Introduction: Presence of ST-segment elevation in the context of acute coronary syndrome is fundamental for patient’s management. However, particular electrocardiogram (ECG) such as de Winter pattern have been highlighted without obvious ST-segment elevation. It signifies proximal left anterior descending artery occlusion and is often unrecognized by physicians.

Methods: A 57-year-old male patient without history of cardiac disease was admitted to referral hospital for acute onset of chest pain. Hypertension was the only patient’s risk factor for coronary artery disease. ECG at 30-minute after symptom onset (Fig. 1) showed significant ST-segment depression at the J point in leads V4-V6 with tall, positively symmetrical T waves with slight ST-segment elevation (0.1 mm) in the aVR lead. The patient developed ventricular fibrillation and managed by defibrillation. The patient was referred for further treatment. On arrival the patient was hemodynamically stable and physical examination revealed no abnormalities. Repeated ECG showed normal sinus rhythm with normalization of the ST depression. Urgent coronary angiography showed total occlusion in the proximal LAD coronary artery (Fig 2). Percutaneous coronary intervention was performed with three drug eluting stent resulted TIMI-2 graded flow. No complication developed in hospital after the procedure and the patient was discharged five days later.

Result: de Winter described a novel ECG pattern in 2008. Specific criteria for this ECG pattern consistently include: 1 to 3 mm upsloping ST-segment depression at the J-point in leads V1 to V6 that continue into tall, positive symmetrical T-waves and 1 to 2 mm ST-segment elevation in lead aVR. However, it is rare case that occurs in approximately 2% of patients with LAD occlusion. De winter et al purposed that there is an idiopathic anatomical variant of the Purkinje fibers, leading to delayed endocardial conduction. They also hypothesized the absence of ST-segment elevation is due to lack of activation of sarcolemmal ATP-sensitive potassium channels by ischemic ATP depletion. Controversy exists whether this pattern only occurs as a transient or persistent. That ECG changes may be missed or misdiagnosed as nonspecific, reversible ischemia. This can significantly lengthen reperfusion therapy. It is imperative that all practitioners learn to identify this ECG pattern to ensure appropriate intervention in the cardiac catheterization laboratory.

Conclusion: Patient presenting with typical angina symptoms, ST-segment depression and peaked T waves, combined with the absence of classic ST-segment elevation in the precordial leads of the 12-lead ECG, suggest that a significant LAD coronary artery occlusion is present. The de-Winter T waves should be expressed clearly in educational courses and in the guidelines such that patients would receive appropriate treatment in time and morbidity and mortality can be reduced.