Case Report

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Electrocardiographic findings of Wellens syndrome due to coronary artery–pulmonary artery fistula

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Abstract

A coronary artery fistula (CAF) is an abnormal connection between a coronary artery and any of the four cardiac chambers, the large vessels, or other vascular structures. Wellens syndrome is an ST-segment elevation myocardial infarction equivalent. Although both Wellens syndrome and CAFs have been reported in the literature, they have rarely been reported in the same patient. We herein report a case clinically diagnosed as Wellens syndrome by electrocardiography (ECG) findings; coronary angiography subsequently showed a fistula originating from the left anterior descending artery and draining into the pulmonary artery. The ECG findings then returned to normal after the fistula had been closed by controlled-release coils. These events confirmed that the abnormal ECG findings of Wellens syndrome were due to the CAF.

Keywords

Coronary artery fistula, Wellens syndrome, electrocardiography, ST-segment elevation myocardial infarction equivalent, coronary angiography, case report

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Introduction

Coronary artery fistulas (CAFs) have been described as a direct connection between a coronary artery and one of the cardiac chambers, large vessels, or other vascular structures.[1] This abnormality accounts for 0.27% to 0.40% of all congenital cardiac

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defects.[2] Most infants and children with CAFs are asymptomatic. Symptoms tend to occur more frequently in older age groups (>20 years of age); such patients may present with fatigue, dyspnea, angina, and congestive heart failure.[3]

First reported by de Zwaan et al.[4] in 1982, Wellens syndrome is a pre-infarction stage of severe stenosis or occlusion of the proximal left anterior descending artery (LAD) and may lead to extensive anterior wall myocardial infarction without timely intervention. Wellens syndrome was reported in 26% of patients with suspected coronary artery disease undergoing coronary angiography.[5] Therefore, electrocardiography (ECG) findings of Wellens syndrome can be used to detect acute coronary syndrome.[6] We herein describe a patient who had ECG findings of Wellens syndrome and was clinically diagnosed with Wellens syndrome, which was later confirmed to be due to a fistula originating from the LAD and draining into the pulmonary artery.

Case presentation

A 67-year-old man with a 4-day history of chest pain was admitted to the hospital. His medical history and physical examination findings were unremarkable. ECG on admission revealed T-wave inversion in leads V1 to V3 and biphasic T waves in lead V4 (Figure 1(a)). He was asymptomatic when the ECG examination was performed. His serum level of cardiac troponin T was $=0.013$ ng/mL (normal value, <0.10 ng/mL), creatine kinase level was

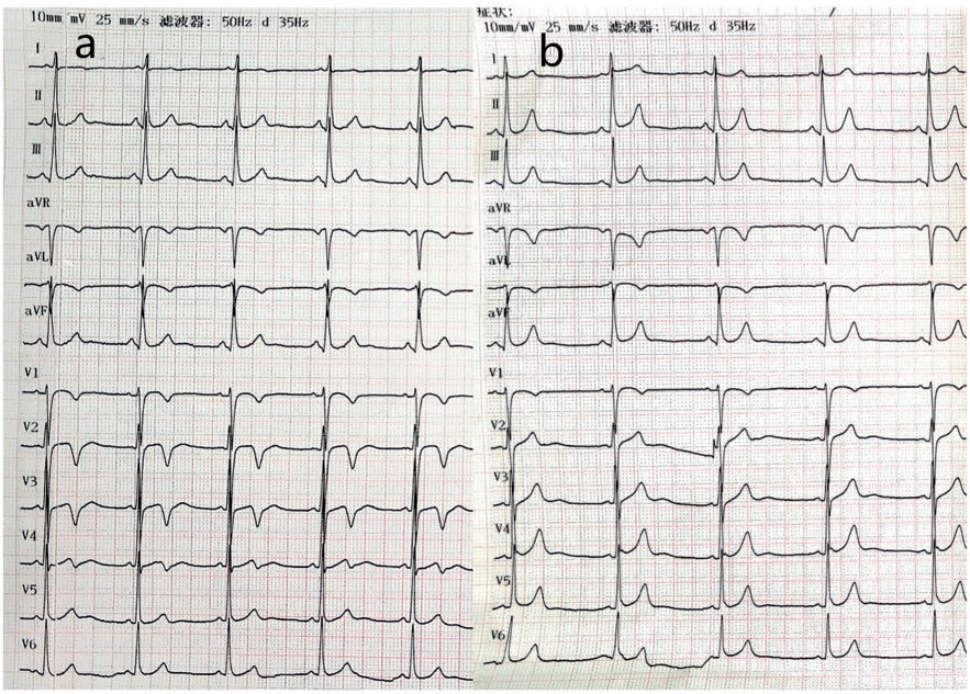

Figure 1. (a) Electrocardiogram at presentation with a regular sinus rhythm, heart rate of 64 beats/minute, normal axis, normal QRS interval, and normal QTc interval. Deep symmetric T-wave inversions were present in leads V1 to V3, and biphasic T waves were present in lead V4. (b) Electrocardiogram after the intervention showed sinus rhythm and improved T-wave inversions in the anteroseptal leads.

255 U/L (normal value, <310 U/L), and creatine kinase-MB level was 17 U/L (normal value, <25 U/L). Transthoracic echocardiography showed no obvious abnormalities. Three days later, the patient underwent coronary angiography, which showed a fistula originating from the LAD and draining into the pulmonary artery (Figure 2(a)). Two controlled-release coils (2.0×40 mm) were implanted into the dilated fistula. Coronary angiography showed that the fistula had been successfully closed by the controlled-release coils (Figure 2(b)), and postoperative ECG showed that the inverted or biphasic T waves had returned to normal (Figure 1(b)). After 3 months of follow-up, the patient had experienced no recurrence of chest pain.

Discussion

CAFs are rare congenital heart malformations caused by abnormal cardiovascular development during the embryonic period. The incidence of CAFs is very low; these fistulas are present in 0.002% of the general population and represents about 0.4% of all cardiac malformations. Most CAFs are small, and patients are asymptomatic because myocardial blood flow is not compromised. Small CAFs in children tend to grow with age. If untreated, fistulas cause clinical symptoms in 19% of patients aged <20 years and in 63% of older patients.[7,8] According to previous reports, the ECG findings are normal in about 50% of surgical patients; the remaining patients may show right or left ventricular hypertrophy caused by volume overload. Atrial fibrillation may be present in older patients with fistulas to the right atrium. An ischemic pattern is more likely if the coronary steal involves a major branch.[7] Because the incidence of CAFs is very low, however, CAFs cause only a few ECG changes; thus, CAFs are rarely suspected based only on ischemic ECG manifestations.

Wellens syndrome is identified by a specific ECG pattern in the absence of chest pain and is typically indicative of severe stenosis or occlusion of the LAD. Therefore, Wellens syndrome is commonly referred to as an ST-segment elevation myocardial infarction (STEMI) equivalent.[9] An ECG examination in a patient with Wellens

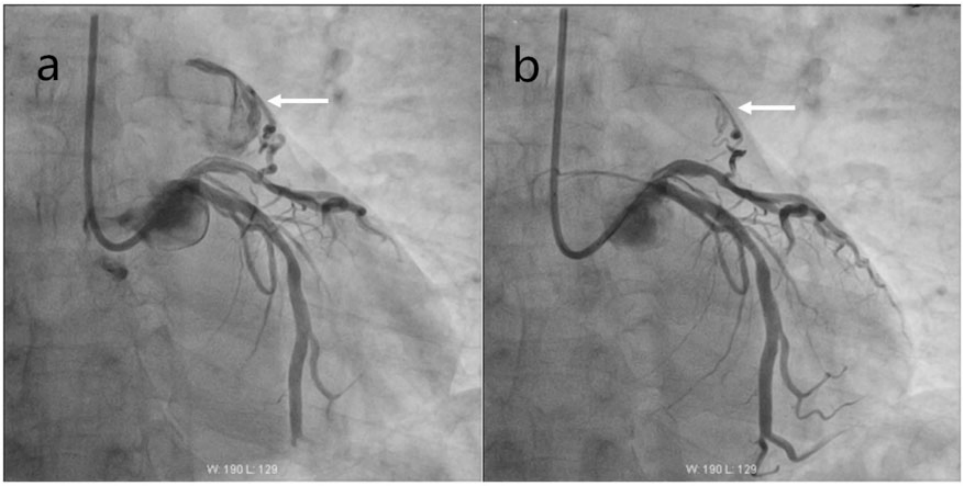

Figure 2. Left coronary angiography. (a) Right anterior oblique 30° + caudal 30° view showing a fistula originating from the left anterior descending artery and draining into the pulmonary artery. (b) Right anterior oblique 30° + caudal 30° view showing that the fistula has been closed by controlled-release coils.

syndrome can exhibit two patterns according to the T-wave changes in chest lead. Pattern A is characterized by biphasic T waves in leads V2 and V3 and accounts for 25% of cases, and Pattern B is characterized by symmetric and deeply inverted T waves in the chest leads and accounts for 75% of cases. The diagnostic criteria for Wellens syndrome include the presence of ECG Pattern A or Pattern B during a pain-free period plus a history of angina, little or no elevation of the ST segment, no Q waves in the chest leads, and normal or minimal elevation of cardiac enzymes. The presence of T-wave inversion has 69% sensitivity, 89% specificity, and an 86% positive predictive value for significant LAD occlusion. Even in challenging situations such as the presence of pre-existing left bundle branch block, ECG patterns consistent with Wellens syndrome can be used to detect acute coronary syndrome.[6]

In our patient, who had a 4-day history of angina, no ST-segment elevation was present, the chest leads had no Q waves, and the cardiac enzymes were normal. During the pain-free period, ECG showed symmetric and deeply inverted T waves in leads V2 and V3, consistent with Pattern B of Wellens syndrome. Additionally, the V4 lead had biphasic T waves, consistent with Pattern A of Wellens syndrome. Before concluding that our patient's T-wave inversions had resulted from anterior wall myocardial ischemia, we explored other etiologies. The patient had normal echocardiography findings and no history of hypertension; left ventricular hypertrophy and Takotsubo cardiomyopathy were excluded. Noncardiac pathologies, such as intracranial hemorrhage, pulmonary embolism, electrolyte abnormalities (hypokalemia), and medications (digitalis or cocaine toxicities) can also present with shallow T-wave inversion[10]; however, none of these were found in our patient. Thus, our patient was clinically diagnosed with Wellens syndrome. Coronary angiography subsequently showed a fistula originating from the LAD and draining into the pulmonary artery, and none of the three main coronary arteries showed stenosis of >50%. We considered that the patient's symptoms and ECG findings were associated with coronary artery steal caused by a CAF, and interventional therapy was successfully performed. Postoperative ECG showed that the biphasic and inverted T waves had returned to normal, and the patient experienced no recurrence of chest pain. These findings also indicate that the patient's symptoms and ECG findings were caused by coronary artery steal due to the CAF.

Conclusions

CAFs are rare coronary malformations. Wellens syndrome is a STEMI equivalent. However, a CAF that presents as Wellens syndrome is rare in the medical literature. Appropriate diagnostic testing in such patients should include coronary angiography.

Declaration of conflicting interest

The authors declare that there is no conflict of interest.

Ethics approval and consent to participate

This study complied with the Declaration of Helsinki and was approved by the Institutional Ethics Committee of the People's Hospital of Guangxi Zhuang Autonomous Region. Written informed consent was obtained from the patient.

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