Extracorporeal cardiopulmonary resuscitation after pediatric cardiac surgery

To the Editor,

We genuinely appreciate Erek et al. (1) for their study. Extracorporeal cardiopulmonary resuscitation (ECPR) has become a widely used procedure in cardiac arrest situations. The authors should definitely admit this procedure if they use cardiac arrest after pediatric cardiac surgery, a highly catastrophic condition. We believe that their results are very successful considering that the rate of post-cardiopulmonary bypass without cardiac arrest after discharge from the hospital is 20%–45% (2-4). However, we want to comment on a different topic. We believe that some obvious complications could have developed because of cannulation sites utilized by the authors. Because the ascending aorta is placed in the outlet cannula, left ventricular failure can be triggered by increasing afterload. Heart failure after ECPR is almost inevitable because of systemic phenomena caused by heart failure due to cardiac arrest in patients in the study by Erek et al. (1). Our questions to Erek et al. (1) are focused on this stage. If the causes of cardiac arrest in patients can be determined, what is the rate of heart failure in these patients? Further, if heart failure occurs, does it affect survival after ECPR? We would be very grateful if the authors have any explanation for these questions.

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Author`s Reply

To the Editor,

We thank Dr. Gökalp et al. (1) entitled "Extracorporeal cardiopulmonary resuscitation for refractory cardiac arrest in children after cardiac surgery." published in Anatol J Cardiol 2017; 17: 328-33. We agree with their comment that increasing afterload caused by veno-arterial (V-A) ECMO may impact left ventricular (LV) function. Increased LV afterload, together with severe systolic dysfunction, may result in LV overload with subsequent increase in left atrial pressure and severe pulmonary edema (2). This is especially true for patients with biventricular physiology, intact atrial septum, and severe left ventricular dysfunction, such as that in dilated cardiomyopathy. Although the experiences of left atrial decompression during V-A ECMO in children are limited, Hacking et al. (3) have suggested that the elective decompression of the left ventricle reduces ECMO duration and increases survival. However, in their study, almost all patients had biventricular physiology, only half of whom had congenital heart disease. As our study included a small number of patients with biventricular physiology, no patient required left atrial decompression. However, after submitting our study, we experienced two patients requiring left atrial decompression. One of them was a 15-year-old boy with dilated cardiomyopathy, and the other was a 3-year-old boy with ventricular septal defect closure and subaortic resection. Both patients received ECPR, and the indication for left atrial decompression was unresolved pulmonary edema. Left atrial decompression was achieved with a second venous cannula inserted through the left atrial appendage, which was connected to the venous line with a "Y" adapter. Atrial septostomy and left ventricular cannulation are other alternatives for left heart decompression during ECMO support (2, 3).

We again thank Dr. Gökalp et al. (1) for giving us the opportunity to emphasize the importance of left heart decompression during V-A ECMO support.

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Epicardial adipose tissue and atrial fibrillation: The other side of the coin

To the Editor,

Epicardial adipose tissue, a specialised visceral adipose tissue, produces numerous pro-inflammatory and pro-atherogenic mediators that promote the initiation and progression of coronary atherosclerosis (1). Increased epicardial adipose tissue is related to the presence and angiographic severity of coronary artery disease and coronary plaque vulnerability and independently predicts major adverse cardiovascular events (2). Furthermore, in visceral obesity, the epicardial adipose tissue undergoes conformational and functional changes, leading to the secretin of pro-inflammatory and pro-atherogenic adipokines (e.g., interleukin-6, tumor necrosis factor α, adiponectin, leptin, and plasminogen activator inhibitor) (2), which are involved in a causal relationship between inflammation and atrial fibrillation (3). Consequently, beyond classical cardiovascular risk factors, a causative link between the epicardial adipose tissue and atrial fibrillation has also been suggested because of the structural and functional interplay between atrial fibrillation and the epicardial adipose tissue and the existing evidence of abnormal atrial architecture, adipocyte infiltration, and atrial fibrosis that predispose the myocardial tissue to arrhythmic genesis (4).

In their very interesting and well-conducted clinical research article entitled "An increase in epicardial adipose tissue is strongly associated with carotid intima-media thickness and atherosclerotic plaque, but LDL only with the plaque" recently published in the Anatolian Journal of Cardiology 2017; 17: 56-63, Kocaman et al. (2) emphasized that the epicardial adipose tissue had a stronger association with carotid intima-media thickness than other risk factors. The epicardial adipose tissue has a complex pathophysiological function; potential direct interactions through paracrine or vasocrine mechanisms between the epicardial adipose tissue and myocardium are strongly suggested because of its metabolically active role as a source of several both pro- and anti-inflammatory adipokines (5). Therefore, it is reasonable to assume its additional role in the modulation of biochemical and metabolic triggers leading to atrial fibrillation (5). The association between the epicardial adipose tissue amount and atrial arrhythmia is supported by a consistent body of evidences suggesting a strong relationship; moreover, the presence of other cardiovascular risk factors does not weaken this link, clearly indicating that the epicardial adipose tissue depot can play a role in the complex pathophysiological scenario of atrial fibrillation (5).

Hence, one could hypothesize that the role of epicardial adipose tissue as a novel cardiovascular risk predictor involves both coronary artery disease and atrial fibrillation. Considering that this probable role in providing continuous pro-atherogenic and pro-inflammatory stimuli could be involved in both the initiation and progression of atherosclerosis, in addition to that a modulator in the arrhythmia genesis and as a possible substrate or trigger, this relationship is not clinically negligible and should be considered a very important element in the prevention/management of cardiovascular disease. In conclusion, based on these evidences, we can suggest that the epicardial adipose tissue is a novel and comprehensive surrogate of cardiovascular risk. Therefore, further consensus on the definition and method to assess and quantify the epicardial adipose tissue should be reached; the epicardial adipose tissue can become a therapeutic target, and evaluating the epicardial adipose tissue amount can become a major need, both for the diagnostic work up and for the assessment of therapy response.

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