Bangladesh Journal of Medicine (BJM)
ISSN : 1023 - 1986
eISSN : 2408 - 8366

CASE REPORT

A PERPLEXING CASE OF PITUITARY APOPLEXY MASQUERADING AS MENINGOENCEPHALITIS

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Abstract:

Pituitary apoplexy (PA) is extremely rare in children and adolescents. It is a life-threatening condition usually results from sudden hemorrhage or infarction induced swelling in a pituitary adenoma. The clinical manifestations of PA include severe headaches, impaired consciousness, fever, visual disturbance, and variable ocular paresis. Therefore, the presence of meningeal irritation may lead to misdiagnosis as a case of meningoencephalitis or spontaneous subarachnoid hemorrhage, and delay in the proper management of the disease. We report a case of 17-year-old pubertal boy who developed sudden severe headache, vomiting, slurring of speech and abnormal behaviour followed by impaired sensorium with fever. The patient who was initially diagnosed with meningoencephalitis (ME) based on clinical presentation and cerebrospinal fluid (CSF) analysis, which was consistent with bacterial meningitis. MRI of brain was performed, confirming a pituitary macroadenoma with hemorrhage and ischemic changes in both basal ganglia and pons. A complete analysis of the pituitary hormones revealed decreased cortisol and thyroid hormone level and hyperprolactinemia and he was subsequently started on placement corticosteroid and L-thyroxine therapy and cabergoline. After 14/ days of antimicrobial therapy with ceftriaxone and ampicillin, the patient improved and was discharged on hormone replacement therapy and surgical advised. Hereby, we report our case with a review of literatures.

Keywords: *Apoplexy, neurosurgery, panhypopituitarism, pituitary.*

Received: 22.10.2021 *Accepted: 09.11.2021*
DOI: https://doi.org/10.3329/bjm.v33i1.56795

Citation: *Rahman A, et al. A Perplexing Case of Pituitary Apoplexy Masquerading as Meningoencephalitis.. Bangladesh J Medicine 2022; 33: 85-88.*

Introduction:

Pituitary apoplexy is a rare, but life-threatening disorder that usually results from intratumoral hemorrhage or infarction in pituitary adenomas.[1] The clinical manifestations of PA include severe headaches, impaired consciousness, fever, visual disturbance, and ophthalmoplegia.[2] However, signs of meningeal irritation are not typical findings.[3] So the presence of meningeal irritation may lead to misdiagnosis as a case of meningoencephalitis or spontaneous subarachnoid hemorrhage, and delay in the proper management of the disease. Hereby,

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we report a case of pituitary apoplexy with an initial presentation mimicking meningoencephalitis. We also highlight the importance of careful clinical and radiological correlation, which led to a timely diagnosis and treatment in our patient.

Case report:

A 17- year- old pubertal boy with no past medical history, visited the emergency unit of in a tertiary care hospital with a headache, slurring of speech, dysphagia, abnormal behaviour followed by impaired sensorium with fever. On admission, he was confused with Glasgow Coma Scale of 11 (E4, V3, M4), fever (temperature 38. 5/ °C) and neck stiffness, but without neurological deficit including cranial nerve palsies. His skin was well perfused with no rashes, cardiovascular, respiratory and abdominal examinations were normal. MRI of brain (Figure: 1) showed a hyperintense pituitary macroadenoma with hemorrhage and ischemic changes in both basal ganglia & pons.

Lumbar puncture was done and showed turbid cerebrospinal fluid (CSF) white cell count 4500/ mm3 (90%polymorph, 10% lymphocytes), red cell count 30/mm3, protein 0.8/ g/L and glucose 0.22/ g/L; no organisms on gram stain. He was started on intravenous ceftriaxone at 4/ g daily and ampicillin for bacterial meningitis. Over the next 5/ days his temperature regularly spiked to 38.5–39/ °C. A complete analysis of the pituitary hormones (Table:1) revealed decreased serum cortisol level (Cortisol - 21.42ng/ml at 8:00 A.M.) and thyroid hormone level (TSH- 0.201 ulU/ml, FT4 0.94ng/dl, ACTH- 0.5 pg/ml, Growth hormone: 2.30 ng/ml, LH-2.30mU/ml, FSH-6.54mIU/ml and Prolactin: 190.44 ng/ml level) and Serum Electrolytes- normal(143 mmol/L K+ -4.03 mmol/l). Those results consisted with revealed decreased cortisol level and hypothyroidism with hyperprolactinemia. His course and investigations were consistent with pituitary apoplexy, and he was subsequently started on replacement corticoid (dexamethasone intravenously, 10 mg per 8 hours) and L-thyroxine and cabergoline therapy with good outcome. The patient was discharged on day 24 without significant neurologic deficits and was referred to neurosurgery department & advised for surgical decompression.

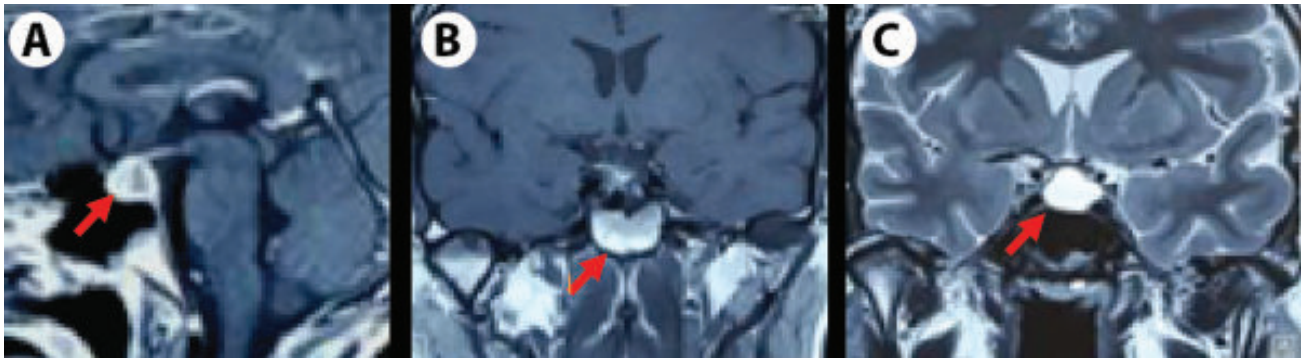

Fig.-1: *Initial sellar magnetic resonance imaging (MRI) findings. T2-weighted axial image shows a sellar mass with heterogenous internal high intensity signals (A). Non-contrast T1-weighted sagittal and coronal images show a pituitary mass extending suprasellar area without typical findings compatible with intralesional hemorrhage (B and C)*

Table-I

Complete analysis of the pituitary hormones

Parameter	At presentation	Normal Values
GH	2.30 ng /ml	<5 ng/ml
RBS	5.07mmol/l	3.67-7.80 mmol/l
LH	2.03 mIU/L	2-12 mIU/L
FSH	6.54 mIU/L	<2 mIU/L
Prolactin	190.44ng/ml	2.10-17.70 ng/ml
FT4	0.94 ng/dl	0.71-1.85 ng/dl
S TSH	0.201 uIU/ml	0.47-5.01 uIU/ml
ACTH	0.5pg/ml	5-46 pg/ml
Cortisol(morning)	21.42 ng/ml	72.6-322.8ng/ml
Na+	143 mmol/l	135-145 mmol/L
K+	4.03 mmol/L	3.5-5.0 mmol/L

Discussion:

Pituitary macroadenomas represent around 10% of all primary brain tumors [4]. Pituitary apoplexy was first described in 1950 by Brougham et al. [5]. The frequency of pituitary apoplexy reported is variable which ranges from 0.6% to 27.7% [6]. There are only 25 cases that have identifiable prepping factors. Head trauma, unforeseen changes in arterial or intracranial pressure, cabergolin administration or pullout, anticoagulant remedy or bleeding diseases or cardiac bypass, diabetes, and postpartum hemorrhage has been cited as inciting apoplectic occurrences [7]. Still, utmost pituitary apoplexies are considered to idiopathic etiologies. A vast maturity (60–80%) of pituitary tumour apoplexy occurs in else asymptomatic cases with secretory adenomas being the most common underpinning tumours, [8.] Leakage of necrotic cellular debris into the subarachnoid space following pituitary apoplexy may lead to chemical meningitis with resultant triad of fever, meningism and photophobia [9]. Such a situation would be delicate to label as definite bacterial meningitis unless the CSF Gram staining is positive, CSF antigen discovery tests are positive or CSF culture grow the pathological organism. Numerous times this criterion isn't completely met in the clinical setting. Still, the presence of particularly high CSF leukocyte count (2.775 × 109/ L) with 80 neutrophils, low CSF to serum glucose rate (0.4), and the fact that the first blood culture grew an implicit bacterial pathogen, all explosively point towards the contemporaneous circumstance of bacterial meningoencephalitis in our case and can be labeled as probable bacterial meningoencephalitis [10]. CSF study may not be helpful in distinguishing pituitary apoplexy from meningoencephalitis, since chemical meningitis may have rebounded from leakage of blood or necrotic towel into the subarachnoid space [8]. In our case, the sugar content of the primary CSF analysis was not low enough to suggest bacterial meningitis and no microorganism was detected. Thus, we were ready to conclude that the abnormal findings of CSF examination were attributed to chemical meningitis. [11].

Neuroimaging studies demonstrate atypical findings not compatible with a pituitary apoplexy in the veritably early stages, and meningeal vexation signs may be the presenting symptoms [12]. Brain MRI is presently the foremost accurate tool for the opinion of pituitary excrescence apoplexy, the study of the conterminous structures which will be involved, and also the discovery of the intrasellar hemorrhage. MRI is superior to CT imaging for delineating pituitary hemorrhage and hence is the procedure of choice [6]. Cerebral infarction associated with PA has been reported uncommonly. In a recent literature review of cerebral infarction associated with PA, the pathogenesis was reported as being due to direct intracranial vascular contraction or vasospasm, either independently or together[13]. In our case, brain MRI revealed a slightly miscellaneous lesion intrasellar heterogeneous lesion iso/ hyperintense T2 suggesting intratumoral pituitary haemorrhage and ischemic changes in both basal ganglia & pons.

Conclusion:

This case demonstrates the occurrence of pituitary apoplexy presenting as meningoencephalitis. It reinforces the importance of including pituitary apoplexy in the differential diagnosis of infectious meningoencephalitis and cerebral infraction in patients presenting with acute headaches associated with pyrexia, meningism and altered sensorium. In such cases, early evaluation and treatment may decrease recurrences and reduce or avoid the morbidity and cost burden associated with this condition.

Acknowledgments: The authors are grateful to all doctors and staffs of Neurology Department, Sir Salimullah Medical College, Mitford, Dhaka, Bangladesh.

Declaration of interest:

The authors report no conflict of interest.

Funding:

The author(s) received no financial support for the research, authorship, and/or publication of this article. The patient is aware that there is no patient identifiable information provided in the article and has consented to this publication. The clinical tests were all done as part of clinical practice.

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