Circulation

CASES AND TRACES

Acute Coronary Syndrome With Tall R Waves and Inverted T Waves in the Precordial Leads

An Ignored Entity

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ECG CHALLENGE

A 79-year-old man with diabetes presented after he had experienced chest pain and shortness of breath of approximately 4 hours duration. His medical history included myocardial infarction and an unknown type of coronary intervention that was performed abroad. The ECG performed during ongoing chest pain is presented in Figure 1. The high-sensitivity troponin T level exceeded five times the upper limit of normal.

What is the most likely diagnosis? What could be the cause of the ECG abnormalities? How should one treat this patient?

Please turn the page to read the diagnosis.

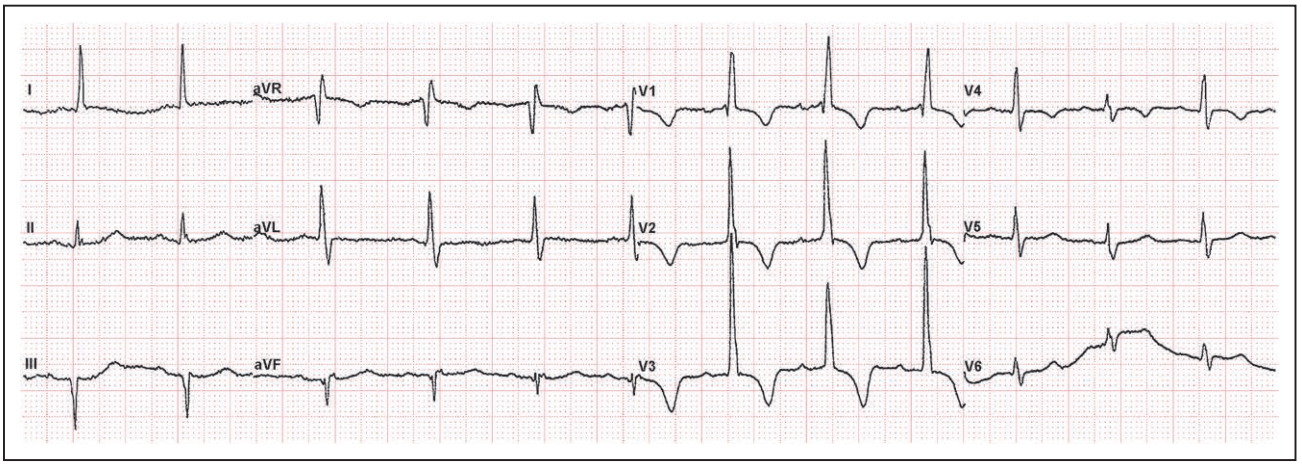

Figure 1. Twelve-lead ECG during angina.

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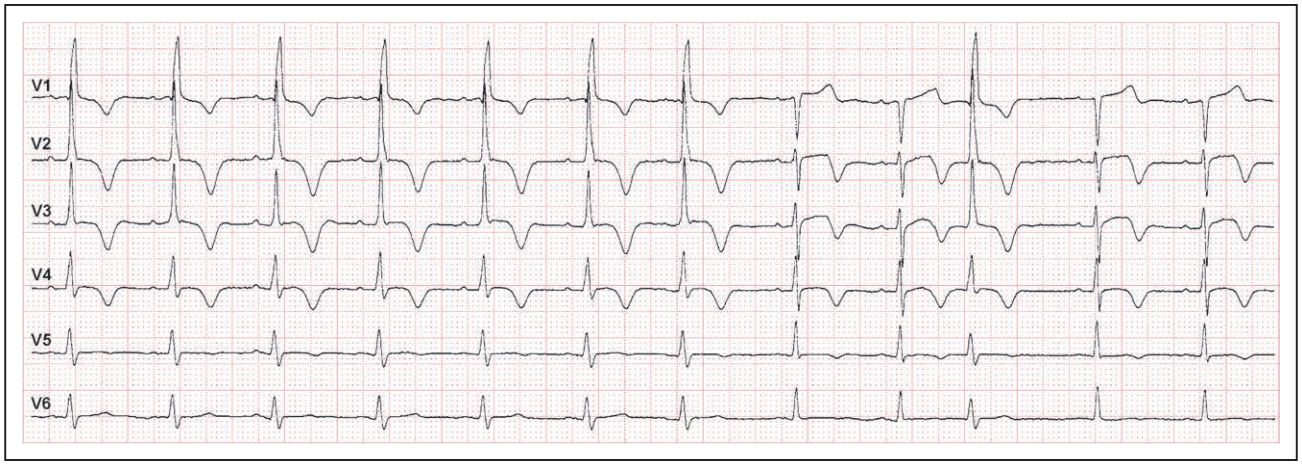

Figure 2. Rhythm strip with carotid sinus massage.
Note the increased RR interval after the seventh QRS complex with disappearance of high R waves in leads V1–V3, whereas negative T waves remain unchanged in leads V2–V4. After the ninth QRS complex, the shorter RR interval results in the initial QRS morphology, which clearly demonstrates acceleration dependent behavior.

RESPONSE TO ECG CHALLENGE

The ECG on presentation showed normal sinus rhythm and normal PR intervals (Figure 1). The QRS complexes in the precordial leads V1–V3 demonstrated tall R waves but no S waves. In lead V2, the height and width of R waves were 18 mm and 0.10 s, respectively. Monophasic R waves were followed by deeply inverted T waves in leads V1–V4. The differential diagnosis of narrow QRS complexes with tall R waves in the anterior chest leads (predominant anterior forces) includes postero-lateral myocardial infarction, right ventricular hypertrophy, septal hypertrophy, ventricular preexcitation, right bundle-branch block, dextroposition, misplaced precordial leads, and left septal fascicular block (LSFB).[1] The most important diagnostic criteria of the latter condition are tall R waves in leads V1 and V2 exceeding 5 mm and 15 mm, respectively. In addition, the PR interval cannot be shortened, the QRS duration must be normal or minimally prolonged (≤110 ms), and there cannot be slurring or notching of the tall R waves.[1]

To further clarify the clinical and ECG diagnosis, we performed carotid sinus massage. It demonstrated that a slight slowing of the sinus rate eliminated the tall R waves in the anterior chest leads. Of the listed causes of predominant anterior forces, only acceleration-dependent LSFB fits this picture (Figure 2).

Note that although a slower heart rate eliminated the tall anterior precordial R waves, the deep negative T waves, characteristic for the Wellens syndrome,[2] remained unchanged (Figure 2). The clinical diagnosis of non–ST-segment–elevation myocardial infarction with acceleration dependent LSFB and Wellens syndrome was established.

After guideline-based initial medical therapy, the patient became asymptomatic, the LSFB disappeared, and the T wave inversion in the anterior chest leads markedly improved (Figure 3). Coronary angiography performed 12 hours later revealed significant left main disease and critical proximal left anterior descending coronary artery (LAD) stenosis (Figure 4). The diagonal branch of the LAD and the proximal right coronary artery also demonstrated severe stenosis. The culprit LAD lesion was treated with balloon angioplasty, which was followed 1 week later by coronary artery bypass grafting.

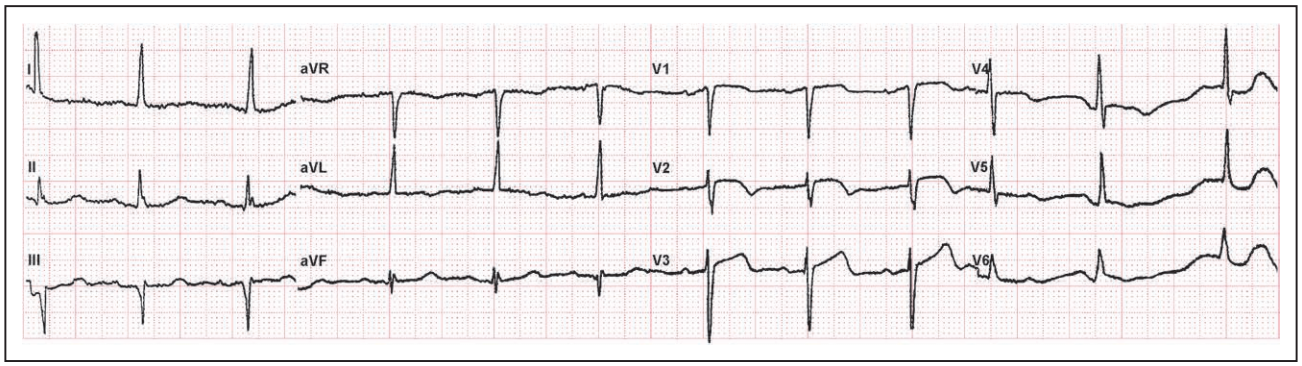

Figure 3. Twelve-lead ECG after angina.
The left septal fascicular block disappeared, and the T wave inversion in the anterior chest leads is markedly improved.

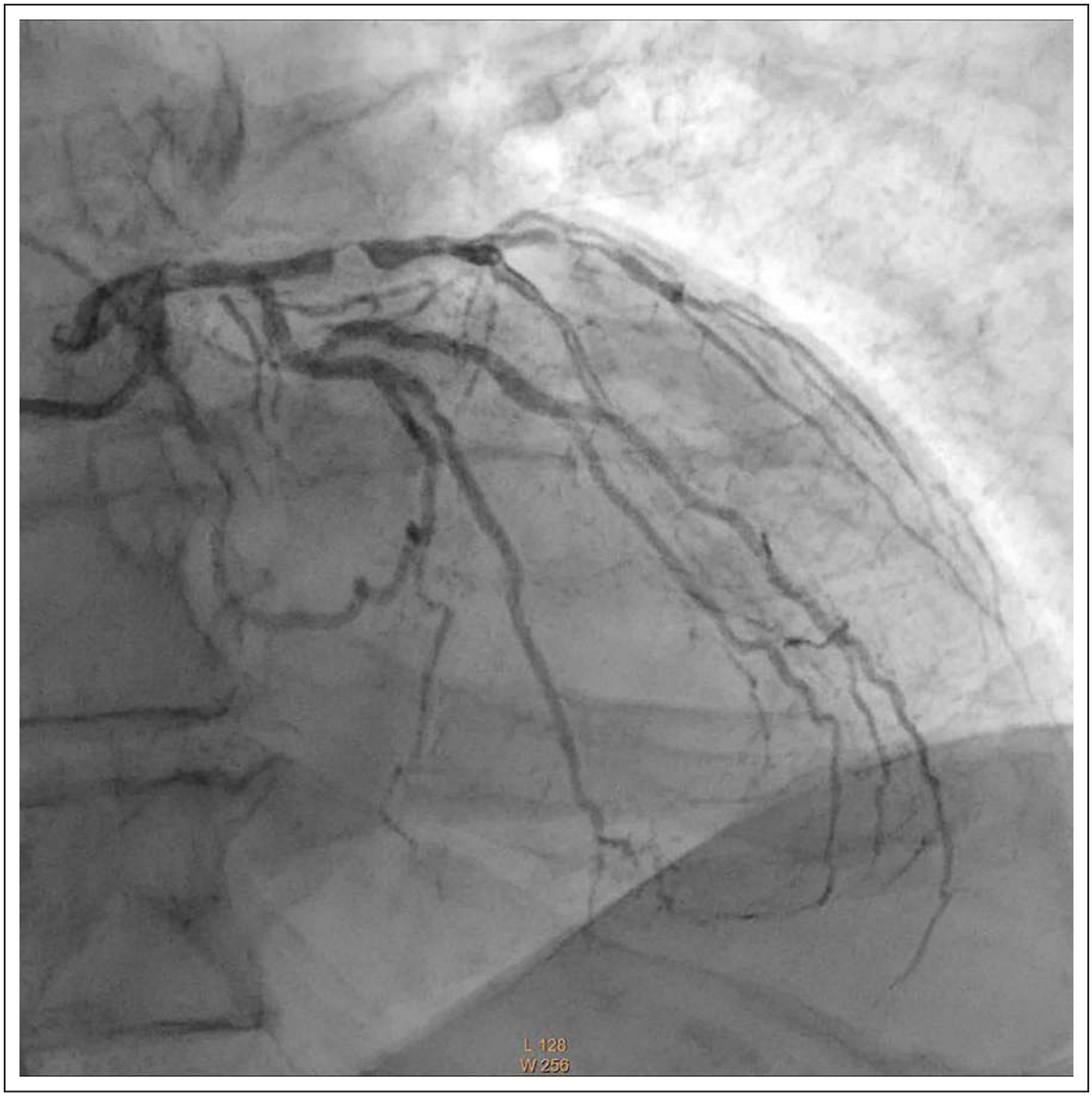

Figure 4. Angiographic view of left coronary artery.
Subtotal occlusion of proximal left anterior descending artery before the first septal perforator branch.

Growing evidence supports the concept of a trifascicular structure of left bundle branch. According to that, the left bundle branch branches into three fascicles: the left anterior, the left posterior, and the left septal fascicles. The fact that left septal fascicle has blood supplied exclusively by the septal branches of the LAD explains why critical lesion of proximal LAD is 1 of the 2 main causes of LSFB. The other is Chagas cardiomyopathy.[1] The ECGs during angina (Figures 1 and 2) also fulfill criteria for Wellens syndrome, which reflects critical stenosis in the proximal LAD and high risk of myocardial infarction and death without revascularization.[2]

Our case calls the interested readers' attention to severe septal ischemia–related LSFB, whose clinical significance is that it represents a possible extension of the Wellens syndrome.[3] Meanwhile, the differential diagnostic utility of carotid sinus massage in cases of suspected LSFB is also highlighted.

ARTICLE INFORMATION

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Disclosures

None.

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