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Case Report

Metastatic Pulmonary Calcification with the Coexistence of known Esophageal Carcinoma: Case Report and Literature Review

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Abstract

Metastatic pulmonary calcification, also called MPC, is a metabolic abnormality resulting in pulmonary calcium deposition. Our patient is a known biopsy-proven SCC of the mid-esophagus with extensive widespread alveolar calcifications on a background of renal and ureteric stones. It is important to note that, although the disease is known as metastatic, it is a rather benign lung illness with an excellent long-term prognosis. To our knowledge, this kind of cohabitation has never been documented before.

Keywords: Metastatic Pulmonary Calcification; Esophageal Carcinoma; Metastasis.

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Introduction

One metabolic lung condition called metastatic pulmonary calcification (MPC) is characterized by calcium deposits in the pulmonary parenchyma. It is most frequently associated with conditions that cause hypercalcemia, either directly or indirectly [1]. The interstitial accumulation of calcium salts, primarily in the alveolar epithelial basement membranes, is a characteristic of the illness process [2]. Chronic renal failure, hypercalcemia, and increased tissue alkalinity are risk factors. Metastatic and dystrophic calcifications are the two main categories of pathological pulmonary calcification. MPC, which has a connection to a persistently high serum calcium-phosphate product, is described as calcium deposition in healthy lung tissue without previous tissue injury. On the other hand, even when there is no rise in serum calcium levels, dystrophic calcification necessitates damaged tissue, such as inflammatory or infected lung tissue. While most MPC patients are asymptomatic, there have been a few documented occurrences of respiratory failure [3]. But it wasn't until the development of computed tomography (CT), on which MPC was initially reported in 1989, that antemortem diagnosis became commonplace. Since then, computed tomography has taken center stage in diagnosis, usually eliminating the necessity for a lung sample [4].

Case Presentation

46-year-old male, tractor driver by profession, a known case of well-differentiated SCC esophagus, presented to the clinic with a complaint of cough. Upper GI endoscopy showed ulcerative and friable lesions noted at 24 cm from incisor teeth, extending to 39 cm in mid-esophagus LES at 42 cm from incisors, consistent with biopsy-proven well-differentiated SCC. Rest was normal up to D2. Extracorporeal shock wave lithotripsy for urinary bladder stones or renal stones was performed two years ago.

CT chest and abdomen performed showing fluffy pulmonary alveolar-based opacities with sparse interstitium in patchy distribution were seen bilaterally, with few marginal calcifications in the bilateral upper lobes, suggesting metastatic pulmonary calcifications (Figure 1, 2, 3).

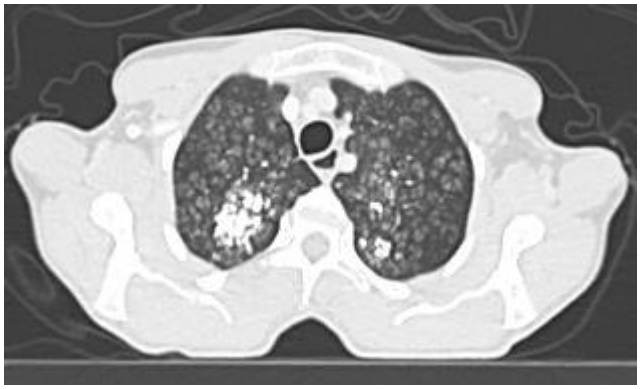

Figure 1: Pulmonary alveolar-based opacities with calcifications

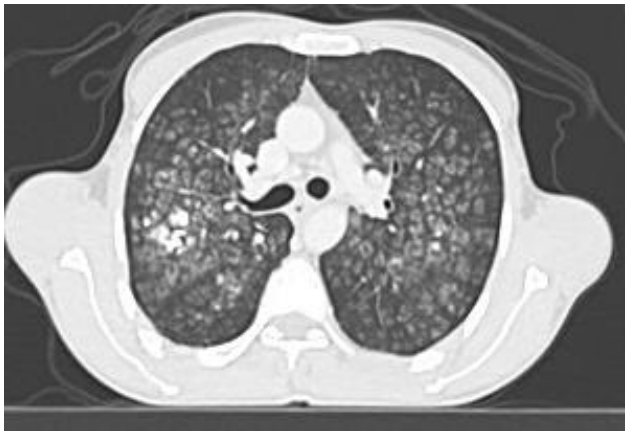

Figure 2: Pulmonary alveolar-based opacities with calcifications

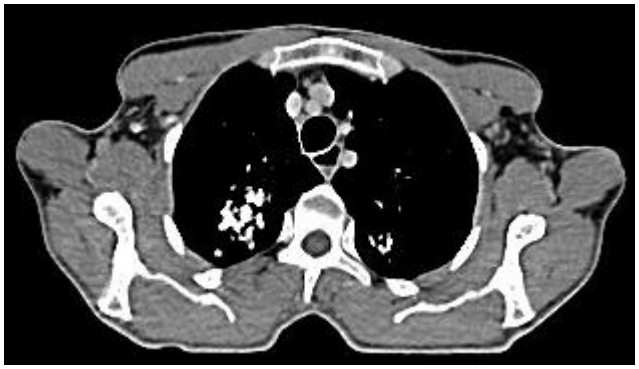

Figure 3: Bilateral pulmonary calcifications

Multiple pulmonary nodules were noted, bilaterally largest in the left lung, measuring up to 8 mm. There was no pneumothorax or pleural effusion on either side. There was no extensive supraclavicular or axillary lymphadenopathy. Mostly sub centimeter-sized, a few prominent, partly calcified mediastinal and hilar lymph nodes were noted. The heart and major mediastinal vessels were normally contrast-opacified and appeared patent. There was no pericardial effusion.

Marked circumferential thickening and luminal narrowing were seen involving the esophagus, extending from the subcarinal location until the gastroesophageal junction, consistent with the well-known well-differentiated squamous cell carcinoma of the esophagus (Figure-4). This mass was anteriorly causing a mass effect on the left atrium and posteriorly on the thoracic aorta, with a loss of fat plane, an AP diameter of almost 4.4 cm, a transverse diameter of approximately 5.4 cm, and a craniocaudal extent of 11 cm. Index mass, bulky tumor involving esophagus commencing from subcarinal location till gastroesophageal junction, 11 cm in craniocaudal dimension compression on the left atrium with loss of fat plane with aorta with aortic encasement off its anterior aspect. Index tumor merging with the nodes in the posterior mediastinum. Additional sub centimeters of chronically calcified nodes are present in the mediastinum. A couple of small sub centimeter nodes in the gastroesophageal junction were seen. No metastases in the liver were noted.

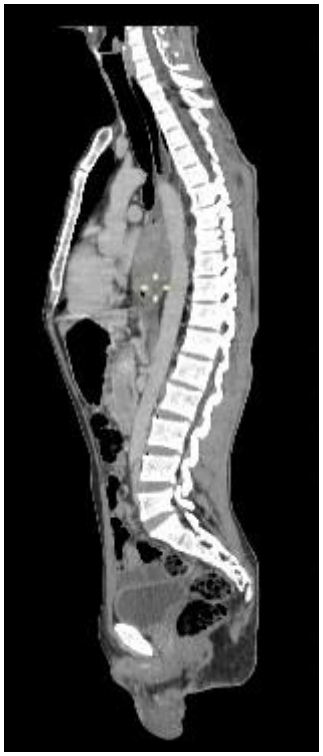

Figure 4: circumferential thickening and luminal narrowing involving the esophagus

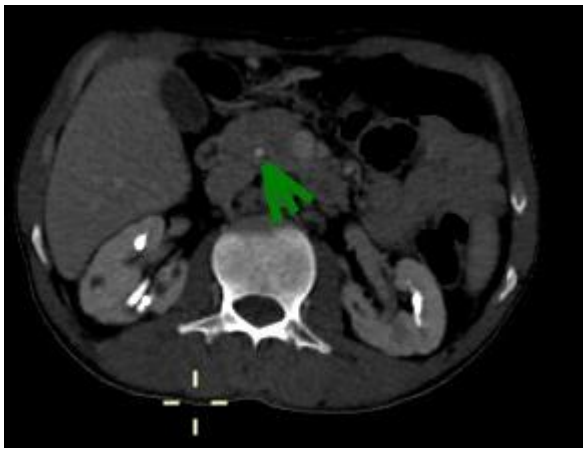

Figure 5: Tiny calcification in the pancreatic head inseparable from CBD likely representing choledocholithiasis

Multiple calculi with areas of scarring are seen involving bilateral kidneys (Figure 6). The largest calculus, measuring 2 cm, is seen in the line of the right ureter (Figure7). There was no evidence of hydroureteronephrosis on either side. The urinary bladder was adequately distended and appears unremarkable. The prostate and seminal vesicles appeared unremarkable.

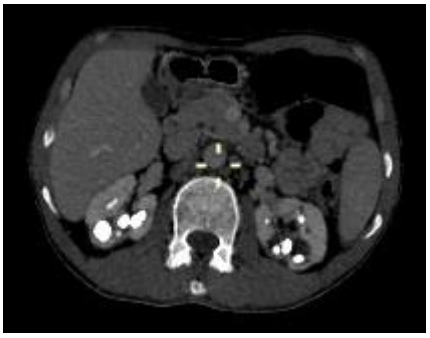

Figure 6: Multiple calculi with areas of scarring involving bilateral kidneys.

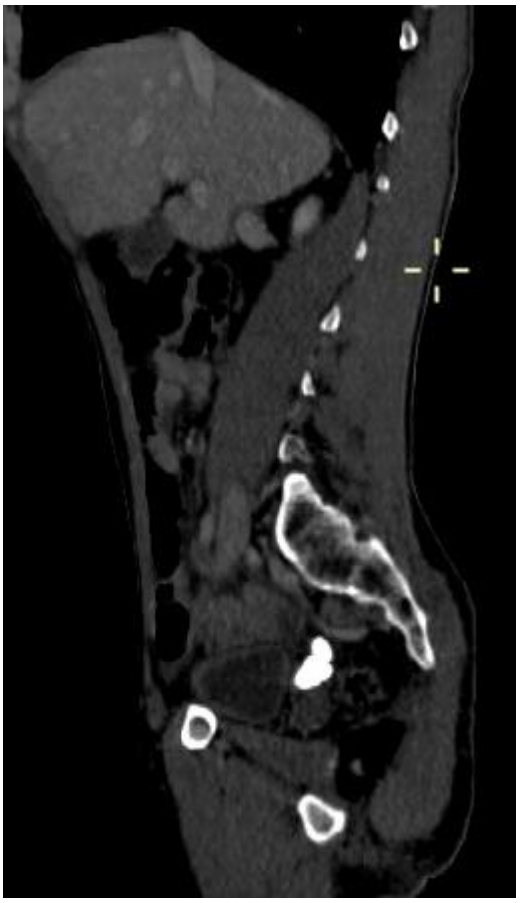

Figure 7: Calculus, measuring 2 cm, in the line of the right ureter.

Background changes of extensive widespread alveolar calcifications; these findings were due to background ongoing changes of metabolic abnormality resulting in pulmonary calcification called metastatic pulmonary calcification'; lab correlation was advised as this needs to be correlated with metabolic underlying abnormality and processes as kidneys also showed medullary calcification as well as multiple calculi in the pelvic calyceal system as well as suggestion of it in the right distal ureter. Lab findings reported intact PT: 116 pg/mL (15–65 normal value), 25-hydroxyvitamin D: 16 ng/mL (Vitamin D Deficiency: <20 ng/ml, Vitamin D Insufficiency: 21–29 ng/ml, Desirable Level: 30–150 ng/ml), calcium: 9.5 mg/dL (Adult 8.4–10.2 mg/dL), phosphorus: 2.8mg/dl (2.-4.5 mg/dl reference range). The renal profile of the patient was creatinine 0.91 mg/dl (male, 0.75 to 1.25 normal range) and eGFR= 90.97 ml/min (normal >60 ml/min); excluding chronic kidney disease as a cause of MPC. Other causes were also excluded including sarcoidosis and multiple myeloma by clinical picture and imaging correlation. There was no evidence of IV or oral calcium therapy. Increased parathyroid hormones, decreased vitamin

D levels, and normal calcium levels suggested secondary hyperparathyroidism, a common cause resulting in metastatic pulmonary calcification, the process further augmented by metabolic stress due to tumour burden. As the patient's primary underlying pathology was squamous cell carcinoma of the esophagus, management was initiated with a combined chemoradiotherapy approach, including carboplatin. Following 10 sessions of radiotherapy, the patient reported significant improvement in dysphagia. After the patient lost to follow-up.

Discussion:

Metastatic pulmonary calcification (MPC) is a lung disease defined by the accumulation of calcium in normal lung tissue under conditions that lead to hypercalcemia, either directly or indirectly [5]. This differs from dystrophic calcifications, which result from calcium deposits in already damaged tissue [6]. Chronic renal failure is the leading cause of MPC [7]. Other potential causes are primary and secondary hyperparathyroidism, excessive administration of calcium and vitamin D, sarcoidosis, significant osteolysis from metastases or multiple myeloma, orthotopic liver transplantation, and cardiac surgery (5,8,9,10). Secondary hyperparathyroidism was the potential cause in our case.

The lesions primarily affected the alveolar septa, accompanied by varying degrees of fibrosis and thickening of the alveolar septa [11]. The accumulation of calcium can be interstitial, peribronchial, and perivascular, and is often associated with significant interstitial fibrosis [12]. Calcium deposits more readily in relatively alkaline tissues, so the lung apex is more commonly affected than the lung base [13].

The clinical manifestations of MPC are usually minimal, with most patients being asymptomatic. A small number may experience dyspnea and a chronic dry cough (14). Severe respiratory failure and death are rare but possible outcomes [15]. Diagnosis chiefly depends on imaging findings. Because radiographs are insensitive in detecting small amounts of calcium, it is frequently normal or shows non-specific results [16]. HRCT is due to its high sensitivity in detecting small calcifications, is useful for diagnosis of MPC. HRCT can demonstrate characteristic findings that suggest a diagnosis of MPC, potentially eliminating the need for lung biopsy [17]. Belem et al describe the most common HRCT patterns due to MPC; centrilobular ground-glass nodules (60.9%), high-attenuation consolidation (43.5%), small dense nodules (39.1%), peripheral reticular opacities with small, calcified nodules (21.7%), and ground-glass opacities without centrilobular ground-glass nodular opacity (21.7%). Centri-lobular ground-glass nodules were also seen in our case. MPC is potentially reversible, and symptom resolution has been observed with the correction of hypercalcemia [14].

In conclusion, characteristic patterns of MPC on HRCT are key to diagnosis. In the presence of hypercalcemia and associated conditions such as renal failure or hyperparathyroidism, these imaging findings may eliminate the need for lung biopsy.

Conclusion:

Normal pulmonary parenchyma experiences calcium deposition, which leads to an uncommon ailment known as MPC. MPC is frequently caused by problems in the metabolism of calcium and phosphate like hyperparathyroidism and ongoing metabolic stresses as in our case. It is important to note that, although the disease is known as metastatic, it is a rather benign lung illness with an excellent long-term prognosis.

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