Correspondence

Primary hyperparathyroidism presenting with cough and dyspnea

To the editor: Primary hyperparathyroidism (PHPT) is an occasionally encountered disease caused by parathyroid adenoma, hyperplasia or cancer which secretes much parathyroid hormone leading to high serum calcium and low serum phosphorus level. The disease mainly involves bones and urinary system. The clinical symptoms such as seldom attacks of lassitude, fatigue, vomiting and calcification in normal tissues are rare. We present a patient with PHPT who developed bilateral metastatic calcification of the lungs, primarily presented with a chronic cough and dyspnea on exertion, as a reminder to clinicians of this possible complication.

A 56-year-old woman presented with cough and dyspnea. The chest CT scan showed multiple, bilateral infiltrates and calcification in the left lung and ventricular wall (Figure 1 A, B). Laboratory results were as follows: an increased serum creatinine level of 108 µmol/L (reference: 44–106 µmol/L), normal calcium level of 2.56 mmol/L (2.25–2.75 mmol/L), abnormal phosphate level of 0.79 mmol/L (0.96–1.62 mmol/L) and an increased parathyroid hormone level of 128 pmol/L (1.1–7.3 pmol/L). Right solid thyroid lesion was found through B ultrasonic imaging, and parathyroid hyperactive adenoma was observed by $^{99m}$Tc-MIBI parathyroid imaging. After the transbronchial lung biopsy, we got the pathological result of the patient that there was numerous calcification in the alveoli and interalveolar septum (Figure 1C). A $^{99m}$Tc-methylene diphosphate (MDP) whole body scan showed pulmonary uptake mostly (Figure 1D). A diagnosis of metastatic pulmonary calcification (MPC) due to primary hyperparathyroidism was made.

MPC is a process in which calcium deposition occurs in pulmonary tissues as a result of an excessively high serum calcium level. Such calcification can also occur in the stomach, kidneys, heart, and blood vessels, with the lungs being particularly susceptible to such an occurrence. Two CT patterns of pulmonary calcification have been described: diffuse or patchy areas of ground-glass opacity, and multiple diffuse calcified nodules. Multiple, bilateral infiltrates and calcification in the left lung and ventricular wall demonstrated in the CT scanning of our patient were consistent with the second pattern, which enabled us to consider the diagnosis of MPC.

This is an unusual case of MPC in a patient with PHPT, who primarily presented with a chronic cough and dyspnea. It is easily misdiagnosed as pulmonary disease. We should consider the possibility of MPC with calcium deposition in lungs in patients with PHPT.

DOI: 10.3760/cma.j.issn.0366-6999.20132453

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(Received September 22, 2013)
Edited by Wang Mouyue