Elevated troponin and left bundle branch block in the setting of suspected septicemia and demand ischemia: to treat or not to treat

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Abstract

Elevated troponin and atypical chest pain in the setting of septicemia and Type II Non ST elevation myocardial infarction is frequently encountered. These cases are not necessarily scheduled for emergent cardiac catheterization. High index of clinical suspicion and continuous in-patient cardiac monitoring with serial trending of cardiac enzymes are important in such cases. Subsequent sudden development of electrocardiogram changes requires prompt investigation with emergent coronary catheterization. These types of cases may be missed especially in females who present with atypical chest pain and in patients with Left bundle branch block.

Introduction

Patients with myocardial infarction (MI) and left bundle branch block (LBBB) have been shown to have higher in-hospital mortality (22.6%) compared to patients without LBBB (13.1%).1 After adjusting for potential confounders, patients with LBBB associated with a 34% increased risk for in-hospital death. However, it is not uncommon for physicians to deduce patients’ electrocardiogram (ECG) as non-diagnostic and compromise treatment via delaying medication or coronary intervention.2 In the setting of suspected septicemia and a possible Type II Non ST segment elevation MI (NSTEMI), definitive treatment for MI may further be delayed.

Patients with LBBB and MI are generally older (76 vs 68 years) and are more likely to be female (50% vs 41%) compared to patients with MI in absence of LBBB.3 These patients may present without typical chest pain and may have more atypical symptoms which pose a greater risk of undertreatment for MI.4 We present a case of atypical chest pain and mildly elevated troponin that was initially suspected to be Type II NSTEMI in the setting of sepsis. High index of clinical suspicion and continuous in-patient cardiac monitoring with serial trending of troponin helped to detect subsequent development of LBBB which required management with emergent cardiac catheterization and coronary stenting.

Case Report

A 47-year-old woman with history of hypertension, diabetes mellitus type II, hyperlipidemia, COPD, fibromyalgia and Post traumatic stress disorder presented to the emergency department complaining of chest pain, fever, diaphoresis and chills. She also reported a chronic productive cough with yellowish/whitish phlegm and a two-day history of dizziness, lightheadedness, decreased appetite, generalized weakness and malaise. At 2 AM on the day of admission, she woke up from sleep and felt severe chest pain associated with shortness of breath. Her chest pain was substernal, radiating to bilateral shoulders, and was constant with no exacerbating or relieving factors. Her axillary temperature was 101 F on admission, Blood pressure 102/60 mm Hg, respiratory rate 14 per minute, heart rate 98-100 beats per minute and saturation >92% at room air. She received single doses each of 325 mg of aspirin, 0.4 mg of sublingual nitroglycerin and 4 mg of intravenous morphine. Her chest pain did not completely get relieved. Initial blood work revealed an elevated Troponin I at 0.56 ng/mL (Normal reference range: 0.00 to 0.07 ng/mL), a white count elevated at 14.2 x 10³ cells/µL (Normal reference range: 4 x 10³ to 10 x 10³ cells/µL) Her renal function test was normal. Her initial ECG revealed sinus tachycardia with poor R-wave progression across leads V1-3 and mild ST depression in the inferolateral leads (Figure 1). CT angiography of the chest did not reveal pulmonary embolism or aortic dissection, but did depict pulmonary fibrosis and bronchiectasis within the apical segment of the right upper lobe.

She was subsequently admitted in the telemetry with suspicion of sepsis and a possible demand ischemia and was started on empiric intravenous antibiotics. Repeat Troponin I 6 hours later increased to 0.63 ng/mL. A repeat 12-lead ECG showed new onset LBBB (Figure 2). In view of these findings and continued intermittent chest pain, she was referred for emergent cardiac catheterization. The procedure revealed 95% narrowing of proximal to mid LAD in which a drug-eluting stent was placed (Figures 3-5). Luminal narrowing with up to 90-95% stenosis along with dissection was noted in the proximal part of the RCA, but this lesion appeared to be chronic since there were some collateral circulations to the distal RCA from the LAD and was thus not stented. After cardiac catheterization, the patient had significant relief in her symptoms and a follow up ECG depicted resolution of LBBB (Figure 3). Antibiotics were discontinued because her symptoms were attributed to myocardial infarction and suspected septicemia was a mere confounder. She was later discharged on aspirin, prasugrel, metoprolol and atorvastatin.

Discussion

LBBB is a pattern seen on the ECG that is produced when the normal electrical activity is interrupted in the His-Purkinje system. It occurs most commonly in patients with underlying heart disease and may indicate progressive disease of the conducting system. LBBB can also be present in an asymptomatic patient without any structural heart disease.5 It is associated with approximately 7% of all infarctions yet patients with this condition are less likely to receive timely medical or coronary intervention and have higher in-hospital mortal-
LBBB can complicate the diagnosis of MI and interfere with interpretation of stress testing as it delays ventricular depolarization to later stages in the QRS complex making it difficult to recognize myocardial ischemia. Thus, it is prudent to give utmost attention to clinical history and examination besides serial monitoring of cardiac enzymes.

The Sgarbossa criteria help to make a diagnosis of acute MI in the presence of underlying LBBB with three independent ECG criteria. An ST-segment elevation of more than 1mm that is concordant with the QRS complex has a score of 5; a ST-segment depression of more than 1mm in leads V1, V2, or V3 has a score of 3; and a ST-segment elevation of more than 5mm that is discordant with the QRS complex will have a score of 2. Although a score of 3 or more is highly specific (98%) for acute MI, the absence of these criteria does not rule it out completely (20% sensitivity).

In a study by Frans J. Wackers, a correlation between ECG changes in LBBB with localization of the infarct by thallium scintigraphy was made. In his study, it was found that serial changes of QRS complexes or ST segments on ECG had 67% sensitivity for MI; ST segment elevation had 54% sensitivity for MI; and abnormal Q waves had 31% sensitivity for MI. Initial positive deflection of the R wave in V1 with an abnormal Q-wave in V6 was highly specific for an anteroseptal MI, but was shown to have 20% sensitivity. Cabrera’s sign, referring to a prominent notching in the ascending limb of the S wave in leads V3 and V4, was found to have 47% sensitivity for anteroseptal MI. Our patient presented with minimal ST segment depression in inferolateral leads and poor R-wave progression in V1-V3. Serial monitoring of cardiac enzymes and continuous monitoring of telemetry rhythm, with close observation in the hospital, helped us to identify MI under the presence of new onset LBBB.

It is also possible that this patient may have had type 2 MI secondary to sepsis. Type 2 MI results secondary to ischemia due to the presence of increased or decreased oxygen supply. 24% of type 2 MI cases are due to sepsis and cardiac troponins are frequently elevated as in this patient. Thus, sepsis may have been a significant confounding factor in the patient’s acute MI.

Conclusions

Patients with LBBB and acute MI can sometimes present with atypical symptoms. This is more relevant in cases of female

Figure 1. Sinus tachycardia, minimal ST-segment depression in infero-lateral leads and poor R wave progression V1-V3.

Figure 2. Sinus tachycardia with new left bundle branch block.

Figure 3. Sinus rhythm, resolution of left bundle branch compared to EKG two days ago.
patients. These patients are less likely to receive timely thrombolytic therapy or catheter-based reperfusion therapy. It is more likely to happen in presence of confounding factor such as suspected septicemia which itself can give rise to demand ischemia. High index of clinical suspicion, continuous in-patient cardiac monitoring and serial monitoring of cardiac enzymes within the context of a relevant clinical presentation should guide a clinician towards subjecting patients for a definitive test to rule out acute coronary syndrome.

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