Author's response to reviews

Title: A case report of ventricular dysfunction post pericardiocentesis: Stress Cardiomyopathy or Pericardial Decompression Syndrome?

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Author's response to reviews: see over
Dear Professors Sicari, Varga and Picano,

Re: ‘A case report of ventricular dysfunction post pericardiocentesis: Stress Cardiomyopathy or Pericardial Decompression Syndrome?’

We thank you for your careful review of the above-named manuscript and consideration for publication as a Case Report (with comprehensive literature review) in the Journal of Cardiovascular Ultrasound. Please find below responses to address the reviewers’ considered points.

Reviewer 1:

This is an interesting and well conducted case report with important clinical implications. Authors provide a comprehensive review of the available cases. They should probably expand on the pathophysiologic mechanisms. More images would be useful for the readership of the journal

- Please find expansion of pathophysiologic mechanisms on pp. 5 and 6 of the revised manuscript. This includes the following for pathogenesis of SCM (p.6): “A stressor leading to sympathetic overdrive and excessive catecholamine release is the currently accepted trigger in the development of SCM. The catecholamine surge precipitates 1) peripheral arterial vasospasm leading to increased afterload and transient increase in LV end-systolic pressure, 2) acute
multiple coronary artery vasospasm causing myocardial ischaemia, and 3) direct catecholamine-\(\beta\)-adrenoceptor-mediated myocardial stunning in the apex. These three pathophysiologic pathways are thought to contribute to the ischaemia, morphologic features and potential haemodynamic sequelae that may be seen in SCM”.

- Please find additional images (new figures 3 and 8 on pp 11 and 13 respectively) that demonstrate the physiological effects of tamponade as seen by significant trans-mitral inflow variation and parasternal long axis view additionally showing resolution of apical ballooning 2 weeks after the event

**Reviewer 2:**

*The case is well documented, that quality of the video samples is very good. I have only minor comments to the Authors:

Please, provide a follow-up ECG. The disappearance of the electrical alternans is obvious on the second ECG, but the other changes are less clear. A follow-up ECG would be useful for the enhancement of the differences.*

- Thank you. Please refer to p.14 with additional ECG (new figure 9) 2 months post event that demonstrates resolution of ischaemic changes. This now indeed better shows the reader the improvement in ischaemic changes, and the difference reinforcing the spontaneous resolution of the cardiomyopathy.

*The heart rate showed on the second ECG seems to be less than 110 beats/min as was reported in the text.*

- Thank you, this is well noted. This ECG was taken after the resolution of chest pain, and so the patient was not tachycardic as he had been at the immediate time of chest pain, which was a sign that helped herald the new cardiomyopathy. We have amended the text on p. 4 to more correctly reflect this. Unfortunately ECGs taken at the immediate time of the chest pain are not available in file.

*It was very interesting that there were only small changes on the ECG despite the biventricular involvement of the disease. Any explanation?*

- It is indeed interesting that more pronounced changes were not seen on our patient’s ECG. Samardhi et al in their series of SCM reported 41% having ST elevation and 44% having diffuse TWI, and Sing et al reported 25% with ST changes, 27% with deep TWI, and 10% developing Q waves. So whilst the majority of patients who are subject to this condition do develop more significant ischaemic ECG changes, there are some who do not. Whilst we cannot account for why our patient specifically did not have more
pronounced changes on ECG, we do note the loss of R waves in anterior leads (figure 6), which although more subtle, were nonetheless new, and then resolved (figure 9).

*Please, shorten the conclusion part of the discussion section.*

- Thank you, the conclusion on p.8 has been shortened to more succinctly convey the teaching points arising from this report

We thank you for your review and suggestions, and feel the revised manuscript is the better for them. We hope you find the revised manuscript worthy of publication in the Journal of Cardiovascular Ultrasound.

Yours Sincerely,

Professor Len Kritharides
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