Atypical presentation of primary hypoparathyroidism

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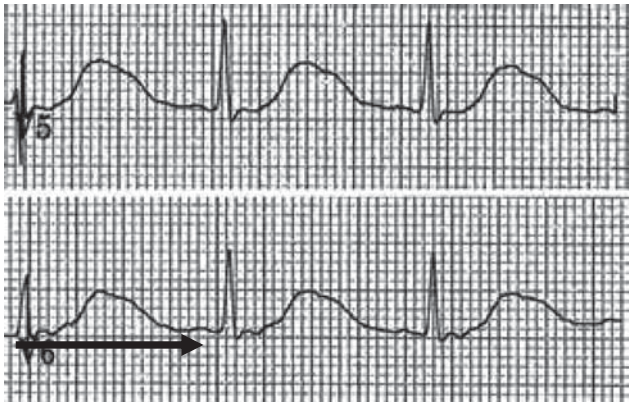
Case report

A 50-year-old housewife presented to the internal medicine clinic with bilateral lower limb painless edema for 2 months associated with exertional dyspnea with no orthopnea or paroxysmal nocturnal dyspnea. She also had a history of recurrent attacks of painless dysphagia to both fluids and solids associated with weight loss and frequent attacks of vague generalized abdominal pain not related to meals, with no specific precipitating factors, not associated with vomiting or change in bowel habits; the pain was found to be relieved spontaneously. Five years back, she had developed attacks of bronchial asthma and had received regular inhalation steroid therapy. Examination indicated an underweight patient (45 kg) with generalized muscle wasting, normal vital signs, of the head and neck facial muscle spasm, loss of teeth, and high jugular venous pressure. The heart, chest, and abdomen showed cardiomegaly, bilateral mild pleural effusion, and an enlarged congested liver. ECG indicated a prolonged QT interval (Fig. 1), plain chest radiograph showed global cardiomegaly (Fig. 2), and echocardiography showed dilated cardiac chambers with global hypokinesia and ejection fraction 28% (Fig. 3). She was diagnosed with congestive heart failure and, upon starting frusemide therapy, she went into carpopedal spasm, generalized tonic–clonic seizures, and disturbed conscious level.

Brain computed tomography was normal and both Chvostek and Trousseau signs were absent.

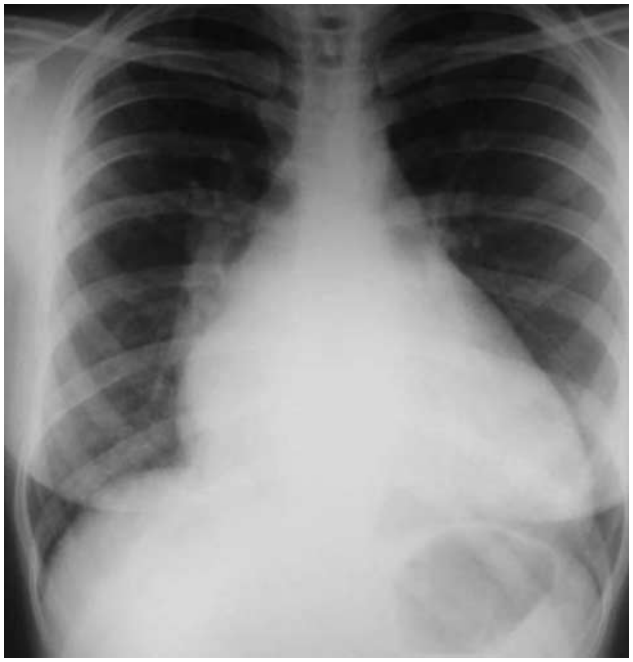
(1) Total and ionized calcium was low (4.7 and 2.8 mg/dl); magnesium was also low (1.0 mmol/l). Phosphorus was high (8.4 mg/dl) with normal renal function (creatinine 0.7 mg/dl) and parathyrod hormone was in a low normal range (27.3 pg/ml). The patient was diagnosed with primary hypoparathyroidism that presented with congestive heart failure and recurrent attacks of bronchial spasm, esophageal spasm, abdominal muscle, and bowel spasm.
(2) She received intravenous calcium gluconate and intravenous $MgSO_4$ infusion as a rescue treatment; 3 hours after receiving the infusion, she developed acute pulmonary edema, and hypertension. Acute myocardial infarction was excluded. This was because

Figure 1



ECG indicated a prolonged QT interval.

Figure 2



Plain chest radiograph showed global cardiomegaly.

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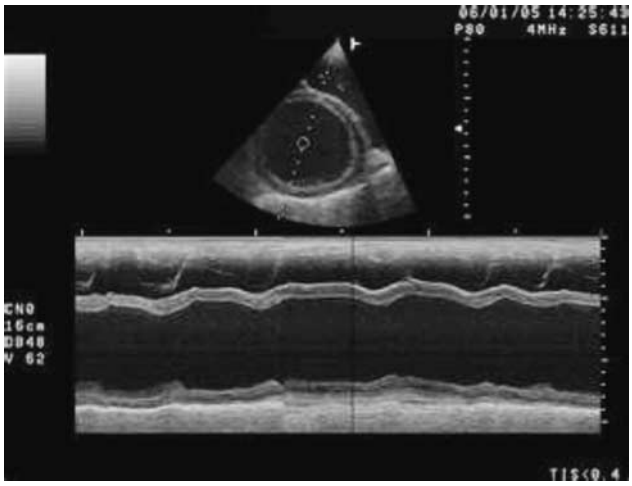
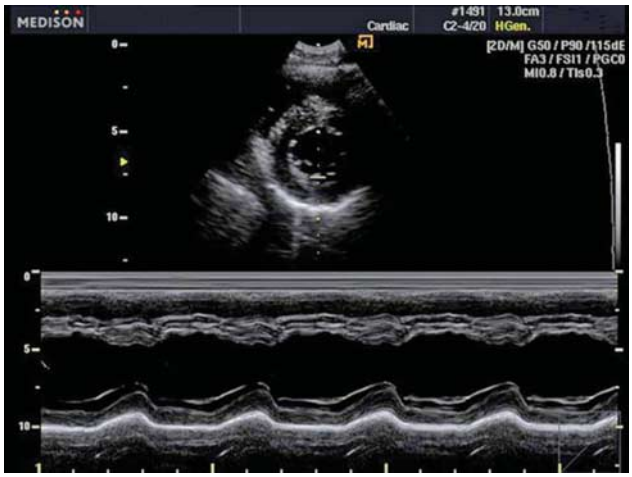
Figure 3



Echocardiography showed EF 28%.

Figure 4



Echocardiography indicated a marked improvement EF of 45%.

of the use of a calcium infusion at a concentration less than it should be. Consequently, the serum calcium level decreased considerably and this aggravated heart failure. She improved partially with an intravenous injection of frusemide and oxygen. She was discharged home and was prescribed oral vitamin D, calcium, and magnesium. One month after discharge, she came for follow-up and showed a marked improvement in symptoms, and normalization of the serum calcium level (9.4 mg/dl), phosphorus level (3.8 mg/dl), and magnesium level (1.8 mmol/dl), and repeated echocardiography indicated a marked improvement in myocardial contractility, with an ejection fraction of 45% (Fig. 4).

Discussion

The patient had congestive heart failure, attacks of dysphagia, abdominal spasm, and bronchial spasm associated with severe hypocalcemia, hyperphosphatemia, and hypomagnesemia. The differential diagnosis was primary hypoparathyroidism versus renal failure with secondary hyperparathyroidism; normal renal function excluded the second possibility early. Consequently the only diagnosis that could explain the clinical and laboratory findings in our patient was primary hypoparathyroidism. This atypical presentation and the development of convulsions after starting frusemide therapy and also the development of pulmonary oedema after intravenous calcium infusion was challenging. The development of carpopedal spasm and convulsions after frusemide can be attributed to the aggravation of hypocalcemia, and the development of pulmonary edema after calcium infusion was also because of a marked decline in the calcium level with the use of a subtherapeutic calcium concentration level in the infusion fluid. In the literature, previously reported eight patients with hypocalcemia presented with congestive heart failure, and also hypocalcemia was found to be a rare cause of abdominal wall spasm, intestinal muscle spasm, sphincter of oddi spasm, and even bronchial smooth muscle spasm. Correction of low serum calcium level by vitamin D and calcium supplementation was expected but, normalization of high serum phosphate was buzzling and it was explained only by the fact that hypomagnesemia decreases secretion and action of parathyroid hormone and in this case we corrected hypomagnesemia so parathyroid hormone level increased which increases renal phosphate excretion.

Improvement in myocardial contractility and ejection fraction after the correction of the calcium level indicates that hypocalcemia was responsible for the poor cardiac function and also absence of further attacks of dysphagia, abdominal pain, and bronchial asthma. This confirms that these were induced by hypocalcemia.

This case shows the diagnostic challenges in cases of reversible heart failure, asthmatic attacks, abdominal pain, and dysphagia and if there is no obvious cause of the patient's complaint, assessment of the mineral level should be carried out. This patient was being treated for years with steroids for asthma and with analgesics for abdominal pain, which was relieved completely with the correction of the mineral level.

Acknowledgements

Conflicts of interest

There are no conflicts of interest.