Calcium carbonate overdose

Hypercalcaemia, acute cerebral infarction and reversible cerebral vasoconstriction syndrome: case report

A 59-year-old woman developed hypercalcaemia, acute cerebral infarction and reversible cerebral vasoconstriction syndrome (RCVS) following accidental overdose of calcium carbonate supplements [*time to reactions onset not stated*].

The woman presented to the emergency department via emergency medical services after she was found unresponsive in her home. Her medical history included essential hypertension, hypothyroidism and dyspepsia. Four months prior, she was hospitalised for left tibia and fibula fractures after a fall from standing height. During that time, she was hypercalcaemic, which was rapidly corrected with IV fluids. Specified aetiology for hypercalcaemia was not found during that hospitalisation. After arriving to emergency department, her Glasgow Coma Scale score was 6. Eye opening or verbal response to tactile or verbal stimuli was not observed. Withdrawal to pain was observed only in the left upper extremity, and she had right hemiplegia. Cranial nerve examination revealed right gaze preference with spontaneous, conjugate and intermittent roving eye movements. Thereafter, she was intubated for airway protection. She had severe hypertension with systolic BP ranging from 200-230mm Hg and diastolic BP ranging from 105-125mm Hg. Laboratory tests revealed calcium level 17.3 mg/dL and mildly elevated protein with normal WBC count, RBC count and glucose. An MRI (with diffusion-weighted images and apparent diffusion coefficient maps) demonstrated acute infarction of the left parieto-occipital cortex, hippocampus, anterior temporal cortex and insula and left posterior thalamus with no vasogenic oedema on T2 fluid-attenuated inversion recovery. A CT angiography (CTA) of the neck and head showed multifocal intracranial vascular narrowing. Cerebral angiography was delayed by 2 days expecting reversibility of findings on CTA in parallel with the observed clinical improvement. Cerebral angiography demonstrated largely normal appearing intracerebral vessels and almost complete resolution of the focal narrowing, which was observed during CTA. Considering her altered mental status, focal neurologic deficit and hypercalcaemia, there were broad differential diagnoses. She started receiving empiric unspecified broad-spectrum antibacterials until her lumbar puncture excluded CNS infection. Her son had brought her supplements and medications from home, which included a nearly empty bottle of calcium carbonate, which was purchased shortly before the presentation. It was reported that she had been ingesting up to 10 calcium carbonate tablets several times a day [dose not stated] before the admission.

Along with ventilator management and general intensive care, the woman was treated with broad-spectrum parenteral unspecified antibacterials. She also received clevidipine to control BP with gradual resolution of hypertension over the several days after the admission. Permissive hypertension with systolic BP up to 180mm Hg was allowed for effective cerebral perfusion. Her serum calcium levels normalised with crystalloid solution treatment. Following negative cultures, antibacterials were stopped. She started receiving aspirin and unspecified statins for secondary prevention. For the treatment of RCVS, she started receiving a 14-day course of nimodipine. Considering the rapid correction in serum calcium levels and negative investigations for alternative aetiology, her hypercalcaemia, acute cerebral infarction and reversible cerebral vasoconstriction syndrome were thought to be related to accidental calcium carbonate overdose. Subsequently, she was extubated, following which physical, occupational and speech therapies were started. Her mental status improved, and she started following commands. She continued to have right-sided weakness and expressive aphasia. Over the course of hospitalisation, her neurologic deficits improved with a near-complete resolution at the time of discharge. She did not require inpatient rehabilitation, and was directly sent home after discharge.

Schertz AR, et al. Reversible cerebral vasoconstriction syndrome (RCVS) caused by over-the-counter calcium supplement ingestion. BMJ Case Reports 14: No. 1, 28 Jan 2021. Available from: URL: http://doi.org/10.1136/bcr-2019-233877 803542016