

A Case of Timely Recognized Aortic Stenosis in Primary Health CareMuharrem Ak¹, Murat Yüksel²¹University of Illinois at Chicago'²Dicle University, Diyarbakır**ABSTRACT****A Case of Timely Recognized Aortic Stenosis in Primary Health Care**

Aortic stenosis (AS) prevalence increases with age, affecting 2–3% of the population older than 65 years of age. If not recognized early and treated appropriately, it may cause significant morbidity and consequently even lead to death. Herein we are presenting a case of sixty five-year-old male patient admitted to our clinic with the complaint of recently developed syncope attack. He was evaluated by complete physical examination and electrocardiogram ECG was ordered. 3/6 systolic murmur was heard and ECG revealed ST-segment elevation, later on the patient was diagnosed as severe calcific aortic stenosis in a secondary care center.

Key Words: Aortic Stenosis, Syncope, Primary Care

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Introduction

Geriatric population with aortic stenosis (AS) is no surprise in rise and seeks remedy for prolonged life.¹ Calcific aortic stenosis is the most common cause of aortic valve replacement in developed countries.¹ However there are no data regarding the unrecognized patients who even had no opportunity to have an operation to live longer. If not recognized and treated appropriately on time, it may cause significant morbidity and consequently even death. The most common etiologic culprit is the calcific degeneration pertinent to the aging process. A harsh murmur over right sternal border sometimes radiating to neck and accompanied by a thrill should warn the physicians about incidental aortic stenosis without any symptom, however in older age it may present with a symptom. The pathologic causes usually occur at the level of the aortic valve^{2,3} AS commonly presents with left ventricular hypertrophy and left atrial enlargement electrocardiographically (ECG). ST-segment elevation is frequently seen in the ECG of the patients with acute coronary syndrome (ACS). Nevertheless ST-segment elevation is not specific only for myocardial infarction. Conditions such as aortic stenosis, pericarditis, cardiomyopathies and electrolyte disturbances may also cause ST-segment elevation.⁴

It is crucial to determine the patients who would benefit from immediate reperfusion therapies in case of ST-segment elevation due to ACS.

Herein we present a 65-year-old male patient admitted with syncope, harsh systolic murmur and ST-segment elevation on ECG who was later diagnosed as severe calcific aortic stenosis.

Case Report

A sixty five-year-old man new to practice was brought to family medicine outpatient clinic with the complaint of recurrent episodes of fainting.

Past medical history was unremarkable except for inguinal hernia operation 10 years ago. Thorough systemic physical examination was rather normal other than unremarkable dyspnea. The cardiovascular risk factor was not closely associated with the current symptoms of the patient. The patient did not mention any previous noteworthy diagnosis from his previous doctor visits. Cardiovascular examination revealed harsh 3/6 systolic murmur extending up to his neck from the upper right sternal border, and peripheral pulses were hardly palpable. ECG revealed ST-segment elevation in chest derivations and ST-segment depression and, T wave inversion was seen in lateral derivations (Figure1). Then the patient was immediately referred to cardiologist for further evaluation in a secondary care center. Transthoracic echocardiography revealed a severe aortic stenosis (mean gradient was 52 mmHg). The

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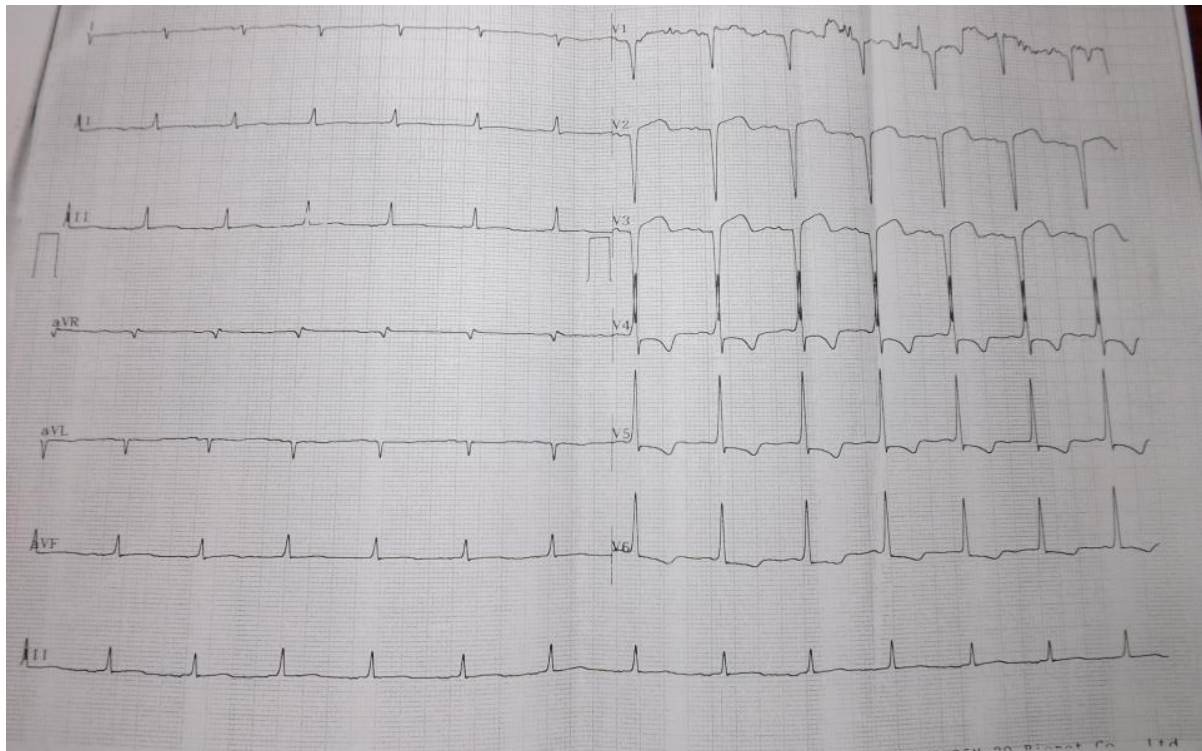


Figure 1. ECG of the patient

aortic leaflets were thickened, calcified, and had a reduced excursion. Electrolytes, liver and kidney function tests, hemogram and coagulation parameters were in normal range. In follow up ST-segment elevations on ECG remained unchanged. STEMI was ruled out because of absence of chest pain and normal levels of cardiac biomarkers. The patient was referred to a tertiary center for further evaluation.

Discussion

AS is narrowing of the aortic valve area through which blood moves from heart to aorta. AS has several etiologies including congenital unicuspid or bicuspid valve, rheumatic fever, and degenerative calcific changes of the valve. Degenerative calcification causing restriction in valvular movement is the most common etiological factor in geriatric age group. Aortic valve area which is normally 3-4 cm² can even decrease below 1 cm² in severe aortic stenosis. Once symptoms develop, survival duration decreases to less than 5 years.⁵ Patients with exertional angina or syncope survive 3 years in average. After development of heart failure, life expectancy is approximately 1 year. The primary ECG findings in aortic stenosis are related to the presence of left ventricular hypertrophy and left atrial enlargement. ECG of patients with

significant aortic stenosis most likely shows evidence of left ventricular hypertrophy with or without a strain pattern, and T-wave inversion. In addition, ST-segment depression and higher QRS voltage also may be seen. A rapid and accurate diagnosis of acute STEMI has critical importance for the early initiation of reperfusion therapy but various conditions may present with ECG patterns mimicking STEMI in clinical practice including acute pericarditis, myocarditis, hyperkalemia, Brugada syndrome, pulmonary embolism, Prinzmetal's angina, cardiomyopathies and cerebrovascular diseases. For instance Khan et al. reported a case of hypertrophic cardiomyopathy with ST-segment elevation mimicking STEMI.⁶ Prinzmetal's angina should be considered in case of ST-segment elevation but persistent pattern of ST-segment in our case ruled out this condition. Nitroglycerine is contraindicated in severe aortic stenosis and should not be given to rule out Prinzmetal's angina in patients with harsh systolic murmur.

The delay in the timely diagnosis of AS is multifaceted. Inclination to oversimplify the symptoms as the "natural phenomenon of aging process" may be associated with the delayed diagnosis of diseases in geriatric population. Coronary angiography revealed no coronary stenosis nor vasospasm, and cardiac biomarkers

didn't elevate in our case. He didn't notice any chest pain. Recently developed syncope attacks concomitant with ST-segment elevation and harsh systolic murmur provided sufficient clinical data for the recognition of aortic stenosis. Although not the chief presenting symptom, dyspnea as an accompanying symptom needs elaboration which may give valuable clues about the presence of non-cardiac causes such as COPD, pulmonary emboli, asthma, neuromuscular disorders and anxiety disorders. The patient was referred to a tertiary center for further evaluation. Aortic valve replacement operations today have excellent outcomes, however deciding whether a patient should go to surgery does not merely depend on physicians but it is a multifaceted issue to be addressed and is beyond the scope of this case. Nevertheless family physicians often follow up patients who have cardiac surgeries. An evidence-based approach to daily practice for a family physician is to close monitoring of the patients' international normalized ratio levels under Coumadin treatment.

Conclusion

Acute coronary syndrome should always be kept in mind in case of ST-segment elevation on ECG. However in the geriatric age group, recently developed syncope with ST-segment elevation without chest pain deserves particular attention for further evaluation in order to rule out aortic stenosis.

Despite new developments in medical science, thorough physical examination and evaluation of ECG of patients in primary health care have paramount importance for early diagnosis of disorders such as the case presented here. In addition to effective use of basic tools "time and the opportunity to help patients are very powerful diagnostic tools in the hands of family physicians".⁷

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