Shot to the Heart: Cardiac Manifestations of SLE

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CardioCase presentation

Nolan’s Pain

Nolan, 56, is active with a history of:
• hypertension,
• hypercholesterolemia and
• a family history of premature coronary artery disease.

He presents to the hospital with a gradual onset of retrosternal, oppressive chest pain of four hours duration. The pain is non-radiating with a pleuritic component and is associated with diaphoresis and mild dyspnea.

He reports a three-month history of fatigue and arthralgia.

On arrival at the emergency department (ED), Nolan’s symptoms have resolved, following treatment with oxygen and sublingual nitroglycerin administered by emergency medical services.

- Heart rate: 69 beats per minute (bpm)
- Blood pressure:
  - Right arm: 132/84 mmHg
  - Left arm: 136/80 mmHg
- Respiratory rate: 12 breaths per minute
- Oxygen saturation in arterial blood: 97% on room air
- Body temperature: 37°C

His cardiac exam is unremarkable.

Nolan’s left calf is swollen; when measured, it is found to be 3 cm greater in circumference than the right calf.

An electrocardiogram (ECG) is performed (Figure 1).

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Figure 1. Nolan’s ECG upon presentation.
CardioCase presentation

Nolan’s Pain continued...

Nolan’s ECG (Figure 1) shows normal sinus rhythm, with non-specific ST-T wave changes. A chest X-ray is performed (Figure 2) and is normal.

Nolan is admitted to the care of the Cardiac Care team and treated with acetylsalicylic acid (ASA), intravenous heparin, metoprolol, ramipril and simvastatin. Serial cardiac enzymes are drawn and are all within normal limits. Because of the unilateral swollen calf, a D-dimer is performed (the results are negative), as well as a duplex ultrasound, which does not reveal any evidence of deep vein thrombosis.

Because of the strong pre-test probability of coronary disease, and a presentation consistent with a potential acute coronary syndrome, arrangements are made for an in-patient cardiac catheterization. The cardiac catheterization reveals normal epicardial coronary arteries, with no angiographic evidence of coronary atherosclerosis. His ejection fraction is estimated to be normal at 60%. Nolan does not suffer any further episodes of in-hospital pain and is discharged to follow up with his family physician.

Two days later, Nolan suffers worsened chest pain and returns to the ED. He describes the pain as sharp and pleuritic in nature, with relief by sitting up and leaning forward.

- Heart rate: 85 bpm
- Blood pressure: 162/98 mmHg (the right side is equal to the left)
- Respiratory rate: 10 breaths per minute
- Oxygen saturation in arterial blood: 99% on 3L O₂
- Body temperature: 38°C
- Pulsus paradoxus: 20 mmHg

A physical examination reveals new telangiectasias on the face, with a normal cardiac exam. The heart sounds are not muffled, the jugular venous pressure is not distended and there are no discernible murmurs or cardiac rubs. On his respiratory exam, dullness to the left base with a pleural rub is noted.

An ECG is performed and reveals a normal sinus rhythm, with no change from the previous ECG. A chest X-ray reveals a left-sided pleural effusion and increased cardiac silhouette (Figure 3).

For more on Nolan, go to page 24.

What’s your CardioCase diagnosis?

CardioCase discussion

What’s wrong with Nolan?

Cardiac manifestations of systemic lupus erythematosus (SLE) are varied and common (Table 1). As many as 75% of patients with SLE will have a degree of cardiac abnormality at some point during the course of their disease. While many of the underlying cardiac associations are clinically silent, important associations...
exist. The cardiac manifestations of SLE do not necessarily reflect the duration, severity nor activity of disease and may precede the clinical signs and diagnosis of SLE (including benign pericarditis).

Valvular heart disease is the most common cardiac manifestation, followed by:
• pericardial disease,
• coronary artery disease (CAD),
• myocardial disease and
• conduction abnormalities.

Although valvular disease is the most common association, pericarditis is the most common symptomatic cardiac manifestation in SLE. Thromboembolic disease is another possible manifestation of SLE, and must be considered in patients presenting with chest pain, especially in patients with antiphospholipid antibodies (who are predisposed to vascular thrombosis). Pulmonary hypertension occurs with increased frequency in patients with SLE.

**Coronary artery disease**

CAD is common in patients with SLE. An increased incidence of traditional CAD risk factors have been noted in patients with SLE. However, the increased risk of CAD cannot be solely associated with traditional risk factors. Exposure to:
• glucocorticoids,
• endothelial dysfunction,
• platelet hyperactivity and
• impaired fibrinolysis

have all been implicated in the increased incidence of CAD in this population. In addition to atherosclerosis, patients with SLE may suffer myocardial events from coronary artery vasculitis and coronary emboli.

**Table 1**

<table>
<thead>
<tr>
<th>Common cardiac manifestations of SLE</th>
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<tbody>
<tr>
<td>• Mitral regurgitation (12% to 38%)</td>
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<td>• Mitral valve prolapse (25%)</td>
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<td>• Verrucous (Libman-Sacks) endocarditis (10%)</td>
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<tr>
<td>• Pericardial effusion (55%, often silent)</td>
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<tr>
<td>• Myocarditis (8% to 25%)</td>
</tr>
<tr>
<td>• Coronary artery disease (up to 45%)</td>
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<tr>
<td>• Conduction abnormalities, most commonly 1st AVB (34% to 70%)</td>
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SLE: Systemic lupus erythematosus
AVB: Atrioventricular block

**Infective endocarditis**

Infective endocarditis is more common in patients with SLE. Following the American Heart Association guidelines, patients with significant valvular lesions should be considered for bacterial endocarditis prophylaxis.

**DID YOU KNOW...**

A family history is considered to be positive for early coronary artery disease (CAD) if an individual has a first degree male relative younger than 55 years of age or a first degree female relative younger than 65 years of age with CAD.

**DID YOU KNOW...**

Echocardiography is recommended in all cases of acute pericarditis where the cause is unknown, but especially if cardiac tamponade or purulent pericarditis is suspected.
prior to invasive procedures with a risk of bacteremia. Because of the frequency of valvular abnormalities in patients with SLE, some experts advocate that all patients with SLE undergoing invasive procedures be considered for antibiotic prophylaxis. However, consensus on this strategy does not yet exist.

**Pericarditis**

The course of acute pericarditis in patients with SLE is typically benign. Symptomatic pericarditis often responds well to nonsteroidal anti-inflammatory agents (NSAIDs). As in other forms of pericarditis, patients who fail to respond or who are intolerant to an NSAID can be treated with prednisone and he was discharged with Rheumatology follow-up.

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### More about Nolan

Nolan’s presentation was consistent with pleuro-pericarditis. Because of the pulsus paradoxus noted on his exam, a concern existed regarding possible cardiac tamponade and an urgent echocardiogram was requested. Transthoracic echocardiography revealed a moderate-sized pericardial effusion, without any echocardiographic evidence of cardiac tamponade.

Nolan was admitted to the Cardiac ward for pain management and further work-up. His chest pain was treated with standard antipericarditis therapy—ASA, 650 mg, orally, four times daily. His pericarditic pain quickly resolved.

Because of the prodrome of fatigue, arthralgias, photosensitivity and current serositis, the Rheumatology team was consulted to investigate for a possible autoimmune etiology. Serologic testing revealed a positive anti-nuclear antibody (1:160) and a positive anti-phospholipid antibody. Nolan was diagnosed with systemic lupus erythematosus (SLE). Under the guidance of Rheumatology, his SLE was treated with azathioprine and prednisone and he was discharged with Rheumatology follow-up.

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### DID YOU KNOW...

**Etiology of pericarditis includes:**
- Viral
- Auto-immune
- Neoplastic
- Tuberculosis
- Uremic
- Idiopathic

Resources

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### About the authors...

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