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Spontaneous Resolution of Pituitary Adenoma after Apoplexy: A Rare Case Report

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Abstract: ***Resolution of pituitary adenoma after pituitary apoplexy is rare. Even in people with a known pituitary tumor, only 0.6–10% experience apoplexy; the risk is higher in larger tumors. Ischemia of tumor and pituitary apoplexy are some causes of decrease in tumor size. In addition, for spontaneous resolution, lymphocytic hypophysitis should be considered in the differential diagnosis. Here, we discuss a 42-year-old male diagnosed with a non-functioning pituitary macroadenoma (NFPA) which had almost completely regressed spontaneously after eight months and only small residual pituitary tissue was noted.***

Keywords: Pituitary Adenoma, Apoplexy, Spontaneous Resolution

1. Introduction

Resolution of pituitary adenoma after pituitary apoplexy is rare.[(1)(2)] Even in people with a known pituitary tumor, only 0.6–10% experience apoplexy; the risk is higher in larger tumors.[(3)]The term pituitary apoplexy represents a complex series of clinical events occurring as a consequence of the fulminant expansion of a pituitary tumour by infarction, haemorrhage, or haemorrhagic infarction of the tumour and the adjacent pituitary tissue.[(4)] The sequelae to pituitary apoplexy are extremely varied, ranging from spontaneous recovery to fatal outcome, and include spontaneous remission of endocrinopathy, hypopituitarism, development of empty sella, and fracture of the dorsum sellae.[(4)] In many instances, the apoplectiform insult constitutes the first conclusive evidence that a pituitary tumour is present.[(5)]The most common initial symptoms are sudden headache, decrease sensorium , vomiting, associated with a rapidly worsening visual field defect or double visioncaused by compression of nerves surrounding the gland. Spontaneous remission of endocrinopathy following an apoplectic event is a known phenomenon in cases with hormonally active pituitary adenomas.[(6, 7, 9, 12)] However, spontaneous complete resolution of a nonfunctioning adenoma is a rare occurrence.[(8)]Even in people with a known pituitary tumor, only 0.6–10% experience apoplexy; the risk is higher in larger tumors.[(10)] Here in, we report a rare case of endocrinologically inactive pituitary macroadenoma which resolved in time following an apoplexic event without causing hypopituitarism.

2. Case Report

A 42 year old male patient initially presented to an outside institution emergency department with sudden headache, vomiting, and decrease of vision in left eye approx 8 months back. No history of decrease in sensorium, trauma or fever was present. Ophthalmologic examination revealed vision 6/9 on right eye and only perception of light (PL) +ve on left eye , with complete ptosis and opthalmoplegia in left eye. Once stabilized, patient plain Ct scan of head (figure 1), revealed a large mass in the sellar suprasellar region with widening of sella and destruction of sellar floor but no evidence of heamorrhage was seen. MRI brain revealed a 3.6x3.7x2.7 cm3 sellar suprasellar mass (figure 2,3,4). The mass was inseparable from normal pituitary tissue and extended inferiorly into sphenoid sinus and superiorly into the suprasellar cistern, with mass effect on the optic chiasm. The mass was hypointense on T1 and hyperintense on T2 with inhomogenous contrast enhancement without signal characteristics to suggest hemorrhage. His basal serum level of pituitary hormones were normal. During the course of time patients ptosis improved completely and has also got improvement in his vision in left eye. The patient was then referred to our institution for outpatient evaluation of his sellar/suprasellar lesion. On neurologic examination, he was found to have vision 6/24 vision in right eye and 6/36 in left eye; however, the rest of his neurologic examination was normal. Based on his laboratory values, MRI findings, and physical examination a nonfunctioning pituitary macroadenoma was the diagnosis. Given the degree of chiasmal compression and subjective visual field deficit, a microscopic endonasal transsphenoidal approach was indicated. As is our protocol, a planning MRI and computed tomography (CT) scan of the brain and skull base were ordered and performed. The repeat imaging was done eight months after his initial MRI. On repeat ct head, we found enlargement and destruction of sellar floor with no mass identified (figure 5). On mri brain (figure 6,7,8) reveals a small T2 hyperintense soft tissue mass (8*5)mm in the sellar region without any significant enhancement with widened sella. The optic chiasm had normalized in position. There was no signs of acute or chronic blood products within the sella. Patient was then discharged and kept for follow up.

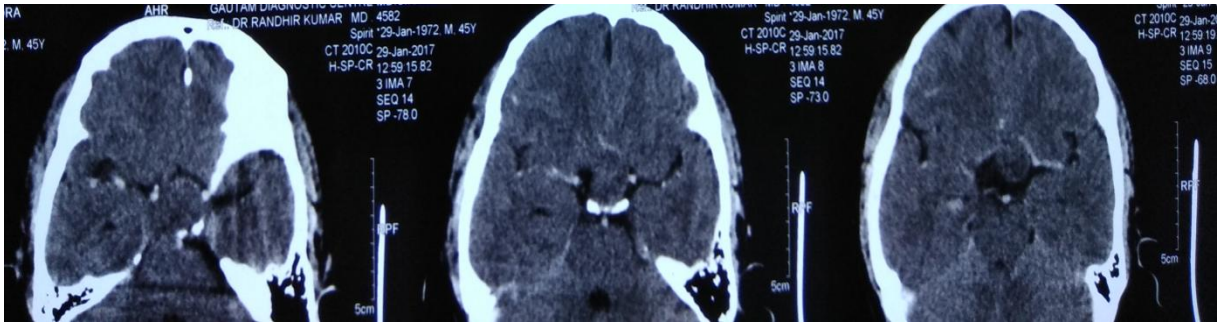

Figure 1: Ncct head: initial CT head showing isodense mass in sellar suprasellar region more in left side without any hyperdense signal (haemorrhage).

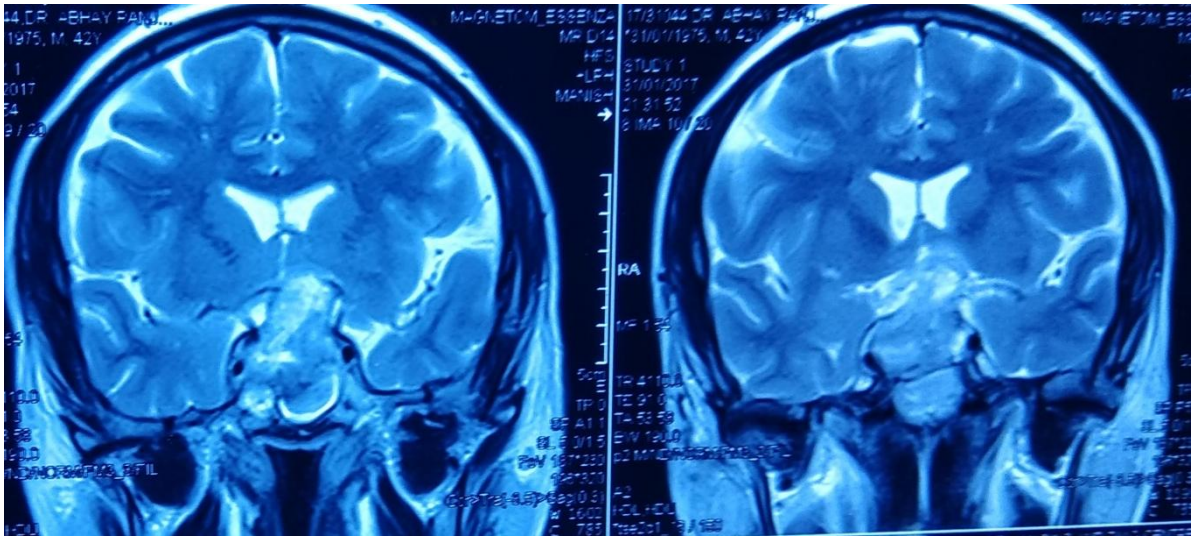

Figure 2: Initial MRI obtained at the time of diagnosis. T2 coronal image showing heterogenous mass in sellar and suprasellar region.

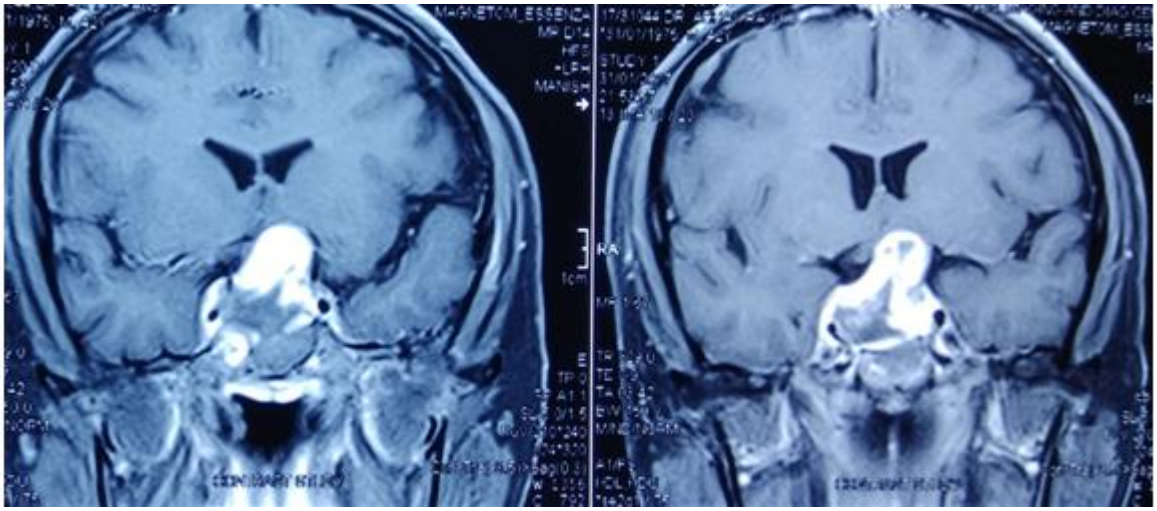

Figure 3: Contrast MRI brain coronal image showing heterogenous contrast enhancing sellar and suprasellar mass

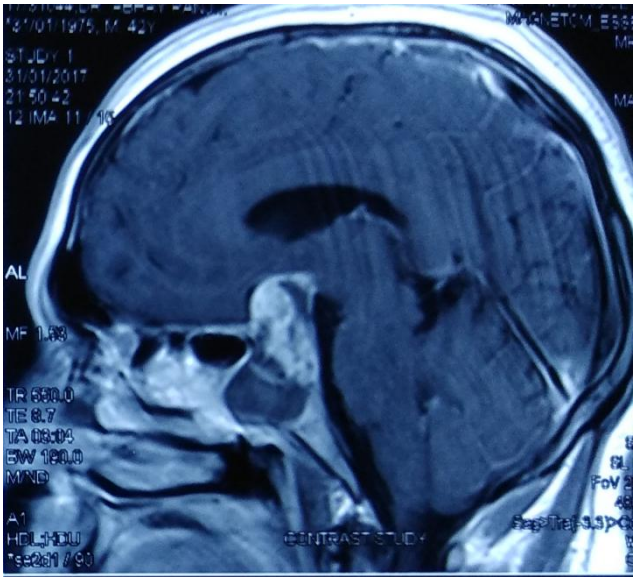

Figure 4: Contast mri saggital view showing heterogenous contrast enhancing mass in sellar and suprasellar region with widened sellar floor

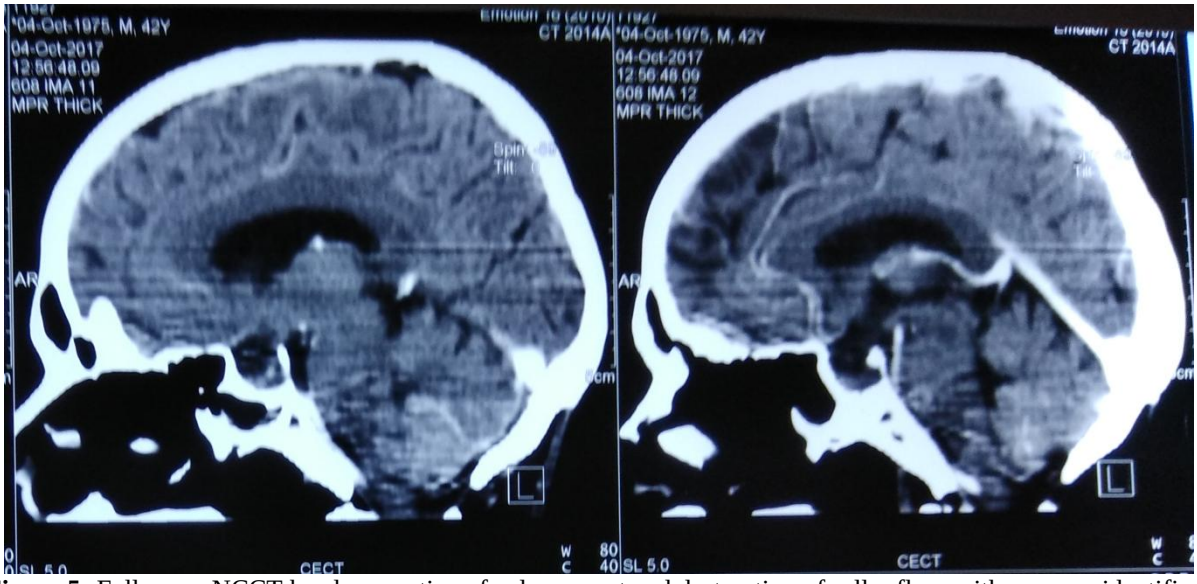

Figure 5: Follow up NCCT head suggestive of enlargement and destruction of sellar floor with no mass identified.

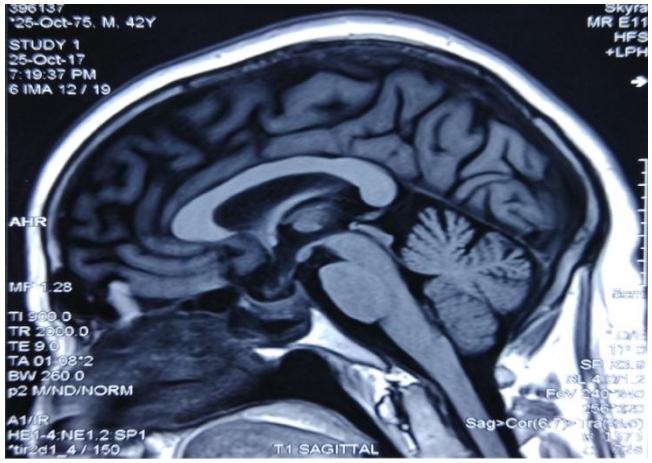

Figure 6: Follow up MRI obtained after 8 months of initial MRI. T1 MRI saggital view showing resolved sellar and suprasellar mass.

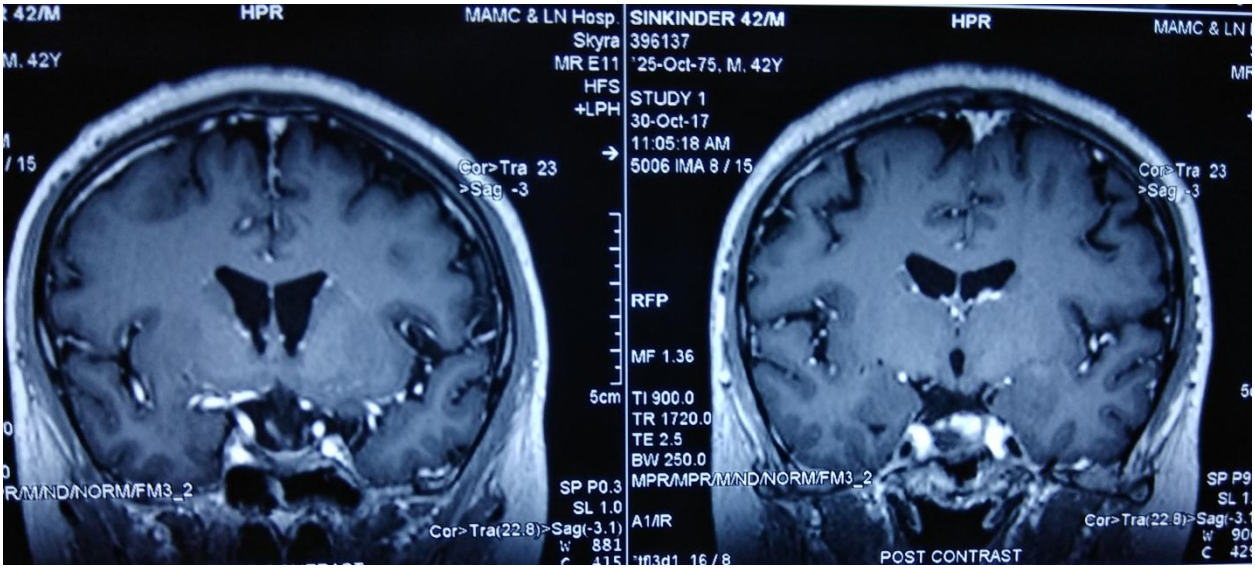

Figure 7: Contast MRI brain coronal view showing resolved suprasellar mass with small residual sellar mass.

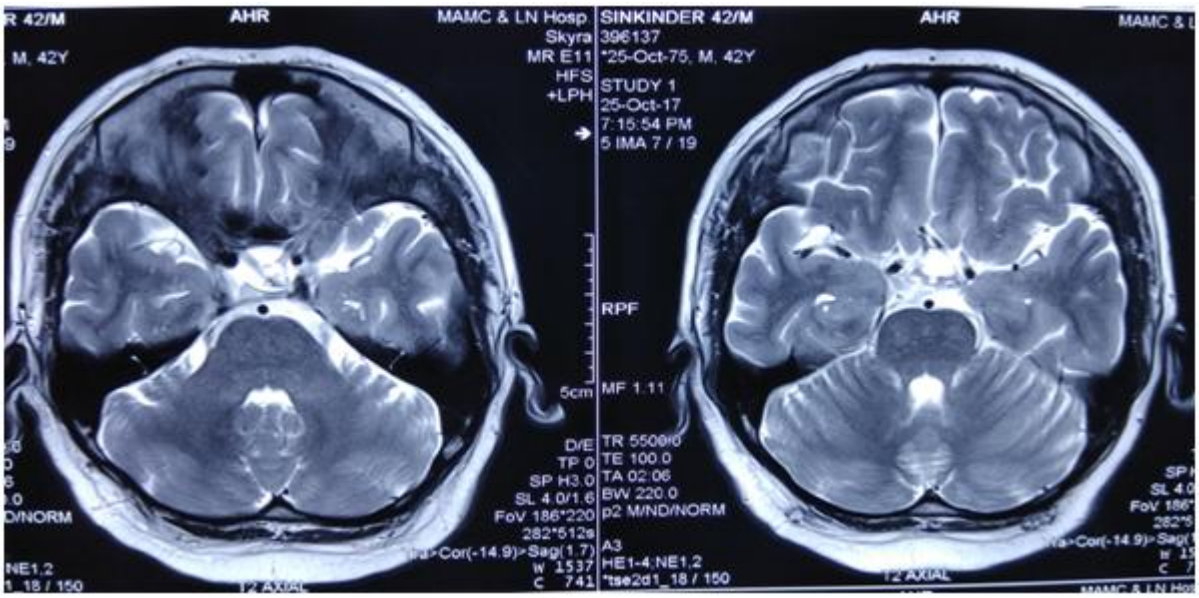

Figure 8: Follow up MRI T2 axial images showing resolved sellar suprasella

3. Discussion

We present a patient with an incidentally discovered NFPA, which spontaneously regressed over the course of approximately eight months. The patient had an hormonal profile and MRI with imaging characteristics classic for a NFPA and documented visual field defects. A routine preoperative planning MRI demonstrated complete disappearance of the NFPA.

Some studies were done for finding the factors that could predict tumor growth or regression. Igarashi[11] classified MRI findings into cystic and solid type and reported that cystic type frequently will shrink or even disappear spontaneously but solid type will often grow. In our patient, tumor type was solid but regressed in short time. A meta-analysis by Dekker, et al.[12] suggests that approximately 10% of nonfunctioning pituitary macroadenomas show some degree of shrinkage with follow-up imaging, however only one reported case describing complete spontaneous regression of pituitary macroadenomas has been reported in the literature. Bahar, et al.[13] discussed the case of a 46-year-old woman who presented with a pituitary macroadenoma that spontaneously regressed. Unlike our patient, whose NFPA was incidentally found due to acute onset of symptoms of pituitary apoplexy, their patient presented with amenorrhea, weight loss, and malaise. Her macroadenoma, measuring 1.8 x 1.5 x 1.5 cm was also non-functioning; however, her endocrine workup revealed pan hypopituitarism whereas our patient had a normal endocrine workup. Finally, unlike our patient, she had no visual deficits. Despite these differences, the patient in the study by Bahar, et al. also had an NFPA with compression on the optic chiasm that completely spontaneously regressed by the three-month follow-up MRI with no signs of acute blood or pituitary apoplexy on this follow-up imaging. Maltby et al.[14] described spontaneous cure of pituitary adenoma after apoplexy with csf sella syndrome in a 11 yr old girl. She presented with sudden onset vomiting, moderate headaches, lethargy, weight loss, and tall stature above her mid-parental height, unlike this girl our patient did not develop empty sella and his age is 42years.

Amr morsi et al.[15] reported a case of pituitary apoplexy after leuprolide administration for carcinoma of the prostate , suggesting leuprolide to be a possible cause of apoplexy. Pinto et al·[16] described a case with apoplexy in a prolactin secreting adenoma causing resolution of adenoma leading to hypopituitarism. Although most cases occur spontaneously, there are some apparent predisposing factors such as bromocriptine therapy[17] arteriography[18] and gadolinium administration[19] . It has also beenreported after pituitary function tests.[20] Our patient had a clinically non-functioning adenoma which was recognized only after he presented as an apoplexy. The present case never had any hypofunction of pituitary, following resolution of adenoma, and the apoplexy was the only manifestation of the tumour.

The differential for sellar and suprasellar lesions is broad and includes not only neoplastic entities such as pituitary adenoma, metastases, Rathke's cleft cyst, craniopharyngioma, and meningioma but also vascular lesions and inflammatory lesions such as sarcoid and autoimmune hypophysitis.[21] One must certainly consider this broad differential when evaluating this case. While the clinical, endocrinologic, and imaging characteristics supported a diagnosis of NFPA for our patient, there is alternative diagnosis of autoimmune hypophysitis that warrant discussion. Autoimmune hypophysitis[22] is a disorder with a strong temporal relationship to pregnancy where there is lymphocytic infiltration of the pituitary gland and infundibulum frequently leading to hypopituitarism, diabetes insipidus or hyperprolactinemia.[23] Our patient did not match the classical clinical picture of autoimmune hypophysitis (young peripartum females presenting with pituitary dysfunction).

This report adds to the spontaneous regression of adenoma following an apoplexy in an endocrinologically inactive adenoma without producing hypopituitarism.

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