Appendicitis Causing Plaque Rupture - An Unusual STEMI Presentation of Appendicitis

Introduction:

Acute surgical abdomen is noticed to show EKG changes. This case highlights the relation between acute systemic event triggering plaque rupture, leading to STEMI

Case description:

Our patient is a 65-year-old Caucasian male with a past medical history of NSTEMI, 10 years back with status post DES in RCA and hypertension. He presented to us from the outside emergency department (ER) for an emergent PCI after being found to have inferior STEMI and elevated troponin of 14.0. He presented to the ER with generalized abdominal pain, diaphoresis and sob. He required 5 L of O2 (oxygen) on initial presentation. Patient had leukocytosis at 15.9k/ul. His EKG showed significant ST elevations in inferior leads. His initial troponin was <0.02, repeat troponin was 15.80 within the next hour. Patient was taken emergently to the catheterization lab. Total occlusion of the dominant RCA was seen in proximal to mid segment. Collateral circulation was noted from the left coronary system. DES of 2.75 x 24 mm was placed in acutely occluded proximal to mid RCA. Patient was started on aspirin and Brillinta per protocol. Post PCI, the EKG STEMI findings have resolved. Patient complained of abdominal pain still persisting and worsening. His leucocyte count is up to 24.6k/ul, lactic acid is 1.7mmol/ and blood cultures were drawn which later showed no growth. Abdominal CT was done which showed severe appendicitis. He is continued on the DAPT. He was found to have a gangrenous appendix which was removed. Post appendectomy, his pain was relieved, his leucocyte count trended down and was discharged the next day.

Discussion:

Appendicitis is systemic inflammatory process. Cytokines and interleukins like IL-1, IL-6, IFN-γ and TNF-α are studied to be significantly higher in serum of patients with appendicitis[6]. These inflammatory markers are also significantly elevated in STEMI patients [7]. Animal studies have shown that pro inflammatory cytokines can cause plaque instability leading to rupture. These inflammatory markers like IL-1, IL-6, IFN-γ and TNF-α promote apoptosis and are studied to

enlarge the lipid core, inhibit plaque stabilizing factors and matrix metalloproteinases. These factors ultimately can cause plaque rupture [8]. This could serve as a hypothesis as why an inflammatory process such as appendicitis could lead to a STEMI

Abstract:

This is a case of a 65-year-old male who presented with generalized abdominal pain and diaphoresis. EKG on presentation showed inferior wall STEMI (ST elevation myocardial infarction) with significant troponin elevation. Patient was transferred for PCI (percutaneous coronary intervention) and had RCA (right coronary artery) DES (drug eluting stent) placed after complete occlusion of RCA. With persistent abdominal pain, further evaluation showed the patient to have acute appendicitis. Appendectomy was performed without withholding DAPT (dual antiplatelet therapy)..

Case report:

Discussion:

. Several theories have been put forward, such as severe electrolyte disturbances, hypotension, high vagal activity [1]. A case report of a patient with gangrenous appendicitis was found to have EKG changes of anteroseptal MI with no troponin elevation [2]. A case of ruptured appendicitis and peritonitis was found to have ST elevations in inferior lead [3]. Similar case was reported with resolving ST elevations in lead V1-V3 after removal of appendix [4]. But these cases did not have a troponin elevation along with the EKG changes. Isolated EKG changes would have given our patient time for further evaluation, but with elevated troponin he was rushed to our hospital for PCI. Our patient had resolution of ST elevations in inferior leads after the PCI. The cases reported have presented a type 2 MI picture which is secondary to an ischemic balance. But our case was found to have ruptured plaque on PCI which points towards the type I MI [5]

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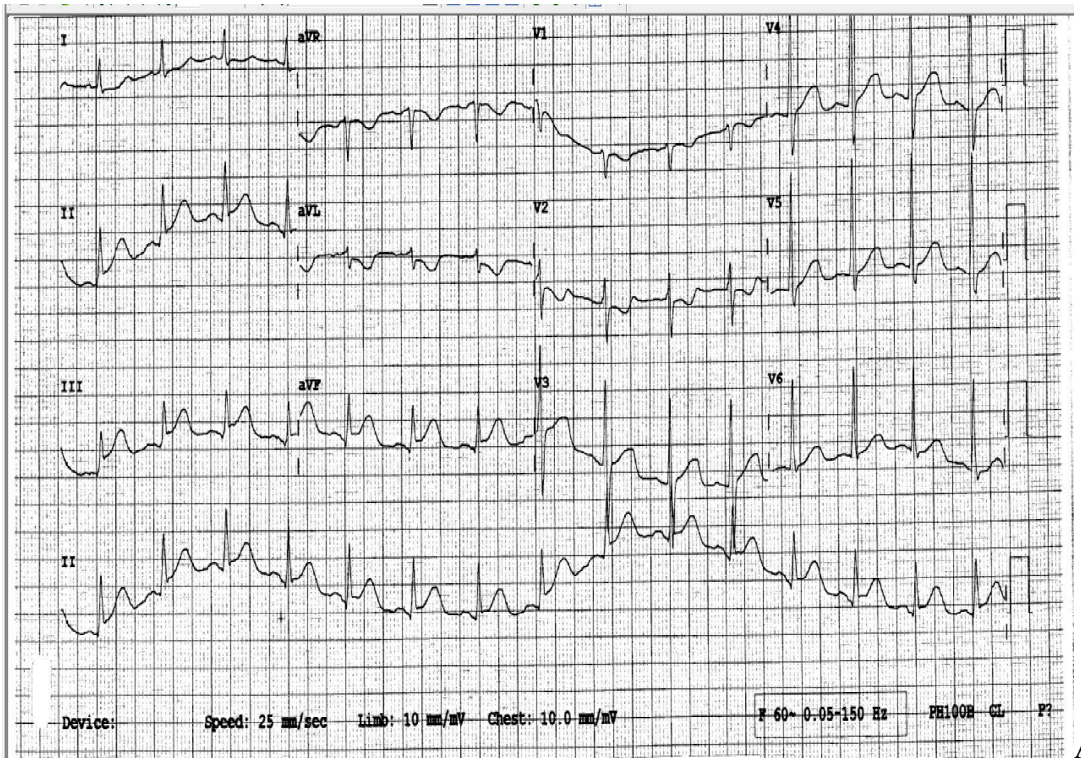
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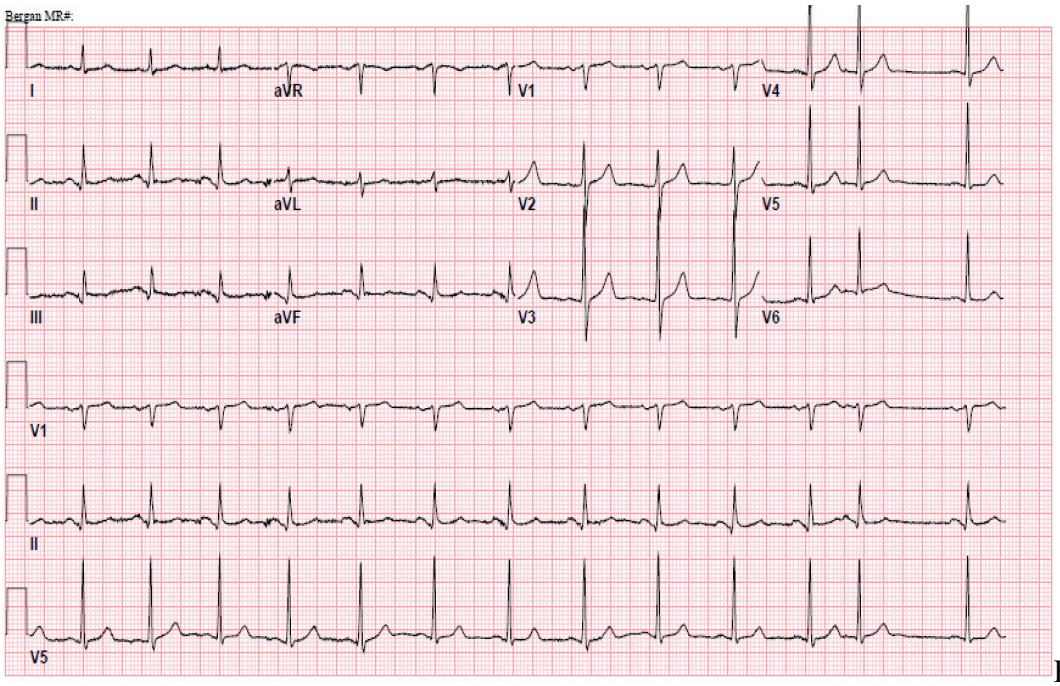

B

Figure 1- The figure shows A. EKG during initial presentation showing ST elevations in II, III, aVF with reciprocal changes in I and aVL. B. EKG shows resolution of the ST changes.

References:

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