Rare case of secondary hypoparathyroidism with extensive intracranial calcifications

Clinical History:

A 68 year old man was brought by his relatives to the emergency department with mild paresthesia, fatigue, weakness, headache and cramps of the calves. He was also complaining for episodes of loss of consciousness for the last three months.

Imaging Findings:

From his personal history, Ca of larynx was diagnosed two years ago, graded T2 N2a which was treated with topical radiotherapy. The brain CT at that time was normal. There was no surgical intervention in the neck area for any reason. On clinical examination he was unwell with difficulty on erection and on walking and rigidity of the large joints. He also had Barre and Babinski on the right. Further investigation included a new brain CT, in order to exclude metastases, and full blood examination. CT was negative for metastases but revealed diffuse calcifications at the basal ganglia, at the white matter and at the cerebellum bilaterally. From blood investigation he had hypocalcaemia (Ca 4.6 mg/dl), hyperphosphoremia (P 7.1 mg/dl), low level of PTH (8 pg/dl). Clinical examination revealed also positive Chvostek and Trousseau signs (hypo clinical tetany). The ECG showed mild prolongation of Q-T due to hypocalcaemia and from the eyes examination bilateral cortical cataract. The diagnosis of Hypoparathyroidism after radiation therapy was established. That was based on the plasma level of the calcium, phosphorus and PTH.

Discussion:

Hypoparathyroidism after radiation therapy is a very rare condition with non-typical presentation and neurological findings, usually delays and makes the diagnosis difficulty. Vascular degeneration of exopyramidal system is a possible cause of calcium crystal deposit and clinical manifestations are usually due to increase of neuromuscular stimulation. The complications may be serious and rarely fatal (tetany can cause respiratory obstruction) Also, stunted growth, malformed teeth, and slow mental development can occur if Hypoparathyroidism develops in childhood, while there is an increased risk of pernicious anemia, Addison's disease, cataract development, and Parkinson's disease. CT is the diagnostic modality of choice for the depiction of intracranial calcifications. The goal of treatment is to restore the calcium and mineral balance within the body. Oral calcium carbonate and vitamin D supplements are usually life-long therapy. Blood levels are measured regularly to make sure that the dose is correct. A high-calcium, low-phosphorous diet is recommended. Others causes of Hypoparathyroidism are: surgical extraction or destruction of the glands, congenital insufficiency, idiopathic, neonatal presentation of DiGeorge syndrome (dysgenic thymus and parathyroid glands), low level of magnesium, autoimmune cause (MEN 2) and
deposition of Cu or Fe or Al. Causes of intracranial calcifications at basal ganglia are: primary or secondary Hypoparathyroidism, metabolic disorders (Fahr disease), inflammatory conditions (toxoplasmosis, CMV), congenital or neurodevelopment diseases, carbon monoxide poisoning and hyperparathyroidism.

**Differential Diagnosis List:** Extensive intracranial calcifications due to secondary hypoparathyroidism after radiation therapy.

**Final Diagnosis:** Extensive intracranial calcifications due to secondary hypoparathyroidism after radiation therapy.

**References:**

Figure 1

Description: 1a.jpg, calcifications at the cerebellum bilaterally. Origin:

Description: 1b.jpg, calcifications at the dentate nucleus. Origin:
**Figure 2**

**Description:** 2a.jpg, diffuse, marked, calcifications bilaterally at the territory of the head of caudate nucleus, at the putamen, and at globus pallidus. **Origin:**
Description: 2b.jpg, diffuse calcifications at the territory of the thalamus and internal capsule. Origin:
Description: 3a.jpg multiple areas of calcifications at the white matter and corona radiata. Origin:
Description: 3b.jpg, calcifications at the gray-white junction’s areas. Origin: