# Atypical Radiological Manifestation of Pulmonary Metastatic Calcification

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Eun Hae Kang, MD, Division of Respiratory and Critical Care Medicine, Department of Internal Medicine, Korea University College of Medicine, 126-1, 5ka, Anam-dong, Sungbuk-ku, Seoul 136-705, Korea. Tel. (822) 920-6611 Fax. (822) 929-2045 e-mail: keunhae@unitel.co.kr Metastatic pulmonary calcification refers to calcium deposition in the normal pulmonary parenchyma and this deposition is secondary to abnormal calcium metabolism. The most common radiologic manifestation consists of poorly-defined nodular opacities that are mainly seen in the upper lung zone. We present here a case of metastatic pulmonary calcification that manifested as atypical, dense, calcium deposition in airspaces within the previously existing consolidation in the bilateral lower lobes, and this process was accelerated by pneumonia-complicated sepsis in a patient with hypercalcemia that was due to hyperparathyroidism.

**W** etastatic pulmonary calcification is a condition of calcium deposition in the normal pulmonary parenchyma, and this is secondary to abnormal calcium metabolism without any prior soft tissue damage. The predisposing factors for this condition include chronic renal failure, hypercalcemia and increased tissue alkalinity. The most common radiologic manifestation consists of poorly defined nodular opacities in the upper lung zone. These opacities reflect the deposition of calcium salts in the pulmonary interstitium (1-4). We present here a case of metastatic pulmonary calcification in a patient who recovered from pneumonia with sepsis and whose high-resolution CT (HRCT) images demonstrated localized parenchymal airspace calcification that was limited to the bilateral lower lobes. These lower lobes had been involved with pneumonic consolidation without calcification, as seen on the previous CT scan.

# CASE REPORT

A 48-year-old female patient presented with weight loss, fever and general weakness for about one month. She had been admitted to another hospital and was diagnosed with hyperparathyroidism. She had had an episode of febrile shock during her hospitalization in that hospital; therefore, she was transferred to our hospital. The initial vital signs at admission were as follows: a blood pressure of 86/54 mmHg, a pulse rate of 136/min, a body temperature of 38.5 °C and a respiratory rate of 28/min. The physical examination demonstrated coarse breathing sounds with crackles in both lower lung zones. She complained of severe dyspnea. The laboratory results were as follows: serum urea 37 mg/dL (normal range: 10-40), creatinine 2.3 mg/dL (normal range: 0.6 - 1.1), calcium 14.1 mg/dL, ionized calcium 7.8 mg/dL, phosphorus 2.3 mg/dL, intact PTH 635.30 pg/mL and WBC 24,460/mm<sup>3</sup>. The arterial blood gas analysis values under oxygen therapy via a face mask with a flow rate of 10 L/min were pH 7.235, PaCO<sub>2</sub> 61.8 mmHg, PaO<sub>2</sub> 75.9 mmHg, HCO<sub>3</sub> 25.6 mmol/L and O<sub>2</sub>

saturation 94%.

Admission chest radiograph showed patchy bilateral airspace consolidation that was mainly in the lower lung zones. The precontrast chest CT demonstrated airspace consolidation in the bilateral lower lobes (Fig. 1A) and multifocal patchy ground-glass opacities in both lungs. Neck CT showed a 2.5  $\times$  1.8 cm sized ovoid soft-tissue nodule at the right infrathyroid area, which was suggestive of parathyroid adenoma. Thus, the patient was diagnosed to have hyperparathyroidism-associated hypercalcemia



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and pneumonia-complicated septic shock. She was moved to the intensive care unit and received mechanical ventilator care with assisted controlled mandatory ventilation (ACMV) and continuous renal replacement therapy (CRRT). She underwent bronchoscopy and bronchoalveolar lavage (BAL) to search for clues for her sepsis, and the result of quantitative culture using the BAL fluid revealed that the pathogen was methicillin-resistant Staphylococcus aureus (MRSA).

Parathyroidectomy was performed after she had improved from the sepsis and had been weaned from mechanical ventilation. However, the bilateral pulmonary airspace consolidations remained. Therefore, HRCT was performed to further evaluate the persistent consolidative lesions in the both lower lobes. The follow-up CT demonstrated geographic high-attenuation consolidation and ground-glass opacities with a lower lobe predominance, which was suggestive of metastatic calcifications (Fig. 1B). Bone scintigraphy using Tc-99m methylene diphosphonate (MDP), demonstrated a prominently dense accumulation of the radioactive tracer in the bilateral lower lung zones in the thorax, and diffusely increased uptake was seen along the stomach wall without any abnormal bone uptake; this was all compatible with metastatic calcification (Fig. 1C). She complained of exertional dyspnea and her pulmonary function was compatible with severely restrictive physiology and a low diffusion capacity. She underwent percutaneous lung biopsy and the histologic specimen revealed metastatic pulmonary calcification (Fig. 1D).

At the 9th month after surgical resection of the parathyroid, her serum calcium level was maintained within the normal range and her pulmonary function test showed nearly complete recovery to the normal value. She has almost recovered her health without any symptoms.

# DISCUSSION

Pathologic pulmonary calcification can be broadly divided into metastatic calcification and dystrophic calcification. Metastatic pulmonary calcification is defined as calcium deposition in normal lung tissue without prior tissue damage and it is secondary to abnormal calcium metabolism. On the other hand, dystrophic calcification requires injured tissues such as infected or inflamed lung for the calcification to occur even in the absence of increased serum calcium levels (5).

The predisposing pathophysiologic states for metastatic calcification are reported to be hypercalcemia and a local alkaline environment. For that reason, elevated calciumphosphate production, including hypercalcemia, is usually discovered in most cases of metastatic calcification (6, 7). Meanwhile, the calcium salts precipitate in an alkaline environment, and so pulmonary calcific disorders have the upper lobe predilection due to the higher blood pH and lower  $PaCO_2$  at the apex as compared with the relatively lower pH at the base (5, 8, 9).

The radiological findings of metastatic calcification have generally been reported to be centrilobular ground-glass nodular opacities with numerous fluffy, poorly defined nodules that measure 3 to 10 mm in diameter on the high resolution CT scans (1-4). Patchy areas of consolidation that might also simulate pneumonia were reported in a few cases (1).

However, our case presented with unusual manifestations in several aspects such as the distribution of lesion and the radiologic findings. For our case, the site of calcification was the base of both lungs where a relatively less alkaline or slightly acidic environment is maintained. In addition, the ectopic calcification of this case occurred at a prior inflamed lung lesion, as opposed to the definition of pulmonary metastatic calcification. Radiologically, our case showed well-defined highly attenuated dense airspace consolidation like that in a report by Hartman et al. (1). So, we have validated another type of metastatic pulmonary calcification that developed in underlying consolidation.

We suppose some potential pathophysiologic mechanism in our case, such as the influence by previous cellular and tissue injury as occurs in dystrophic pulmonary calcification, may have contributed to the calcium accumulation in the consolidative lesions in the lower lobes (5). During the process of cellular injury, the pH falls during the early phase and it shifts to a neutral or alkaline pH as the injury continues. The latter state would promote ectopic calcification. Our case had significant predisposing factors for ectopic calcification: excess serum calcium and the continuous tissue injury caused by lobar pneumonia (10). Therefore, this case would correspond to metastatic pulmonary calcification that occurred in her pneumoniainjured tissue.

In summary, we report here on an atypical presentation of metastatic pulmonary calcification that showed dense airspace consolidation localized to the bilateral lower lobes in a patient with primary hyperparathyroidism and pneumonia.

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