Regional Pericarditis Status Post Cardiac Ablation: A Case Report

Dear Editor,

We sincerely appreciate the response to our recently published article[1] regarding regional pericarditis as it is a very difficult diagnosis and requires the exclusion of the more common etiologies of chest pain and ST-segment elevation.

Numerous articles[2-6] have confirmed the fact that the frequency of pericarditis would be underestimated if required the diagnosis of a pericardial rub; however, a pericardial rub is often present but transient in nature and is often not heard during physical exam. Although a pericardial rub was not appreciated during physical exam of our patient, it very well could have been, and likely was, present at some point during hospitalization.

Transmural post-infarction regional pericarditis (epistenocardiac pericarditis) is the most common and documented etiology resulting in regional pericarditis.[7] For this reason, our case is of great interest, as in our case regional pericarditis is due to post-atrial fibrillation ablation resulting in transmural myocardial cell death.

The electrocardiogram (ECG) prior to cardiac ablation was relatively unremarkable [Figure 1]. Patient underwent elective atrial fibrillation ablation. During the ablation, aggressive radiofrequency, and cryoablation of the pulmonary veins, left atrial posterior wall, and the left and right isthmus was required for adequate treatment of the arrhythmia. The patient was then discharged home 24 hours after the ablation. Shortly after returning home, patient developed chest pain and presented to the emergency department 36 hours after the procedure. At time of presentation, the patient had an elevated troponin of 17.2 ng/ml, which may be consistent with a post-cardiac ablation.[8] The ECG at time of presentation to the emergency department revealed normal sinus rhythm with a heart rate of 78 beats per minute with anterolateral ST-segment elevations in leads I and aVL and V2-V4, reciprocal inferior ST-segment depressions in leads III and aVF and subtle PR-segment depressions [Figure 2]. However, after coronary-artery angiography revealed no significant obstructive coronary artery disease, the ECG revealed resolving ST-segment depressions in the inferior leads and increased ST-segment elevation in leads V5-V6 [Figure 3] consistent with regional pericarditis. Furthermore, a 2D echocardiogram...
was performed prior to discharge, and after further review from the previous article,[1] revealed a left ventricular ejection fraction of 55-60%, no wall motion abnormalities and a new trace regional pericardial effusion around the posterolateral wall compared to the echocardiogram two days prior during the pre-procedural ischemic work-up [Figure 4]. Two-week follow-up ECG revealed normal sinus rhythm with heart rate of 81 beats per minute with a left-posterior fascicular block and no ST-segment abnormalities other than T-wave inversions isolated in leads I and aVL [Figure 5]. This is consistent with a pericarditis as T-waves may become inverted around the third week and resolve within several weeks thereafter.[6]

A 3-month follow-up ECG revealed a normal sinus rhythm with heart rate of 82 beats per minute with a left posterior fascicular block and resolution of non-specific T-wave abnormalities [Figure 6].

Oliva et al.,[3-5] explained the T-wave evolution and morphology following a transmural myocardial infarction related regional pericarditis. It is known that T-wave inversions are seen during an ischemic event and accompany myocardial infarction.[3-5] However, in post-infarction regional pericarditis, the T-waves either remain positive for 48 hours despite infarction or they undergo pre-mature gradual reversal of inversion.[2-5] Our patient did not have T-wave inversions at time of presentation or at time of discharge. This strongly suggests that myocardial ischemia was not the culprit for this case of regional pericarditis.

Coronary vasospasm was ruled out as seen on coronary angiography [Figure 7]. This is a likely differential diagnosis when considering regional pericarditis; however, we did not feel coronary angiography was suggestive of such a phenomenon.

Cardiac magnetic resonance imaging (CMR) is an appropriate and great diagnostic tool and would have been beneficial in this case to confirm the diagnosis of regional pericarditis, but may not have assisted in the etiology.[7,9]

In conclusion, all evidence as mentioned within is suggestive of a regional myo-pericarditis secondary
to transmural myocardial cellular death due to an aggressive atrial-fibrillation ablation, which resolved with conservative therapy for traditional pericarditis.

Joseph Orme, Moneer Eddin¹, Akil Loli¹
Departments of Internal Medicine, and ¹Cardiology, Banner Good Samaritan Medical Center, Phoenix, Arizona, USA.
E-mail: Geoffrey.orne@bannerhealth.com

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