CASE REPORT

Alternating Bundle Branch Block or Pyridostigmine-induced Mobitz Type II Block Masquerading as Acute Coronary Syndrome

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ABSTRACT

Background: “ST-T changes in the ECG!!” These words are enough to get the emergency doctor to spring into action. These changes can be diffuse and/or non-specific but we should rule out all emergent and urgent causes before shifting the patient to the specialist. To err on the side of dangerous etiology is the dictum.

Introduction: Out of all emergency department (ED) patients with undifferentiated chest pain, 7% will have ECG findings consistent with acute ischemia or infarction, and 6–10% of those in whom cardiac markers are ordered will have initially positive results. Of all patients with the possible acute coronary syndrome (ACS), 5–15% ultimately prove to have ACS. 1 Shortness of breath with chest pain mostly has a cardiac origin in the presence of dynamic ECG changes. We had managed a patient with rapidly evolving ECG changes, chest pain, palpitations, and grade III–IV dyspnea. In the chaotic environment of a busy ED, the most probable diagnosis here will be ACS. Comorbid conditions like diabetes mellitus, hypertension, and prior coronary artery disease (CAD) are commonly enquired. However, other long-standing illnesses like myasthenia gravis (MG), as in our patient can be easily missed if a patient is not forthcoming with history. We experienced a similar confusion when in the cacophony of chest pain, dyspnea, and T wave inversions with bundle branch blocks, ACS protocol was initiated and a simple diagnosis was missed. The significance of the alternating bundle branch block (ABBB) will be presented to the readers.

Keywords: Coronary syndrome, Infarction, Retrosternal.

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INTRODUCTION

Out of all emergency department (ED) patients with undifferentiated chest pain, 7% will have ECG findings consistent with acute ischemia or infarction, and 6–10% of those in whom cardiac markers are ordered will have initially positive results. Of all patients with the possible acute coronary syndrome (ACS), 5–15% ultimately prove to have ACS. 1 Shortness of breath with chest pain mostly has a cardiac origin in the presence of dynamic ECG changes. We had managed a patient with rapidly evolving ECG changes, chest pain, palpitations, and grade III–IV dyspnea. In the chaotic environment of a busy ED, the most probable diagnosis here will be ACS. Comorbid conditions like diabetes mellitus, hypertension, and prior coronary artery disease (CAD) are commonly enquired. However, other long-standing illnesses like myasthenia gravis (MG), as in our patient can be easily missed if a patient is not forthcoming with history. We experienced a similar confusion when in the cacophony of chest pain, dyspnea, and T wave inversions with bundle branch blocks, ACS protocol was initiated and a simple diagnosis was missed. The significance of the alternating bundle branch block (ABBB) will be presented to the readers.

CASE DESCRIPTION

A 69-year-old male patient presented to an ED with a history of breathlessness and chest pain for 1 day. The dyspnea was associated with non-productive cough. He was neither tachypneic nor cyanosed at the time of ED admission. The chest pain was sudden in onset, localized to the left side of the chest, dull aching, and pressure type, and had no alleviating and relieving factors, not radiating and not associated with syncope, diaphoresis, and nausea and vomiting. The patient was connected to a cardiac monitor and vital signs were recorded. The airway of the patient was patent, breathing at 14/minute with a saturation of 96% on room air. The heart rate (HR) was 128/minute, BP was 110/60 mm Hg, and GRBS was 265 mg/dL. Pallor raised jugular venous pressure and pedal edema were absent. There was no prior history of CAD. The patient was a known diabetic on metformin, hypertensive on telmisartan, and thymoma negative MG on pyridostigmine and wosolone. There were no murmurs, gallop rhythm, and previous surgical incisions on the chest wall.

The first ECG (Fig. 1a) showed a rate of 126, sinus rhythm, left axis deviation, QRS complex >120 ms, dominant S wave in V1, broad monophasic R wave in lateral leads (I, aVL, V5–V6), absent Q wave in lateral leads (I, V5–V6), and poor R wave progression in the chest leads. These changes were indicative of the left bundle branch block (LBBB). Left ventricular hypertrophy is a strong differential diagnosis for LBBB with widened QRS and ST depression-T inversion in lateral leads.

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Adding to Confusion in a Busy Emergency Department

Bedside echocardiography was the next plan. However, the monitor showed a sudden decrease in HR, and ECG was repeated. Figure 1b depicts the second ECG suggestive of a rate of 52 bpm, second degree AV block (Mobitz type II) with narrow QRS complexes, and deeply inverted T waves in V1–V3. The arterial blood gases and troponins were unremarkable (Tables 1 and 2). Point of care (POC) creatinine and electrolytes sodium and potassium were also normal. Another ECG (Fig. 1c) was ordered which showed ABBB in 30 minutes. These changes were dynamic, changing morphology very quickly, and hence were suspicious to be ominous.

The 2D echocardiography was fairly normal (no regional wall motion abnormality, good LV systolic function, grade I diastolic dysfunction, no mitral regurgitation, pulmonary arterial hypertension (PAH), vegetation, pericardial effusion, clot, and an ejection fraction of 58%). The chest radiograph was suggestive of left lower zone consolidation (Fig. 2).

The history of pyridostigmine therapy for MG was shared by attendants with a cardiologist. The patient was given 325 mg of enteric-coated aspirin and 40 mg atorvastatin. His pyridostigmine was stopped and he was kept on continuous neuro-hemodynamic observation. He was started on antibiotics and O2 therapy. The patient underwent repeat troponins after 6 normal hours. He underwent a coronary angiogram 14 days later which showed normal coronary anatomy.

Table 1: Cardiac biomarkers profile

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<tr>
<td>PH</td>
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<tr>
<td>Lactates</td>
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Figs 1A to C: ECGs depicting LBBB (A), RBBB (B) and ABBB (C)

Fig. 2: Chest radiograph suggestive of left lower lobe pneumonia

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**Discussion**

The low probability of ACS is a well-established identity. These patients are observed with serial ECG and repeat troponin over the next 6–24 hours. If they remain pain-free, they can undergo treadmill testing within 72 hours. Our patient had non-classic chest pain. The classic pain is retrosternal, left, crushing, squeezing, tightness, and pressure type. It comes with exertion and gets relieved on rest. The duration is 2–30 minutes. Non-classic pain lasts for seconds, has a constant character for 12–24 hours, no waxing/waning factors, stabbing in character, well-localized or positional, and pleuritic. He had three major risk factors as an elderly male, diabetes, and hypertension for ACS. The other risk factors to look for are postmenopausal females, tobacco use, hypercholesterolemia, truncal obesity, family history, and sedentary lifestyle. Those who will have a poor outcome are female gender, racial minorities, alcoholics, psychiatric illness, and those on multiple prescription medications.

Our patient had rapidly evolving ECG changes from LBBB to RBBB to ABBB. European Society of Cardiology ST-segment elevation myocardial infarction (STEMI) guidelines from 2012 state that urgent coronary angiography is indicated in patients with STEMI and patients with new or presumed new LBBB. American College of Cardiology/American Heart Association guidelines on the management of STEMI from 2013, however, do not recommend that new or presumed new LBBB be considered diagnostic of acute myocardial infarction (AMI). New or presumably new, transient ST-segment deviation (>1 mm) or T wave inversions in multiple precordial leads have a high likelihood of ACS. The intermediate likelihood is recognized by the presence of fixed Q waves, ST depression 0.5 to 1 mm, or T wave inversion >1 mm. Flattening of T wave or inversion <1 mm in leads with dominant R waves or normal ECG points toward a low likelihood of the disease. An ABBB could, in an appropriate clinical setting, be an indicator of myocardial reperfusion, thus changing the management and urgency of an ACS patient. Bundle-branch blocks are a perplexing entity that often blurs the AMI diagnosis, but there are ways to recognize myocardial ischemia (MI) despite the presence of a BBB. In patients with LBBB, the diagnosis can be made with the original or modified Sgarbossa criteria. In RBBB, however, relying on ST elevations could lead to a significant under-diagnosis of AMI.

Mobitz type II block is the failure of the conduction system at the level of His-Purkinje fibers. Broad QRS complexes are a result of blocks distal to the bundle of His, whereas blocks within the bundle of His show as narrow QRS complexes. This is secondary to anterior MI, idiopathic fibrosis of the conducting system, cardiac surgery, inflammatory diseases, autoimmune disease, infiltrative myocardial disease, hyperkalemia, and drugs like beta-blockers, calcium channel blockers, digoxin and amiodarone. There is a high risk of progression to asystole.

The cardiologist brought to our notice that the history of MG in this patient was extremely important. A decrease in the number of available nicotinic acetylcholine receptors at neuromuscular junction leads to defective neuromuscular transmission in skeletal muscles that manifest as muscle weakness. Heart muscle is a target for autoimmune inflammation in MG in approximately 48% of patients who develop antistriational antibodies. These patients clinically show features of myocarditis, arrhythmias, cardiomyopathy, and sudden cardiac death. The dense cholinergic innervation of the AV node makes it more susceptible to conduction slowing secondary to vagomimetic drugs, such as, pyridostigmine.

A transient reversible form of left ventricular dysfunction often presenting as ACS in the absence of significant coronary stenosis is seen in takotsubo cardiomyopathy seen with severe MG. It is more common to develop in patients on plasmapheresis therapy for MG.

Autonomic dysfunctions seen in patients on pyridostigmine therapy show wide fluctuations of HR. Common manifestations are atrial fibrillation, ventricular and supraventricular extrasystoles, and prolonged corrected QT interval due to the presence of ganglionic AChR antibodies.

Diffuse spasm of left anterior descending (LAD) seen due to anticholinesterase therapy and coronary vasospasm causing non-obstructive MI presenting as non-specific T wave changes shortly after starting pyridostigmine are reported in literature.

**Conclusion**

Long-term therapy with pyridostigmine may be associated with potential side effects, such as, bradyarrhythmias, asystole, and sinus arrest.

Hyoscyamine, a muscarinic antagonist in a dose of 0.125–0.25 mg qid is recommended as the first-line therapy. The persistence of bradyarrhythmias may warrant pacemaker placement. Emergency/critical care physicians should be well versed with the skill of insertion of a temporary transvenous pacemaker. Magnetic resonance (MR) myocardial perfusion imaging and coronary CT angiography have complementary roles in the evaluation of patients who are suspected of having CAD. Coronary CT angiography can be used to reliably rule out CAD, but its capability to demonstrate hemodynamically significant CAD is limited. The combination of both techniques enables the clinician to evaluate the morphology and functional relevance of CAD comprehensively and non-invasively. The algorithms and scoring systems in cardiology patients can be confusing at times so more important is to stick to history, physical examination, and cardiac markers for diagnosing ACS. Each ED should discuss the protocol for low-risk ACS patients with the interventional cardiology team and display it in triage areas. The chest pain and breathlessness in our patient was explained by community-acquired pneumonia.

**References**


