

## The Winter gets Well(ens) – a rare pattern of left anterior descending artery occlusion

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### Summary

De Winter syndrome is a rare presentation in patients with acute left anterior descending artery occlusion, thus an equivalent of anterior ST elevation myocardial infarction (STEMI) requiring immediate coronary angiography. We present a case of de Winter syndrome, where, after successful revascularisation, the patient's ECG proceeded to Wellens syndrome while he complained of atypical chest pain. Follow-up coronary angiography showed good results, so possibly the Wellens ECG pattern here might indicate myocardial reperfusion.

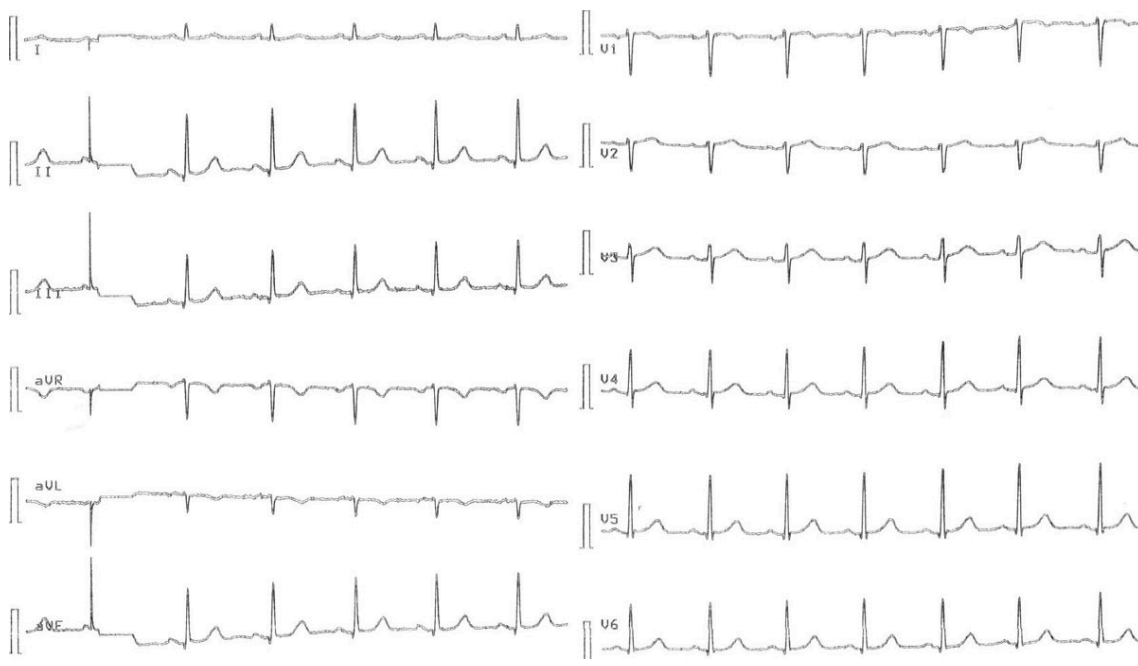
**Keywords:** *de Winter syndrome, Wellens syndrome, myocardial infarction*

### Case report

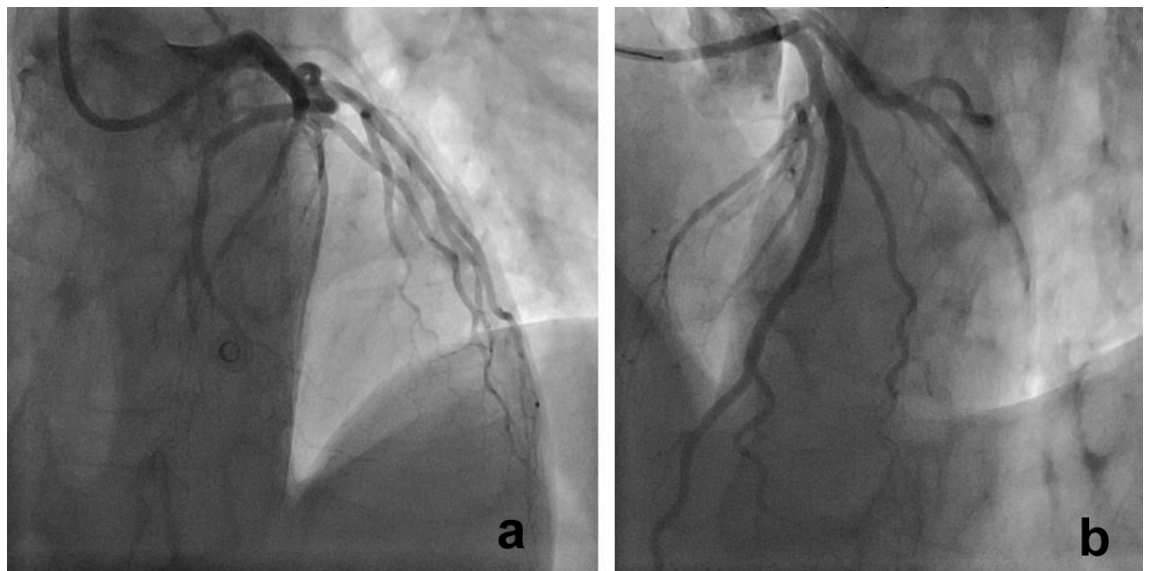
A 53-year-old male, previously in good health, presented to a primary care hospital because of severe acute chest pain with onset of symptoms 2 days previously and with

increasing intensity. The first ECG (fig. 1) was normal and the cardiac troponin I concentration, measured using a conventional assay, as well as creatine kinase (CK), aspartate aminotransferase, lactate dehydrogenase and D-dimer levels, were within the reference range. Because of increasing chest pain, aortic dissection and pulmonary embolism were ruled out by means of computed tomography. The patient continued to have ongoing severe thoracic pain and a follow-up ECG, 75 minutes after the initial one, displayed de Winter syndrome, with J point depression in leads V4 to V6 followed by relatively prominent, symmetrical T waves (fig. 2). The patient was therefore transferred to our tertiary referral centre and immediate coronary angiography was performed, showing acute occlusion of the mid left anterior descending artery (LAD) (fig. 3a). The lesion was pre-dilated and two drug-eluting stents were implanted. In addition, the small first diagonal branch was dilated using a final kissing-balloon technique, finally resulting in TIMI III flow in both vessels (fig. 3b). Echocardiogra-

Figure 1: The first, normal ECG.



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**Figure 2:** Follow-up ECG, 75 minutes after the initial one, showing de Winter syndrome.**Figure 3:** (a) Coronary angiogram showing acute occlusion of the mid left anterior descending (LAD) artery. (b) Final coronary angiogram, showing TIMI III flow in both the LAD artery and the small first diagonal branch.

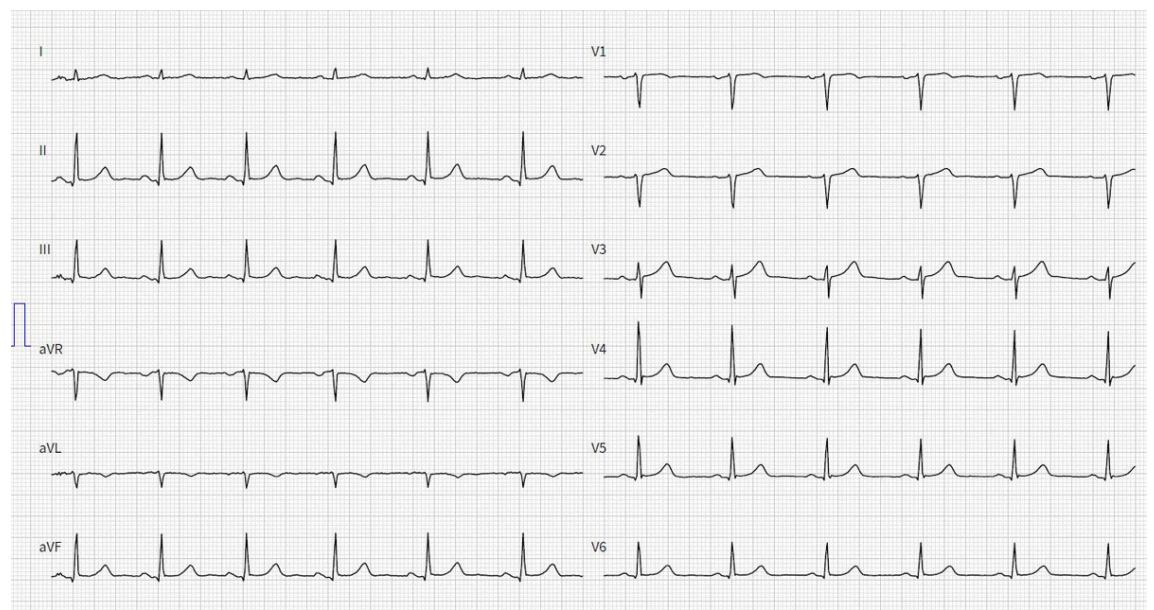
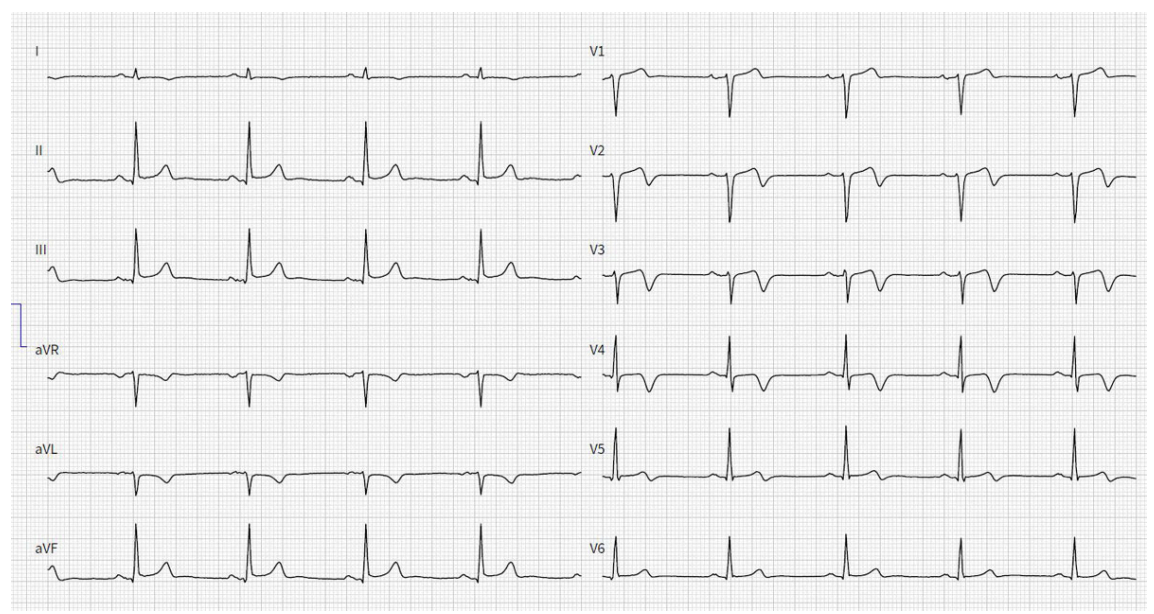
phy showed preserved ventricular function (ejection fraction 60%) with anterior and apical hypokinesia. The ECG after the intervention was normal (fig. 4). After revascularisation the patient was admitted to our intensive care unit for 2 days without occurrence of haemodynamic or rhythmic events. Maximum concentration of CK was 222 U/l (reference range 50–200 U/l) and high sensitivity cardiac troponin T (Hs-cTnT) peaked at 865 ng/l (reference value <14 ng/l). Investigation of cardiovascular risk factors revealed mild dyslipoproteinaemia (cholesterol 4.78 mmol/l; low-density lipoprotein 2.59 mmol/l; high-density lipoprotein 1.61 mmol/l) and a positive family history for stroke.

Three days after admission to the hospital the patient complained again of, now atypical, recurrent thoracic pain. The episodes lasted for about 30 seconds and were described by

the patient as burning sensations below the left clavicle. Interestingly, the patient's ECG now displayed Wellens syndrome type A (biphasic T-waves in precordial leads; fig. 5). Hs-cTnT, which had already decreased to 610 ng/l, showed a second peak (839 ng/l), while CK and CK-MB (52 and 1.9 U/l, respectively) continued to decrease. This pain responded well to analgesics.

Five days after admission the patient was discharged in good general condition to an outpatient rehabilitation centre.

Seventy-five days after the index event, elective coronary angiography was performed because of disturbing ventricular extrasystoles. Angiography was able to rule out ischaemia-induced arrhythmia, showing good results after

**Figure 4:** Normal ECG after the intervention.**Figure 5:** ECG 3 days after admission, showing Wellens syndrome type A (biphasic T-waves in precordial leads).

stenting of LAD without signs of stent thrombosis or restenosis. Left ventricular function remained normal with anteroapical scarring. An ECG at that time revealed slight biphasic T waves in leads V1 and V2 (fig. 6).

## Discussion

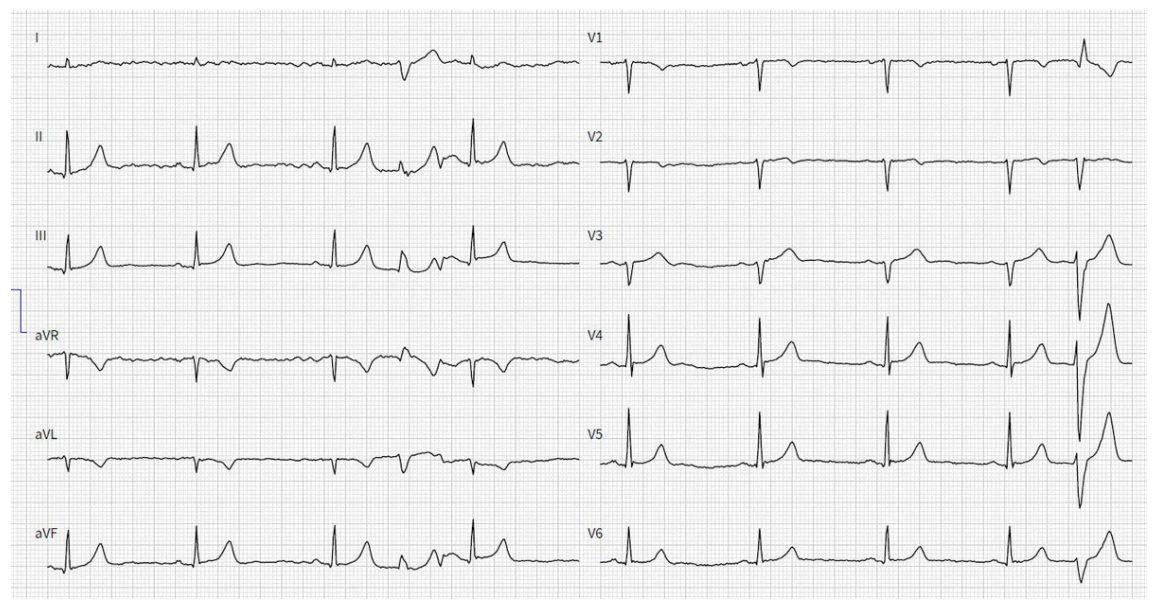
We present an interesting case of de Winter syndrome, an equivalent of acute anterior ST segment elevation myocardial infarction. An ECG typically shows 1–3 mm upsloping ST segment depression at J point in precordial leads, merging to tall symmetrical T-waves. In most cases, although not necessary for diagnosis, there may be an additional small (1–2 mm) ST segment elevation in lead aVR. This ECG configuration is pathognomonic for acute LAD occlusion, requiring immediate coronary angiography [1–3].

Interestingly, our patient's ECG recorded 3 days after successful revascularisation showed Wellens pattern while he complained about atypical chest pain.

Usually, Wellens syndrome, consisting of either biphasic (type A) or deeply inverted (type B) T waves in anterior leads, is considered highly specific for proximal stenosis of the LAD [4–6]. In contrast to our patient, Wellens ECG can usually be found in pain-free episodes, and in patients with normal or only slightly elevated (up to two times the upper reference limit) cardiac enzymes. On the other hand, there are several case reports of “pseudo-Wellens syndrome” caused by congenital myocardial bridge, takotsubo cardiomyopathy, cholecystitis or substance abuse, in the absence of critical LAD stenosis [7–11].

In contrast to most, but not all, cases reported, our patient's ECG did not show preserved R wave progression in ante-

**Figure 6:** ECG 75 days after the index event, showing slight biphasic T waves in leads V1 and V2.



rior leads in the presence of typical biphasic T waves. The mechanism of Wellens ECG is unclear, but it is hypothesised that repetitive transmural ischaemia-reperfusion leading to myocardial oedema, or coronary artery spasm are possible causes [12].

To our knowledge, this is the first case report of de Winter and Wellens ECG in the same patient, and of a Wellens ECG after successful revascularisation. Follow-up coronary angiography revealed a good short-term result after revascularisation, with preserved left ventricular function. Our case highlights the possibility that a Wellens ECG pattern could not only represent a pre-infarction state of coronary artery disease, but might also indicate myocardial reperfusion after successful dilatation and stenting. Other possible explanations might be coronary artery spasm, or dissolution of a possible thrombus with established dual antiplatelet therapy.

Our findings might help to prevent unnecessary cardiac catheterisation procedures in the future. Nevertheless, until there is further evidence, we do emphasise the importance of invasive diagnostics in any patient presenting with Wellens ECG and intermittent typical angina. Furthermore, our case report highlights the necessity of repeated ECG recordings in patients with high probability of acute coronary syndrome and a normal first ECG, and underlines the significance of knowledge of de Winter syndrome.

#### Disclosure statement

No financial support and no other potential conflict of interest relevant to this article was reported.

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