

Editorial



Post-pericardiocentesis Constrictive Physiology in Cancer Patients With Pericardial Effusion

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Pericardial effusion with tamponade, which frequently occurs in patients with cancer, is caused by several mechanisms, including a direct extension or metastatic spread of the underlying malignancy, a complication of radiation or chemotherapy, or an opportunistic infection during antineoplastic therapies.¹⁾ Although pericardial effusion is often associated with poor outcomes, direct cancer invasion of the pericardium comprises less than half of all cases, while up to two-thirds of pericardial effusions are caused by etiologies other than direct cancer involvement.²⁾

Concurrent pericardial effusion and pericardial constriction are defined as effusive-constrictive pericarditis. The original definition of effusive-constrictive pericarditis was made based on data of patients undergoing pericardiocentesis in tamponade, in whom the right atrial pressure by invasive measurement did not fall below 50% or 10 mmHg after normalization of the pericardial pressure.³⁾ As cardiac catheterization based diagnosis is not easy to perform in current clinical practice, the presence of typical echocardiographic Doppler findings of constrictive physiology (CP) after pericardiocentesis could be a clue for effusive-constrictive pericarditis.⁴⁾ Effusion-constrictive pericarditis diagnosed by echocardiography is increasingly recognized in patients with pericardial effusion⁵⁾ as well as in those with cancer.⁶⁾⁷⁾

In this issue of the Journal, Park et al.⁸⁾ reported the characteristics and outcomes of 133 cancer patients who underwent pericardiocentesis with comprehensive echocardiography before and after. In this population, 36.8% of patients had CP after pericardiocentesis. Patients with post-pericardiocentesis CP showed the following characteristics: (1) features suggestive of pericardial inflammation: pericardial enhancement, fever, ST segment elevation, higher C-reactive protein level, and higher pericardial fluid leukocyte counts; and (2) features suggestive of cancer invasion, including a malignant mass abutting the pericardium and positive cytology. The most prominent echocardiographic characteristic before the pericardiocentesis was a higher septal e' velocity (mean value, 8.6 cm/s) in patients with post-pericardiocentesis CP who eventually experienced worse overall survival than those without CP. The authors concluded that CP frequently develops after pericardiocentesis, is associated with poor survival in cancer patients, and can be predicted by clinical, imaging, and laboratory findings before pericardiocentesis.

Data Sharing Statement

The data generated in this study is available from the corresponding author(s) upon reasonable request.

The contents of the report are the author's own views and do not necessarily reflect the views of the *Korean Circulation Journal*.

Currently, however, more investigations are needed on the clinical significance of CP after pericarditis. First, simultaneous echocardiography–cardiac catheterization is still critically needed to better understand the underlying hemodynamics of CP after pericardiocentesis. Echocardiography is likely to identify patients with slight hemodynamic alterations. Mild respiratory septal shift or minor variations in early diastolic mitral velocities are not uncommon among patients with cardiac tamponade and normalization of jugular venous pressure after pericardiocentesis.⁴⁾ Therefore, caution should be given to diagnosing CP after pericardiocentesis in patients with subtle constriction findings on echocardiography, especially those with a normal-sized inferior vena cava and normal jugular venous pressure.⁴⁾ Second, it was previously reported that CP after pericardiocentesis showed a differential predictive impact on outcomes according to the pericardial fluid cytology results.⁶⁾ The presence of CP in patients with negative cytology findings conferred the most favorable outcome, which was even better than that of patients with negative cytology and no CP.⁶⁾ This implies that according to etiologies post-pericardiocentesis CP might result in different outcomes. Therefore, different therapeutic strategies might be required depending on the etiology of the CP, as it can result from completely different causes such as pericardial inflammation or cancer invasion. Third, it remains to be determined whether pre- and post-pericardiocentesis CP can be used to guide therapy in cancer patients undergoing pericardiocentesis. In a single-center study, Kim et al.⁷⁾ reported that cancer patients with a malignant pericardial effusion who received colchicine after successful pericardiocentesis showed significant improvement in clinical outcomes among a population showing pericardial adhesion or constriction in 80.3% of patients on post-pericardiocentesis echocardiography. Therefore, further investigations of echocardiographic findings of CP or adhesions after pericardiocentesis are needed to readily identify those who would benefit from anti-inflammatory therapies such as colchicine, non-steroid anti-inflammatory drugs, or steroids.

In conclusion, the study by Park et al.⁸⁾ provides valuable insights about echocardiography before and after pericardiocentesis in patients with cancer and pericardial effusion. Since they showed that cancer patients with post-pericardiocentesis CP were associated with poor survival and high levels of inflammatory markers, further larger studies are needed to unveil the clinical role of echocardiographic CP and define whether it can guide therapeutic strategies in cancer patients with pericardial effusion.

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