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Reply to: ECPR in acute aortic dissection - Really a no-go?



We thank the authors for their reply and interest to our case report on using transthoracic echocardiography to identify an acute aortic dissection during ECMO initiation [1].

The creation of an emergency department ECMO program is a complex process that relies on buy-in and agreement from multiple specialties including emergency medicine, cardiothoracic surgery, cardiology, and anesthesiology. This process requires predefined inclusion and exclusion criteria for initiation and termination agreed upon by all parties. As the authors state with regard to aortic dissection in ECPR, “no studies on feasibility, safety and efficacy of ECPR in this condition exist”. While the authors report three previous case reports of aortic dissection in ECPR, with one favorable outcome, we do not think this constitutes a level of evidence that would merit change to protocols.

The authors acknowledge the risk of perfusing the false lumen, which they estimate to be low based on non-emergent femoral cannulation cases. We hesitate to extrapolate this limited data to patients that suffer cardiac arrests from an acute dissection and are actively undergoing CPR.

With regard to diagnosis accuracy of TEE for Type A aortic dissection, we refer to Alter et al., which is cited by the authors, which states that TEE has a “sensitivity of 90–98% that is equal to CT or magnetic resonance imaging (MRI)” [2]. We agree that there are potential artifacts that can mimic acute aortic dissection, however, we believe if using TEE, providers should have knowledge of these potential pitfalls and obtain views in multiple imaging planes to confirm diagnosis. We refer to our accompanying videos for multi-view confirmation.

We overall agree that the concept of using ECPR for aortic dissection patients is intriguing and may warrant further investigation. We do, however, hesitate a change to established protocols for a perceived benefit without supporting data, especially given the resource and time expense as well as risk of body fluid exposure and needle sticks to providers.

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Pulmonary embolism in the differential diagnosis of right ventricular myocardial infarction

When right ventricular failure occurs as a result of right ventricular myocardial infarction (RVMI) [1], its manifestations can be closely simulated by right ventricular failure attributable to pulmonary embolism (PE) [2,3]. In the latter example of PE-related right ventricular failure a 35 year old previously healthy woman (a non-smoker) presented with severe abdominal pain and abdominal distension, but denied dyspnea or chest pain. Exploratory laparotomy revealed that she had a congested and very enlarged liver, and ascites as well. Further investigations included echocardiography, which disclosed a right ventricular systolic pressure of 77 mm Hg, and tricuspid regurgitation. These derangements were attributable to right ventricular failure resulting from massive PE [3]. According to Namana et al., RVMI most closely simulates PE when it presents with “chest pain, diaphoresis, nausea and vomiting with [the] hemodynamic triad: hypotension, jugular venous distension and clear lung fields” [2]. Dysrhythmias which occur in RVMI, such as atrial fibrillation and complete heart block [4], also occur in PE [5,6].

Hypoxemia is another manifestation of RVMI which can lead to a mistaken diagnosis of PE. The occurrence of RVMI-related hypoxemia was exemplified by a 70 year old man who presented with chest tightness and dyspnea. On examination his blood pressure was 100/60 mm Hg and jugular venous pressure was elevated. Oxygen saturation was 82% while he was breathing room air. The electrocardiogram disclosed ST segment elevation in leads V4–V6. Right sided precordial leads indicated 0.5 mm ST-segment elevation suggestive of RVMI. Transthoracic echocardiography showed infero-posterior wall akinesis. The right ventricle was dilated, with akinesis of the free and inferior wall. Furthermore, he had severely impaired right ventricular systolic function. Coronary angiography revealed total occlusion of the proximal segment of the right coronary artery. The persistence of hypoxemia (arterial partial pressure of oxygen amounting to 60 mm Hg), in spite of up-titration of inspired oxygen, prompted a search for coexistence of either PE or a right-to-left shunt. A computed pulmonary angiogram did not reveal PE. Transesophageal echocardiography (TEE) showed spontaneous constant flow from the right to the left atrium through a patent foramen ovale (PFO). This was a consequence of RVMI-related acute elevation in right atrial pressure. During his subsequent hospital stay hypoxemia gradually resolved. Repeat TEE, performed 6 weeks later, did not reveal any spontaneous right-to-left shunt through the PFO, presumably because right atrial pressure had reverted to normal levels [7]. In their account of the occurrence of a right-to-left shunt in a patient with inferior wall myocardial infarction, Albaghdadi et al. alluded to the occurrence of RVMI-related right-to-left shunting in eight cases reported in the literature [8]. They recommended that right-to-left shunting should be considered as the underlying cause of RVMI-related hypoxemia, after excluding more common causes such as pulmonary edema, underlying pulmonary disease, or pulmonary embolism [8].

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