*Case Reports*

# Posttraumatic Subacute Effusive-Constrictive Pericarditis

## After a Motor Vehicle Accident

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*Effusive-constrictive pericarditis is typically caused by tuberculosis or other severe inflammatory conditions that affect the pericardium. We report a case of effusive-constrictive pericarditis consequent to a motor vehicle accident.*

*A 32-year-old man with gastroesophageal reflux disease presented with severe substernal chest pain of a month's duration and dyspnea on exertion for one week. Echocardiograms revealed a moderate pericardial effusion, and the diagnosis was subacute effusive-constrictive pericarditis. After thorough tests revealed nothing definitive, we learned that the patient had been in a motor vehicle accident weeks before symptom onset, which made blunt trauma the most likely cause of pericardial injury and effusion. Medical management resolved the effusion and improved his symptoms.*

*To our knowledge, this is the first report of effusion from posttraumatic constrictive pericarditis associated with a motor vehicle accident. We encourage providers to consider recent trauma as a possible cause of otherwise idiopathic pericarditis.* ***(Tex Heart Inst J 2020;47(3):233-5)***

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Effusive-constrictive pericarditis (ECP) can develop after certain cardiac injuries. Classically, a pericardial effusion causes enough hemodynamic compromise to warrant pericardiocentesis.[1] This sequence promotes pericardial remodeling in a manner that reduces pericardial compliance and causes a constrictive physiologic state.[1] Effusive-constrictive pericarditis occurs rarely and is typically seen in high inflammatory states such as tuberculosis infection; however, it has also been associated with hemopericardium. We report a case of ECP consequent to trauma from a motor vehicle accident (MVA).

## Case Report

In late 2018, a 32-year-old man with attention deficit hyperactivity disorder, chronic allergic rhinitis, and gastroesophageal reflux disease (GERD) had experienced persistent substernal chest pain for one month. He had no family history of cardiovascular disease, nor did he smoke. A few weeks of pantoprazole therapy had brought little relief. The patient's pain was presumed to be from uncontrolled GERD, and he was scheduled for elective esophagogastroduodenoscopy. When presenting for that procedure, he reported a week's history of progressive dyspnea on exertion (most noticeable when he climbed stairs) and that his substernal pain worsened when he lay flat and improved when he arose. He had not experienced this magnitude of either symptom before. The patient's heart rate of 120 to 130 beats/min prompted his referral to the emergency department.

On evaluation, the patient had tachycardia, a blood pressure of 102/64 mmHg, distant heart sounds, a sedimentation rate of 67 mm/hr, a C-reactive protein level of 317.2 mg/L, a cardiac troponin I level <0.03 ng/mL, and a brain-type natriuretic peptide level of 80 pg/mL. An electrocardiogram showed diffuse PR depressions. A transthoracic echocardiogram (TTE) showed thickened pericardium with a pericardial and left pleural effusion (Fig. 1A). The diagnosis was constrictive pericarditis.

The patient was admitted to the hospital, and therapy with ibuprofen (600 mg 3×/d) and colchicine (0.6 mg 2×/d) was begun. On hospital day 3, a pericardial friction rub developed, and dyspnea persisted. A TTE showed fibrinous material within the pericardial effusion (Fig. 1B), indicating ECP. Prednisone (60 mg/d) was added

to the patient's therapy, after which his symptoms and physical examination findings improved.

We initially presumed that a viral infection had caused the pericarditis, because the patient reported having had symptoms of an upper respiratory tract infection a week before; however, his rhinitis clouded the clinical picture. Results of respiratory viral, influenza A and B, and coxsackie B panels were negative, as were tests for respiratory syncytial virus, human immunodeficiency virus, and tuberculosis. Extensive autoimmune and inflammatory evaluations also revealed no precipitating factors.

We questioned the patient further and learned that, 3 months earlier, he had been in an MVA; his chest had hit the steering wheel and the airbag had deployed. He remembered having no symptoms beyond shoulder pain and stiffness. We concluded that blunt trauma to the chest had led to the subacute pericarditis and indolent fibrinous effusion.

The patient was discharged from the hospital 5 days later with instructions to continue taking ibuprofen and colchicine as prescribed, as well as a prednisone taper starting at 60 mg/d. A TTE 3 weeks later showed substantial resolution of the pericardial effusion (Fig. 1C). At the one-month follow-up examination, the patient reported symptomatic improvement, and he was able to walk and exercise as usual.

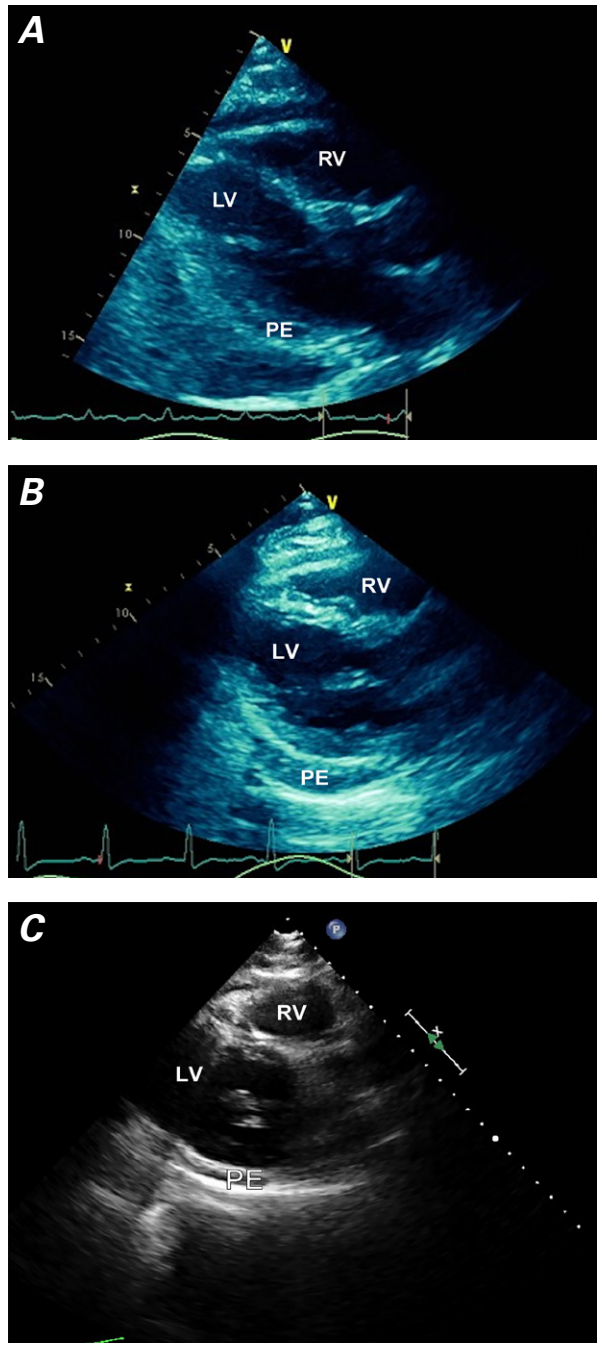


***Fig. 1*** *Transthoracic echocardiograms (parasternal long-axis views) show **A**) thickened pericardium with pericardial and left pleural effusions on admission, **B**) fibrinous material within the pericardial effusion on hospital day 3, and **C**) substantial resolution of the pericardial effusion after 3 weeks of medical therapy.*

*LV = left ventricle; PE = pericardial effusion; RV = right ventricle*

## Discussion

Our patient's ECP was most likely caused by blunt trauma to the chest wall during an MVA. Pericarditis development after any type of pericardial injury, including myocardial infarction, pericardiotomy, and trauma, is often called postcardiac injury syndrome.[2]

What causes postcardiac injury syndrome is unclear. Dressler[3] suggested that injury to the myocardium after infarction produces new antigens, provoking an autoimmune hypersensitivity reaction in susceptible individuals. Similarly, trauma to pleural and pericardial mesothelial cells may enable novel antigens to cause a delayed inflammatory response. In a case series of cardiac surgical patients,[4] anti-heart antibodies were detected in all who had postpericardiotomy syndrome (fever, leukocytosis, and pericarditis). However, these antibodies were also found in 20% of the other patients and were therefore nonspecific.

Traumatic pericarditis is typically iatrogenic, resulting from pacemaker or stent implantation, radiofrequency ablation, or Swan-Ganz catheterization.[2] Pericarditis after blunt trauma is less well described.[5] In one of the 2 cases that we found of posttraumatic pericarditis after an MVA,[6] the pericardial injury went unnoticed for more than 2 weeks.

Constrictive pericarditis usually occurs after cardiac surgery, pericardiocentesis, or end-stage renal disease; however, tuberculosis is another known cause. Less often, injuries from an MVA, falls, and penetrating trauma have led to constrictive pericarditis that became symptomatic after days or weeks.[7] In one patient, the constrictive condition developed 2 weeks after an MVA caused trauma to the chest wall.[8] In another patient, it

developed more than a year after thoracic trauma from a fall onto a trailer hitch.[9]

In the previous cases and in our patient, extensive infectious, inflammatory, and autoimmune evaluations produced negative or inconclusive results. We made the diagnosis of ECP secondary to trauma, confirmed by symptomatic and echocardiographic resolution after triple therapy. We think that this is the first report of effusion in constrictive pericarditis consequent to blunt trauma in an MVA, and we hope that medical teams will consider trauma as an additional possible cause of otherwise idiopathic pericarditis.

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