

# Clinical update no. 532

6 March 2019



European Society of Cardiology European Heart Journal (2018) 39, 119–177 doi:10.1093/euroheart/ehx393

ESC GUIDELINES

## 2017 ESC Guidelines for the management of acute myocardial infarction in patients presenting with ST-segment elevation

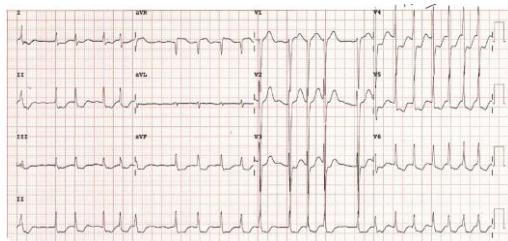
**Table 3** Atypical electrocardiographic presentations that should prompt a primary percutaneous coronary intervention strategy in patients with ongoing symptoms consistent with myocardial ischaemia

### Ischaemia due to left main coronary artery occlusion or multivessel disease

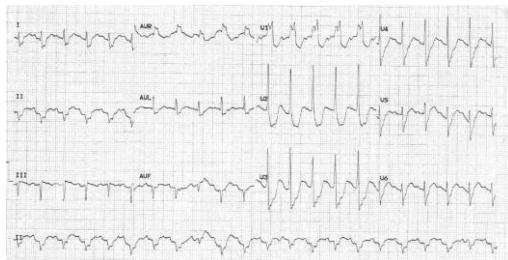
ST depression  $\geq 1$  mm in eight or more surface leads, coupled with ST-segment elevation in aVR and/or V<sub>1</sub>, suggests left main-, or left main equivalent- coronary obstruction, or severe three vessel ischaemia

STEMI Guidelines include, as indications for acute reperfusion, ST elevation in aVR together with widespread ST depression.

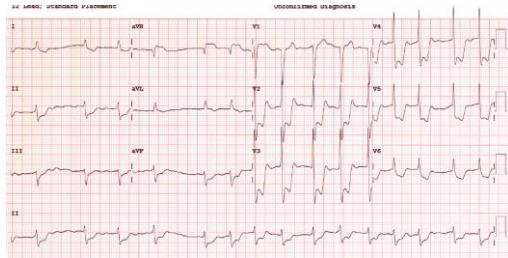
The incidence of acute coronary occlusion may not be as high as has been thought, warranting some further thought as to whether urgent PCI will help.



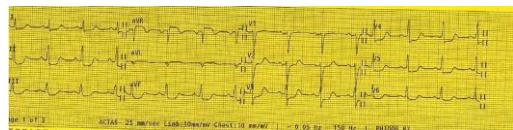
97yr-F; palliated.



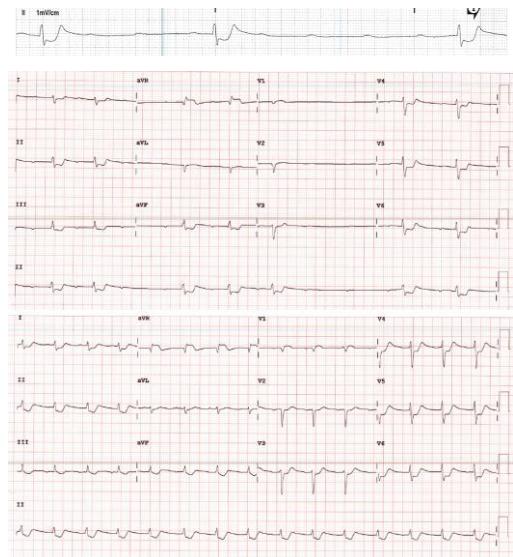
78yr-F; underlying massive PE



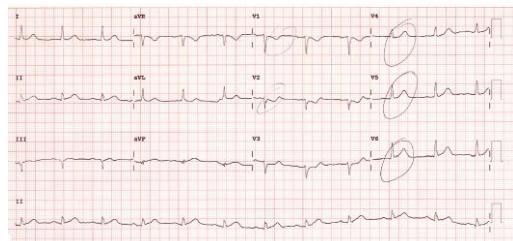
95yr-F; palliated.



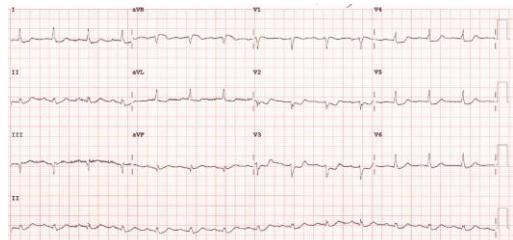
64M, post arrest. Likely ACS, but delayed ROSC and severe hypoxic brain injury.



66yr-M, syncope. K 7.6 mmol/L; responding to treatment. Troponin 203. Not a massive MI.



68yr-M, chest pain. Abrupt ST take off in lateral leads is typical for ischaemia. Progress ECG below ; hs-troponin I >50,000.



There is a differential for the ECG pattern. It can be from ACS amenable to reperfusion, other causes of global cardiac ischaemia without a discrete culprit lesion amenable to intervention, or non-ischaemic causes (see list below).

**ACS: LMCO, triple vessel disease, prox LAD**  
**Any other causes of global cardiac ischemia**

- TAD, massive PE, severe anemia, early post-arrest (w/i 15 min of EPI or shocks)

LVH with strain, esp. with severe htn  
 LBBB, pacers  
 SVTs (esp. AVRT)  
 Severe hypoK<sup>+</sup>  
 Sodium channel blockade (incl. TCAs, hyperK<sup>+</sup>, Brugada, etc.)



#### aVR ST Segment Elevation: Acute STEMI or Not? Incidence of an Acute Coronary Occlusion

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<https://doi.org/10.1016/j.amjmed.2018.12.021>

99 of 847 STEMI activations had ST elevation in aVR with widespread ST depression; 36 were post cardiac arrest. Urgent angiography was done in 80.

A likely culprit lesion was found in 8 (10% where angiogram was done), and none were left main or LAD. 60% of angiograms found severe CAD (40% did not).

Having STE aVR/ST depression was associated with 31% in-hospital mortality compared to 6.2% mortality in those without.

The majority of patients with aVR-STE and ST depression have severe multivessel coronary disease as opposed to an acute coronary occlusion.

[Dr. Smith's ECG Blog](#)

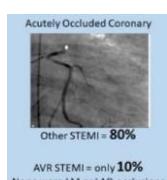
A useful discussion

<http://hqmeded-ecg.blogspot.com/2018/02/st-elevation-in-a vr-with-diffuse-st.html>

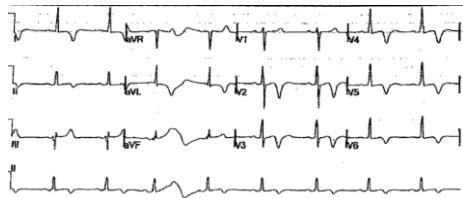
The pattern is diffuse subendocardial, not transmural, ischaemia. There may be coronary occlusion, but there are other causes that do not benefit from urgent coronary cath.

#### CLINICAL SIGNIFICANCE

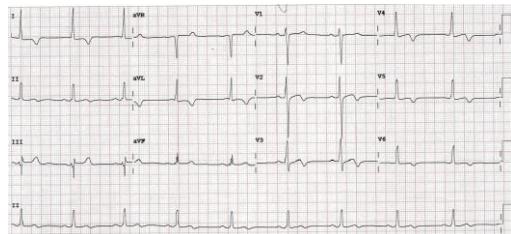
- Of patients with standard ST segment elevation myocardial infarction (STEMI), 80% have an acutely occluded coronary artery.
- Only 10% of patients with ST segment elevation in the electrocardiographic augmented vector right (aVR) lead had an acutely occluded coronary.
- Urgent cardiology evaluation is warranted, but routine activation of the STEMI emergency response team for ST segment elevation in the electrocardiographic aVR lead should be reconsidered.



Not so fast.



76yr-M referred by GP because of above ECG. There was mild chest discomfort which had resolved. Progress ECG -

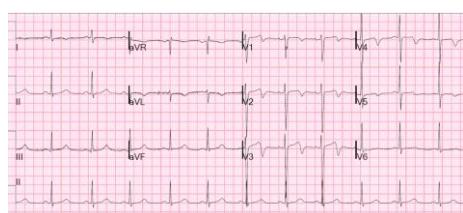


hs-Troponin I 119, with Wellens pattern on ECG. Referred for PCI. PCI wasn't done; an echo showed no regional wall motion abnormality, so no acute ischaemia or need for reperfusion, and there was no ongoing pain. Serial ECGs showed no change from the deep T wave inversion that was of concern to the GP. It was not clear why the ED ECG showed some variation (? lead placement).

The lab then notified that the raised troponin I was a false +ve due to assay interference from antibodies. Troponin T was 17, with no rise. A false +ve troponin I is unusual but happens. The rest requires the whole clinical picture to be taken in context.

#### Wellens' waves are NOT equivalent to Wellens' syndrome:

<http://hqmeded-ecg.blogspot.com/2015/01/wellens-waves-are-not-equivalent-to.html>



Wellens pattern on ECG does not always indicate acute LAD occlusion. The above is from a 20yr-M with end stage renal disease with hypertension and pulmonary oedema. It is type 2 demand ischaemia.

These updates are a review of current literature at the time of writing. They do not replace local treatment protocols and policy. Treating doctors are individually responsible for following standard of care.