New onset of epileptic seizures induced by Fahr’s syndrome secondary to idiopathic hypoparathyroidism.

Cristina Alina Silaghi1,2, Carmen Emanuela Georgescu2, Cristina Ghervan2, Ana Valea2, Ramona Irimia1, Daniel Tudor Cosma3, Sanda Petrus4, Alina Filip5, Horațiu Silaghi6, Vasile Țibre7

1 County Emergency Hospital, Cluj-Napoca, Romania; 2 Department of Endocrinology, ”Iuliu Hatieganu” University of Medicine and Pharmacy, Cluj-Napoca, Romania; 3 Diabet, Nutrition and Metabolic Diseases Clinical Center Cluj-Napoca, Romania; 4 County Hospital, Târgu Mureș, Romania; 5 County Hospital, Bistrița, Romania; 6 5th Department of Surgery, ”Iuliu Hatieganu” University of Medicine and Pharmacy, Cluj-Napoca, Romania; 7 Department of Neurology, ”Iuliu Hatieganu” University of Medicine and Pharmacy, Cluj-Napoca, Romania.

Introduction

Hypocalcemia due to hypoparathyroidism leads to a large spectrum of clinical manifestations but a rare and unusual presentation is onset or aggravation of epilepsy. Physiological intracranial calcification occurs in about 0.3-1.5% of cases. It is asymptomatic and detected incidentally by neuroimaging. Pathological basal ganglia calcification is due to various causes, such as: metabolic disorders, infectious and genetic diseases. Hypoparathyroidism and pseudohypoparathyroidism are the most common causes of pathological basal ganglia calcification. We present a representative case study of a spectacular extensive bilateral brain calcifications in the basal ganglia in a patient with idiopathic hypoparathyroidism with neurological manifestations.

Clinical case

We report a 77-year-old man who was found to have profound hypocalcaemia and idiopathic hypoparathyroidism when investigated for epileptic seizures and loss of consciousness. He had affective disturbances and only mild neurocognitive disorders. Cataract was present. The neurological examination showed an extrapyramidal syndrome with postural tremor and cerebellar ataxia. The deep tendon reflexes were nonoactive in all four limbs. Chvostek’s sign was present but Trousseau sign was not observed.

Laboratory analysis showed: low concentration of serum ionized calcium at 2.9 mg/dL (normal: 4.2-5.4 mg/dL), total calcium at 5.9 mg/dL (normal: 8.8 to 10.0 mg/dL), hyperphosphorhemia at 6.4 mg/dL (normal: 2.3 to 4.7 mg/dL) and 2.5 pg/mL intact-parathyroid hormone (normal: 11.0 to 67.0 pg/mL). Alkaline phosphatase, magnesium, calcitonin, serum thyroid and thyroid-stimulating hormone levels were normal.

Brain computed tomography demonstrated a symmetric, extensive, bilateral calcification of the basal ganglia, centrum semiovale, and bilateral dentate nuclei of the cerebellum, typical for Fahr’s syndrome. The red nucleus and substantia nigra appeared normal. The diagnosis of Fahr’s syndrome, secondary to hypoparathyroidism was posed. A preponite meningioma was also found (Figure 1 A and B).

The electrocardiogram showed normal QTc interval and the interictal electroencephalography and electromyography were normal. Bone densitometry showed increased bone mineral density.

After the patient was treated with oral calcium and active vitamin D (1- alphahydroxy vitamin D3), serum calcium levels returned to normal and seizure attacks ceased progressively resulting in stopping antiepileptic drugs.

Conclusions:

These cases illustrate the importance of search for disrupted phosphocalcic metabolic but also emphasizes the importance of the role of neuro-imaging in patients with new-onset epileptic seizures in order to detect hypocalcemia secondary to hypoparathyroidism.

Bibliography

3. Engin Deniz Arslan, Fazil Yılmaz, Süleyman Koca, Burcan Topal, Engin Arslan, Sercan Kahveci, Fahr’s Disease and Its Relationship with Hypoparathyroidism: Case Report, Department of Emergency, Ankara Numune Training and Research Hospital, Ankara, Turkey; JEMCR 2013, 4: 95-7 doi: 10.5005/jemcr-2013.50954