Echocardiographic progression of a subepicardial aneurysm after inferior myocardial infarction

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Introduction

Subepicardial aneurysm is a rare complication of myocardial infarction. Subepicardial aneurysms of the left ventricle usually feature sudden interruption of the myocardium, a narrow neck, and a
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Case report

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On admission, he had a heart rate of 82 bpm
and a blood pressure of 111/67 mmHg. The 12-
lead electrocardiogram showed inverted T waves
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abnormal Q waves in leads III and aVf. Labora-
tory tests revealed that serum total cholesterol
was 210 mg/dl, triglycerides were 45 mg/dl, low-
density lipoprotein was 159 mg/dl, high-density
lipoprotein was 42 mg/dl, uric acid was 4.5 mg/dl,
and hemoglobin A1c was 7.1%. Transthoracic
echocardiography revealed akinesis of the infe-
rior wall of the left ventricle. The infarction area
remained in the myocardium (Figure 1A and B). We
diagnosed inferior acute myocardial infarction, and
emergency cardiac catheterization was performed.
Heparin was administered (100 U/kg). Emergency
coronary angiography (CAG) showed total occlusion
of the right coronary artery (RCA). We performed
percutaneous coronary intervention using a 7Fr JR4
guide catheter (Launcher®; Medtronic Ltd., Min-
esota, MN, USA) and guide wire (Runthrough®,
Terumo Co. Ltd., Tokyo, Japan) to cross the culprit
lesion in the RCA. We then performed stent-
ing of the culprit lesion. TIMI grade 2 flow was
achieved and the vessel wall distal to the lesion
was thin.

After that, his chest symptoms were resolved.
We administered nicorandil (0.5 mg) and heparin
(400 U/h) by continuous intravenous infusion. His
peak serum creatinine kinase level after coro-
nary intervention was 4088 IU/l. He was switched
to oral medication with aspirin (100 mg/day) and
ticlopidine (200 mg/day). After 7 days, chest
roentgenography showed slight cardiomegaly and
echocardiography revealed a moderate pericar-
dial effusion. However, there was no abnormal
flow from the left ventricle to the epicardium.
Echocardiography revealed almost the same view
as on admission, except pericardial effusion. He
had no symptoms and his condition was stable
with a heart rate of 92 bpm and a blood pres-
sure of 110/70 mmHg, so we administered diuretics
and observed him carefully. The pericardial effu-
sion gradually decreased in volume and his general
condition remained good. Follow up CAG revealed
patency of the stent and the patient was discharged
from hospital on March 26.

Four months later, two-dimensional echocardi-
ography revealed a large anechoic cavity behind the
inferior wall of the left ventricle that communi-
cated with the left ventricular lumen (Figure 1C).
The aneurysm of inferior wall of the left ventricle
was sudden interruption of the myocardium. Blood
flowed from the left ventricle into the cavity during
systole and flowed in the opposite direction during
diastole on color Doppler imaging. The maximum
diameter of the lesion was 28 mm. We concluded
that this was a pseudoaneurysm and recommended
urgent surgical resection. However, the patient had
no symptoms, so he rejected surgery.

After one month, echocardiography revealed
expansion of the aneurysm (Figure 1D), with the
maximal diameter increasing to 32 mm.

After two months, echocardiography revealed
rapid expansion of the aneurysm (Figure 1E). Its
maximal diameter was now 48 mm and the or-
ifice measured 22 mm. Multidetector row computed
tomography (MDCT) showed a huge aneurysm lying
below the inferior surface of the left ventricle
(Figure 2A and B). Blood flow from the left ventricle
into the cavity during systole and reverse flow dur-
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tomography (MDCT) showed a huge aneurysm lying
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(Figure 2A and B). Blood flow from the left ventricle
into the cavity during systole and reverse flow dur-
ing diastole was observed more clearly than that by
echocardiography. The walls of the aneurysm were
thin, and the myocardium showed sudden discon-
tinuity at the neck of the aneurysm. We feared
that the aneurysm would rupture or would cause
thrombosis or a ventricular arrhythmia, so we rec-
ommended immediate resection and the patient
agreed at this time.

On November 20, ventriculoplasty was per-
fected. There was a huge aneurysm on the
inferior surface of the left ventricle with epi-
Massive subepicardial aneurysm

Figure 1  (A) Echocardiography showed akinesis of the inferior wall of the left ventricle on admission. (B) The enlargement of the inferior wall. The infarction area remained in the myocardium. (C) At four months after discharge, echocardiography revealed an aneurysm behind the inferior wall of the left ventricle. The maximal diameter of the aneurysm was 28 mm (arrows). (D) After one month more, the aneurysm was larger and its maximal diameter was 32 mm (arrows). (E) After two months more, there was rapid expansion of the aneurysm and the maximal diameter was 48 mm (arrows).

cardium adherent to its walls. The aneurysm was excised and the defect was closed with a pericardial patch. Pathologic examination revealed the complete myocardium within the aneurysm wall and thrombus in the cavity (Figure 3). Postoperative echocardiography and MDCT showed that the aneurysm was no longer detectable and abnormal blood flow had disappeared. His postoperative course was uneventful.

Discussion

Mechanical complications of myocardial infarction are less frequently encountered in the current era of early revascularization [3]. However, these can still be catastrophic complications, especially ventricular free wall rupture.

Pathological examination is needed to distinguish a true ventricular aneurysm from a pseudoaneurysm after myocardial infarction. The former type has myocardium in its wall and the latter does not. Epstein and Hutchins first advocated the concept of subepicardial aneurysm [2]. A typical subepicardial aneurysm of the left ventricle arises at the site of sudden discontinuity of the myocardium, has a narrow neck, and shows a propensity to rupture spontaneously regardless of the mural components. It is diagnosed from morphologic findings. It is important to remember that subepicardial aneurysm can cause sudden death, even though this is a rare complication.
of myocardial infarction. Echocardiography is the method for diagnosing this type of aneurysm [4].

In our patient, a pericardial effusion was detected in the subacute phase of acute myocardial infarction. We suspected that this was due to oozing left ventricular rupture, but the patient’s condition was stable and echocardiography did not reveal abnormal flow from the left ventricle. After treatment with a diuretic and observation for several days, his pericardial effusion resolved. This suggested that partial rupture of the infarcted ventricular wall had occurred, which formed a subepicardial aneurysm at four months after his discharge from hospital. The aneurysm enlarged markedly after another three months of observation. This is the first report about serial findings of a subepicardial aneurysm observed by echocardiography.

There have been reports that hemorrhagic infarction caused by thrombolysis is associated with incomplete myocardial rupture [5]. Friedman et al. reported that early treatment with aspirin, heparin, and beta-blockers after myocardial infarction may limit infarct size, thereby reducing the risk of infarct expansion and aneurysm formation. On the other hand, steroids, non-steroidal anti-inflammatory drugs, and hypertension may promote aneurysm formation [6]. Our patient was administered aspirin and an angiotensin-converting enzyme inhibitor soon after diagnosis, but an aneurysm formed. He was referred to our hospital about 40 h after infarction occurred, so late revascularization may explain the development of a ventricular aneurysm. Dzavik et al. reported that late revascularization did not reduce infarct size or improve left ventricular function [7]. However, our patient had persistent chest symptoms, so we performed revascularization. Furthermore, we could not achieve TIMI 3 flow. Murakami et al. reported TIMI grade was an important factor for salvaging myocardium in patients with acute myocardial infarction [8]. So, it may be one of the reasons for forming subepicardial aneurysm.

Two-dimensional echocardiography is generally considered to be the technique of choice for diagnosing subepicardial aneurysm. Giltner et al. reported a case of subepicardial aneurysm diagnosed by computed tomography [9]. In our case, MDCT was useful for diagnosis and localization of the aneurysm.

Figure 2 (A) Multidetector row computed tomography shows a huge aneurysm behind the inferior wall of the left ventricle. Blood flows from the aneurysm into the left ventricle during diastole (arrow). (B) Blood flows from the left ventricle into the aneurysm during systole (arrow).

Figure 3 Pathologic examination revealed all of the myocardial elements in the aneurysm wall and thrombus in the lumen of the aneurysm (arrow).
In conclusion, the clinical significance of a subepicardial aneurysm is a high risk of rupture even in the chronic phase and regardless of its size. Accordingly, prophylactic surgical correction is necessary and is usually effective [10].

References