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Case Report

An uncommon case of acute kidney injury with severe hyperkalaemia and polyuria in a patient with pituitary apoplexy

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Introduction

Pituitary apoplexy, due to acute haemorrhage or ischemia within a pituitary tumour, is an endocrinological emergency [1]. Acute onset headache, vomiting and neuro-ophthalmic manifestations are common presentations of this condition whereas bilateral proptosis is rare [2]. A high degree of clinical suspicion should be maintained, especially in the background of a known pituitary tumour. Cortisol deficiency is the most disastrous complication which can lead to severe haemodynamic consequences and acute kidney injury (AKI) if not promptly corrected [1]. Thus, empiric initiation of steroid treatment on clinical suspicion is essential, without awaiting diagnostic confirmation. AKI could be masked by polyuria secondary to transient diabetes insipidus due to apoplexy. Here, we present a case of acute kidney injury with severe hyperkalaemia and polyuria requiring haemodialysis in a patient with pituitary apoplexy due to delayed initiation of steroids with a focused review of the available literature.

Case report

A 48-year-old farmer from the Central Province was admitted with a 3-day history of headache, vomiting and altered consciousness. He denied any history of recent head injury, surgical intervention, febrile illness or anticoagulant use. He had a previous diagnosis of acromegaly with a pituitary macroadenoma, complicated by diabetes and hypertension. Initial imaging of the brain is shown in Figure 1. He had been scheduled for surgical intervention but had defaulted follow-up and was being managed at a local hospital for diabetes.

On admission to hospital, his Glasgow coma scale (GCS) was 14/15 and he had bilateral proptosis. conjunctival oedema, bilateral ophthalmoplegia and left sided partial ptosis (Figure 02).

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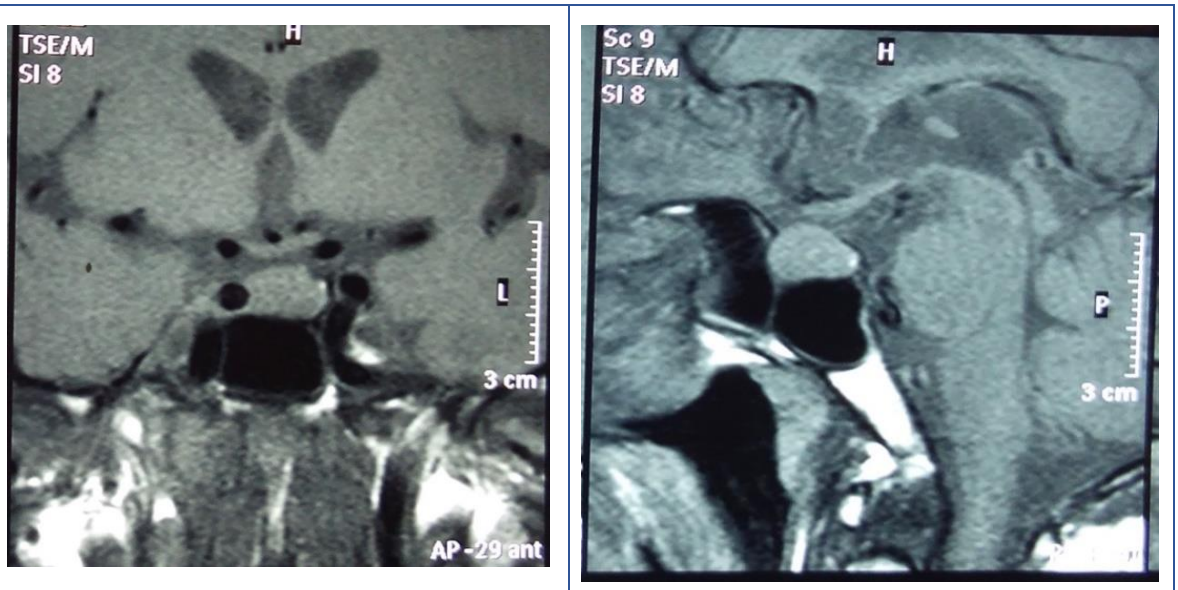

Figure 01: Initial MRI brain T1 weighted non contrast enhanced-Pituitary gland enlarged with convex sella turcica measuring 14*14.1*14.2. There is a hypointense non enhancing intrasellar lesion with peripheral ring enhancement compressing the pituitary parenchyma.

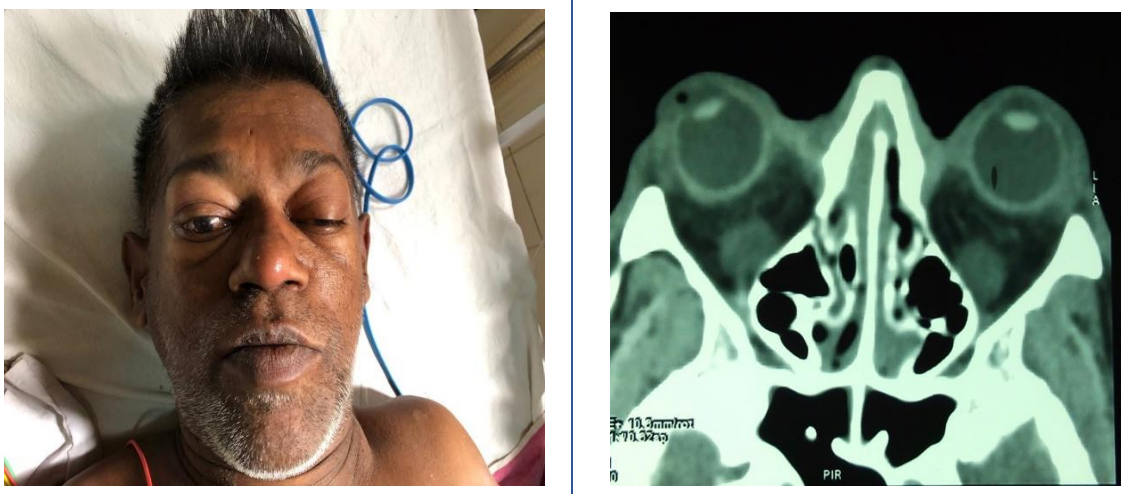

Figure 02: Acute presentation with bilateral proptosis and left sided partial ptosis and MRI revealing bilateral proptosis

Crude visual field assessment by the 'blink to threat' test was normal bilaterally. His visual acuity was reduced on both sides. He had no other focal signs and bilateral plantar reflexes were flexor. He was tachycardic with a pulse rate of 110 beats per minute and his blood pressure was 76/40 mmHg. His lung fields were clear, and the abdomen was soft to palpation. Bedside ultrasound scan (USS) revealed a collapsed inferior vena cava. His random blood sugar level was 37mmol/L. He was immediately initiated on intramuscular hydrocortisone 100mg with fluid resuscitation. Hydrocortisone at the same dosage was continued 6 hourly. Blood

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pressure improved rapidly with production of dilute urine 150-200 ml/hr. His investigations on admission are recorded in Table 1.

Table 01: Investigations on admission to a tertiary care hospital

Investigation	Result
ECG	Tall tented T waves
Serum K+	7.9mmol/L
Serum Na+	139mmol/L
Serum creatinine	7.6mg/dL
Blood urea	22mmol/L
HCO_3	6.2mmol/L
pH	7.01
Serum Ca	2.02mmol/L
Serum PO_4	2.61mmol/L
Serum lactate	9mmol/L
Procalcitonin	0.5ng/mL
CPK	217U/L
CRP	40mg/L
Liver profile	Albumin3.9g/dl Total protein6.8 g/dl AST 12 U/L ALT 17 U/L GGT 27
Serum osmolality	312mosm/kg
Urine osmolality	247mosm/kg
Urine electrolytes	Na74 mmol/L K10 mmol/L
UFR	Protein trace, Pus cells 2-4/HPF, Red cells nil

USS revealed bilateral hyperechogenic kidneys. Non-contrast computed tomography (NCCT) of the brain revealed bleeding into a pituitary macroadenoma with evidence of bilateral proptosis (Figure3).

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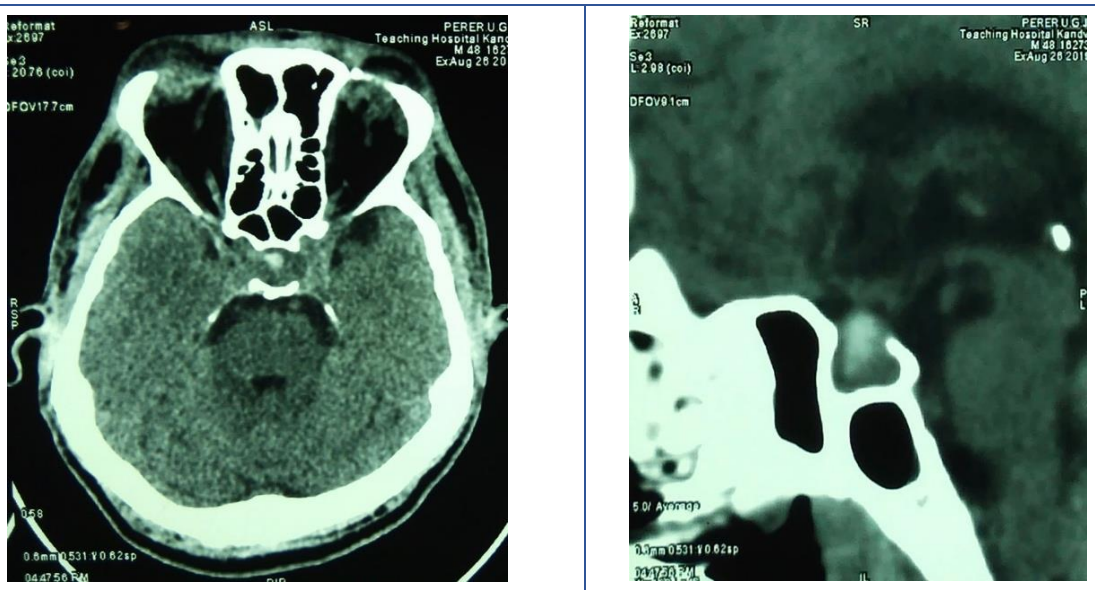

Figure 03: Pituitary apoplexy on NCCT brain axial and sagittal views 12.3*9.1*9.8 mm pituitary mass with a hyperdense centre suggestive of pituitary apoplexy

With investigations revealing evidence of hyperkalaemia and AKI, he was started on medical management of hyperkalaemia with intravenous calcium gluconate and an insulin-dextrose regime. Since he had a high Apoplexy Score with reduced level of consciousness, low visual acuity and bilateral ocular paresis, we obtained urgent neurosurgical opinion on decompression surgery. However, conservative management was initiated with close observation for neurological deterioration. He was managed in a high dependency unit with regular monitoring of blood pressure, blood sugar, urine output, conscious level and neuro-ophthalmic parameters.

According to his clinic records, his baseline serum creatinine level had been 1.2 mg/dl one month prior to admission. Although he had a high urine output throughout, he was initiated on haemodialysis with minimum ultrafiltrate, without heparin due to worsening hyperkalaemia, acidosis and a rising serum creatinine

Post haemodialysis, serum potassium normalised, and serum creatinine came down to 3.2 mg/dl. IV hydrocortisone was continued, and a second haemodialysis was arranged one day later. He gradually improved and by day 5 his GCS was 15, proptosis was settling and the ophthalmoplegia was improving. His serum electrolytes were within the normal range and creatinine was stable at around 1.6 mg/dl without further haemodialysis. Urine output was maintained at 2.5-3 L/24h.

Pituitary investigation profile revealed an FT4 level of 7.1 pmol/L (reference range 10-28 pmol/L) and IGF1 level of 152.5ng/ml (reference range 68-225 ng/ml). He was discharged on day 6 on oral hydrocortisone, thyroxine with a plan for repeat IGF1 and MRI scan in 3 months to assess biochemical and radiological resolution.

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Discussion

Pituitary apoplexy due to acute haemorrhage or ischemic infarction of a pituitary adenoma is an endocrinological emergency. It complicates 2-12% of pituitary adenomas, commonly non-functioning pituitary adenomas followed by prolactinomas and growth hormone secreting adenomas. Our patient had the latter [3,4].

A precipitating factor is identified in only 10-40% of cases of pituitary apoplexy [1,5]. No precipitating factor was identified in the current patient. Clinical manifestations of pituitary apoplexy are highly variable, largely determined by the extent of haemorrhage, necrosis and oedema and compression of neighbouring structures [1]. Headache is the most prominent symptom and is present in more than 90% of patients [1,5]. It is often associated with vomiting and nausea. Visual disturbances are also common and are present in more than 50% of patients with apoplexy [1,4,7,8]. A variable degree of visual field impairment can occur of which bitemporal hemianopia is the commonest [1]. Reduced visual acuity or even blindness are less common sequelae [1,2]. Ocular paresis is also a frequent manifestation, affecting 52% of patients and the third cranial nerve is the most commonly affected nerve [9]. Variable degrees of impaired consciousness may be observed ranging from lethargy to coma [6]. Less commonly, focal neurological deficits may result due to cerebral ischaemia secondary to compression of the intra cavernous internal carotid arteries or vasospasm [1,4,7]. On presentation, our patient had the classic neuro-ophthalmic manifestations as well as less common manifestations like bilateral proptosis which is a rare but well recognised feature of pituitary apoplexy [10]. The presence of classic neuro-ophthalmic manifestations, especially with the background history of pituitary macroadenoma, should raise suspicion of a pituitary apoplexy and the patient should be managed accordingly to prevent any subsequent catastrophic events.

Multiple acute endocrine deficiencies can occur in apoplexy, due to the destruction of the anterior pituitary or impaired release of hypothalamic hormones due to pressure on the pituitary stalk [111]. Cortisol deficiency is the most common deficit, occurring in 50-80% of patients and it is the hormonal deficiency that may lead to the most life-threatening complications (6). In the absence of steroids, blood vessels are insensitive to endogenous and exogenous catecholamines that can result in severe hypotension and AKI[12]. AKI is a less recognised complication associated with apoplexy [13]. It is characterized by a rise in serum creatinine of more than 0.3 mg/dl or a decreased urine volume of less than 0.5 ml/kg/hr. Pre-renal failure due to impaired renal perfusion is the commonest aetiology[14]. Patients with pre-existing renal injury are more prone to develop AKI. On presentation, the patient had haemodynamic instability with severe hypoglycaemia as a consequence of acute cortisol deficiency and the three-day delay before administration of steroids led to the development of AKI secondary to the severe haemodynamic compromise. Haemodialysis was required due to hyperkalaemia resistant to medical management. It is possible that he may have had an element of diabetic nephropathy as his most recent serum creatinine level was at the upper end of the normal range. Empiric steroid supplementation should be given to all patients with suspected pituitary apoplexy, even before confirmation to prevent disastrous consequences such as haemodynamic instability and AKI[1].

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Diabetes insipidus (DI) is a rare complication of pituitary apoplexy, occurring in less than 5% of patients. It may be masked and only emerge following steroid replacement [1,4,7,15]. The suggested mechanisms for DI are diminished perfusion of the posterior pituitary due to compression of the inferior hypophysial artery or impairment of the flow of ADH from hypothalamic nuclei to posterior pituitary by the pressure on the infundibulum (7). After initiation of steroid replacement, our patient had persistent polyuria in spite of a significant rise in serum creatinine and hyperkalaemia. His osmolality studies were suggestive of diabetes insipidus, although the Na levels remained normal due to fluid replacement. So, it is possible that he may have had a transient episode of diabetes insipidus which settled spontaneously without requiring treatment with desmopressin. In this scenario, the AKI could have been masked if no active monitoring of renal function was done. Thus, in the background of prolonged haemodynamic instability, exclusion of renal injury is important, with monitoring of renal function as urine output may be misleading in the presence of transient DI.

Pituitary apoplexy should be managed by an expert, multi-disciplinary team with the involvement of an endocrinologist, a neurosurgeon, a neuroradiologist and a neuro-ophthalmologist [16]. The mainstay of management is IV steroid replacement as soon as the diagnosis is made independent of whether the initial lesion is treated medically or surgically [1,17]. The optimal management strategy for acute pituitary apoplexy is controversial [1]. Neuro-ophthalmic complications and a reduced level of consciousness are indications for neurosurgical decompression and scoring tools, such as the pituitary apoplexy score, are available to guide management decisions [16]. Reports of spontaneous clinical improvement and shrinkage of tumour suggest that conservative management may be appropriate in selected cases[1]. Our patient had good neurological recovery with conservative management although he had a high apoplexy score on admission. Thus, patients with a stable clinical presentation and visible improvement in the first few days after admission could be safely managed conservatively.

Disappearance of the tumour mass or marked shrinkage after conservative management of pituitary apoplexy has been described [18]. Cases of spontaneous biochemical remission have also been reported in hormonally active tumours [13,15]. In our patient, IGF1 levels done after the acute episode were within the normal range. This could be an early indication of biochemical remission but re-evaluation both biochemically and radiologically is needed for confirmation [19].

Conclusion

Pituitary apoplexy due to acute haemorrhage or ischaemia within a pituitary tumour is an endocrinological emergency. A high degree of clinical suspicion should be seen in a patient with a pre-existing pituitary adenoma. Cortisol deficiency may be life threatening, leading to severe haemodynamic consequences if left untreated. Thus, corticosteroids must be initiated immediately on suspicion without waiting for diagnostic confirmation. AKI is a rare devastating complication of the hemodynamic consequences of acute cortisol deficiency. It could be masked by the polyuria secondary to the transient diabetes insipidus that may be seen in pituitary apoplexy and should be managed with haemodialysis when indicated.

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