

Two Cases of Acute Myocardial Infarction in Patients with Severe Aortic Stenosis and Normal Coronary Arteries

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Manuscript submitted: June 29, 2019; Revised and accepted: August 24, 2019

ABSTRACT

In patients with aortic stenosis (AS), the development of left ventricular systolic dysfunction and heart failure predicts poor prognosis. Myocardial ischemia, particularly of the circumferential sub endocardial region, commonly occurs in patients with severe AS during hemodynamic stress, even in the setting of angiographically documented normal coronary arteries. We report two case patients who experienced of ischemic chest pain with ST-changes and undergoing a coronary angiography but we found normo coronary arteries and echocardiography with nomokinetic. These cases highlight the importance of the correlating between history taking, physical examination and other supporting examination, especially focused on bedside investigation like echocardiography in the management of patients presenting with chest pain.

Keywords: aortic stenosis; acute myocardial infarction

INTISARI

Pada pasien dengan stenosis aorta (AS), perkembangan disfungsi sistolik ventrikel kiri dan gagal jantung memprediksi prognosis yang buruk. Iskemia miokard, terutama dari daerah subendokardial sirkumferensial, biasanya terjadi pada pasien dengan AS berat selama stres hemodinamik, bahkan saat didapatkan arteri koroner yang normal secara angiografi. Kami melaporkan dua pasien kasus yang mengalami nyeri dada khas iskemik dengan perubahan segmen ST dan menjalani angiografi koroner tetapi kami menemukan arteri koroner yang normal dan ekokardiografi dengan nomokinetik. Kasus ini menyoroti pentingnya korelasi antara anamnesis, pemeriksaanfisik, dan pemeriksaan penunjang lainnya, terutama berfokus pada pemeriksaan klinik seperti ekokardiografi dalam manajemen pasien yang mengalami nyeri dada.

INTRODUCTION

In patients with aortic stenosis (AS), the development of left ventricular systolic dysfunction and heart failure predicts poor

prognosis, including a less favorable outcome after valve replacement, the onset of heart failure is preceded by structural and functional alterations in the heart muscle with left ventricular hypertrophy followed by

degeneration and death of the cardiac myocytes.¹ Myocardial ischemia, particularly of the circumferential sub endocardial region, commonly occurs in patients with severe AS during hemodynamic stress, even in the setting of angiographically documented normal coronary arteries.² Serum cardiac troponins I is a heart specific contraction regulating proteins released into the circulation from injured myocytes, Severe AS has been reported to cause elevations in cardiac biomarkers such as troponin I.¹

However, examination of the coronary arteries should be performed to definitely rule out transient coronary artery obstruction as a causative factor. We report a case of acute myocardial infarction in a patient with severe AS and normal coronary arteries documented a day later after admission.

CASE PRESENTATION

Case 1

Severe AS was suspected in a 52-year-old male who was referred to our hospital with typical chest pain. He had a 1-month history of chest pain and shortness of breath during exertion. Two days before admission, he had experienced these

symptoms while farming his fields. On admission, Physical examination revealed a systemic blood pressure of 133/90 mmHg on dobutamine support and 96/67 after dobutamine is stop, his pulse rate of 124 beats/min. On auscultation, a grade IV/VI systolic ejection murmur was heard best at the upper right sternal border. Laboratory work revealed a troponin I rise from an initial level of >40.000 mg/ml within >24 hours presentation of chest pain. Anelectrogram (ECG) showed high voltage in the left ventricle and ST-segment elevation in leads aVR, V1-V3, concomitant with marked ST-segment depression in leads V4–V6 (Figure 1).

A chest X- ray disclosed an enlarged cardiac with a cardiothoracic ratio of 55% and pulmonary vascular congestion (Figure 2). The classic physical exam findings of aortic stenosis lead us to an urgent echocardiography, rather than an early invasive approach of cardiac catheterization.

Echocardiography shows (Figure3) an ejection fraction about 61% and calcific aortic valve stenosis with aortic valve area 0.6 mm and concentric left ventricular hypertrophy were found. Elective coronary angiography done a day later revealed normal coronaries (Figure 4).

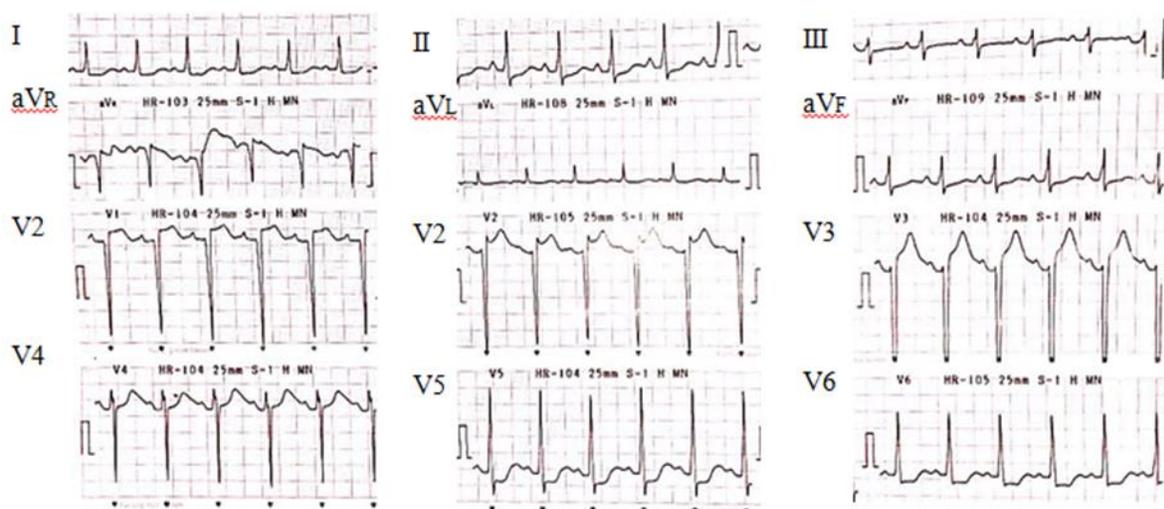


Figure 1. ECG on admission shows high voltage in the left ventricle and ST-segment elevation in leads aVR, V1- V3, concomitant with marked ST-segment depression in leads V4–V6.

Case 2

A 65-year-old male was referred to our hospital with Non ST elevation myocardial infarction and cardiogenic shock. He had a 2-month history of chest pain during exertion. 11 days before admission, he felt typical chest pain followed with dizziness while playing with his grandson and was brought to district hospital and diagnosed with Non ST elevation myocardial infarction. Three days before he was referred, this patient had a low systolic pressure about 80 mmHg and he was assessed with cardiogenic shock and treated with 8 mcg/kg/minute of dopamine and then he was referred to our hospital. On admission, physical examination revealed a systemic blood pressure of 100/50 mmHg, his pulse rate of 85 beats/min. On auscultation, a grade III/VI systolic ejection murmur was heard best at the upper right sternal border. Laboratory

work revealed a troponin I rise from an initial level of 8055.90 mg/mL. An electrogram (ECG) from previous hospital showed high voltage in the left ventricle and ST-segment depression in leads I, II, III, aVF and V2-V6 (Figure 5), but the ECG at our hospital showed left ventricle hypertrophy with changes of ST depression in leads I, aVL, V3-V6 and also at the posterior leads (Figure 6).

A chest X-ray disclosed an abnormal cardiac thoracic ratio and pulmonary vascular congestion (Figure 7). Patient was planned to echocardiography first than an early invasive approach of cardiac catheterization. Echocardiography shows (Figure 8) an ejection fraction is 54% and calcific aortic valve stenosis with aortic valve area 0.4 mm, AV Vmax 4.38 m/s, AV mean PG 41 mmHg and concentric left ventricular hypertrophy were found. Elective coronary angiography done and the result is normal coronaries (Figure 9).



Figure 2. Chest X-ray on admission shows cardiomegaly and lungs congestion.



Figure 3. Short-axis views show the aortic valve area was 0.6 cm^2 , which is suggestive of severe aortic stenosis

DISCUSSION

Patients with severe AS may be asymptomatic for many years despite severe obstruction, but once symptoms, such as angina, heart failure and syncope, manifest in such patients, the prognosis is poor. Myocardial ischemia, particularly of the left ventricular sub endocardium, commonly occurs in cases of severe aortic stenosis during hemodynamic stress, even in the setting of coronary arteries documented angiographically as normal.³

Angina episodes are reported by about half of the patients with a severe degree of aortic stenosis, and this occurs despite the evidence of normal epicardial coronary arteries at angiography.⁴ Angina may be caused by mismatched demand and supply of myocardial oxygen, particularly in response to stress. Besides mismatched demand and supply of myocardial oxygen, development of left ventricular hypertrophy (LVH) in patients with aortic valve stenosis is an adaptive response that attempts to reduce wall stress in the left ventricle, the

development of LVH also affects the coronary circulation, it may result in a reduction in coronary flow reserve (CFR), which is the ratio of maximal to basal coronary blood flow.⁵ Coronary flow reserve is reduced in aortic stenosis due to a combination of mechanisms, including: (i) reduced time of diastolic coronary filling; (ii) increased LV diastolic filling pressure and intra myocardial pressure during diastole, both contributing to impairment of perfusion selectively in the sub endocardium; (iii) reduced capillary density; (iv) a low coronary perfusion pressure when compared with intra cavitory pressure; and (v) increased intra myocardial systolic pressure and delay in myocardial relaxation at the end of systole, which further reduces time of coronary filling and perfusion.⁴

Angina with aortic stenosis almost always occurs with exercise and tachycardia, which affect the diastolic filling period, a close linear correlation would likely be found between the diastolic filling time at anginal threshold and severity of aortic stenosis (valve area). An increase in left

ventricular (LV) peak systolic pressure (LVPSP), LV mass, and LV ejection time (LVET) increases myocardial oxygen demand; decreased diastolic time resulting from the increased LVET and increased LV end-diastolic pressure (LVEDP) decreases myocardial oxygen supply.²

The combination of left ventricular hypertrophy, tachycardia, and decreased perfusion pressure in this patient will

significantly damage the sub endocardial perfusion which can lead to ischemia, and serum troponin I that is related to the contraction of the heart muscle which will be released by the heart muscle during damage from the myocytes.

The usefulness of an accurate and thorough clinical exam in the emergency department is of paramount importance in such patients.¹

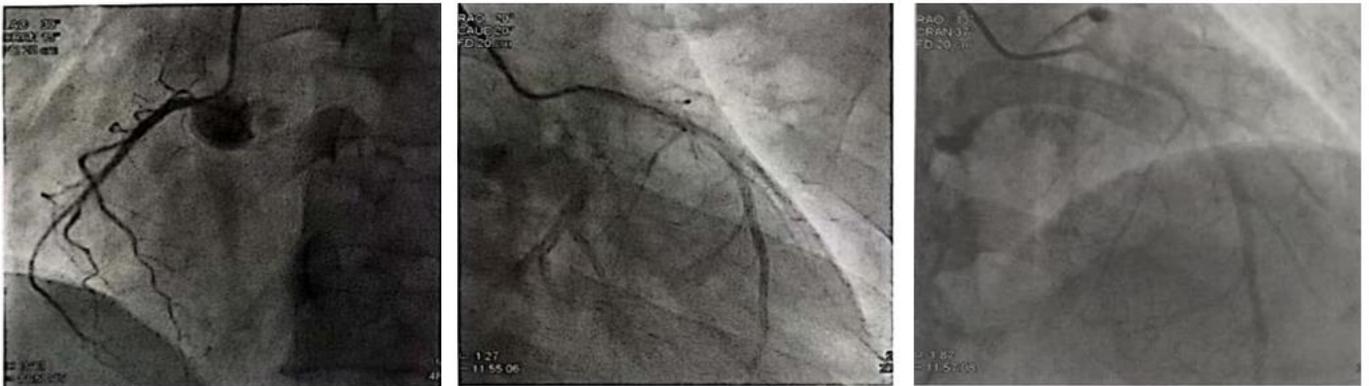


Figure 4. Angiogram of the left coronary artery and the right coronary artery taken the day later of admission show a normal coronary arteries.

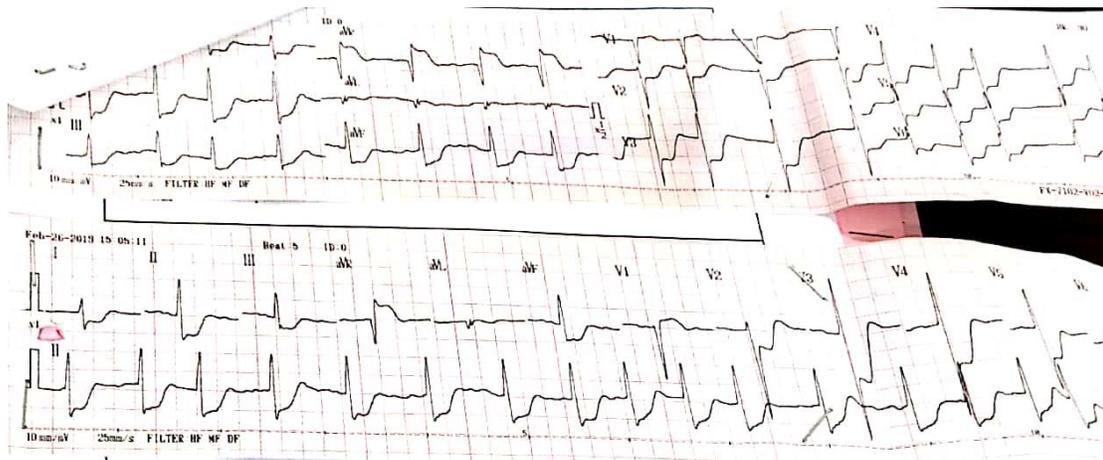


Figure 5. An ECG from previous hospital showed high voltage in the left ventricle and ST-segment depression in leads I, II, III, aVF and V2-V6.

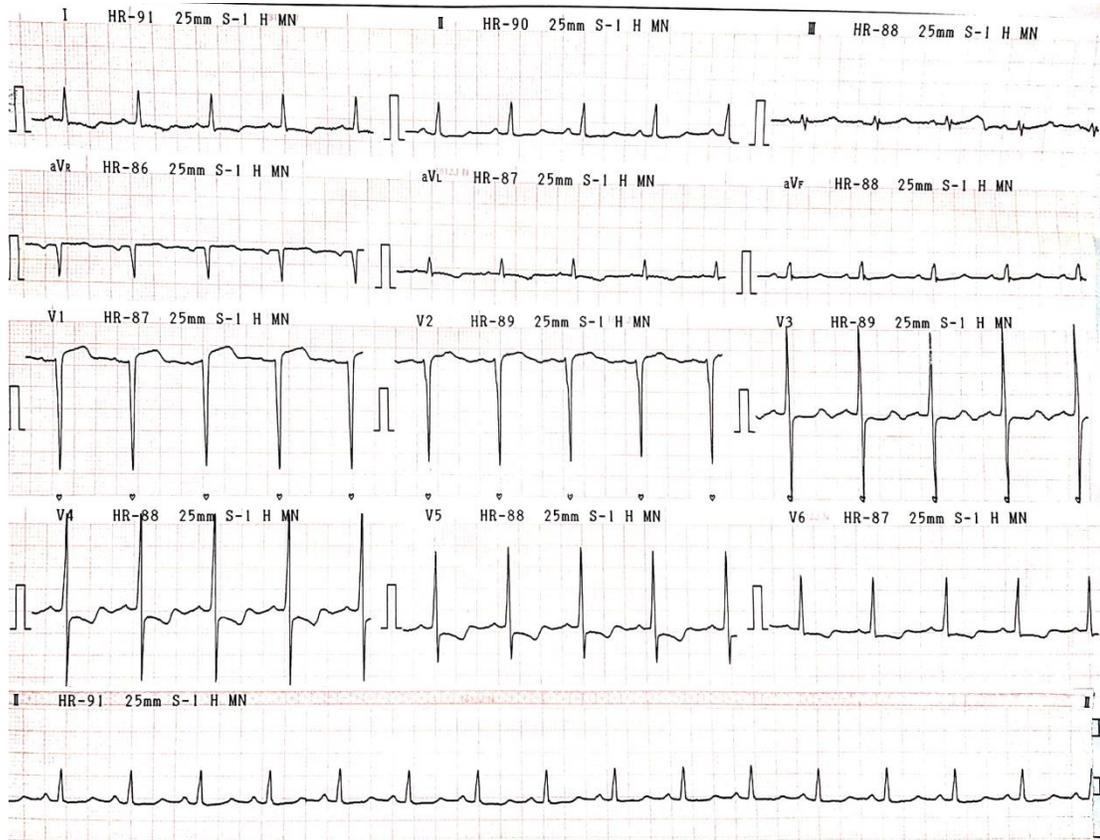


Figure 6. An ECG at our hospital show left ventricle hypertrophy with changes of ST depression in leads I, aVL, V3-V6 and also at the posterior leads.

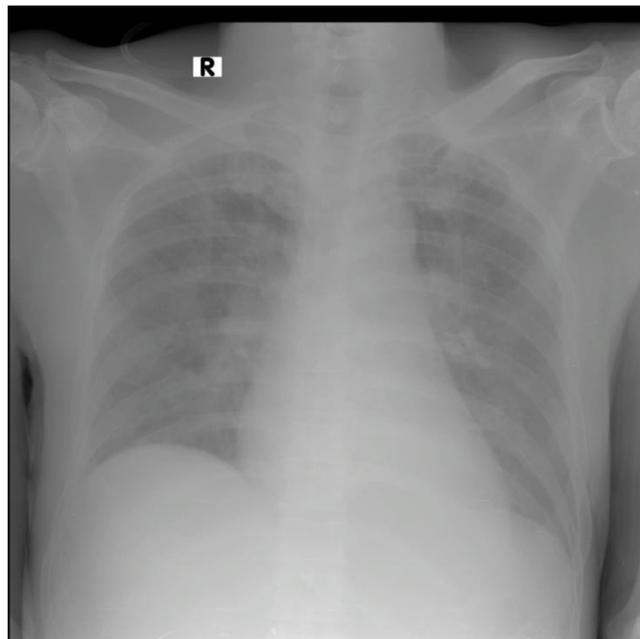


Figure 7. A chest X- ray disclosed an normal cardiac thoracic ratio and pulmonary vascular congestion.

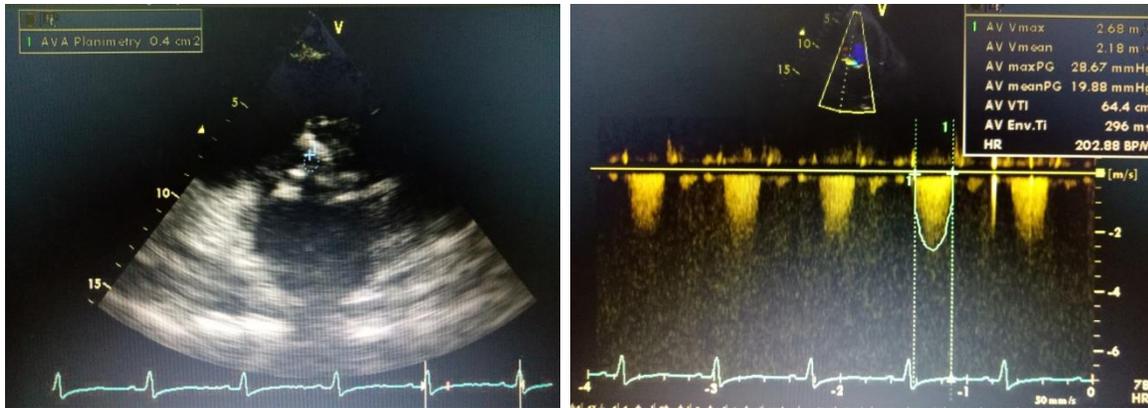


Figure 8. Short-axis views show the aortic valve area was 0.4 cm², which is suggestive of severe aortic stenosis



Figure 9. A coronary angiogram of the left coronary artery and the right coronary artery taken show a normal coronary arteries.

CONCLUSION

This case is interesting because it is a documented case of acute myocardial infarction involving the circumferential sub endocardial wall of the left ventricle in a patient with severe aortic stenosis due to angina micro vascular caused by left ventricular hypertrophy, marked LVH in echocardiography and angiographically normal coronary arteriograms.

This case highlights the importance of the correlating between history taking, physical examination and other supporting examination, especially focused on bedside investigation like echocardiography in the management of patients presenting with chest pain.

REFERENCES

1. Wayangankar S.A., Dasari T.W., Lozano P.M., Beckman K. J. 2010. A case of critical aortic stenosis masquerading as acute coronary syndrome. *Cardiol Res Pract*, doi:10.4061/2010/423465.
2. Kawamoto R., Imamura T., Kawabata K., Date H., Ishikawa T., Maeno M., et al. 2001. Microvascular angina in a patient with aortic stenosis. *Jpn Circ J*, 65:839-841
3. Kodama-Takahashi K., Ohshima K., Kurata A., Yamamoto K., Uemura S., Watanabe S., et al. 2003. Myocardial infarction in a patient with severe aortic stenosis and

- normal coronary arteriograms: involvement of the circumferential subendocardial wall of the left ventricle. *Circ J*, 67:891-894
4. Crea F., Camici P.G.,Bailey Merz C.N. 2014. Coronary microvascular dysfunction: an update. *Eur Heart J*, 35:1101-1111.
 5. Rajappan K., Rimoldi O.E., Dutka D. P., Ariff B., Pennell D. J., Sheridan D. J. et al. 2002. Mechanisms of coronary microcirculatory dysfunction in patients with aortic stenosis and angiographically normal coronary arteries. *Circulation*, 105:470-476.