Rare case of anaphylaxis causing st elevation myocardial infarction-epinephrine better avoided, ils myocardial bridge a trigger?

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Background

Myocardial infarction occurring in the context of an anaphylactic reaction ccausing allergic myocardial infarction, is known as Kounis syndrome.

Epinephrine is considered as the mainstay in the management of anaphylaxis. Intramuscular injection of 1:1000 adrenaline to a maximum dose of 0.5 mg is recommended in the management of anaphylaxis ... There have been a few cases of myocardial infarction occurring as a result of therapeutic doses adrenaline for anaphylaxis, reported in the previous case reports .The mechanism of myocardial injury was considered to be due to coronary vasospasm secondary to epinephrine as the coronary angiograms were normal in these occasions.

Here we report a case of a young healthy female who developed myocardial infarction following ingestion of Beef to which she was previously allergic to.

Case

34 year old female with known to haves ero positive rheumatoid arthritis presented to the emergency department with history of syncope within an hour of consumption of red meat (beef) to which she was known to be allergic.

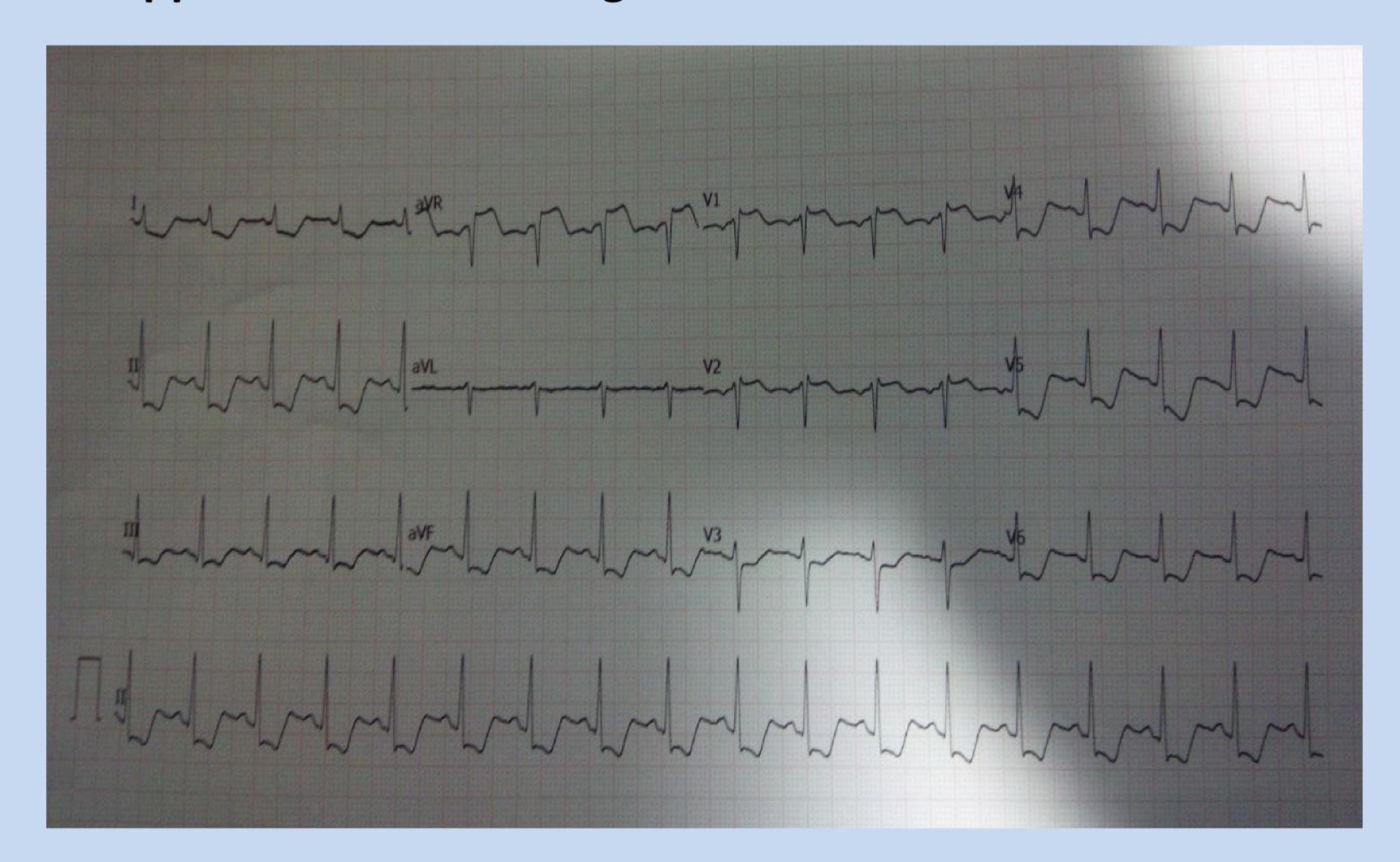
On presentation she had a patent airway and was not in respiratory distress. HR 102 bpm, RR 20 per minute, BP 70/50 mm Hg, Saturation 98% in room air.

She was diagnosed to be in anaphylactic shock

cardiac monitor attached showed significant ST depression in limb leads.

Adrenaline injection was deferred in view of the ST changes and the patient was treated with Injection Hydrocortisone 200 mg intravenously and with 25 md of intravenous Pheniramine maleate and intravenous Ranitidine 50 mg along with bolus fluid infusion.

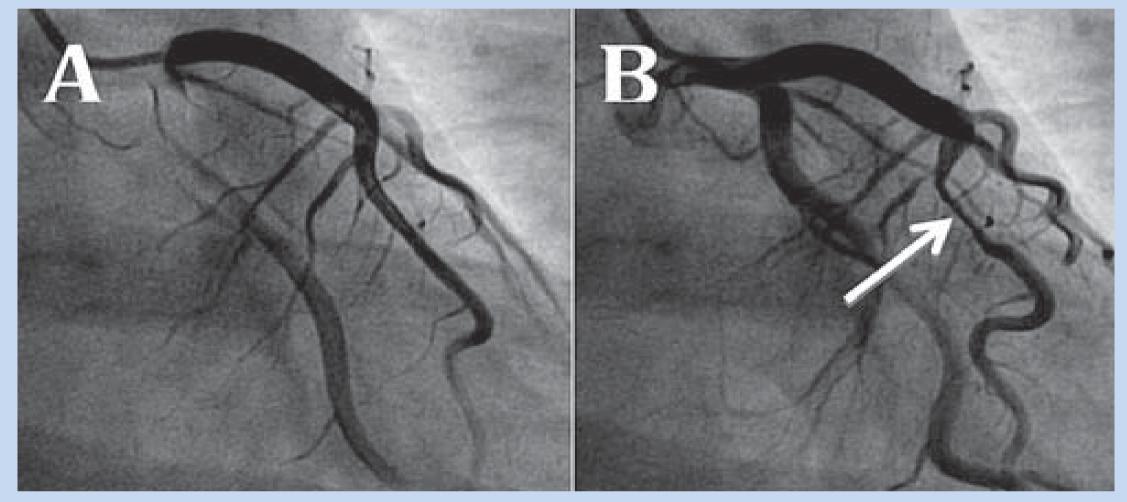
A12 lead ECG was immediately obtained which showed ST segmant elevation in leads a VR and V1, V2, and ST depression in leads I, II, III, a VL, V3 to V6, suggestive of LMCA occlusion or Proximal LAD occlusion or Diffuse sub endocardial ischemia. Cardiac Troponin was elevated and hence a diagnosis of myocardial infarction was confirmed. Patient improved hemodynamically and a repeat 12 lead ECG taken after 30 minutes of arrival to ED showed disappearance of ST changes.



She was administered oral loading dose of antiplatelets and Cath Lab was alerted.

Senior cardiologist assessed the patient in the emergency department and a bed side echo was done, which was completely normal.

In view of the significant ECG changes a coronory angiogram was done which showed a normal LMCA, LCX and RCA with a myocardial bridge in the mid LAD



Conclusion

Considering few case reports of cardiac arrests following administration of epinephrine in Kounis syndrome it would be preferable to look at the ecg changes in anaphylaxis before administering epinephrine.

There is one case report suggesting myocardial bridge as a possible trigger for Kounis syndrome. But in the above case the myocardial bridge was found in the mid LAD segment but ECG changes were suggestive of occlusion in the left main coronory artery and not in the LAD.

Anaphylaxis presenting as STelevation MI should be carefully assessed and treated with extreme caution.