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Case Report

Extensive myocardial calcifications in a dialysis patient: A porcelain heart manifesting with abdominal pain ☆

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ABSTRACT

This case report describes a 41-year-old male patient with chronic kidney disease on peritoneal dialysis presenting with upper abdominal pain and mild thigh numbness. CT chest demonstrated extensive myocardial calcifications and left atrial thrombus. This case emphasizes the clinical relevance of myocardial calcifications, especially in patients with end-stage renal disease. It also highlights the potential association between these calcifications and complications such as atrial fibrillation and thromboembolic events. The findings emphasize the need for diagnostic vigilance and an improved understanding of the pathophysiology of myocardial calcifications in the context of renal disease.

Introduction

Myocardial calcification, although a relatively rare condition, can result from various cardiac and systemic diseases. This finding can be identified during diagnostic imaging studies, especially CT scans, with variable imaging appearances. It is important to identify these calcifications, as they are frequently associated with adverse outcomes. These calcifications are commonly linked to calcium deposition in normal myocardial tissue, particularly in patients with end-stage renal disease (ESRD) on hemodialysis. Although the imaging findings may be non-specific, the correlation of calcification patterns, clinical history, and alteration in serum calcium and

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phosphorus levels can offer critical insights. This enables clinicians to recognize the origin and clinical relevance of myocardial calcifications more effectively.

Case presentation

A 41-year-old Caucasian male, with chronic kidney disease secondary to membranous proliferative glomerulonephritis (baseline creatinine level, 4.4 mg per deciliter; glomerular filtration rate, 14 mL/min/1.73 m^2 of body surface) presented to the emergency department with a 2-week history of the left upper abdominal pain and mild left thigh numbness. He reported no fever, chills, nausea, weight loss, or abnormal bowel habits. He had no history of smoking, alcohol abuse, or illicit drug use.

Physical examination revealed an irregularly irregular cardiac rhythm and decreased left femoral and dorsalis pedis pulses. His abdomen was mildly distended, with focal tenderness in the left upper quadrant. The complete blood counts revealed mild anemia (Hemoglobin of 10.5 g/dL, reference range: 12.0-16.0 g/dL) and leukocytosis (white cell count of 12.200 /μL, reference range: 4500-11,500/ μL). An electrocardiogram showed atrial fibrillation.

He received his first kidney transplant at age 22, which was rejected after three years. He was then placed on hemodialysis for two years before having a second kidney transplant. This was rejected after six years. Following this, he resumed hemodialysis for another six years and then switched to peritoneal dialysis for the last two years. The patient's calcium levels were consistently low, between 7.1 and 7.7 mg/dL (reference: 8.6-10.2 mg/dL); Simultaneously, his phosphorus levels were consistently elevated, varying between 6.2 and 7.5 mg/dL (normal range: 2.5-4.5 mg/dL), and his creatinine levels were persistently high, ranging from 1.9 to 4.4 mg/dL (normal range: 0.66-1.25 mg/dL). The patient underwent a plain and IV and oral contrast-enhanced CT scan of the chest, abdomen, and pelvis due to suspicion of an acute abdominal condition. The chest CT revealed cardiomegaly, left ventricular hypertrophy, and extensive calcifications affecting both atriums, ventricles, and atrioventricular valves (Fig. 1). On contrast CT, an oval-shaped, mobile filling defect, measuring 18 × 12 mm was noted in the left atrium and atrial appendage, which was consistent with thrombus (Fig. 2). There was a subpleural, high attenuation consolidative opacity with punctate calcifications in the right lung base (Figs. 1A and B).

At the level of abdomen, there was mild splenomegaly with a peripheral, wedge-shaped, hypoenhancing region (Figs. 3A and B) reflecting embolic splenic infarction. Moderate free fluid was in the peritoneal cavity. Atrophic calcified kidneys with multiple small cortical cysts (acquired renal cystic disease) and rejected transplanted kidneys with extensive amorphous calcifications in the pelvis (Fig. 1D) were also noted. The finding of the intraluminal filling defect of the left superficial femoral artery with partial luminal obstruction (Fig. 4) suggested a cardioembolic origin in the context of atrial fibrillation. The referring physician was immediately notified of the CT scan findings. Given the poor acoustic window and extensive myocardial calcification, which caused significant posterior acoustic shadowing, the transthoracic echocardiography (TTE) was associated with very low-quality images. The patient declined to undergo transesophageal echocardiography.

Anticoagulant therapy, according to established guidelines, was initiated, and a consultation with a vascular surgeon was arranged.

Table 1 – Summary of etiology of cardiac calcification.

Dystrophic calcification
Ischemic:
Myocardial infarction and complication (aneurysm/pseudoaneurysm)
Traumatic and Iatrogenic:
Cardiac surgery
Irradiation [6]
Hemorrhage
Infectious:
Myocarditis (mostly fulminant viral)
Myocardial abscess
Sepsis [26]
Granulomatous infections (Tuberculosis, fungal) [46]
Echinococcal disease [47]
Inflammatory:
Rheumatic heart disease
Endomyocardial fibrosis
Heart transplant (rejected) [48,49]
Neoplastic:
Metastatic disease
Primary cardiac tumors
Other
Drugs, including cyclosporine, steroids, calcium chloride, cocaine [50]
Calcification of the mitral annulus [7]
Pulmonary hypertension
Metastatic Calcification
Renal failure
Hyperparathyroidism[11,51]
Oxaluria[52]
Aluminum intoxication (related to hemodialysis)[53]
Dietary calcium and/or vitamin D deficiency
Sarcoidosis (related to vitamin D hyperactivation)
Idiopathic Calcification[18]
Mimics
Valvular/annular calcification[7]
Pericardial calcification[27]
Vascular (coronary or great vessel) calcification[3,27]
Calcified intraluminal thrombus
Calcified amorphous tumor[54]

Discussion

Myocardial calcification or calcinosis, is a rare cardiac pathology, first reported by Simmonds in 1908 [1], Recognition of this pathology in clinical practice has been significantly enhanced with the increased use of cardiac CT. This pathology may be caused by different mechanisms (Table 1), which are typically classified based on the homeostasis of calcium (Ca), phosphorus (P) metabolism, and damaged myocardium [2].

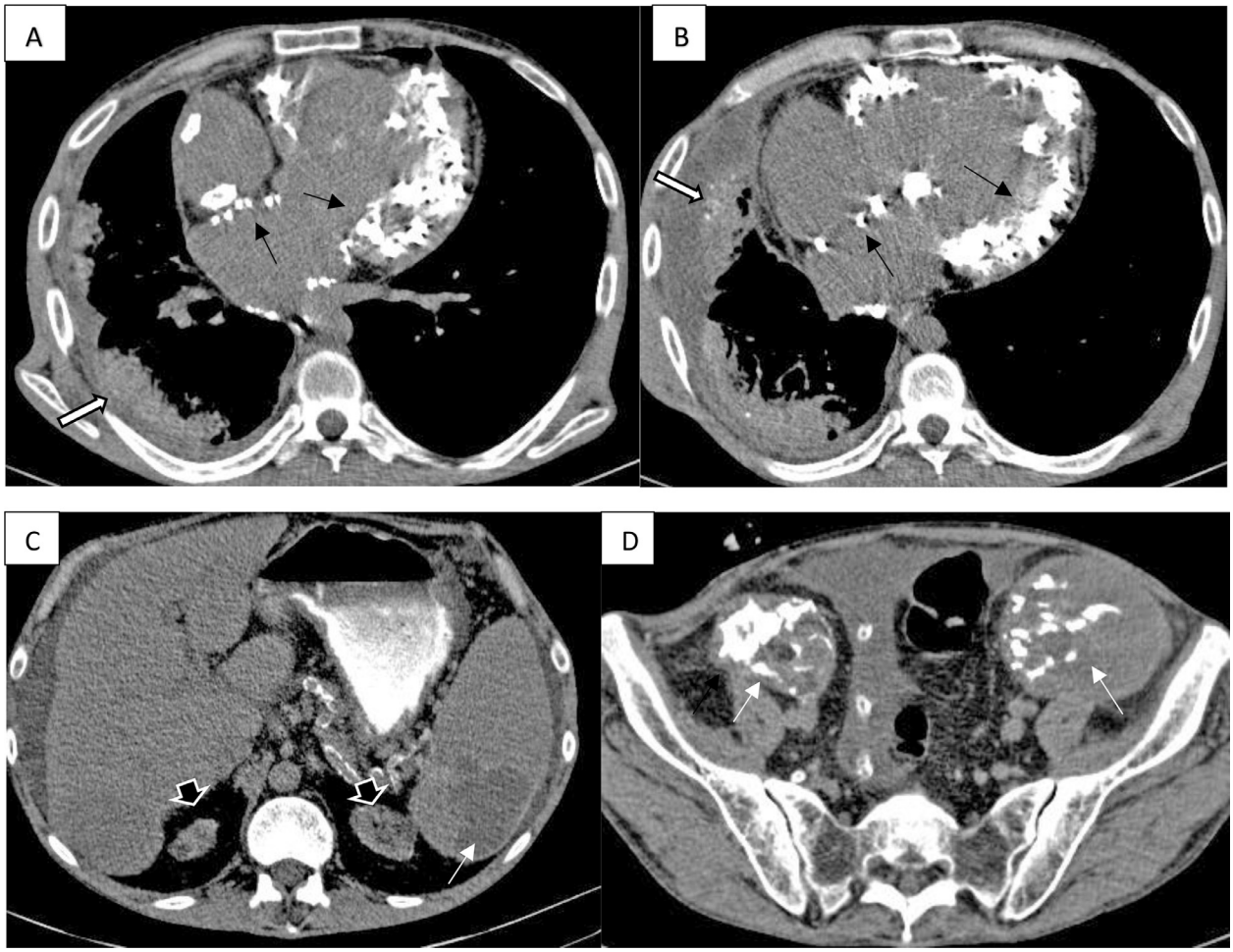

Fig. 1 – (A, B) Axial slices of unenhanced CT of the chest reveals increased myocardial wall thickness, diffuse nodular and amorphous calcifications, throughout the endocardial surfaces and myocardium of both atria and ventricles (black arrows), Calcification of the interatrial septum and mitral valve are shown. Metastatic pulmonary calcification appears as high attenuated consolidation with small punctate calcifications in the right lung base (white arrow). (C) Axial slice of non-enhanced CT of the abdomen at the level of spleen shows peripheral, wedge-like hypoattenuated lesion in the lower pole of the spleen (arrow). Both native kidneys are atrophic with multiple small cortical cysts (wide arrows). (D) amorphous calcifications of the rejected transplanted kidneys on both sides of the pelvis (white arrows).

Dystrophic calcification is the most common type and usually occurs following myocardial infarction [3,4]. This condition is characterized by the deposition of calcium in the injured myocardium in the setting of normal calcium and phosphorus levels. The prevalence of dystrophic calcifications is estimated to be about 8% in myocardial infarctions older than 6 years [5]. Other causes of dystrophic calcification include trauma, radiation [6], infectious and/or inflammatory causes, and neoplastic pathology [2]. Rarely, there have been reports where the primary disorders originate from the adjacent myocardial tissue, as recognized in cases like exuberant and extensive mitral annulus calcification [7], or pericardial calcification extending into the surrounding myocardium [2].

In the second type, metastatic calcification involves calcium deposition in otherwise healthy myocardium, indicating a systemic disruption in calcium metabolism. The locations and patterns of this type of calcification vary. It can appear faint or dense and usually presents as nodular and/or amorphous. The deposition may be widespread or localized, affecting a single heart chamber or all of the cavities [3]. This form is most commonly seen in chronic kidney disease (CKD) with an estimated prevalence of 59% in postmortem studies of those patients with CKD on maintenance hemodialysis [8,9]. Other causes include acute kidney disease (AKD), increased bone turnover, oxaluria [10], primary or secondary - hyperparathyroidism related to dietary deficiency of calcium and Vitamin D (11), and other Vitamin D-related disorders [4]. There are multiple CKD and dialysis-related factors contributing to dystrophic calcification, including elevated phosphorus levels, and secondary or tertiary hyperparathyroidism which results in hypercalcemia and culminates in an abnormal calcium and phosphorus homeostasis manifesting as a high product of Calcium-Phosphorus (Ca x PO4) with a normal range is less than 55 mg/dL [12–14], as well as side effects of treatment with calcium-containing phosphate binders and vitamin D analogs [15].

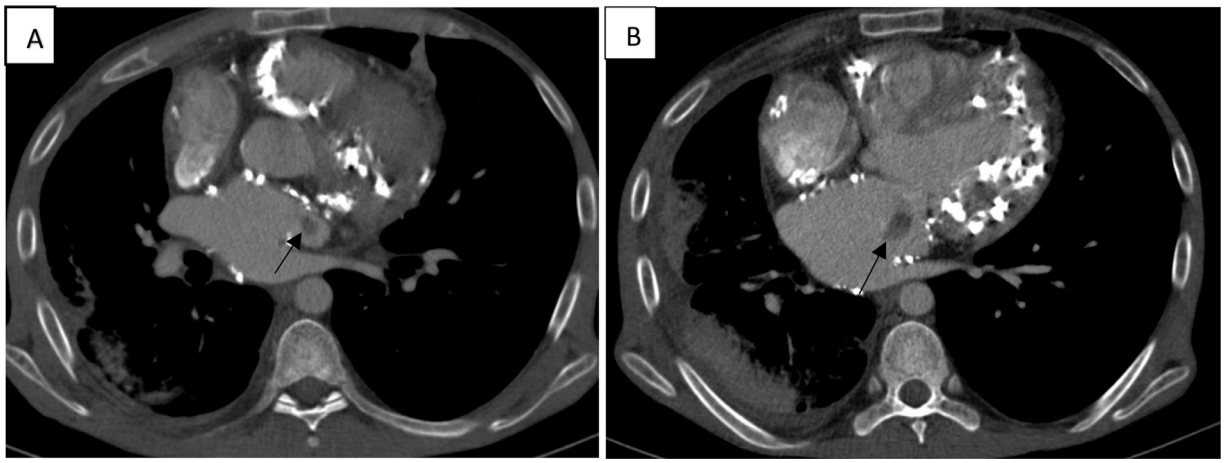

Fig. 2 – (A, B) Axial contrast enhanced CT scan at the level of heart: An oval-shaped, low-attenuation filling defect signifies a thrombus in the left atrium and atrial appendage (arrow). Nodular myocardial calcifications affect both atria, left ventricle, and papillary muscles.

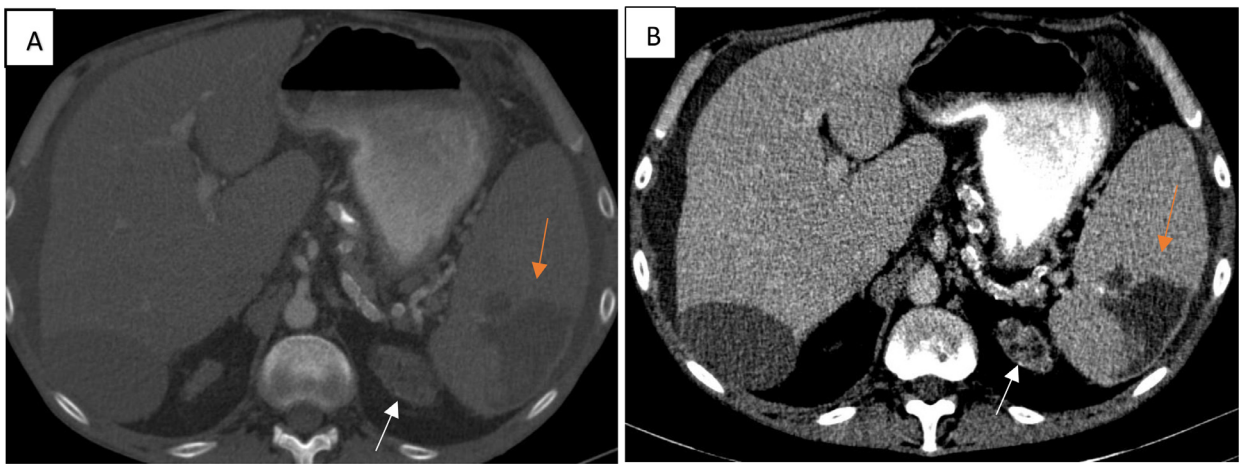

Fig. 3 – (A, B) Axial CT scan of upper abdomen with IV and oral contrast on portal and delayed phases shows mild splenomegaly with a focal, wedge- shaped, capsular based and hypoenhanced region (black arrow), reflecting splenic infarction. Moderate free fluid in the peritoneal cavity. Atrophic calcified kidneys accompanied with multiple small cortical cysts (white arrow).

The duration of hemodialysis has also been identified as a strong risk factor, primarily contributing to accentuated imbalance in blood calcium and phosphorus levels [16]. In extreme conditions, calcium depositions can extend beyond the heart, affecting various extracardiac structures including skin, systemic arteries, pulmonary veins, soft tissue, lungs, stomach, and kidneys [3,17].

Idiopathic calcification is a third type of myocardial calcification, with unrecognized prevalence, etiology, and definitive mechanism [18]. This type of myocardial calcification is most likely a manifestation of dystrophic or metastatic calcifications secondary to a clinically occult or unrecognized pathologic process [2,18]. It is hypnotized that various types of myocardial calcification originate from a common pathway. In this process, calcium enters cardiomyocytes, either due to an increased concentration gradient from hypercalcemia or cellular membrane defect secondary to myocardial injury (ischemic or nonischemic causes). As a result, calcium hydroxyapatites begin to establish as crystals or noncrystalline amorphous forms and accumulate in mitochondria, then diffuse throughout the cardiomyocyte and subsequently spread to the extracellular interstitial space [19]. It is clinically important to use imaging for early identification of the cause of myocardial calcification, it may help to prevent progression and treat complications. For example, the timely detection of rapid onset myocardial calcification in the setting of septicemia, which likely results from a combination of myocardial injury and renal dysfunction, can guide the management strategy [4]. For patients with CKD, an essential preventive strategy against metastatic calcification is the meticulous management of calcium and phosphorus levels. A wide range of treatment options are currently available, including renal

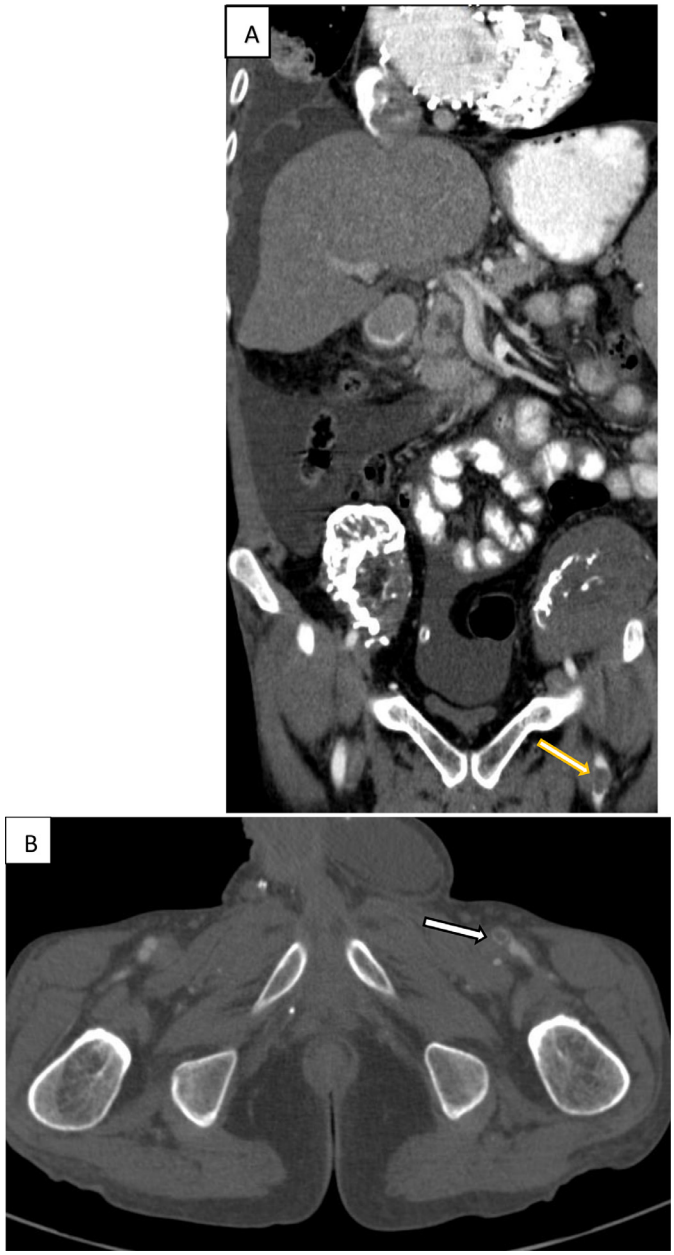

Fig. 4 – (A, B) Coronal reformat and axial contrast-enhanced abdominal and pelvis CT scan shows: A low - attenuated intraluminal embolism in the proximal left superficial femoral artery (arrow). Moderate ascites and calcified rejected transplanted kidneys are noted.

transplant, dietary adjustments, modifying the calcium content in dialysate, utilizing phosphate binders, administering vitamin D, and considering parathyroidectomy [20]. However, achieving optimal management remains challenging, and no single treatment offers a comprehensive solution.

Myocardial calcification, though infrequent, is not always a benign sequelae of derangements in mineral metabolism. It can cause mechanical and electrical abnormality of cardiac function, typically leading to restrictive physiology with normal function or heart failure with preserved ejection fraction (HFpEF), ventricular and supraventricular arrhythmias, coronary events, and even sudden cardiac death, with arrhythmias being the most frequent cause [17,21].

CT, particularly ECG-gated cardiac CT, is the gold standard imaging modality for detecting and characterizing myocardial calcifications due to high spatial and contrast resolution and its ability in reducing heart-beat-related motion artifacts [2,10,22]. However, standard nongated chest CT can also reliably identify and localize such calcifications. In recent years, advancements in CT technology, especially the introduction of dual-energy CT (DECT) and spectral CT, have significantly enhanced in vivo tissue characterization. These advancements provide a significant improvement in detecting subtle myocardial calcification or coronary plaque characterization by offering detailed material composition [23,24]. The superiority of DECT to assess material density (MD) maps, which can se-

lectively demonstrate or exclude specific materials including iodine, calcium or water significantly improves both subjective and objective diagnostic leverage [25]. By suppressing the high-density calcium in tissues, DECT can identify subtle myocardial calcification as a low attenuation area within the myocardium [26]. Myocardial calcium appears as echogenic foci with posterior acoustic shadowing on echocardiography. MRI can also suggest myocardial calcifications through findings of low signal intensity on T1-W and T2-W images, although it is not a specific sign.

The patterns and specific localizations of these calcifications can provide clues into the underlying cause. Generally, dystrophic calcifications have a tendency to be localized and linear, whereas metastatic calcifications may present as diffuse, globular, or coarse amorphous patterns [2,27]. Atrial involvement is usually associated with rheumatic heart disease along with mitral valve engagement, but it can also occur in cases of metastatic calcifications. Various pathologies like vascular, valvular, pericardial, and tumoral calcification can mimic myocardial calcification. The morphology and distribution of calcification play a crucial role in discerning the precise anatomical location. For instance, coronary calcifications appear as linear or tram-track patterns along their typical anatomical path, while pericardial calcifications exhibit a curvilinear pattern, often globally involving the pericardium. The application of cardiac CT scans is pivotal for distinguishing calcifications situated within the myocardium from those in extramyocardial locations.

Cardiovascular disease accounts for more than 50% of deaths in patients with end-stage renal disease (ESRD) receiving dialysis [14]. There are various factors that have been suggested to contribute to elevated risk, but recently roles of dialysis-specific pathogenetic factors such as hyperphosphatemia, hypercalcemia that lead to high levels of serum calcium x phosphorus product(Ca x PO4), and hyperparathyroidism in the development of coronary artery calcification, myocardial calcification and aortic valve calcification has been raised [14,15,28]. Regmi et al. [29] found that elevated product of serum calcium and phosphorus (Ca x PO4) exceeding 55 mg/dL is an independent risk factor for cardiovascular events in CKD patients.

Atrial fibrillation (AF) as the most common arrhythmia is reported to occur in approximately 7% to 20% of patients with end-stage renal disease (ESRD), (14)a rate that is 2-3 times higher than observed in the general population [14]. This association is particularly concerning as AF in ESRD patients is linked with adverse outcomes, including thromboembolic events, ischemic strokes, and mortality [14,15]. Multiple potential mechanisms might account for the increased prevalence of AF in this group, such as ischemic heart disease, heart failure, and myocardial or valvular calcification [16]. Left atrial (LA) thrombus, most commonly seen in the left atrial appendage (LAA), is a known complication of atrial fibrillation, occurring in 13%-15% of cases [30]. This thrombus formation in the LAA is mainly attributed to slow flow velocity and prolonged LAA flow time, often referred to as atrial stunning [31,32]. The LA/LAA thrombi in the presence of atrial fibrillation significantly increased the risk of cerebral strokes or peripheral embolism, with an annual risk ranging from 3% to 6% [33,34]. Therefore, diagnosing LA/LAA thrombus, especially in those with atrial fibrillation, is crucial before the cardioversion to prevent systemic embolization [35]. Currently transesophageal echocardiography (TEE) is considered the gold standard modality to detect thrombi in the LA/LAA [36]. However, it is a semi-invasive procedure with potential serious complications. Another challenge in using TEE to diagnose and estimate the size of LAA thrombus arises from the complex anatomy and various morphology of the LAA [37]. There are multiple noninvasive alternative modalities recently available for the detection of LAA thrombus. Contrast-enhanced ECG-gated cardiac computed tomography (CCT) and Cardiac MRI (CMR) are widely investigated for diagnosis of LA/LAA thrombus. On CCT, LAA thrombus appears as a filling defect on arterial and more appreciably on 6-minutes delayed phase because reduced LAA filling rates can cause false-positive or pseudofilling defects during arterial phase [38]. A meta-analysis by Romero et al. [33] found that CCT's diagnostic accuracy for detecting of LA/LAA thrombus had an average sensitivity of 96% and specificity of 92%, respectively. However, CCT has notable considerations, including the potential nephrotoxicity of iodinated contrast agents, radiation exposure risks, and beam-hardening artifacts in extensively calcified myocardium. Additionally, acquiring high-quality images of CCT can be challenging in AF patients with irregular or rapid heart rates [39]. Recent studies have demonstrated the superior accuracy of dual-energy CT (DECT) over TEE in distinguishing LAA thrombus from blood stagnation [25,40]. This is achieved using iodine density (MD) maps, which can detect even minimal iodine concentrations within the LAA cavity [40–42]. Cardiac magnetic resonance imaging has been established as a noninvasive, standard modality to assess cardiac function and tissue characterization, although it has a lower spatial resolution when compared to CCT. For patients with atrial fibrillation, CMR has emerged as an accurate diagnostic modality for diagnosis and assessment of LA/LAA thrombus [43]. Among noncontrast and contrast-enhanced sequences, Late gadolinium enhancement (LGE) technique with a long inversion time had the highest sensitivity, specificity, and diagnostic accuracy [44,45].

Patient consent

We confirm that informed consent for publication of this case report was obtained from the patient.

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