

## A case report “Chest discomfort in a patient with dengue – is it an acute myocardial infarction?”

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Dear Editor-in-Chief

I read with great pleasure the recently published case report by Koh KC et al. in the *Malaysian Family Physician*, Volume 13, Issue 2, 2018. The authors have reported on an interesting case of Dengue myocarditis mimicking an acute myocardial infarction (MI). I would like to congratulate the authors on their great job in writing this case report.

However, there are a few important points that need to be clarified. First, Dengue myocarditis mimicking acute coronary syndrome (ACS) has been reported in the literature.<sup>1,2,3</sup> Among the three cases reported, two are from Singapore, and one is from Thailand. These patients were much younger than the current patient at presentation (ages ranged from 13 to 33 years old versus 56 for the current patient), and all of these patients had no coronary risk factors.<sup>1,2,3</sup> In the absence of coronary risk factors, I certainly agree that the most plausible explanation for the ECG and biomarker changes is Dengue myocarditis. However, it is also sensible to consider the possibility of silent ischemia in a post-menopausal woman. For this patient, I think Dengue myocarditis is a diagnosis of exclusion. It is of great importance to exclude coronary ischemia in an older patient with the symptoms, signs, ECG changes and biomarkers suggestive of ACS, either through a non-invasive imaging study (e.g., myocardial perfusion scan) or coronary angiography.

Second, I would like to emphasize that cardiac magnetic resonance (CMR) with myocardial contrast delayed enhancement sequences is a useful, noninvasive imaging test which can differentiate myocarditis from MI.<sup>4</sup> In myocarditis, the most typical finding is a subepicardial late gadolinium enhancement (LGE), sparing the subendocardium. In acute MI, LGE typically exhibits a subendocardial or transmural enhancement, and the edema is localized to the territory of the culprit vessel. Nonetheless, the main drawback of CMR is its high cost, and the test is not readily available.

Third, the authors described the patient's clinical signs of heart failure at presentation, including tachycardia, raised jugular venous pressure, crepitations at lung bases and pedal edema. Nevertheless, the clinical diagnosis of heart failure was not supported by the echocardiographic findings, i.e., a normal ejection fraction (60%). One possibility for such a presentation could be a diastolic dysfunction, but it was not described in the echocardiography report.

**Conflict of interest:** None

### References

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