



More Than "Just Another Conversion Reaction!" A Case of Hyperventilation Syndrome

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Summary

Although hyperventilation occurs in many different clinical specialties, family physicians differ in that they have the responsibility to provide their patients the best available health care without increasing the burden of the health system by asking for unnecessary referrals. Hyperventilation syndrome is a clinical entity with the potential to be easily overlooked as conversion reaction or referred to higher centers with the doubt of potentially serious disease.

In this paper, we report a case admitted to the emergency unit. She was first considered as having a conversion reaction, but history, physical examination, and laboratory test revealed decreased ionized calcium levels caused by respiratory alkalosis, induced by a hyperventilation syndrome. She rapidly recovered by re-breathing into a bag.

If family physicians become alert to the diagnosis and knowledgeable about the treatment of hyperventilation syndrome, such clinical presentations can easily be handled with high patient satisfaction at the primary care level.

Introduction

Hyperventilation occurs in many different specialties including neurology, cardiology, chest medicine, psychiatry, and family practice. The importance of family practice comes from the responsibility to provide patients the best available health care without increasing the burden of the health system by asking for unnecessary referrals. The prevalence of hyperventilation syndrome in primary care is 6-11% (1). Hyperventilation can be described as breathing more than metabolic needs of body resulting excess CO₂ excretion, respiratory alkalosis and elevation of the blood pH (2). Hyperventilation syndrome is characterized with somatic symptoms emerging with volunteer inappropriate hyperventilation.

Diagnostic criteria of hyperventilation syndrome are as follows:

1. The patient should hyperventilate and have low PaCO₂,
2. Somatic diseases causing hyperventilation should have been excluded, and
3. The patient should have a number of complaints that are, or have been, related to the hypocapnia (2).

Hypocalcemic crisis presents with the classical symptomatology of tetany plus extrapyramidal symptoms and a disordering of consciousness extending even to coma.

Clinical findings of hypocalcaemia are as follows:

1. Nonspecific symptoms such as fatigue, irritability, acral or oral paresthesia, muscle spasms and fasciculations,
2. Overt tetany,

3. Convulsions,
4. Latent tetany which can usually appear by provocation (Chvostek and Trousseau signs), and
5. Elongation of Q-T and S-T intervals; very rarely a 2:1 A-V block.

It develops when the concentration of ionized serum calcium declines rapidly, and is very rarely found in chronic hypocalcaemia. In terms of its etiology, various forms of parathyroid deficiency, and nonparathyrogenic diseases associated with hypocalcaemia may be involved (3). Since in the latter, the concentration of albumin is also diminished, and thus ionized calcium decreases to only a small extent, hypocalcemic crisis in these conditions is rare. The most common clinical form is normocalcemic tetany, that occurs within the framework of the hyperventilation syndrome (3). In hyperventilation syndrome, the ionized calcium fraction is temporarily reduced by marked alkalosis.

In hyperventilation syndrome, symptoms related to a decline in ionized serum calcium levels are often evaluated inadvertently as conversion reaction. In this paper, a case characterized with symptoms due to metabolic changes triggered by hyperventilation is presented to emphasize the importance of comprehensive and analytic patient approach.

Case: A 26 years old woman was admitted to the emergency unit of Trakya University Hospital (Edirne, Turkey), which is 8 km from the city center. The first encounter at this center is made by a family physician with special training in emergency medicine. Other specialists are asked for consultation according to necessity.

The patient was admitted with complaints of spasms of hands and feet, and paraesthesia at her whole body. Her complaints began four hours ago and she had vomited once. She had spasm like twisting in the hands. She reported a similar history, but in a milder form without contractions 8 years ago, when she entered the national university entrance exam. Her personal medical history was unremarkable. She underwent two cesarean sections and had two healthy children at the ages 2 years and 6 months. Family history and drug usage history was unremarkable as well. She looked anxious and had flexion contractions in both hands. Her respiration was fast (24/min) and deep. Chvostek finding was positive. Other systemic physical examination findings were normal. Her clinical picture was in concordance with hypocalcaemia. After obtaining a blood sample for laboratory investigation, she was asked to breath into a bag and simultaneously an IV calcium gluconate infusion was started. The laboratory analysis including electrolytes was normal (Table 1). Electrocardiography showed a slight increase in the QT interval (0.46s). Arterial blood gas analysis was performed, which revealed respiratory alkalosis (pH: 7.63, pCO₂: 15.4 mm Hg, pO₂: 122 mm Hg, HCO₃: 16.5 mEq/L). The clinical picture was considered as decreased ionized calcium levels caused by respiratory alkalosis, which in turn was induced by a hyperventilation syndrome. She rapidly recovered by re-breathing into a bag. Her symptoms started to improve within the first 5 minutes of the therapy and she completely recovered after 1 1/2 hours. Her control blood gas analysis was normal (pH: 7.45, pCO₂: 26.3 mm Hg, pO₂: 84.7 mm Hg, HCO₃: 19.7 mEq/L).

Table 1: Initial laboratory evaluation results.

Item	Value
Total calcium	9 mg/dl
Magnesium	1,9 mg/dl
Serum glucose	102 mg/dl
Serum urea	28 mg/dl
Serum creatinine	0.8 mg/dl
Total protein	6.6 mg/dl
Albumin	3.7 mg/dl
Total kalsiyum:	9 mg/dl
AST	30 U/L
ALT	11 U/L
Na	134 mmol/L
K	3.8 mmol/L
Serum chloride	103 mmol/L

Discussion

Conversion is a frequently encountered clinical presentation at the emergency units. Its signs and symptoms are typically neurological (4). Due to the contractions in her hands and paresthesias throughout her body, our first image of this patient as having a conversion reaction. Sensorial symptoms, diffuse pain, carpopedal spasm, and spasms at the facial muscles led us to more vigorous evaluation and the diagnosis of hypocalcaemic tetany. Tetany is usually presented with sensorial symptoms such as paresthesias at the lips, tongue, fingers, and feets, a carpopedal spasm, which may be long lasting, diffuse myalgia, and spasms at the face muscles. Tetany is typically the result of severe hypocalcaemia. However, also a decrease in the ionized fraction of plasma calcium can lead to tetany without the presence of hypocalcaemia, such as in the case of severe alkalosis (5).

Whatever the cause, increased serum pH may lead to a decline of ionized calcium levels by increasing the binding capacity of calcium to serum albumin. Symptoms of hypocalcaemia may be seen parallel to and depending on the speed and intensity of the fall in ionized calcium levels. Breathing of our patient was fast and deep, with arterial blood gas results concordant with respiratory alkalosis. Her total serum calcium level was normal. However, the ionized calcium level was calculated indirectly (we did not have the facility to analyze ionized calcium levels) as 2.5 mg/dl according to the following formula: $[\text{protein bound Ca (mg/dl)} = (0.8 \times \text{albumin}) + (0.2 \times \text{globulin}) + 3]$. Every 0.1 unit increase in the blood pH causes a 0.2 mg/dl additional decrease in ionized calcium levels.³ Hence, the ionized serum calcium level of our patient was 2.1 mg/dl.

Tetany is encountered usually at a serum total calcium level of 5 mg/dl and an ionized serum calcium level of 2.5 mg/dl. The essential determinant in the development of symptoms is the ionized fraction. The clinical picture of hyperventilation ensues after a few minutes of rapid and deep ventilation.

The clinical picture of the patient was evaluated as a hyperventilation syndrome induced by respiratory alkalosis causing a decline of ionized calcium levels.

The therapeutic approach to hyperventilation syndrome has several stages and/or degrees of intervention: psychological counselling, physiotherapy and relaxation, and finally drug therapy. Depending on the severity of the problem, one or more therapeutic strategies can be chosen. Reassurance and breathing into a bag are enough in the treatment.

Understandable and satisfactory explanation on the self-limiting nature and the cause of the disease should be provided to the patients. Also our treatment strategy for this patient was composed of reassurance and breathing into a bag. In order not to omit more serious causes of carpopedal spasm, we started IV calcium gluconate infusion until we obtained initial results from the laboratory. Her symptoms started to improve with treatment within five minutes and completely recovered after 1 1/2 hours.

Figure 1: Carpopedal spasm at the hand of the patient when admitted to the emergency unit.



We conclude that, patients with suspected conversion reaction should be carefully evaluated before referral to higher centers. Appropriate clinical management of hyperventilation syndrome by primary care physicians will lead to an increase in patient satisfaction as well as a decrease of unnecessary patient load to the emergency units and cause economical savings.

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