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Case Report

Successful surgical treatment of delayed left ventricular pseudoaneurysm related to *Candida albicans* infection after repair of ventricular septal rupture complicating acute myocardial infarction



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ABSTRACT

Left ventricular (LV) pseudoaneurysm is a rare complication after postinfarction repair of ventricular septal rupture (VSR), and surgical treatment of this condition due to mycosis has rarely been reported. We report a rare case of successful surgical treatment of delayed LV pseudoaneurysm related to *Candida albicans* infection after repair of VSR due to myocardial infarction. A 75-year-old woman was admitted for fever and severe inflammatory reaction. Two and a half years previously, she had undergone postinfarct VSR repair and was treated for mycotic infective endocarditis due to *C. albicans*. Transthoracic echocardiography and computed tomography revealed a LV pseudoaneurysm (maximum transverse diameter 6.2 cm). The cause of the LV pseudoaneurysm was suspected to be infectious, and broad-spectrum antibiotic treatment was started. Fourteen days after admission, she developed acute abdominal pain and an elevated β -D-glucan level because the LV pseudoaneurysm ruptured. Emergency surgical treatment was performed with antimycotic drug therapy. The LV wall defect was reconstructed using bovine pericardium under cardiopulmonary support. Her postoperative course was good, and she was discharged to home. Echocardiography revealed no recurrence of the LV pseudoaneurysm at 4 months postoperatively. During 1 year of follow-up, the patient had been doing well without any infection or adverse event.

<Learning objective: The development of a left ventricular (LV) pseudoaneurysm after repair of postinfarction ventricular septal rupture (VSR) is rare, and the cause of this condition is often unknown. Moreover, LV pseudoaneurysm due to mycosis has rarely been reported and is thought to have a poor prognosis. We report a rare case of successful surgical treatment of delayed LV pseudoaneurysm related to Candida albicans infection after repair of VSR complicating acute myocardial infarction.>

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Introduction

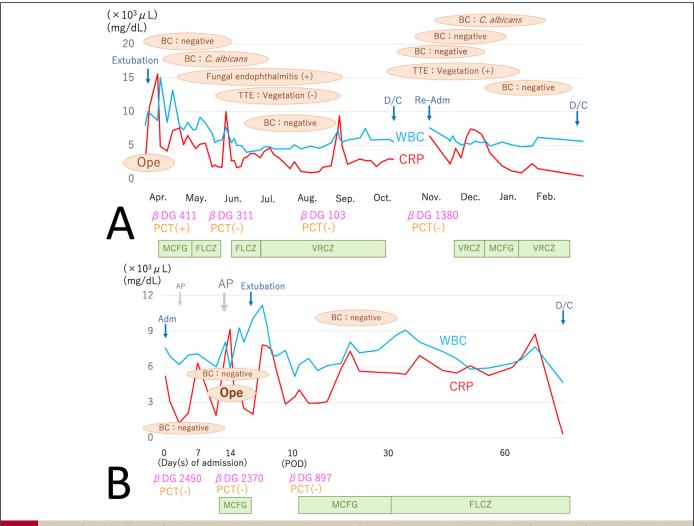
An acquired left ventricular (LV) pseudoaneurysm is a myocardial free-wall rupture contained by pericardial adhesion or the epicardial wall, and is caused by myocardial infarction, heart surgery, trauma, or infection [1, 2]. Cases of LV pseudoaneurysm after repair of postinfarction ventricular septal rupture (VSR) are rare, and the cause of this condition is often unknown. Moreover, LV pseudoaneurysms due to mycotic infection have rarely been reported and the prognosis might be poor [3]. We describe the successful surgi-

* Corresponding author. E-mail address: hitosuha@gmail.com (H. Suhara). cal treatment of a delayed LV pseudoaneurysm related to *Candida albicans* infection after repair of a VSR complicating acute myocardial infarction.

Case report

A 75-year-old woman was admitted to our hospital because she had a fever of 39.0°C. She had a history of osteoporosis, hypertension, dyslipidemia, and cervical spondylosis. Two and a half years previously, she had undergone an infarct exclusion operation via the LV approach and coronary artery bypass graft to the left circumflex artery, with a saphenous vein graft used to repair VSR that occurred because of acute myocardial infarction. After VSR repair,

Fig. 1.



History of *Candida albicans* infection in this patient. (A) Postoperative course after previous VSR repair and re-admission for mycotic infective endocarditis due to *C. albicans*. (B) Pre- and postoperative course of the present admission for pseudoaneurysm repair.

VSR, ventricular septal rupture; Ope, operation; BC, blood culture; TTE, transthoracic echocardiography; D/C, discharge; WBC, white blood cell count; CRP, C-

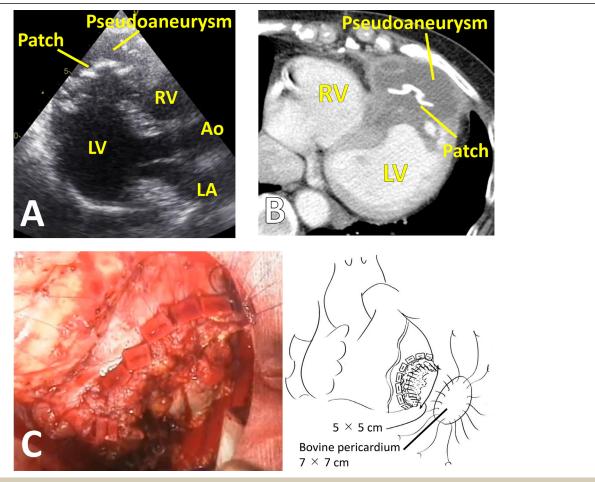
VSR, ventricular septal rupture; Ope, operation; BC, blood culture; TTE, transthoracic echocardiography; D/C, discharge; WBC, white blood cell count; CRP, C-reactive protein; BT, body temperature; β DG, β -D glucan (pg/ml); PCT, procalcitonin; MCFG, micafungin; FLCZ, fluconazole; VRCZ, voriconazole; Adm, admission; CT, computed tomography; AP, abdominal pain; POD, postoperative day.

the patient had developed candidemia and fungal endophthalmitis. *C. albicans* had been detected in the second blood culture. She was treated with micafungin (150 mg/day for 3 weeks), fluconazole (200 mg/day for 5 weeks), and voriconazole (400 mg/day for 6 weeks) (Fig. 1A). At 6 months after VSR repair, the patient had been re-admitted and diagnosed with fungal infective endocarditis with vegetations on the bovine pericardial patch due to *C. albicans* infection. She was administered voriconazole (400 mg/day for 10 weeks) and micafungin (150 mg/day for 4 weeks) (Fig. 1A). The infection had recurred twice after two discontinuations of the antifungal drugs. Fig. 1A shows the postoperative course after VSR repair and re-admission for mycotic infective endocarditis due to *C. albicans*. Fig. 1B shows the course of the present admission.

At the time of the present admission to our hospital for fever, the patient was no longer receiving antimycotic drug therapy. Laboratory data at this admission showed a marked inflammatory response (C-reactive protein 9.18 mg/dL) and a markedly elevated β -D-glucan level (2,450 pg/ml), but a normal white blood cell count (5,900/ μ L) and negative results for procalcitonin. The patient also had renal dysfunction (serum creatinine 1.25 mg/dL), but

no coagulopathy (platelet count 254 \times 104/ μ L, prothrombin time 100%). Electrocardiography showed a regular sinus rhythm with a heart rate of 64 beats/min, and chest radiography showed a cardiothoracic ratio of 57%, with no congestion or pleural effusion. Transthoracic echocardiography (TTE) and computed tomography (CT) revealed a LV apex pseudoaneurysm (Fig. 2A, B). TTE showed 'to and fro' blood flow between the LV cavity and the pseudoaneurysm. Echocardiography did not show any vegetation. Her cardiac function was relatively preserved, with an LV ejection fraction of 50%, and no significant valve dysfunctions. CT showed a large LV pseudoaneurysm with a maximum transverse diameter of 6.2 cm. The pseudoaneurysm formation was not seen on CT performed 1.5 years before the present hospitalization, or on TTE performed 2 days before this admission. The cause of the LV pseudoaneurysm was strongly suspected to be infection, and broad-spectrum antibiotic treatment was started.

At 14 days after hospital admission, the patient developed acute abdominal pain with re-elevation of the inflammatory response. CT showed enlargement of the LV pseudoaneurysm and a growing large hematoma in the epigastric space communicating with the LV pseudoaneurysm (Fig. 1B). We clinically diagnosed this patient



Preoperative transthoracic echocardiography and contrast-enhanced computed tomography, and intraoperative findings. (A) Preoperative transthoracic echocardiography and (B) contrast-enhanced computed tomography revealed a LV apex pseudoaneurysm outside of the intraventricular patch used in previous ventricular septal rupture repair. Computed tomography showed a large LV pseudoaneurysm (maximum transverse diameter 6.2 cm). (C) The LV wall defect (diameter 5 × 5 cm) was reconstructed using bovine pericardium (diameter 7 × 7 cm) and 16 pairs of 4-0 Nespilene mattress sutures with a felt pledget. LV, left ventricle; RV, right ventricle; LA, left atrium; Ao, aorta.

with LV pseudoaneurysm rupture related to *C. albicans* infection, in view of the markedly elevated β -D-glucan level and history of infective endocarditis due to *C. albicans*.

Emergency surgical treatment for LV pseudoaneurysm rupture was performed and antimycotic drug therapy (micafungin 150 mg/day) was initiated. Following median full re-sternotomy, cardiopulmonary bypass was routinely established via cannulation of the femoral artery and femoral vein. Part of the LV wall was dehisced with the felt strip used in the previous operation, and the intraventricular patch used in the VSR repair was completely exposed. Intraoperative examination revealed that an abscess had formed around the felt and the pseudoaneurysm. The infected felt strip and the dehisced LV wall were completely resected. The defect (diameter 5 \times 5 cm) was reconstructed using bovine pericardium (diameter 7 \times 7 cm) with 16 pairs of 4-0 Nespilene® (Alfresa Pharma Corporation, Osaka, Japan) mattress sutures with a felt pledget and 4-0 Nespilene® running suture (Fig. 2C). The culture samples collected intraoperatively from the felt, pseudoaneurysm wall, abdominal wall hematoma, and wound were all negative because of the antifungal drugs administered before surgery. Postoperatively, the β -D-glucan level decreased to 897 pg/ml. The antimycotics administered at the time of the pseudoaneurysm repair were 150 mg/day of micafungin for 4 weeks from the day before the operation, and 100 mg/day of oral fluconazole from when the patient was discharged to home at 2 months postoperatively (Fig. 1B). This was the third recurrence, and this recurrence was strongly suspected to be due to antimycotic discontinuation. Therefore, we recommended ongoing antifungal drugs for the patient's lifetime. During 1.5 years of follow-up, she has been doing well without any infection, recurrence, or adverse event.

Discussion

LV pseudoaneurysm is a result of myocardial free-wall rupture contained by pericardial adhesion, scar tissue, or the epicardial wall, while a true aneurysm affects all the layers of the cardiac wall. LV pseudoaneurysm is often caused by myocardial infarction, heart surgery, trauma, or infection [1, 2]. There are few reported cases of LV pseudoaneurysm arising from the LV suture line after postinfarction VSR repair [3–6] (Table 1), and the causes of this condition are often unknown.

In three previously reported cases, there was no evidence of infection and the cause of LV pseudoaneurysm after VSR repair was unknown [4–6]. There was only one case of mycotic LV pseudoaneurysm after VSR repair, and this patient died from sepsis [3]. Fungal infective endocarditis is rare, accounting for 1%–2% of all cases of infective endocarditis, but has a worse prognosis than bacterial infective endocarditis, with a surgical mortality rate of 30%–50%. A positive blood culture is only detected in about 50%–75% of patients with fungal infective endocarditis, which makes diag-

 Table 1

 Previous cases of left ventricular pseudoaneurysm arising from the left ventricular suture line after postinfarction ventricular septal rupture repair.

Author	Age·Sex	Primary MI	VSR location	Previous operation approach	Recurrence interval	Pseudoanurysmal site	Infection	Operation	End point
Ridley PD, et al. [4]	69·F	Inferior wall	Inferior	Inferior	3 years	Inferior	-	Direct suture	Home
Prêtre R, et al. [3]	72·M	-	-	Inferior	-	Inferior	Mycosis	Direct suture	Death, sepsis
Ghatak A, et al. [5]	74·F	Anterior wall	Apical	Apical	1.5 years	Anterior	-	Medication	Home Stable condition after 3 years
Song Y, et al. [6]	77·F	Anterior wall	-	Anterior	4 years	Apical	-	Patch plasty using bovine pericardium	Home Stable condition after 6 months
Current case	75·F	Anterior wall	Anterior	Anterior	2.5 years	Anterior	Mycosis	Patch plasty using bovine pericardium	Home Stable condition after one years

LV, left ventricle; VSR, ventricular septal rupture; MI, myocardial infarction.

nosis difficult [7]. Fungal infective endocarditis is difficult to control and surgery is recommended as the first-line treatment [8]. However, as the mortality rate reportedly does not differ between patients receiving surgical versus non-surgical treatment, clinicians must carefully select the best treatment method.

Our patient had a history of infective endocarditis due to *C. albicans* infection and had imaging changes consistent with endocarditis at the site of the cardiac prosthesis, with marked reelevation of the β -D-glucan level. For these reasons, despite the negative blood culture, we clinically diagnosed this patient with LV pseudoaneurysm rupture related to *C. albicans* infection in accordance with the Clinical Practice Guideline for the Management of Candidiasis: 2016 Update by the Infectious Diseases Society of America [9] and EORTC/MSG [10].

Although mycotic LV pseudoaneurysm after VSR repair may carry a poor prognosis, we described a rare case of successful surgical repair of LV fungal pseudoaneurysm rupture. Clinicians should closely monitor patients for signs of infection after VSR repair and should consider not only bacterial but also mycotic infection. Follow-up echocardiography and CT may be necessary after VSR repair, especially in patients with a history of fungal infections because of the possibility of pseudoaneurysm formation. If infection is suspected, it is important to initiate antibiotic and/or antimycotic therapy. The optimal timing of pseudoaneurysm repair surgery is as soon as possible after the diagnosis. However, if infection is present, the surgery should be performed after the infection has been controlled with antimycotic or antibacterial drugs.

Conclusion

LV pseudoaneurysm after previous repair of postinfarction VSR due to mycosis has rarely been reported and the prognosis might be poor. We report a rare case of successful surgical treatment of delayed LV pseudoaneurysm related to *C. albicans* infection after repair of VSR complicating acute myocardial infarction.

Declaration of Competing Interest

The authors declare that there is no conflict of interest.

Acknowledgments

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