CLINICAL VIGNETTE

An Outpatient STEMI

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Case

A 71-year-old male with hypertension, dyslipidemia, and type 2 diabetes mellitus presented to the outpatient cardiology office complaining of dyspnea on exertion and generalized weakness for several days. He was comfortable at rest and with light exertion. He denied chest pain, jaw or arm pain, nausea, and diaphoresis. He had no history of smoking, no family history of early onset coronary artery disease, and no significant alcohol use. His vitals showed a blood pressure of 114/72 mmHg and pulse of 50 bpm. His physical exam was otherwise unremarkable.

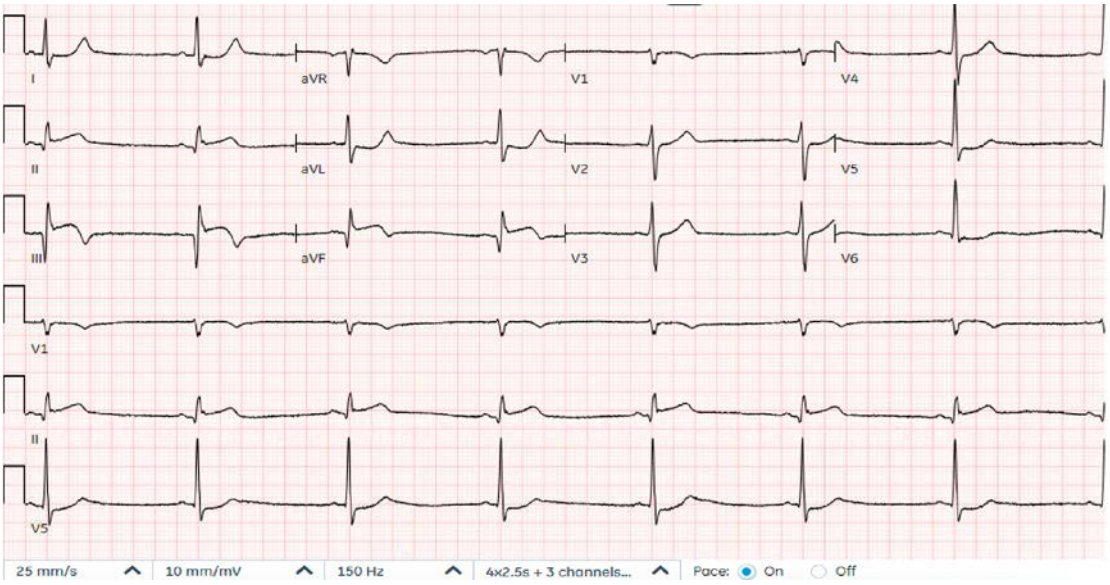
An electrocardiogram was obtained (Figure 1), as is routine for all new cardiology patients. This showed sinus bradycardia, ST segment elevations and early Q-wave formations in the inferior leads (II, III, & aVF), as well as reciprocal ST segment depressions in lead aVL.

The EKG was interpreted as: findings consistent with an acute ST-elevation inferior myocardial infarction. The patient was emergently sent to the hospital where code STEMI was activated. He was rushed to the cardiac catheterization lab and found to have an acute, 100% thrombotic occlusion of the proximal right coronary artery (RCA) with Thrombolysis in Myocardial Infarction (TIMI) flow grade of zero. He underwent mechanical thrombectomy with Penumbra® device followed by percutaneous coronary intervention (PCI) with three overlapping drug-eluting stents (2.5 x 28 mm, 3.0 x 38 mm, and 3.5 x 15 mm; post dilated to 3.25 mm) in the RCA. Post-PCI, grade 3 TIMI flow was restored. His coronary anatomy was right-dominant with otherwise non-obstructive disease, notable for a 50% obstruction in the left anterior descending artery and mild diffuse disease in the left circumflex artery.

Shortly post-PCI, an echocardiogram showed a mildly decreased left ventricular systolic function with an ejection fraction of 45%, as well as hypokinesis of the entire inferior and inferolateral walls. Two days later, repeat echocardiogram showed normalization of his left ventricular systolic function with an ejection fraction of 60%, as well as resolution of the inferior and inferolateral wall motion abnormalities.

The patient's hospital course included an initial troponin of 3.83 ng/mL which subsequently peaked at 6.72 ng/mL. Notably, his hemoglobin A1c was 13.2%. His lipid profile, prior to initiating statin therapy, showed a total cholesterol of 165 mg/dL, HDL of 40 mg/dL, LDL of 95 mg/dL, and a triglyceride count of 149 mg/dL.

The patient did well during his hospital stay, with improvement in his presenting symptoms of weakness & exertional dyspnea on follow-up.



Discussion

While most patients with acute coronary syndromes (ACS) present with chest pain, there is a proportion that present with atypical, minimal, or even no symptoms.[1] Diabetic patients especially are likely to present with atypical or absent symptoms with ACS,[2] and thus may have a greater delay before presenting to clinical attention.[3]

Several mechanisms have been proposed to explain the prevalence of atypical or absent symptoms in diabetics with ischemic heart disease. Early clinical and autopsy studies have shown evidence of cardiac autonomic neuropathy in these patients, which can impair the translation of myocardial ischemia into nociceptive signals.[4] Diabetics have, also been shown to have reduced myocardial flow reserve, which is inversely related to glycemic index control.[5] Multi-vessel disease and complex lesions are also more common among diabetics, leading to greater preconditioning.[6] Consequently, these patients have a prolonged or absent anginal threshold, leading to silent ischemia or infarction.

Silent ischemia may be prevalent in up to 1 in 5 diabetics despite contemporary medical care and close follow-up. There was strong evidence for this in the Detection of Ischemia in Asymptomatic Diabetics (DIAD) study,[7] a large, well-designed, prospective, controlled trial that randomized over 1,100 diabetics with no known coronary disease to be screened for underlying ischemia with myocardial perfusion imaging (MPI). Of the screened subjects, 22% had a perfusion defect, including 6% with a moderate or large sized defect.

Notably, in the absence of symptoms, screening for ischemia with stress testing in these subjects does not seem to improve outcomes. During the nearly 5 years of clinical follow-up in the DIAD trial, subjects had similar rates of myocardial infarction or cardiac death regardless of their randomization to screening or non-screening (2.7% versus 3.0%, respectively). Routine or universal screening for asymptomatic ischemic disease in these patients, therefore, has not been recommended in clinical guidelines. However, this remains controversial. This recommendation is partially because diabetes should already be considered coronary artery disease equivalent. As such, these patients should already be receiving intensive medical therapy– an approach that has been repeatedly demonstrated to be equally beneficial as invasive revascularization in stable patients. Current guidelines therefore only recommend that screening for silent ischemia may be considered in high-risk patients with diabetes and concurrent proteinuria, elevated calcium artery score, and peripheral artery disease.

In conclusion, this vignette highlights the fact that diabetic patients may have minimal or no symptoms in the setting of an acute coronary syndrome, even one involving a transmural infarct with acute ST-elevations. Clinicians should be aware of recognizing such patients rapidly, and reducing delays in diagnosis and definitive management.

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