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Case Report

A case of hypophysitis in a young male patient

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Introduction

Lymphocytic hypophysitis, which is autoimmune inflammation of pituitary gland, is initially characterised by lymphocytic infiltration and enlargement followed by destruction of pituitary gland. Although most cases occur in females during pregnancy and early post-partum period, it may rarely be seen in males.[1] We describe a young male patient who presented with acute onset pituitary enlargement with left third cranial (oculomotor) nerve palsy, which resolved completely without any residual deficit.

Case report

A 42-year-old male patient presented with history of acute onset bifrontal headache with projectile vomiting of 3 days duration. There was no history of fever, visual symptoms, altered sensorium or focal neurological deficits. On day 3 of illness, he developed sudden onset binocular diplopia. On examination, he was afebrile and normotensive with no postural fall of blood pressure. He had left-sided third cranial nerve palsy without pupillary involvement. Other cranial nerves were normal. There was no papilledema or any other neurological deficit. Urgent non-contrast computerised tomography scan brain was normal with no intracerebral haemorrhage. Magnetic resonance imaging (MRI) scan of the brain revealed enlarged pituitary gland measuring 12 mm × 19 mm × 10 mm with convex superior border protruding into suprasellar cisterns but not reaching optic chiasma and thickened infundibular stalk, which was in midline (Figs. 1 and 2). Cerebrospinal fluid examination revealed WBC 20/mm^3 (lymphocytes), RBC 6/mm^3, protein 87 mg/dl and glucose 65 mg/dl (blood glucose 102 mg/dl). Hormonal evaluation revealed basal hypocortisolism (3.2 mcg/dl) with normal ACTH levels (26 pg/ml) suggestive of basal hypocortisolemia or secondary adrenal insufficiency. He showed a normal cortisol response to ACTH stimulation (22 mcg/dl). The rest of the pituitary hormone profile including LH, FSH, prolactin, TSH and free T4 was normal. His urine output was normal and osmolality was 687 mOsm/kg (300–900) suggesting no evidence of diabetes insipidus (DI). Serum electrolytes, glucose levels and renal and liver function tests were normal. Markers of autoimmunity (ANA, dsDNA and anti-TPO antibody) were negative. Serum angiotensin converting enzyme was normal. He was started on oral corticosteroids in anti-inflammatory dose with tablet prednisolone 60 mg daily. Within a week, he had complete resolution of symptoms and left third cranial nerve palsy

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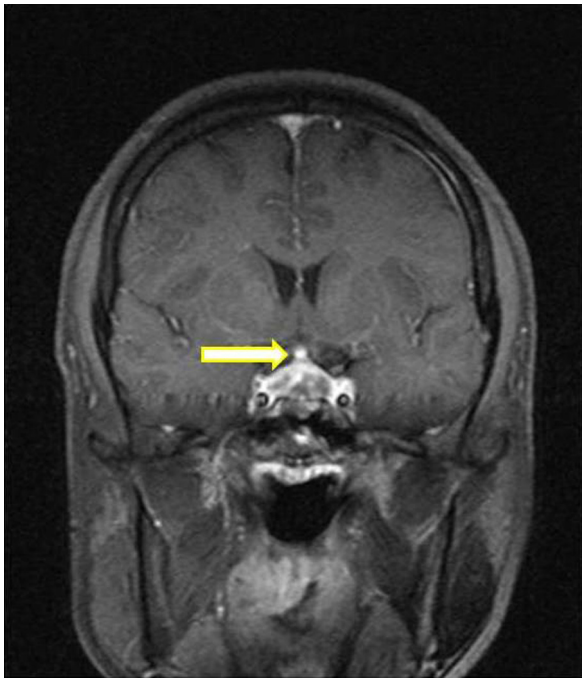

Fig. 1 – Contrast enhanced Magnetic resonance imaging (MRI) scan (Coronal view) of the brain showing enlarged pituitary gland with homogenous enhancement measuring 12 × 19 × 10 mm with convex superior border protruding into suprasellar cisterns with thickened infundibular stalk in midline (Arrow).

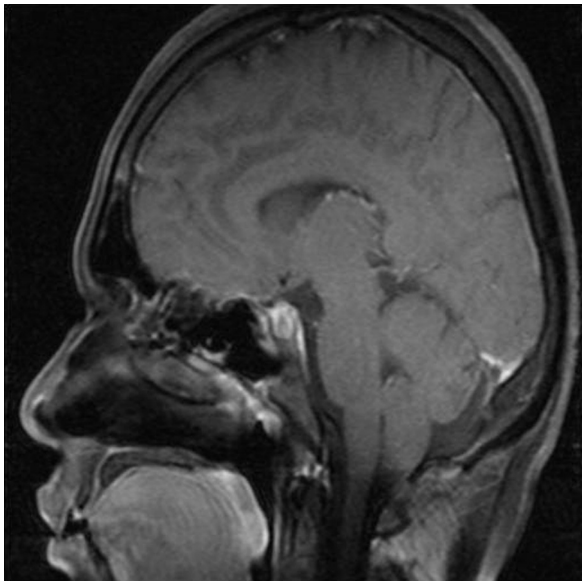

Fig. 2 – Contrast enhanced Magnetic resonance imaging (MRI) scan (Sagittal view) of the brain showing enlarged pituitary gland with homogenous enhancement.

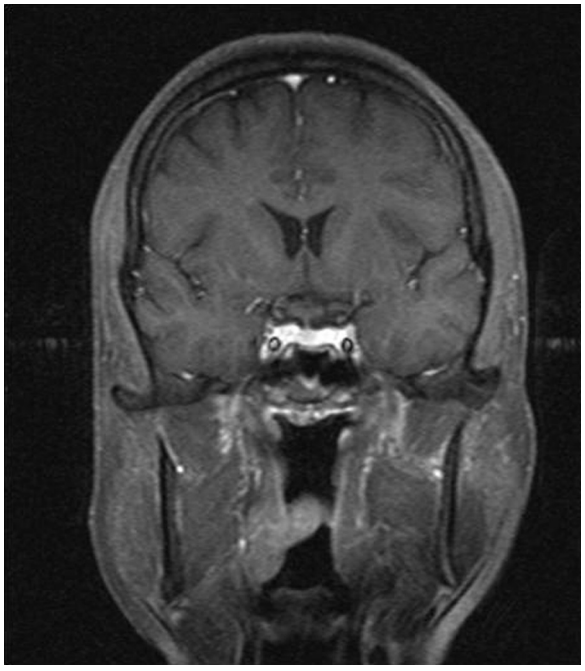

Fig. 3 – Follow up post treatment contrast enhanced MRI brain (Coronal view) showing resolution of the pituitary mass.

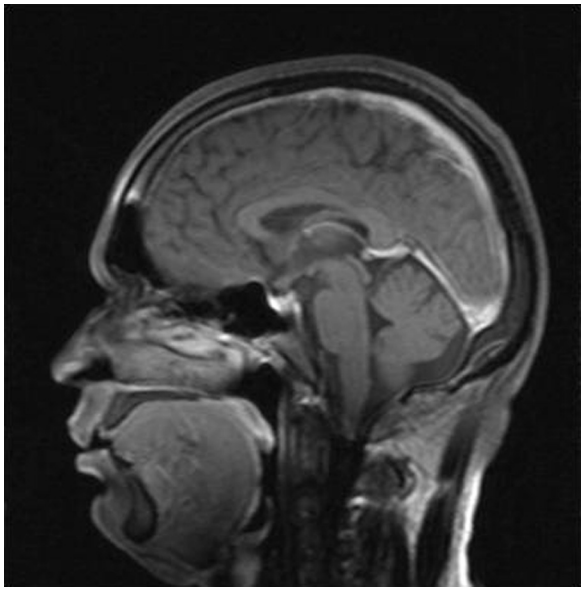

Fig. 4 – Follow up post treatment contrast enhanced MRI brain (Sagittal view) showing resolution of the pituitary mass.

resolved. Prednisolone was tapered off over the next 8 weeks. His repeat MRI done after 6 months showed complete resolution of the pituitary mass (Figs. 3 and 4) and basal and post ACTH cortisol levels were normal. There was no residual pituitary hormone deficit. Investigations are tabulated in Table 1. He has been advised regular follow-up for recurrence of hypophysitis and monitoring for pituitary hormone deficiency.

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