

Pseudo-myocardial Infarction Pattern with Lax Esophageal hiatus with Severe Erosive Gastritis and Normal Coronary Vessels: a Case Report

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Abstract: The case is presented of a 20 year old male who attended the emergency department with sudden onset of choking discomfort in the throat & vague epigastric pain following a brief history of shortness of breath on exertion. Patient had no history of Ischemic heart disease. A routine electro-cardiograph (EKG) demonstrated an ST elevation in V1 to V4 with Q waves in II, III, and AVF consistent with acute myocardial infarction. Troponin I at admission & 12 hours later were normal (8.2 ng/ml). CT coronary angiography was reported normal. EKG changes compatible with Acute Myocardial Infarction have been reported with a number of non-cardiac manifestations. To our knowledge, it has never been reported in relation to lax esophageal hiatus with severe erosive gastritis. Possible mechanisms of Pseudo-myocardial Infarct & its importance is discussed.

Key Words: EKG – Electrocardiograph, Pseudo-myocardial infarction, CT coronary angiography, Troponin I, Lax esophageal hiatus, Erosive gastritis

I. Introduction

We present a case of 20 years young male patient with apparent EKG findings of acute myocardial infarction but an urgent CT coronary angiography revealed normal coronary arteries (no abnormality), a pattern consistent with diagnosis of pseudo-myocardial infarction. The possible mechanism by which this occurs is unclear. Stress response secondary to pain of erosive gastritis likely mechanism. Alternatively, we also speculate that, the EKG changes may as well be a reflection of vagal nervous system. The importance of treating the patient & not the EKG findings is emphasized.

II. Case Report

A 20 years old male was admitted with sudden choking discomfort in the throat, epigastric pain for past 4 hours. He denied history of shortness of breath on exertion, any major illness or psychological stress. Patient's history was non-contributing for Diabetes mellitus, Hypertension, dyslipidemia, smoking, alcohol consumption. On initial assessment, he looked apprehensive. Pulse rate was 100/min, regular, respiratory rate 24/min, BP - 120/80 mmHg. The systemic examination was unremarkable. Routine Electrocardiograph (EKG) demonstrated an ST elevation in lead V1 to V4 with Q waves in II, III, and AVF consistent with acute myocardial infarction. Repeat EKG showed an identical pattern of acute myocardial infarction. He had no other symptoms beyond discomfort in the throat & burning sensation. His serial Troponin & CPK-MB levels were normal. Blood sugar & electrolytes were also within normal limits. ECG findings were same even after 3 days with ST elevation in lead V1 to V4 with Q waves in II, III, and AVF. CT angiography undertaken revealed normal coronary arteries. 2-D Echo/Color Doppler did not reveal any evidence of chamber hypertrophy, HOCM, Pericardial disease or deficient pericardium.

Esophago-duodenoscopy was undertaken on 4th day of admission. Patient was noted to have evidence of lax esophageal hiatus; however no evidence of hiatus hernia was noted. Stomach revealed severely inflamed gastric mucosa with multiple erosions. The duodenum was normal. CT abdomen did not reveal any evidence of pancreatitis or adrenal pathology. 24 hours urine metanephrins were also noted to be within normal limits. Patient was managed comfortably & conservatively and discharged on day 5 of admission with a diagnosis of Pseudo-myocardial infarction.

III. Discussion

Our patient, who presented with a lax esophageal hiatus with severe erosive gastritis, has EKG findings suggestive of a myocardial infarction but urgent CT coronary angiography revealed normal coronary arteries. To the best of our knowledge this is the first case in the literature involving Pseudo infarction pattern due to severe erosive gastritis.

There is increasingly documented phenomenon of pseudo-myocardial infarction that needs to be considered especially in those cases where the thrombolytic therapy has disastrous consequences. EKG changes

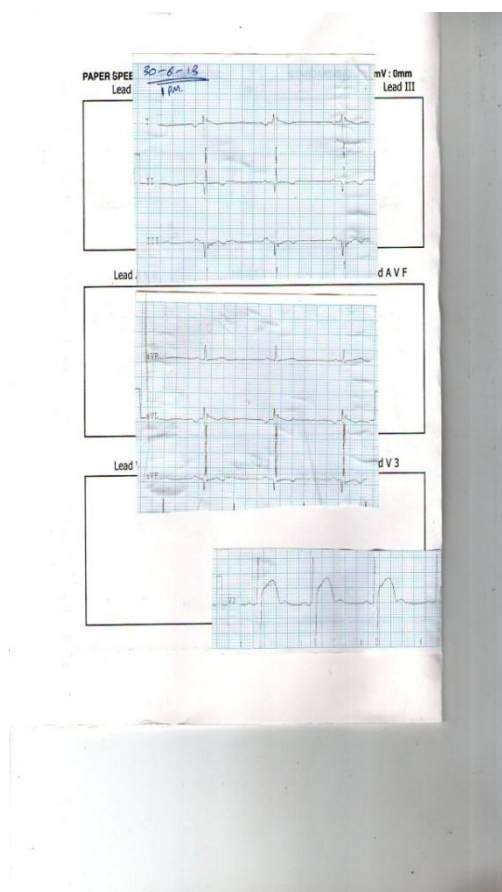
compatible with acute myocardial infarction have been reported in association with Acute Abdomen, presenting with pancreatitis, gangrenous appendix, perforated duodenal ulcer, rectus sheath hematoma, and sepsis with shock⁽¹⁾. Pseudo-myocardial infarction pattern has also been demonstrated in relation to diabetic Ketoacidosis⁽²⁾, Intracellular shift of potassium, Pulmonary embolism & Adrenergic hyper stimulation; all cases lead to picture of sick cardiomyocytes mimicking an injury current seen – Myocardial Infarction Necrosis.⁽³⁾

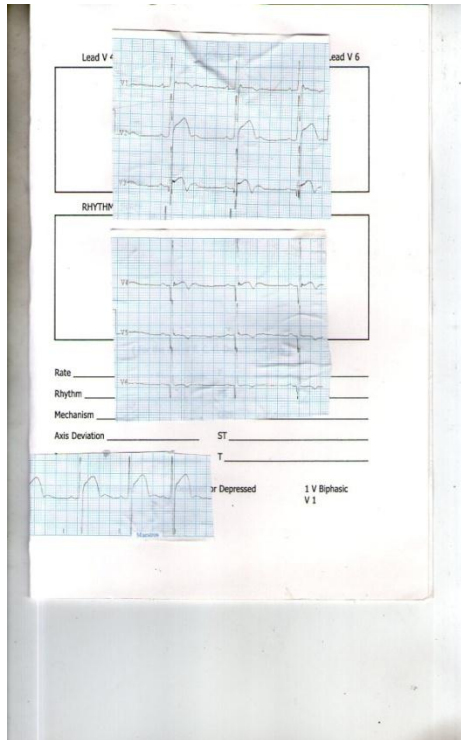
The possible mechanism by which myocardial pseudo infarct occurs is unclear. Stress response secondary to pain of erosive gastritis is likely mechanism in this case. Alternatively we also speculate that EKG changes could well be a reflection of the Vagal Nervous System causing artery spasm indirectly affecting coronary blood flow. Pseudo-myocardial infarct pattern on EKG changes have also been reported in Wolf-Parkinson-White syndrome, Left Ventricular Hypertrophy, HOCM, Pericardial disease or a deficient pericardium. 2-D Echo & Color Doppler did not reveal any evidence of these disorders.

It should also be appreciated that the relationship of Pseudo-myocardial infarction and lax esophageal hiatus with severe erosive gastritis is yet another cause in the expanding list of disorders related to Pseudo-myocardial infarction. Failure to appreciate such relationship could lead to disastrous consequences during management of patient.

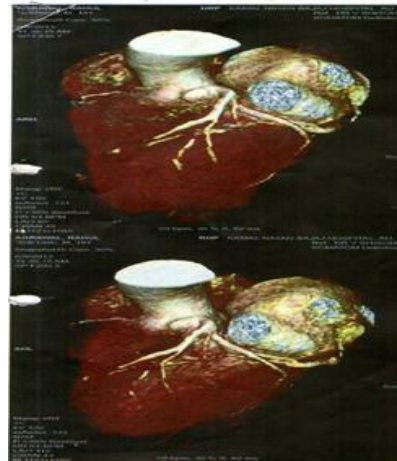
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1) EKG showing ST elevation in lead V1 to V4 with Q waves in II, III, and AVF consistent with Antero-septal myocardial infarction



2) Coronary CT angiography showing normal coronary vessels



3) Esophago-duodenoscopy images showing Lax esophageal hiatus with severe erosive gastritis