

An unusual case of constrictive pericarditis

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CASE

A 61-year-old retired clerical worker, WM, presented to his family doctor in 2003 with a several month history of worsening exertional dyspnea and abdominal bloating. Previous medical history consisted of childhood pneumonia and a twenty-seven pack-year smoking history, having quit twenty years previously. His previous chest x-rays had shown bilateral pleural thickening and plaque formation, suggestive of prior asbestos exposure acquired while working for thirteen years in a foundry.

On examination, abdominal distension and ascites were noted. A thorough cardiac work-up was ordered, including a CT and echocardiogram. The CT revealed more extensive pleural plaques than shown in previous chest x-rays, with significant pleural effusions and some pleural calcification. The pericardium was also noted to be thickened. The echocardiogram with colour Doppler suggested the diagnosis of constrictive pericarditis (CP). A subsequent cardiac catheterization indicated significant coronary artery disease (75% stenosis in the left anterior descending artery) and a hemodynamic profile consistent with constrictive pericarditis. MRI identified pericardial thickening measuring 5 mm in thickness, along with pleural thickening and calcification.

Thoracentesis revealed that the pleural effusions were benign. As WM was feeling increasingly fatigued and short of breath by early 2004, he elected for surgery. He underwent a total pericardiectomy, however his LAD stenosis was not bypassed as his tissue was considered very friable. Subsequent pathology of the pericardial tissue revealed severe chronic inflammation and fibrosis consistent with constrictive pericarditis. WM had an immediate improvement in respiratory status and energy levels post-operatively. He submitted a claim to the Workplace Safety and Insurance Board of Ontario (WSIB) for compensation for asbestos exposure resulting in constrictive pericarditis. The claim was subsequently accepted and awarded by the WSIB in 2005.

WHAT IS CONSTRICTIVE PERICARDITIS?

The pericardium is a fibroelastic sac that surrounds the heart. In constrictive pericarditis (CP), scarring and fibrotic changes alter the pericardium and prevent it and the heart from expanding.

The majority of constrictive pericarditis cases are idiopathic or post-cardiac surgery or post-radiation therapy in etiology.¹⁻³ In the developing world, post-infectious causes (i.e. tuberculosis or purulent pericarditis) are most common. A handful of cases – only 12 reported in literature⁴ within the last twenty years – have been related to asbestos exposure.

HOW DO WE RECOGNIZE CONSTRICTIVE PERICARDITIS?

As always, the history and physical exam play an important role in the diagnosis. Knowing that WM had pneumonia as a child may help estab-

lish associated artifacts that may appear on chest x-ray (e.g. granuloma from TB), but isolating the etiology would not help with definitive diagnosis or treatment in this case. CP patients often report symptoms of fluid overload and diminished cardiac output³ in response to exertion. For instance, WM had been referred to a cardiologist when he experienced ascites, and his symptoms of shortness of breath and fatigue were present up until his surgery. On physical exam prior to his surgery, WM had only two of the six most common signs of CP,³ which include elevated JVP, ascites, peripheral edema, pulsus paradoxus, Kussmaul's sign, and pericardial knock. His CT scan also revealed the fairly common CP finding of pleural effusions.

HOW DO WE DIAGNOSE CONSTRICTIVE PERICARDITIS?

The initial steps of any cardiac work-up usually include EKG and chest x-ray. EKGs are not very useful for diagnosing CP, as there are no pathognomonic electrographic findings for CP.⁴ Pericardial calcification may occasionally be caught on x-ray, however this is a rare finding.⁵ Transthoracic echocardiography is the diagnostic test of choice for CP.⁶ However, invasive hemodynamic evaluation during cardiac catheterization is often used to confirm the diagnosis, as was done with WM.

WHAT IS THE TREATMENT FOR CONSTRICTIVE PERICARDITIS?

Although total pericardiectomy is the definitive treatment for chronic CP, diuretics can be used as a temporary measure. WM's abdominal ascites was controlled by diuretic therapy for several months until surgery. Total pericardiectomy is the treatment of choice, but partial pericardiectomy has also been conducted for some CP patients with prior asbestos exposure.⁷

CONSTRICTIVE PERICARDITIS DUE TO ASBESTOS

WM is believed to be the first patient in Sarnia, Ontario to have developed CP in association with asbestos-related disease – one of only a handful reported in the medical literature. Without the concurrent development of pleural plaques, which are indicative of asbestos exposure, it is likely that the etiology of his CP would have remained idiopathic. Indeed, were it not for the increased prevalence of asbestos-related diseases in Sarnia, making both healthcare teams and city residents more aware of its consequences, the association of CP with asbestos exposure may have been missed altogether.

Several recent autopsy reports of workers at the Sarnia Occupational Health Clinic for Ontario Workers have described pericardial fibrosis with asbestos-related lung diseases and it is possible this finding is more prevalent than is realized. While asbestos-related pleural plaques will often increase physician's vigilance for asbestosis, lung cancer, and mesothelioma, it is worth considering the possibility of pericardial fibrosis and CP in workers with cardiorespiratory symptoms.

Although the medical literature has linked asbestos exposure with CP, major pieces of the puzzle have not been elucidated. It is unknown whether CP patients with prior asbestos exposure possess unique signs or symptoms that differentiate them from the majority of patients with CP. A more extensive case series could be conducted for clarification.

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