Turk J Phys Med Rehab 2022;68(x):i-iv
DOI: 10.5606/tftrd.2022.8328
Available online at www.turkishjournalpmr.com

Case Report

Electromyographic changes in a patient with hypocalcemia after thyroidectomy: A case report

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Received: February 01, 2021 **Accepted:** April 19, 2021 **Published online:** December 10, 2021

ABSTRACT

Hypocalcemia is a rather uncommon condition that may be encountered in the outpatient setting. It may be associated with a wide range of clinical symptoms and signs. It is unclear that the primary cause of symptom is hypocalcemia, as these symptoms are usually discrete and ambiguous. Signs of muscle cramp and tetany are considered the expressions of overexcitability of peripheral nerves or central nervous system in case of hypocalcemia in the nerve. In this case, we present a 59-year-old female who presented with persistent muscle cramp and pain of both upper and lower extremities and underwent an investigation that revealed electromyographic changes due to hypocalcemia during an electromyographic study.

Keywords: Electromyography, hypocalcemia, muscle cramp.

Hypocalcemia can be caused by several clinical conditions, with hypoparathyroidism being the most common cause of hypocalcemia, as the parathyroid hormone is a key regulator of calcium level.[1] Hypoparathyroidism can be inherited or caused by immune system-related damages. It can also occur after surgical destruction of the parathyroid gland, such as thyroid or parathyroid gland surgery.[2] Hypocalcemia may be associated with a wide range of clinical symptoms and signs. Mildly decreased calcium levels or slowly changed calcium levels may have no definite clinical symptoms, as symptoms vary depending on how quickly and how severely the calcium level is decreased.[3,4] Common symptoms of hypocalcemia include fatigue, numbness, seizures, muscle spasms, and even cardiac arrest.[5] It is unclear that the primary cause of symptom is hypocalcemia, as these symptoms are usually discrete and ambiguous. In nerves, low calcium concentrations allow sodium to enter nerve cells, increasing nerve irritability and resulting in spontaneous contractions and muscle fasciculations.[6] Signs of muscle cramp and tetany are considered the expressions of overexcitability of peripheral nerves or central nervous system caused by disturbances in calcium, magnesium, or potassium concentrations.[7]

Until now, there is no report showing electromyographic (EMG) changes due to hypocalcemia in the normal range of creatine kinase (CK) after thyroidectomy. In this article, we report the importance of needle EMG in the diagnosis of muscle cramp-induced hypocalcemia, for which other examination could not reveal the cause of the symptom.

CASE REPORT

A 59-year-old female patient was referred to the Department of Rehabilitation Medicine by the Department of Anesthesiology, due to a four-month

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Cite this article as:
Min H, Lee CH. Electromyographic changes in a patient with hypocalcemia after thyroidectomy: A case report. Turk J Phys Med Rehab 2022;68(x):i-iv.

TABLE 1
Laboratory tests

Test	Result	Reference values
Parathyroid hormone (pg/mL)	1.20	15-65
Calcium^{2+} (mg/dL)	3.6	4.5-5.3
Calcium total (mg/dL)	7.1	8.6-10.2
Phosphorus (mg/dL)	5.1	2.7-4.5
Lactate dehydrogenase (U/L)	276	135-225
Magnesium (mg/dL)	2.1	1.5-2.5
Creatine kinase (U/L)	168	0-190
Creatine kinase-MB (ng/mL)	0.8	0-3.6
Triiodothyronine (T3) (ng/dL)	97.23	80-200
Free thyroxine (FT4) (ng/dL)	1.57	0.93-1.70
Thyrotropin (TSH) (mlU/L)	0.04	0.27-4.2
Rheumatoid factor (IU/mL)	8.3	0-15
Fluorescent antinuclear antibody	Negative (<1:40)	Negative

history of muscle cramp and pain of both upper and lower extremities. She did not complain of any weakness or gait disturbance. Her physical examination findings were normal. Neurological examination indicated no evidence of abnormal muscle tone or involuntary movements, except for equivocal Trousseau's and Chvostek's signs. She had a total thyroidectomy for a papillary thyroid cancer 10 years ago. Since then, she had been taking a calcium-based drug to raise calcium levels. Otherwise, she had no history of any tumor, trauma, or other diseases.

Before being transferred, she underwent magnetic resonance imaging (MRI) of cervical spine to evaluate the cervical lesion. She received only pain control medicines, as there was no definite finding on MRI. After being transferred, we consulted the patient to the Department of Rheumatology to rule out possible rheumatological diseases. Lower leg-enhanced MRI was performed to rule out myopathy. There was no definite finding on MRI.

Laboratory analysis was performed (Table 1). The results showed low parathyroid hormone levels indicating hypoparathyroidism with low serum calcium and high phosphate levels suggestive of severe hypocalcemia. Magnesium level was within the normal

TABLE 2
Findings of needle electromyography

Muscle	Doublet	Triplet	NMD	Fibrillation	PSW	MUAP	Recruitment
Right biceps brachii	+	+		None	None	Normal	Mildly reduced
Right deltoid	+	+		None	None	Normal	Mildly reduced
Right flexor carpi radialis	+	+		None	None	Normal	Mildly reduced
Right abductor pollicis brevis	+	+		None	None	Normal	Mildly reduced
Right first dorsal interosseous	+	+		None	None	Normal	Mildly reduced
Left biceps brachii	+	+		None	None	Normal	Mildly reduced
Left deltoid	+	+		None	None	Normal	Mildly reduced
Left flexor carpi radialis	+	+		None	None	Normal	Mildly reduced
Left abductor pollicis brevis	+	+	+	None	None	Normal	Mildly reduced
Left first dorsal interosseous	+	+		None	None	Normal	Mildly reduced
Right vastus medialis	+	+		None	None	Normal	Mildly reduced
Right rectus femoris	+	+	+	None	None	Normal	Mildly reduced
Right tibialis anterior	+	+	+	None	None	Normal	Mildly reduced
Right extensor digitorum brevis	+	+		None	None	Normal	Mildly reduced
Right gastrocnemius	+	+	+	None	None	Normal	Mildly reduced
Left vastus medialis		+		None	None	Normal	Mildly reduced
Left rectus femoris	+	+		None	None	Normal	Mildly reduced
Left tibialis anterior	+	+		None	None	Normal	Mildly reduced
Left extensor digitorum brevis	+	+	+	None	None	Normal	Mildly reduced
Left gastrocnemius	+	+		None	None	Normal	Mildly reduced

NMD: Neuromyotonic discharge; PSW: Positive sharp wave; MUAP: Motor unit action potential.

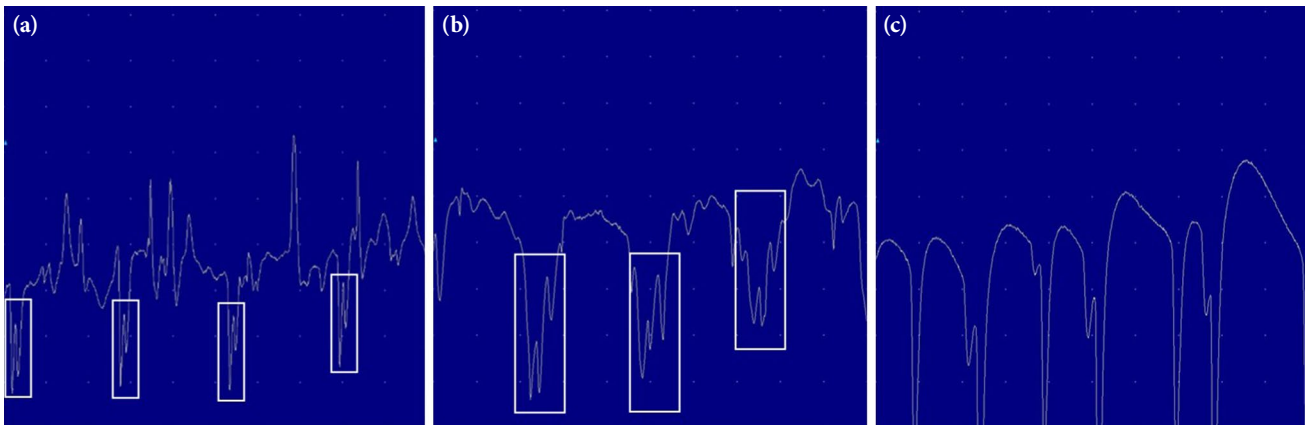

Figure 1. Electromyographic recordings obtained from the patient with hypocalcemia. **(a)** Doublets from the right gastrocnemius muscle, **(b)** triplets from the left deltoid muscle, and **(c)** neuromyotonic discharges from the right vastus medialis muscle at a frequency of 75 Hz. Each division represents 100 μV (y-axis) and 10 milliseconds (x-axis).

range. The patient was in an euthyroid state with adequate thyroid hormone supplement. No evidence of myopathy was observed, as her CK and lactate dehydrogenase (LDH) levels were normal.

We requested nerve conduction studies with needle EMG to rule out peripheral polyneuropathy or cervical radiculopathy. A Medelec® Synergy System (Oxford Instruments, Oxford, UK) was used for the patient lying on a bed in a quiet and warm room, and the limb temperature was maintained at 33°C. Nerve conduction studies of median, ulnar, superficial peroneal, and sural nerves were performed and showed no remarkable findings. However, when the muscles of both upper and lower extremities were used in the needle EMG (Table 2), abnormal spontaneous activities appeared as doublets (Figure 1a), triplets (Figure 1b), and neuromyotonic discharges (Figure 1c). We recommended a physical therapy and drugs. She agreed our suggestion. Two months later, pain of both upper and lower extremities was not completely resolved, so she is still under follow-up.

A written informed consent was obtained from the patient.

DISCUSSION

Clinical symptoms of hypocalcemia can be produced in various forms. They may result in tetany, muscle cramp, carpopedal spasm, laryngospasm, and seizures. Tetany and muscle cramp can be characterized, when hypocalcemia is classically associated with hyperexcitability at the neuromuscular junction.[5] The present case indicates that changes in functions of the neuromuscular system may be present in patients with hypocalcemia after thyroidectomy.

Electromyographic examination is regarded the most sensitive way to examine tetany or muscle cramp, as it enables quantification of increased neuromuscular excitability.[7] Decreased serum calcium concentration causes a shift in the cellular activation potential toward the resting potential.[8] Therefore, less current is required to elicit an action potential, and muscle cramp and tetany can occur. In our case, an abnormal spontaneous activity during needle EMG was encountered abundantly in all muscles of upper and lower extremities. In particular, neuromyotonia was detected in all muscles. Doublet or triplets (multiplets) were encountered frequently. Neuromyotonic discharges usually display high frequencies of 150 to 250 Hz. However, it should be noticed that the frequency may drop to 75 Hz and that the amplitude may also decrease as shown in this patient with chronic hypocalcemia.[9] The activity representing neuromyotonic discharge can be seen in chronic motor neuron disorders or syndrome of acquired neuromyotonia. However, a severe hypocalcemic state may be the most suggestive cause of the EMG changes in this case. In case of nerve conduction studies, there was no remarkable finding. It is considered that an altered extracellular ionic environment does not substantially affect the velocity of conduction for either motor or sensory nerves, although the patient may have significant symptoms.

A few case reports on hypocalcemia have shown hypocalcemic myopathy in hypoparathyroidism.[1,2] showing the elevated CK or LDH levels. However, none of these have been examined through needle EMG. There was one case report showing myotonia on needle EMG in a patient of hypoparathyroidism with hypocalcemia.[10] However, the patient in the case report was with idiopathic hypocalcemia rather than iatrogenic or postoperative hypocalcemia as in our case. The patient had elevated CK levels suggesting myopathy. In our case, CK and LDH levels that could suggest myopathy were within the normal range. At three months later, follow-up test of CK and LDH levels showed normal levels. Only calcium levels decreased.

The lower leg-enhanced MRI was performed to rule out the muscle origin, as serum CK could be normal or mildly elevated in some patients.[11] We did not have a definite finding on MRI. However, the etiology of a rare myopathy associated with hypoparathyroidism is even less well understood. Elevated serum CK and mild histological abnormalities on muscle biopsy are usually considered secondary to muscle damage from muscle cramp or tetany.[11] We, therefore, follow this patient in the outpatient setting.

In conclusion, hypocalcemia is a rare condition that may be encountered in a clinic. We report the first case of EMG changes due to hypocalcemia in the normal range of CK after thyroidectomy. This case illustrates the need to consider needle EMG for diagnosis of cause in a patient presenting with muscle cramp or tetany.

Declaration of conflicting interests

The authors declared no conflicts of interest with respect to the authorship and/or publication of this article.

Funding

The authors received no financial support for the research and/or authorship of this article.

REFERENCES

1. Policepatil SM, Caplan RH, Dolan M. Hypocalcemic myopathy secondary to hypoparathyroidism WMJ 2012;111:173-5.
2. Nora DB, Fricke D, Becker J, Gomes I. Hypocalcemic myopathy without tetany due to idiopathic hypoparathyroidism: Case report. Arq Neuropsiquiatr 2004;62:154-7.
3. Bilezikian JP, Khan A, Potts JT Jr, Brandi ML, Clarke BL, Shoback D, et al. Hypoparathyroidism in the adult: Epidemiology, diagnosis, pathophysiology, target-organ involvement, treatment, and challenges for future research. J Bone Miner Res 2011;26:2317-37.
4. Fong J, Khan A. Hypocalcemia: Updates in diagnosis and management for primary care. Can Fam Physician 2012;58:158-62.
5. Cooper MS, Gittoes NJ. Diagnosis and management of hypocalcaemia. BMJ 2008;336:1298-302.
6. Gordon A. Molecular basis of contraction. In: Patton HD, Fuchs AF, Hille B, editors. Textbook of physiology: Excitable cells and neurophysiology. Philadelphia: WB Saunders: 1989. p. 177-8.
7. Starita A, Giusti A, Rossi B, Sartucci F. Evaluation of the EMG spontaneous activity in tetany: Further development of a method of automatic analysis. Electromyogr Clin Neurophysiol 1984;24:619-23.
8. Frankenhaeuser B, Hodgkin AL. The action of calcium on the electrical properties of squid axons. J Physiol 1957;137:218-44.
9. Preston DC, Shapiro BE, editors. Electromyography and neuromuscular disorders: Clinical-electrophysiological correlations. 3rd ed. Philadelphia: Saunder; 2012.
10. Mathukumalli NL, Kanikannan MA, Shaik JA, Kandadai MR. Idiopathic hypoparathyroidism with clinical and electrophysiological myotonia- An unusual occurrence. Neurol India 2018;66:1486-8.
11. Hower J, Struck H, Tackmann W, Stolecke H. CPK activity in hypoparathyroidism. N Engl J Med 1972;287:1098.