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Case Report

Left Septal Fascicular Block without and with ST-Segment Elevation

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ABSTRACT

We present a case where left septal fascicular block precedes anterior STEMI. Our case calls the attention to severe ischaemia-related left septal fascicular block could be an early red flag sign for proximal left anterior descending coronary artery critical stenosis. In our case the causative mechanism of prominent anterior forces in the setting of an acute anterior ST-elevation is left septal fascicular block.

KEYWORDS

Acute Coronary Syndrome, Left Septal Fascicular Block, Wellens Syndrome, Anterior Myocardial Infarction

INTRODUCTION

A patient in their late 30s presented to the emergency department with intermittent compression-like chest pain. The patient had a history of psoriasis treated for two years with the biological agent ixekizumab. They also had untreated mild hypercholestremia. Physical examination showed a blood pressure of 135/80 mmHg, heart rate 75 beats/min, arterial oxygen saturation of 96%. Chest auscultation revealed normal breath sounds and normal heart sounds. The rest of the physical examination yielded normal results except for the typical skin changes associated with psoriasis. The admission ECG (Figure 1) was initially felt to display right bundle branch block with negative T-waves in the precordial leads. The D-dimer was slightly elevated at 0.8 ng/ml (normal < 0.5). CT angiography of the chest was negative for pulmonary embolism. The initial serum high sensitivity troponin T was in the gray zone at 28 ng/L (normal < 14).

Interpretation

The ECG on presentation revealed sinus rhythm with high and tall monophasic R-waves in leads V2 and V3 leads (Figure 1) accompanied by negative T-waves. The QRS complexes were narrow with a QRS width of 100 ms. The clinical picture and the ECG were highly suggestive of Wellens syndrome complicated by left septal fascicular block characterized by tall upright but narrow R-waves in the precordial leads [1,2].

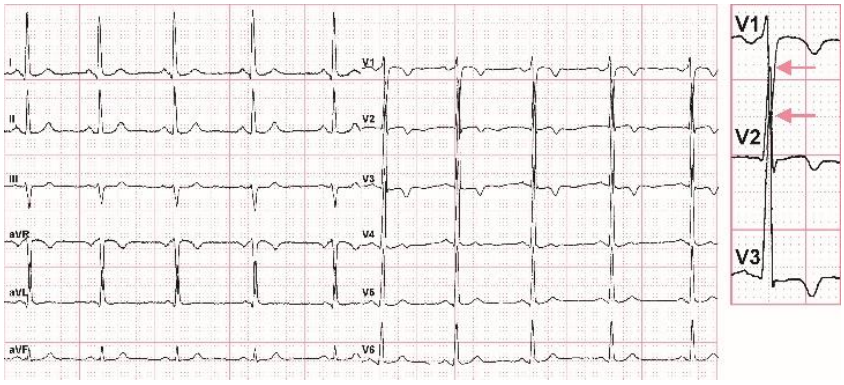

Figure 1: Twelve-lead electrocardiogram on presentation. Right, the same chest leads enlarged with arrows indicating the end of R-waves in leads V2 and V3.

Clinical Course

In the 8th hour of observation, in the morning, the patient again indicated intense chest pain. The ECG at this time revealed extensive ST-elevation accompanied by tall R-waves (Figure 2). Urgent coronary angiography showed critical obstruction of the left anterior descending coronary artery before the take-off of the septal perforating branch (Figure 3). Following successful coronary intervention, the ECG no longer displayed left septal fascicular block (Figure 4). The maximum creatinine kinase increased to 1307 U/L (normal < 170 U/L). The patient was discharged on hospital day 5 day with secondary prevention medications.

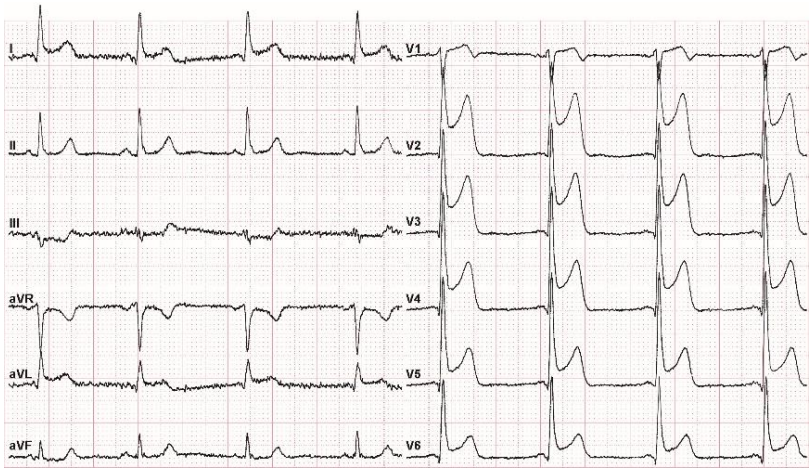

Figure 2. Twelve-lead ECG 8 hours later during severe, 7/10 chest pain. The precordial tall R-waves are now accompanied by marked ST-segment elevation.

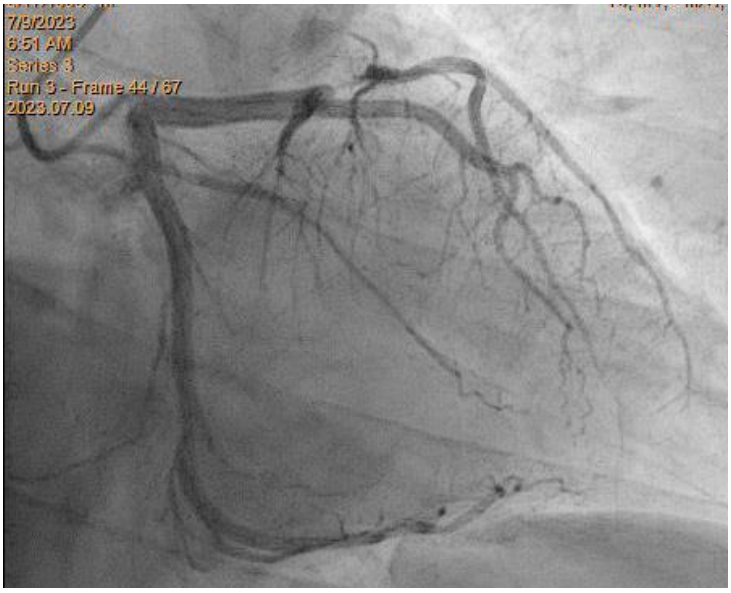

Figure 3. Angiographic view of the left coronary artery shows critical obstruction of the left anterior descending artery before the take-off of the first septal perforator branch.

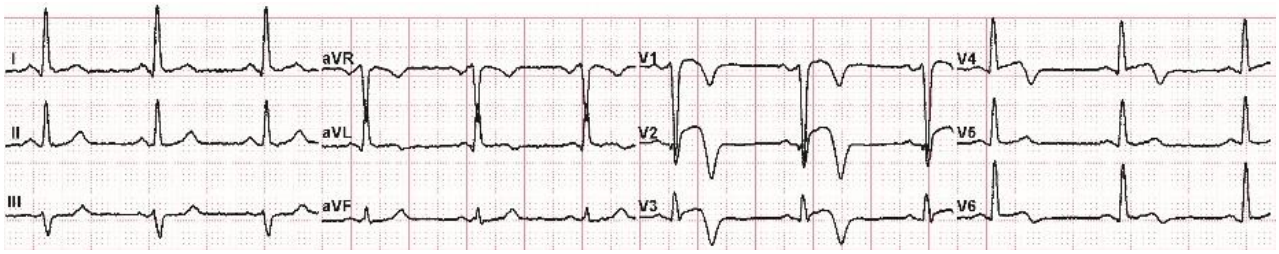

Figure 4. Twelve lead ECG following coronary intervention. The precordial high R-waves disappeared. In lead V2, there are now QS complexes; in V3, the R wave amplitude has decreased. There is minimal ST-elevation and deep negative T-waves in the precordial leads.

DISCUSSION

This case highlights that in a case of precordial high and narrow R-waves appearing alongside negative T-waves indicating Wellens syndrome, it is important to consider critical LAD stenosis proximal to the septal branch [3,4,5]. The case clearly demonstrates that the tall R-waves in extensive anterior infarction are not caused by the marked ST-elevation, but they are related to left septal fascicular block. This allows a more precise localization of the culprit lesion. The case highlights that the risk of MI is increased among patients with psoriasis, and remains unchanged during biologic therapy [6].

Our case calls the attention to severe ischaemia-related left septal fascicular block whose clinical significance is that it represents an extension of the Wellens syndrome and allows a more percise localization of the culprit lesion in MI and an early red flag for proximal critical LAD stenosis.

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