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Case Report

Severe Aortic Stenosis Mimicking STEACS at Hospital in Remote Areas: A Case Report

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ABSTRACT

Background: This case highlights the need for a comprehensive and accurate physical examination in patients with angina pectoris. **Case Summary:** A 46-year-old man was admitted to the hospital with chest pain and anterior ST segment elevation on electrocardiogram. Given the limited drugs and equipment in remote hospitals, initial treatment for ACS was given immediately. However, no significant improvement was seen with this treatment. An echocardiogram was performed right away and the results showed a severe AVS. B-blockers, ACE-I, diuretics, and MRA antagonists were administered, and the patient had good clinical results, and was recommended for immediate referral to a hospital with primary facilities for valve replacement surgery. **Conclusion:** Severe AVS can cause subendocardial ischemia, so the clinical findings may correspond to ACS. A thorough physical examination allows us to provide more accurate treatment.

Highlights:

1. The value of a detailed physical assessment in patients with angina pectoris is crucial, as emphasized in this report.

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Introduction

Aortic valve stenosis (AVS) is a narrowing of the aortic valve that inhibits blood flow from the left ventricle to the ascending aorta during systole, which can cause symptoms such as chest pain and angina pectoris. Since angina occurs due to increased oxygen demand in the myocardium, the heart controls it by increasing afterload and left ventricular hypertrophy (LVH) [1]. Blockage of left ventricular blood flow due to AVS results in increased afterload, prolongation of left ventricular ejection time, decreased of aortic pressure, and increased of left ventricular end-diastolic pressure. Increased afterload causes LVH and ultimately leads to left ventricular dysfunction and failure [2]. Myocardial oxygen consumption increases along with the increment of left ventricular systolic pressure, mass, and ejection time. Therefore, a decreased in left ventricular function lowers the cardiac output.[3]

Thickening of the LVH causes an increase in the heart's electrical current voltage. This phenomenon is reflected on the ECG as an elevation in the amplitude of the R wave in the left chest leads (I, aVL, V5, V6) and an extend in the depth of the S wave in the right chest leads (III, aVR, V1, V2). LVH can also increase the time of ventricular depolarization which causes widening of the QRS complex and disrupting the repolarization phase as demonstrated by abnormalities in the ST-T wave and a dominant cardiac axis towards the left ventricle.[2,4]

In the coronary arteries, blood-filling and blood flow actually occur during the ventricular diastolic phase. This is because in the systolic phase, the myocardial tissue suppresses the two coronary arteries, refraining the coronary arteries to dilate maximally. When there is a decrease in cardiac preload, it ultimately results in a decrease of myocardial perfusion time. In some cases, patients with decreased myocardial perfusion have typical symptoms of chest pain [5]. This is because the heart muscle has begun to experience damage in the form of myocardial infarction (MI) type 2. MI type 2 is defined as MI secondary to ischemia caused by an increase in demand or decrease in oxygen supply.

Based on the background above, this AVS case report highlights the need for a comprehensive and accurate physical examination in patients with angina pectoris. The majority of patients with ACS have coronary artery stenosis and/or thrombus on angiography, but the subgroups range from 1% to 13%. Subendocardial ischemia can occur in severely hemodynamically compromised aortic stenosis (AS) even in patients with normal coronary arteries and may present clinical findings correspond to ACS. In this case report, we present a patient with severe AVS who had angina at rest, in which increased with exercise, and clinically masqueraded as ACS until coronary angiography ruled out the latter possibility [6]. The preparation of this case report is intended as

a lesson to be more vigilant in handling these cases, especially in remote health facilities and hospitals. In addition, the prevalence of AVS suspected as STE-ACS was also reduced.

Case Presentation

A 46-year-old male patient came to the emergency room with left-side chest pain that radiated to the back, right hand, and left hand. The discomfort followed by shortness of breath that was felt since approximately one month before and worsened in the last two days prior to admission. The pain did not improve with rest and was accompanied by nausea. The patient had a history of acute coronary syndrome, diagnosed at another hospital, and was taking ACS medication after discharge. Shortness of breath worsened by activities. Patient mostly found comfort with more than one pillow to sleep and sometimes had a trouble sleeping because the discomfort in the chest. Patient was conscious and restless when admitted to the hospital.

Initial clinical examination revealed blood pressure (110/80 mmHg), heart rate (111 beats/min), respiratory rate (24 breaths/min), axillary temperature (36.7°C). Systolic murmurs were heard in the aorta area 3/6 and mitral area 4/6. The electrocardiogram showed pathological Q accompanied by ST elevation in leads V1-V4 and ST depression in leads V5, V6, I, and aVL. Based on clinical and ECG findings, plaque rupture was suspected. Hence, ST elevation acute coronary syndrome (STE-ACS) suggested as diagnosis. Laboratory findings such as complete blood count, kidney function, and blood sugar were within normal limits. Cardiac enzyme tests could not be carried out due to the limited laboratory test at our hospital.

Subsequent to Acute Coronary Syndrome (ACS) concluded as diagnosis, dual antiplatelet, anti-ischemic, and anticoagulant were administered. However, the patient's condition did not show significant progress. Further treatment such as fibrinolytic ACS and PCI could not be conducted considering the limited drugs and facilities at our hospital. Due to stable hemodynamics, the patient was transferred to the ICU for further follow-up. Thenceforth, an echocardiography examination was performed in the ICU room. Echocardiogram revealed ejection fraction 50%, with severe aortic stenosis, AVA Echo ≤ 0.8 cm2, AV Vmax 4.9m/s. AV mean PG 63 mmHg, mild mitral regurgitation, TAPSE 22 mm, and CO 2.4 L. Based on these findings, the patient was diagnosed with low output syndrome caused by aortic stenosis, with severe aortic stenosis, and mild mitral regurgitation. Further follow-up in the ICU room, the patient complained of chest pain returning. ACS therapy with dual antiplatelet was stopped, followed by treatment with B-blocker and Diuretic. The patient showed an improvement upon this treatment. However, shortness of breath still occurred intermittently. Therefore, hospital referral was suggested to the

patient with the aim of definitive therapy by replacing the aortic valve with a new one.

Discussion

Aortic valve stenosis (AVS) is a narrowing of the aortic valve that inhibits blood flow from the left ventricle to the ascending aorta during the systolic phase. The etiologies include congenital, calcification, and rheumatic diseases [1]. The three classic symptoms of AVS are angina, syncope, and heart failure. However, the symptoms are often subtle and may vary greatly among patients, depending on the degree. In Ross and Braunwald's original report, median survival after the onset of angina, syncope, and heart failure was five, three, and two years, respectively [4]. Typical chest pain in acute coronary syndrome (ACS) is left-sided chest pain that feels like a pressure, may radiate to the left shoulder and/or arm and is accompanied by dyspnea, nausea, vomiting, diaphoresis, or mild headache.

Echocardiography is the gold standard for the diagnosis and evaluation of AVS, and is the primary non-invasive imaging method for AVS assessment. Transthoracic echocardiography (TTE) remains as the key for the definition of severe AVS, based on aortic valve area (AVA) < 1.0 cm2 or AVA indexed to body surface area (AVAindex) < 0.6 cm2/m2 and transpulmonary pressure gradient (TPG) ≥ 40 mmHg or peak aortic valve velocity (Vmax) ≥ 4 m/s [7]. Some patients with severe AVS based on AVA have a relatively low gradient despite left ventricular ejection fraction (LVEF) is preserved.[1,8]

In this case report, the patient's clinical findings met the diagnostic criteria for severe AVS, which is assessed from patient's complaint of worsened shortness of breath in the last two days before admission. Shortness of breath aggravated by activities. Patient had to be propped by more than one pillow to find comfort while lying down and sometimes had trouble sleeping caused by the discomfort on the chest. The patient experienced a chest pain which characterized as if heavy weight was crushing his chest. Cardiac auscultation helps in differentiating ACS and aortic stenosis. In aortic stenosis, mid systolic murmurs are heard most clearly at the second intercostal space, radiating to the right neck. However, the high frequency component can spread to the apex in calcified aortic valves, which is called as the Gallavardin phenomenon. The murmur may be heard milder in some cases such as left ventricular failure and decreased stroke volume. In this case, there was a typical chest pain of infarction, accompanied by a grade III/IV systolic murmur heard at the aortic area and a grade IV/VI systolic murmur at the mitral area.[9]

The ECG in this case showed pathologic Q waves and ST segment elevation in V1-V4, concurrently accompanied by LV strain. The SEAS study also showed that LVH and LV strain on the ECG were

independent predictors of poor prognosis in patients with asymptomatic AVS [10]. LVH according to the Romhilt and Estes criteria was found to be an independent predictor of the early development of AVS symptoms, although the sensitivity of LVH detection by ECG was found to be as low as 40% [11]. ST segment elevation in the precordial leads is frequently seen in AVS patients. It has been shown that ST segment elevation in leads V1–V2 independently predicts a poor prognosis and more frequent need for AVR in patients with severe AVS.[12]

The patient's clinical and supporting examinations suggest ACS. Considering that ACS requires immediate treatment, we implemented an ACS management protocol for this patient by administering a loading dose of dual anti-platelet, anti-ischemic, anti-coagulant, and statin. The patient had not shown significant improvement. Hence, the diagnosis soon changed after an echocardiography was carried out, which revealed a picture of severe aortic stenosis. The patient was educated to go to a hospital where primary facilities for valve replacement surgery are provided. Angiotensin-converting enzyme inhibitors (ACEI) and angiotensin 2 receptor blockers (ARB) are very useful in the treatment of arterial hypertension and systolic heart failure in the general population. However, its use in patients with severe aortic stenosis should be carefully monitored due to the risk of hypotension and syncope. There is experimental and clinical evidence that ACEIs and ARBs prevent hemodynamic disturbances caused by aortic stenosis [9]. This patient had to undergo valve replacement surgery, because of limited facilities in our hospital, so we provided optimal medical treatment and then prepared the patient to be referred for valve replacement surgery. During treatment in the ICU, optimal medical treatment was given for the management of aorta stenosis, including beta blockers and ACE inhibitors. The patient showed sufficient improvement with stable hemodynamics. The patient was sent home and prepared to be referred to a hospital with valve replacement facilities.

Conclusion

Chest pain and ST elevation on electrocardiogram do not always indicate an Acute Coronary Syndrome, nonetheless, these clinical findings may show in patient with severe aortic stenosis. Doctors have to be more careful in carrying out in clinical assessment and physical examinations to differentiate among those diseases. This is a rare case of severe AVS with ACS-like findings.

Considering there are some clinical conditions with identical symptoms, better facilities and infrastructures are needed in emergency room, such as echocardiography is obligatory for earlier diagnosis, so that patient management becomes

more accurate. Given this case is quite rare to happen, it is necessary to evaluate additional tools such as echocardiography in the emergency room, especially in remote hospitals, to avoid errors in diagnosis and treatment implementation.

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