Not all new Left Bundle Branch Block (LBBB) requires Cardiac Catheterization Laboratory Activation

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Abstract:
Presence of concomitant left bundle brunch block (LBBB) in patients presenting with possible acute myocardial infarction (AMI) showcases a unique diagnostic and therapeutic challenge. Current guidelines recommend that patients with new or presumed new LBBB undergo emergency reperfusion therapy. However, only a minority of patients prove to have occluded artery at cardiac catheterization. Alternative strategies have been proposed to triage patients to select those suitable for acute reperfusion therapy, invasive and non-invasive cardiac investigations. We report a case of a 65-year-old man with atypical chest pain and new onset left bundle branch block on his electrocardiogram (ECG), who was managed without emergency reperfusion therapy, but instead, with observation and subsequent outpatient non-invasive cardiac investigation.

Key Words: left bundle branch block, acute coronary syndrome, myocardial infaction, cardiac catheterization, Sgarbossa criteria

Introduction:
Between 1% and 9% of patients seeking evaluation for suspected acute myocardial infarction (AMI) have left bundle branch block (LBBB) (chronicity uncertain). Patients presenting with a new or presumed new LBBB, with suspected AMI present as an important diagnostic and management challenge (1,2). An electrocardiographic (ECG) diagnosis of ST-segment elevation AMI (STEMI) in these patients is difficult as altered ventricular depolarization in LBBB interfere with ST segment elevation analysis, due to ST-segment discordant changes which obscure or mimic ECG manifestations of STEMI. Complex algorithms created to assist diagnosis also do not always provide diagnostic certainty (3-6). LBBB is essentially a manifestation of disruption in the electrical conduction system of the heart. It has been noted in patients with cardiomyopathy as well as those without any known cardiac disease (2,6,7). Studies show that in patients with a clinical suspicion of ongoing myocardial ischemia, LBBB may suggest higher risk for mortality, sudden deaths, ischemic heart disease and congestive heart failure, compared to patients without a LBBB. This is because the left bundle branch is an anatomically large and diffuse structure, deeper in the myocardium than the right bundle branch, hence, a large insult leading to acute ischemic injury is typically involved. A LBBB has been considered to be a STEMI equivalent, and thought to require optimal management of urgently identifying coronary occlusion and promptly providing reperfusion therapy. LBBB can also arise in healthy hearts with no myocardial infarction, secondary to ischemic, structural heart disease or fibrotic conduction system, in conditions such as chronic ischemic heart disease, left ventricular hypertrophy, or cardiomyopathy. As such, LBBB also defines a subset of patients with chronic cardiac disease at high risk of morbidity and mortality when there is concomitant AMI (6-12). There are also observations of transient LBBB in some patients. This may be a step in the progression from a normal ECG to a chronic LBBB as has been suggested (2,7,8). It has also been observed that between 6% to 51% of all patients with suspected AMI and LBBB will ultimately be diagnosed with AMI based on
angiographic evidence. Although a significant proportion of patients will not have an occluded coronary artery at cardiac catheterization, previous guidelines recommended for LBBB to be an activating criterion for coronary angiography, in view of higher mortality in LBBB patients with AMI. A major disadvantage to this approach is overtreatment with a high false-positive catheterization laboratory activation rate among patients presenting with new-onset LBBB and exposure of a significant proportion of these patients to the risk of fibrinolytic therapy or invasive procedures (1,4,5,7-9).

The Case Report:

A 65-year-old Chinese gentleman, Mr. A, presented to the Emergency Department (ED) after routine health screening for the evaluation of his chest symptoms. He complained of a few months history of intermittent, localized left sided, sharp chest pain, radiating to left shoulder, with each episode lasting less than 10 minutes. This pain was worse with pressure over the area, but not on inspiration, and was relieved with rest. It was not progressively worsening with time, and was not associated with shortness of breath, diaphoresis, palpitations, syncope, nausea, or vomiting. The last episode of chest pain was four hours before arriving at the ED. He did not have any symptoms suggestive of acute decompensated heart failure, such as lower limb edema, reduced effort tolerance, orthopnea, or paroxysmal nocturnal dyspnea. Mr. A has a background of intermittent chest pain for the past 10 years, which he has never been formally evaluated for. However, this history was not consistent, as there were days when he could walk relatively long distances with no symptoms. He is a smoker of 50 pack-years, and has no other cardiovascular risk factors. He has a history of rectal cancer which had been resected more than 10 years ago. Mr. A is not on any medications, and does not have a family history of sudden cardiac deaths. The physical examination done was normal, as were his vital signs.

The first ECG done is shown in Fig 1, with normal sinus rhythm. He was asymptomatic during consultation and the doctor sent off blood investigations which included, renal panel, full blood count, HbA1C, lipid panel and a chest X-ray was done.

His 12 ECG was repeated 20 minutes after the first one and this is shown in Fig 2. This time a new LBBB was noted but the patient remained asymptomatic and vital signs were stable.
All the initial blood investigations came back normal. A bedside 2D echocardiography noted a grade 1 left ventricular diastolic dysfunction, with ejection fraction 64% and no regional wall motion abnormalities. He was on telemetric monitoring and it was noted that he had reverted to normal sinus rhythm again. Thus, another 12 lead ECG was done. (Fig 3).

Figure 3: The 12 lead ECG which was repeated when his telemetry recording was noted to have reverted back to normal sinus rhythm

The cardiac catheterization laboratory was not activated in view that Mr. A remained asymptomatic. He was admitted to the inpatient Cardiology ward, where he was monitored and had another two serial cardiac enzymes done, which were also normal (not elevated) After evaluation by an electrophysiologist specialist, Mr. A was discharged with a scheduled outpatient myocardial perfusion imaging scan and Holter monitoring.

Discussion:

Currently, new guidelines no longer recommend treating new or presumably new LBBB as a STEMI equivalent with planned emergent reperfusion therapy. ACCF/AHA 2013 STEMI guidelines suggest not to consider a new LBBB at presentation alone diagnostic of STEMI. ESC 2017 STEMI guidelines recognize that the presence of a new LBBB does not predict AMI, but likely, ongoing myocardial ischemia and LBBB should be managed in a way similar to STEMI patients with early reperfusion therapy in the appropriate patients. Historically, the Sgarbossa ST-segment concordance criteria is used to assess the probability of AMI in new-onset LBBB. The initial study noted that a Sgarbossa's score >3 has a specificity of 96% for AMI but a sensitivity of only 36%. However, a recent meta-analysis suggested sensitivity of only 20%. Other new algorithms have also been proposed in recent years which include non-invasive test to counter the potential to overtreat...
as according to ESC 2017 guidelines, and undertreat as according to ACCF/AHA 2013 guidelines. Cai et al. and Neeland et al. proposed a triage algorithm, suggesting for

i) patients who present with hemodynamic instability or acute heart failure to be triaged to percutaneous coronary intervention (PCI) or fibrinolytic reperfusion;

ii) hemodynamically stable patients with Sgarbossa score >3, ST/S ratio <−0.25 to be triaged to reperfusion therapy based on additional diagnostic tools including serial ECGs, serial troponin, and bedside echocardiogram;

iii) patients with Sgarbossa score <3 and ST/S ratio >0.25 to be also evaluated with the above mentioned additional investigation before deciding on whether they should receive angiography or non-invasive cardiac testing (8,9).

This triage algorithm was again used in another study by Wyant et al., which concluded that its use facilitates more accurate diagnosis, fewer complications, better resource allocation, and improved risk stratification for patients (13). Our patient, Mr. A, experienced atypical chest pain, with a new-onset LBBB seen on ECG. Mr. A’s Sgarbossa score was 0, ST/S ratio >0.25. Mr. A was also hemodynamically stable. The management of Mr. A took into account ACC/AHA 2013 and ESC 2017 STEMI guidelines, which suggest not to consider new-onset LBBB alone as diagnostic of AMI. In this case, the new triage algorithm by Cai et al. and Neeland et al. was applicable to evaluate Mr. A’s new onset LBBB (8,9). According to the algorithm, Mr. A was not managed with perfusion therapy, but serial ECGs, troponin levels, as well as a bedside echocardiogram were done. Since results were unremarkable, Mr. A is less likely to have a NSTEMI or STEMI, and he was scheduled for outpatient non-invasive cardiac investigations. Mr. A represents a significant group of patients who presents with new-onset LBBB, but do not actually have acute coronary occlusion (7). This case report is a positive validation of how a stable patient with new-onset LBBB and suspected AMI can be triaged using non-invasive investigations to conclude a low probability of STEMI, and subsequently, managed conservatively. This is as opposed to more invasive recommendations of present ESC 2017 STEMI guidelines, which do not advocate for use of other investigations to select patients for reperfusion therapy. On the other hand, the ACCF/AHA 2013 STEMI guidelines lead to undertreatment by failing to recognize patients with chronic LBBB who actually do have STEMI, delaying reperfusion and increasing morbidity due to under-treatment. Hence, we recommend that noninvasive diagnostic tools included in the proposed triage algorithm to be used to aid in accurately determining appropriate interventions for these patients. This will ensure that patients with the highest likelihood of STEMI will receive emergency perfusion therapy with little delay, whilst a more careful and judicious approach to rule out STEMI or NSTEMI and reduce overtreatment risks is taken for patients where clinical picture is less clear. Further research can be done to explore additional diagnostic and therapeutic strategies such as other cardiac biomarkers to predict AMI in the presence of new onset LBBB. A study by Wahab, M. suggests that ischemia modified albumin (IMA) can be used as an additional parameter to exclude cardiac ischemia in patients with LBBB (12). As more research is conducted on excluding AMI in new onset LBBB patients, the evaluation and ultimate management of patients with new onset LBBB can be modified and improved. More evidence can also be gathered for the proposed approach to suspected AMI with LBBB (13-18).

Conclusion:
Patients with suspected AMI and LBBB present as a unique diagnostic and therapeutic challenge. Current guideline recommendations do not account for the complexity and heterogeneity of this group of patients. Given the potential to reduce overtreatment (reduce rate of false positive catheterization laboratory activation, exposure to risk of fibrinolytic therapy and costs) and undertreatment (reduce mortality and morbidity), we recommend that the new triage algorithm to be taken into consideration to better evaluate new-onset LBBB with stable hemodynamics and no prominent ECG changes characteristic of STEMI (8, 9, 17-19).

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