Cautionary Tale

Pituitary apoplexy after stimulation tests

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Summary: Pituitary apoplexy occurs after infarction of a non-neoplastic pituitary or sudden expansion of an adenoma following haemorrhage or infarction. It usually occurs spontaneously but can follow a number of causes including pituitary stimulation tests. Since this complication is potentially life-threatening, the benefits of subjecting patients who might have pituitary tumours to such tests should be considered.

Introduction

Pituitary apoplexy due to haemorrhage or infarction of a pituitary adenoma can occur after dynamic pituitary function testing. It has been observed with a combination of luteinizing hormone-releasing hormone (LHRH), thyrotrophin-releasing hormone (TRH) and insulin, after LHRH alone and TRH alone.¹ It has not been previously reported with the combination of LHRH, TRH and levodopa.

Case report

An 81 year old man presented with dyspnoea due to heart failure. He was otherwise asymptomatic but was noted to be very pale with no hair in the axillae or on the face. He had a left hemiparesis caused by a stroke 4 years ago. There was no visual field defect and he had slow relaxing reflexes. His free thyroxine (FT₄) was a 9 pmol/l (normal range 12–30 pmol/l) and thyroid stimulating hormone (TSH) was 1.5 mU/l (normal range 0.15–3.2). Other investigations included normal urea and electrolytes, liver function tests, electrocardiogram and skull radiograph. A chest radiograph confirmed heart failure.

An initial diagnosis of sick euthyroid state due to heart failure was made, but there were no changes in his thyroid hormone levels despite good control of the heart failure with diuretics, nitrates and an angiotensin converting enzyme inhibitor.

A combined test of pituitary function was performed with intravenous LHRH 100 µg and TRH

200 µg, levodopa 500 mg orally (to stimulate growth hormone release) and a short Synacthen test. Insulin hypoglycaemia was avoided due to his age and heart failure.

Two hours after the test began he developed frontal headache, nausea, vomiting, blurred vision and progressive drowsiness. There were no meningeal signs or papilloedema but he developed an incongruous left partial hemianopia. His TSH was 0.05 mU/l with a FT₄ of 7 pmol/l, and a short Synacthen test now showed a cortisol of 51 nmol/l at 0 minutes and 274 nmol/l at 30 minutes. A T₁-weighted magnetic resonance image (MRI) scan with gadolinium showed a ring-enhancing pituitary tumour with suprasellar extension and a cystic low density centre (Figure 1).

He was treated conservatively with fluids, intravenous hydrocortisone and tri-iodothyronine. His condition improved over the following days, and he was discharged on thyroxine and steroid replacement.

Discussion

Pituitary apoplexy occurs after infarction of a non-neoplastic pituitary or sudden expansion of an adenoma following haemorrhage or infarction.² It usually occurs spontaneously since the blood supply to the tumour is precarious, but it has been reported complicating trauma, raised intracranial pressure, anticoagulants, bromocriptine, diabetic ketoacidosis, angiography, radiation, open heart surgery, pregnancy, upper respiratory tract infections and pituitary stimulation tests.³ TRH is the likely causal agent, although it has occurred after LHRH,⁴ and the exact mechanism is unknown.

Pituitary apoplexy is a syndrome characterized by bilateral frontotemporal headache, vomiting,

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Figure 1 T_1 -weighted MRI showing a pituitary adenoma with suprasellar extension, ring enhancement with gadolinium and a cystic centre.

cranial nerve palsies, visual field defects, hyperpyrexia and decreased consciousness. Plain radiographs and computed tomographic (CT) scanning with enhancement are usually sufficient to make a diagnosis. On CT an infarcted adenoma becomes an enlarging hypodense mass due to oedema. In haemorrhage there are increased attenuation values from the tumour or rarely a blood fluid level. A ring-enhancing lesion with a low-density centre is suggestive of pituitary apoplexy as long as there is no history of radiation. T_1 -weighted MR images reveal low signals from infarcted areas and gadolinium enhancement from non-infarcted tissue.⁵ Suspected cases should be given intravenous steroids, and a close monitor on fluids and electrolytes should be kept since, uncommonly, diabetes insipidus or the syndrome of inappropriate anti-diuretic hormone secretion may develop.

Unfortunately, the indications for surgical treatment are not so clear, since the clinical course is unpredictable and spontaneous resolution is

known to occur. Patients with severe deterioration of level of consciousness, visual acuity or visual field should be referred for surgery. Ophthalmoplegia is not an absolute surgical indication.⁶

Some reports suggest that pituitary apoplexy due to infarction follows a less aggressive course and a conservative approach is best.⁵

This is a case of pituitary apoplexy following a particular combination of pituitary stimulation tests including levodopa, which has not previously been described.

This condition is rare, but since this is a potentially fatal complication, the benefits of subjecting patients who might have pituitary tumours to such tests should be carefully considered, particularly as it appears to be a risk associated with a number of different stimulation tests. Indeed, it could be argued that the test performed in this case was inappropriate, and a better approach to management would have been replacement therapy, and imaging of the pituitary with CT or MRI.

References

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