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Difficult diagnosis of co-existence of constrictive pericarditis and cardiomyopathy due to rheumatoid arthritis

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Introduction: Constrictive pericarditis (CP) is the most common cardiac involvement of rheumatoid arthritis (RA). Heart failure (HF) may develop due to CP through pericardial stiffening that hampers diastolic ventricular filling. Cardiomyopathy (CM) is another manifestation of RA and may also cause HF. RA-associated cardiomyopathy is complex and it is not necessarily easy to assess its association and the type of cardiomyopathy accurately. This is particularly true if the patients with RA had both CP and CM because these cause in similar hemodynamic alteration. We present such a case.

Case: A 62-year-old female patient with a history of RA was administered prednisolone and tacrolimus for 8 years, and was considered to have associated CP 5 years ago. Her pleural effusion (PE) and dyspnea on exertion (DOE) due to CP was incremental recent several months, and a work-up for surgical treatment was initiated. She was in sinus rhythm with a heart rate of 88. Her jugular venous pressure with sitting-up position increased to the level of the right earlobe. Laboratory findings showed normal renal function with brain natreutic peptide (BNP) of 345 pg/ml. The PE was transudative. Transthoracic echocardiology (TTE) revealed a septal bounce. Chest CT showed thickened pericardium. Invasive hemodynamics showed dip and plateau pattern in the bi-ventricles. According to these results, she underwent pericardiectomy. Histological finding of the resected pericardium was obviously fibrous thickening with calcification, supporting the diagnosis of CP. After the surgery, she had residual DOE with a further elevation in BNP (818 pg/ml). TTE showed a disappearance of the septal bounce without LV dilatation. Invasive hemodynamics was re-assessed a month after the operation. Mean pulmonary artery wedge pressure was 25 mmHg, mean right atrial pressure, 22 mmHg, right ventricular end-diastolic pressure, 23 mmHg, and left ventricular end-diastolic pressure, 29 mmHg, respectively. There was no ventricular interdependence by Valsalva maneuver. Histological finding of the myocardial biopsy showed moderate interstitial fibrosis with inflammatory cell infiltration, suggesting CM due to RA. Then, she received a titration of drugs for heart failure.

Discussion and Conclusions: This case was initially considered to have only CP because there was clear evidence of CP in imaging modalities such as echocardiography and cardiac CT, and because hemodynamic alterations were explained solely by the presence of CP. However, there was little improvement in HF symptoms after surgical treatment, and the association of CM became evident. A slight increase in BNP level may have been only a clue of the association of CM with CP before surgery in this particular case. Future studies are definitely required to assess the presence or absence of CM and CP in a sophisticated fashion in patients with RA.