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CASE REPORT

MRI and CT findings of metastatic pulmonary calcification

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ABSTRACT. Metastatic pulmonary calcification is a consequence of calcium deposition in the normal pulmonary parenchyma, secondary to abnormal calcium metabolism. The most characteristic radiological manifestation is poorly defined nodular opacities that are mainly seen in the upper lung zone. The aim of this report is to describe the CT and MRI findings observed in two patients with metastatic pulmonary calcification. The disease may present in CT as consolidations with calcification, and with a high lesion/muscle signal intensity ratio on T_1 weighted imaging without contrast in MRI. The high signal on T_1 weighted imaging probably occurs because the low calcium concentration of the lesion changes the surface effects of diamagnetic calcium particles, causing T_1 shortening of water protons. MRI is a good option for characterising calcium accumulation caused by a metabolic disorder.

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Metastatic pulmonary calcification (MPC) is a consequence of calcium deposition in pulmonary parenchyma [1]. The most common cause is chronic renal failure [1, 2], but MPC can also occur in a variety of disorders, including primary and secondary hyperparathyroidism, intravenous calcium therapy and massive osteolysis from metastases or multiple myeloma [2]. Clinical symptoms are extremely variable. Patients frequently are asymptomatic, but the process may lead to fulminant respiratory failure and early death [1, 3, 4].

The aim of this work was to report the MRI and CT findings of two patients with MPC.

Case reports

Case 1

A 26-year-old female with end-stage renal disease presented at our hospital with dyspnoea. She had developed nephrotic syndrome at the age of 14 years. By the age of 22 years, she required a bilateral nephrectomy and received a transplant from her father, which failed. She was on haemodialysis for 3 years. Chest auscultation did not reveal adventitious sounds and the rest of the clinical examination was unremarkable. A chest radiograph showed patchy consolidation areas in both lungs. Laboratory studies revealed creatinine levels to be 625.25 μmol l^{-1} (normal range, 30–70 μmol l^{-1}); serum urea, 40.34 mmol l^{-1} (normal range, 3.0–6.5 mmol l^{-1}); calcium, 2.35 mmol l^{-1} (normal range, 2.20–2.50 mmol l^{-1}); phosphate, 3.71 mmol l^{-1} (normal range, 0.80–1.60 mmol l^{-1}); magnesium, 1.03 mmol l^{-1} (normal range, 0.80–1.20 mmol l^{-1}); potassium, 5.5 mmol l^{-1} (normal range, 3.5–5 mmol l^{-1}); sodium, 139 mmol l^{-1} (normal range, 135–147 mmol l^{-1}); alkaline phosphatase, 3.57 μkat l^{-1} (normal range, 0.5–2 μkat l^{-1}); albumin, 20 g l^{-1}; (normal range, 40–60 g l^{-1}); and parathormone, 227 ng l^{-1} (normal range, 14–72 ng l^{-1}). Flexible bronchoscopy with bronchoalveolar lavage showed no evidence of acute inflammation, granulomatous disease or malignancy. A CT scan revealed consolidation with calcification in the right upper lobe (Figure 1a). MRI of the chest was performed. The signal intensity of the pulmonary lesions was greater than the skeletal muscle on T_1 weighted imaging with fat saturation (Figure 1b). The signal intensity of the lesions was similar to that of skeletal muscle on T_2 weighted imaging (Figure 1c). The patient underwent a surgical biopsy, for which a histological examination demonstrated MPC. The patient underwent a partial parathyroidectomy and is in follow-up.

Case 2

A 35-year-old male with chronic renal failure secondary to chronic glomerulonephritis was on haemodialysis for 8 years. During clinical evaluation for transplantation, he underwent a chest radiograph that showed bilateral upper lobe consolidation. A CT scan also demonstrated consolidation with homogeneous calcification in the upper lobes (Figure 2a,b). The physical examination was normal. Laboratory studies revealed creatinine levels to be 510.88 μmol l^{-1} (normal range, 10–40 μmol l^{-1}); serum urea, 35.7 mmol l^{-1} (normal range, 3.0–6.5 mmol l^{-1}); calcium, 2.49 mmol l^{-1} (normal range, 2.20–2.58 mmol l^{-1}); phosphate, 3.71 mmol l^{-1} (normal

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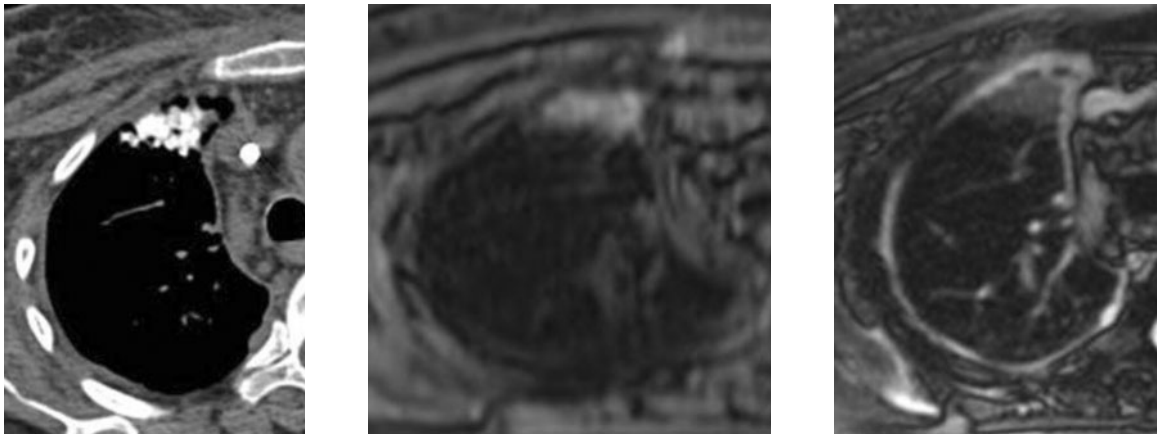

(a) (b) (c)

Figure 1. (a) Axial CT scan with mediastinal window showing a consolidation area with calcification in the right upper lobe. (b) Axial T_1 weighted image with fat saturation showing that the lesion has a higher signal intensity than skeletal muscle. (c) Axial T_2 weighted image showing the lesion has a similar signal intensity to skeletal muscle.

range, 0.80–1.60 mmol l^{-1}); potassium, 5.0 mmol l^{-1} (normal range, 3.5–5 mmol l^{-1}); sodium 140 mmol l^{-1} (normal range, 135–147 mmol l^{-1}); and parathormone, 340 pg ml^{-1} (normal range, 67–135 ng l^{-1}). MRI showed high signal intensity on T_1 weighted imaging and a signal intensity that was similar to that of skeletal muscle on T_2 weighted imaging (Figure 2c,d). Surgical biopsy was performed and a histological examination confirmed the diagnosis of MPC. The patient underwent a renal transplant and is in follow-up.

Discussion

Calcification is the deposition of calcium salts in tissue [5]. The two types of tissue calcification are metastatic and dystrophic. Metastatic calcification occurs when calcium salts are deposited in previously normal tissue, whereas dystrophic calcification occurs in previously damaged tissue [5]. Most cases of MPC occur in patients with hypercalcaemia, particularly those with hyperparathyroidism secondary to chronic renal failure [1]. MPC is found in 60–75% of haemodialysed patients at autopsy [6–8]. The histopathology features are calcium deposits located in the alveolar epithelial basement membranes, alveolar capillary walls, bronchial walls and media of pulmonary arterioles [4, 6].

On CT scans, MPC is characterised by centrilobular ground-glass nodular opacities, with numerous poorly defined nodules measuring 3–10 mm in diameter [1, 2, 8, 9]. Airspace consolidation is rarely seen [9]. Distribution of pulmonary calcification can be punctuate within the nodular opacities or ring-like and involve the entire nodule or the consolidation area [1, 2, 9]. Metastatic calcification tends to involve mainly the upper lung zones. This distribution is presumed to be related to the higher ventilation–perfusion ratio in the upper zones compared with the lower zones, resulting in a lower partial pressure of carbon dioxide and therefore a higher pH in the upper lung zones [1, 2, 5]. Chest radiographs are usually normal or show poorly defined bilateral opacities without signs of calcification [1, 2].

MRI is an increasingly widespread method and has the inherent advantage of not employing ionising radiation. The accuracy of MRI *vs* CT in detecting metastatic calcification has yet to be tested in subsequent studies. However, the great advantage of MRI over CT is the possibility to perform follow-up imaging in these patients without radiation exposure. Moreover, the contrast medium employed in MRI is less nephrotoxic than the iodinated contrast used in CT [10]. There is generally either no effect of calcium on signal intensity in MRI or a reduction in signal intensity [5]. In our cases, lung MRI showed a high lesion-to-muscle signal intensity ratio on T_1 weighted imaging. The MRI appearance in these cases was unusual and similar to certain calcified brain lesions that appear hyperintense on T_1 weighted MRI [11]. A previous study reported increased signal intensity in a case of MPC [12]. This increase in signal intensity is due to a shortening of the T_1 relaxation of protons, and the degree of T_1 shortening is directly related to the surface area of the calcium crystals [12]. The T_1 shortening of water protons in calcified tissues can be attributed to the surface effects of diamagnetic calcium particles. The water protons adhering to the surfaces of crystals relax more quickly than those distant from the crystal surface [12], although calcium also causes a change in T_2 relaxivity and proton density [11]. The latter factors reduce MR signal intensity and nullify any potential increase in signal intensity caused by T_1 shortening. Only in cases in which the microscopic crystal surface area is very high can the T_1 effect predominate, causing a net increase in MR signal intensity [11]. These MR signal variations are directly influenced by the concentration and surface of calcium salts [11]. As previously demonstrated, a decrease in hydrogen proton density and a reduction in T_2 relaxivity are not able to overcome the signal intensity increase from T_1 shortening [11]. For this reason, we observed a high signal in T_1 weighted images. With calcium concentrations above 30–40%, T_1 weighted signal intensity progressively decreases [11]. Although a pathological study has been performed in our patients, as well as

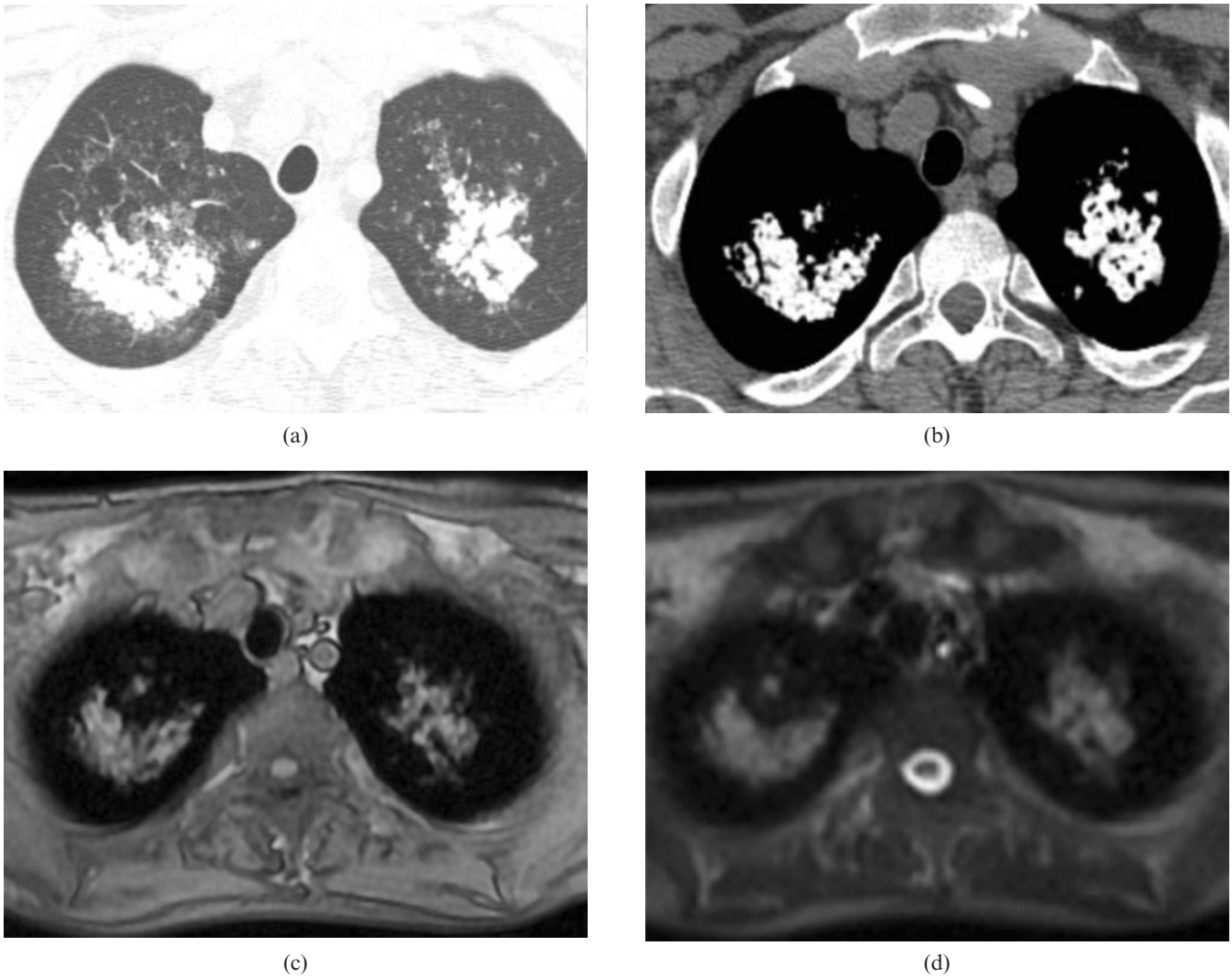

Figure 2. Axial CT scan with (a) lung and (b) mediastinal windows showing consolidation areas with calcification in the upper lobes. (c) Axial T_1 weighted images demonstrated higher signal intensity for the lesions than for skeletal muscle. (d) Axial T_2 weighted imaging showing that the lesions presented with hypersignal.

in several cases reported in the literature [1, 3, 7–9], we believe that the clinical history associated with the characteristic high-resolution CT findings is sufficient to establish the diagnosis and avoid a lung biopsy.

Conclusion

MPC may present with calcified consolidations on CT that show as a higher signal intensity than skeletal muscle on MRI. This probably occurs because the low calcium concentration of the lesion changes the surface effects of diamagnetic calcium particles, resulting in T_1 shortening of water protons. Therefore, MRI is a good option for characterising lung calcium accumulation caused by a metabolic disorder. This condition should be kept in mind when dialysis patients develop unexplained radiographic changes or pulmonary symptoms.

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