Primary pituitary tubercular abscess mimicking as pituitary adenoma

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ABSTRACT

Tubercular abscess of the pituitary fossa is rare and may lead to diagnostic uncertainty in a patient with absence of tuberculosis elsewhere in the body. We present a rare case report of a young lady who presented with sellar and suprasellar cystic mass. She was diagnosed as a case of pituitary macroadenoma and was intraoperatively found to harbor pus in the lesion. She did not have any symptoms of infection. The case underlines the importance of considering such a possibility in the differential diagnosis of cystic sellar lesions and further diagnostic tests should be done for confirmation and treatment of this rare and potential life threatening illness.

Key words: Pituitary abscess, primary tubercular pituitary abscess, sellar tuberculosis, tubercular abscess, tuberculoma

INTRODUCTION

Primary pituitary abscess is a rare disease.^[1] The radiological presence of cystic lesion in the sella usually leads to consideration the diagnosis of pituitary adenoma or occasionally rathke's cyst and craniopharyngioma. The suspicion of abscess may not be entertained in the absence of constitutional symptoms and the differential diagnosis of a tubercular abscess may not be entertained even in endemic regions of the world. We discuss such a case in which diagnosis of tubercular abscess was suspected after surgery and stress the need of considering the diagnosis as a possible pathology for cystic lesions of the pituitary.

CASE REPORT

A 27-year-old lady presented to our hospital with complaints of throbbing headache of three months duration. The

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headache was bifrontal in location, severe in intensity and was associated with occasional episodes of vomiting. There was no history of visual disturbance or symptoms suggestive of pituitary endocrinopathy. She was married and had a 10-months-old daughter. The menstrual cycle was regular. There were no constitutional symptoms of fever, anorexia, weight loss or other systemic illness. Her endocrine evaluation by an endocrinologist revealed decreased thyroid stimulating hormone of 0.02 µIU/ml (normal range 0.27-4.20 μ IU/ml) and moderately raised prolactin level of 70 ng/ml (normal range 4.79-23.3 ng/ ml). The detailed thyroid profile included T3 1.3 ng/dl (normal range 60-180 ng/dl) and T4 14.9 μ g/dl (normal range 5.5-12.5 μ g/dl). Growth hormone level was 0.32 ng/ ml (normal range 0-18 ng/ml). Serum cortisol at fasting 8 AM was 611 nmol/l (normal range 171-536 nmol/l). Serum LH was 1.8 mIU/ml (normal range 2.4-12-6 mIU/ ml) and FSH was 6.3 mIU/ml (normal range 3.5-12.5 mIU/ml). Serum ACTH level was 19.5 pg/ml (9-52 pg.ml). She was put on replacement therapy with thyroxine (100 µgm/day). She also received capergoline for control of tumor but the symptoms had worsened over the period of time. General physical examination was unremarkable. Her blood pressure was 110/70 mg of Hg and there was no postural variation. Visual perimetry examination with Goldman's applicator was normal. MRI examination of the brain revealed a cystic sellar lesion with suprasellar

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extension with dimension of $15 \times 21 \times 23$ cm [Figure 1a]. T2 weighted image showed hyper intense signal in the cyst cavity [Figure 1b]. The lesion showed ring enhancement with gadolinium [Figures 2a and b]. Posterior pituitary spot was well visualized. Her hematological investigations were within normal limits. It was decided to remove the lesion through trans-sphenoidal route. During surgery, the sphenoid sinus was well pneumatized. The cyst wall was entered after incising the duramater. The cyst cavity contained creamy pus. After evacuation of about 4 ml of pus, arachnoid pulsations were well visualized. There was no evidence of pituitary tumor. The gram stain and the Zeihl-Neelson stain for acid fast bacilli (AFB) were negative. Histopathological examination of the pus showed presence of pus cells and macrophages. There was no evidence of tumor. She was put on hydrocortisone in the peri-operative period and was stopped on post operative day 5 when cortisol level was normal (846 nmol/l). Post



Figure 1: T1 weighted (a) sagittal and T2 weighted (b) axial magnetic resonance imaging images of the brain showing the cystic lesion in the sellar and suprasellar region

operative erythrocyte sedimentation rate was 90 mm in the first hour and Mantoux test was strongly positive (17 mm). Keeping the clinical picture, the pituitary abscess was considered to be tubercular in nature. The bacterial culture on Lowenstein-Jensen medium at six weeks showed mycobacterial colonies. She was treated with anti tubercular therapy with four drugs which included rifampicin (10mg/ kg/day), isoniazid (5-10mg/kg/day), ethambutol (15 mg/ kg/day) and pyrazinamide (20-35 mg/kg/day). Pyridoxine 20 mg/day was given along with isoniazid. No steroids were used in the treatment regime. Post operative imaging did not reveal any residual cyst [Figure 3]. She was treated with thyroxin replacement (100µg/day) and repeat thyroidal function study at three months revealed normal hormonal values. Thyroxine replacement was subsequently reduced to 50 µg/day. Pituitary hormone status (TSH and prolactin) has recovered to normal post anti tubercular therapy of six months. Patient has been doing well at 12 months follow up.

DISCUSSION

Primary pituitary abscess is a rare disease.^[1] A literature search for sellar and suprasellar tuberculoma reveals the presence of about 54 cases.^[2] However, search for pituitary tubercular abscess or sellar tubercular abscess yielded two cases.^[3,4] The need to distinguish between tuberculoma, bacterial abscess and tubercular abscess assume importance in terms of reaching a clinical and radiological diagnosis and direct their management.

Dutta *et al.*, reported a 13-year-old boy with meningitis, deteriorating vision and panhypopituitarism.^[3] MRI of brain revealed a solid-cystic lesion with suprasellar extension. He underwent stereotactic aspiration and biopsy which yielded acid fast bacilli. The patient was treated with



Figure 2: T1 weighted contrast enhanced magnetic resonance imaging images of the lesion in sagittal (a) and coronal (b) sections showing ring-like enhancement of the abscess wall



Figure 3: Post-operative contrast enhanced CT scan of the brain showing complete decompression of the sellar cystic lesion

antitubercular therapy and steroids. Patient responded well to the treatment in the initial six months but expired during later period.

Behari *et al.*, reported a patient who presented with holocranial headache of two-years duration with features of hypogonadism.^[1] MRI revealed a sellar lesion with enhancing rim of solid tissue and necrotic core. A diagnosis of pituitary adenoma was made and the patient underwent a sub labial trans-sphenoidal decompression which yielded yellowish pus. Histology revealed the presence of tuberculoma. However, the pus did not reveal any acid fast bacilli on Ziehl-Neelsen staining. The pus culture showed growth of mycobacterial colonies. The patient received anti tubercular treatment and became asymptomatic in follow-up.

While the tubercular abscess of the brain has been reported, primary tubercular abscess of the pituitary is extremely rare. The literature review yields only few cases of primary tubercular lesion of the pituitary and the diagnosis was reached only after surgery.^[2,3] Our patient was a young lady who had progressive headache with hypothyroidism and hyper-prolactinemia. She had a primary pituitary tubercular abscess and was treated with trans-sphenoidal drainage and decompression of the cyst. The symptoms recovered during the follow up.

Of all the patients with pituitary tubercular abscess/ tuberculoma, headache is the most common symptom followed by visual and endocrine abnormalities.^[4] Headache may be bifrontal, retro-orbital or over vertex. The headache of tubercular abscess may be disproportionate secondary to stretching of diaphragma sellae. The other causes may be due to the presence of meningitis and hydrocephalous.^[1,2]

About 30-50% of patients may have anterior pituitary hormone deficiencies or central diabetes insipidus at the onset.^[5] The earliest manifestation is growth hormone (GH) deficiency, followed by gonadotropin (LH/FSH) and ACTH deficiency.^[5] However, in cases with tuberculoma of the pituitary gland, the most frequent deficiencies encountered are ACTH, TSH and hyperprolactinemia.^[2,3] The high incidence of endocrinopathy mandates hormonal evaluation of the pituitary gland in all patients prior to surgery.^[2,3]

There may be few distinct clinical and radiological features to distinguish tubercular abscess from tuberculoma and pyogenic abscess. The pyogenic abscess can develop in an otherwise normal pituitary gland or due to hematological seeding or by direct extension of adjacent infection, either in cerebrospinal fluid (CSF) or sphenoid sinus.^[1,4] They may also occur as a complication of cavernous sinus thrombosis. Other risk factors include underlying immunocompromised condition, previous pituitary surgery or irradiation of pituitary gland.^[2] The central nervous system tuberculosis occurs due to hematogenous spread from else where in the body. Tubercular meningitis can occur via lymphatic spread. Tubercular bacilli are immobilized in the end arteries which lead to formation of sub meningeal tubercular foci. In tuberculoma the bacilli get lodged in brain with rich blood supply. It evokes a secondary reaction which leads to formation of thick capsule. In rare cases of central caseation, liquefaction and formation of abscess occurs. Radiologically and clinically they may behave similar to pyogenic abscess. Tubercular abscess is characterized by an encapsulated collection of pus containing viable tubercle bacilli and without typical tubercular granuloma and epithelioid cells.^[6]

Both tubercular and pyogenic abscess can present with cystic mass in the sellar region in the absence of any systemic manifestation. Hematological parameters (leucocyte count, ESR) may not be helpful and Mantoux test may be falsely positive in patients living in endemic area.^[4] Radiologically, pituitary tumors would account for 91% of all sellar tumors.^[7] The differential diagnosis of a cystic lesion in sella includes adenoma, carcinoma, arachnoid cyst, abscess, rathke's cleft cyst, craniopharyngioma, colloid cyst and metastasis.^[7] The classical description of multiple coalescing contrast-enhancing lesion points to tuberculoma. However, the findings of a single cystic ring enhancing mass lesion may not exclude pathology like neurocysticercosis and abscess.^[2] The enhancing and beaded appearance of the pituitary stalk is suggestive of infiltrative rather than infective cause. The normal pituitary may be seen as a rim of tissue surrounding an adenoma, but usually the rim is thicker when compared to that of an abscess.^[1] The confirmation of the diagnosis based on clinical and radiological findings may be extremely difficult because of similar presentation of all these lesions. The need for surgical intervention may arise to reach a pathological diagnosis in the absence of other clinical features and to alleviate the raised intracranial pressure. Trans-sphenoidal decompression provides the most effective means to reverse the symptoms of visual deficits and headache. The recovery of the endocrine abnormalities may be partial and patients must be followed up closely during the treatment.

CONCLUSION

Primary pituitary tubercular abscess is a rare disease and can be life threatening. The presence of a cystic lesion with contrast enhancing wall in the sella should lead to a suspicion of an abscess even if the patient has no systemic symptoms of any infection. Histopathological and microbial examinations confirm the diagnosis but may be inconclusive. A high clinical suspicion, timely intervention and anti-tubercular therapy may help in alleviation of symptoms of visual disturbances, headache and hormonal abnormality.

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