Intraprocedural left ventricular free wall rupture diagnosed by left ventriculogram in a patient with infero-posterior myocardial infarction and severe aortic stenosis

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Abstract

**Background:** Left ventricular wall rupture remains a major lethal complication of acute myocardial infarction and hypertension is a well-known predisposing factor of cardiac rupture after myocardial infarction.

**Case presentation:** An 87-year-old man was admitted to our hospital, diagnosed as acute myocardial infarction (AMI). The echocardiogram showed a 0.67-cm² aortic valve, consistent with severe aortic stenosis (AS). A coronary angiography showed a chronic occlusion of the proximal left circumflex artery and a 99% stenosis and thrombus in the mid right coronary artery. During percutaneous angioplasty of the latter, transient hypotension and bradycardia developed at the time of balloon inflation, and low doses of noradrenaline and etilefrine were intravenously administered as needed. The patient suddenly lost consciousness and developed electro-mechanical dissociation. Cardio-pulmonary resuscitation followed by insertion of an intra-aortic balloon pump (IABP) and percutaneous cardiopulmonary support were initiated. The echocardiogram revealed moderate pericardial effusion, though the site of free wall rupture was not distinctly visible. A left ventriculogram clearly showed an infero-posterior apical wall rupture. Surgical treatment was withheld because of the interim development of brain death.

**Conclusions:** In this patient, who presented with severe AS, the administration of catecholamine to stabilize the blood pressure probably increased the intraventricular pressures considerably despite apparently normal measurements of the central aortic pressure. IABP, temporary pacemaker, or both are recommended instead of intravenous catecholamines for patients with AMI complicated with significant AS to stabilize hemodynamic function during angioplasty.

**Keywords:** Left ventricular wall rupture, Acute myocardial infarction, Aortic stenosis, Angioplasty, Intra-aortic balloon pump, Catecholamine
Background
Left ventricular wall rupture remains a major lethal complication of acute myocardial infarction despite the recent progress in primary percutaneous coronary intervention era. The incidence of cardiac rupture has been reported to be 1.7–4.8 % of patients with acute myocardial infarction (AMI) [1–3]. Hypertension is a well-known predisposing factor of cardiac rupture after myocardial infarction [1, 4, 5]. Although a few reports have described the intraventricular hypertension associated with the increased risk of myocardial rupture in the presence of significant aortic stenosis (AS), [6–8] none were able to perform left ventriculography during percutaneous angioplasty for a patient with AMI and AS complicated by cardiac rupture because of its emergency and difficulties. We discuss this case in the context of a) the usefulness of left ventriculogram for identifying the site of left ventricular wall rupture, and b) a problem pertaining to catecholamine use for a patient presenting with acute myocardial infarction and significant aortic stenosis.

Case presentation
An 87-year-old man with histories of severe AS and diabetes mellitus was admitted to our hospital complaining of cold sweat and chest pain of 4 h duration. His heart rate was 98 bpm, blood pressure (BP) 94/57 mmHg and oxygen saturation on room air 98 %. A systolic murmur in the aortic area and a 3rd heart sound were audible on auscultation. The 12-lead electrocardiogram (Fig. 1) showed 1st degree atrioventricular block, complete right bundle branch block and ST segment and T wave changes consistent with an infero-posterior AMI. The white blood cell count was 13.4 × 10^3/μl, platelet count 16.9 × 10^4/μl, hemoglobin concentration 9.5 g/dl.
troponin-T 1,614 ng/l, creatine and creatine-MB concentrations 681 and 38.9 IU/l, respectively, and the serum C-reactive protein concentration 1.30 mg/dl. A transthoracic echocardiogram showed a severely hypokinetic infero-posterior left ventricular (LV) wall, a 40 % LV ejection fraction without pericardial effusion, and a severely limited opening of the aortic valve (Fig. 2a, b). A coronary angiography performed in emergency showed a chronic occlusion of the proximal left circumflex artery and a 99 % stenosis and thrombus in the mid right coronary artery (Fig. 3a, b). During percutaneous angioplasty of the latter, hypotension and bradycardia developed at the time of balloon inflation, and etilefrine and noradrenaline were administered in order to keep optimal blood pressure and heart rate.

1 ml (once) and 2 ml (twice) intravenous boluses of a) 1 mg of etilefrine, and followed by 2 ml (4 times) of b) 1 mg of noradrenaline, each diluted in 19 ml of saline, were administered during the procedure. The aortic blood pressure rose to 120–130/60–70 mmHg during the procedure, when the patient suddenly lost consciousness and developed electro-mechanical dissociation. Cardio-pulmonary resuscitation followed by insertion of an intra-aortic balloon pump (IABP) and percutaneous cardiopulmonary support were initiated. A repeat transthoracic echocardiogram revealed the presence of a moderate pericardial effusion, though the site of free wall rupture was not distinctly visible (Fig. 4a, b). Despite an immediate pericardiocentesis followed by continuous drainage, the effusion accumulated rapidly. While waiting for an operation of cardiac surgery, we performed a left ventriculography to detect the site of the LV wall rupture, using ACIST® (ACIST Medical Systems, Eden Prairie, Minnesota, United States).
Contrast agent was injected by 10 ml/s with the total amount of 20 ml. A left ventriculogram in right and left anterior oblique views clearly showed the presence of an infero-posterior apical wall rupture (Fig. 5a, b; Additional file 1: movies 1 and 2). The final coronary angiogram showed no rupture or wire perforation of the right coronary artery. The patient was transferred to the operating room. However, surgical treatment was withheld because of the interim development of brain death.

Discussion
This is a rare case of intraprocedural blowout type of LV wall rupture diagnosed by left ventriculogram after an infero-posterior AMI in a patient with severe AS. Hyper-tension is one of the well-known risk factors for LV wall rupture after AMI [1, 4, 5]. In this patient, who presented with severe AS, the administration of noradrena-line and etilefrine to stabilize the BP probably increased the intraventricular pressures considerably despite apparently normal measurements of the central aortic pressure. Catecholamines increase myocardial contractil-ity and cause peripheral vasoconstriction, both of which increase the left intraventricular pressure. On the other hand, IABP counterpulsation lowers the systolic pressure by unloading the left ventricle, and increase the mean arterial BP by diastolic augmentation [9–11]. Diastolic augmentation does not increase the left intraventricular pressure because, in absence of aortic regurgitation, the aortic valves are closed during diastole.

This case highlights the risk of intraprocedural LV wall rupture in patients presenting with AMI and severe AS by, a) increasing the intraventricular pressures, and b) attempting to preserve a normal aortic BP by the administration of catecholamines. It is also noteworthy that the use of high-speed injectors for left ventriculograms must be avoided because, by increasing the intraventricular pressure, they may rupture the left ventricle. IABP, a temporary pacemaker, or both are recommended instead of intravenous catecholamines to stabilize hemodynamic function during percutaneous coronary interventions. In this case, the insertion of an IABP and the implant of a temporary pacemaker should have been considered before proceeding with angioplasty since, in patients presenting with inferior AMI, ST segment elevation in lead aVR is not only a manifestation of basal septal infarction, but also of diffuse non-transmural apical and lateral wall ischemia from multiple vessels disease, and a sign of poor prognosis [12].

Conclusion
Left ventriculography was useful for identifying the site of LV wall rupture after AMI complicated with AS. For such patients, IABP, a temporary pacemaker, or both are recommended instead of intravenous catecholamines to stabilize hemodynamic function during percutaneous angioplasty.

Additional file
Additional file 1: Movies 1 and 2 of left ventriculography were showed.

(Accessed file 7571 kb)

Abbreviations
AMI, acute myocardial infarction; AS, aortic stenosis; BP, blood pressure; IABP, intra-aortic balloon pump; LV, left ventricular

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Authors’ contributions
TK drafted the manuscript and performed the angioplasty procedure. NF, TY, HN, DH, KK, HY and KO critically revised the manuscript for important intellectual content. All authors read and approved the final manuscript.

Competing interests
The authors declare that they have no competing interests.

Consent for publication
Written informed consent was obtained from the patient’s family for publication of this case report and any accompanying images.

Ethics approval and consent to participate
Not applicable.

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References
1. Honda S, Asaumi Y, Yamane T, Nagai T, Miyagi T, Noguchi T, et al. Trends in the clinical and pathological characteristics of cardiac rupture in patients with acute myocardial infarction over 35 years. J Am Heart Assoc. 2014;3(5):e000984.
2. Figueras J, Alcalde O, Barabes JA, Serra V, Alguersuari J, Cortadellas J, et al. Changes in hospital mortality rates in 425 patients with acute ST-elevation myocardial infarction and cardiac rupture over a 30-year period. Circulation. 2008;118(25):2783–9.
3. Sutherland FW, Guell FJ, Pathi VL, Naik SK. Postinfarction ventricular free wall rupture: strategies for diagnosis and treatment. Ann Thorac Surg. 1996;61(4):1281–5.
4. Naeim F, De la Maza LM, Robbins SL. Cardiac rupture during myocardial infarction. A review of 44 cases. Circulation. 1972;45(6):1231–9.
5. Yip HK, Wu CJ, Chang HW, Wang CP, Cheng CJ, Chua S, et al. Cardiac rupture complicating acute myocardial infarction in the direct percutaneous coronary intervention reperfusion era. Chest. 2003;124(2):565–71.
6. Tanaka M, Goto Y, Suzuki S, Mori I, Ohtsuka Y, Miyazaki S, et al. Postinfarction cardiac rupture despite immediate reperfusion therapy in a patient with severe aortic valve stenosis. Heart Vessels. 2006;21(1):59–62.
7. Kadri MA, Kakadellis J, Campbell CS. Survival after postinfarction cardiac rupture in severe aortic valve stenosis. Eur Heart J. 1994;15(1):140–2.
8. Duke M. Aortic stenosis, myocardial infarction and cardiac rupture: an unusual triad. Tex Heart Inst J. 1984;11(1):96–7.
9. Stone GW, Ohman EM, Miller MF, Joseph DL, Christenson JT, Cohen M, et al. Contemporary utilization and outcomes of intra-aortic balloon counterpulsation in acute myocardial infarction: the benchmark registry. J Am Coll Cardiol. 2003;41(11):1940–5.
10. Scheidt S, Wilner G, Mueller H, Summers D, Lesch M, Wolff G, et al. Intra-aortic balloon counterpulsation in cardiogenic shock: report of a cooperative clinical trial. N Engl J Med. 1973;288(19):979–84.
11. Kern MJ, Aguine F, Bach R, Donohue T, Siegel R, Segal J. Augmentation of coronary blood flow by intra-aortic balloon pumping in patients after coronary angioplasty. Circulation. 1993;87(2):500–11.
12. Wong CK, Gao W, Stewart RA, Benatar J, French JK, Aylward PE, et al. aVR ST elevation: an important but neglected sign in ST elevation acute myocardial infarction. Eur Heart J. 2010;31(15):1845–53.