

The Clinical Picture

Stiff, numb hands



FIGURE 1. Carpopedal spasm.

A 45-YEAR-OLD WOMAN with no chronic medical problems presented to the emergency room with a 1-day history of cramps and paresthesias in both hands and feet, mainly involving the fingers and toes. She said that after an argument with her daughter she began feeling anxious, and this was accompanied by shortness of breath and palpitations as well as generalized weakness, fatigue, and body aches. She also reported nausea and repeated vomiting but no abdominal pain, distention or change in bowel movements. She had had no loss of consciousness, confusion, incontinence, headache, dizziness, diplopia, or facial paresthesia.

She is a cigarette smoker, is alcohol-dependent, but does not use illicit drugs and is not on any medications.

Examination revealed a temperature of 37.1°C (98.8°F), blood pressure 150/75 mm Hg, heart rate 105 bpm, respiratory rate 24 breaths per minute, and oxygen saturation 97% on room air. She appeared very



FIGURE 2. The Trousseau sign, carpopedal spasm provoked by inflating a blood-pressure cuff on the patient's arm.

fatigued, thin, and in mild distress due to her cramps. Her mucous membranes were dry, but she had no orthostatic changes. She had noticeable carpopedal spasms (FIGURE 1), reproducible by inflating a blood-pressure cuff placed on her arm (Trousseau sign) (FIGURE 2). Also noted was the Chvostek sign—contraction of the ipsilateral facial muscles when the facial nerve is tapped just in front of the ear. The rest of the systemic evaluation was normal. Laboratory investigations were as listed in TABLE 1. Electrocardiography showed a prolonged QTc interval (0.5 sec). The chest radiograph was normal.

■ HYPERVENTILATION AND TETANY

The presumptive diagnosis was latent tetany caused by an electrolyte derangement, in this case a combination of hypocalcemia, hypomagnesemia, and hypokalemia as the result of alcohol abuse, repeated vomiting, and

hyperventilation brought on by a severe attack of anxiety.

Tetany results from increased excitability of nerves and muscles, leading to painful muscle cramps.^{1,2} Typical symptoms include circumoral and distal paresthesias, stiffness, clumsiness, myalgia, carpopedal spasm, laryngospasm, bronchospasm, and generalized seizure. The Chvostek and Trousseau signs help to confirm the diagnosis of tetany.^{3,4}

The differential diagnosis of carpopedal spasm includes other conditions of involuntary muscle contraction, such as myotonic disorders; myokymia from Isaac syndrome (writhing movements of the muscles under the skin visualized by continuous “rippling” movements of the muscle); stiff-man syndrome (an autoimmune-antiglutamic acid decarboxylase antibody-associated muscle rigidity that waxes and wanes with concurrent spasms); and snake envenomation.

In addition, our patient’s symptoms were probably brought on by hyperventilation. In general, patients with hyperventilation syndrome are young females who display various manifestations of underlying anxiety and can develop tetany even after a brief episode of hyperventilation. At the time of presentation, our patient was found to have mixed respiratory and metabolic alkalosis. The anxiety-induced hyperventilation likely contributed to the respiratory alkalosis. She had no other symptoms or signs to suggest an acute organic respiratory illness such as pulmonary embolism, pneumothorax, or infection. Vomiting likely caused the metabolic alkalosis and hypokalemia.

Tetany is usually triggered by acute hypocalcemia and is uncommon unless the serum ionized calcium concentration falls below 4.3 mg/dL (1.1 mmol/L), which usually corresponds to a serum total calcium concentration of 7.0 to 7.5 mg/dL (1.8 to 1.9 mmol/L). Patients with a gradual onset of hypocalcemia tend to have fewer symptoms.^{3,4}

Although alkalosis alone can cause tetany, it also enhances tetany by reducing the level of ionized calcium in the serum. Alkalemia causes hypocalcemia by an intravascular chelative mechanism in which the decrease in concentration of hydrogen ions leaves the negatively charged binding sites on albumin available to bind ionized calcium.³

TABLE 1

Our patient’s laboratory test results

TEST	RESULT	REFERENCE RANGE
Complete metabolic profile		
Sodium	137 mmol/L	136–145
Potassium	3.1 mmol/L*	3.5–5.0
Chloride	94 mmol/L*	98–107
Bicarbonate	32 mmol/L	23–32
Urea nitrogen	5 mg/dL*	7–18
Creatinine	0.6 mg/dL	0.6–1.0
Glucose	90 mg/dL	74–107
Albumin	3.3 g/dL*	3.4–5.0
Calcium	6.0 mg/dL*	8.5–10.1
Phosphorus	2.3 mg/dL*	2.5–4.9
Magnesium	0.7 mg/dL*	1.8–2.4
Aspartate aminotransferase	155 U/L*	15–37
Alanine aminotransferase	81 U/L*	30–65
Total bilirubin	0.7 mg/dL	0.2–1.0
Alkaline phosphatase	95 U/L	50–136
Complete blood count		
Hemoglobin	10.1 g/dL*	12.0–15.0
Hematocrit	31.2%*	36.0–48.0
Mean corpuscular volume	109.1 fL*	79.0–98.0
Platelets	151 × 10 ⁹ /L	140–440
Coagulation profile		
International normalized ratio	0.95	0–1.2
Partial thromboplastin time	27.0 sec	23.1–33.6
Arterial blood gasses		
pH	7.55*	7.35–7.45
Partial pressure of CO ₂	37 mm Hg	34–46
Bicarbonate	32.3 mmol/L*	22–26
Other values		
Intact parathyroid hormone	8 pg/mL*	10–65
25-hydroxyvitamin D	15 ng/mL*	32–100

*Value outside the reference range

The same happens to the magnesium, a cation with the same size and valence. Significant hypomagnesemia is common in tetanic patients with hyperventilation attacks and may, by itself or in combination with hypocalcemia, cause tetany.^{2,5,6} Hypokalemia can develop in patients with hypomagnesemia or metabolic alkalosis and may lead to tetany.^{6,7} Furthermore, our patient was dependent on alcohol, and this is known to cause hypomagnesemia from the excessive urinary excretion of magnesium. This defect of alcohol-induced

tubular dysfunction is reversible within 4 weeks of abstinence. Magnesium depletion can cause hypocalcemia by producing resistance to parathyroid hormone or by decreasing its secretion, and this occurs in severe hypomagnesemia, ie, when the serum magnesium concentration falls below 1.0 mg/dL (0.4 mmol/L).

IDENTIFY AND TREAT THE UNDERLYING CAUSE

The management of tetany consists of identifying and treating the underlying cause. Infusion of calcium or magnesium is effective as acute therapy for tetany, and, if appropriate, vitamin D supplementation should also be provided.^{3,4,7} However, if associated hyperventilation syndrome is present, patients benefit from reassurance and treatment for underlying psychological stress. The traditional treatment of rebreathing into a paper bag is no

longer recommended because of the potential risk of hypoxia. Sedatives and antidepressants should be reserved for patients who have not responded to conservative treatment.

Our patient was given an explanation of the condition together with breathing exercises. She received lorazepam and was immediately treated with intravenous hydration, along with intravenous infusion of magnesium, calcium, and potassium. These interventions led to an immediate resolution of her symptoms.

Her low level of intact parathyroid hormone may also have been caused by hypomagnesemia. She was given oral magnesium, potassium, calcium, and vitamin D to continue at home. In addition, she was advised to avoid excessive alcohol consumption and to see us or her primary care doctor should the symptoms recur. As expected, all the laboratory values normalized within 1 month of abstinence from alcohol, and she has been well since. ■

REFERENCES

1. Macefield G, Burke D. Paraesthesiae and tetany induced by voluntary hyperventilation. Increased excitability of human cutaneous and motor axons. *Brain* 1991; 114:527–540.
2. Moe SM. Disorders involving calcium, phosphorus, and magnesium. *Prim Care* 2008; 35:215–237.
3. Tohme JF, Bilezikian JP. Hypocalcemic emergencies. *Endocrinol Metab Clin North Am* 1993; 22:363–375.
4. Cooper MS, Gittes NJ. Diagnosis and management of hypocalcaemia. *BMJ* 2008; 336:1298–1302.
5. Tong GM, Rude RK. Magnesium deficiency in critical illness. *J Intensive Care Med* 2005; 20:3–17.
6. Smets YF, Bokani N, de Meijer PH, Meinders AE. Tetany due to excessive use of alcohol: a possible magnesium deficiency [in Dutch]. *Ned Tijdschr Geneesk* 2004; 148:641–644.
7. Huang CL, Kuo E. Mechanism of hypokalemia in magnesium deficiency. *J Am Soc Nephrol* 2007; 18:2649–2652.

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