Delayed Clinical Presentation of Hemorrhagic Pericardial Effusion in a Patient Receiving Warfarin Sodium

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Acute hemorrhagic pericardial effusion is a condition that may coexist with other injuries in patients with multiple trauma. Pericardial tamponade caused by hemorrhagic effusion is an emergent condition that requires prompt treatment.[1] Subacute presentation has been reported,[1] and anticoagulation of a patient with blunt chest trauma may lead to delayed presentation.[2]

In treating patients with multiple trauma, some orthopedic surgeons may assume the role of primary attending physician after other trauma issues are thought to be resolved. Not infrequently, patients with blunt chest injury will also have lower extremity fractures. Anticoagulation may be indicated in these patients, and orthopedic surgeons may become responsible for managing this aspect of patient care. In this scenario, orthopedic surgeons may be the first to encounter signs and symptoms associated with hemorrhagic pericardial effusion or tamponade.

In this report, I present a case of hemorrhagic pericardial effusion with tamponade in a patient in his late 30s with blunt chest trauma and multiple fractures who was anticoagulated with warfarin sodium but did not clinically manifest the effusion until 45 days after injury.

The patient was involved in a motor vehicle accident. He was found to have bilateral rib fractures, right pneumothorax, right sacroiliac joint dislocation, right pubis fracture, symphysis pubis dislocation, T-configured left acetabular fracture, closed left lateral tibial plateau fracture, and grade I open right comminuted bicondylar tibial plateau and shaft fracture.

Initial treatment at an outside facility involved insertion of a right chest tube, percutaneous placement of a right iliosacral screw, anterior pelvic external fixation, percutaneous screw fixation of the left lateral tibial plateau fracture, and débridement and closure of the open right tibial plateau fracture wound with application of a spanning external fixator.

An inferior vena cava filter was placed on hospital day 2. The chest tube was removed on hospital day 4. No anticoagulation was administered. On hospital day 7, the patient was air-transported to LDS Hospital in Salt Lake City, Utah.

On hospital day 8, as a part of a preoperative evaluation, the patient had both pulmonary and cardiology consultations because of complaints of chest and sternal discomfort. The electrocardiogram showed sinus tachycardia (rate, 107) and frequent multifocal premature atrial contractions. The supine chest x-ray showed no pneumothorax, mild cardiomegaly, and an elevated right hemidiaphragm (Figure 1). Rib fractures were not seen. A right subclavian catheter was in the mid superior vena cava. The cardiology consultant noted the ectopy but found no contraindication to further surgery.

On hospital day 9, the patient was taken to surgery for removal of the external fixator and operative repair of the posterior aspect of the left acetabular fracture. A chest x-ray on hospital day 10 showed "mild to moderate enlargement of the cardiac silhouette," and a ventilation-perfusion lung scan done the same day indicated low probability for pulmonary embolus. On hospital day 12, the patient had repair of the right tibial plateau fracture by percutaneous screw fixation. A postoperative chest x-ray report also mentioned generalized cardiomegaly. On hospital day 14, the symphysis pubis and anterior aspect of the left acetabulum were repaired. A chest x-ray on hospital day 19 again showed "mild cardiac enlargement" with the distal tip of the subclavian catheter in the superior vena cava.

Enoxaparin 30 mg subcutaneous every 12 hours was initiated after surgery on hospital day 9 but was withheld on days of subsequent surgeries. Warfarin was started on hospital day 19, after all anticipated surgeries were completed; enoxaparin was stopped when the international normalized ratio (INR) was above 2.0 on hospital day 28. The subclavian catheter was removed on hospital day 24.

The patient was moved to the in-hospital skilled nursing facility floor on hospital day 27, as social issues precluded further convalescence at home. The combination of injuries and repair prevented standing or ambulation. The patient's INR was checked periodically through hospital day 41 while he received a continuous dose of 10 mg warfarin daily. INR values ranged from 2.1 to 2.6.

On hospital day 45, after several weeks of being afebrile, the patient developed a temperature of 38°C. He also developed a slightly productive cough, fatigue, and malaise. On suspicion of a respiratory origin of the symptoms and findings, a chest x-ray and a medicine

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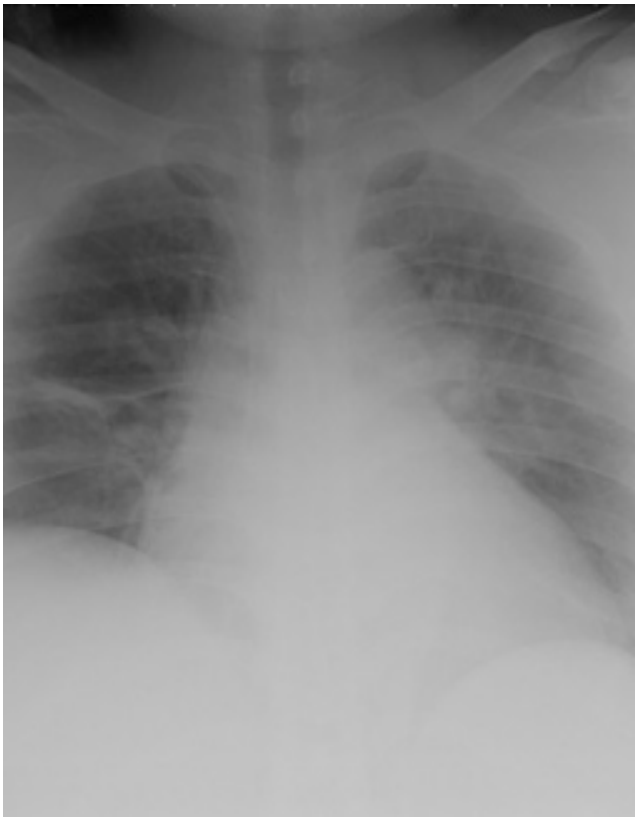

Figure 1. Chest radiograph on hospital day 8 demonstrating mild cardiomegaly.

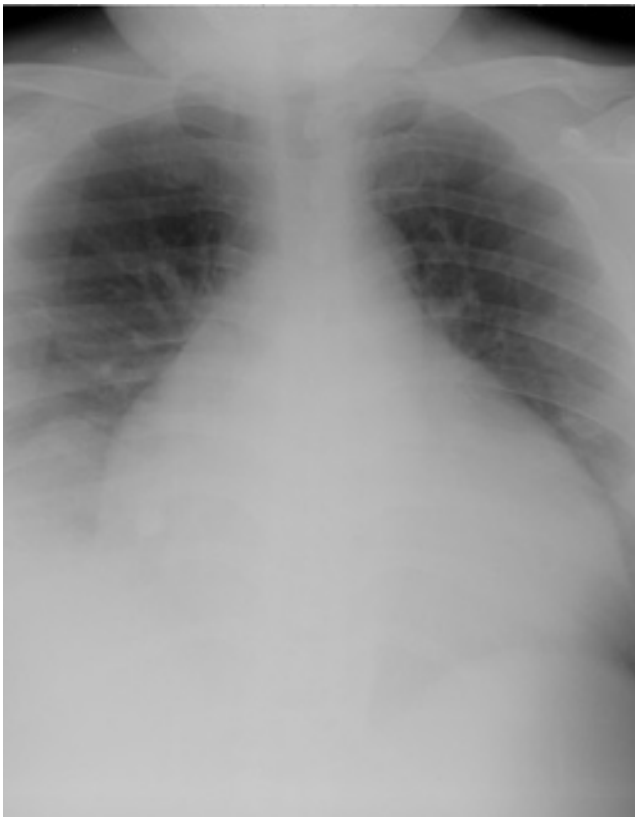

Figure 2. Chest radiograph on hospital day 45 demonstrating marked enlargement of the cardiac silhouette.

consultation were obtained. The INR on hospital day 46 was 3.5. The chest x-ray showed "marked enlargement of the cardiac silhouette" (Figure 2). Cardiology was consulted again, the patient was transferred to the medicine floor, and an effort to reverse his anticoagulation was initiated using fresh-frozen plasma and vitamin K. An echocardiogram showed a "large pericardial effusion with some suspicion of tamponade but no drop in flow across the mitral valve." On hospital day 47, he was noted to have increased jugular venous pressure, increasing pulsus paradoxus, and increasing oxygen needs. A repeat echocardiogram showed "increasing pericardial effusion with signs of tamponade now present."

Pericardiocentesis was performed with the return of 1100 mL of hemorrhagic fluid. A pigtail catheter was left in place for 2 days and then removed after only another 5 mL of fluid was produced. A repeat echocardiogram showed no reaccumulation of pericardial fluid. The patient was not anticoagulated for 1 month, and then warfarin was restarted because of delayed healing of the fractures, continued non–weight-bearing, and the permanent inferior vena cava filter.

Discussion

In previous reports, pericardial effusion has manifested with symptoms of weakness, chest pain, dyspnea, and cough. Signs may include fever, hypotension, elevated central venous pressure, distended neck veins, muffled heart sounds, weak apical pulse, and pulsus paradoxus.[2-5] A classic presentation, however, may not be evident, and the orthopedic surgeon may not initially consider hemorrhagic pericardial effusion as a diagnosis for fever and cough in a multitrauma patient weeks after injury and surgery.

Hemorrhagic pericardial tamponade may also result from central venous catheter misplacement.[6] My patient had a subclavian catheter placed early in the course of his hospitalization, but all x-ray evaluations showed that it remained in the superior vena cava, and the hemorrhagic effusion did not develop until almost 3 weeks after its removal.

Gabram and colleagues[2] noted hemorrhagic pericardial effusion in trauma patients anticoagulated with warfarin. Effusions manifested in 2 patients 4 and 7 days after starting warfarin. The effusion in my patient did not develop symptoms until 27 days after starting warfarin and 18 days after an INR of 2.0 was first achieved. This patient received some form of anticoagulation for 37 days before the effusion was symptomatically manifest. Why a bleed occurred after a long time of stability on warfarin anticoagulation was not immediately clear. The patient's INR rose slightly above the usual therapeutic range at the time the effusion became manifest. Although the elevation was not excessive, it may have been a factor. The patient was in a hospital-type setting, where he was observed and did not have new trauma. The hemorrhagic effusion did not subsequently recur when the anticoagulation was eventually restarted.

It is also notable that the patient had an enlarged cardiac shadow on the supine chest x-rays done early in his hospital care. As echocardiography was not part of the initial cardiology evaluation, it cannot be determined whether the patient had a pericardial injury with a small effusion as a result of the initial blunt chest trauma. It is curious that, if he did, the cardiac silhouette did not enlarge, as assessed by the serial chest x-rays during the initial hospital stay, when he was receiving low-molecular-weight heparin. However, it was notably larger when he developed fever, cough, and malaise more than 6 weeks after injury.

Bleeding complications, in a nontrauma setting associated with warfarin sodium administration, have been reviewed by several authors, and their findings are reviewed in the Box at the top right.[7-13]

The series of chest x-rays taken during the initial part of this patient's hospitalization raises the possibility that a pericardial effusion was present from the time of the first chest x-ray. The eventual large effusion and tamponade may have represented a worsening of the initial problem rather than a new occurrence 6 weeks after admission.

Given the possibility that the patient had a pericardial effusion after blunt chest trauma, one may appropriately ask what different treatment could have been provided. In retrospect, certainly an echocardiogram would have provided useful information. On the possible finding with echocardiography of a mild to moderate effusion, should pericardiocentesis have been performed initially? Studying nontraumatic large pericardial effusions (sum of echo-free pericardial spaces in diastole >20 mm), Mercé and colleagues[14] reviewed 71 patients who did not fit an established protocol for pericardial drainage. Twenty-six underwent pericardial drainage, and 45 did not. No patient developed cardiac tamponade, and only 2 of the 45 had persistent effusions after "conservative" treatment. The authors indicated that routine pericardial drainage would be justified only if it provided relevant diagnostic information or helped to avoid further complications.

In a study of 28 patients with "large" idiopathic chronic effusions, Sagristá-Sauleda and colleagues[15] concluded that the effusion itself was well tolerated by patients but that severe tamponade could develop unexpectedly. Twenty-nine percent of patients in this group developed tamponade a mean of 4 years after the effusion developed.

LeWinter and Kabbani[16] stated, "Patients with possible bacterial effusions or bleeding into the pericardial sac, whose effusions are no more than moderate in size, may in some cases be suitable for initial conservative management and careful monitoring, especially because risks associated with closed pericardiocentesis are increased with smaller effusions."

Holt and Faulx[17] wrote, "Drainage of a pericardial effusion is usually unnecessary unless purulent pericarditis is suspected or cardiac tamponade supervenes, though pericardiocentesis is sometimes needed to establish the etiology of a hemodynamically insignificant pericardial effusion. Anticoagulants should be discontinued temporarily if possible to reduce the risk of cardiac tamponade. In patients on chronic oral anticoagulation, heparin should be used, since its effect can be reversed rapidly. Large effusions may respond to nonsteroidal anti-inflammatory drugs, corticosteroids, or colchicine."

Bleeding Rates With Warfarin in the Nontrauma Setting

Bleeding complications associated with warfarin administration in a nontraumatic setting were reviewed by several authors. Palareti and colleagues[7] reported on 2745 consecutive patients with 153 bleeding complications (23 major, 125 minor). None involved a pericardial effusion. The bleeding complication rate was thus 7.6 per 100 patient-years. Landefeld and Beyth[8] reviewed 6 studies involving inception cohorts and found 187 instances of major and minor bleeding. Five (2.6%) of these were listed as thoracic but were not delineated further. Launbjerg and colleagues[9] reviewed 551 outpatients receiving oral anticoagulant therapy for a total of 1010 treatment-years. They identified 27 bleeding episodes in 21 patients for a rate of 3.8% of patients. There was 1 pericardial bleed (0.18%). Forfar[10] reviewed 501 patients comprising 1199 patient-years and found 41 patients with 51 hemorrhages for a rate of 8.2% among patients and 4.3% per treatment-year. There were no cases of hemopericardium.

Van der Meer and colleagues[11] reviewed 6814 patients with 1003 bleeds, of which 162 were major—representing 2.7 per 100 treatment-years. Of these 162 bleeds 35 were classified other, while none was classified pericardial. Hylek and colleagues[12] reviewed outpatients with an INR of more than 6.0 and studied bleeding complications. Of 114 patients identified, 10 had bleeding episodes, 5 of which were major (4.4%). McMahan and colleagues[13] reviewed "major hemorrhage" as a complication in 579 patients. Forty patients were identified (6.9%), but none was identified as pericardial.

In summary, it appears that the bleeding complication rate for patients receiving oral anticoagulation therapy in the nontrauma setting is less than 10%, and the incidence of pericardial bleeding is much less than 1%.

Conclusions

Orthopedic surgeons attending anticoagulated patients with a history of blunt chest trauma who develop somewhat nonspecific cardiopulmonary symptoms should include hemorrhagic pericardial effusion in the differential diagnosis. Anticoagulation in trauma patients with known or possible pericardial effusion should be carefully considered.

Author's Disclosure Statement

The author reports no actual or potential conflict of interest in relation to this article.

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