Letters to Editor

Isolated Quadriceps Weakness: A Case of Hypokalemic Periodic Paralysis

Dear Editor,

Episodic weakness of muscles, which is unrelated to exercise, can be seen in genetic autosomal dominant channelopathies, secondary causes such as thyrotoxicosis and neuromuscular junction diseases. Hypokalemic periodic paralysis (HPP) is a channelopathy affecting the calcium channels in the body.

We present the case of a 25-year-old male who came to the OPD with three episodes of bilateral lower limb weakness in the past 1 week. Each episode lasted for 6–8 h, with this current episode lasting for 14 h. There was no history suggestive of altered sensorium, convulsions, visual, respiratory, bulbar weakness or thyroid dysfunction. There was no preceding history of fever, diarrhea, vomiting, trauma, heavy carbohydrate meal, prior strenuous exercise, recent vaccination or medication like diuretics. No family member had similar symptoms.

On examination, his vitals were within normal limits, all cranial nerve functions, sensory and motor functions in the upper limb were intact. In both lower limbs, there was decreased range of motion (ROM) at the level of the knees, the patient was unable to extend the knee from 90° to 0°. The power of knee extension was 3/5. Knee jerk and plantar reflex (flexor) were diminished on both sides. The tone of limbs and all sensory sensations were intact. There were no signs of cerebellar dysfunction or meningeal irritation. Systemic examinations revealed no obvious abnormalities.

Based on the history and clinical findings, HPP, Guillain–Barre syndrome (GBS), and subacute combined degeneration (SCD) of the spinal cord were suspected, and relevant tests were ordered. Laboratory investigations showed a potassium level of 3.25 mEq/L, sodium level of 135.9 mEq/L, and creatine kinase was 201 U/L. Baseline laboratory values were normal. Blood gas analysis showed pH of 7.34, pCO₂ of 36 mm Hg, and HCO₃ of 23 mmol/L. Thyroid function, liver function, kidney function, and Vitamin B₁₂ levels were within normal limits. His ECG did not show any changes associated with hypokalemia. Nerve conduction studies for bilateral femoral nerves were normal, but electromyography (EMG) of both the vastus lateralis and vastus medialis showed a myopathic pattern of weakness.

Based on the above reports, the patient was started on intravenous potassium for 3 days that lead to clinical recovery and also biochemical improvement to some extent. Keeping in view of the improvement, we did a repeat EMG, which showed normal amplitudes and durations. Hence, this allowed us to revert to our differential diagnosis of HPP.

HPP is seldom considered as the diagnosis in a patient presenting with weakness and decreased muscle power. Since HPP only affects 1 in 100,000 population, there are many clinical differential diagnoses such as GBS, acute transverse myelitis, poliomyelitis, stroke, and SCD which are also considered. Patients with HPP often present with flaccid muscle weakness which is sudden in onset and occurs upon waking up without any sensory signs, facial, bulbar or autonomic involvement. This flaccid muscle weakness may have been precipitated by a large carbohydrate meal, strenuous exercise the day before or medication such as insulin. The serum potassium level will be <3.5 mmol/L. Our patient presented after 14 h with a serum potassium level of 3.25 mEq/L, so the potassium level had probably hit its nadir near the start of the episode and risen up to 3.25 mEq/L at 14 h. This case shows that even with a potassium level that is not greatly reduced at presentation, HPP, although rare, should still be considered in a patient presenting with isolated quadriceps weakness.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

Financial support and sponsorship

Nil.

Conflicts of interest

There are no conflicts of interest.

Ritika Agarwal, Ravinder Singh Aujla, Amit Gupta
Department of Medicine, Chandra Laxmi Hospital, Ghaziabad, Uttar Pradesh, India

Address for correspondence: Dr. Ravinder Singh Aujla, Plot No. 337, Sector 4 Vaishali, Ghaziabad - 201 010, Uttar Pradesh, India. E-mail: raujla@live.ca

© 2020 Journal of Emergencies, Trauma, and Shock | Published by Wolters Kluwer - Medknow
Letters to Editor

REFERENCES

1. Barohn RJ, Dimachkie MM, Jackson CE. A pattern recognition approach to patients with a suspected myopathy. Neurol Clin 2014;32:569-93.
2. Fontaine B, Vale-Santos J, Jurkat-Rott K, Reboul J, Plassart E, Rime CS, et al. Mapping of the hypokalaemic periodic paralysis (HypoPP) locus to chromosome 1q31-32 in three European families. Nat Genet 1994;6:267-72.
3. Ajith D, Jalawadi VM, Yadav U. Study of incidence and prevalence of hypokalemic periodic paralysis. Int J Res Med Sci 2019;7:810.

How to cite this article: Agarwal R, Aujla RS, Gupta A. Isolated quadriceps weakness: A case of hypokalemic periodic paralysis. J Emerg Trauma Shock 2020;13:97-8.

Submit: 03-Sep-2019. Revised: 24-Sep-2019. Accepted: 18-Oct-2019. Published: 19-Mar-2020.

©2020 Journal of Emergencies, Trauma, and Shock | Published by Wolters Kluwer - Medknow