Tertiary hyperparathyroidism presenting as posterior reversible encephalopathy syndrome

A man with hereditary hypophosphatemic rickets presented with seizures and coma. Brain MRI revealed edema typical of posterior reversible encephalopathy syndrome (PRES) and ECG showed ST elevations (figure). Coronary angiography was normal. Blood pressure was 165/90 mm Hg on admission; other known causes of PRES were absent. Severe hypercalcemia (3.83 mmol/L) was detected while parathyroid hormone was highly elevated (1,119 pg/mL). Sonography revealed an enlarged parathyroid gland. Subtotal parathyroidectomy led to prompt normalization of parathyroid hormone and calcium. The patient recovered fully.

Tertiary hyperparathyroidism and hypercalcemic crises can be rare sequelae of long-term substitution treatment in hereditary hypophosphatemic rickets.1 Severe hypercalcemia has been recognized as a trigger for PRES (and ST elevations), yet the pathophysiologic mechanisms involved are unknown.2

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