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Case Report

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Iliopsoas Hematoma Following Anticoagulant and Thrombolytic Agent in the Intensive Care Unit: A Case Report and Discussion of Current Treatment Strategy

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Iliopsoas hematoma (IPH) may lead to intravascular coagulation and hypovolemic shock with increased mortality rate. Symptoms of IPH are nonspecific, and diagnosing IPH with computed tomography scan may be challenging because of the unstable conditions of critically ill patients. Here we report a 66-year-old female who developed IPH in the intensive care unit (ICU) after receiving anticoagulant and thrombolytic therapy for congestive heart failure and intracardiac thrombus. After stopping thrombolytic therapy, the symptoms subsided, and the patient later underwent removal of intracardiac thrombus with uneventful recovery. For patients with risk factors of IPH, such as anticoagulation, antiplatelet therapy, old age, hemodialysis, obesity, and early ICU rehabilitation, the intensivist should pay attention to an unexplained hemodynamic instability or drop of hematocrit, and consider IPH in the differential diagnoses.

Key words: iliopsoas hematoma, anticoagulation, thrombolytic therapy, intensive care unit

Introduction

Iliopsoas hematoma (IPH) is a collection of blood in the iliopsoas muscle with spontaneous or traumatic etiology. The incidence of IPH in patients receiving anticoagulation is reported to range from 0.1% to 0.6%.[1] For patients in the intensive care unit (ICU), IPH is a potentially life-threatening complication. It may lead to disseminated intravascular coagulation and further hemodynamic instability, which may increase ICU mortality and prolonged ICU stay. Here we report a rare case of IPH in patient receiving anticoagulant and thrombolytic therapy for large left atrial thrombus while staying in the ICU.

Case Reports

A 66-year-old woman was admitted to the ICU due to severe congestive heart failure with the left ventricle ejection fraction being 33% and a large thrombus in the left atrium (Fig. 1). She underwent mitral valve replacement with a mechanical prosthesis due to rheumatic mitral

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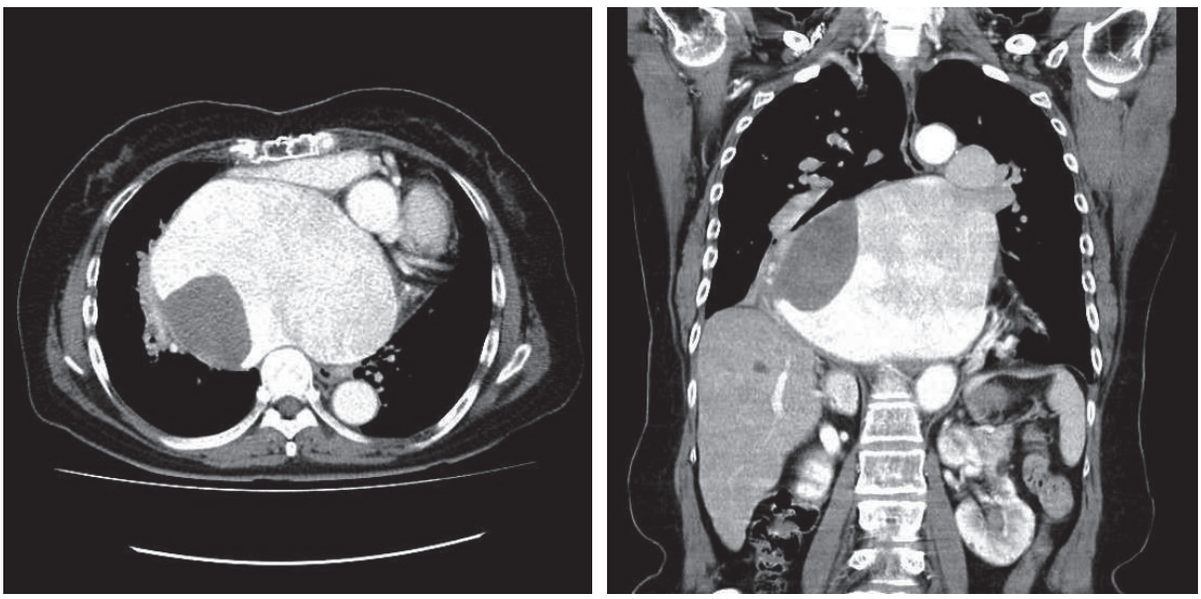

Fig. 1 Contrast-enhanced computed tomography. Revealing a large intracardiac thrombus in the severely dilated left atrium.

valve stenosis 15 years prior to presentation. Besides, she had chronic atrial fibrillation and took long-term vitamin K antagonist (VKA) therapy (Cafarin, Gentle Pharma, Taiwan).

Continuous infusion of inotropic agent and heparin were given, and the heparin infusion rate was adjusted every 6 hours to keep activated partial thromboplastin time (aPTT) in the range of 40 to 60 seconds. The patient decided not to undergo surgical removal of the intracardiac thrombus, hence thrombolytic treatment with urokinase 1,200,000 international units divided into 2 doses per day was given.

However, on the second day in the ICU, the patient complained of severe left groin pain radiating to the left flank. The pain was exacerbated by movement of the left hip joint, but there was no muscle weakness or paresthesia of lower limbs. Contrast-enhanced computed tomography (CT) demonstrated a high-density mass over the left iliopsoas muscle with anterior displacement of the left kidney (Fig. 2), and IPH was diagnosed. Urokinase and heparin were discontinued immediately, and fresh frozen plasma was transfused to correct the bleeding tendency. The clinical condition of the patient, including symptoms, hemodynamic status, and hematocrit, was closely monitored. The serum aPTT returned to 42 seconds in 4 hours, and the left groin pain was alleviated 2 days later.

The patient later received surgical removal of the intracardiac thrombus and replacement of the mechanical mitral valve with a porcine prosthetic valve. She was extubated 2 days after the operation, and VKA therapy was restarted on the 5th postoperative day with a therapeutic target international normalized ratio of 2.5 – 3.0. After stopping the inotropic agent, the patient was transferred to the ward on the 7th postoperative day and was discharged from the hospital on the 15th postoperative day. One month later, echocardiography showed normal function of the prosthetic valve without intracardiac thrombus.

Discussion

Combination of anticoagulant and thrombolytic therapy may be considered for treating intracardiac thromboses. However, the prevalence of extracranial hemorrhagic complications of anticoagulants is approximately 2.1%

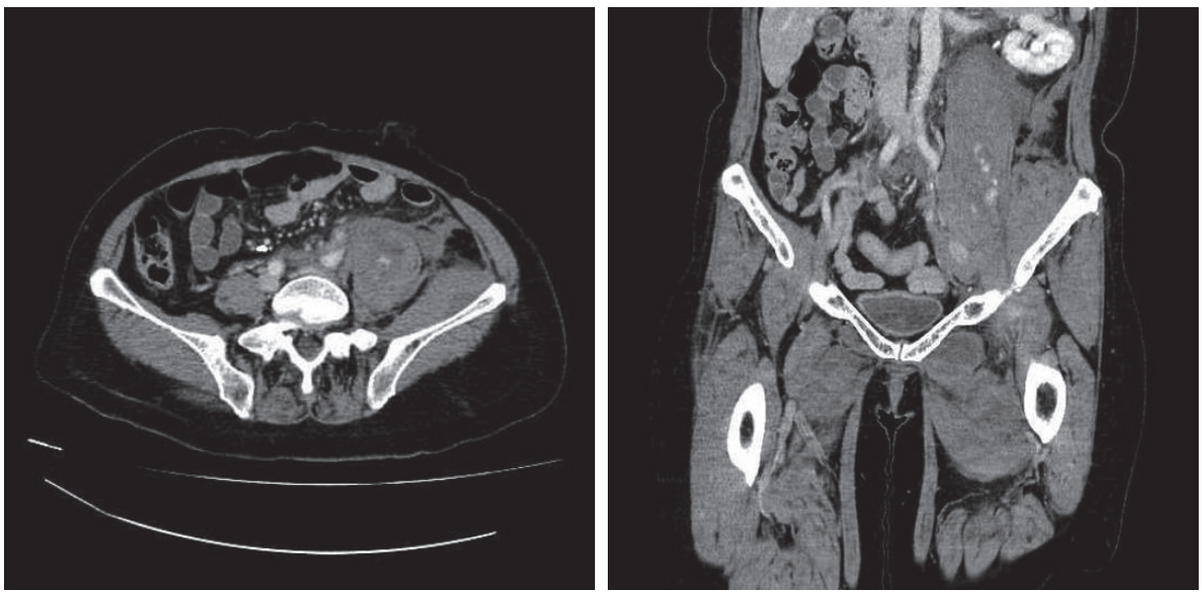

Fig. 2 Hematoma in the left iliopsoas muscle with anterior displacement of the left kidney.

per year.[2] For critically ill patients, iliopsoas hematoma is an uncommon but potentially lethal complication of anticoagulant therapy, with the incidence ranging from 0.30% to 0.38%.[3]

Risk factors of IPH include anticoagulant therapy, advanced age, obesity, and hemodialysis. Besides, early physical rehabilitation may be a risk factor of developing IPH in patients undergoing extracorporeal membranous oxygenation or anticoagulation therapy.[4] Decreased serum fibrinogen level could be an indicator for monitoring hemorrhagic complications of fibrinolytic therapy. But bleeding complications may happen despite adequate monitoring and strict controlled dose of anticoagulant agents.[5] Artzner T. et al. reported that for IPHs observed in the ICU, only 10% of cases had aPTT above the therapeutic range.[6]

Common symptoms of IPH, such as groin pain, are nonspecific. Early detection of IPH in the ICU remains difficult and challenging because patients in the ICU are usually unconscious, immovable, or under deep sedation. The most useful tool for diagnosis of IPH is CT scan, which provides information about presence of active bleeder, the volume of blood, or the severity of local compression.[7] The management of iliopsoas hematoma remains controversial. Medical treatment, such as close observation and blood transfusion when clinically indicated, is usually provided first. Surgical decompression, ultrasound or CT-guided percutaneous drainage, and transcatheter arterial embolization of the inferior epigastric artery or lumbar artery, should be considered in patients with femoral neuropathy or hemodynamic instability.[8,9]

IPH will not only increase ICU stay for at least 3 days but also increase ICU mortality rate to 30 – 50%. While arranging bedside rehabilitation and early mobilization program for critically ill patients receiving anticoagulant or antiplatelet treatment, the risk of IPH should be taken into account. The intensivist should always pay attention to an unexplained drop of hematocrit or hemoglobin level, and hemodynamic instability, for patients with risk factors of IPH to identify this possible lethal complication in time.

Conclusion

For patients in the ICU, IPH is a potentially life-threatening complication and may prolong the ICU stay. Risk factors of IPH

include anticoagulation, antiplatelet therapy, old age, hemodialysis, obesity, and early ICU rehabilitation. If a patient with risk factors of IPH develops an unexplained hemodynamic instability or drop of hematocrit, IPH should be considered in the differential diagnoses. IPH is usually treated conservatively first. Transarterial embolization and surgical decompression should be considered in patients with femoral neuropathy or hemodynamic instability.

Author Contributions

Hsin-Hung Shih: conception and design of study, acquisition of data, drafting the manuscript, literature search; Shih-Hsien Yang: acquisition of data, drafting the manuscript; Ming-Chieh Yang: drafting the manuscript, literature search; Szu-Ying Chen: manuscript preparation; Yu-Wei Huang: manuscript preparation; Yi-Ming Wang: manuscript preparation; All authors have read and agreed to the published version of the manuscript.

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Conflicts of Interest

The authors declare no conflict of interest.

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