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RESEARCH ARTICLE

DE WINTER'S PATTERN: AN UNUSUAL ELECTROCARDIOGRAPHIC PATTERN TO RECOGNIZE

Dr. Manish Ruhela, Dr. Indira Sahu, Dr. Rakesh Kumar Ola and Dr. Rajeev Bagarhatta

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Abstract

De Winter syndrome is a rare electrocardiographic (ECG) pattern that makes the diagnosis of ST-segment elevation myocardial infarction (STEMI) very challenging. Our case indicates that early identification and diagnosis of such ECG's and timely reperfusion therapy of De Winter syndrome as an ST-segment elevation myocardial infarction (STEMI) equivalent are required to improve the prognosis of such patients.

Introduction:-

Electrocardiogram (ECG) play a pivotal role in the diagnosis of patients with suspected ST-segment elevation myocardial infarction(STEMI), allowing rapid treatment. In some cases, patients may have an initial ECG without ST-segment elevation, making the diagnosis very challenging. These ECG patterns are known as STEMI equivalents, such as hyperacute T waves, Wellens syndrome, de winter's sign, and posterior STEMI. (1) Our case report aims to reveal de winter's ECG pattern as a STEMI equivalent and early identification of these patterns by physicians is very crucial to improve the prognosis of such cases.

Case Report:

A 50-year-old male patient with a history of smoking was presented to the emergency department for severe retrosternal chest pain accompanied by diaphoresis, nausea and vomiting. His symptoms lasted for 6 hours. He had neither significant past medical history including diabetes, hypertension or hyperlipidemia nor a family history of coronary artery disease. An admission electrocardiogram(ECG) was performed in the emergency department showed upsloping ST-segment depression at J-point with tall and symmetrical T waves from V2 to V6 precordial leads, as well as ST-segment elevation in lead aVR.(Figure1) The cardiac biomarker (troponin-I) test was slightly elevated. He was admitted to the coronary care unit(CCU) with the diagnosis of non-ST-elevation myocardial infarction(NSTEMI) made by an emergency department physician. The attending cardiologist recognizes this ECG pattern as De winter's pattern, the equivalent of anterior STEMI. Both aspirin and ticagrelor were given and then taken to the catheterization laboratory. Emergency coronary angiography revealed complete occlusion of the proximal left anterior descending coronary artery(LAD). (Figure 2A) The patient underwent immediate percutaneous coronary intervention(PCI) with successful stenting of the LAD using a 3.0x28mm stent. Repeat angiography showed TIMI grade III flow in distal LAD(Figure 2B). The patient had no complications during the procedure and shifted to CCU. The post-procedural ECG showed persistent negative T waves in V2 to V5(Figure 3). This is commonly seen in patients that have had an acute anterior wall myocardial infarction. The left ventricular ejection fraction(LVEF) of the patient at the time of admission was 40%. The patient was discharged 3 days after the PCI with an LVEF of 55%.

Corresponding Author:- Dr. Manish Ruhela

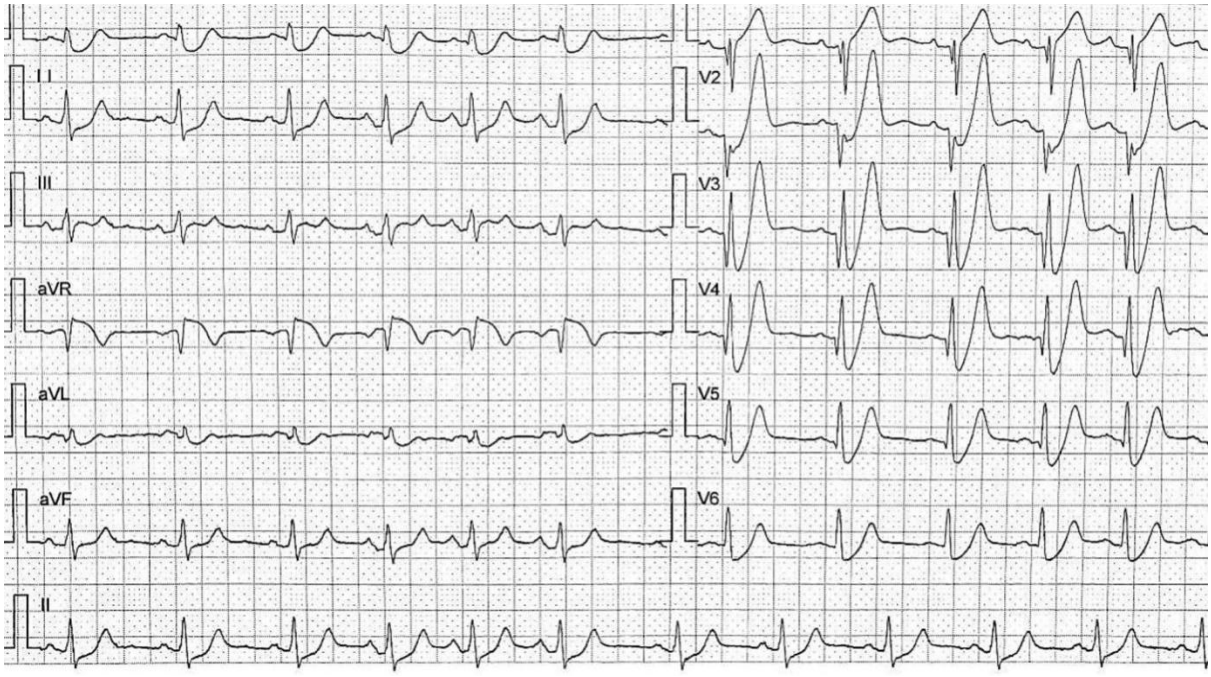

Figure 1:- Admission electrocardiogram (ECG) showing upsloping ST-segment depression from V2 to V6 leads, and tall and symmetrical T-waves in the precordial leads and ST-segment elevation in lead aVR.

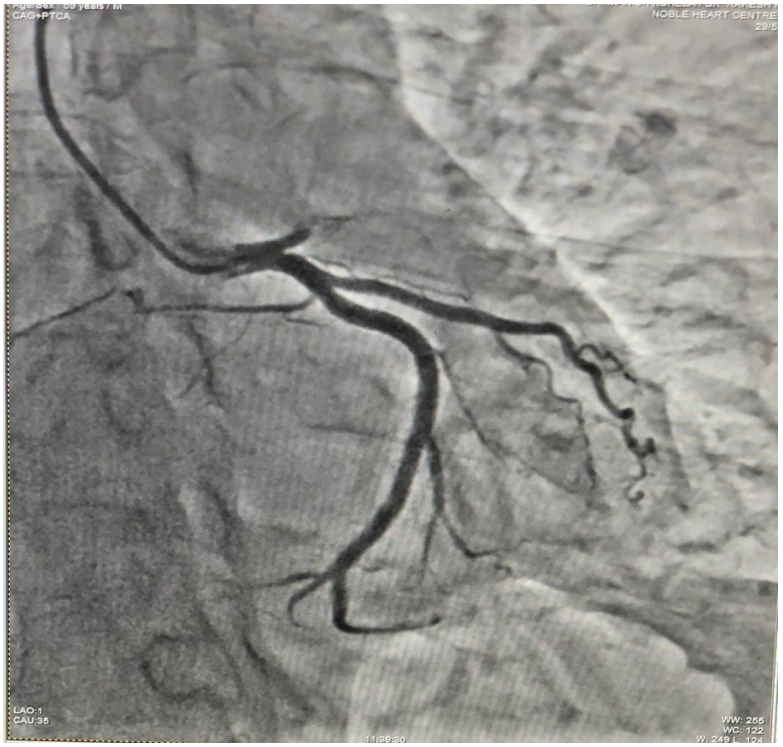

Figure 2 (A):- Coronary artery angiography showing complete thrombotic occlusion of the proximal left anterior descending artery (LAD).

Figure 2 (B):- Coronary angiogram after the left anterior descending artery (LAD) stenting.

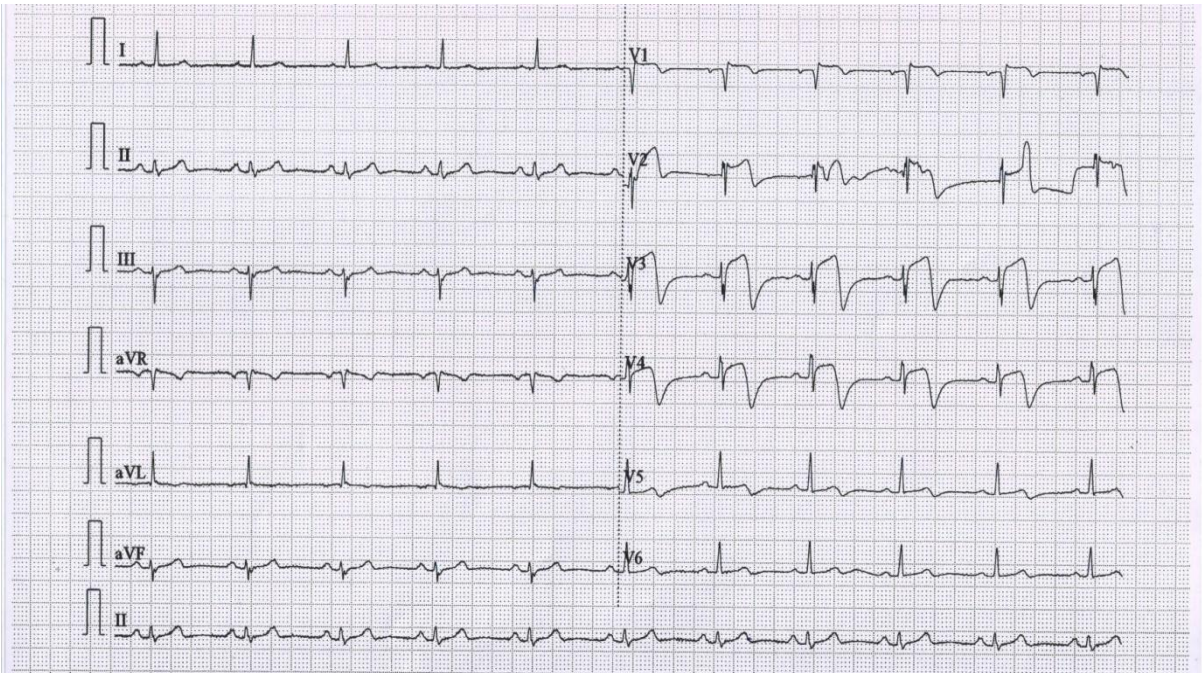

Figure 3:- Post Percutaneous coronary intervention (PCI) electrocardiogram (ECG) showing persistent negative T-waves in V2 to V5.

Discussion:-

The De winter's sign was first described by de winter and colleagues(2) in 2008 as an equivalent of STEMI due to proximal left anterior descending(LAD) coronary artery occlusion. Its prevalence is approximately 1.6% to 2.1% of anterior STEMI(2,4) but a recent study suggests a higher prevalence. (3) This pattern is associated with a mortality of approximately 27% within the first week. (4)

The diagnostic criteria for De Winter syndrome include (i) a 1 to 3 mm upsloping ST-segment depression at the J point in leads V1 to V6 that continues into tall, positive symmetrical T waves; (ii) QRS complex usually not wide or

only slightly widened; (iii) in some patients, a loss of precordial R wave progression; (iv) a 1 to 2 mm ST-segment elevation in aVR. (2)

A clear explanation of this ECG pattern remains elusive. The lack of activation of the sarcolemmal adenosine triphosphate-sensitive potassium channel is believed to be the cause of the absence of ST-segment elevation, as observed in adenosine triphosphate-sensitive potassium channel knock-out animal models. (2) It has also been proposed that the absence of ST-segment elevation could be due to sub-endocardial localization of the ischemia. According to this explanation, the loss of R waves in the precordial leads and the notch in the negative limbs of QRS complexes would be due to conduction slowing over the anterior subendocardium with initial activation of the opposite wall and late activation of the anterior subepicardium. The ST-segment depression would be related to the negative voltage difference between the subendocardial and subepicardial action potentials during the plateau phase, and the peaked T waves would be the expression of the shorter time duration between subendocardial and subepicardial repolarization. (5)

Besides the classic De Winter ECG T-wave changes must be differentiated from ST-T segments of an acute myocardial infarction during the hyperacute period. (6) Characteristics of the latter include tall T waves at precordial leads with/without symmetrical shapes and accompanied by a wide base. These types of changes occur due to early-phase occlusions of coronary arteries, and the ECG might evolve to show ST-segment elevation myocardial infarction (STEMI) as cardiac muscles become more seriously injured. On the other hand, the De Winter ECG indication thrombosis at LAD occlusion is identified based on the tall T waves along with upsloping ST-segment depression. More importantly, the De Winter ECG remains consistent, showing no dynamic ECG changes. It will eventually directly evolve into transmural myocardial infarction instead of evolving into STEMI. Thus, patients with De Winter sign-on ECG require immediate PCI to prevent expansion to large areas of anterior myocardial infarction with no evidence of ST-segment elevation on precordial leads.

The American College of Cardiology (ACC)/American Heart Association (AHA) released non-STEMI guidelines in 2012 and STEMI guidelines in 2013 and neither mention the de Winter ECG. (7) Thus, it is not a widely recognized type of acute myocardial infarction. Coronary angiography shows that the de Winter ECG is a manifestation of complete/partial LAD occlusion. Thus, upon identification of the de Winter ECG, PCI should be performed immediately.

Conclusion:-

Our case report underlines the importance of promptly recognizing de Winter's sign on ECG as a STEMI equivalent pattern to advance the patient to a rapid reperfusion strategy and confirms to higher risk and the probable evolutive feature of this sign. Recognition of this specific ECG sign is quite crucial for physicians, and such cases should not be confused with either STEMI or non-STEMI to improve the prognosis of such patients.

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