Early postoperative carbamazepine-induced tetany in a patient with trigeminal neuralgia

Madam,
Carbamazepine-induced hypocalcemia is a known association but is usually mild and asymptomatic.[1,2] However, its conversion to severe symptoms such as tetany is extremely rare and is not described in literature. We report a case of carbamazepine-induced tetany in a patient with trigeminal neuralgia and its rapid reversibility following administration of intravenous calcium gluconate.

A 48-year-old male (65 kg, 170 cm) without pre-existing comorbidities underwent microvascular decompression (MVD) for trigeminal neuralgia refractory to oral carbamazepine 200 mg twice and pregabalin 75 mg once daily respectively. The surgery and anesthesia periods were uneventful. Intraoperative bleeding was minimal and blood products were not transfused. At the end of the surgery, the patient was fully conscious, oriented, and hemodynamically stable. The patient was shifted to the intensive care unit (ICU) for observation.

In the ICU, 4 h later, the patient complained of tingling sensations in both hands which distributed rapidly over his face and also in the lower limbs. Within few minutes, his hands developed severe spasticity (carpal spasm) characteristic of tetany. Chvostek’s and Trousseau’s sign were positive. There were no signs of meningeal irritation or hyperventilation.
Hemodynamic parameters were within normal limits. An arterial blood gas (ABG) analysis was done initially which showed normal acid-base status except decrease in ionized calcium (0.65 mmol/L). Computed tomography (CT) of brain was also done which excluded any new onset pathology. Initially, 10 mL of 10% calcium gluconate was administered intravenously over 15 min followed by another 10 mL over another 15 min titrated to achieve ionized calcium between 1 mmol/L and 1.2 mmol/L. The symptoms subsided dramatically after administration of calcium gluconate.

Later on, laboratory reports showed normal serum sodium, potassium, magnesium but decreased corrected calcium level (6.2 mmol/L). The serum levels of thyroid and parathyroid hormones including kidney and liver function tests were within normal limits. In addition, serum level of 1, 25-dihydroxyvitamin D was also normal (43 ng/L, range 15–45 ng/L); however, there was decreased 25-hydroxyvitamin D level (7.2 μg/L, range 14–70 μg/L). Repeat analysis of serum calcium (post calcium gluconate infusion) was done which increased to 9.2 mmol/L. The patient was started on oral calcium 1 g and calcitriol 0.25 mcg once daily. Rest of the patient’s hospital stay was uneventful.

Carbamazepine-induced hypocalcemia has been linked to long standing effect of the drug on vitamin D catabolism and inhibition of cellular response to parathyroid hormone[1-3]. In our patient, the unusual manifestation of severe symptomatic hypocalcemia in the form of tetany could have been due to stress related to surgery and anesthesia. In addition, genetic susceptibility and alteration in calcium-protein binding could have been other contributing factors.

Other known causes of tetany such as hypokalemia, hypomagnesemia, hyperventilation, and alkalosis were excluded in our patient. Our patient’s dietary intake was normal prior to operation. The serum concentration of phosphate and parathyroid hormone including urinary calcium and phosphate excretion were also normal. Osteodystrophy in the bones of hands and feet which could suggest pseudohypoparathyroidism was also ruled out. Any major intracerebral event following the surgery could have been another aggravating factor, but was ruled out by a normal computed tomography (CT) scan.

Thus, in our case, there was a decrease of both ionized calcium and corrected serum calcium levels even though isolated decrease of ionized calcium has been shown to cause tetany.[4] As anesthesiologists, one should be vigilant of this potential complication of carbamazepine therapy in the perioperative period. The timely diagnosis and prompt intervention can prevent undue catastrophes. Moreover, it can be said that the supplementation of calcium and vitamin D is effective in management of such patients.[5]

Declarations of patient consent
The authors certify that they have obtained all appropriate patient consent forms. In the form, the patient has given her consent for her images and other clinical information to be reported in the journal. The patient understands that name and initial will not be published and due efforts will be made to conceal identity, but anonymity cannot be guaranteed.

Financial support and sponsorship
Nil.

Conflicts of interest
There are no conflicts of interest.

References
1. Hoikka V, Alhava EM, Karjalainen P, Keränen T, Savolainen KE, Riekkinen P et al. Carbamazepine and bone mineral metabolism. Acta Neurol Scand 1984;70:77-80.
2. Tjellesen L, Nilas L, Christiansen C. Does carbamazepine cause disturbances in calcium metabolism in epileptic patients? Acta Neurol Scand 1983;68:13-9.
3. Mintzer S, Boppana P, Toguri J, DeSantis A. Vitamin D levels and bone turnover in epilepsy patients taking carbamazepine or oxcarbazepine. Epilepsia 2006;47:510-5.
4. Williams A, Liddle D, Abraham V. Tetany: A diagnostic dilemma. J Anaesthesiol Clin Pharmacol 2011;27:393-4.
5. Misra A, Aggarwal A, Singh O, Sharma S. Effect of carbamazepine therapy on vitamin D and parathormone in epileptic children. Pediatr Neurol 2010;43:320-4.

How to cite this article: Singh S, Roy H, Singh GP, Khandelwal A. Early postoperative carbamazepine-induced tetany in a patient with trigeminal neuralgia. J Anaesthesiol Clin Pharmacol 2018;34:405-6.

© 2018 Journal of Anaesthesiology Clinical Pharmacology | Published by Wolters Kluwer - Medknow

Access this article online
Quick Response Code:  
Website: www.joacp.org
DOI: 10.4103/Joacp.JOACP_245_17