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Left Bundle Branch Blockade in Suspected Acute Myocardial Infarction

Nasyrova Zarina Akbarovna
DSc, Acting Assistant Professor, Department of Internal Medicine and Cardiology №2
Samarkand State Medical University, Samarkand, Uzbekistan

Ergashzoda Erkinjon Ergashevich
Resident of the master's program of the Department of Internal Medicine and Cardiology №2
Samarkand State Medical University, Samarkand, Uzbekistan

Abstract: The diagnosis of acute myocardial infarction (AMI) in the setting of left bundle branch block of the Hiss left bundle branch block (LBBB) is challenging for the clinician. Despite current guidelines for therapy, early reperfusion may not be appropriate for all patients with new or suspected new BLNPG because only a minority are due to acute myocardial infarction with true arterial occlusion. Current guidelines recommend that patients with probable myocardial infarction (MI) who have a new or suspected new BLNPG should be considered diagnostic for AMI and should receive early reperfusion therapy. Despite this recommendation, early reperfusion may not be appropriate for all patients with new BLNPG because only a minority are diagnosed with myocardial infarction. The prevalence of false-positive catheterization laboratory activation is frequent among patients with BLNPG, and a significant proportion of patients with BLNPG with AMI do not have a blocked culprit artery at catheterization. Careful clinical evaluation is essential in the diagnosis and management of patients with acute MI and BLNPG. Avoiding unnecessary burdens and risks resulting from early reperfusion therapy can have a significant impact, especially in a center with limited modality options.

Keywords: Left bundle branch blockade, reperfusion, American College of Cardiology, modified Sgarbossa criteria.

Introduction. BLNPG often alters ventricular depolarization, which can mask changes in the ST/T teeth and eliminate the pathological Q wave, which usually localizes the infarction area on the ECG in acute myocardial infarction (AMI). In the recommendations of ESC STEMI (2017), it is proposed to use 3 ECG criteria of Sgarbossa in the diagnosis of myocardial infarction in conditions of BPH. The criteria include: 1) concordant elevation of the ST segment ≥ 1 mm in the presence of a positive QRS complex, 2) depression of the ST segment ≥ 1 mm in leads V1, V2 or V3, and 3) discordant elevation of the ST segment with a ST/S ratio ≥ -0.25. All measurements are made from the PR segment, and the ST segment is measured from the J point. These criteria have an overall sensitivity of 91% and a specificity of 90%[1,13,15].

Meanwhile, current guidelines recommend that patients with probable myocardial infarction (MI) who have a new or suspected new BPH are considered diagnostic for AMI and should undergo early reperfusion therapy. Despite this recommendation, early reperfusion may not be appropriate for all patients with new BPH, since only a minority are diagnosed with myocardial infarction. The prevalence of false positive activation of the catheterization laboratory is common among patients with BPH, and a significant proportion of patients with BPH with AMI do not have a blocked culpable artery during catheterization. In fact, true acute BPH associated with MI is rare, but failure to perform adequate reperfusion therapy in this group is associated with a high risk of congestive heart failure and death (Wahab, 2017). These considerations create a dilemmatic solution in the diagnosis and management of patients with BPH and suspected AMI[2,4,8,12].

The consequences are even more complicated in centers that do not have immediate access to early reperfusion therapy, where false-positive fibrinolytic or catheterization activation imposes a burden on the referral process, unnecessary risks and, on the other hand, leads to huge costs[3,9,14,22].

Blockade of the left leg of the Gis bundle is one of the diseases of the conductive tissue caused by delay or blocking of conductivity within the branches of the Gis-Purkinje system, which leads to QRS anomalies. In adults, BPH is defined as (Kusumoto et al., 2018):

QRS duration is 120 ms

A wide jagged or indistinct R tooth in leads I, aVL, V5 and V6, as well as a random RS pattern in V5 and V6 associated with the offset transition of the QRS complex, there are no Q teeth in leads I, V5 and V6, but a narrow Q tooth may be present in the aVL lead in the absence of myocardial pathology During the peak of the R wave >60 ms in leads V5 and V6, but normal in leads V1, V2 and V3, when small initial R teeth can be distinguished in precardial leads;

The ST and T teeth are usually opposite in the QRS direction;

The blockade of the left leg of the Gis bundle affects ventricular repolarization, which leads to a deviation of the ST segment from the QRS direction, and in myocardial infarction, the blockade of the left leg of the Gis bundle masks ECG changes characteristic of myocardial infarction, such as changes in the ST/T teeth and a decrease in the Q wave[5,7,15,20,25].

Sgarbossa ECG criteria were introduced to help doctors diagnose myocardial infarction with blockage of the left leg of the Gis bundle, which were then modified by Smith and co-authors:

Concordant elevation of the ST segment by ≥ 1 mm in the presence of a positive QRS complex;

ST segment depression ≥ 1 mm in leads V1, V2 or V3;

Discordant ST segment elevation with a ST/S ratio ≥ -0.25.

All measurements are made from the PR segment, and the ST segment is measured from the J point. These criteria have an overall sensitivity of 91% and a specificity of 90%.

In the presented case, the patient presented with typical symptoms of angina pectoris in acute coronary syndrome (ACS). Although symptoms were observed for 6 days prior to admission, the patient described that chest pain had increased in the last 12 hours. Chest pain increased with activity, did not go away at rest, became more prolonged and deteriorated in quality, which indicates an acute destabilization (crescendo) of stable angina pectoris in ACS (Ibanez et al., 2018). A twelve-channel ECG at the initial treatment showed the presence of a blockade of the left leg of the Gis bundle with a pathological Q wave in the lower leads. Applying modified Sgarbossa criteria to indicate MI under conditions of blockage of the left leg of the Gis beam, 3 criteria were negative on this primary ECG. There was no concordant ST segment elevation, there was no ST segment depression, and the ST/S ratio was -0.125[10,11,17,24,30].

The American College of Cardiology (ACC) has proposed an algorithm in which an echocardiogram and serum cardiac biomarkers should be followed in a patient with MI and BLNPG (new or old) and negative criteria for Carbarbossa without heart failure or hemodynamic instability, whereas patients with heart failure or hemodynamic instability should undergo emergency percutaneous coronary intervention (PCI) or fibrinolysis regardless of the criteria of Sgarbossa (Beaty & Park, 2020)[32].

Kusumoto et al. (2018) in the AHA/ACC guidelines stated that several cohort studies have demonstrated an association between BPH in the development of coronary heart disease and heart failure. Damage to the left leg of the Gis-Purkinje bundle as a result of ischemia or fibrosis can lead to BLNPG in patients with myocardial damage in the past (Biti and Park, 2020). Other structural heart diseases that can cause chronic BPH by damaging the Gis-Purkinje system and its branches are left ventricular hypertrophy due to prolonged or uncontrolled hypertension, congestive heart failure leading to ventricular remodeling, or valvular heart disease and cardiomyopathy leading to dilation, fibrosis and progressive remodeling of the heart. Impaired conduction due to a degenerative disease

causing Lenegre disease or sclerosis and calcification of the cardiac skeleton (Lev's disease) can also lead to chronic BPH (Cai et al., 2013; Neeland et al., 2012)[33].

Anatomically, the left leg of the Gis bundle is a large and diffuse structure. BLNPG most often masks ECG changes in the anterior or anteroposteroid region involving a large infarction zone compared to any other localization. In this case, the possibility of a new lower MI causing BPH was less likely, since lower and posterior infarction are less likely to cause a new BPH (Parekh et al., 2014). Moreover, instead of being the result of a small or focal infarction zone, true new BLNPG in AMI is often caused either by a very proximal coronary occlusion affecting the septal perforating arteries feeding the proximal left leg of the Gis bundle, or by extensive myocardial infarction affecting most of the distal conduction system, including both bundles, resulting in acute damage. Thus, true patients with BPH associated with AMI will usually be hemodynamically unstable and associated with a very high mortality rate. Taking this into account, a new blockage of the left leg of the Gis bundle caused by IMpST is less likely in this case, and the diagnosis, as a rule, was as follows: STEMI with chronic blockage of the left leg of the Gis bundle caused by IMO[19].

According to a study by Neeland et al. (2012), less than half of all patients diagnosed with suspected ACS and BPH will eventually be diagnosed with AMI. Kontos et al. (2011) reported that MI was diagnosed in 29% of patients with BPH with no significant difference in the prevalence or size of MI among 3 groups: chronic BPH, new BPH, or suspected new BPH. Chang et al. An even lower incidence of AMI was reported in patients with new or suspected new BPH — 7.3%, and among these patients, only 19.2% had documented coronary heart disease by angiography, and 7.8% underwent coronary revascularization. In a more recent study, Pera et al. (2018) reported that new BLNPG was detected in 3.3% of patients with suspected STeMI who underwent PCI, and culpable lesion was less present (54.2%) in patients with new BLNPG compared to those who did not have it (83.3%). Farre et al. (2015) reported in a study where only 35.8% of patients with BPH underwent primary PCI thrombotic occlusion of the coronary artery compared to 85.8% of patients with STeMI. These studies suggest that the prevalence of true new BPH caused by AMI may be low in reality and, conversely, signaling a high prevalence of false positive activation of the catheterization laboratory. In fact, among patients with STeMI with new BPH, 44% had no culpable coronary occlusion during emergency angiography[9]. Patients with BPH and MI can have a wide range: a minority who have the equivalent of STEMI, patients with the equivalent of STEMI without STEMI who have myocardial necrosis but without a completely blocked vessel, or patients with acute decompensated heart failure with minor myocardial necrosis who are mistakenly classified as having MI. Thus, early reperfusion treatment may not be appropriate for all patients with new or suspected new BPH, and only in patients with the real equivalent of STEMI, urgent reperfusion treatment is really useful[4].

The current recommendation of early reperfusion for all patients with new or suspected BPH can have a big impact, especially in a center where immediate access to early reperfusion is not readily available. False catheterization leads to an increased risk of complications associated with the invasive procedure, which leads to longer hospital stays and higher costs. On the other hand, unnecessary fibrinolytic therapy may impose on patients the risks of bleeding and more serious complications in elderly patients, hypertension and other concomitant diseases. Therefore, it is very important to distinguish whether BLNPGS are classified as new "STEMI equivalents" or old ones resulting from a chronic process, and whether early reperfusion is more beneficial to a patient with typical ACS, since any decision-making therapy really strongly affects the outcome for the patient. This practice and application are even more important for the rural center, where the acuteness of clinical judgment will benefit the results for the patient, while preventing the burden and risks of the early reperfusion option[5].

Diagnosis of AMI in the context of BPH is a serious problem for clinicians, especially in rural areas where diagnostic capabilities are limited. Despite the recommendation of early reperfusion for patients with new or suspected new BLNPG with AMI, only a small number of patients have proven to have a blocked artery, indicating a high prevalence of false activation of the catheterization laboratory. Instead, chronic BPH represents a large proportion, which is often the result of an ischemic and

degenerative process due to structural heart disease. A thorough medical history from the present and past medical history, a physical examination taking into account hemodynamic stability and a thorough sequential ECG analysis would be the best approach to decision-making and management of patients with BPH and possible AMI in conditions of limited assistance. The acuteness of clinical judgment will benefit both patients and healthcare providers, while avoiding unnecessary burdens, risks and costs resulting from false reperfusion therapy.

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