

Images in Nephrology
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Uremia-associated cardiovascular and lung injury

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A 23-year-old woman presented to the emergency department with severe dyspnea. She reported progressively worsening dyspnea during the previous 2 weeks. She had a history of end-stage renal disease (ESRD) due to chronic glomerulonephritis. The patient had been maintained on peritoneal dialysis (PD) for 3 years. The PD prescription consisted of three 2 L exchanges per day, 1.5% dextrose solutions. Chemistries were significant for a parathyroid hormone of 146.7 pmol/L (normal, 1.3–9.3 pmol/L), phosphorous of 3.52 mmol/L (normal, 0.8–1.65 mmol/L) and serum calcium of 2.54 mmol/L (normal, 2.1–2.6 mmol/L). The chest radiograph showed a typical picture of congestive heart failure with cardiomegaly and ground glass opacity changes in both lung fields. Transthoracic echocardiography revealed a dilated left ventricle and left atrium with cardiac muscle and left ventricular papillary muscle echo enhancement (Figure 1). Chest computed tomography (CT) images showed left atrium and ventricle dilatation. CT images also showed myocardium, lung and coronary artery calcification (Figure 2). The results of single-photon emission CT technetium-99m methoxyisobutylisonitrile (SPECT ^{99m}Tc-MIBI) showed that the mild reductions of

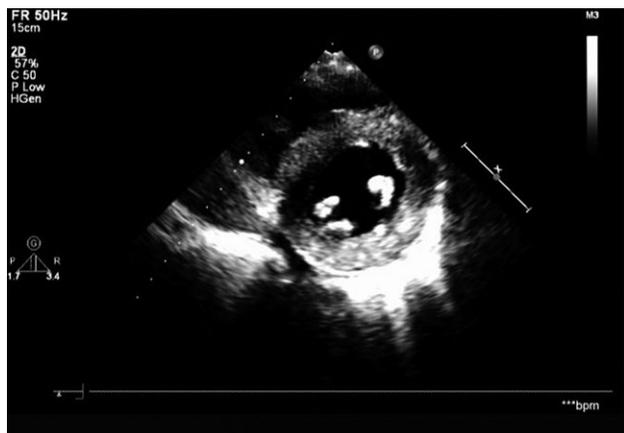


Fig. 1. Echocardiography showing cardiac muscle and left ventricular papillary muscle echo enhancement.

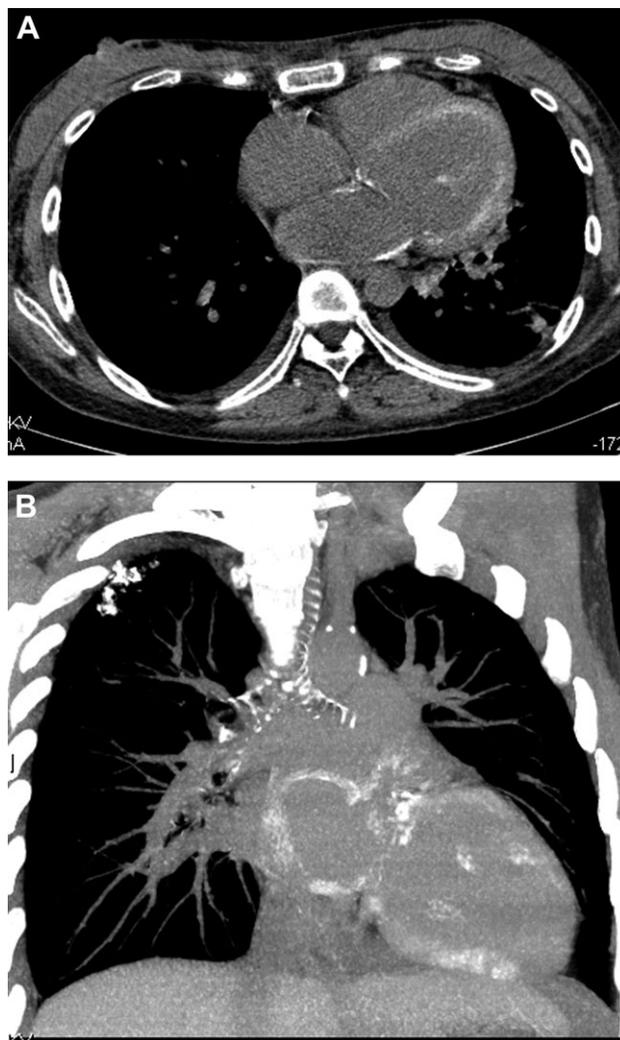


Fig. 2. CT images showing left atrium and ventricle dilatation (A) and myocardium, lung and coronary arteries calcification (B).

^{99m}Tc -MIBI uptake were found in anterior, posterior and posterolateral segments of the left ventricular due to myocardial damage.

Due to the diminished ability of the kidneys to excrete a phosphorus load as glomerular filtration decreases, most patients with ESRD have a predisposition toward elevated levels of serum phosphorus. The development and progression of secondary hyperparathyroidism are the primary consequence of this elevated serum phosphorus. An additional consequence is a predisposition to metastatic calcification when the product of serum calcium and phosphorus is elevated. Both of these may contribute to the high morbidity and mortality of patients with end-stage renal failure [1]. The former results in the development of renal osteodystrophy and exposes the patient to excessively high serum levels of parathyroid hormone, which is a cardiovascular risk factor in moderate chronic kidney disease [2]. The latter often results in calcification of soft tissue, joints, blood vessels and internal viscera, such as myocardium, lung, liver and kidney. It is certainly conceivable that vascular and cardiac calcification in particular leads

to complications and increased mortality, especially in children and young adults with ESRD [3].

Conflict of interest statement. None declared.

References

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