Causes of pseudo inferior Myocardial Infarction

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1) Normal variant: in the horizontal heart and with the tip backward (S1 Q3 pattern). Besides, both the q or Q wave of III and aVF, may be influenced by the respiratory moment. Thus, a deep inspiration causes a verticalization of the heart and a decrease of depth in the cases in which there are no infarctions and persists in the presence of infarction;
2) Hyperkalemia
3) Acute pulmonary embolism;
4) Pre-excitation of WPW type;
5) Left Posterior Fascicular Branch (LPFB);
6) In certain LVH;
7) Possibly in End Conduction Delay (ECD) by one of the divisions of the right branch of the His bundle;
8) In CLBBB with extreme shift of SAQRS to the left.
9) In doubtful cases, VCG is more sensitive than ECG to diagnose inferior necrosis, because the analysis of the initial forces in the FP that displays closer dashes, suggests infarction.
10) In brief, the diaphragmatic infarction manifests more frequently in the inferior leads III and aVF, while the II lead is less constant, except extensive infarction of this wall. All emergency physicians will be aware of the ECG changes associated with acute MI. However, the increasingly documented phenomenon of pseudo MI needs to be considered, especially in those cases in which thrombolytic therapy could have disastrous consequences.

ECG changes compatible with acute MI have been reported in association with acute surgical abdomen (pancreatitis, gangrenous

appendix, perforated duodenal ulcers), and also with shock, severe metabolic stress, herpes zoster, pheochromocytomas, hyperkalemia secondary to diabetic ketoacidosis, and WPW syndrome, and rectus sheath haematoma. The mechanism by which this occurs is unclear. Several mechanisms: A “stress response” secondary to circulating catecholamines has been linked to pseudo MI, either secondary to the pain of the initial presentation (such as pancreatitis) or in relation to a pheochromocytoma. This appears to a likely cause in this patient, in the absence of any other abnormalities.

Vagal stimulation causing coronary artery spasm (Possible Hipotesis Master Samuel) or indirectly altering coronary blood flow has been considered, as has the effect of circulating proteolytic enzymes or electrolyte abnormalities.

Myocarditis such as Chagasic myocarditis. has also been linked to the ECG changes, but would have been unlikely with normal cardiac enzymes, left ventricular hypertrophy and Wolf−Parkinson−White syndrome.