Annulus Reversus Caused by Transmural Scar in a Patient With Myopericarditis

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INTRODUCTION

Pericarditis, an inflammation of the pericardial sac, can be caused by infectious, autoimmune, metabolic, traumatic, iatrogenic, and drug-related entities.[1,2] Not infrequently, the inflammatory process also involves the contiguous myocardium, with associated elevation in cardiac enzymes. In some instances, pericarditis is associated with constrictive pericarditis, the diagnosis of which can be challenging. Cardiac ultrasound is critical to the diagnosis of constrictive pericarditis.[3] The finding of annulus reversus—the medial early diastolic tissue Doppler velocity exceeding the lateral velocity—can be of particular diagnostic value.[4] It is hypothesized that this finding is due to a reduction in the longitudinal motion of the lateral annulus by the constrictive diathesis.[4] In the case we are presenting herein, we demonstrate an example of annulus reversus whose origin may be related more to myocardial scar than to the inflammatory changes in the pericardium.

CASE PRESENTATION

A 30-year-old woman with a medical history notable for recurrent myopericarditis and pericardial effusion of unclear etiology presented to the emergency department with left shoulder pain of 3 days' duration. Three weeks prior, the patient had undergone Roux-en-Y gastric bypass for gastric outlet syndrome. Surgical complications were ultimately ruled out as a cause for the discomfort, and the patient was thereupon admitted to the cardiology service. Laboratory tests showed elevated troponin I (5.66 ng/mL; normal, 0.01-0.04), brain natriuretic peptide (323 pg/mL; normal, <100), and D-dimer (3.83 mg/L; normal, <0.5). Inflammatory markers were elevated as well: erythrocyte sedimentation rate was 25 mm/hour (normal, <20), and C-reactive protein was 156 mg/L (normal, <10). Electrocardiogram revealed sinus tachycardia with nonspecific inferolateral changes, and a Q wave in lead aVL (Figure 1). Chest computed tomography angiography ruled out pulmonary embolism but revealed a moderate pericardial effusion and small left pleural effusion.

Transthoracic echocardiography (TTE) findings included left ventricular (LV) ejection fraction of 60% with hypokinetic to akinetic anterior and anterolateral LV walls that appeared tethered to the pericardium (Video 1). The latter was thought to be associated with an inflamed lateral pericardium. Tissue Doppler velocities were medial e′ 10 cm/sec and lateral e′ 5 cm/sec, consistent with annulus reversus (Figure 2); however, there was no major respirophasic variation of mitral or tricuspid inflow velocities, nor was there a respirophasic septal shift. Myocardial deformation analysis showed global longitudinal strain of −10.8%, with greater involvement of basal and mid anterolateral and inferolateral segments (Figure 3). Cardiac magnetic resonance (cMR) imaging showed LV ejection fraction of 50%, global hypokinesis with sparing of septal segments, and late gadolinium enhancement (LGE) involving the pericardium and myocardium of basal lateral, apical lateral, and apical segments of the LV consistent with transmural scarring (Figure 4).

The clinical presentation, biomarkers, TTE, and cMR findings were strongly suggestive of myopericarditis, and the patient was therefore treated with colchicine 0.6 mg twice a day with improvement in symptoms. Repeat troponin I, erythrocyte sedimentation rate, and C-reactive protein were within normal limits; a complete blood count and metabolic panel were unremarkable. Three months after the initial presentation, cMR showed mild LGE improvement of basal lateral and apical lateral segments, with disappearance of pericardial enhancement. Rheumatologic workup was negative for underlying inflammatory processes.

DISCUSSION

The identification of annulus reversus has been posited to be of diagnostic help in suspected constrictive pericarditis.[4] It has been hypothesized that tethering of adjacent scarred pericardium mainly affects the lateral wall.[4] Thus, an inflammatory process, which might create adhesions between the pericardium and lateral wall of the LV, might reduce the longitudinal motion of the lateral annulus relative to that of the septal annulus. The case of annulus reversus presented herein shows that there was also clinically occult *myocardial* dysfunction in a patient with pericardial disease, the full extent of which required assessment by multimodality imaging.

That myocardial as well as pericardial disease might be present among patients with annulus reversus has been suggested in the past. For example, pericardiectomy does not completely eliminate this finding. Patil *et al*[5] described resolution of annulus reversus in only 50% of patients with chronic constrictive pericarditis who underwent pericardiectomy, despite evidence of a favorable clinical response. This strongly suggests the existence of an underlying localized lateral myocardial scar, which was first described in electrocardiographic studies.[6] A recent animal study confirmed the presence of myocardial fibrosis in an experimental model aimed to primarily induce pericardial constriction.[7] Moreover, myocardial dysfunction

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Keywords: Annulus reversus, Constrictive pericarditis, Transthoracic echocardiography, Global longitudinal strain, Cardiac magnetic resonance imaging

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VIDEO HIGHLIGHTS

Video 1: Two-dimensional echocardiographic video clip of 4-chamber view zoomed at left ventricle shows hypokinesis to akinesis of anterolateral wall and possible adhesion to adjacent pericardium.

View the video content online at www.cvcasejournal.com.

appears to play an important role in the pathophysiology of constrictive pericarditis, mainly due to subepicardial involvement of the pericardial process and localized myocardial ischemia, in the setting of high transmural pressures and impairment of coronary blood flow.[8] Since the ventricular apex remains relatively stationary throughout the cardiac cycle, mitral annular assessment allows for measurement of overall longitudinal LV function.[9] Therefore, annular tissue Doppler, which measures peak myocardial velocities, may be abnormal in lateral annular dysfunction due to an intrinsic myocardial process or pericardial disease with myocardial involvement such as myopericarditis; we believe that to be the case here.

Additional etiologies primarily affecting lateral e′ velocity include processes involving the LV lateral wall such as conduction delay, lateral wall myocardial infarction with transmural scar, and predominant lateral mitral annular calcification. Although "annulus reversus" is a term conventionally used when referring to reversal of normal annular tissue Doppler velocity in constrictive pericarditis, it may also be seen in any of these conditions if the LV lateral wall is severely affected, decreasing lateral e′ below medial e′ velocity. Nonetheless, identification of a medial e′ velocity ≥ 9 cm/sec was associated with an optimal receiver operating characteristic curve for the diagnosis of constrictive pericarditis in the largest comparative trial to date. It has been conjectured that the enhanced medial velocity may be due to an exaggerated longitudinal motion of the medial mitral annulus as lateral expansion is limited by the constrictive process.[10,11] It is critical to point out that annulus reversus is not, by itself, diagnostic of constrictive pericarditis, and highlights the importance of searching for other classic echocardiographic findings, such as ventricular septal shift, hepatic vein expiratory diastolic reversal, and inspiratory changes in mitral and tricuspid valve inflow velocities.[10]

We believe that our case shows the following evidence for a primary myocardial process contributing to annulus reversus:

- Transmural scar in the lateral segments by cMR.
- Akinetic lateral LV segments by two-dimensional TTE and myocardial strain analysis corresponding to those identified as scarred by cMR.
- The absence of ventricular interdependence by Doppler analysis, mitral and tricuspid valve inflow velocities with inspiration, and expiratory diastolic reversal of hepatic vein flow.

To reiterate, we believe that the relative lack of longitudinal motion of the lateral wall in this patient was caused by inflamed and scarred

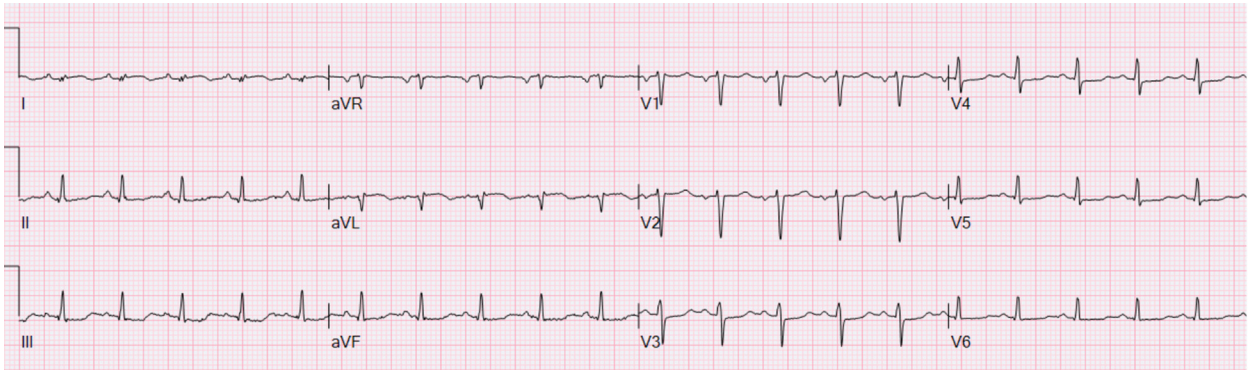

Figure 1 Initial electrocardiographic tracing revealed sinus tachycardia and nonspecific ST-T changes in leads II, III, aVF, V4-V6. Note the absence of generalized ST-segment elevations or PR-segment depressions. There is a prominent Q wave in lead aVL.

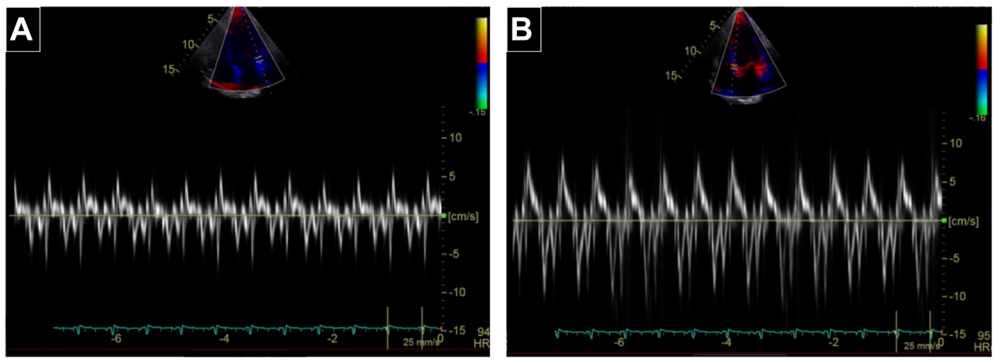

Figure 2 Tissue Doppler velocity analysis of medial and lateral early diastolic mitral annular velocities shows lateral e' velocity of 5 cm/sec **(A)** and medial e' velocity of 10 cm/sec **(B)**, a reversal of normal findings, known as 'annulus reversus.'

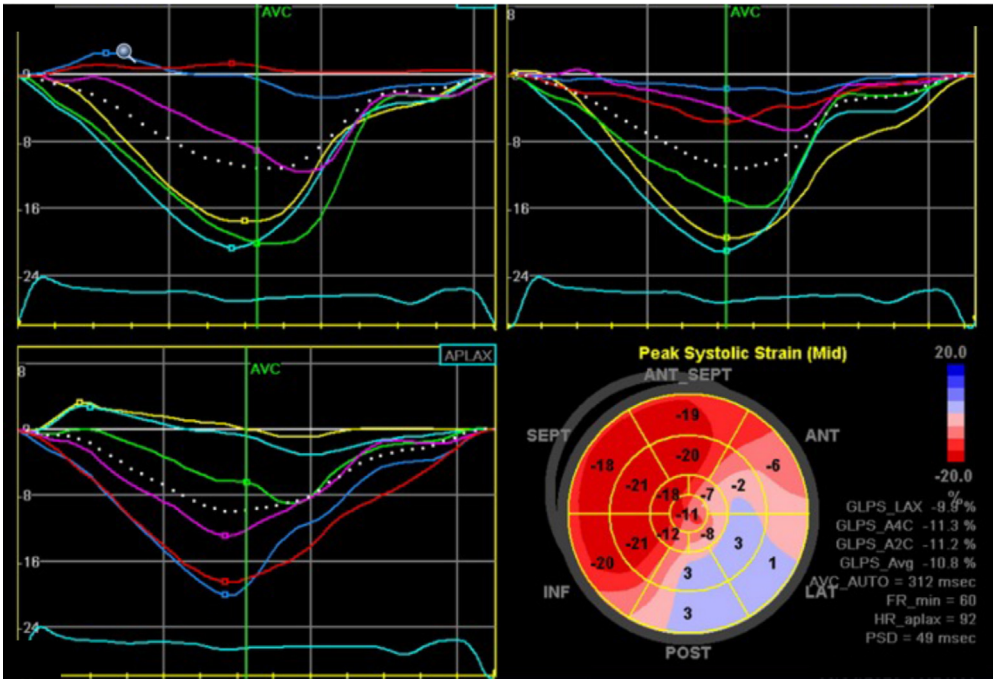

Figure 3 Myocardial strain analysis showing global longitudinal strain of –10.8%. There is relative sparing of septal segments with abnormal strain more pronounced in basal and mid anterolateral and inferolateral LV segments. Longitudinal strain is markedly abnormal in the lateral segments, indicated by *blue color*, signifying systolic expansion.

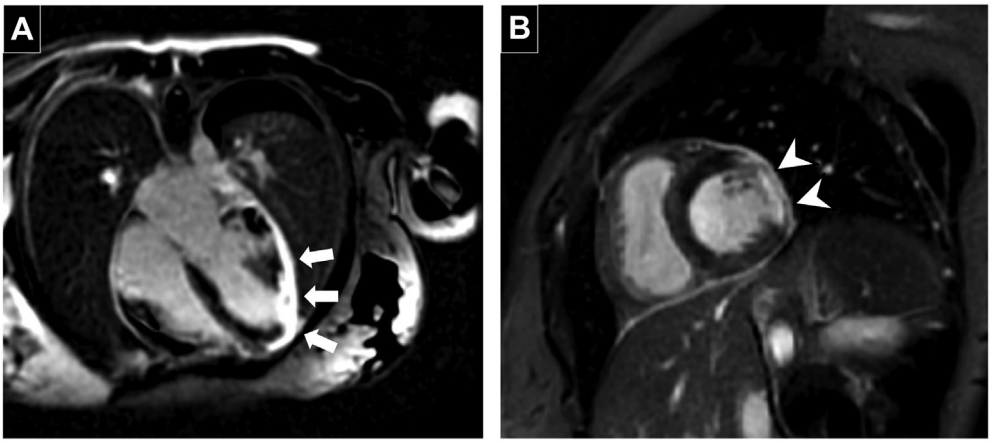

Figure 4 Cardiac magnetic resonance imaging. **(A)** Four-chamber view shows diffuse LGE more prominent at both the visceral and parietal pericardium, as well as basal anterolateral, apical lateral, and apical segments. The basal lateral and apical lateral segments show areas of transmural hyperenhancement *(arrows)*. **(B)** Short-axis view shows patchy transmural hyperenhancement of the mid anterolateral segment, with LGE of adjacent pericardium *(arrowheads)*.

myocardium, rather than a primary pericardial process, and highlights the value of multimodality imaging in some patients with presumed constrictive pericarditis. The case also points out that annulus reversus can be due, at least in part, to a primarily myocardial injury.

CONCLUSION

We report a complex clinical case of a patient found to have annulus reversus on tissue Doppler velocity analysis that was thought to be caused by myocarditis and scarring. Further evaluation with multimodality imaging after identification of annulus reversus may reveal alternative underlying pathophysiology.

ETHICS STATEMENT

The authors declare that the work described has been carried out in accordance with The Code of Ethics of the World Medical Association (Declaration of Helsinki) for experiments involving humans.

CONSENT STATEMENT

The authors declare that since this was a non-interventional, retrospective, observational study utilizing deidentified data, informed consent was not required from the patient under an IRB exemption status.

FUNDING STATEMENT

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DISCLOSURE STATEMENT

The authors report no conflict of interest.

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SUPPLEMENTARY DATA

Supplementary data to this article can be found online at https://doi.org/10.1016/j.case.2022.11.002.

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