

CASE REPORT

Cognitive impairment and epilepsy seizure caused by hypoparathyroidism

Titlic M¹, Tonkic A², Jukic I², Filipovic-Grcic P¹, Kolic K³

Department of Neurology, Split University Hospital, Split, Croatia. marina.titlic@gmail.com

Abstract: We report a 46-years-old man with idiopathic hypoparathyroidism who presented with gradually cognitive impairment and generalized tonic clonic epilepsy. Computed tomography (CT) and magnetic resonance imaging (MRI) of brain showed calcification in the basal ganglia, thalamus and cerebral white matter. Calcium level was low and phosphor was increased in serum, but parathormon level was low. Following introduction of replacement therapy, cognitive functions improved as well. After treatment with calcium and vitamin D supplementation he showed clinical, cognitive impariment and laboratory improvements (Fig. 2, Ref. 13). Full Text (Free, PDF) www.bmj.sk.

Key words: hypoparathyroidism, cognitive impariment, CT, MRI, calcium, phosphor.

Hypoparathyroidism can result from lack or decreased secretion of parathormon (PTH) or from defective responsiveness of effector organs to PTH. Neurological signs of hypoparathyroidism include symptomatic or latent tetany, seizures, hemiparesis, difficulty in speaking, ischemic attacks, in case of basal ganglia and cerebral calcifications also choreatetosis, parkinsonism and cerebellar syndrome (1–3).

Clinical, laboratory and radiological features of cases of hypoparathyroidism make basis of diagnostics of the primary disease with various neurological signs. Latoratory findings revealed severe hypocalcemia and hyperphosphatemia, and serum low levels of parathyroid hormone. Computed tomography (CT) and magnetic resonance imagind (MRI) of brain showed calcification in the basal ganglia, thalamus, cerebral white matter and cerebellum (4–7).

We report a very rare case of a patient with hypoparathyroidism who manifested cognitive impairment and epilepsy seizure, We compare our case with others in medical literature.

Case report

46-year man is hospitalized because of two generalized tonic clonic seizures. The patient's friends who escorted him reported gradual decrease of his intellectual functions over the last two years. He consumed alcohol moderately and smieks up to 60 cigarettes a day. Neurological signs are dysmetria and tongue

bite. The Chvostek's and the Trousseau's signs are negative. The basic blood laboratory tests are normal. The heart and lungs x-ray shows lime saturation of medical and apical, right handside, parts of the pleura.

EEG showed the continuous generalized 2–4 Hz spike and wave complexes. CT of the brain shows infra- and supratentorially, temporo-parieto-occipitally, large lacunar and punctiform intracerebral calcifications. The basal ganglia and the corona radiata are completely saturated with lime salts. The ventricular structures are centrally located and normally shaped (Fig. 1).

MRI of the brain shows bilateral, in the area of the coronae radiatae, globus pallidus, and posterior parts of the thalamus, lamilar calcificates that indicate lime saturation of the basal ganglia blood vessel walls. More marked are cortical atrophic changes and periventricular leukomalation (Fig. 2).

Additional diagnostical laboratory tests were performed: serum calcium level 1,15 mmol/l (reference values 2.3–2.8 mmol/l), phosphorus 2.57 mmol/l (refernce values 0.6–1.5 mmol/l), magnesium level 0.70 mmol/l (refence values 0.8–1.1 mmol/l), ionized calcium 0.59 mmol/l. Parathormon is under 1.0 pg/l (reference values 6.2–29.0 pg/ml). Blood cortisol level 372.2 (reference values 260–720), aldosterone level 0.13 mmol/l (reference values 0.10–0.87), testosterone 16.2 nmol/l (reference values 10.4–41.6), DHEA-SO₄ 7.1 μmol/l (reference values 0.95–11.9), and androstendione 6.6 mmol/l (reference values 1.1–10.8), estradiol 0.383 nmol/l (reference values <0.20 in men), progesterone 0.4 (reference values 0.4–4.0 in men), prolactin 246 mIU/l (reference values 50–440). Thyroidea hormones: T3 1.3 nmol/l (reference values 1.3–2.5), T4 107.4 nmol/l (reference values 69–141), TSH 0.422 mIU/l (reference values 0.63–4.19).

The upper abdomen sonography is normal. Electromyographical test for tetany is negative. Echographic examination shows the thyroid gland to be normally homogenous, of isoecho-

¹Department of Neurology, Split University Hospital, Split, ²Department of Internal Medicine, Split University Hospital, Split, and ³Department of Radiology, Split University Hospital, Split, Croatia

Address for correspondence: M. Titlic, MD, PhD, Dept of Neurology, Split University Hospital, Spinciceva 1, Split 21 000, Croatia.
Phone: +385.21.556426, Fax: +385.21.556675

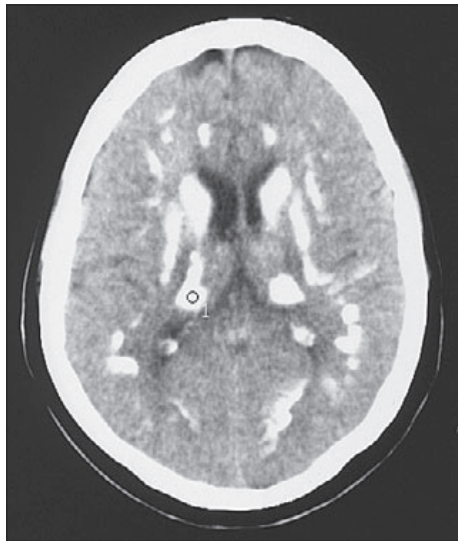


Fig. 1. CT of the brain – diffusely situated numerous calcificates (periventricular, subcortical, centrum semiovale).

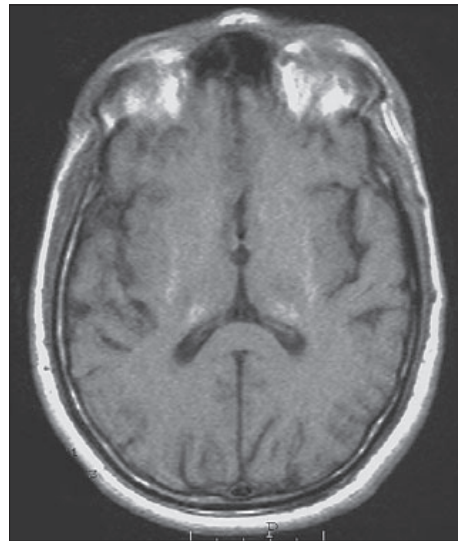


Fig. 2. MRI of the brain – T2 weighting – periventricular and subcortical calcifications.

genous structure, free of nodes. The parathyroid glands are echographically normal. Biomicroscopy established bilateral presenile cataract. Psychological tests indicate marked mental deterioration, dominating in the fields of thinking, planning and organization of mental activities, with damaged flexibility. Simple visual perception is preserved, marked deficits are noticed in complex spatial functions, visoperceptive organization and visomotor coordination disorders, with mental and motoric deceleration. There are markedly decreased learning processes with recalling difficulties, but memory is preserved at the recognition level. There is high anxiety and impulsivity, and lowered frustration tolerance.

After treatment with calcium and vitamin D supplementation he showed clinical, neurophysiological and laboratory improvements.

Discussion

Clinical, laboratory and radiological features of our case of hypoparathyroidism presenting with pure neurological disease are described.

Hypoparathyroidism occurs due to insufficient production of parathyroid hormone to maintain extracellular calcium levels within the normal range. The acute clinical symptoms and signs of hypoparathyroidism are those of hypocalcaemia, ranging from tingling and numbness of extremities to intractable seizure (7). Seizures may occur at any age and are usually generalized tonic-clonic ones (1, 8). Generalized tonic-clonic seizures are often secondary signs of brain lesion, but rarely makes a cause of hypoparathyroidism. Our patient had two sequential seizures, which indicates a brain lesion, requiring radiological examination, CT and MRI. CT better defined the site and extent of the calcification which may be found in both idiopathic and postoperative hypoparathyroidism, in symptomatic

or asymptomatic patients. MRI seems to be capable of depicting the various stages of calcium deposition on the basis of the presence of reduced or increased SE T2 weighted images (3). CT and MRI of the brain of our patient agrees with the above and adds to the clinical diagnosis. Laboratory findings revealed severe hypocalcemia and hyperphosphatemia, and parathyroid hormones levels were low in serum. This is in line with other authors' researches (4, 5). The usual EEG findings in patients with generalized tonic clonic seizures, as well as the cataract, have already been described in patients with brain calcificates caused by hypoparathyroidism. Clinical, laboratory and radiological features of our case of hypoparathyroidism are further completed with neuropsychiatric tests that showed a cognitive impairment. Pathogenesis of cognitive and motor changes in Fahr's disease and hyperparathyroidism with calcification of the brain are based in a dysfunction of corticobasal connections and their interhemispheric relations. This defines a subcortical dementia secondary to mineral deposits in subcortical structures (10, 11). The cognitive impairment and neurological deficit incidence in patients with intracerebral calcification caused by hypoparathyroidism is very much present, according to other author's researches (12, 13). Joint cognitive impairment and epilepsy seizure in adult patients require additional radiological and laboratory examinations for a possible hypoparathyroidism.

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