Surviving Case of a Blowout-Type Left Ventricular Free Wall Rupture During Percutaneous Coronary Intervention for a Lateral Acute Myocardial Infarction

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Summary
A 76-year-old man suffering from chest pain was admitted to our hospital with a suspected acute myocardial infarction (AMI). Emergent coronary angiography revealed a totally occluded proximal left circumflex artery (LCX). During primary percutaneous coronary intervention, his blood pressure suddenly fell within seconds, and he developed pulseless electrical activity (PEA). Surprisingly, the 12-lead electrocardiogram (ECG) findings including the heart rate remained unchanged before and after the PEA, but a heart rate reduction and asystole occurred a few minutes after developing PEA. After tracheal intubation and mechanical assistance by venoarterial extracorporeal membrane oxygenation (VA-ECMO), the sudden onset of PEA appeared to be caused by cardiac tamponade due to a blowout-type left ventricular free wall rupture (BO-LVFWR) diagnosed by transthoracic echocardiography. While pericardiocentesis was performed and the drained blood was directly continuously perfused intravenously to keep the VA-ECMO flow, the patient was moved to the operation room. The surgical findings revealed a solitary BO-LVFWR due to a lateral AMI, and a direct closure was performed. Successful perioperative management, oral medication administration, and rehabilitation lead to the patient being transferred to a rehabilitation hospital without any serious cerebral damage. This case report suggested the detailed onset pattern of a BO-LVFWR followed by a rapid diagnosis by echocardiography and lifesaving treatment.

Key words: Transthoracic echocardiography, Mechanical circulatory support, Continuous vital sign monitoring

A blowout-type left ventricular free wall rupture (BO-LVFWR) is a fatal complication of an acute myocardial infarction (AMI). Treatment of this complication remains challenging due to the limited time to perform further lifesaving therapeutic interventions following the onset of the BO-LVFWR. We herein experienced a case of a BO-LVFWR occurring during percutaneous coronary intervention (PCI) for an AMI, which could be diagnosed and treated early and eventually discharged without any serious cerebral damage.

Case Report
A 76-year-old man with hypertension experienced a sudden onset of chest pain. The chest pain persisted, and he was transferred to our hospital 6 hours after the onset. There were no signs of shock according to his vital signs and physical findings during the initial examination (Table I). A synthesized 18-lead electrocardiogram (ECG) during the initial examination revealed complete right bundle branch block with an elevated ST segment in leads I and aVL and ST depression in leads III and aVF. Transthoracic echocardiography revealed a preserved global left ventricular function with slight hypokinesis due to a posterolateral lesion without a D-shape, echo-free space, or valvular disease. A chest X-ray revealed no notable findings, including congestion. The laboratory data showed increased creatine kinase (CK) and troponin I serum levels, which were checked during catheter examination. An AMI was diagnosed, and an emergent coronary angiography via the left radial artery revealed no significant stenosis in the right coronary artery, but a total occlusion in the proximal left circumflex artery (LCX), which coincided with the culprit inferred from the 18-lead ECG and echocardiography (Figure 1). We immediately moved to a primary PCI of the proximal LCX. First, we tried to wire the obstructed lesion in the LCX with a floppy wire (SION blue guide wire, Asahi Intecc Co., Ltd., Aichi, Japan); however, it did not pass due to the tortuosity and insufficient backup and penetration. Subsequently, wiring was attempted with a taper wire (XT-A guide wire, Asahi Intecc) under the support of a micro-catheter (Caravel MC...
proximal left circumflex coronary artery distal to the left main trunk南昌Boston Scientific Corp.) imaging from the left anterior de-
ultrasonography (IVUS) (Opticross, Boston Scientific, wiring, but no extravasation was observed. Intravascular
was performed to rule out any vascular perforation due to
echocardiography was ready, a left coronary angiogram
den onset of PEA was evaluated. Until the transthoracic
ECMO and right ventricular pacing, the cause of the sud-
by tracheal intubation and mechanical assistance by V A-
formed at 80 bpm. After establishing respiratory support
femoral approach, and right ventricular pacing was per-
was immediately placed in the right ventricular apex via a left
micro-catheter, Asahi Intecc). However, his blood pressure
suddenly fell within seconds while attempting to wire the
obstructed lesion (Figure 2). When no cardiac output was
present, cardiopulmonary resuscitation was started with a
rapid response system. Rapid tracheal intubation by an
emergency physician was performed, and venoarterial extracorporeal membrane oxygenation (VA-ECMO) via a
right femoral approach was simultaneously established. The
time from the cardiopulmonary arrest (CPA) to the
VA-ECMO drive was 17 minutes (Table II). Surprisingly,
the 12-lead ECG findings including the heart rate before
and after the PEA remained unchanged (Figure 3), but a
heart rate reduction and asystole occurred a few minutes
after developing PEA. A temporary pacing catheter was
immediately placed in the right ventricular apex via a left
femoral approach, and right ventricular pacing was per-
formed at 80 bpm. After establishing respiratory support
by tracheal intubation and mechanical assistance by VA-
ECMO and right ventricular pacing, the cause of the sud-
den onset of PEA was evaluated. Until the transthoracic
echocardiography was ready, a left coronary angiogram
was performed to rule out any vascular perforation due to
wiring, but no extravasation was observed. Intravascular
ultrasonography (IVUS) (Opticross, Boston Scientific, Boston Scientific Corp.) imaging from the left anterior de-
sending coronary artery distal to the left main trunk
ostium was performed to rule out a coronary artery dis-
section induced by the PCI procedure, but no significant
dissection was found. On echocardiography, it was judged
that the PEA was caused by cardiac tamponade because
there was a pericardial effusion that was not recognized in
the initial examination; thus, a pericardiocentesis was per-
formed from an apex approach. The pericardial effusion
was bloody (hemoglobin level was 12.0 g/dL in a blood
gas analysis in later), and the systemic blood pressure in-
creased temporarily after drainage, but as soon as the
drainage was stopped, the pericardial effusion reappeared,
and the blood pressure decreased. When the amount of
drainage exceeded 300 cc, it was judged that the cardiac
tamponade was not caused by a coronary artery injury due
to the PCI procedure but was due to a BO-LVFWR that
complicated after the AMI, and we consulted a cardiovas-
cular surgeon. An intra-aortic balloon pump (IABP) was
placed via a left femoral approach, and a central venous
catheter was placed from the right internal jugular vein.
Up until the operation was ready, because blood transfu-
sions were not rapidly available, in addition to the rapid
infusion of extracellular fluid, a VA-ECMO flow was
maintained by returning the drained blood directly from
the central venous catheter placed in the right internal
jugular vein. Eventually, the patient was able to be moved
to the operation room while maintaining the VA-ECMO
flow.

The surgical findings revealed a solitary BO-LVFWR
due to a lateral AMI (Figure 4). There was a 2-cm rupture
hole in the peripheral myocardium of the LCX. A direct
closure was performed at the same site, and a central
ECMO was attached to complete the procedure. The op-
eration time was 244 minutes (Table II). After that, the
CK level peaked out at 2895 IU/L, and the myocardium
stunned after the CPA and gradually improved day by
day, and it was possible to withdraw him from the VA-
ECMO 6 days after the surgery and the IABP 7 days after
the surgery. Regarding the neurological course, convul-
sions were observed on the day after the operation, and a
consultation was given by a neurologist. Based on the
neurological examination, head computed tomography
scan, and electroencephalogram findings, the patient was
diagnosed with convulsions due to hypoxic encephalopa-
thy and was treated with antiepileptic drugs. Considering
the need for long-term artificial respiration management
for the prolongation of a consciousness disorder due to
convulsions, a tracheostomy was performed on the 19th
postoperative day. After that, the convulsions disappeared,
and the disturbance of consciousness improved until com-
munication was achieved. Eventually, oral administration,
including a beta-blocker and angiotensin II receptor blocker (ARB), and rehabilitation could be performed, and the patient was then transferred to a rehabilitation hospital on the 35th postoperative day for the purpose of continued rehabilitation.

**Discussion**

LVFWRs are clinically classified as either an oozing type or a blowout type. The oozing type is characterized by a perforation, allowing enough time for a diagnosis and surgery, while the blowout type is characterized by rapid, irreversible, electromechanical dissociation, shock, and death within a few minutes due to massive hemorrhages into the pericardial cavity. According to the previous reports regarding LVFWRs, most cases are the oozing type, and blowout-type cases are relatively rare. Although the blowout type is a rare but fatal complication of an AMI leading to sudden and devastating death, the clinical data for its characteristics and course are lacking. More-
Figure 3. Twelve-lead electrocardiograms before and after pulseless electrical activity (PEA). Note that there are no significant changes in the heart rate or 12-lead electrocardiograms before (left figure) and after (right figure) the PEA. A few minutes after a sudden drop in the blood pressure, a heart rate reduction, and asystole occur.

Figure 4. Intraoperative findings of a 2-cm rupture hole blowing blood out into the peripheral myocardium of the left circumflex coronary artery (arrow in the left figure) and a direct closure at the same site (arrow in the right figure).

Moreover, as even case reports are limited in the previous studies about BO-LVFWRs, there has been a lack of consensus on a rapid diagnosis and treatment strategy. We experienced a rare case of a BO-LVFWR successfully treated without any serious cerebral damage despite its occurrence during a PCI for an AMI.

Clinical studies have reported that the frequency of LVFWRs is 1-4% of patients after an AMI, but it accounts for 10-20% of patient deaths.3,4) LVFWRs occur within the first 5 days after an AMI in about half of the cases and within 2 weeks in over 90% of patients.5,7) Nakatsuhi Y et al. divided LVFWRs into an early phase (< 72 hours) and late phase (> 96 hours).5) The risk factors for an LVFWR were an older age (> 65 years), female gender, hypertension, one-vessel disease (usually a totally occluded vessel with poor collateral circulation), first AMI, absence of reperfusion therapy, and delayed admission.5) LVFWRs occur mainly in the left ventricle with a fairly even distribution between the anterior, inferior, and lateral walls.5) In the present case, it occurred in the early phase, and the risk factors for an LVFWR included hypertension, and one-vessel disease with a total occlusion and poor collateral circulation.

In this case, his life was saved without any serious cerebral damage through a prompt diagnosis by echocardiography and appropriate treatment, including the insertion of mechanical support and pericardiocentesis, leading to maintaining the VA-ECMO flow until the operation.
Transthoracic echocardiography is the most important diagnostic tool for LVFWRs, with a sensitivity and specificity of the diagnosis between 93% and 98%. The detailed onset pattern of the BO-LVFWR could be observed because it occurred during the PCI. That is, the blood pressure suddenly decreased during the PEA within seconds, i.e., electromechanical dissociation, in spite of having no significant change in the 12-lead ECG or heart rate. The pulse slowed down, and asystole developed a few minutes after the sudden drop in the blood pressure. These findings provided the clinical importance of continuous blood pressure monitoring in AMI patients with the risk of an LVFWR, because it would be too late to respond after the pulse had slowed. It was considered that an immediate confirmation of a pericardial effusion by echocardiography is the most important diagnostic method for a BO-LVFWR in this situation. Furthermore, the prompt use of mechanical support (VA-ECMO and IABP) is needed. In such a situation, maintaining the VA-ECMO flow requires the constant release of the cardiac tamponade and securing of the blood vessel volume. In this case, because blood transfusions were not in time, the bloody epicardial fluid from the pericardial drain was administered intravenously to maintain the VA-ECMO flow, and it managed to maintain the circulation until the surgery. Although the pericardial fluid drainage was returned directly to the vein, there were few significant demerits, and it helped maintain the VA-ECMO flow.

An increased PCI rate, better control of the blood pressure, and use of beta-blockers and ACE inhibitors (ACEI)/ARB have contributed to a lower incidence and death rate from LVFWRs. The utilization of beta-blockers and ACEIs/ARBs in the AMI treatment strategy is correlated with a higher in-hospital survival rate from LVFWRs, especially in LVFWR cases that occur in the late phase. In this case, the uncontrolled high blood pressure during the PCI might have contributed to the BO-LVFWR, and the careful introduction of the beta-blockers and ARBs during the perioperative period was performed, even though this BO-LVFWR occurred in the early phase. Additionally, it was unclear whether those medications could have decreased his mortality as in the late phase.

Conclusion

Here, we experienced a surviving case from a BO-LVFWR occurring during a PCI for an AMI. The present case initially had a sudden blood pressure drop within seconds without any change in the heart rate or 12-lead ECG when the BO-LVFWR occurred. After a few minutes, a heart rate reduction and asystole occurred. These findings suggested that continuous vital sign monitoring could be useful for an early detection of a BO-LVFWR and that an early diagnosis by echocardiography and maintaining the circulation by VA-ECMO until the operation could be lifesaving.

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Disclosure

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References