



International Journal of Emergency Medicine

Editor-in-Chief: Latha Stead

ISSN: 1865-1372 (print version)

ISSN: 1865-1380 (electronic version)

Journal no. 12245



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A delayed diagnosis: stridor secondary to hypocalcemia

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Received: 19 June 2010 / Accepted: 9 August 2010 / Published online: 23 October 2010
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Abstract Hypocalcemia with stridor is a well-known condition in the pediatric age group but has rarely been reported in the elderly. We report an elderly patient who presented with dyspnea and laryngeal stridor attack caused by hypocalcemia. The patient had been suffering from stridor and dyspnea episodes for 2 years, and the etiology had not been determined until the evaluation in our department. The cause of stridor was hypocalcemia secondary to thyroidectomy. Complete resolution of stridor was achieved by calcium replacement therapy.

Keywords Hypocalcemia • Laryngospasm • Stridor • Postoperative complications

Introduction

The most common causes of stridor in adults are abscesses or swelling of the upper airway, tumors and paralysis or malfunction of the vocal cords. Laryngospasm due to hypocalcemia is a rare cause of stridor [1]. We report an elderly patient who presented with dyspnea and laryngeal stridor attack because of hypocalcemia. The patient had been suffering from stridor and dyspnea episodes for 2 years, and

the etiology had not been found until her evaluation at our department. The cause of stridor was hypocalcemia secondary to thyroidectomy. Complete resolution of stridor was achieved by calcium replacement therapy.

Case report

A 54-year-old woman complaining of dyspnea and stridor was admitted to our hospital. She had a history of intermittent stridor and dyspnea for 2 years. In her medical history, it was learned that she had undergone a thyroidectomy operation 2 years ago. After that operation, she had not gone to the control examinations, and she had not used any medical treatment. But she had visited the pulmonologist because of stridor and dyspnea several times. In her previous examinations, fiberoptic bronchoscopy, thorax computed tomography and respiratory function tests were applied, and these tests were evaluated as normal by the pulmonologist, but her PTH level, thyroid function tests and calcium level had not been checked yet.

On examination in our emergency department, she was tachypneic, and her accessory muscles were joining in respiration. She had inspiratory stridor, and the inspiration duration was lengthened. On auscultation of her chest, normal vesicular breath sounds were heard; there was no wheezing or rhonchus. Arterial blood gases demonstrated respiratory alkalosis (pH: 7.46, pCO₂: 33.2, pO₂: 120.9, HCO₃: 23.7, sO₂: 98.7, iCa: 0.272 mmol/l). The blood calcium level was 3.3 mg/dl; other blood tests including thyroid function tests were normal: parathormone (PTH): 2.56 pg/ml (15–65 pg/ml), 25-OH vitamin D3: 9.91 ng/ml (11.1–42.9 ng/ml). A 20-ml calcium gluconate 10% solution (180 mg elemental calcium) was given to the patient in 4 h. After this replacement therapy, the severity of her stridor decreased. The calcium level at that moment was 4.9 mg/dl, iCa: 0.879 mmol/l. An additional 40 ml of

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calcium gluconate was given for 16 h. Her calcium level returned to baseline (7.6 mg/dl) after 20 h of calcium replacement therapy, and complete resolution of the stridor was achieved. Pulmonary auscultation was normal, and the patient was discharged from the emergency department for further evaluation at the endocrinology outpatient department. Oral calcium repletion was initiated by the endocrinologist. The patient was evaluated after 2 months, and during this period there were no stridor attacks. Also her calcium level was within normal range.

Discussion

The main postoperative complications of thyroidectomy include injury to the parathyroid glands and to the laryngeal nerves [2]. Injury to the parathyroid glands was manifested by temporary or permanent hypocalcemia. Transient hypocalcemia is commonly seen after thyroid surgery, as biochemical disorders are described in more than 83% of the cases. Clinical hypocalcemia is however less frequently observed [3]. Hypocalcemia leads to increased neuromuscular irritability and may present with circumoral numbness, paresthesias of the hands and feet, and muscular cramps, or when severe, with laryngospasm, focal or generalized tonic muscle cramps, or seizures. Myocardial dysfunction and prolongation of the QT interval may also occur [1].

In the literature, post-thyroidectomy hypoparathyroidism has been reported with a varying incidence, ranging from less than 1% to as high as 15% [4]. The extension of the thyroid resection procedure is a risk factor for the onset of clinical and laboratory hypoparathyroidism, while age above 50 years is a risk factor for clinical hypoparathyroidism [3]. Patients must be carefully observed in the postoperative period and have their laboratory workup done, especially those categorized as high risk patients [5]. In the reported case we explained that the patient had not gone to her control examinations, and her parathyroid hormone level had never been evaluated. Consequently, hypocalcemia had not been recognized for 2 years.

Severe hypocalcemia and hypomagnesemia resulting from malabsorption syndrome secondary to celiac disease in an elderly woman leading to laryngospasm has been reported [6]. Hypomagnesemia also results in increased

neuromuscular irritability and perioperative laryngospasm from hypomagnesemia, and secondary hypocalcemia in a diabetic patient has also been reported [7].

In conclusion, it is suggested that in case of recurrent laryngospasm, electrolyte measurements of magnesium and calcium might be helpful in the diagnosis and treatment of the patients. Hypocalcemia should be considered in the differential diagnosis of stridor for elderly patients. Additionally, the patients must be followed for complications such as hypocalcemia after thyroidectomy.

Conflicts of interest None.

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