DELAYED ONSET OF DEMENTIA AND PARKINSONISM IN A POSTOPERATORY HYPOPARATHYROIDISM CASE

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ABSTRACT

Introduction. Brain imaging is mandatory for dementia diagnosis. In various guidelines and clinical studies MRI stands as the first choice, with the highest sensitivity and specificity. Our goal here is to show the exception to the rule. CT remains an imaging method important for diagnosis of pathology characterised by basal ganglia calcifications.

Case history. A female patient,aged 60 years, came to the hospital with symptoms of mild cognitive impairment and parkinsonian syndrome. A medical history of hypertension and total thyroidectomy were recorded. The first imaging option for patient was to perform a brain MRI, which did not bring any information about the cause of dementia. Fortunately a CT was performed as well and bilateral basal ganglia massive calcifications were seen, which were due to total thyroidectomy/parathyroidectomy in the past.

Conclusion. To diagnose secondary congnitive disorders of basal ganglia calcifications a standard head CT seems to be the method of choice.

Keywords: dementia, Parkinson disease, brain imaging

INTRODUCTION

Basal ganglia calcification is seen in approximately 1% of all CT scans of the brain and is often an incidental imaging finding without clinical significance. The most common cause of pathological basal ganglia calcification is hypoparathyroidism (1). We report a case of postoperatory hypoparathyroidism with bilateral extensive intracranial calcification and only mild clinical symptoms.

CASE REPORT

We report a case of a 60 year old woman admitted in the Neurology Department of Colentina Hospital for dysarthria and dysphagia to both solids and liquids, that lasted ten hours and was resolved before presentation. From her past medical history we recall hypertension (well controlled with pharmacological therapy) and a total thyroidectomy 10 years ago for toxic multinodular goiter followed by

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hormone replacement therapy (T4-levothyroxine). The patient's clinical course after surgery is without notable events. She reported a calcium supplementation for 4 months after the procedure, which she stopped by her own decision. The neurological examination found a neurocognitive disorder (mini-mental state examination 22/30) and extrapyramidal signs (mild rigidity and bradykinesia). The family did not report any change in the pacient's behavior and no familly history of dementia, movement disorders or other neurological or psychiatric diseases.

The first step to evaluate the patient was to perform an MRI (Fig. 1), that did not show any changes to explain the patient's symptoms.

Due to the thyroid surgery history, a standard head CT was performed. This revealed symmetrical calcification of the basal ganglia (head and tail of caudate nucleus, putamen), thalamus, cerebellum, and also radially disposed subcortical white matter calcifications (Fig 2, 3). However, contrary to what is traditionally said for massive brain calcifications, the skull radiography was normal and no calcium depositswere seen (Fig. 4). The blood panel revelead that his serum calcium was 2.07 mg/dL (3,8-5.6 mg/dL), PTH 8,7 pg/dL (15-65 pg/mL), serum phosphate 6,7 mg/dL (2,7-4,5 mg/dL), TSH was normal 2,2.

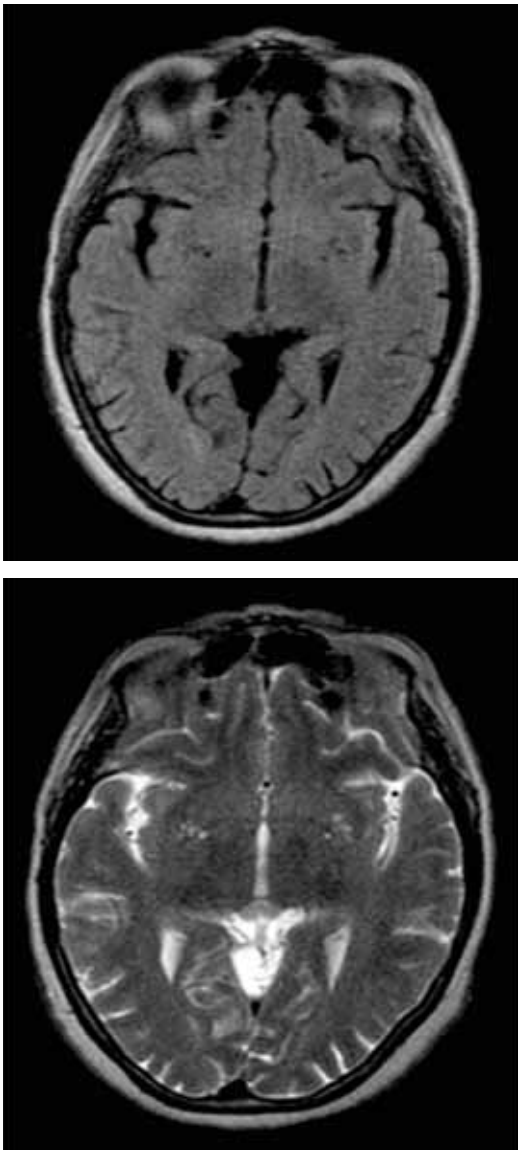

FIGURE 1. *Brain MRI-normal*

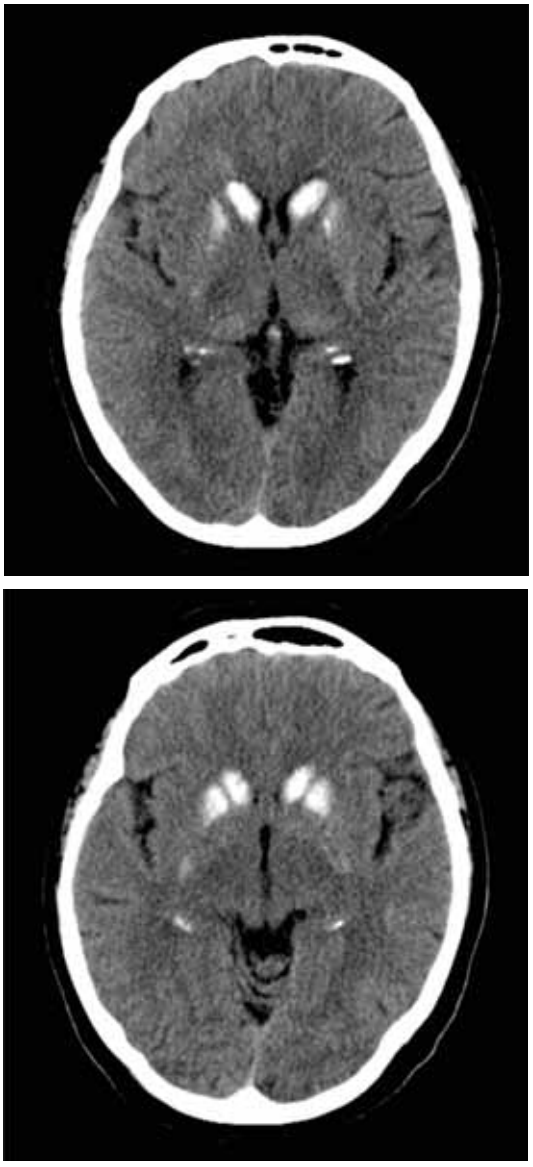

FIGURE 2. *Brain computed tomography revealed severe calcification of basal ganglia*

With these investigations, the clinical presentation of our patient was attributed to hypoparathyroidism. We treated the patient with intramuscular calcium gluconate and calcitriol.

The patient returned to control after three months where we relief on the neurological examination improving on the parkinsonian syndrome and the MMSE was 28/30. Repeated CT showed

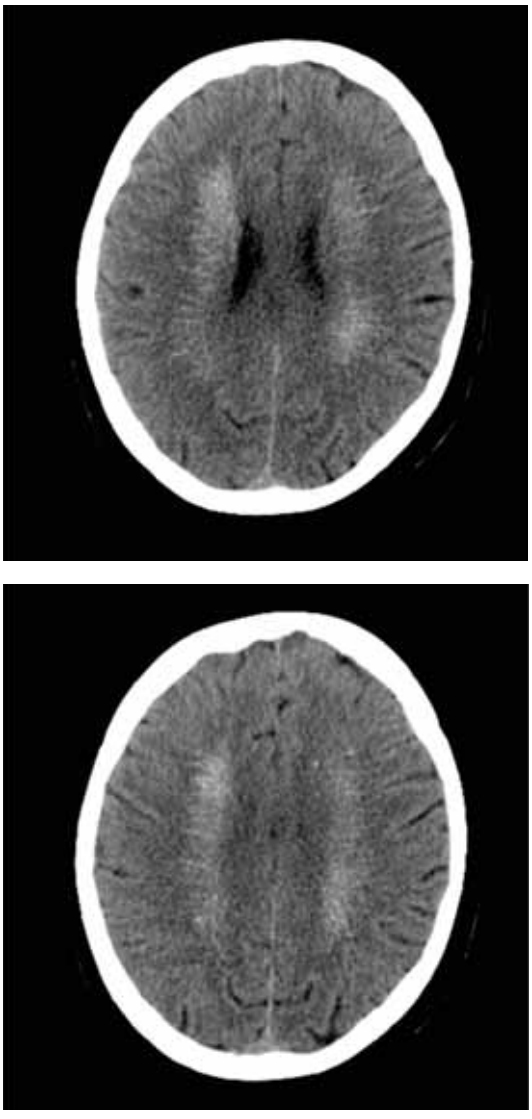

FIGURE 3. *Computed tomography revealed radially disposed subcortical white matter calcifications*

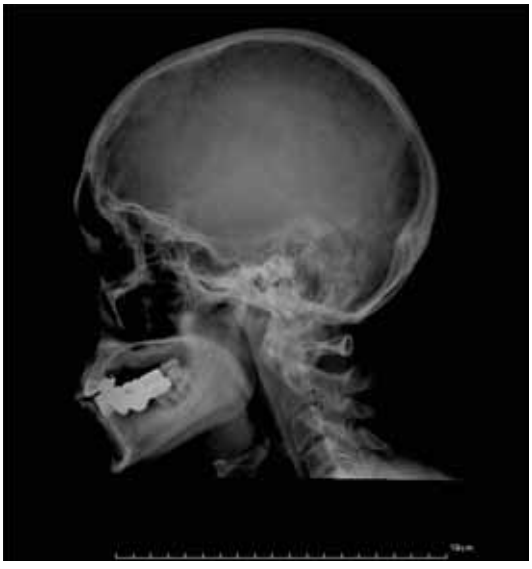

FIGURE 4. *Normal skull radiography*

that calcifications in the basal nuclei, thalamus and cerebellum decreasedin size and density and the same did the radially disposed subcortical white matter calcifications.

DISCUSSION

There are several types of hypoparathyroidism: postsurgical (the most common cause, associated with oncological surgery of the neck, total thyroidectomy, parathyroidectomy), familial, autoimmune or idiopathic. The biochemistry panel reveals hypocalcemia, hyperphosphatemia and low or undetectable PTH levels. There are two clinical situations that can occur postoperatively: tetany can appear 1 or 2 days after the intervention (yet 50 % of pacients have a good recovery so they do not need further therapy) or, in some pacients, hypocalcemia is obvious only years after the procedure. The first presumpsion in such pacients with a scar on the neck and hypocalcemia is surgical hypoprathyroidism (2).

Neurological manifestations of hypocalcemia include tetany, seizures, headaches and papilledema, basal ganglia dysfunction with a variety of hypokinetic and hyperkinetic movement disorders (parkinsonism), behavioral and neurasthenic manifestations.

The most common location of intracranial calcifications in hypoparathyroidism is the basal ganglia (3). In our pacient, the calcifications extend past the common location and are seen in the subcortical white matter.Few case reports regarding postoperative hypoparathyroidism with white matter subcortical calcification are to be found in the literature (4).

Causes of symmetrical calcifications of the basal ganglia can be summarized as follows:

- idiopathic – aging and Fahr's disease;
- toxic – carbon monoxide poisoning, lead poisoning, radiation therapy and chemotherapy;
- infectious –TORCH, CNS tuberculosis, AIDS, neurocysticercosis, CNS toxoplasmosis;
- metabolic – parathyroid disorders and hypoxia at birth;
- inherited-mitochondrial diseases, Cockayne syndrome and Hallervorden-Spatz syndrome;
- methotrexate treatment.

The mechanisms of extrapyramidal signs of idiopathic hypoparathyroidism remain unclear. Parkinsonism associated with basal ganglia calcification differs clinically from idiopathic parkinsonism. Although suffering from extrapyramidal signs

(which may be associated with basal ganglia calcification), the pacientsare non-responsive to levodopa treatment (5).

Dilemma of the clinician when facing aneurocognitive deficit and parkinsonism in a postoperatory hypoparathyroidism case is what we perform first– MRI or CT?

In the study of Colliot(7) it was suggested that MRI should be the standard imaging in early diagnosis of cognitive disorders (e.g. Alzeimer's disease). In particular, magnetic resonance imaging (MRI) allows detecting different types of structural and functional abnormalities at an early stage of the disease (such as hippocampal atrophy).

On the other hand, Sarmiento (8) recommended as a diagnostic method in detriment of MRI the CT for calcified lesions of basal ganglia, because the group of patients with MRI have an unspecific appearance and the CT can be identified and characterized.

CONCLUSION

The best choice in detecting intracranial calcifications are computed tomography, rather that conventional skull radiographs or MRI (6).

The treatment of hypocalcemia should be directed at the underlying disorder. In all cases, replacement with exogenous calcium should be instituted (3).

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