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Case Report

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A Case of ST Segment Elevation of Precordial Lead in ECG Caused by Occlusion of Right Ventricular Branch

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Abstract

St segment elevation in ECG V1-V3 (or V4) is usually caused by occlusion of the left coronary artery, and a few criminals' blood vessels come from the right coronary artery. In our report, the ST segment of lead V1-V4 was significantly elevated, but coronary angiography revealed a rare right ventricular branch occlusion. We have read the related literature and analyzed the causes of ST segment elevation in the original lead caused by occlusion of right ventricular branch, hoping to provide help for clinical decision-making.

Keywords: Acute myocardial infarction; Right ventricular branch, ST segment elevation of ECG

Established Facts

ST segment elevation in lead V1-V3 (or V4) is usually considered as myocardial infarction of anterior septal wall (anterior wall), and the culprit blood vessel is usually the anterior descending branch or its main branch.

Novel Insights

Acute right ventricular branch occlusion can lead to ST segment elevation in leads V 1-V4 of Electrocardiogram (ECG), which may be related to isolated right ventricular myocardial infarction caused by right ventricular branch occlusion.

Introduction

Acute Myocardial Infarction (AMI) is a fatal disease caused by acute coronary artery occlusion, which leads to myocardial ischemic necrosis in blood supply areas. ECG has important guiding significance for the diagnosis of acute myocardial infarction. In general, experienced doctors can roughly determine the culprit vessels through the ST segment elevation leads. For example, the elevation of the ST segment in lead V1-V3 (or V4) is usually considered to be an anterior septal wall (anterior wall) myocardial infarction, and the culprit vessel is usually the anterior descending branch or its main branch. However, we observed a case of acute myocardial infarction with elevated ST segment in lead V1-V4. Coronary angiography showed occlusion at the opening of right ventricular branch, which is rare in clinical practice.

Case Presentation

The patient is a 66-year-old male with a history of hypertension and hyperlipidemia for 3 years. This time, he entered the chest pain center of our hospital for treatment because of "chest pain 3.5 hours". In the state of rest at home, the patient had sudden chest pain, located in the precordial area, showing compression-like pain, accompanied by sweating, dizziness and weakness of the extremities, and took 0.5 mg nitroglycerin tablets, but the symptoms persisted. On admission, blood pressure was measured at 122/74 mmhg, heart rate was 78 beats /min, jugular vein was not filled and enraged, lung breathing was clear, dry and wet rale was not heard, arrhythmia was not heard, no murmur and murmur were heard in each valve auscultation area, and there was no edema in both lower limbs. The first ECG **(Figure 1A)** after admission showed that the ST segment of lead V1-V4 and V3R-V5R was raised. The diagnosis of "acute anterior wall, right ventricular ST segment elevation myocardial infarction class I (Killip)". Emergency coronary angiography showed that **(Figure 2A and B)**: coronary artery showed right dominant type, localized stenosis of the middle part of the anterior descending branch was 50-60%, and the proximal diffuse stenosis

of the first diagonal branch (lumen diameter about 1.0 mm) and the second diagonal branch (lumen diameter about 2.0 mm) was about 90%. No obvious stenosis was found in the circumflex branch, and the most severe local stenosis in the middle part of the second obtuse marginal branch (lumen diameter about 1.0 mm) was about 90%; there was no obvious stenosis in the right coronary artery; and the forward blood flow of the three coronary vessels was TIMI3 grade. At that time, considering that the diagonal branch of stenosis was small, the forward blood flow reached TIMI level 3, and after consultation with the patients and their families, the drug balloon dilatation was abandoned. In the follow-up, we gave patient intensive antithrombotic therapy, including oral aspirin enteric-coated tablets 100 mg, once a day, ticagrelor tablets 90 mg, twice a day, atorvastatin calcium tablets 40 mg, once a day, and continuous intravenous drip of tirofiban hydrochloride (0.1 ug/kg/min). On the second day of admission, the reexamination of ECG showed that **(Figure 1B)** the ST segment of lead V1-V4 and V3R-V5R decreased significantly, and the degree of chest pain was relieved, but there were still typical symptoms of angina pectoris. Combined with the patient's ECG and coronary angiography images, considering that the patient's second diagonal branch is severely narrowed, various stress factors that lead to increased work of the heart may lead to the blood supply and oxygen supply of the diagonal branch failing to meet the myocardial demand of the corresponding area, resulting in angina pectoris. However, the second coronary angiography showed that the lesions of the anterior descending branch, circumflex branch and its branches were almost the same as before, the right coronary artery trunk in the same position was the same as before, and the occluded right ventricular branch vessels were recirculated. Comparing the two coronary angiography before and after, it was confirmed that the criminal vessel causing chest pain was the right ventricular branch occluded by opening, and the modified diagnosis was "Acute right ventricular st-segment elevation myocardial infarction cardiac function grade I (Killip)". Considering that the right ventricular branch of the patient is small (the lumen diameter is about 1.5 mm), the TIMI3 grade blood flow has been restored, there is no further revascularization of the right ventricular branch, and the second diagonal branch is not a criminal vessel, so further intervention is abandoned. In the follow-up, we gave patient standard secondary prophylaxis for coronary heart disease, including aspirin enteric-coated tablets 100mg, once a day, ticagrelor tablets 90 mg, twice a day, atorvastatin calcium tablets 40 mg, once a day, perindopril 4mg, once a day, metoprolol succinate

sustained-release tablets 95 mg, once a day, and trimetazidine hydrochloride 20 mg, three times a day. After treatment, the patient's condition improved and he was discharged from the hospital. Up to now, the patient had no angina pectoris.

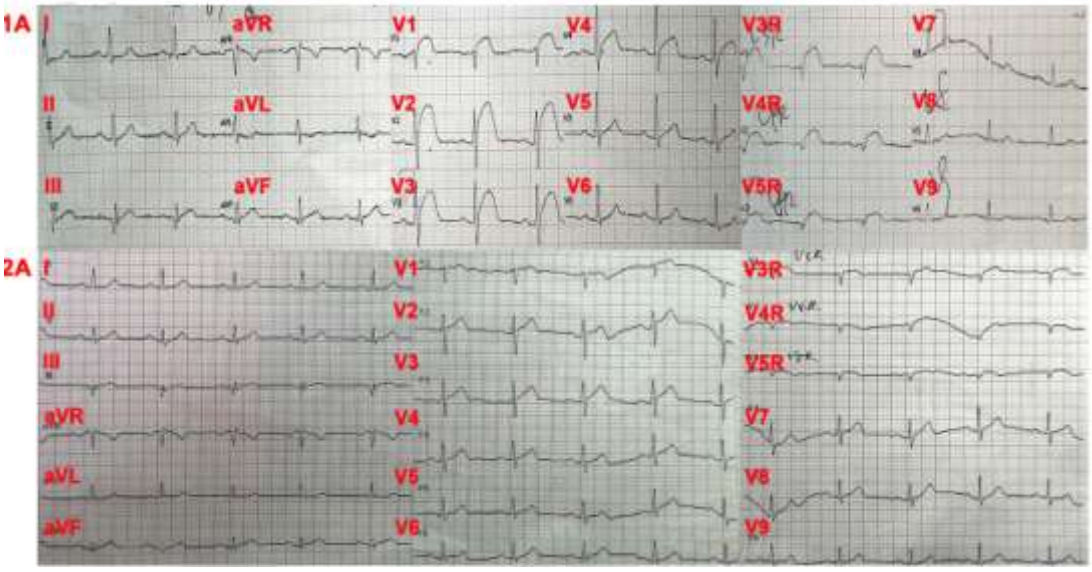

Figure 1: ECG. A) The first ECG of admission showed that the ST segment in lead V3Rmure V5R was significantly elevated. B) On the second day after admission, the ST segment of V1-V4 lead was at the baseline level, and the V3R-V4R was elevated about 0.05-0.1 MV.

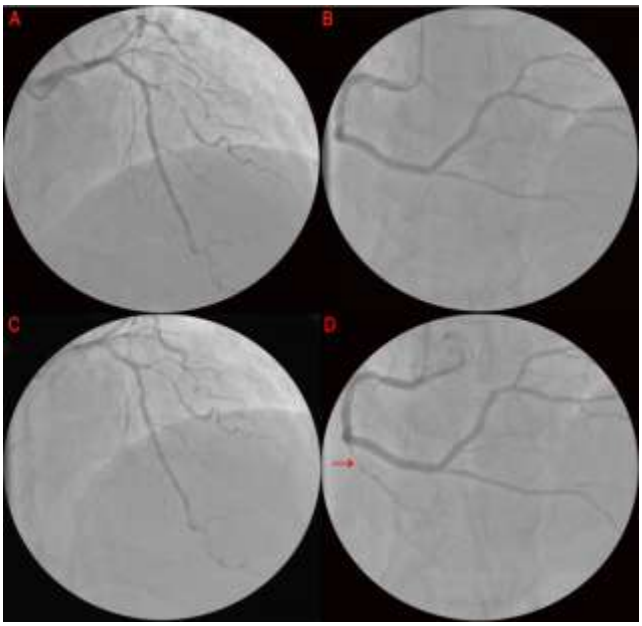

Figure 2: A, B) Emergency coronary angiography images on the first day of admission. C, D coronary angiography images on the second day of admission.

Discussion

AMI is a common and fatal disease in clinic. In China, despite the government's efforts to promote the construction of chest pain centers in recent years and the more standardized treatment of AMI in primary medical institutions, the mortality rate of AMI is still on the rise. The data show that the average mortality rate of AMI during hospitalization is 6.42% [1]. ECG is one of the important methods to diagnose acute ST-segment elevation myocardial infarction, and it has important reference significance for judging the infarct location and infarct-related vessels [2]. Usually, the ST segment elevation of leads V1-V3 (or V4) in the primordial region is considered as myocardial infarction in the anterior wall (or anterior wall), and the culprit vessels basically come from the left anterior descending branch and its branches, while the ST segment elevation of leads V 3R-V 5R indicates the right ventricular myocardial infarction, and the culprit vessels usually come from the right coronary artery. The patient was admitted to the hospital because of typical angina pectoris, and the ECG showed that leads V1-V4 and V3R-V5R were elevated. So, before operation, we thought that the right coronary artery or superior left anterior descending branch was very likely to be occluded. However, the results of the subsequent emergency angiography were surprising. The patients' right coronary artery and left anterior descending branch were not obviously narrowed, while the severely narrowed first diagonal branch and second diagonal branch did not dominate the right ventricle. Moreover, clinically, it is not common that the ST segment elevation of the primordial lead was caused by the acute occlusion of the diagonal branch. More importantly, the blood flow in front of the oblique branch of our patients is TIMI 3, which couldn't explain the continuous elevation of the ST segment in the wide leads of ECG. Therefore, it was basically excluded that the oblique branch was a criminal blood vessel. During the follow-up, we chose conservative treatment with drugs, but the patient still had typical angina pectoris. After excluding non-coronary factors such as aortic dissection, pulmonary embolism, pericarditis, coronary angiography was selected again, and the blood supply of diagonal branches was planned to be reconstructed. More surprisingly, during the second coronary angiography, the operator found the developed right ventricular branch in the same position and considered intensive antithrombotic therapy leading to thrombolysis at the opening of the right ventricular branch. Combined with the dynamic changes of ECG and coronary angiography images before and after, it was finally confirmed that the occlusion of right ventricular branch is the cause of ST segment elevation in leads V 1-V 4 and V 3R-V 5R of ECG. This extensive elevation of the ST segment of the precordial lead caused by right ventricular branch occlusion is clinically rare. At present, the mechanism of ST segment elevation of precordial leads caused by occlusion of right ventricular branch is not very clear, and the reason may be related to right ventricular

infarction. When the right ventricle is infarcted, the ejection capacity of the right ventricle decreases, the volume load of the right ventricle increases, and the pressure of the right ventricle increases, which leads to the acute dilatation of the right ventricle and the clockwise transposition of the heart. Most of the free wall of the right ventricle points forward, which may lead to the injury current in the precordial lead, showing the ST segment elevation of leads V1-V3. However, this kind of ECG usually appears in isolated right ventricular myocardial infarction. When the left ventricular inferior wall myocardial infarction is combined, because the human left ventricle is heavier than the right ventricle, the ECG vector is more advantageous. Therefore, the injury current generated from the inferior wall of the left ventricle may counteract the ST segment elevation of the precordial lead during the right ventricular myocardial infarction **[2-3]**. This is verified in animal models. Geft et al. **[4]** used dogs to build isolated right ventricular myocardial infarction model, and its ECG showed ST segment elevation of precordial leads, but when inferior myocardial infarction was combined, ST segment elevation of precordial leads couldn't be detected by ECG. Researchers believe that this is related to the thin right ventricular wall and the large mass of inferior left ventricular wall, which determines that the main injury current caused by ischemia is mainly inferior wall, which inhibits ECG changes caused by right ventricular ischemia. This patient suffered from isolated right ventricular myocardial infarction due to simple occlusion of right ventricular branch **[5,6]**. After operation, color Doppler echocardiography suggested that the right heart was enlarged to support this theory. Through the study of the clinical data of this patient, it is suggested that doctors should pay attention to isolated right ventricular myocardial infarction and obtain right ventricular lead electrocardiogram in time when they encounter ST segment elevation of the chest lead. When coronary angiography shows no clinically significant lesions in the main right coronary artery, multi-position projection is needed to find branches with occluded openings as much as possible. Recognizing this situation in time and taking correct treatment measures may change the outcome of the disease, reduce the occupation of medical resources and benefit patients.

Acknowledgement

None.

Statement of Ethics

All treatments received by the patient are agreed and signed by the patient and his family. The publication of the manuscript was approved by the patient.

Conflict of Interest Statement

All the authors declare no conflict of interest.

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Author Contributions

Li Liang: collected data, and was a major contributor in writing the manuscript; Jinhua Chen, Yuangui Zhang, Shanyin Wei, Yinzhi Song: collected data, supporting contribution; Zhicong Zeng: reviewed and guided the intellectual content of the manuscript. All authors read and approved the final manuscript.

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