Timely embolectomy in acute massive pulmonary embolism prevents catastrophe: An experience from two cases

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Abstract

Acute massive pulmonary embolism is a life-threatening emergency that must be promptly diagnosed and managed. Over the last several years, the use of computed tomography scanning has improved the clinician’s ability to diagnose acute pulmonary embolism. We report two cases of acute massive pulmonary embolism who presented with sudden onset of dyspnea and underwent successful open pulmonary embolectomy. The first case presented with acute onset of dyspnea of 2 days duration, in view of hemodynamic deterioration and two-dimensional echocardiography, it revealed clot in right ventricular (RV) apex and right pulmonary artery; the patient underwent cardiopulmonary bypass and open pulmonary embolectomy with RV clot extraction. The second case presented with a sudden onset of dyspnea on the 15th postoperative day for traumatic rupture of urinary bladder, in view of recent surgery, the patient was subjected to surgical embolectomy. Following surgical intervention, both the patients made a prompt recovery.

Key words: Computed tomography, pulmonary embolism, surgical embolectomy

INTRODUCTION

Pulmonary embolus (PE) refers to obstruction of the pulmonary artery or one of its branches by material (e.g., thrombus, tumor, air, or fat) that originated elsewhere in the body. Despite the high-incidence of pulmonary embolism, its diagnosis continues to be difficult primarily because of the nonspecific nature of the presenting signs and symptoms. The most common sources of pulmonary embolism are the pelvic veins or deep veins of the thigh.

Hemodynamically unstable PE results in hypotension, which by definition is systolic blood pressure <90 mmHg for a period >15 min or that requiring vasopressors or inotropic support and not explained by other causes including sepsis, arrhythmia, left ventricular dysfunction from acute myocardial ischemia or infarction, or hypovolemia. Hemodynamically stable PE is defined as PE that does not meet the definition of hemodynamically unstable PE. Majority of the cases of pulmonary embolism can be managed with medical therapy alone. For more than 30 years, thrombolytic agents have been used to dissolve or reduce the embolus and improve the circulation. However, embolectomy is indicated in patients with hemodynamically unstable PE in whom thrombolytic therapy is contraindicated. It is also a therapeutic option in those who fail thrombolysis and surgical intervention may be indicated in certain instances. Currently, only few indications for surgical embolectomy have been described which include contraindications to thrombolytic therapy, presence of persistent thrombi in the right heart or pulmonary arteries, or deteriorating hemodynamic status. We report two cases of acute massive PE, which was managed successfully with surgical embolectomy.

CASE REPORT

A 60-year-old female presented with dyspnea since 2 days duration. She is also a k/c/o hypertension. On examination, saturation was 60% and there was hypotension. Electrocardiogram (ECG) showed sinus tachycardia, right axis deviation, and right bundle branch block. Routine investigations were normal with erythrocyte sedimentation rate (ESR) −90 mm 1h
Westergren. D-dimer was elevated (10 mg/L). Venous Doppler revealed was normal. Two-dimensional (2D) echocardiography revealed dilated right atrium/right ventricular (RA/RV), severe Tricuspid Regurgitation (TR) with pulmonary artery systolic pressure of 70 mmHg, and clot in RV apex and right pulmonary artery (RPA) [Figure 1a and b]. The patient was subjected to surgical embolectomy with RV and RPA clot extraction [Figures 2a, b and 3]. The patient had prompt recovery on follow-up.

A 65-five-year-old male patient presented with acute onset of dyspnea on the 15th postoperative day for traumatic rupture of urinary bladder. He is a k/c/o hypertension with chronic obstructive pulmonary disease. On examination, saturation was 50% and hypotensive. ECG showed sinus tachycardia, nonspecific ST-T changes. Routine investigation revealed hemoglobin of 12 g%, ESR — 70 mm 1st h Westergren. D-dimer was elevated (12 mg/L). 2D echocardiography revealed dilatation of RA, RV, RPA ostium, and RA fuzzy hypoechoic lesion [Figure 4]. Computed tomography pulmonary angiogram revealed pulmonary thrombus in proximal RPA and normal main pulmonary artery [Figure 5]. In view of recent surgery for traumatic rupture of urinary bladder and clot in RA, thrombolysis was not considered, and the patient was subjected to surgical embolectomy [Figure 6]. The patient had prompt recovery on follow-up.

**DISCUSSION**

Massive PE is defined as RV dysfunction or a systolic blood pressure <90 mmHg or a drop in systolic blood pressure of 40 mmHg from baseline for a period >15 min. In patients with massive PE, 50% patients die within 30 min, 70% die within 1 h, and more than 85% die within 6 h of the onset of symptoms. Clinically, diagnosis is difficult due to the nonspecific nature of the symptoms and signs of PE and investigations including routine blood, arterial blood gas measurement, electrocardiography, and chest radiography, however all lack specificity; thereby limiting their utility in reaching a definitive diagnosis. The differential diagnosis for PE is many which include pneumothorax, pleuritis, pneumomediastinum, pericarditis, acute myocardial infarction, aortic dissection, esophageal rupture, lung cancer, rib fractures, and metastatic deposits. The most

![Figure 1: (a) Two-dimensional echo apical four chamber view shows clot in right ventricular apex and (b): Two-dimensional echo short axis shows thrombus in the right pulmonary artery](image)

![Figure 2: (a) Extraction of the right ventricular clot. (b) Extraction of the thrombus in right pulmonary artery](image)

![Figure 3: Right ventricular and right pulmonary artery thrombus](image)

![Figure 4: Two-dimensional echo short axis shows thrombus in the ostium of right pulmonary artery](image)
common sources of pulmonary emboli are the pelvic veins or deep veins of the thigh.[4] Pulmonary arterial obstruction by clot causes platelets to release vasoactive agents such as serotonin, which may lead to further elevation of pulmonary vascular resistance. This redistribution of blood flow increases alveolar dead space with a resultant ventilation/perfusion mismatch that impairs gas exchange.[5] Right ventricular after load increases, tension rises in RV wall and may lead to dilatation, dysfunction, and ischemia of right ventricle.[6] In the absence of absolute contraindications, thrombolysis is the indicated treatment for patients with acute massive PE, who present with shock and is reported to achieve good outcomes.[7,8] In those patients whose condition worsens despite thrombolysis, surgical intervention should be strongly considered.[9] Currently, only a few indications for surgical embolectomy have been described which include contraindications to thrombolytic therapy, presence of persistent thrombi in the right heart or pulmonary arteries, or deteriorating hemodynamic status. Early surgical intervention may be associated with a survival benefit in such situations.[10] In one nonrandomized case series of patients with massive PE complicated by shock, surgical therapy yielded a slightly lower mortality when compared with thrombolysis.[11] The main predictor of mortality in patients undergoing surgical pulmonary embolectomy is preoperative cardiac arrest requiring resuscitation. Patients with hemodynamic collapse prior to surgery have an operative mortality ranging from 43% to 84%.[12]

CONCLUSION

Massive PE requires immediate therapy to restore pulmonary arterial flow. In the absence of treatment, mortality from PE remains high. Anticoagulation is the standard of care for PE, with the addition of thrombolysis or surgical embolectomy when the clot burden and clinical presentation warrant more aggressive treatment. Surgical embolectomy in hemodynamic instability due to acute PE is reserved for patients in whom thrombolysis (systemic or catheter-directed) is contraindicated, and is an option in those in whom thrombolysis has failed. However, surgical embolectomy may show favorable outcomes in cases such as echocardiographic evidence of an embolus trapped within a patent foramen ovale, or present in the RA, or right ventricle. Surgical embolectomy, therefore, should not simply be considered as a treatment of last resort, but may warrant earlier consideration in certain conditions.

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Conflicts of interest
There are no conflicts of interest.

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Figure 5: Computed tomography pulmonary angiography shows thrombus in the right pulmonary artery

Figure 6: Extraction of thrombus in the right pulmonary artery
Organ transplant recipients under immunosuppressive therapy have a highly increased risk of acquiring unusual opportunistic infections. Diagnosis of the etiology of infection may be difficult in clinical manifestations, which need further histological and biological investigations. Here in we report, for the 1st time in the Iran, a Morganella morganii isolate harboring bla VIM, bla CTX-M, and bla SHV genes after kidney transplantation with persistent urinary infections.

Key words: Carbapenems, kidney transplant, Morganella morganii, opportunistic infection

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possesses the ability to develop resistance on exposure to broad-spectrum cephalosporins. [3] Urinary tract infection (UTI) after kidney transplantation is a common cause of patient morbidity and represents a potential risk factor for poorer graft and recipient outcome. [4] Several species of bacteria that cause UTI in kidney transplant patients have been isolated from Enterobacteriaceae family have been reported as the main isolates in UTI among transplant patients. [5] However, there

Persistent infection with metallo-beta-lactamase and extended spectrum beta-lactamase producer Morganella morganii in a patient with urinary tract infection after kidney transplantation

INTRODUCTION

Morganella morganii is a Gram-negative facultative anaerobe that is commonly found in the environment and in the intestinal tracts of humans as normal flora and belongs to the family of Enterobacteriaceae. Despite its wide distribution, it has been considered a rare cause of human infections. [1] M. morganii is naturally resistant to tetracyclines, tigecycline, polymyxins, and nitrofurantoin. [2] Moreover, by chromosomally encoded AmpC beta-lactamases and 

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