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CASE REPORT

Pituitary apoplexy masquerading as meningitis

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Pituitary apoplexy is an uncommon condition and medical emergency that classically presents with abrupt onset of severe headache, nausea, fever, impaired level of consciousness, visual disturbances and a variable degree of ocular paresis. The constellation of signs, symptoms and cerebrospinal fluid findings in keeping with meningitis is considered a rare presentation of pituitary apoplexy and often leads to a misdiagnosis of infectious meningoencephalitis. We present the case of a 52 year old male admitted with a diabetic ketoacidosis who subsequently developed a severe headache with features of meningism. Haematological tests revealed an elevated white cell count and C-reactive protein, and lumbar puncture demonstrated a marked neutrophilic pleocytosis and elevated protein. Ceftriaxone was commenced for the presumptive diagnosis of bacterial meningitis, but resulted in no improvement in the patient's condition and so imaging studies were performed which revealed the presence of an incidental pituitary macroadenoma with intra-lesional haemorrhage, thus providing the diagnosis of pituitary apoplexy. The patient recovered with conservative management consisting of hormonal replacement therapy and continues to follow up at our facility.

Keywords: apoplexy, endocrinology, neurosurgery, panhypopituitarism, pituitary

Case study

A 52-year-old man with poorly controlled type 2 diabetes mellitus and hypertension presented with a three-day history of headaches, vomiting and a productive cough. The examination revealed the man to be obese, with *acanthosis nigricans* and a blood pressure of 147/97 mmHg, tachycardia of 91 beats per minute, but no pyrexia. The remainder of the examination was normal, with no features of meningism or focal neurological deficit. The finger-prick glucose result was 25 mmol/l. An arterial blood gas test revealed a high anion gap metabolic acidosis, with a pH of 7.25 (normal range 7.36–7.44), base excess of −14.7 (normal range −2 to + 2 mEq), and serum bicarbonate of 18.6 mmol/l (normal range 23–29 mmol/l). Ketonuria (2+) and glycosuria (3+) were demonstrated following urine dipstick testing. Formal laboratory investigations at the time of admission revealed normal calcium, magnesium and phosphate levels, and normal liver function and cardiac enzymes. The full blood count, C-reactive protein (CRP), and urea and electrolyte results are presented in Table 1. A diagnosis of diabetic ketoacidosis (DKA) was made, and therapy was instituted according to local protocol.

The patient's DKA responded rapidly to therapy, and had resolved by day 1 post admission. However, he then complained of marked worsening of his headaches. He had clinical signs of meningism with neck stiffness, photophobia and pyrexia. A lumbar puncture was performed. The results are shown in Table 2. Concomitantly, there was a marked increase in the CRP to 206 mg/dl. Bacterial meningitis was suspected, and ceftriaxone was initiated pending the cerebrospinal fluid (CSF) culture results. There was minimal improvement in his symptoms three days later. The CSF culture was negative, so a computed tomography brain scan was performed. It demonstrated a mass in the pituitary fossa

Table 1: Laboratory findings for the patient on admission

Parameter	Result	Reference range
White cell count (× 10^9/l)	8.95	3.92–10.40
Haemoglobin (g/dl)	18.30	13.40–17.50
Platelets (× 10^9/l)	340.00	171.00–388.00
C-reactive protein (mg/l)	< 10.00	< 10.00
Sodium (mmol/l)	130.00	136.00–145.00
Potassium (mmol/l)	4.00	3.50–5.10
Chloride (mmol/l)	91.00	98.00–107.00
Carbon dioxide (mmol/l)	26.00	23.00–29.00
Urea (mmol/l)	11.10	2.10–7.10
Creatinine (umol/l)	192.00	64.00–104.00
Haemoglobin A_{1c} (%)	14.10	

Table 2: The patient's cerebrospinal fluid results

Parameter	Result
Glucose (mmol/l)	11.30
Protein (g/l)	1.24
Clarity	Turbid
Colour	Colourless
Clots	Absent
Polymorphs (/µl)	535.00
Lymphocytes (/µl)	0.00
Erythrocytes (/µl)	280.00
Unidentified cells (/µl)	0.00
Indian ink	Negative
Gram stain	No bacteria observed
Cryptococcal latex antigen test	Negative
VDRL test	Nonreactive

VDRL: Venereal Disease Research Laboratory

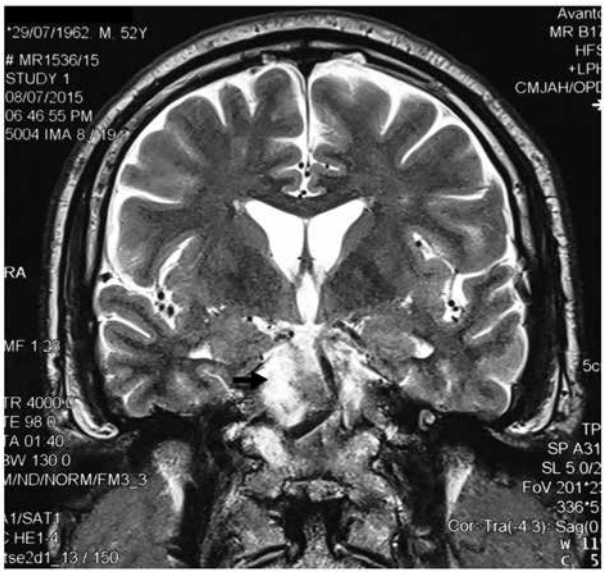

Figure 1: T2-weighted coronal magnetic resonance imaging, demonstrating a pituitary mass measuring 1.3 cm × 1.8 cm × 1.25 cm, causing expansion of the *sella turcica*, with an area of haemorrhage (arrow).

Table 3: Results of the pituitary panel

Parameter	Result	Reference range
Prolactin (µg/l)	2.10	2.10–17.70
Testosterone (nmol/l)	< 0.70	4.50–26.60
FSH (IU/l)	4.00	1.40–18.10
LH (IU/l)	0.90	1.50–9.30
GH (µg/l)	0.20	0.00–3.00
IGF-1 (µg/l)	56.70	87.00–238.00
TSH (mIU/l)	0.56	0.35–5.50
FT_4 (pmol/l)	10.90	11.50–22.70
Cortisol (nmol/l)	84.00	Morning: 145.00–619.00
ACTH (pmol/l)	< 1.10	1.10–10.20

Notes: ACTH: adrenocorticotropic hormone, FSH: follicle-stimulating hormone, FT_4: free thyroxine, GH: growth hormone, IGF-1: insulin-like growth factor-1, LH: luteinising hormone, TSH: thyroid stimulating hormone

measuring at least 1.2 cm × 1.8 cm. Hypodensity was seen, consistent with a recent haemorrhage. Subsequent magnetic resonance imaging confirmed the presence of a pituitary macroadenoma measuring 1.8 cm × 1.3 cm × 1.25 cm (Figure 1). A pituitary profile was performed, which confirmed hypopituitarism involving the thyrotropes, lactotropes, corticotropes, somatotropes and gonadotropes, with concomitant decreases in the effector hormones (Table 3). Visual field testing revealed bitemporal haemianopia. The patient was started on pituitary hormone supplementation, and completed a 10-day course of antibiotics. He was referred to the neurosurgical service and is awaiting resection of the tumour. He was stable on discharge and continues to follow-up at our outpatient clinic.

Discussion

First described by Bailey et al. in 1898 (though only named by Broughman et al. in 1950),[1] pituitary apoplexy is a well-known, but uncommon, endocrine emergency involving haemorrhage or infarction into a pre-existing pituitary adenoma or rarely normal pituitary gland.[2–4] It is classically characterised by the abrupt onset of visual field deficit, impaired visual acuity, headaches and nausea, and with or without associated endocrine abnormalities. However, the clinical manifestations may vary, depending on the predominating pathophysiological mechanism, i.e. tumour expansion, extravasation of the blood into the subarachnoid space, or partial or total destruction of the pituitary gland. Meningism, as a constellation of signs and symptoms mimicking bacterial meningitis, as occurred in our patient, is rare.[2,4] Secondary hypoadrenalism due to deficient adrenocorticotropic hormone and cortisol responses frequently occurs, and may result in an acute hypocortisolaemic crisis.[5]

Huang et al. reported on a case in 2009 involving a 57-year-old Taiwanese man who presented with fever and meningism, with CSF features of acute bacterial meningitis, who was unresponsive to empiric antibiotic therapy.[6] Minor differences in presentation were also reported in similar patients in two subsequent cases by Oh et al. and Lee et al.[2,3]

Although well described in the literature, the symptom of meningeal irritation is not considered a classic feature of pituitary apoplexy.[2,3] The pathophysiology behind this symptom complex involves leakage of blood into the subarachnoid space, which, in conjunction with the necrotic tissue in the pituitary itself, induces a cytokine response, resulting in meningeal irritation and the subsequent CSF picture, as previously described.[2–4]

Both medical and surgical intervention is necessary in the management of pituitary apoplexy. Medical management includes prompt glucocorticoid therapy to replace any cortisol deficiency, as well as the replacement of other hormones, as indicated, based on the laboratory testing. Such conservative therapy may be all that is required in mild disease. Surgical intervention is indicated in severe disease with neuro-ophthalmic signs or reduced level of consciousness,[5] and involves drainage of the haemorrhage with removal of the tumour.[2,3]

Conclusion

This case demonstrates the occurrence of pituitary apoplexy presenting as bacterial meningitis. It reinforces the importance of including pituitary apoplexy in the differential diagnosis of infectious meningitis and subarachnoid haemorrhage in patients presenting with acute headaches associated with pyrexia, meningism and visual abnormalities.

Declaration – None.

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