Gyriform calcifications after ischemic stroke in a patient with primary hyperparathyroidism

Calcifications gyriformes après infarctus cérébral chez un patient souffrant d’hyperparathyroïdie primaire

Cerebral calcification has been observed in association with diverse lesions such as tumors, infections, and vascular malformations [1]. Vascular calcifications are a frequent finding in brains of elderly patients and has also been observed in association with disturbances of calcium and phosphate metabolism [1, 2]. Calcification of cerebral infarcts is a very rare occurrence [1]. Hypercalcemia and cerebral calcifications may be predisposing factors of poststroke seizures.

A 44-year-old man was hospitalized from 01.27.2017 to 02.10.2017 for a convulsive seizure followed by a regressive left side motor palsy.

In his history, we noted the implantation of aortic and mitral mechanical valve prostheses in June 2016. The surgery had been simple, with no postoperative neurological deficit. Hypercalcemia on parathyroid adenoma was discovered preoperatively in May 2016 (serum calcium level at 5.39 mmol/L [2.15–2.50], serum parathyroid hormone level at 85.15 pmol/L [1.6–6.9]). A surgical indication on this adenoma had been given by the endocrinologists at a distance from cardiac surgery (Parathyroidectomy postponed to at least 1 month) but the patient had not carried out the prescribed endocrinological follow-up.

7 months after cardiac surgery, in January 2017, the patient presented sudden onset of headache followed by a left upper limb partial seizure, secondarily generalized, followed by a left predominantly brachiofacial hemiparesis. He was cared for at the local hospital emergency room and then transferred to our facility. The treatment at entrance included Furosemide 40 mg/d, Bisoprolol 2.5 mg/d, acetylsalicylic acid 75 mg/d, lanzoprazole 40 mg/d, fluindione 50 mg/d.

On arrival in service, there was a complete recovery of the postcritical deficit. The clinical examination was unremarkable. Biological tests found an INR at 3.4 (TP: 23.0%) under treatment with VKA, hypercalcemia (serum calcium level 3.26 mmol/L [2.15–2.50]) with hypophosphoremia (serum phosphorus level 0.43 mmol/L [0.8–1.45]), hyperparathyroidism with serum parathyroid hormone level at 29 pmol/L (1.6–6.9). The remainder of the biological check-up was unremarkable.

The brain CT scan performed in the emergency department found a spontaneous gyriform cortical hyperdensity at the right external temporal level, next to a subcortical hypodensity; The appearance was suggestive of a calcified laminar necrosis on the sequelae of a right external temporal stroke (see figures 1 and 2). The control CT carried out 2 days later noted a stability of the calcified cortical hyperdensity, with no argument for bleeding. The brain MRI noted an aspect of right temporal laminar necrosis on old ischemic sequelae without hemorrhage in T2*-weighted sequences (figures 3 and 4). The TOF-weighted sequences noted distal arterial branches of the right middle cerebral artery less visible than left ones. It was concluded to cortical calcifications on old silent ischemic injury, which may have occurred during its cardiac surgery in May 2016, in a context of chronic hypercalcemia.

The EEG found an asymmetric pattern, slower and depressed on the right hemisphere, without epileptic element. The cervical ultrasound noted the presence of a solid-cystic, extra-thyroid nodular formation next to the right lobe, whose aspect was compatible with a right parathyroid adenoma.

Discussion

Our case presents a patient with underlying primary hyperparathyroidism (PHP) who suffered from silent cerebral infarction with prolonged hypercalcemia. A few months later, the patient developed post-infarction seizures and intracranial calcifications were discovered.

The CT scan has a high sensitivity for detecting calcium deposits in the brain [1, 3]. Small, focal calcifications have been associated with vascular malformations (arteriovenous malformation, cavernous angioma), aneurysms, brain tumors, and infections (for example, toxoplasmosis and cytomegalovirus infection) [1]. Calcifications restricted to the basal ganglia have been associated with different conditions among whom parathyroid disorders [1]. Swartz et al. described two patients with renal failure and secondary hyperparathyroidism who had basal ganglia and diffuse subcortical calcifications [4]. Small calcium deposits are more commonly seen as the residua of intracerebral hematomas [5]. Cortical and white matter calcification are rarely seen after ischemic stroke [4, 7]. In different reported cases, a CT scan showed parenchymal calcifications 4 months [6]; 6 weeks [1], 45 days [7], and even as early as 18 days [2] after acute stroke. However, most calcifications are typically discovered years after the acute event [8]. Our patient had a ischemic stroke, with
calcification occurring in a relatively short time (probably a few months).
Intracranial calcification may have been promoted by hypercalcemia. We postulate that our patient’s elevated calcium level probably accelerated and exaggerated the process of calcification in the presence of a disrupted blood-brain barrier. Cerebral calcifications, combined with hypercalcemia itself, may have played a role in the development of poststroke epilepsy in this patient. In a recent study, the number of cases with epilepsy secondary to stroke was approximately 11%, and cortical involvement and large lesion size increase the risk [9]. Patients with ischemic stroke concurrent with PHP should receive parathyroidectomy as early as possible to avoid potential predisposing factors of poststroke seizures.

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**Disclosure of interest**

The authors declare that they have no competing interest.
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References


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