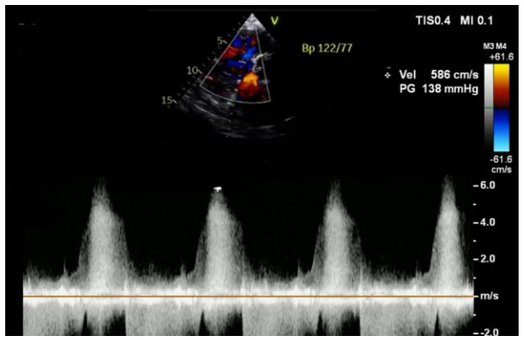
SDI EDITORIAL COMMENTS FORM

EDITORIAL COMMENT'S on revised paper (if any)	Authors' response to editor's comments
1. I'm quite surprised by the clinical presentation of the patient who did not actually require any pressor support or temporary mechanical support as an acute ventricular septal defect (VSD) in the setting of an acute myocarditis will signify a fulminant myocardial inflammation with prominent necrosis to cause septal destruction. The clinical presentation in the scenario of an VSD usually exhibits florid pulmonary edema and cardiogenic shock and most of these patients end up requiring pressors in addition to ionotropes and even mechanical support. How sure were the authors that his was not an acute VSD and just an incidental finding of a patient with congenital VSD with acute myocarditis (this differential needs to be discussed in detail in the discussion part). 2. I would be prudent if the authors could also provide a color doppler image and continuous wave doppler study of the VSD in figure 1 for better appreciation of the hemodynamics of the VSD. 3. I would request the authors to provide the doi of references 7,8 as I was not able to get these from the pubmed to be sure that these are the rightly quoted references. 4. The authors should also emphasize the implications of diffuse ST elevation in this scenario and this should differentiate from a presentation with STEMI.	**the patient did develop cardiogenic shock and was put under vasoactive drugs however he quickly recovered without the need for surgical intervention given the restrictive nature of the septal defect and the amelioration of ejection fraction.**  **well, the diffuse ST elevation is normally seen in both myocarditis and STEMI and in EKG there are practically no differences between the two of them. That is why we proceeded with a coronary angiography to rule out the implication of an Acute coronary syndrome.**