The Heart Surgery Forum 2022-4759
25 (4), 2022 [Epub July 2022]
doi: 10.1532/hsf.4759

Online address: http://journal.hsforum.com

Reversible Myocardial Calcification Following Acute Heart Failure and Kidney Injury Caused by Valsalva Sinus Rupture

Weichi Chen, MD, Jinzhou Liu, MD, Guotao Ma, PhD, Xingrong Liu, PhD, Qi Miao, PhD, Chaoji Zhang, PhD

Department of Surgery, Peking Union Medical College Hospital, Chinese Academy of Medical Sciences, Beijing, China

ABSTRACT

A 47-year-old previously healthy man was referred to a local hospital with chest tightness, oliguria, and lower extremity edema for seven days. An initial investigation revealed acute heart failure and kidney injury. The patient was intensively treated with cardiac and renal replacement therapy, and cardiorenal function improved one week later. Two months later, echocardiography was performed because chest tightness and edema had not resolved. Echocardiography showed Valsalva sinus rupture, and the patient was transferred to our center. Myocardial calcification was observed in the left ventricular wall on computed tomography after admission. The patient underwent cardiac surgery and recovered smoothly. At the three-year follow up, the patient was asymptomatic with normal renal function and serum electrolytes. Imaging revealed a significant reduction in diffuse calcification of the left ventricular wall. This case indicates that this rare form of reversible myocardial calcification may be associated with acute heart and renal failure caused by Valsalva sinus rupture. The results of this case will guide clinicians about further management and timely referral of such patients to appropriate specialties.

INTRODUCTION

Myocardial calcification can be either dystrophic (occurring in infarcted, non-viable tissue) or metastatic (due to abnormalities in calcium-phosphorus metabolism or elevated parathyroid hormone) [Nance 2015]. However, myocardial calcification secondary to acute heart and renal failure caused by congenital heart disease rarely has been reported. We herein report a case of reversible myocardial calcification associated with Valsalva sinus rupture. We performed serial follow up of myocardial calcification for three years. The results of this case will guide clinicians about further management and timely referral of such patients to appropriate specialties.

Received March 29, 2022; accepted April 25, 2022.

Correspondence: Chaoji Zhang, PhD, Department of Surgery, Peking Union Medical College Hospital, Chinese Academy of Medical Sciences, Beijing 100730, China, Telephone 86-10-69152820 (e-mail: chaojizhang@163.com).

CASE PRESENTATION

A 47-year-old man with chest tightness, oliguria, and lower extremity edema for seven days was admitted to a local hospital in March 2018. The patient had no history of cardiac, renal, or other disorders. On admission, the patient's vital signs were as follows: temperature, 36.5°C; respiratory rate, 33 breaths/min; heart rate, 120 beats/min; and blood pressure, 89/56 mmHg. The patient's laboratory test results were as follows: prothrombin time, 17 seconds (international normalized ratio, 1.5); albumin, 2.9 mg/dL; aspartate transaminase, 520 U/L; total bilirubin, 28 mg/dL; lactate dehydrogenase 516 U/L; creatine kinase, 800 U/L; creatine kinase isoenzyme, 20 µg/L; cardiac troponin I, 20 µg/mL; sodium, 129 mmol/L; potassium, 5.2 mmol/L; blood urea nitrogen, 20 mmol/L; creatinine, 180 mmol/L; magnesium, 2.5 mmol/L; calcium, 1.80 mmol/L; phosphorus, 1.89 mmol/L; N-terminal pro-B-type natriuretic peptide, 8420 pg/mL; high-sensitivity C-reactive protein, 25 mg/L; erythrocyte sedimentation rate, 30 mm/h; anti-ds-DNA antibody (+). Computed tomography (CT) showed small pericardial effusion, bilateral pleural effusion, and abdominal effusion. The patient required vasopressor therapy for hemodynamic support and continuous renal replacement for acute renal insufficiency. The patient's clinical condition gradually improved, and renal replacement was withdrawn on days 3 and 5. Urine volume was restored by oral diuretics, acreatinine concentration was maintained at 120–135 µmol/L. Lupus nephritis was suspected because of the high C-reactive protein concentration, and the patient was treated with an experimental regimen (prednisolone and hydroxychloroquine). One month later, echocardiography was performed because chest tightness and edema had not resolved. Echocardiography showed Valsalva sinus rupture, and the patient was transferred to our center in May 2018. Repeated transthoracic echocardiography (TTE) demonstrated enlargement of both atria and ventricles. The rate of inspiratory change of the inferior vena cava was >50%, left ventricular ejection fraction was 64%, left ventricular wall movement and systolic function were normal, the left ventricular myocardium was unusually hyperechoic, and two strong echogenic masses were found in the myocardium at the base

of the interventricular septum, indicating Valsalva sinus rupture (left-to-right shunt). Mild aortic insufficiency with small pericardial effusion was observed. Coronary CT angiography revealed no significant coronary artery disease. Dilatation of the right coronary sinus was communicated with the outflow tract of the right ventricle with a width of 12.9 mm. The main pulmonary artery was widened. Chest CT showed an enlarged heart shadow and an increased diffuse density of the left ventricular wall.

After active preoperative preparation, cardiopulmonary bypass was performed under general anesthesia on May 10, 2018. Ruptured sinus aneurysm located in the right coronary sinus (10 mm in diameter) was detected intraoperatively, with calcification of the expansive aneurysm wall. The ruptured orifice was repaired with Dacron pieces. Aortic valve replacement was performed owing to aortic valve calcification and prolapse. The extracorporeal circulation was smoothly weaned. The extracorporeal circulation time was 150 minutes, and the clamp time was 99 minutes. The patient recovered well postoperatively, and no postoperative renal replacement therapy was required. The patient was discharged seven days postoperatively. Because of insufficient evidence of connective tissue disease after the immunologist's consultation, postoperative hospital hormone therapy was stopped. At the three-year follow up, the patient was asymptomatic and was not taking any medication other than warfarin. The patient had normal renal function and serum electrolytes. TTE showed slightly enhanced echo of the left ventricular wall, with a left ventricular ejection fraction of 60% and normal wall motion. CT revealed significantly reduced diffuse calcification of the left ventricular wall, a normal cardiac shadow, and no dropsy of the serous cavity. (Figure 1)

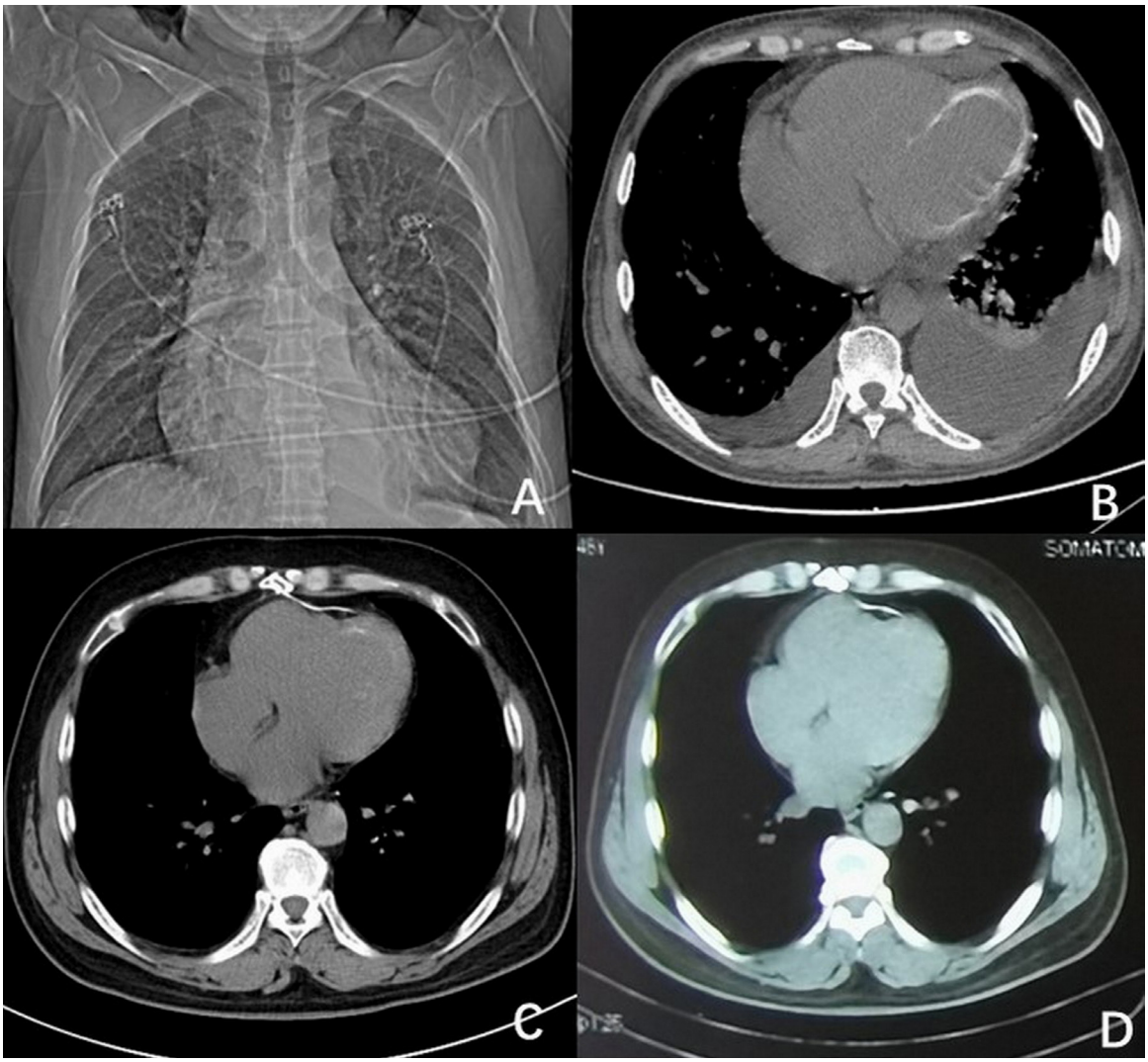

Figure 1. A) Chest X-ray showing no calcification at the border of the cardiac silhouette at disease onset. B) CT two months after onset showing calcification of the left ventricle and interventricular septum. C) CT 12 months after discharge showing a significant reduction in diffuse calcification of the left ventricular wall. D) CT 36 months after discharge showing complete resorption of calcification deposits in the heart.

DISCUSSION

In terms of dystrophic calcification, there have been various reports of myocardial calcification associated with either coronary artery disease or a history of myocardial damage. In a study of 21 patients with myocardial calcification, Ananthakrishna et al. [Ananthakrishna 2016] demonstrated stenosis of >75% in at least one major coronary artery in all 21 patients, with three-vessel disease in over half of patients. Approximately 8% of individuals who suffer a sizable myocardial infarction will develop myocardial calcification after six years [Gowda 2004]. In our case, an elevation in myocardial enzymes showed acute myocardial necrosis resulting from sudden Valsalva sinus rupture. Previous studies have reported that cardiac myocyte necrosis results in increased membrane permeability and an influx of calcium ions [Schellhammer 2002; Li 2021], with consequent development of myocardial calcification.

Metastatic myocardial calcification refers to calcium deposition in non-infarcted tissue, resulting from impaired calcium phosphate metabolism, which is most frequently described in patients with chronic renal failure, oxalosis, and secondary hyperparathyroidism [Lasser 1983]. Patients with chronic renal failure are more likely to manifest these calcifications in the cardiac, renal, and vascular systems. In an autopsy study of dialysis patients, Kuzela et al. [Kuzela 1977] reported that 58.9% of patients had myocardial calcification. A possible explanation is that patients with acute renal failure are initially hypocalcemic due to an inability to produce 1,25(OH)2D3 (the active form of vitamin D), leading to an imbalance in calcium and phosphorus and subsequent myocardial calcification [Wada 1993; Zakouta 2013]. Hemodialysis also has been shown to cause calcium and phosphorus imbalance, which may contribute to the development of myocardial calcification. However, the imbalances caused by end-stage renal disease and hemodialysis are typically thought to lead to more insidious development of calcification, rather than onset within a matter of weeks, as was the case with our patient [Jing 1998; Lee 2012].

Kapandji et al. [Kapandji 2018] reported that three critically ill patients underwent extracorporeal membrane oxygenation (ECMO) and had extensive myocardial calcification, which was confirmed by multimodal imaging. The combination of multiple factors, such as prolonged hemodynamic failure, profound acidosis, a high vasopressor dose, and renal failure may lead to this unusual and severe complication. Myocardial damage secondary to other risk factors might also be a catalyst for calcium deposition.

Our case is distinctly unusual because the massive deposition of calcium in the myocardium occurred in the absence of any known metastatic or dystrophic initiating factors. Coronary CT angiography demonstrated no apparent coronary artery disease, and the patient also did not have any history of childhood illness resembling chronic kidney disease. Although the serum creatinine concentration increased to a concentration that would indicate kidney disease, the patient's creatinine concentration returned to baseline (1.0 mg/dL) after cardiac surgery. Furthermore, the patient's urine output and urinalysis were within normal limits. It seems likely that multiple factors contributed to the development of myocardial calcification with sudden rupture of the Valsalva sinus. We postulate that myocardial calcification occurred secondary to acute cardiac damage and acute kidney failure caused by Valsalva aneurysm rupture.

The prolonged natural course of myocardial calcification has not been fully elucidated because of the poor outcomes of affected patients [Zakouta 2013; Kimura 2019]. In our case, left ventricular calcification almost completely disappeared on chest CT over a period of three years. This finding suggests that massive myocardial calcification can completely regress.

CONCLUSION

In conclusion, the results of this case will guide clinicians about further management and timely referral of patients to appropriate specialties. In patients with myocardial calcification, positive treatment may prevent further detrimental structural and functional changes to the cardiovascular system and reverse myocardial calcification.

ACKNOWLEDGEMENT

We thank Emily Woodhouse, PhD, from Liwen Bianji (Edanz) (www.liwenbianji.cn) for editing the English text of a draft of this manuscript.

REFERENCES

Ananthakrishna R, Moorthy N. 2016. Dystrophic myocardial calcification. Indian Heart J. 68 Suppl 2: S180-S181.

Gowda RM, Boxt LM. 2004. Calcifications of the heart. Radiol Clin North Am. 42(3):603-17, vi-vii.

Jing J, Kawashima A, Sickler K, Raval BK, Oldham SA. 1998. Metastatic cardiac calcification in a patient with chronic renal failure who was undergoing hemodialysis: radiographic and CT findings. AJR Am J Roentgenol. 170(4):903-5.

Kapandji N, Redheuil A, Fouret P, et al. 2018. Extensive Myocardial Calcification in Critically Ill Patients. Crit Care Med. 46(7): e702-e706.

Kimura Y, Seguchi O, K KA, et al. 2019.Massive Biventricular Myocardial Calcification in a Patient with Fulminant Myocarditis Requiring Ventricular Assist Device Support. Intern Med. 58(9):1283-1286.

Kuzela DC, Huffer WE, Conger JD, Winter SD, Hammond WS. 1977. Soft tissue calcification in chronic dialysis patients. Am J Pathol. 86(2):403-24.

Lasser A. Calcification of the myocardium. 1983. Hum Pathol. 14(9):824-6.

Lee HU, Youn HJ, Shim BJ, et al. 2012. Porcelain heart: rapid progression of cardiac calcification in a patient with hemodialysis. J Cardiovasc Ultrasound. 20(4):193-6.

Li J, Chelala L, Hossain R, Jeudy J, White C. 2021. Rapid Onset Development of Myocardial Calcifications in the Setting of Renal Failure and Sepsis. Radiol Cardiothorac Imaging. 3(2):e200549.

Nance JJ, Crane GM, Halushka MK, Fishman EK, Zimmerman SL. 2015.Myocardial calcifications: pathophysiology, etiologies, differential diagnoses, and imaging findings. J Cardiovasc Comput Tomogr. 9(1):58-67.

Schellhammer F, Ansen S, Arnold G, Brochhagen HG, Lackner K. 2002. Myocardial calcification following septic shock. Cardiology. 98(1-2):102-3.

Wada A, Nakata T, Tsuchihashi K, et al. 1993. Massive myocardial calcification of right and left ventricles following acute myocarditis complicated with rhabdomyolysis-induced acute renal failure. Jpn Circ J. 57(6):567-72.

Zakouta R, Carmo Perloirob MD, Freitasc P. 2013. Reversible Myocardial Calcification Following Severe Leptospirosis Complicated with Rhabdomyolysis-Induced Acute Kidney Injury and Magnesium-Wasting Nephropathy. J Med Cases. 4(2):92-97.