Internal Carotid artery aneurysm.

Fig-3.MR Angio: Obstruction of flow in supraclinoid internal carotid artery.

Fig 4:Diffusion image showing involvement of subcortical area, internal capsule, lentiform nucleus, suggestive of complete right middle cerebral artery infarct.

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