Unusual presentation of more common disease/injury

Idiopathic hypoparathyroidism: an unusual cause of acute encephalopathy

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Summary

Though hypoparathyroidism is not an uncommon diagnosis, it is an unusual cause of seizures in adults. The authors report a case of late onset epilepsy due to idiopathic hypoparathyroidism in a 50-year-old man. This case reiterates the importance of CT scan and serum calcium in the evaluation of patients with recurrent seizures.

BACKGROUND

Middle-aged adults presenting with intractable seizures primarily due to an endocrinological condition is distinctly uncommon in medical literature. There are only isolated case reports of adults presenting with seizures secondary to idiopathic hypoparathyroidism, although postsurgical hypoparathyroidism causing acute hypocalcemic seizures is a frequent clinical occurrence. Idiopathic hypoparathyroidism has distinct clinical, biochemical and radiological manifestations which are simple to recognise and the seizures amenable to treatment without routine use of antiepileptic drugs.

CASE PRESENTATION

A 50-year-old man presented to the medicine emergency with recurrent seizures of 1 day duration, postictal confusion and altered sensorium. There was no history of fever, headache, head trauma or focal neurological deficit. He had similar episodes of seizures since last 2 years for which he was prescribed phenytoin. However, despite being compliant, he continued to have frequent breakthrough seizures with a recent increase in their frequency. Relatives gave a history of increased irritability and decreased judgement. He was operated for bilateral cataract 5 years back. There was no neck surgery in the past. There was no family history of similar illness. Subsequent examination of this patient revealed dystrophic nails, brisk reflexes and focal dystonia of upper limbs. The Trousseau sign was positive. There was no evidence of oral candidiasis. Rest of the systemic examination was unremarkable.

INVESTIGATIONS

On investigation, the haemogram was within normal limits. The blood urea was 24 mg/dl and creatinine was 0.9 mg/dl. The serum sodium was 139 mEq/l and potassium was 4.4 mEq/l respectively. The serum calcium concentration (normal range: 8.0–10.4 mg/dl) was found to be 4.5 mg/dl (corrected 5.3 mg/dl for hypoalbuminemia). The ionised Ca value was 3.4 mg/dl (normal range: 4.6–5.4 mg/dl). The serum phosphate level was 5.6 mg/dl (normal range: 2.5–4.5 mg/dl). The liver function tests revealed total bilirubin

of 0.8 mg/dl; alkaline phosphatase 147 IU/l (normal range: 54–369 IU/l); aspartate aminotransferase (AST): 36 IU/l, alanine aminotransferase (ALT): 38 IU/l (normal range: AST: 5–35 IU/l; ALT: 5–40 IU/l). The total proteins were reduced at 6.4 g/dl and serum albumin was 3.1 g/dl. The arterial blood gas analysis showed a pH of 7.494; PCO₂: 44.5; PO₂: 80.5; HCO₃: 32.8 mEq/l; SAT: 97%, meaning thereby metabolic alkalosis with respiratory compensation. 24 h urinary data revealed a normal loss of calcium in urine as measured to be 0.2 g (normal range: 0.1–0.3 g) and a decreased phosphate loss of 0.09 g (normal range: 0.4–1.3 g). There was also mild proteinuria of 460 mg. The serum magnesium levels were 1.9 mg/dl (normal range: 1.2–2.5 mg/dl). The intact parathormone levels were found to be 3 pg/ml (normal range: 8–51 pg/ml). The ECG of the patient did not reveal any QT prolongation. EEG revealed generalised 2–4 Hz spike and wave pattern.

A non-contrast imaging of the brain revealed extensive calcifications of the brain parenchyma (figure 1). Bilateral and mostly symmetrical calcifications were noted in the basal ganglia, cerebral cortex, subcortical white matter and cerebellum. The imaging of the forearm was suggestive of interosseous membrane calcification. Abdominal imaging was unremarkable.

DIFFERENTIAL DIAGNOSIS

A diagnosis of idiopathic hypoparathyroidism with bilateral extensive intracranial calcifications and hypocalcemic seizures was made.

TREATMENT

The patient was started on 1 g of elemental calcium, 0.5 µg of calcitriol per day which slowly restored his serum calcium levels. He was also started on sodium valproate 500 mg twice a day and discharged seizure free in satisfactory condition.

OUTCOME AND FOLLOW-UP

He has had no fresh seizures for the last 1 year and is maintaining normal calcium and phosphate levels.

Figure 1 Non-contrast CT brain depicting calcifications of cerebral cortex and subcortical white matter.

DISCUSSION

Idiopathic hypoparathyroidism presents most commonly in the age group of 5–10 years.¹ It may occur as an isolated entity or may be associated with the autoimmune polyendocrine syndrome type 1 encompassing thyroid disease, autoimmune hypoadrenalism and muco-cutaneous candidiasis. There are few case reports in literature of adults presenting with intractable seizures secondary to idiopathic hypoparathyroidism.² Our patient had developed symptoms of hypoparathyroidism quite late in life. Low serum calcium, low intact PTH and high phosphate values all point towards the diagnosis of idiopathic hypoparathyroidism. History of premature cataract, dystrophic nails, extensive intracranial calcifications, interosseous membrane calcifications all indicate chronic hypocalcaemia. Although

asymptomatic basal ganglia calcifications are a common occurrence in adults, such extensive bilateral symmetrical intracranial calcifications should alert the physician to the above diagnosis.

Idiopathic hypoparathyroidism is a treatable cause of epilepsy. Treatment of hypocalcaemia generally controls the seizure disorder and in some cases anticonvulsant treatment may be withdrawn. Therapy for hypoparathyroidism has been recently extensively reviewed.³ The aims of treatment are symptom control, maintaining serum albumin, a 24-h urinary calcium below 300 mg and a calcium-phosphate product below 55. Treatment is generally with calcitriol and calcium supplementation, although some clinical trials and isolated cases have administered subcutaneous synthetic human parathyroid hormone (PTH 1–34) with conflicting results.^{4 5}

Learning points

- ▶ Idiopathic hypoparathyroidism is a condition that manifests with symptoms and signs of acute and chronic hypocalcaemia.
- ▶ Acute hypocalcaemia may have protean manifestations varying from limb paresthesias to intractable seizures.
- ▶ Idiopathic hypoparathyroidism needs to be short listed in the differential diagnosis of adult-onset, generalised, tonic-clonic seizures.

Competing interests None.

Patient consent Obtained.

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Please cite this article as follows (you will need to access the article online to obtain the date of publication).

Gupta M, Lehl SS, Sachdev A. Idiopathic hypoparathyroidism: an unusual cause of acute encephalopathy . *BMJ Case Reports* 2011;10.1136/bcr.12.2010.3593, date of publication

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