Case Report

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Rapid Development of Diffuse Myocardial Calcification in a Patient after Recovery from Sepsis and Renal Failure: A Case Report

패혈증과 신부전에서 회복된 환자에서 급속하게 발생한 미만성 심근 석회화: 증례 보고

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Myocardial calcification can develop owing to several conditions. It is a rare complication following sepsis and renal failure. We report a case of rapid development of left ventricular mid-wall calcification observed using CT and cardiac MRI in a patient after recovery from sepsis and acute renal failure.

Index terms Magnetic Resonance Imaging; Computed Tomography, X-Ray; Sepsis; Renal Failure

INTRODUCTION

Myocardial calcification can arise from a number of conditions with focal myocardial calcification usually observed after myocardial infarction (1, 2). Diffuse myocardial calcification is generally rare. It is sometimes seen as a complication of sepsis and renal failure (3). We observed a case of rapidly developed diffuse and extensive deposition of left ventricular (LV)

mid wall calcification based on CT and cardiac MRI (CMRI) in a patient who recovered from sepsis and renal failure during six days of hospitalization.

CASE REPORT

A 51-year-old male patient with underlying liver cirrhosis complaining of general weakness was admitted to the emergency room. Clinically, he required inpatient treatment due to acute renal failure and septic condition. During five days of hospitalization, he continuously showed abnormal laboratory findings including elevated creatine (average: 5.10 mg/dL, normal range: 0.6–1.1 mg/dL), blood urea nitrogen (average: 79.3 mg/dL, normal range: 8–20 mg/dL), C-reactive protein (11.8 mg/dL), leukocytosis (32322/uL), hypocalcemia (average: 7.3 mg/dL, normal range 8.7–10.6 mg/dL), and hyperphosphatemia (average, 5.3 mg/dL, normal range: 2.5–4.7 mg/dL). *Enterobacteriaceae* and *Escherichia coli* were found on blood culture. He had a low blood pressure and a fever, suggesting sepsis and renal failure based on all available information. He also showed elevated troponin I (over 1000 pg/mL) and creatine kinase myocardial band (over 15 ng/mL) suggestive of heart injury. On the sixth day of hospitalization, a new diffuse and extensive calcific infiltration along the mid wall of LV was noted on covered pre-enhanced liver CT (Fig. 1A), which was not seen on chest CT scanned from outside the hospital one day before admission (Fig. 1B). On the seventh day of hospitalization, he underwent chest CT which showed the same finding of diffuse calcification involving only the LV wall with other newly noted ancillary findings of a small amount of bilateral pleural effusion. Cardiologists decided to perform transthoracic echocardiography (TTE) to evaluate cardiac function on the thirteenth day of hospitalization. The initial TTE presented diffuse infiltration echogenic materials in LV myocardium (Fig. 1C). The LV ejection fraction was preserved as 59%. Although mitral inflow Doppler showed impaired relaxation pattern, there was no significant evidence of diastolic dysfunction. We speculated that this rapid and extensively diffuse mid wall LV myocardial calcific deposition might be metastatic calcification relevant to the sequelae of recovery from sepsis and renal failure. Although LV global function and LV contractility were preserved, the patient was scheduled to undergo CMRI to rule out an unknown infiltrative process in the LV myocardium. Balanced short axis steady-state free precession images from CMRI revealed a preserved global LV function without regional LV wall motion abnormality. However, there were multiple, amorphous low signal intensity foci deposits in the mid-wall of LV. On rest perfusion images, there were also rim-like reduced perfusion areas in the LV mid-layer wall on the short axis view. On two-dimensional phase sensitive inversion recovery late enhancement images, there were diffuse basal to apical circumferential rim-like heterogeneous high signal intensity lesions along the mid-layer of the LV myocardium, sparing only the LV apex, in contrast with the normal null LV myocardium (Fig. 1D, E). Furthermore, the average native T1 relaxation time at the mid-LV was 1335 ms. There was a markedly increased average extracellular volume fraction of 54.8% (Fig. 1F, Supplementary Fig. 1 in the online-only Data Supplement). We believe that these high signal intensity areas suggest massive calcific deposits mixed with myocardial damage related to inflammatory edema or fibrosis in this patient with preserved LV function following recovery from sepsis and renal failure. In three months, follow-up TTE showed still pre-

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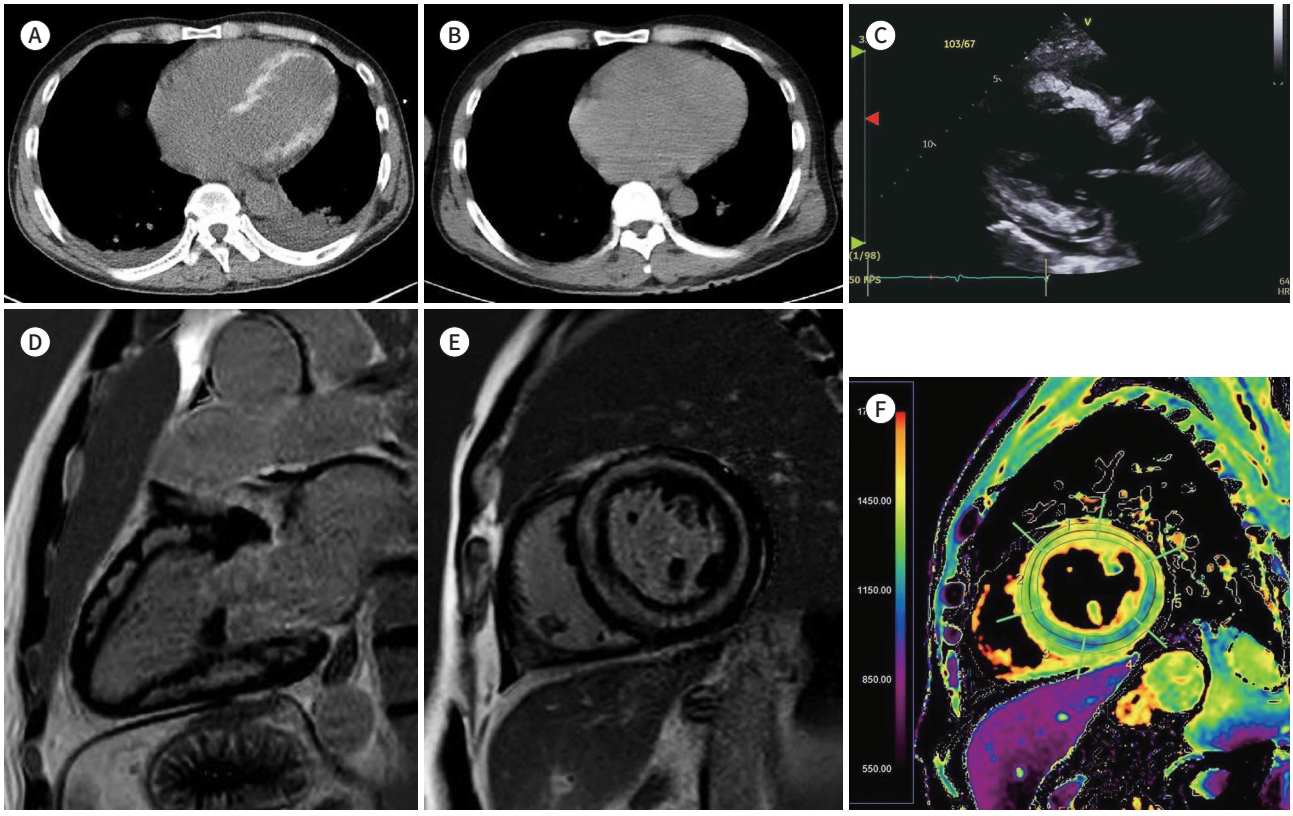
Fig. 1. Rapidly developed diffuse myocardial calcification in a 51-year-old male after recovery from sepsis and renal failure.
A. Diffuse and extensive calcific infiltration along the mid-layer wall of the LV is observed using covered pre-enhanced liver CT on day 6 post-admission.
B. Chest CT performed outside the hospital 1 day before admission does not show these findings.
C. Echocardiography reveals a diffuse echogenic calcification deposit in the mid-layer of the LV myocardium on day 13 post-admission. However, global systolic LV function (ejection fraction = 59%) and contractility were preserved.
D, E. Late gadolinium enhancement (viability) images of PSIR two chamber (D) and short axis (E) views demonstrate diffuse basal to apical circumferential, heterogeneous, and high signal intensity lesions, in contrast with the normal nulled LV myocardium along the mid-layer of the LV myocardium, sparing only the LV apex.
F. A mid-LV short-axis pre-contrast native T1 map shows increased T1 relaxation time (1335 ms) in the corresponding area in a two-dimensional PSIR short-axis image (E). A regionally decreased T1 relaxation time (1120 ms) area at the inferior wall of the LV (the blue area on the color map) is observed.
LV = left ventricle, PSIR = phase sensitive inversion recovery



served global LV function with normal LV contractility. There was no change of diffuse myocardial calcification in follow-up chest CT. The patient was scheduled for regular follow-up due to long term risk of future cardiomyopathy.

This case report was conducted in compliance with the Helsinki declaration.

DISCUSSION

We report a case of early onset, diffuse and extensive metastatic LV myocardial calcification associated with sequelae to sepsis and acute renal failure despite preserved global LV function and contractility based on CT, TTE, and CMRI. Regardless of the length of the preceding period of sepsis or renal failure, the dramatic acute infiltration of calcification in the

mid-layer LV wall in six days on follow-up CT scans, the unique tissue characterization of CMRI findings of native T1 relaxation time, and the markedly increased average extracellular volume fraction with preserved global LV function were notable.

The pathogenesis of myocardial calcification has not been well established yet. It is generally believed that two pathophysiologic mechanisms participate in myocardial calcification (2-4). With regard to metastatic calcification, calcium crystals that deposit in healthy myocardium are associated with disturbances of serum calcium homeostasis in patients with acute and chronic renal disease and hyperparathyroidism. In dystrophic calcification, the calcium typically deposits in necrotic myocardium in cases of sepsis or diffuse chronic myocardial infarction. Usually, serum calcium and phosphorous levels are normal or only slightly altered. Regardless of its mechanism, calcific deposition in myocardium is associated with gradually worsening prognosis attributed to disturbances of myocardial conduction and enhanced risk of arrhythmia, LV dilatation, and cardiomyopathy during follow-up (5). Our case of acute myocardial calcification might be due to a combined mechanism of metastatic and dystrophic calcification associated with serum calcium imbalance in acute renal failure with and possible myocardial damage from sepsis.

Possible differential causes for diffuse myocardial calcification have been reported in cases of sarcoidosis, rheumatoid arthritis, or chemotherapeutic agents. Thus, disease history or background and etiologies are needed (6-8).

Interestingly, in terms of T1 relaxation time and extracellular volume fraction, our case exhibited a very high extracellular volume fraction (54.8%). However, the native T1 relaxation time (1335 ms) was not so high at mid-LV, even in preserved LV function and contractility. Although we are not certain, we believe that these unmatched measurements from the unique tissue characterization of LV myocardium are attributable to the short T1 value of calcium mixed with myocardial edema as a complex pathophysiologic process (9).

Although our case has preserved cardiac function probably due to an acute stage of myocardial damage and still functionally compensatory range state of ventricular contractility, scheduled follow-up is necessary for monitoring gradual systolic LV dysfunction and scar-related arrhythmia.

In conclusion, we present a rare case of diffuse LV calcific infiltration after recovery from sepsis and renal failure with preserved LV function and contractility showing unique tissue characteristics on CMRI.

Supplementary Materials

The online-only Data Supplement is available with this article at http://doi.org/10.3348/jksr.2021.0181.

Author Contributions

Conceptualization, K.M.H., K.S.S.; data curation, K.M.H., K.S.S.; formal analysis, K.M.H., K.S.S; supervision, K.S.S.; validation, K.M.H., K.S.S.; writing—original draft, K.M.H., K.S.S.; and writing—review & editing, all authors.

Conflicts of Interest

The authors have no potential conflicts of interest to disclose.

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REFERENCES

1. Nance JW Jr, Crane GM, Halushka MK, Fishman EK, Zimmerman SL. Myocardial calcifications: pathophysiology, etiologies, differential diagnoses, and imaging findings. *J Cardiovasc Comput Tomogr* 2015;9:58-67
2. Torfs M, Salgado R, Van Herck P, Corthouts B, Robert D, Parizel PM. A curious case of acute myocardial calcifications. *Circulation* 2016;133:e426-e427
3. Gore I, Arons W. Calcification of the myocardium; a pathologic study of 13 cases. *Arch Pathol (Chic)* 1949;48:1-12
4. Maiese A, Manetti F, La Russa R, Di Fazio N, De Matteis A, Frati P, et al. Septic myocardial calcification: a case report. *J Forensic Leg Med* 2019;65:45-47
5. Kempf AE, Momeni MG, Saremi F. Myocardial calcinosis in chronic renal failure. *J Radiol Case Rep* 2009;3:16-19
6. Pejic M, Shifman M, Rose T, Jeong D. Chronic anthracycline-related myocarditis presenting as diffuse myocardial calcification. *J Clin Imaging Sci* 2019;9:47
7. Birnie DH, Nery PB, Ha AC, Beanlands RS. Cardiac sarcoidosis. *J Am Coll Cardiol* 2016;68:411-421
8. Mavrogeni S. Cardiac involvement in small and medium sized vasculitides. *Eur Soc Cardiol* 2007;6:14
9. Saba SG, Bandettini PW, Shanbhag SM, Spottiswoode BS, Kellman P, Arai AE. Characterization of cardiac masses with T1 mapping. *J Cardiovasc Magn Reson* 2015;17:Q32

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심근 석회화는 다양한 원인으로 인해서 발생할 수 있다. 하지만 패혈증과 신부전으로 인해서 발생하는 경우는 흔하지 않다. 저자들은 컴퓨터단층촬영과 자기공명영상에서 패혈증과 신부전을 회복한 환자에서 급속하게 좌심실벽에 미만성 석회화를 보이는 증례를 보고하고자 한다.

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