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Stepping into the Light: Defining Culprit Lesion in Non-ST Elevation Myocardial Infarction

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Cover Page Footnote

None

CASE REPORT

Stepping into the Light: Defining Culprit Lesion in Non-ST Elevation Myocardial Infarction

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Abstract

Identifying the infarct-related artery (IRA) in a non-ST-segment-elevation acute myocardial infarction (NSTEMI) can be very challenging, particularly in a hospital that cannot perform intracoronary imaging due to certain limitations. This is because, by angiography, most patients present with multivessel coronary artery disease (CAD), diffuse disease, or non-significant CAD. We present a case of a 60-year-old female patient presented with substernal chest pain and palpitations of 6 h duration. The first hospital contact 12-lead electrocardiogram (ECG) showed ventricular tachycardia (VT) with unstable hemodynamics, after stabilization patient was transported to the catheterization laboratory for immediate percutaneous coronary intervention (PCI). With a clue of VT morphology, post-converted ECG, and coronary angiography, the patient successfully underwent PCI in the left circumflex artery.

Keywords: NSTEMI, Acute coronary syndrome, Infark-related artery, Culprit, Revascularization, Eptifibatide

1. Introduction

Multivessel coronary artery disease (MVD) is commonly found in patients presenting with non-ST segment elevation myocardial infarction (NSTEMI), which approximately occurs in 40–70% of patients undergoing percutaneous coronary angiography [1]. The presence of MVD in NSTEMI is significantly associated with poorer clinical outcomes compared with single-vessel disease [2,3]. Terminology of culprit lesion or infarct-related artery (IRA) is used to determine the coronary lesion considered to be responsible for acute coronary syndrome (ACS). IRA is characterized by the presence of typical profiles of unstable plaque, including intraluminal filling defects consistent with thrombus, plaque ulceration, plaque irregularity, dissection, and flow impairment [4–6]. The correct identification of the IRA in the setting of NSTEMI has a significant impact on the invasive strategy. Using several modalities including ECGs and coronary angiograms (CA), intracoronary imaging can be useful in determining the IRA. However, challenges arise when certain conditions and limitations prevent hospitals from performing intracoronary imaging, as in our case. In this report, we delineate a case of NSTEMI patients with ventricular tachycardia, in which ECGs accompanied by CA might help identify the IRA.

2. Case report

A 60-year-old female presented with substernal chest pain and palpitations of 6 h duration. The first hospital contact 12-lead electrocardiogram (ECG) showed ventricular tachycardia (VT) with unstable hemodynamics (Fig. 1a), during preparation for cardioversion, the VT was autoconverted to supraventricular tachycardia (SVT) (Fig. 1b). The patient was undergoing electrical cardioversion several times using 50, 100, 150, and 200 J. Unfortunately, the rhythm did not convert until Amiodarone was given (Fig. 2). The patient was referred to our hospital with on going chest pain, hemodynamics were stable, no remarkable physical examination

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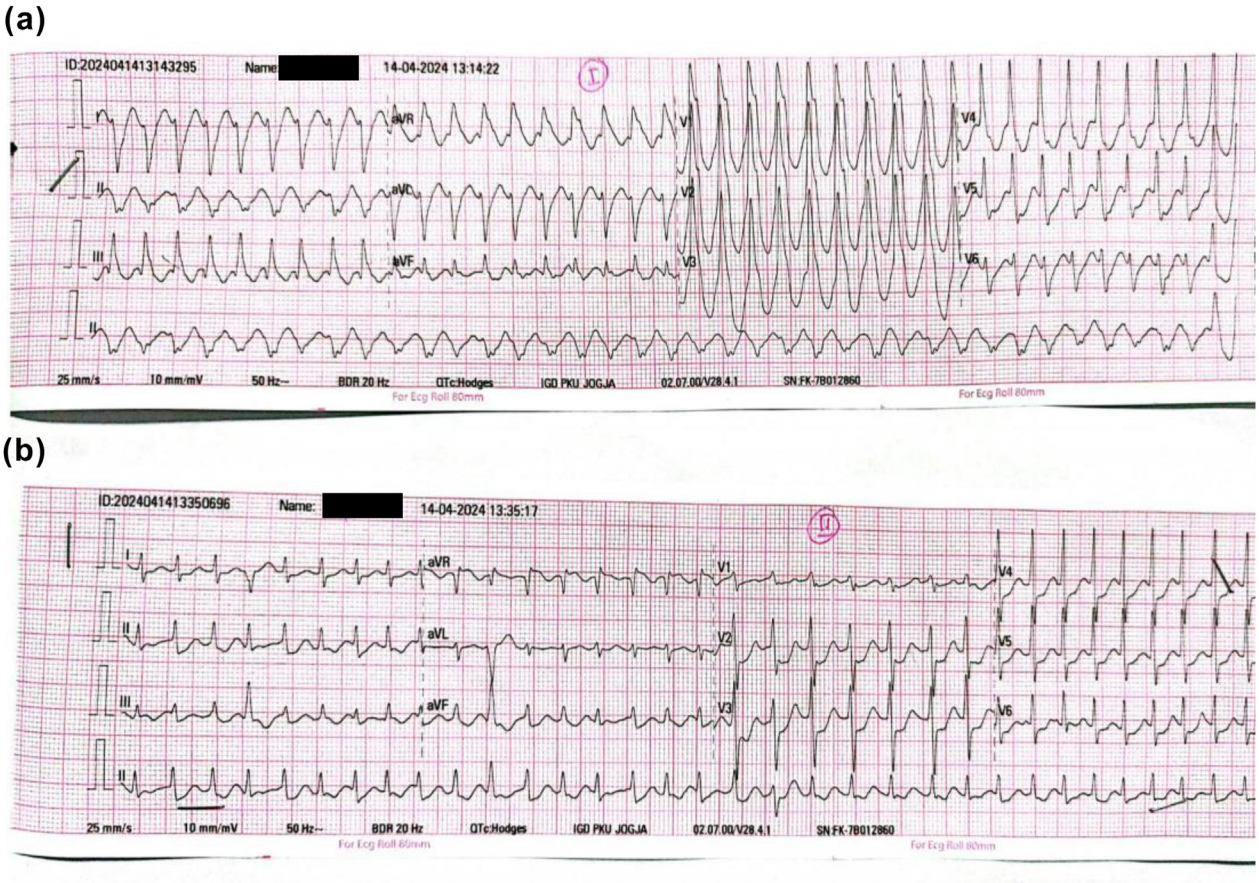

Fig. 1. (a) Documented ventricular tachycardia at first contact ECG with rate of 280 bpm. The ECG showed inferior lead discordance negative axis lead II, positive axis at lead III. R/S ratio at V6 < 1 suggestive for anterolateral papilary muscle (APM) origin (b) Documented supraventricular tachycardia short RP with rate was 150 bpm.

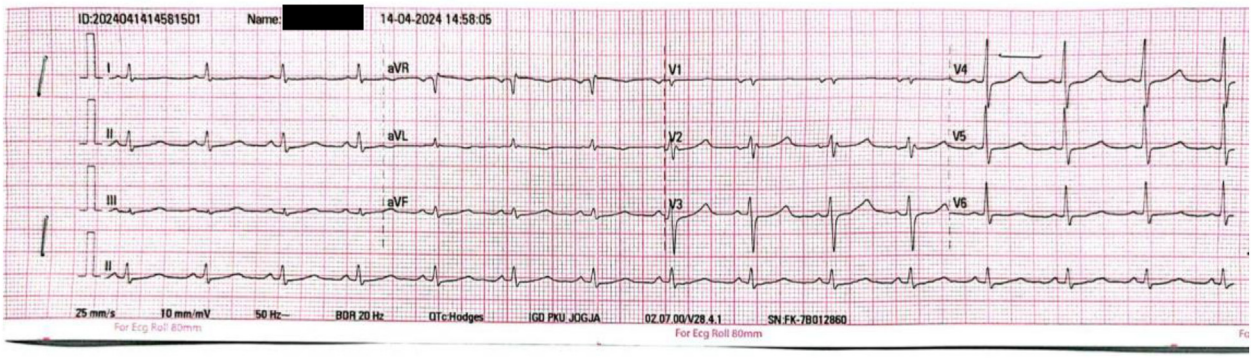

Fig. 2. Post-converted ECG with Normal sinus rhythm 75 bpm, normoaxis with T wave flattening/depressed at I, aVL, V6.

findings, High-sensitivity Troponin T was increased 1599 ng/L (normal <52 ng/L), normal serum creatinine level. Additionally, chest plain radiography was normal.

The patient was transferred to the catheterization laboratory for immediate PCI, where an emergency CA was performed through the right femoral artery approach due to radial artery tortousity. The left coronary artery (LCA) angiogram showed left anterior descending artery (LAD) with moderate stenosis in proximal-to-mid vessel, left circumflex artery (LCx) with subtotal occlusion at proximal, TIMI thrombus grade IV, with TIMI 1–2 flow. The right coronary angiography showed mild stenosis in proximal vessel and severe stenosis in mid vessel (Fig. 3). Based on the ECGs patient showed

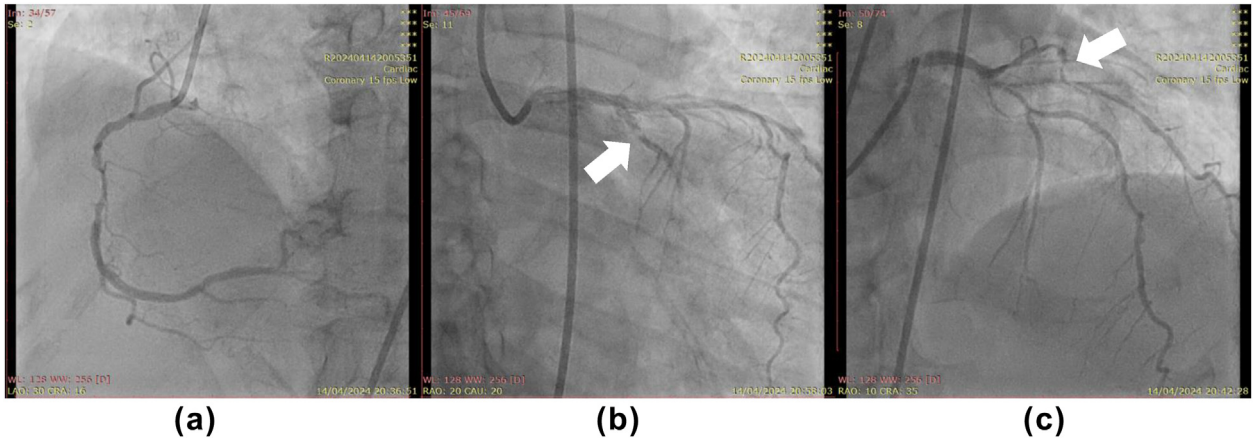

Fig. 3. Corongiography (a) right coronary artery (RCA): stenosis 50% at proximal, stenosis 70% at mid (b) and (c) left main (LM): normal, left anterior descenden (LAD): stenosis 70% at proximal to mid, left circumflex (LCx): subtotal occlusion at proximal, TIMI Thrombus Grade IV, TIMI 1−2 Flow (White Arrow).

monomorphic VT with site prediction at the anterior papillary muscle, and lateral T-wave changes (T-inverted) at I, aVL, V6, which was the territory of LCx. We decided to perform a culprit-lesion only invasive strategy at LCx (see Fig. 4).

A-6 Fr EBU 3.5 guide catheter was used to cannulate into LCA system. Due to significant thrombus. 14 mL eptifibatide was given intracoronary at LCx. Workhorse wire (Marvel) was advanced through a lesion at the proximal LCx; unfortunately, the guidewire protruded into the subintimal vessel. Then, we performed parallel wire technique with a stiffer wire (PILOT 50) and successfully advanced the wire to the distal vessel without resistance. Then, THROMBUSTER II was used to aspirate some red thrombus at the LCx. After the thrombus aspiration procedure, TIMI 2−3 flow was achieved until the distal vessel. Considering that the distal lesion of LCx was dominated by thrombus, with good flow, the intervention procedure (stenting or angioplasty) was not continued. The transthoracic echocardiography findings showed left ventricular concentric hypertrophy and preserved ejection fraction with EF 56% with normokinetic. The patient was observed at the intensive cardiac care unit with a continuous drip of GPIIB/IIIA inhibitor (eptifibatide) for 24 h, then discharged on the sixth day of hospitalization with Aspirin 80 mg O.D, Clopidogrel 75 mg O.D, Atorvastatin 40 mg O.D, Ramipril 2.5 mg O.D, Bisoprolol 5 mg O.D, long acting nitrate 2.5 mg B.I.D.

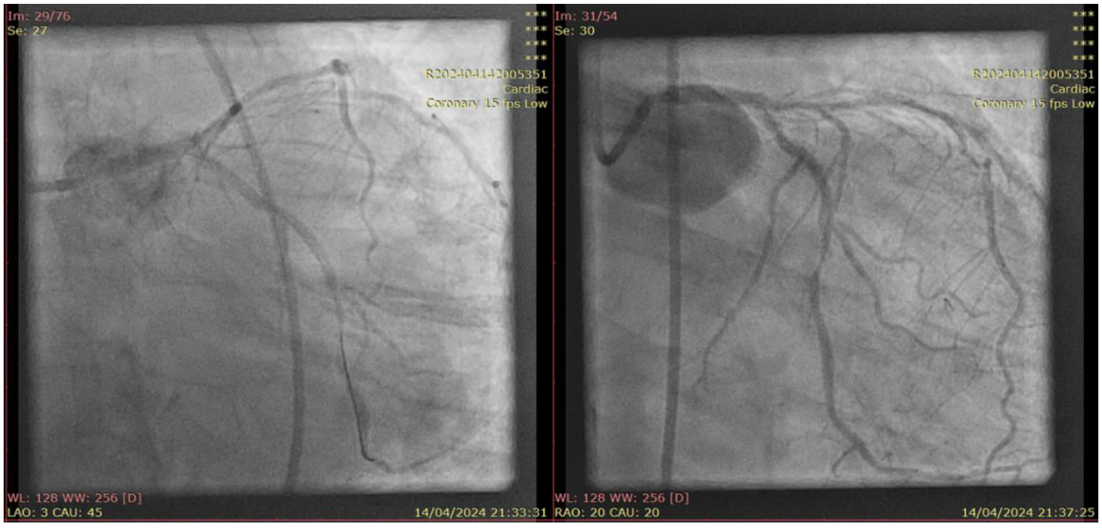

Fig. 4. Coroangiography of LCx after intervention of thrombus aspiration and eptifibatide intracoronary administration showing improvement of coronary flow and small residual thrombus.

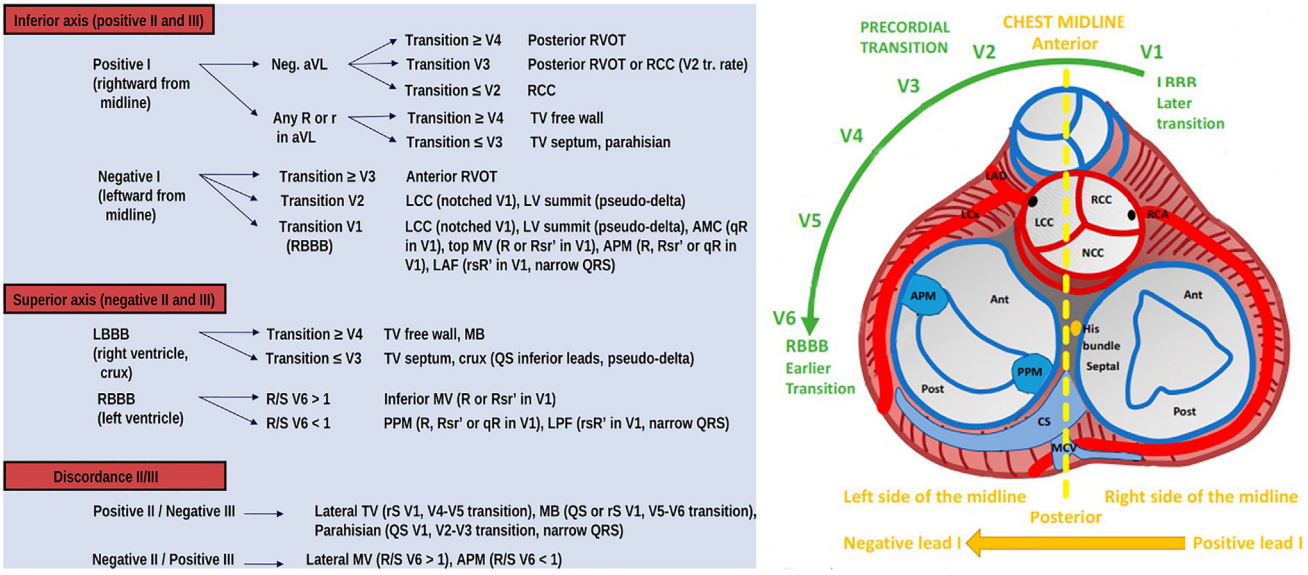

Fig. 5. Schematic figure and algorithm to predict the site origin of ventricular tachycardia (Source: Enriquez et al., 2019; Muser et al., 2021).

3. Discussion

We report a 60-year-old female presenting with NSTEMI and documented ventricular tachycardia also supraventricular tachycardia. According to the latest guideline from the European Society of Cardiology (ESC), patients with symptoms of ACS, ECG, and cardiac biomarkers consistent with NSTEMI, should be classified according to the latest risk stratification [7]. In this patient, it was documented life-threatening arrythmia (ventricular tachycardia) at first presentation. Which was classified as very high-risk NSTEMI. The patient later underwent an immediate invasive strategy in the catheterization laboratory.

In this case, we proposed an approach to defining the culprit lesion using 12-lead ECG during VT and post-converted ECG. Although the guideline clearly stated that complete revascularization in the setting of NSTEMI with multivessel disease is preferable (Class IIa recommendation), it was probably because of no dedicated trial comparing complete revascularization against IRA-only PCI. At first, we analyzed the site of origin of the patient's ECG during VT using the algorithm proposed by previous studies (Fig. 5) [8,9]. The ECG (Fig. 1a) showed that was inferior lead discordance (negative axis at lead II with positive axis at lead III) with R/S at V6 < 1, the prediction site of origin of VT was at anterolateral papillary muscle (APM) which was vascularized commonly by LCx [10]. Second, from post-converted ECG (Fig. 2), there were lateral T-wave changes (T-inverted) at I, aVL, V6 which was the territory of the LCx artery. Third, coronary angiography findings revealed multivessel disease with a subtotal occlusion at proximal LCx with TIMI Thrombus grade IV. Of these findings, we decided to perform PCI with thrombus aspiration due to significant thrombus at LCx.

We used peri-interventional eptifibatide intracoronary to improve blood flow and eliminate thrombus at LCx. Eptifibatide is a small-molecule glycoprotein IIb/IIIa inhibitor, which commonly used in the PCI settings to inhibit platelet aggregation [7]. Immediately after the administration of eptifibatide and thrombus aspiration, the coronary flow was improved and TIMI 2–3 flow was achieved. Intravenous drip administration of eptifibatide was continued until 24 h. There were no complications during the procedure. The patient was discharged on the sixth day of hospitalization without any symptoms and planned to undergo further procedure for full revascularization by elective coronary artery bypass grafting.

4. Conclusion

This study reports a case of NSTEMI with a very high-risk feature (life-threatening arrythmia) which was successfully managed by immediate IRA-only PCI. We highlighted several key points of the symptoms, diagnosis, and our approach in this case. A simple 12-lead ECG is vital in diagnostic and clinical decision-making. Using site prediction of the VT algorithm, we proposed an IRA-only PCI at left circumflex artery. This approach appears to be safe and has optimal outcomes.

Ethics statement

This study was conducted in accordance with the Declaration of Helsinki. Written consent was obtained from the patient.

Author contributions

Conceptualization: ADP, AD. Supervision: AD. Visualization: ADP, AD. Writing − original draft: ADP, AD. Writing − review & editing: HH.

Conflicts of interest

The authors have no potential conflicts of interest to disclose.

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