Dear Editor:

Thank you very much for your suggestions which were very useful to me, I have thought about it carefully and made corresponding changes. The following are the answers I gave.

1. The patient's troponin and CK-MB values (baseline and following) must be given.

troponin 11.663umol/L;CK279U/L; CK-MB28U/L

2.ECG is normal. Is the patient's AMI non-ST-AMI? if ST-elevated-MI adds the ECG showing elevation. Detail the cardiac event in the case report.

There are two ECGs in this article. The first ECG (Fig. 1) which was normal was taken when he was hospitalized in our hospital. The second one(Fig. 2) shows that low T wave on V4-6 limb leads when he was taken to the emergency department of another hospital at the onset of acute myocardial infarction. At that time, the patient's symptoms had alleviated and supported non-ST-segment elevation myocardium infarction. He was considered as acute non-ST-segment elevation myocardial infarction.

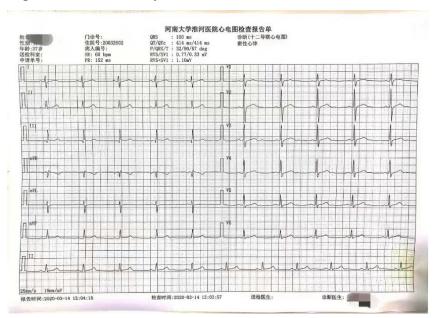


Figure 1 Electrocardiography. ECG tracings shows sinus rhythm with no arrhythmias.

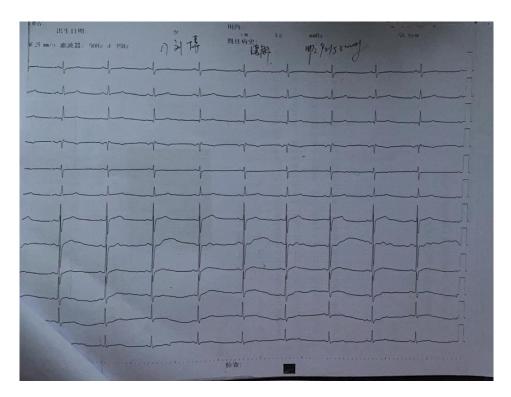


Figure 2 Electrocardiography. ECG tracings shows T wave is low on V4-6 limb lead.

3. Figures 1,2 and 3 are redundant, delete them, should be provided a cardiac angiography image and ECG figure.

I have deleted Figures 1,2 and 3, and provided a cardiac angiography image

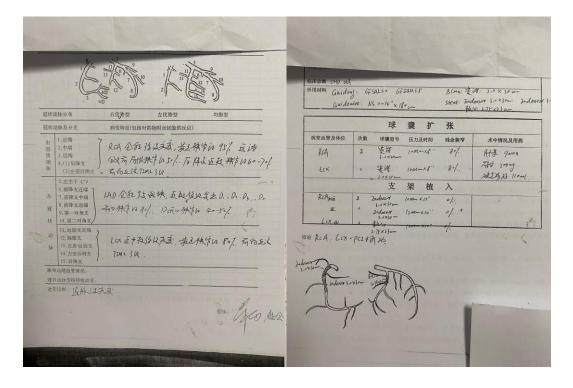


Figure 3 Surgical records of percutaneous coronary intervention. Three coronary arteries are diseased. Diffuse lesions occurred throughout RCA. The most severe degree of stenosis is 95%; 2 stents were placed in the right coronary artery; multiple diseased plaques throughout LAD; diffuse lesions in the near middle of LCX. The most severe degree of stenosis is 80%, 1 stent was placed here.

4. Add the following recent article on the subject to the discussion. doi: 10.1007/s10067-020-05354-3.

This article is very useful for me. After reading the article Characteristics and outcome of a first acute myocardial infarction in patients with ankylosing spondylitis. I got that Patients with AS tend to have a higher comorbidity burden for first AMI at admission. The mortality increased after a first AMI during days 31–365 among patients with AS compared with the general population. I inserted this point into the discussion in my article.

5.The authors should also mention the relationship between thalidomide and AMI.

I did not pay attention to the cardiovascular damage of thalidomide, thank you for your guidance, I have read the relevant literature. Some scholars think Thalidomide is associated with venous and arterial thrombotic events. The related mechanisms have been explored such as serum levels of the anticoagulant pathway cofactor thrombomodulin transiently dropped during the first month of thalidomide therapy, with gradual recovery over the following two months . Patients with multiple myeloma treated with thalidomide had high levels of von Willebrand factor antigen and of procoagulant factor VIII which were associated with an increased risk of thrombotic events in the general population. However, AMI caused by thalidomide generally has no obvious evidence of atherosclerosis through coronary angiography, which may be related to coronary artery spasm. This patient has obvious evidence of coronary atherosclerosis, so whether it is related to thalidomide is currently uncertain, but the rare cardiovascular events

of thalidomide should be considered when choosing DMARDs, and formulate appropriate treatment for the patient Program.

Thank you again for your suggestions and sincerely wish you all the best.

Sincerely,

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