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Case Report

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Electrocardiographic acute inferior myocardial infarction with right ventricle involvement due to acute thrombotic left anterior descending occlusion in a patient with atrial septal defect

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Abstract

Detecting the anatomic location of the lesion with the help of electrocardiography (ECG) is an important and time-saving decision in cases of acute ST-segment elevation myocardial infarction. However, it can be difficult in some patients with different coronary anatomies or underlying structural heart diseases. Here, we reported a 34-year-old male patient with an underlying atrial septal defect (ASD) who presented with acute inferior myocardial infarction with right ventricle (RV) involvement due to acute thrombotic left anterior descending artery occlusion.

Keywords: Electrocardiography, acute coronary syndrome, atrial septal defect

INTRODUCTION

The diagnosis of acute ST elevation myocardial infarction (STEMI) needs a time-sensitive approach based on symptoms and electrocardiography (ECG) findings.[1] Classical expressions of ST elevations in groups over inferior (II, III, aVF), high lateral (I, aVL), precordial (V1-6), posterior (V7-9) or right precordial (V3R-V6R) leads along with new-onset, ongoing angina may easily suggest acute STEMI.[1,2] However, it is not easy to make a diagnosis in case of bundle branch block, pacemaker rhythm, underlying structural heart disease, or anatomically-dislocated heart.[1-3] Additionally, the localization of a culprit artery via looking at the ECG findings can be time-saving in case of unstable patients, but correlation of culprit artery and ECG findings cannot be perfect always, especially in the presence of underlying ECG changes. Thus, reporting this kind of "out-of-standard" example may have potential benefit for the physicians in their clinical judgment. Here, we reported a 34-year-old male patient with an underlying atrial septal defect (ASD) who presented with acute inferior myocardial infarction with right ventricle (RV) involvement due to acute thrombotic left anterior descending occlusion.

CASE

A 34-year-old male patient without chronic illnesses was brought to the emergency department due to approximately 150 minutes of ongoing central chest pain radiating towards the back with a hotline ECG showing acute inferior STEMI with RV involvement (ST elevation over III, aVF, V1-2, V4R-V6R and ST depression over I, aVL, and V4-6). Other findings of the ECG included right ventricle hypertrophy without bundle branch block (**Figure 1A, 1B**).

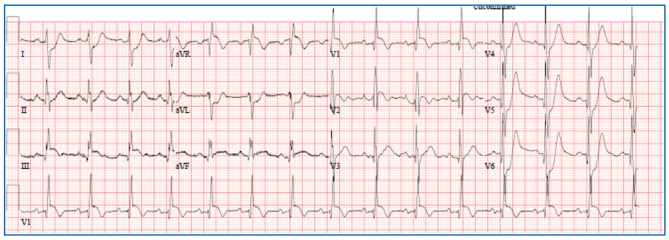

Figure 1A. Standart twelve-lead electrocardiography of the patient at the first medical contact

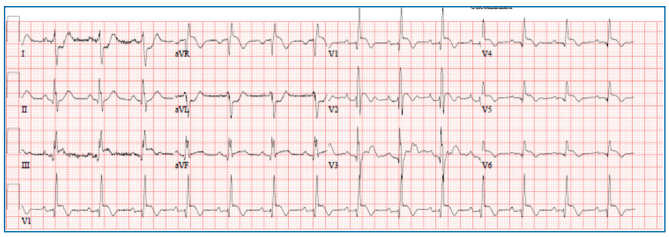

Figure 1B. The right-sided precordial leads (V3R, V4R, V5R, V6R) of the patient at the first medical contact

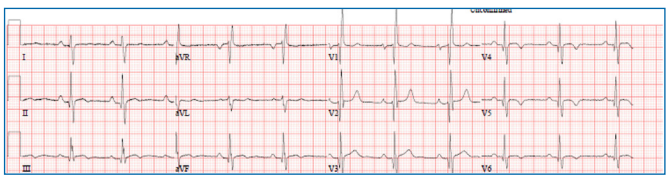

Figure 1C. 12-lead electrocardiography of the patient after the discharge

On arrival, physical examination showed a blood pressure of 102/83 mmHg, a heart rate of 89 bpm with oxygen saturation of 98% at room air, and a soft middiastolic murmur at the upper left sternal border without any crackles or wheezing at chest examination. The bedside echo examination revealed the left ventricular ejection fraction (LVEF) was approximately 40% with a dilated RV chamber without valvular stenosis, or aortic dilation, or pericardial effusion. The patient was transferred to the cath lab promptly. The coronary angiography (CAG) showed normal left circumflex and normal right coronary artery but 100% thrombotic occlusion at the proximal part of the left anterior descending coronary artery (LAD). Primary percutaneous coronary intervention (PCI) was performed to the proximal LAD with a 3.5 mm × 20 mm drug eluting stent following predilation without any complication (**Figure 2A, 2B,** and **2C** respectively) and received guideline-based post-PCI care.

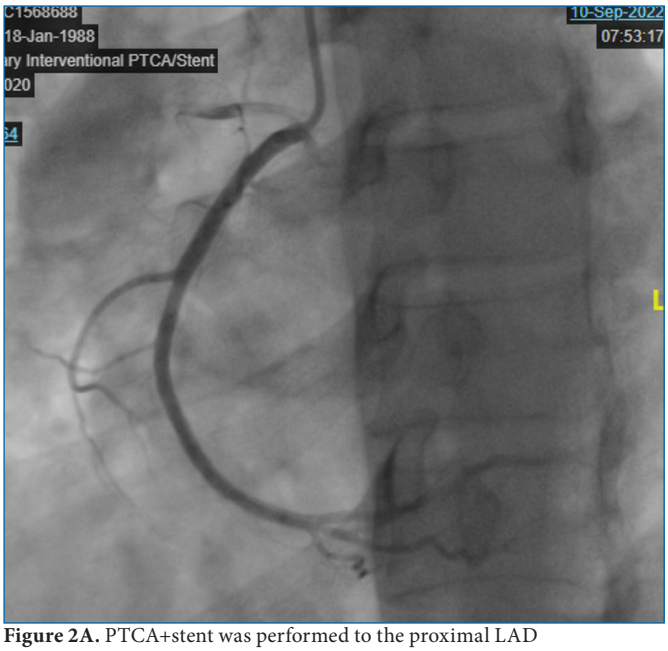

Figure 2A. PTCA+stent was performed to the proximal LAD

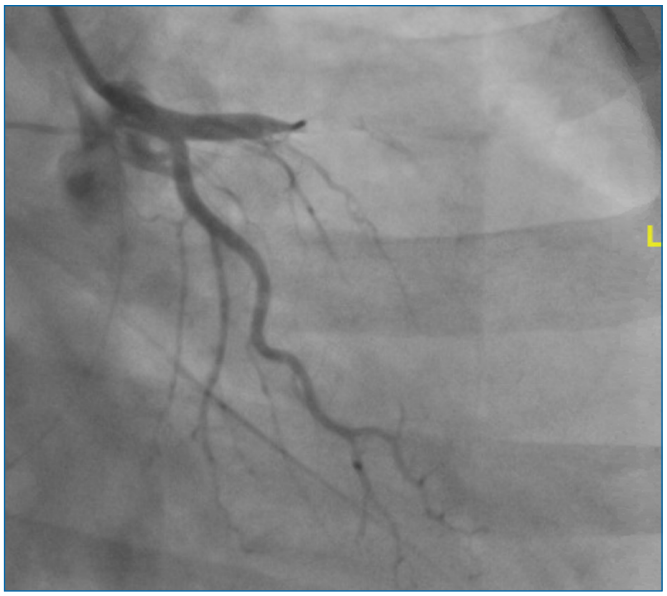

Figure 2B. After PTCA+stent was performed coronary blood flow in LAD

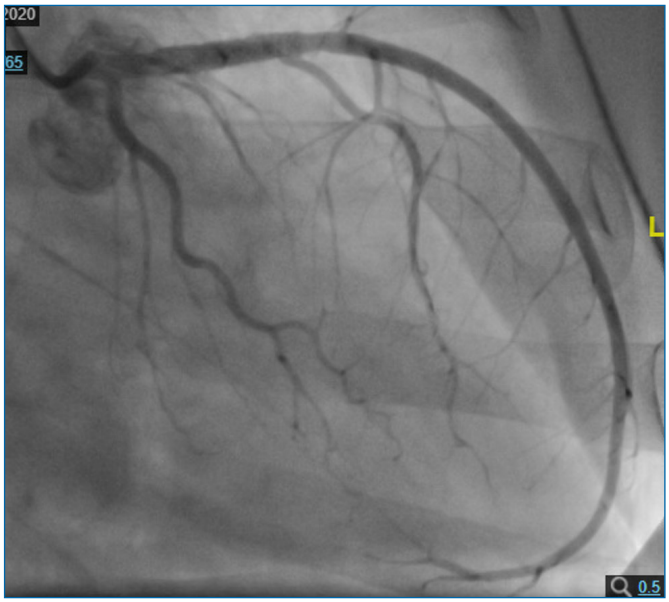

Figure 2C. After PTCA+stent was performed completely coronary blood flow open in LAD

A chest X-ray showed cardiomegaly along with prominent pulmonary vasculature (**Figure 3**). A detailed echocardiographic exam showed LVEF of 42% with regional wall motion abnormalities over the apex and anterior septal segments, a severely dilated RV with normal RV ejection fraction, moderately dilated right atrium, and large secundum ASD with diameter of 3.1 cm (**Figure 4**). RV systolic pressure was 45 mmHg with mild tricuspid regurgitation. In laboratory findings, high-sensitivity troponin T levels were elevated (206 ng/L on admission, 17442 ng/L at peak level six hours after the primary PCI, and 8014 ng/L 24 hours after the primary PCI respectively, with reference values of troponin T of 0-14 ng/L) under normal renal function. The patient had a total cholesterol level of 3.0 mmol/L, LDL of 1.9 mmol/L, triglycerides of 0.8 mmol/L, and HDL of 0.8 mmol/L. His lactate level was 2.6 at arrival (normal range: 0.36 to 1.60 mmol/L). The patient remained clinically stable throughout hospital course and discharged safely. He refused to perform transosephageal echocardiography or ASD closure at the follow-up. ECG changes subsided one month after the discharge, showing T inversions over V4-6, II, III, aVF without any significant ST changes (**Figure 1C**).

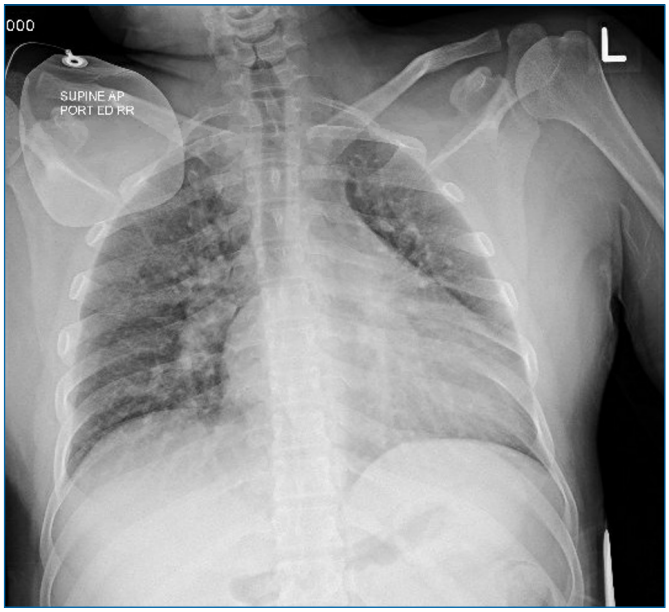

Figure 3. The chest Xray of the patient at admission

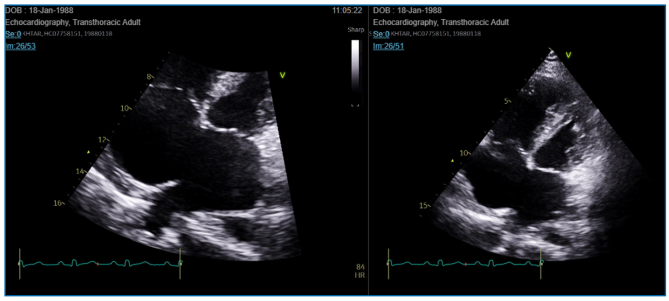

Figure 4. Echocardiographic evidence of secundum type atrial septal defekt at apical four-chamber views with significantly dilated right chambers

DISCUSSION

ST elevation myocardial infarction is a life-threatening condition, and its prompt diagnosis will significantly decrease the related mortality and morbidity. The ECG is the culprit test in the initial evaluation and the keystone in the diagnosis. The ECG will give important information about the culprit lesion and can decrease the-door-to-balloon time. However, some conditions can mask the ECG changes that make diagnosis and locating the culprit artery in the STEMI setting difficult. Here, we report a case of electrocardiographic acute inferior myocardial infarction with right ventricle involvement due to acute thrombotic LAD occlusion in a patient with ASD.

A wrap-around LAD is well-known STEMI that produces ST segment elevation over precordial leads along with ST segment elevation over inferior leads.[4] In our case, the patient had single-vessel disease, and the proximally-occluded LAD turned around the apex but without producing any ST elevation over lead V3-6. The main cause of this situation may be due to anatomic distortion of cardiac chambers by anatomic ASD-related RV changes. The distortion will change RV depolarization forces that overcome the effect of the left ventricular depolarization wave in cases of acute anterior STEMI.[5]

Bodi et al. showed that one-third of the right ventricle may be at risk in cases of extensive anterior STEMI, but the ECG changes of RV involvement will be masked due to dominant LV depolarization vectors in this setting. Following the prompt reperfusion, the infarct size became small enough to be negligible.[6] However, the ECG changes can be more prominent in case of severe right ventricle hypertrophy (RVH). The ECG changes that developed in our case can be explained by this fact.

CONCLUSION

The ECG changes in an acute STEMI setting can be challenging in cases of out-of-normal underlying cardiac states such as RVH. ST elevations in such cases may not be prominent enough to reflect the culprit artery.

ETHICAL DECLARATIONS

Informed Consent: All patients signed the free and informed consent form.

Referee Evaluation Process: Externally peer-reviewed.

Conflict of Interest Statement: The authors have no conflicts of interest to declare.

Financial Disclosure: The authors declared that this study has received no financial support.

Author Contributions: All of the authors declare that they have all participated in the design, execution, and analysis of the paper, and that they have approved the final version.

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