

Dynamic ECG Changes in Myasthenia Gravis

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Abstract: Sudden onset chest pain with shortness of breath and dynamic ECG changes point towards acute coronary syndrome but a case report on 69 years old gentleman presented to ED with one day history of chest pain associated with grade III shortness of breath, ECGs were recorded which showed the initial normal sinus rhythm with LBBB (left bundle branch block) changed to type 2 AV BLOCK [atrio ventricular block]. A new onset LBBB in background of chest pain and SOB (shortness of breath) strongly pointed towards ACS (acute coronary syndrome). But the serum levels of cardiac biomarkers were normal. Past Medical history was suggestive of Myasthenia gravis on pyridostigmine. Pyridostigmine inhibits acetylcholinesterase this results in an increase in the amount of acetylcholine available for the already depleted acetylcholine receptors. Bradycardia and Heart Blocks secondary to muscarinic side effects of pyridostigmine are seen in emergency department

Keywords: Myasthenia gravis, LBBB, AV BLOCK

1. Introduction

Myasthenia gravis (MG) is an autoimmune disorder affecting the neuromuscular junctions. It is characterized by decrease in the number of available nicotinic acetylcholine receptors at postsynaptic membrane due to presence of pathogenic autoantibodies [1, 2]. Reversible acetylcholinesterase inhibitors are used as initial treatment for MG and pyridostigmine is the most widely used drug. They improve symptoms by increasing concentration of acetylcholine at the neuromuscular junction leading to stimulation of nicotinic receptors at postsynaptic membranes [2, 3]. However, use of these drugs is associated with potential adverse effects due to stimulation of muscarinic receptors. In heart, this results in slowing of sino atrial (SA) rate and atrio ventricular (AV) conduction [3].

2. Case Description

We hereby report a case with myasthenia gravis who developed signs and symptoms of cardiovascular involvement. A 69 years old gentleman presented to ED

with one day history of left-sided chest pain sudden onset, dull aching pain, no alleviating and relieving factors, non radiating, no palpitations, no syncope, No diaphoresis, No nausea / vomiting and grade III shortness of breath associated with dry cough. HR of 67 bpm, BP 100/60 mmHg and Spo2 – 96% on room air were the arrival vitals. Sudden onset chest pain is a major red flag presentation to Emergency department (ED). The nature of chest pain, location, aggravating and relieving factors and radiation of the pain is highly important to distinguish between the urgent, emergent and non emergent causes of chest pain. Coexisting shortness of breath and dynamic ECG changes point towards acute coronary syndrome as the most probable cause. ECGs (fig 1, 2 and 3) were recorded over next 2 minutes of ED stay. The initial normal sinus rhythm with broad QRS LBBB changed to type 2 AV BLOCK. A new onset LBBB in background of chest pain and SOB strongly pointed towards acute coronary syndrome (ACS). Fig 4 details the serum levels of cardiac biomarkers, all the markers were normal, serum electrolytes were normal.



Figure 1: Sinus rhythm, HR – 126 bpm
LAD

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QRS > 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)
 Poor R wave progression in the chest leads
 ECG showed LBBB



Figure 2: HR - 52 bpm,
 AV BLOCK: 2nd degree
 Mobitz II
 Narrow QRS complexes
 Deeply inverted T waves in V1- V3



Figure 3 -HR - 52 bpm,
 AV BLOCK : 2nd degree
 Mobitz II
 Deeply inverted T waves in V1- V3

TEST NR: 00070703
 TEST TIME: 6:18PM 19.11.19
 USER ID: 9999999999
 REF. RANGES: S.O.B.

	NORMAL	ABNORMAL
CKMB	0.0- 4.3	> 4.3
MYO	0.0- 107	> 107
TNI	0.00- 0.40	> 0.40
BNP	0.0- 100	> 100
DDIM	0.0- 400	> 400

S.O.B. DLN 10620
 PAT.ID RAMPCHARY

CKMB	< 1.0	ng/mL
MYO	60.5	ng/mL
TNI	<0.05	ng/mL
BNP	58.9	pg/mL
DDIM	400	ng/mL

INT. QC: PASS

Figure 4

Bed side echocardiography was performed. No regional wall motion abnormality was detected with good biventricular function. Past Medical history was suggestive of generalized form Myasthenia gravis under chronic treatment with pyridostigmine and wysolone with no prior history of coronary artery disease. He was also a diabetic and hypertensive on insulin and telmasartan

3. Discussion

Lbbb & it's significance- New onset LBBB is STEMI equivalent also seen in Aortic stenosis, Coronary artery disease, Hypertension, Dialated Cardiomyopathy, Anterior MI, Primary degenerative disease of the conducting system, Hyperkalemia. Pyridostigmine inhibits acetylcholinesterase in the synaptic cleft, thus slowing down the hydrolysis of acetylcholine at the post synaptic junction. This results in an increase in the amount of acetylcholine available for the already depleted acetylcholine receptors. Bradycardia and Heart Blocks secondary to muscarinic side effects is already reported in literature. Cardiac involvement in myasthenia gravis may take several forms, ranging from asymptomatic

ECG changes to ventricular tachycardia, myocarditis, conduction disorders, heart failure and sudden death [4–6]. In Myasthenia gravis decrease in the number of available nicotinic AchR at NMJ- defective neuromuscular transmission in skeletal muscles that manifests as muscle weakness. The dense cholinergic innervation of AV node make it more susceptible to conduction disturbance secondary to vagomimetic drugs, such as pyridostigmine. Long term therapy with pyridostigmine may be associated with potential side effects such as bradyarrhythmias, asystole and sinus arrest. Hyoscyamine, a muscarinic antagonist [7] is recommended as first line therapy. Persistence of bradyarrhythmias may warrant pacemaker placement.

4. Conclusion

Pyridostigmine induced AV block are commonly seen in ED. Emergency physicians should learn the skill of insertion of temporary transvenous pacemaker.

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