CASE REPORT

Acute psychosis as a presentation of hypopituitarism

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SUMMARY

Acute onset neuropsychiatric manifestations in hypopituitarism are uncommon. We report a case of a 60-year-old man who was a follow-up case of macroprolactinoma with hypopituitarism for the last 9 years. He was on medical treatment with cabergoline, thyroxine and depot testosterone. During the last 2 years he was non-adherent to medications especially cabergoline. He was hospitalised for 2 days through emergency services following acute onset psychosis. His pituitary hormone profile was suggestive of adrenal insufficiency, secondary hypothyroidism and hypogonadism. MRI of the hypothalamic pituitary region revealed a pituitary macroadenoma which was larger in size compared to the previous scan. Further, this lesion was compressing on the adjoining structures including optic chiasma. The patient was treated with intravenous fluids, hydrocortisone and thyroxine replacement therapy. With this treatment he completely recovered from psychosis within 48 h.

BACKGROUND

Acute psychosis as presentation of hypopituitarism is rare and has been previously reported in patients with Sheehan's syndrome, following traumatic brain injury and after glucocorticoid therapy.^{1–4} Neuropsychiatric manifestations have also been occasionally reported in cases with macroprolactinoma attributed to hyperprolactinaemia.⁵ We describe a case of macroprolactinoma with hypopituitarism who presented as acute psychosis and recovered completely with intravenous fluids and hormone replacement therapy.

CASE PRESENTATION

A 61-year-old man presented to a private hospital with headache and loss of libido 9 years ago. His serum prolactin levels were 800 ng/mL (normal range: 2–18 ng/mL). MRI of hypothalamic pituitary region revealed an 11×12×9 mm mass lesion in pituitary fossa. Therefore, he was diagnosed with macroprolactinoma with secondary hypogonadism and was treated medically with dopamine agonist therapy and depot preparation of testosterone. He responded well and had a near complete resolution of the mass as per MRI scan after 18 months of therapy. Two and a half years later he was also found to have central hypothyroidism and was given thyroid hormone replacement therapy as well. During the last 2 years, his follow-up with the treating neurosurgeon was very irregular and he became non-adherent to treatment especially to cabergoline. Recently, he presented to the emergency room with headache for the last 6 months and progressive diminution of peripheral vision for

the last 4 months. Two days prior to admission to hospital, the patient had acute onset irrelevant talking, restlessness along with inability to sleep. He also had voices in his head and a feeling of intense heat in his head suggestive of auditory and somatic hallucinations. He was extremely agitated and had to be restrained by his family. There was a history of loss of libido, excessive fatigue and depressed mood for the past few weeks.

On examination, he was conscious, oriented to time, place and person but agitated. There was loss of insight and a loosening of association of his thought. His attention and concentration were impaired as suggested by an inability to perform forward and reverse digit span test. His vital functions such as body temperature, pulse, blood pressure and respiratory rate were within normal limits. There was absence of pubic and axillary hair. On confrontation test for visual field charting he had bilateral heteronymous hemianopia. Bilateral ankle reflexes showed a delayed relaxation time. Rest of the examination including the systemic review was within normal limits.

INVESTIGATIONS

Complete blood count revealed haemoglobin 10.4 g/dL, total leucocyte count 7 800/mm³ with polymorphs 80%, lymphocytes 18%, monocytes 0% and eosinophils 2%. Platelet count was 200×10³/mm³. His random blood glucose was 82 mg/dL. Kidney function and liver function tests were within normal limits. Serum sodium and potassium were 148 and 3.8 mEq/L, respectively. Urine routine and microscopy revealed no abnormality. Chest radiograph and ECG were also normal.

A complete hormonal profile was carried out with a clinical suspicion of hypopituitarism. Serum cortisol (morning fasting) was 3.37 µg/dL (4.30–22.4 µg/dL). Serum thyroid-stimulating hormone, T3 and T4 were 0.12 µIU/mL (0.35–5.5 µIU/mL), 0.99 ng/mL (0.6–1.81 ng/mL) and 8.7 µg/dL (4.5–10.9 µg/dL), respectively. Serum testosterone level was 87 ng/dL (241–827 ng/dL) with low normal luteinizing hormone and follicle-stimulating hormone. Basal serum growth hormone and IGF1 levels were 0.16 mg/mL (0.00–4.21 mg/mL) and 63.2 mg/mL (81–225 mg/mL), respectively. Reported laboratory value of serum prolactin was 1200 ng/mL (2–18 ng/mL). Overall his pituitary hormone profile was suggestive of hyperprolactinaemia with panhypopituitarism. MRI brain revealed a large pituitary mass measuring (36 mm×33 mm×22 mm) compressing on optic chiasma and encasing the bilateral internal carotid arteries (figure 1A,B).

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Figure 1 (A and B) MRI pituitary (contrast-enhanced T1-weighted scans-sagittal and coronal views, respectively) showing a sellar mass (36 mm×33 mm×22 mm) with diffuse enhancement with supra sellar extension causing compression on optic chiasma with encasement of bilateral internal carotid arteries.

TREATMENT

The patient was treated with intravenous fluids and glucocorticoids (hydrocortisone 100 mg intravenously every 6 hourly×48 h and then 50 mg 8 hourly). Along with this the patient was given oral cabergoline 1 mg twice a week, oral thyroxine 100 µg once a day and injectable testosterone depot 250 mg every 3 weeks.

OUTCOME AND FOLLOW-UP

The patient improved dramatically within 48 h with resolution of psychosis. He was then put on oral steroids (tablet prednisolone 20 mg (morning) and 10 mg (evening)) which was then tapered gradually to the present dose of 5 mg in morning and 2.5 mg in the afternoon.

The patient was also put on injectable growth hormone 0.3 mg (1.0 IU) subcutaneous 1 week after his discharge. According to his last follow-up visit he had improved considerably and his serum prolactin had reduced to 200 ng/mL.

DISCUSSION

Psychiatric manifestations have been described in patients with hypopituitarism due to different aetiologies. Psychiatric

morbidity is quite common in adults with hypopituitarism.⁶ In this large series the authors described that 46% cases of hypopituitarism with adult growth hormone deficiency had definite psychiatric illness. In one of the earliest report, Sheehan and Summer⁷ (1949) gave an account of psychiatric symptoms in their survey of 143 patients with hypopituitarism. One-third of their patients had loss of drive and initiative, lack of interest in friends and relatives, and no desire to return home from hospital. Another 22 cases had other psychiatric symptom with delusions being commoner than mania. Patients approaching coma in this case series were excluded. Gutowski and Heron⁸ described three women in late middle age had recurrent episodes of confusion which could not be explained solely on the basis of an associated infection. All three patients had latent hypopituitarism diagnosed on the basis of clinical and hormonal profile. Each patient had a previous history of a severe post-partum haemorrhage followed by two further pregnancies. Majority of patients with hypopituitarism have hyponatraemia which may explain altered sensorium and delirium.⁹

Hypopituitarism very rarely manifests as psychosis in absence of delirium. This has been aptly described by Leo *et al*¹⁰ in 1998 in their two case reports. These two patients presented as psychosis without delirium and recovered with hormonal replacement. Dissanayake and Leiberman¹¹ have also reported two cases of hypopituitarism with paranoid psychosis without memory disturbance or mental confusion. In one patient the psychosis had lasted for 16 years and in other for 2 years despite treatment with phenothiazines. However, there was a complete remission in these patients with hormone replacement. Similar cases have also been reported recently.¹² Our patients also had acute onset psychosis with clear consciousness. Hyperprolactinaemia and hyponatraemia are unlikely to be contributing factor for psychosis in our case as he recovered within 48 h after starting treatment and his serum sodium levels were normal. The possible mechanisms of psychosis in hypopituitarism are combination of hypothyroidism, hypoglycaemia and hypocorticism resulting in complex metabolic and electrolyte changes in the central nervous system. In patients with chronic cognitive dysfunction growth hormone deficiency is likely to play an important role.

In summary, our patient had recurrence of macroprolactinoma due to poor compliance to medical treatment and presented as psychosis, which resolved with intravenous hydrocortisone and fluids. We, therefore, conclude that patients with macroprolactinoma with associated hypopituitarism can present as acute psychosis.

Learning points

- ▶ Patients with panhypopituitarism can have multiple neuropsychiatric manifestations including acute psychosis. Further, these clinical features can occur after prolonged periods of hormonal deficiencies.
- ▶ Acute psychosis in hypopituitarism is reversible with hormone replacement therapy.

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Competing interests None.

Patient consent Obtained.

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