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## Journal of Cardiology Cases

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## Editorial

## Editorial: Pericardial mass: Unusual interference with the right ventricular function



## Keywords:

Pericardium  
Hematoma  
Right ventricular function  
Compression

Right ventricular (RV) diastolic dysfunction is an important indicator for the assessment of heart failure. Abnormalities in RV diastolic function underlie many pathologies. However, the underlying mechanisms remain poorly described, because the term is used to describe many variants in diastolic pathophysiology and function. Since the right ventricle is coupled to the pulmonary circulation with a lower intracavity pressure and had a thin wall, the right ventricle is more compliant than the left ventricle. Thus, the RV volume overload as well as pressure overload may lead to RV distension, which would be restricted by the pericardium. Thus, RV diastolic dysfunction can be caused by any combination of abnormalities in the physiological properties of RV muscle including relaxation as well as compliance, ventricular interdependence [1], and pericardial constraint. RV dysfunction due to the worsening of RV muscle relaxation or RV muscle compliance may occur in the conditions such as RV infarction, restrictive cardiomyopathy, and arrhythmogenic RV cardiomyopathy. However, it is rare that the main cause of RV diastolic dysfunction is due to the impairment of RV muscle itself. Since the right ventricle is surrounded by the pericardium and the RV pressure is much lower compared with left ventricular (LV) pressure, the intrapericardial pressure would practically affect the RV geometrical changes and RV filling. The pericardium has a restrictive effect on the dilated and damaged right ventricle, while the right ventricle could be remarkably distended in the absence of the pericardium [2]. Thus, the pericardium is essential to regulate the RV hemodynamics. The muscle fibers of the RV free wall are orientated transversely and squeeze the blood within the ventricle in a circumferential compression motion [3]. The connections between the muscle fibers of the right ventricle and left ventricle tether the ventricles together through the interventricular septum. The concept of ventricular interdependence is that the size, shape, and compliance of one ventricle can directly affect the size, shape, and hemodynamics of the contralateral ventricle through septal wall reconfiguration, along with the pericardial constraint.

Echocardiographic studies revealed that increase in LV pressures can lead to RV diastolic dysfunction in patients with aortic stenosis [4] and systemic hypertension [5].

As the properties of pericardium directly affect the RV shape and RV hemodynamics, pericardial and intrapericardial abnormalities may induce RV diastolic dysfunction. Cardiac tamponade is a state of impaired distension of the heart due to abnormal accumulation of some substance, typically an increasing amount of fluid in the pericardial space. A constraining effect of the pericardium on the combined volume of the four chambers, respiratory variation in intrapericardial pressure, results in the impairment of right ventricle and LV filling and low output phenomenon followed by hypotension. Furthermore, Beppu et al. reported that one of the causes of low output failure after open heart surgery was the impaired heart distension by pericardial coagula [6,7]. As a possible cause of low output failure, LV dysfunction or cardiac tamponade could be considered. When LV wall motion abnormalities with low LV ejection fraction could be detected, low output failure would be attributed to LV dysfunction, while pericardial effusion with RV collapse implicates the cardiac tamponade. If the bleeding is rapid and large, the blood will spread and cover the entire heart. In contrast, if the bleeding is small and continuous, it may produce a localized blood clot. Thus, the localization of pericardial effusion (coagula) is variable. D'Cruz et al. reported that among 11 patients who required surgical relief of cardiac tamponade after cardiac surgery, 4 had non-localized pericardial effusions, and the other 7 patients had a localized posterior pericardial effusion [8]. Postoperative localized pericardial effusions causing tamponade may selectively and directly compress the left atrium, RV outflow area, and right atrium. Regardless of the localization of coagula, pericardial blood clot might result in cardiac tamponade whose hemodynamics are different from those in fluid tamponade [7]. Furthermore, a localized mass in the pericardial space may induce intraperitoneal pressure elevation, resulting in cardiac tamponade-like hemodynamics [9]. In contrast, the direct compression of right atrium and right ventricle by the coagula localized at the right anterior aspect of the heart could lead to similar hemodynamics to constrictive pericarditis [10]. In the present case report, Nishi et al. [11] demonstrated that a large hematoma located posterior to the pericardial space of LV side could compress the RV cavity to the sternum. It would be extremely rare that localized hematoma posterior to the left ventricle, which was not adjacent to the right ventricle, caused the compression of right ventricle. The authors speculated the two mechanisms for RV dysfunction. One is the elevated intrapericardial pressure by the localized hematoma, and

DOI of original article: <http://dx.doi.org/10.1016/j.jccase.2015.02.007>

<http://dx.doi.org/10.1016/j.jccase.2015.05.013>

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the other is the RV compression by shift of the entire heart to the sternum. The latter might be more plausible, because the elevation of intrapericardial pressure may induce the cardiac tamponade which could not be observed in the present case. Pericardial tumors also could be the cause of compression of the right-sided heart, resulting in the hemodynamic impairment. Arai et al. reported that ectopic thymoma located in the pericardial space compressed the right atrium and caused the right heart failure and cardiac tamponade [12]. Furthermore, the malignant pericardial mesothelioma, and pericardial invasion of the myeloid sarcoma, might form the masses in the pericardial space.

Although pericardial hematoma is not a rare complication after cardiac surgery, it has not been well recognized that a localized hematoma posterior to the left ventricle, which was not adjacent to the right ventricle, caused the RV compression, and RV failure. We should learn from this case report that any type of pericardial mass could cause the hemodynamic dysfunction.

### Disclosure

None.

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7 May 2015