CASE REPORT

Reversible central neural hyperexcitability: an electroencephalographic clue to hypocalcaemia

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SUMMARY

A 23-year-old male patient presented with cognitive decline and seizures. Examination revealed Chvostek's and Trousseau's signs. Investigations revealed hypocalcaemia, hyperphosphatemia and normal intact parathyroid hormone levels. Imaging showed calcifications in bilateral basal ganglia, thalamus and dentate nuclei. Interictal electroencephalogram showed theta range slowing of background activity and bilateral temporo-occipital, irregular, sharp and slow wave discharges, which accentuated during hyperventilation, photic stimulation and eye closure. Appearance of epileptiform discharges after eye closure, hyperventilation and photic stimulation may suggest presence of central neural hyperexcitability due to hypocalcaemia. These features may be an equivalent of peripheral neuromuscular hyperexcitability (Chvostek's and Trousseau's signs) that occurs in hypocalcaemia. The clinical and electroencephalographic features completely reversed with correction of serum calcium without antiepileptic medications. It is important for clinicians to recognise these reversible changes, as it can help to avoid misdiagnosis and long-term administration of antiepileptic becomes unnecessary.

BACKGROUND

To the best of our knowledge, accentuation of epileptiform discharges on eye closure, photic stimulation and hyperventilation, and reversibility of these changes with correction of serum calcium have not been highlighted in literature. Electroencephalogram (EEG) with appropriate activation procedures can be used to substantiate the central neural hyperexcitability associated with hypocalcaemia. Recognition of these reversible changes can help to avoid misdiagnosis and long-term administration of antiepileptic becomes unnecessary.

CASE PRESENTATION

A 23-year-old male patient presented with poor scholastic performance and seizures since 4 years. He had left focal with secondarily generalised seizures since 4 years. The frequency of seizures was once every 2–3 months with daily seizures since last 2 months. On examination, he had positive Chvostek's and Trousseau's signs. His neurological examination was normal except that his deep tendon reflexes were brisk.

INVESTIGATIONS

His serum calcium was 3.5 mg/dL (8.5–11.5), phosphorous was 8.0 mg/dL (2.5–4.5) and serum albumin was normal. His ECG revealed QT prolongation. He was given intravenous calcium and levetiracetam was started in view of daily seizures. Interictal EEG showed theta range slowing of background activity and bilateral temporo-occipital, irregular, sharp and slow wave discharges, which accentuated during hyperventilation, photic stimulation and eye closure. These discharges characteristically appeared as soon as visual fixation was interrupted by eye closure ([figure 1](#)). CT brain showed bilateral basal ganglia, thalamus dentate nucleus and subcortical white matter calcifications which was characteristic of Fahr's syndrome ([figure 2](#)). His intact parathyroid hormone was 16 pg/mL (11–67), vitamin D₃ was 22.3 ng/mL (30–60), magnesium was 2.1 mg/dL (1.6–2.6), thyroid-stimulating hormone was 2.3 µIU/ml (0.35–5.5), Free T3 was 110.5 (81–176 ng/dL), Free T4 was 5.6 µg/dL (4.5–12.5) and cortisol levels was 17.5 µg/dL (5–25).

DIFFERENTIAL DIAGNOSIS

Cerebral calcification can be primary that is, Fahr's disease or secondary to endocrinopathies, mitochondrial diseases, infections or other inherited disorders (eg, Cockayne syndrome) which is known as Fahr's syndrome. In view of very low serum calcium, high phosphorus, normal parathyroid hormone and characteristic CT findings, diagnosis of Fahr's syndrome associated with hypocalcaemia was considered.

TREATMENT

The patient was supplemented with oral calcium and calcitriol.

OUTCOME AND FOLLOW-UP

At the end of 1 month, serum calcium and EEG returned to normal and patient had neither seizures nor abnormal posturing. As patient was asymptomatic and EEG was normal, antiepileptic drug was stopped. At 4-weeks follow-up, EEG showed normal background without any epileptiform discharges on hyperventilation, photic stimulation and interruption of visual fixation ([figure 3](#)). At the end of 2 months, patient was completely asymptomatic.

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Figure 1 Electroencephalogram recording in bipolar montage (10/20 system) shows (A) hyperventilation induced sharp and slow wave discharges over bilateral temporo-occipital region, (B) photic stimulation induced sharp and slow wave discharges over bilateral temporo-occipital region and (C) irregular sharp and wave discharges occurring on eye closure. Recorded at 1 s x 7.5 μ V/mm.

Figure 2 CT brain plain image shows calcification in bilateral basal ganglia, thalamus and subcortical white matter.

Figure 3 Electroencephalogram recording in bipolar montage (10/20 system) shows normal background activity on (A) hyperventilation and (B) after eye closure. Recorded at 1 s x 7.5 μ V/mm.

Learning points

- ▶ Electroencephalogram (EEG) in hypocalcaemia is characterised by generalised theta/delta range slowing and focal or generalised epileptiform discharges.
- ▶ Epileptiform discharges in hypocalcaemia can be accentuated after eye closure, hyperventilation and photic stimulation may suggest presence of central neural hyperexcitability due to hypocalcaemia.
- ▶ These discharges may be equivalent of peripheral neuromuscular hyperexcitability that occurs in hypocalcaemia.
- ▶ EEG abnormalities are completely reversible with correction of serum calcium levels.
- ▶ It is important for clinicians to recognise these reversible changes, as it can help to avoid misdiagnosis and long-term administration of antiepileptic becomes unnecessary.

DISCUSSION

EEG in hypocalcaemia is characterised by generalised theta/delta range background slowing and focal or generalised spike and wave discharges. Hyperventilation and photic stimulation induced epileptiform discharges have been reported in patients with hypocalcaemia.¹⁻³ In our patient, appearance of epileptiform discharges after eye closure, hyperventilation and photic stimulation may suggest presence of central neural hyperexcitability due to hypocalcaemia. These features may be an equivalent of peripheral neuromuscular hyperexcitability (Chvostek's and Trousseau's signs) that occurs in hypocalcaemia. In our patient, these changes completely reversed with normalisation of calcium levels at the end of 1 month, which correlated with clinical improvement. Resolution of clinical features and normalisation of EEG changes were present at 1-month follow-up, after stopping antiepileptic drugs. To the best of our knowledge, accentuation of epileptiform discharges on eye closure, photic stimulation and hyperventilation and reversibility of these changes with correction of serum calcium have not been highlighted in literature. EEG with appropriate activation procedures can be used to substantiate the central neural hyperexcitability associated with hypocalcaemia. It is important for clinicians to recognise these reversible changes, as it can help to avoid misdiagnosis and long-term administration of antiepileptic becomes unnecessary.⁴

Contributors BAP, KVK: drafting of manuscript, analysis/interpretation of neurological/EEG findings and review of existing literature. RTC: drafting of

manuscript and critical analysis of the manuscript. SN: drafting of manuscript and providing clinical details of case.

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