

A Case of Pituitary Macroadenoma with Apoplexy Following Recurrent Pyelonephritis

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Abstract: Pituitary macro adenoma is a rare tumour with varied presentation. It can present acutely due to apoplexy. Here we present a case of 58 years old diabetic male with left pyelonephritis and septicemia who had acute onset of headache, vomiting, ophthalmoplegia and meningism. On clinical suspicion of septic encephalopathy, CSF analysis showed no significant abnormality. MRI Brain showed Pituitary Macro adenoma and intra lesional hemorrhage with intraventricular, subarachnoid hemorrhage and obstructive hydrocephalus. Serum cortisol levels reduced. Our objective is to highlight the importance of pituitary apoplexy that mimics as infective meningitis by presenting a representative case.

Keywords: Apoplexy, pituitary macro adenoma, pyelonephritis, steroids.

INTRODUCTION

Pituitary tumours are rare with an incidence of only 14.4% in around 3500 autopsied patients and 22.5% among 200 patients with imaging studies (overall 16.7%). Among these, macro adenomas are seen only 1 in 600 patients [1]. Many a time, they present as “incidentalomas” on imaging. Pituitary tumour apoplexy is a rare neuro endocrine syndrome resulting from hemorrhage or infarction of a pre existing pituitary adenoma. Apoplexy in pituitary tumour is uncommon and under diagnosed entity. The calculated prevalence from epidemiological surveys of operated patients suggests prevalence of 0.6-12.8 %. [1, 2] Usually, apoplexy presents as neurological and endocrinological signs and symptoms. Headache due to raised intracranial pressure is the commonest complaint (63-100% cases), followed by visual deficits (40-100%), cranial nerve palsies, and vomiting. [1] Here we

report a case of pituitary apoplexy following sepsis and recurrent Pyelonephritis.

CASE REPORT

A 58 years old male who is a known case of type II diabetes mellitus for the past eight years on Oral hypoglycaemic drugs and insulin with previous history of Right ureteric calculus and right Pyelonephritis, treated with surgical drainage and stenting 6 months before was admitted for fever, abdominal pain, for four days. When received in critical care unit patient, febrile, GCS-15 /15, BP-130/80mmHg, HR-120/mt, blood sugar -238 mgs%. CT abdomen showed enlarged left kidney (12 x 6.7cms) with perinephric fat stranding and perinephric fluid suggestive of left pyelonephritis, Right kidney measures 7 x 3.3cms, decreased in size with loss of corticomedullary differentiation (Figure 1).

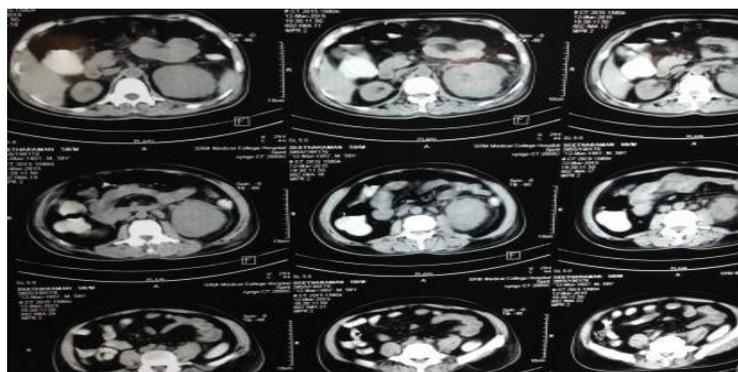


Fig 1: CT abdomen showing enlarged left kidney with perinephric fluid

Urine culture showed heavy growth of enterococcus. After two days patient developed intense head ache and vomiting, neurologically, he had complete unilateral ptosis of left eyelid, left III, IV, VI cranial nerve palsies with sluggishly reacting pupils and neck stiffness. His visual acuity was preserved; visual field examination and fundus examination was normal.

Other cranial Nerves examination and rest of the neurological examination were essentially normal. CT brain showed hypo dense lesions anterior to midbrain with subarachnoid hemorrhage noted adjacent to lesions extending to left lateral ventricle, rest of the brain parenchyma normal. (Figure 2)

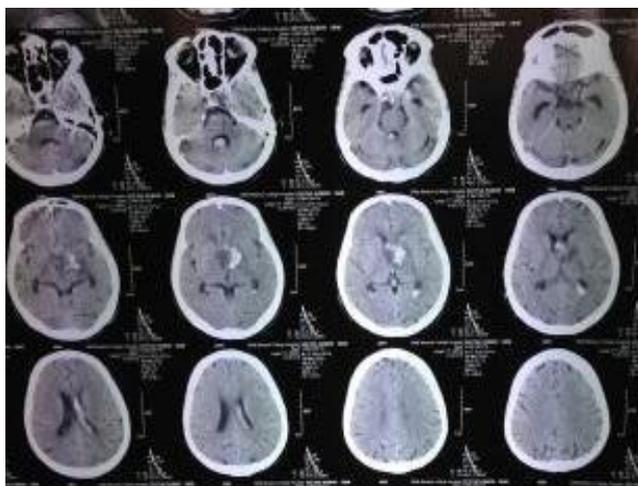


Fig 2: CT Brain showing hypodense lesion anterior to midbrain with hemorrhage in lateral ventricle

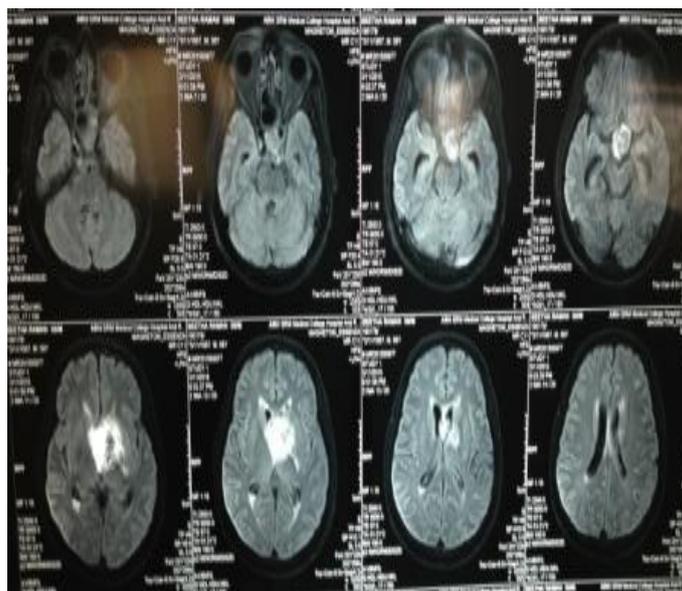


Fig 3: MRI brain coronal view showing pituitary hemorrhage

MRI brain showing iso - hyper intense sella and suprasellar mass lesion of size 3.7 x 2cm with blooming in GRE sequence around the mass lesions(Figure 3), III, IV and lateral ventricles suggestive of hemorrhage (Figure 4 : MRI Brain sagittal view showing pituitary hemorrhage with subarachnoid hemorrhage , compression 3rd ventricle

and dilatation of lateral and 4th ventricle noted., Figure 5: MRI Brain sagittal view showing pituitary macro adenoma with hemorrhage) Diagnosis of Sella – suprasellar mass lesion - Pituitary Macro adenoma with hemorrhage with intraventricular hemorrhage, subarachnoid hemorrhage and obstructive hydrocephalus was made.

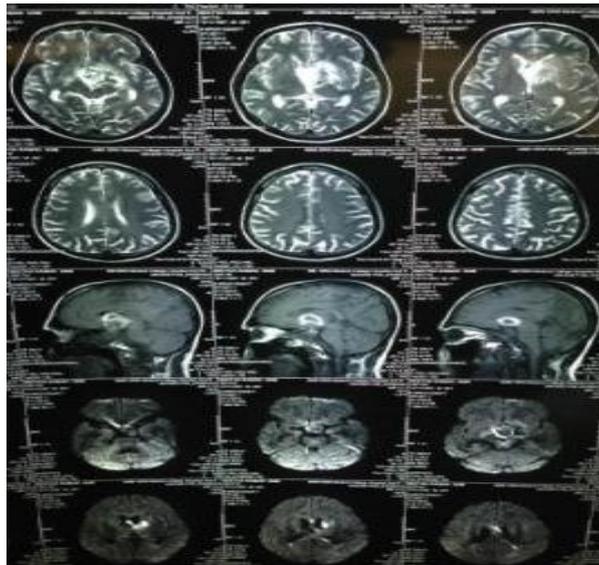


Fig 4: MRI Brain sagittal view with subarachnoid hemorrhage Compression 3rd ventricle and dilatation of lateral and 4th ventricle



Fig 5: MRI Brain sagittal view showing pituitary macroadenoma with hemorrhage

MRA – no communication with cerebral arteries and circle of wills and MRV normal. Serum Cortisol, TSH, FSH, LH were reduced significantly with the following parameters. Serum Cortisol 8:00 AM 1.79 (normal 6.7–22.6 (µg/dL) ,4 PM 1.72 (normal <10 µg/dL),TSH- 0.251 (normal 0.34–5.60 (µIU/mL) Adreno cortico tropic hormone (ACTH) <5.0 (normal <46 (pg/mL), Prolactin 3.83 (normal - 2.6–13.1 (ng/mL), Luteinizing hormone (LH) <0.2 (normal 1.2–8.6 (mIU/mL), Follicle-stimulating hormone (FSH) 1.2 (normal 1.27–19.26 (mIU/ML).The diagnosis of pituitary apoplexy confirmed with the above parameters. ABG showed metabolic acidosis with lactate 8.4. Investigation showed Hemoglobin - 9.7gms%, Total count -32400 cells/mm³ , Platelets count - 80000 cells/mm³ ,Prothrombin time prolonged (30 Sec),INR -4.5, Urea-150 mgs%, Creatinine-4.9 mgs%, Sodium-124 meq/l, Potassium-5.4 meq/l, SGOT-201 U/l, SGPT- 265 U/l, Albumin- 2.1mgs%.

ECG and ECHO were normal. Patient was treated with appropriate sensitive antibiotics, ionotropes, intramuscular hydrocortisone 50 mg 6th hourly, Hemo dialysis, Ventilator support and other supportive treatment measures. Patient worsened with severe acidosis, shock and deteriorating neurological status unfit for any surgical procedures. Despite our intensive treatment measures, patient died due to septic shock and multiorgan failure.

DISCUSSION

Pituitary apoplexy is an endocrine emergency [3]. Its diagnosis rests on the presenting clinical features as well as imaging findings. Typical manifestations include severe headache, visual impairment and ophthalmoplegia. Altered consciousness, hormonal dysfunction and meningeal irritation can also occur .Pituitary imaging (CT or MRI) usually shows signs of intrapituitary or intra adenoma

hemorrhage, stalk deviation, and compression of pituitary tissue [4]. Our patient presented with severe headache, altered consciousness, high fever and neck stiffness, with pyelonephritis, which led to the clinical diagnosis of acute bacterial meningo encephalitis. However, antibiotic treatment did not result in any clinical improvement.

Pituitary apoplexy was recognized only after brain MRI revealed a pituitary tumour with recent hemorrhage and laboratory tests showed secondary adrenal insufficiency. The size of the adenoma appears to be the major risk factor for apoplexy. It is thought to occur when large tumours outgrow their blood supply and develop areas of ischemic necrosis, acute tumour swelling and bleeding [5].

It is also associated with head trauma, radiation therapy, anticoagulation therapy, estrogen or bromocriptine therapy, sudden changes in arterial blood pressure or intracranial pressure, dynamic testing of pituitary function, diabetes and hypertension [6]. It is probably related to elevated intrasellar pressure and reduced blood perfusion from pituitary vessels [7]. There are no case reports pituitary apoplexies following septicemia. Since our patient had diabetes with sepsis we believe that septicemia induced the pituitary apoplexy.

CONCLUSION

Patients with pituitary apoplexies that are hemodynamically unstable or have altered consciousness or have decreased visual acuity and severe visual field defects should be commenced on empirical steroid therapy. In adults, hydrocortisone 100-200 mg as an intravenous bolus is appropriate, followed either by 2-4 mg per hour by continuous intravenous infusion or by 50-100 mg six hourly by intramuscular injection. Also, patients with serum cortisol less than 550 nmol/l are candidates for empirical steroid therapy [8]. As our patient had low serum cortisol levels, steroid was started immediately. The rarity of pituitary apoplexy renders it a difficult subject for audit; hence, there are no evidence-based standards of optimum care for such patients. Indications for surgery are severe neuroophthalmic signs such as severely reduced visual acuity, severe and persistent or deteriorating visual field defects, or deteriorating level of consciousness [8].

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