CASE REPORT

Pseudo-infarct in ECG in A Case of Asymmetric Septal Hypertrophy

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ABSTRACT

A 68-year-old male with a history of type 2 diabetes and hypertension presented for a routine check-up. His routine ECG revealed recent inferior infarct. He was admitted and later echocardiography showed it to as asymmetric septal hypertrophy. The ECG findings of the disease and various causes of pseudo-infarct are discussed.

Keywords: Asymmetric septal hypertrophy, pseudo-infarct, inferior wall

CASE CAPSULE

A 68-year-old male with a history of type 2 diabetes for a decade and hypertension for five years presented for a routine check-up. He was on the following drugs; glipizide, metformin, atenolol, enalapril, atorvastatin and vitamins. He was asymptomatic. A routine ECG revealed inferior infarct of recent onset. There was no evidence of either left atrial overload or left ventricular hypertrophy. He was hospitalized; injection heparin, tablet ecosprin were added. He was asymptomatic. His random blood sugar was 163 mg/dl and blood pressure was 136/84 mmHg. A repeat ECG the next day was similar. His other investigations like blood urea, creatinine and electrolytes were normal; urine ketones were negative. Echo heart on the following day revealed asymmetric septal hypertrophy (ASH) of septal lateral wall and apex. There was no evidence of aortic stenosis or regional wall motion abnormality. Injection heparin was withdrawn after a diagnosis of pseudo-infarct was made. We do not have the facilities for an enzyme analyses or angiography. The patient was discharged the next day at his request. As he was asymptomatic, he was not willing for further evaluation.

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DISCUSSION

ASH may be asymptomatic or manifest as pain in the chest, syncope, congestive heart failure or sudden death. Some patients have left ventricular outflow obstruction, and it is this subgroup which was originally described as having idiopathic hypertrophic subaortic stenosis. Our patient had no symptoms except for very occasional giddiness, which was elicited on repeated probing. It has been suggested that a basic marker for the disease is ASH, defined echocardiographically as a septal to posterior wall thickness ratio >1.3 although diseases other than hypertrophic cardiomyopathy may result in echocardiographic ASH. A variety of ECG findings have been reported in association with hypertrophic cardiomyopathy, including ventricular hypertrophy, 'pseudo-infarct' Q waves, atrial enlargement and abnormalities of the S-T segment and T waves. In a separate study, on description of ECG changes in ASH probable or definite, left ventricular hypertrophy was present in 33% of the patients; Q-wave patterns consistent with transmural myocardial infarction (MI)
were present in 21% of patients. A 35-year-old woman with 40-msec Q waves in both inferior and anterior leads had normal coronary arteries, no disorders of wall motion, and a subaortic gradient only after a premature ventricular contraction occurring during Valsalva's maneuver. A 70-year-old man with 40-msec Q waves in leads V1-V4 had a provokable gradient and coronary atherosclerosis but no myocardial scar at autopsy. Three patients with anterior and four with inferior 40-msec Q-wave patterns did not undergo cardiac catheterization. None of these patients had a clinical history of MI. Our patient had no clinical history of MI.

The various causes of pseudo-infarct patterns are described below.

- In left ventricular hypertrophy, there is often a QS deflection or poor R-wave progression in the right precordial leads that suggests anterior MI. The secondary ST-segment elevation in these leads may be mistaken as a current of injury.

- In pulmonary emphysema, the R waves in the right precordial and sometimes mid-precordial leads become quite small or are absent, suggesting anterior MI. These QRS changes are explained by the vertical displacement of the heart secondary to a low-lying diaphragm and the intervention of hyperinflated lungs.

- The pseudo-infarction pattern may be seen in patients with pneumothorax. The voltage of the QRS complex may be reduced. QS deflection may appear in the right precordial leads.

- In pulmonary embolism, the Q waves in lead III (as part of the S,Q pattern), and sometimes in lead aVF, that are accompanied by ST-segment and T-wave changes are often interpreted as inferior MI. Qs complexes with ST-segment elevation may occasionally develop in these leads and mimic acute anterior myocardial infarction.

- In hypertrophic cardiomyopathy (ASH), abnormal Q waves are often seen, especially in the left precordial leads and lead I. These Q waves have been attributed to ventricular septal hypertrophy.

- Myocardial fibrosis is responsible for the pseudo-infarction pattern in patients with dilated cardiomyopathy, progressive muscular dystrophy, Friedreich's ataxia, scleroderma, amyloidosis and tumors of the heart.

- QS deflections are often seen in the right precordial leads in patients with complete left bundle branch block in the absence of MI.

- Left anterior hemiblock is occasionally associated with small Q waves in the precordial leads that mimic anterior MI.

- The δ waves in Wolff-Parkinson-White syndrome are frequently interpreted as abnormal Q waves of MI.

- Pheochromocytoma may be associated with striking ECG changes mimicking ischemic heart disease.

- Other conditions that may be associated with ECG changes simulating MI include intracranial hemorrhage, hyperkalemia and acute pericarditis.

- Pseudo-infarct patterns in ECG can occur in diabetic ketoacidosis with normokalemia.

- Pseudo-infarct patterns in ECG can occur after intravenous flecainide.

- In postoperative period after cardiac transplantation, 5-10

In our patient, the renal parameters were normal with no evidence of any other systemic illness. Hence, the pattern is probably due to ASH.

CONCLUSION

- Infarct patterns can occur in asymptomatic individuals and ASH is one of the causes.

- We were mistaken because of the presence of associated risk factors like diabetes and hypertension.

- We can be criticized because of the lack of establishment of normal coronaries with angiography.

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REFERENCES