Simultaneously diagnosed pulmonary thromboembolism and hemopericardium in a man with thoracic spinal cord injury

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Background: Simultaneous pulmonary thromboembolism (PTE) and hemopericardium is a rare but life-threatening condition. As hemopericardium is a contraindication to anticoagulation treatment, it is challenging to handle both conditions together.

Objective: The objective of the study was to report a rare case of a man with thoracic spinal cord injury presenting with simultaneous PTE and hemopericardium.

Design: Case report.

Subject: A 43-year-old man with incomplete T9 paraplegia (American Spinal Injury Association Impairment Scale D) complained of fever one and a half months after spinal cord injury sustained in a fall.

Findings: During evaluation of fever origin, chest computed tomography and transthoracic echocardiogram revealed simultaneous PTE and hemopericardium. After serial echocardiograms over 2 days demonstrated stability, intravenous heparin, and oral warfarin were administered and his medical status was observed closely. Ultimately, both conditions improved without significant complications.

Conclusion: We report successful treatment of man with acute spinal cord injury who presented with simultaneously diagnosed PTE and hemopericardium, a rare complication involving two distinct and opposing pathological mechanisms and conflicting treatments.

Keywords: Anticoagulant therapy, Hemopericardium, Paraplegia, Pulmonary thromboembolism, Spinal cord injuries

Introduction

Pulmonary thromboembolism (PTE) is an infrequent but serious complication of acute spinal cord injury (SCI), which may cause significant morbidity and/or mortality.¹ Even though the incidence of PTE in patients with pericardial syndromes has been reported to be 4%,² simultaneous PTE and hemopericardium has not been reported in patients with SCI. As treatment protocols conflict, simultaneous PTE and hemopericardium is a rare life-threatening condition.³ We report the course of a patient with acute thoracic

spinal cord injury presenting with both PTE and hemopericardium.

Case description

A 43-year-old man was transferred to our hospital after sustaining thoracic SCI in a fall. Multi-organ injuries were detected, including fractures of the spinous processes of T5 to T11, tear-drop fracture of the T9 vertebra, multiple rib fractures, bilateral hemothoraces without hemopericardium, and hemoperitoneum with splenic rupture. Transpedicular screw fixation with posterior fusion of the T8 to T11 vertebrae, and splenectomy were performed. No specific postoperative complications were noted and mechanical thromboprophylaxis (compression stockings) instead of anticoagulant thromboprophylaxis was used because of reason

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of high bleeding risk from hemothoraces, hemoperitoneum, and multiple fractures during the acute hospital course.⁴

On the 34th day after admission, he was referred to the rehabilitation ward. At that time, he had incomplete T9 paraplegia (American Spinal Injury Association Impairment Scale D) requiring wheelchair for ambulation and was classified as a maximally dependent activities of daily living (ADL) according to the classification criteria of the Korean Modified Barthel Index.^{5,6} On the 47th day of hospitalization, fever developed suddenly without dyspnea or chest discomfort. On auscultation, mild pericardial friction rub with decreased heart sound was detected. A chest PA radiograph showed normal cardiac silhouette and lung fields. Laboratory findings revealed the increase of several inflammatory markers: C-reactive protein was 13.9 mg/dl (normal range: 0–5 mg/dl), leukocyte count was 18 900/mm³ (normal range: 4000–10 800/mm³), fibrinogen degradation products was 15.8 mg/l (normal range: 0–5 mg/l), and D-dimer was 0.8 mg/l (normal range: <0.4 mg/l). Chest and abdominal computed tomographic (CT) scans were performed. Chest CT displayed low-attenuation filling defects in both main pulmonary arteries and considerable pericardial effusion with high attenuation on pre-contrast image. The final diagnosis was hemopericardium rather than pericardial effusion because the pericardial fluid showed iso-attenuation with intra-cardiac blood (Fig. 1). Electrocardiogram was normal. Transthoracic echocardiogram demonstrated moderate amount of pericardial fluid mainly located in the posterior wall of the left ventricle without right ventricular dysfunction (Fig. 2). Thus, PTE and hemopericardium were diagnosed simultaneously. Several laboratory tests for coagulation (functional assay for factor VIII, IX, protein C,

Figure 2 Transthoracic echocardiogram showed pericardial effusion to the posterior wall of the left ventricle (double-headed arrow).

protein S, and antithrombin III), tuberculin skin test, sputum examination, and blood cultures were performed to exclude other causes of hemopericardium, such as hemorrhagic disease, tuberculosis, and infection. However, no specific causes could be determined. He was tentatively diagnosed with delayed traumatic hemopericardium.

Serial transthoracic echocardiograms twice a day for 2 days revealed no increment in the amount of pericardial effusion and he remained hemodynamically stable throughout. Therefore, intravenous bolus injection of unfractionated heparin 5000 units was administered and a maintenance dose of heparin to achieve 1.5–2.3 times the normal value of activated partial thromboplastin time was infused for the treatment of PTE. In addition, warfarin (oral) at 5 mg/day to adjust dosage as target INR 2.0–3.0 and furosemide at 80 mg/day were administered.⁷ After 8 days of medical treatment, follow-up chest CT showed resolution of filling defects in both main pulmonary arteries and reduction in the amount of pericardial effusion (Fig. 3). There were no

Figure 1 Chest CT with contrast showed low-attenuation filling defects in both main pulmonary arteries (left arrow) and considerable amount of pericardial effusion (right arrow) with iso-attenuation compared with intra-cardiac blood on pre-contrast image.

further episodes of thromboembolic events or aggravation of cardiopulmonary dysfunction during the inpatient rehabilitation in hospital. He was discharged to another rehabilitative unit with assisted ambulation using a monocane and had improved to moderately dependent ADL 4 months after admission. He continued with warfarin therapy over the following 6 months. Ten months later, follow-up chest CT revealed no recurrence of PTE and complete resolution of hemopericardium.

Discussion

Concurrent PTE and hemopericardium as complications of SCI have not yet been reported, probably due to their differing etiologies. Previously known causes of venous thromboembolism include surgery, trauma, long-hour air travel, obesity, cigarette smoking, oral contraceptives, pregnancy, postmenopausal hormone replacement, and medical conditions such as antiphospholipid antibody syndrome, cancer, systemic arterial hypertension, and chronic obstructive pulmonary diseases.⁸ The most common causes of hemopericardium are tuberculosis, rupture of sclerotic coronary vessels, wounds of heart or aorta and rupture of an aneurysm, neoplasm, hemorrhagic diseases, postinfectious sequelae, chronic nephritis and trauma.^{9,10} The only cause common to PTE and hemopericardium is trauma.

Previous blunt chest trauma was likely the cause of hemopericardium, and the combination of spinal cord injury and relative immobilization were risk factors for PTE in this patient. The mechanisms by which blunt chest trauma can induce hemopericardium are pericardial or myocardial contusion, aortic root rupture, or diffuse myocardial hemorrhage.^{11,12} We assume that the pericardial or vessel injury occurred at the time of

the fall. The injured sites may have initially been healed by a clot, but subsequent dislodgement of the clot irritated the surrounding labile tissues, resulting in pericardial hemorrhage.

PTE can be managed with anticoagulation therapy in hemodynamically stable patients. Long-term anticoagulation with warfarin is continued for at least 6 months. Patients with PTE who are hemodynamically unstable should undergo thrombolysis or embolectomy with an intra-vena caval filter.⁸ In contrast to the simple algorithm for treating PTE, the treatment of pericardial effusion depends on the underlying condition, necessitating an individualized approach. In impending cardiac tamponade, pericardiocentesis should be performed immediately, but routine pericardial drainage is not recommended because of the lack of definite therapeutic benefit in most cases.¹³ Furthermore, in the absence of cardiac tamponade, arrhythmias, or congestive heart failure, most cases of isolated traumatic hemopericardium resolve uneventfully.¹¹

An important dilemma of treatment in this case was the simultaneous occurrence of potentially lethal illnesses requiring conflicting treatment strategies. Each can present with tachycardia, hypotension, and signs of heart failure, making the individual diagnosis difficult.^{8,10} Whereas PTE would be managed with anticoagulation, it posed a theoretical risk of worsening the hemorrhagic pericardial effusion and eventually leading to life-threatening cardiac tamponade. As there was no increment in the amount of pericardial effusion on serial imaging studies and the patient remained hemodynamically stable throughout, the decision was made to treat the PTE. The patient received anticoagulation medication with successive follow-up imaging studies to closely monitor the state of hemopericardium. If the symptoms and signs were

Figure 3 Ten months later, chest CT with contrast showed complete absorption of previously seen hemopericardium and no recurrence of PTE.

aggravated to result in cardiac tamponade, anticoagulation must be stopped and pericardiocentesis be pursued. Ultimately, both PTE and hemopericardium were treated successfully without active bleeding or exacerbation of pericardial effusion.

Conclusion

We report successful outcome for a patient with acute SCI with simultaneously diagnosed PTE and hemopericardium, a rare combination with differing etiologies and potentially conflicting treatment strategies.

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