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Letter to the Editor

Before attributing ST-elevation to COVID-19 related pulmonary embolism, myocardial infarction should be ruled out

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We read with interest the article by El-Azrak *et al.* reporting on a 68 years-old male with massive pulmonary embolism which was attributed to an acute infection with SARS-CoV-2^[1]. Since the patient initially complained about chest pain and dyspnoea and since electrocardiography (ECG) showed ST-elevation, right ventricular myocardial infarction was initially suspected^[1]. The patient profited significantly from intravenous thrombolysis with alteplase and could be discharged with only residual symptoms and signs^[1]. The study is appealing but raises concerns that should be discussed.

The main limitation of the study is that myocardial infarction was not adequately ruled out in the index patient^[1]. There was no determination of creatine-kinase (CK) or CK-MB serum levels. Furthermore, no coronary angiography had been carried out. The patient could have had both, myocardial infarction and pulmonary embolism. Given the fact that coagulopathy affecting the arteries and veins is a common complication of SARS-CoV-2 infections, it is crucial that both pulmonary embolism and myocardial infarction are appropriately ruled out. An argument in favour of myocardial infarction is that serum troponin was markedly elevated. Another argument in favour of myocardial infarction is that the initial symptom was chest pain starting three hours prior to admission^[1]. Based on these data, it is crucial to rule out myocardial infarction even if pulmonary embolism was documented on pulmonary artery angiography. Myocardial infarction with ST-elevation is a common complication of SARS-CoV-2 infections^[2] and definitively needs to be ruled out if clinical presentation, ECG, or blood tests are indicative of myocardial infarction.

A further limitation of the study is that the pretended infection with SARS-CoV-2 was not confirmed by a positive RT-PCR^[1]. Ground glass opacities on chest CT are not pathognomonic for COVID-19 as they may be seen in other pulmonary infections as well^[3]. A further argument against an acute SARS-CoV-2 infection is that anti-SARS-CoV-2 IgM antibodies were negative^[1]. Presence of IgG antibodies not necessarily indicates an acute infection as these antibodies can persist for months^[4].

We should know if right ventricular dysfunction was due to pulmonary embolism or due to Takotsubo syndrome (TTS) of the right ventricle. TTS has been reported in association with SARS-CoV-2 and pulmonary embolism^[5]. We should also know if pulmonary artery (PAP) was elevated or not. Elevated PAP is a common feature of pulmonary embolism.

There is a discrepancy between the statement that pulmonary exam was normal and the chest computed tomography showing bilateral, peripheral and lower ground-glass opacities suggesting mild viral pneumonia^[1]. This discrepancy should be solved. In case of pneumonia, the pulmonary exam should be abnormal.

Overall, the study carries obvious limitations that require re-evaluation and discussion. Clarifying these weaknesses would strengthen the conclusions and could improve the study. Before attributing ST-elevation to pulmonary embolism, myocardial infarction needs to be thoroughly ruled out.

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