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Case Report

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Acute myocardial infarction associated with right bundle branch block and changeable trifascicular block: a case report

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ABSTRACT

Rationale: Acute myocardial infarction in the presence of right bundle branch block.
Patient concerns: A 70-year-old, male heavy smoker presented with angina and hypertension.
Interventions: Electrocardiography, intravenous nitroglycerin infusion, intravenous streptokinase infusion.
Diagnosis: Acute myocardial infarction in the presence of with changeable trifascicular heart block.
Outcomes: Dramatic clinical improvement with electrocardiographic ST-segment (whether elevation or reciprocal ST-depression) resolution.
Lessons: Acute myocardial infarction may be associated right bundle branch block. Accompanied trifascicular heart block had pre-streptokinase left anterior fascicular block with left axis deviation and post-streptokinase left posterior fascicular block with right axis deviation.

1. Introduction

Right bundle branch block (RBBB) is defined as a prolongation of QRS duration ⩾120 ms, with an rsr', rsR', or rSR' pattern in the lead of V_1 or V_2 of right chest[1]. The electrocardiographic diagnosis of acute myocardial infarction (AMI) is very difficult if there are associated bundle branch block pattern[2]. RBBB, as a sequel of the acute anterior myocardial infarction (MI), is mostly linked to the proximal of left descending coronary artery (LAD) occlusion with affecting the septal arteries supplying the bundle branches[3,4] or the left main coronary artery[4]. RBBB is statistically present in about 6 percent of MIs[2]. Because of the more RBBB in anterior location of the right ventricle than in the left ventricle, stimulation of the right ventricular free wall can equalize the abnormal septal forces of anteroseptal MI[3]. The right bundle branch block traverses the interventricular septum across the cardiac apex. Indeed, damage of the interventricular septum and the RBB mostly occurs by acute anterior myocardial infarction or severe ischemia due to a very proximal left anterior descending artery occlusion[1]. However, most cases of anteroseptal myocardial infarction with classical RBBB associated with pathological Q-waves in right precordial leads (V_{1-3}) is due to delaying in activation of the right ventricle[5]. Left bundle branch block can mask ST-segment deviations because it can mask the repolarization phase changes or Q-waves, while RBBB can not[6]. In rare conditions, the abnormal Q-waves in the anteroseptal may be masked by early depolarization of the right ventricular free wall[5]. Despite the RBBB does not actually mask the repolarization phase or preexisting Q-waves, minor ST-segments elevation in the anterior leads (*i.e.*, V_1–V_4) may be missed due to

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the pseudonormalization of the negative T-waves[3]. Anyhow, in the cases of myocardial infarction, the RBBB occurs lonely or in conjunction with left anterior hemiblock (Left anterior fascicular block) or left posterior hemiblock (Left posterior fascicular block[4]. Up till now, the studies did not compare the consequence of new-RBBB and the older one[7]. However, newly-onset and chronic RBBB is an important independent risk factor for future adverse in-hospital serious events[6]. The RBBB can be classified according to the onset time, duration, and accompanied fascicular block[6]. RBBB is mostly the finding of large myocardial infarctions[8].

Interrogatively, the RBBB is not recorded as an indication for reperfusion therapy[8]. Interestingly, broadening QRS-duration with RBBB may indicate great damage for the conduction system including right and left bundles from ischemia or infarction yielding a high opportunity for early aggressive reperfusion or revascularization[1]. Certainly, the current European Society of Cardiology Guidelines suggests a primary percutaneous coronary intervention strategy when persistent ischemic symptoms and RBBB occur in patients, but the level of evidence is not high[6]. Undoubtedly, old studies of thrombolytics have revealed a reduction in infarct size, amelioration in late ventricular morphology and function, and decreasing in mortality[6,8]. Early ST-segment elevation restoring post-fibrinolytic therapy despite chronic RBBB is accompanied with reduced death rate[1].

Indeed, the presence of an RBBB in the association with acute anterior myocardial infarction is accompanied by a higher risk of sudden cardiac death on comparison with that of normal conduction[4]. Whatever, the worse prognosis for the cases of widening QRS-duration with RBBB mostly is due to great damage for the conduction system by ischemia or infarction[1]. Several studies regard the combination of AMI and BBB pose the poorer prognosis than the patients with only AMI[6,8]. Many studies had revealed a positive relationship between RBBB and all-cause mortality[4,9,10], while others never reported this association[11,12]. Regarding the blood supply of the RBB is mainly provided by LAD or the proximal septal perforator branch, the newly-onset RBBB may indicate the proximal occlusion of the LAD and large infarction with subsequent acute heart failure, complete heart block, serious arrhythmia, and a high mortality rate[6]. An anterior AMI with RBBB will increase the QRS-duration, which is accompanied by elevated 30-day mortality[1].

Trifascicular heart block is the combination of bifascicular block with first-degree heart block[13]. The most important causes of trifascicular heart block are coronary artery disease, anterior myocardial infarction, hypertension, congenital heart disease, primary degenerative disease of the conducting system such as Lenègre-Lev disease, aortic stenosis, drug toxicity such as digitalis and electrolytes imbalance such as hyperkalemia[13]. There are two categories of trifascicular heart block: the first one is incomplete trifascicular heart block; characterized with bifascicular block with first-degree heart block (most common one), bifascicular block with second-degree heart block, and RBBB with alternating left anterior fascicular block/left posterior fascicular block; The second one is complete trifascicular block; characterized with bifascicular block with third-degree AV block[13]. Asymptomatic bifascicular block with first-degree AV block is not an indication for pacing (class Ⅲ)[13].

2. Case report

A 70-year-old male heavy smoker presented with acute severe chest pain, associated with profuse sweating and headache. Informed consent has been signed by the patient. The patient smoked about 60 cigarettes per day for nearly 30 years. He denied the history of the same attack, cardiovascular diseases, drugs or any other special habits. Chest pain had become anginal, compressible, and progressive. Upon general physical examination, the patient was anxious, severe sweaty, with cold extremities, a regular heart rate of 80 bpm, blood pressure of 160/110 mmHg, respiratory rate of 22 bpm, the temperature of 36.7 ℃, and pulse oximeter of O_2 saturation of 95%. No more relevant clinical data were noted during the clinical examination. He was admitted into the ICU due to anginal chest pain. Urgent ECG was done, which showed complete RBBB, an anteroseptal (V_{1-3}) and high lateral (Ⅰ, aVL) acute ST-segment elevation myocardial infarction and reciprocal ST-depression changes in leads (Ⅱ, Ⅲ, aVF), prolonged PR-interval, sporadic uniformed PVCs (V_{4-6}), left axis deviation, (V_{1-3}) (Figure 1A, B), and sinus arrhythmia (Figure 1A). The case was initially managed as an acute anteroseptal and lateral ST-elevation myocardial infarction in RBBB with a changeable trifascicular block. Sublingual 5-dinitrate tablet (5 mg), O_2 inhalation (100%, by nasal cannula, 5 L/min), pethidine HCL 100 mg (on intermittent Ⅳ doses), and intravenous nitroglycerin infusion (10 μg/min with intermittent titration) were given. Serial ECG tracings were taken. Aspirin 4 chewable oral tablet (75 mg), clopidogrel 4 oral tablet (75 mg), streptokinase IVI (1.5 million units over 60 min) were added after blood pressure control. Clinical improvement and electrocardiographic ST-segment (whether elevation or reciprocal ST-depression) resolution (Figure 2A-C) had happened. Serial ECG tracings were taken that showed complete right bundle-branch block, gradual resolution of anteroseptal (V_{1-3}) and high lateral (Ⅰ, aVL) acute ST-segment elevation myocardial infarction and normalization of reciprocal ST-depression changes in leads (Ⅱ, Ⅲ, aVF), prolonged PR-interval, right axis deviation, (V_{1-3}) (Figure 2A-C). The measured random blood sugar was 189 mg/dL. Troponin test was positive (385 ng/L). D-dimer was 1 029 ng/mL. Later echocardiography was mild anteroseptal and lateral hypokinesia with ejection fraction of 65%. No more workup was done. The patient was continued on captopril tablet (25 mg twice daily), aspirin tablet (75 mg/d), clopidogrel tablet (75 mg/d), nitroglycerin retard capsule (2.5 mg twice daily), and atorvastatin (40 mg/d) until discharged on the 5th day. Planning for both possible pacing and catheter revascularization was the future option.

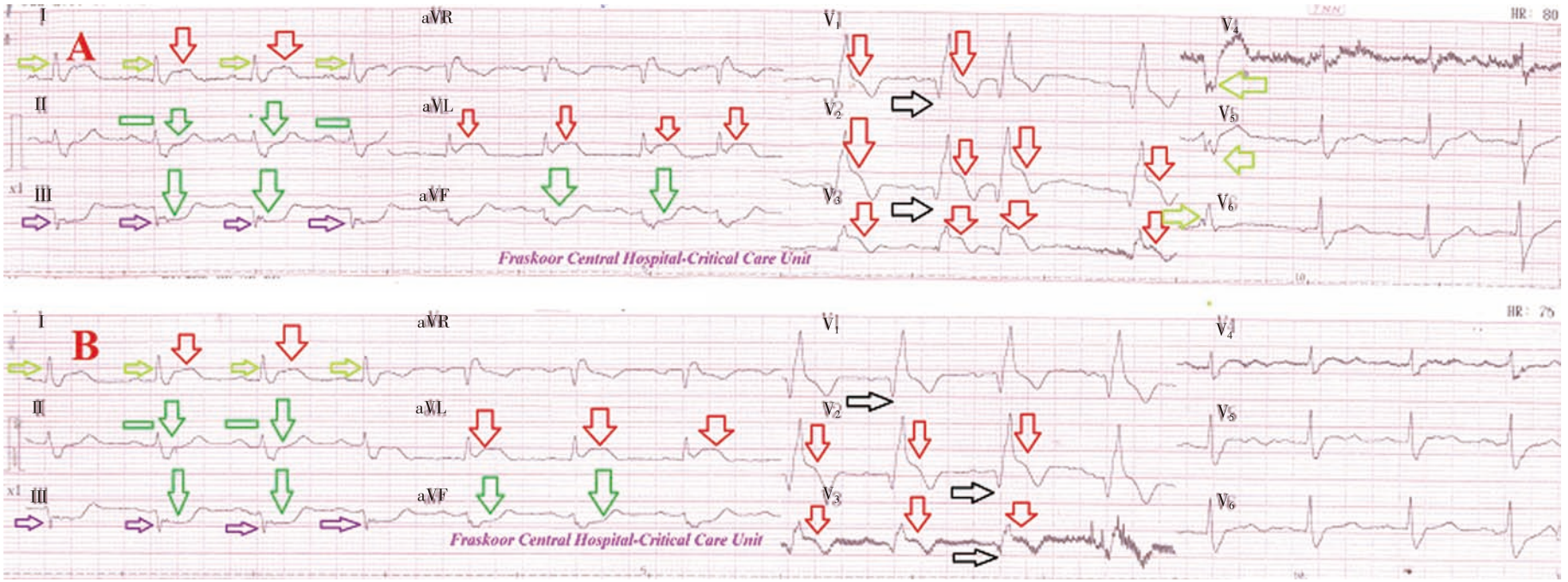

Figure 1. Serial ECG results; A: RBBB, ST-segment elevations in $V_{1\text{-}3}$, I, aVL (red arrows) with Q-wave (black arrows) in $V_{1\text{-}3}$ leads, reciprocal ST-segment depressions in II, III, aVF (green arrows) leads, LAD (lemon and purple arrows), prolonged PR-interval (green rectangles) lead II, sinus arrhythmia, and PVCs in $V_{1\text{-}3}$. B: like "A" but with no sinus arrhythmia and PVCs.

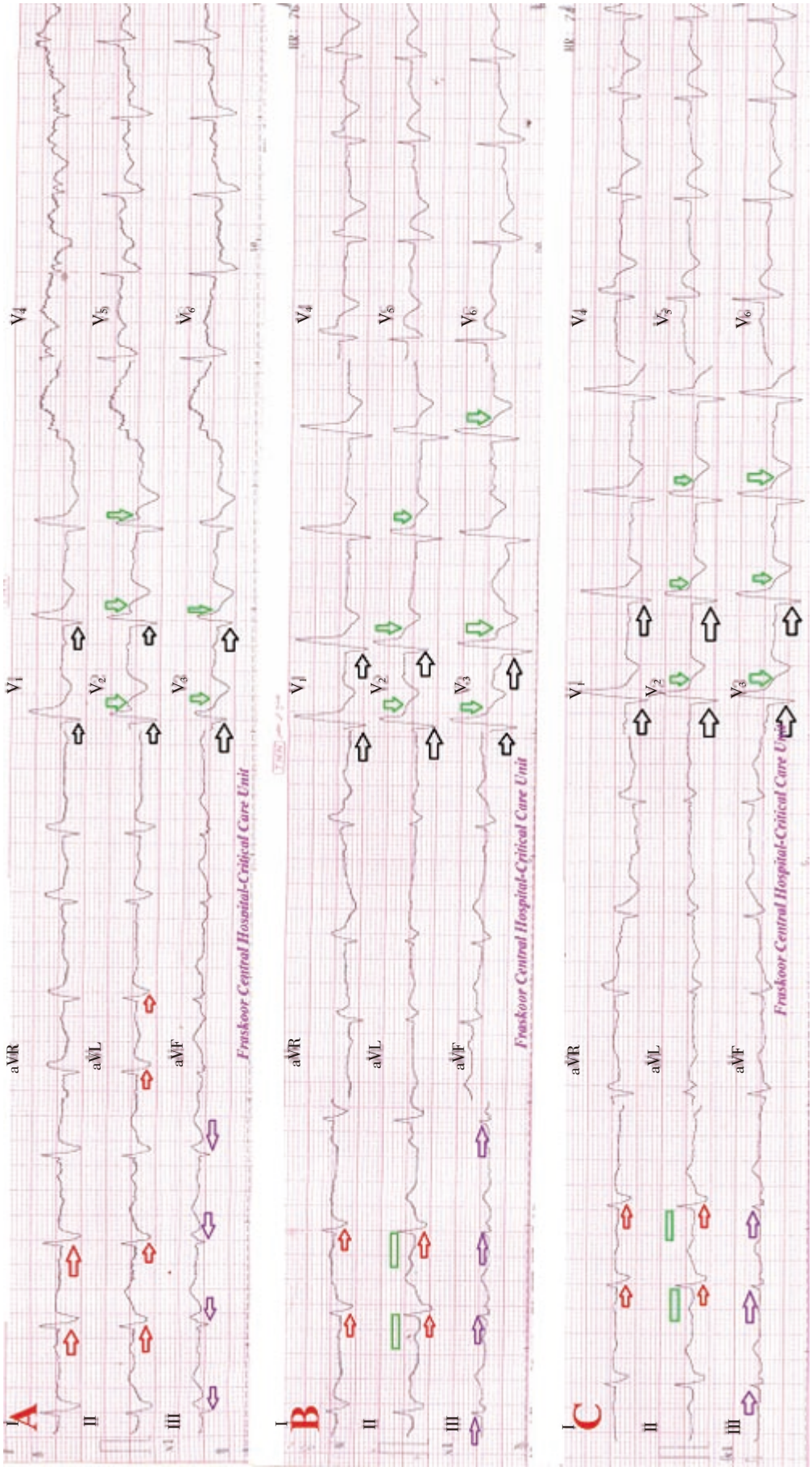

Figure 2. Serial ECG results. A: RBBB, ST-segment resolution in V_{1-3}, I, aVL (green arrows) with increasing the Q-wave (black arrows) in V_{1-3} leads, RAD (red and purple arrows), prolonged PR-interval (green rectangles) lead II. B: like "A" but with sinus and more increasing the Q-wave (black arrows). C: like "A" with more increasing the Q-wave (black arrows).

3. Discussion

Acute anteroseptal and lateral ST-elevation myocardial infarction in the presence of RBBB with hypertension were the initial presentations. Acute myocardial infarction was associated with changeable trifascicular heart block in the present case. Accompanied trifascicular heart block had pre-streptokinase left anterior fascicular block with left axis deviation and post-streptokinase left posterior fascicular block with right axis deviation. An existence of sporadic premature ventricular contractions (Figure 1 A) and sinus arrhythmia (Figure 1 A, 2 B) was benign and insignificant. Typical ischemic chest pain, ST-segment elevation, pathological Q-wave, and positive troponin test are the keys for diagnosis of acute myocardial infarction in this case. Positive d-dimer in the patient may be a marker for associated venous thromboembolism, aortic dissection, and may have a diagnostic role in myocardial infarction in a few studies[14].

The case was stable through the course of management. This was clearer after controlling of hypertension (nitroglycerin) and acute myocardial infarction with both antiplatelet (aspirin, clopidogrel) and thrombolytic (streptokinase). Proximal of LAD occlusion with the septal arteries are mostly attributed to anteroseptal infarction, but the circumflex artery is involving in high lateral infarction. Asymptomatic trifascicular block without complete heart block is not an indication for pacing (class Ⅲ)[13]. Acute pulmonary embolism, other titles of the acute coronary syndrome, aortic dissection, and acute pericarditis are implicated in the differential diagnosis.

The current case can't be compared with similar conditions for there is no similar or known cases with the same management. There are no other known limitations in the study, except for contraindications of streptokinase and nitroglycerin are possible limitations. It is recommended to widening the research in clearing the stabilizing effect of streptokinase in the cases of acute myocardial infarction with RBBB.

4. Conclusion

Acute myocardial infarction with RBBB has dramatic response to streptokinase. Accompanied trifascicular heart block had pre-streptokinase left anterior fascicular block with left axis deviation and post-streptokinase left posterior fascicular block with right axis deviation. This means a change of axis with thrombolytic therapy. Trifascicular heart block is an extreme association. It may be changeable and benign that does not indicate further intervention.

Conflict of interest statement

The author reports no conflict of interest.

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