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A RARE CASE OF ACUTE ONSET FATAL VENOUS GANGRENE

Emergency Medicine

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ABSTRACT

Gangrene of the limbs is mostly attributed to pure acute arterial occlusions or mixed arterial and venous occlusions. Venous gangrene is characterized by progression of deep vein thrombosis (DVT) to limb necrosis. Despite all treatment modalities, venous gangrene remains a life threatening and limb threatening condition. We present a rare case of an elderly woman who presented with acute onset leg pain and shortness of breath and was diagnosed with venous gangrene. She was planned for thrombectomy but succumbed to her illness. It would be prudent for emergency physicians to be aware regarding this rare condition and the different treatment modalities available for the management of the same.

KEYWORDS

Gangrene, Thrombolysis, Thrombectomy

INTRODUCTION

Gangrene of the limbs is mostly attributed to pure acute arterial occlusions or mixed arterial and venous occlusions.[1] Rarely does venous occlusion lead to gangrene of the extremities. Three rare manifestations of acute massive venous thrombosis and obstruction of the venous drainage are phlegmasia alba dolens, phlegmasia cerulean dolens (PCD) and venous gangrene.[2] Venous gangrene was first recognized and described by Fabricious Hidanus in the year 1593 but has not been reported frequently. [3] It is characterized by progression of deep vein thrombosis (DVT) to limb necrosis, despite palpable or Doppler identifiable peripheral pulses. This condition if not recognized timely may have catastrophic results including death.[4] We describe a case of an elderly woman who presented with acute onset leg pain and shortness of breath and was diagnosed with venous gangrene but soon succumbed to her illness.

CASE REPORT

A 65 years old female, non smoker, with history of Interstitial lung disease, COPD and rheumatoid arthritis on regular treatment (methotrexate, prednisolone and pyroxicam) presented to our emergency department with progressive severe left lower limb pain and muscle cramps since two days and acute onset of severe breathlessness since 3 hours before presentation. She did not complain of any fever, cough or chest pain. On presentation the patient was tachypneic, tachycardic and in shock with a respiratory rate of 36/minute, pulse rate of 124 beats/minute and blood pressure of 80/40 mmHg, She had an oxygen saturation of 70 % on room air, temperature of 96.0 deg F and random blood sugar of 102 mg/dL. On examination the patient had bilateral crepts on auscultation and a tender right hypochondrium. Local examination was significant for severe pitting edema of the left thigh, leg and ankle. The left lower limb had black discoloration involving all the toes. (Image 1)

Image 1: Left gangrenous lower limb with marked discolouration

The limb was tender on palpation with intact pressure sensation and proprioception. The local temperature at the time of presentation was conserved but rapidly reduced during the stay in our department. All the peripheral pulses were well palpable. She was started on high flow oxygen, fluid resuscitation, inotropic support, morphine, broad spectrum antibiotics and anticoagulation was done with enoxaparin. An electrocardiograph (ECG) was suggestive of sinus tachycardia and left bundle branch block. 2D echocardiography revealed dyschronus septal motion, ejection fraction of 40%, prominent right ventricle, mild mitral and tricuspid regurgitation. The patient's laboratory tests was significant for Troponin I of 1.03 ng/mL, CPK-MB 19.7 ng/mL, D –Dimer 5250 ng/mL, Urea - 68.8 mg/dL,creatinine-2.33 mg/dL, sodium-135.6 mmol/L, potassium-4.47 mmol/L, SGOT-1432 IU/L,SGPT-980 IU/L, Serum Procalcitonin - 2.43 ng/dl and total leucocyte count - 22.9 10~9/L. Patient was provisionally diagnosed as peripheral arterial disease and suspected pulmonary embolism. After initial stabilization she was shifted for Computed tomography (CT) pulmonary angiography and peripheral angiography. Unexpectedly the CT peripheral angiography revealed hypodense thrombus in the distal most portion of inferior vena-cava extending into left common iliac vein through left external iliac, left internal iliac, left common femoral and all three branches, left superficial femoral, left popliteal and lower down into anterior and posterior tibial veins. (Image 2-4)

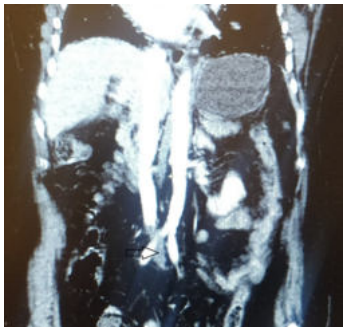

Image 2: Computed tomography (CT) showing thrombus (Black Arrow) in distal part of inferior vena cava

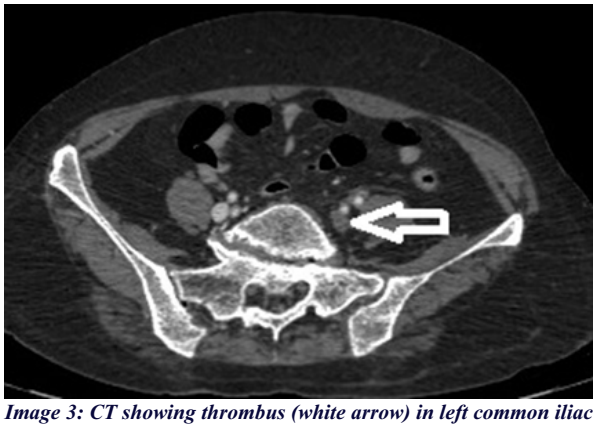

Image 3: CT showing thrombus (white arrow) in left common iliac vein

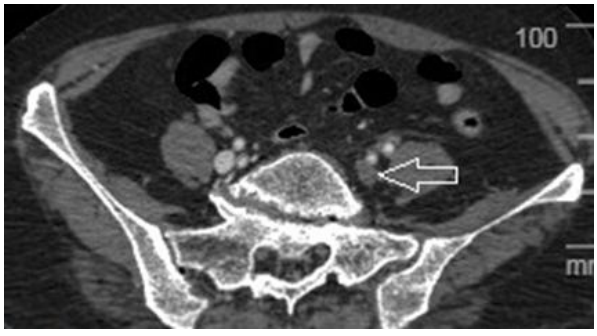

Image 4: CT showing thrombus in left superficial femoral vein (white arrow)

CT Pulmonary angiogram revealed a normal study. The patient was thus diagnosed with the rare condition of venous gangrene with septic shock. Urgent vascular surgeon consult was sought and the patient was planned for urgent high risk thrombectomy and probable amputation. She was shifted to the ICU where she succumbed to her illness before the procedure could be undertaken the same day.

DISCUSSION

The aetiology of venous gangrene includes malignancy, primary hypercoagulable states (antithrombin III, proteins C or S, or plasminogen deficiency) and lupus anticoagulant. It may also occur following surgery or after trauma and postpartum states. Prolonged immobility is also a precipitating factor. [5]

Phlegmasia cerulea dolens and venous gangrene have an overlapping presentation and constitute respectively reversible and irreversible divisions of ischemic thrombophlebitis.[6] Patients with phlegmasia cerulean dolens may present with clinical triad of edema, agonizing pain and cyanosis. Massive fluid sequestration leads to the formation of blebs and bullae.[7] Clinical manifestations of venous gangrene includes (1) onset which is usually sudden; (2) cyanosis appearing early and developing rapidly, extending to the entire extremity; (3) excessive edema of a woody consistency; (4) skin temperature, sometimes conserved, contrasting with the other signs of vascular deficit; and (5) patent peripheral arteries.[1]Venous gangrene is caused by the sudden onset of cellular death due to the prevention of outflow of blood from the area for a long period due to venous occlusion, but arterial tree remains patent which is recognized by palpable pulses, duplex doppler or CT angiogram.

Steep leg elevation, anticoagulation and fluid resuscitation are some of the conservative measures to manage phlegmasia and venous gangrene. Surgical thrombectomy allows instant decompression of venous hypertension, but regardless of thrombectomy, in patients with phlegmasia cerulean dolens, it is associated with high rate of rethrombosis. Thrombolysis may be used as an alternative in the management of venous gangrene. Pacquet et al. were the first to use thrombolysis for treatment of phlegmasia cerulean dolens in 1970.[8] Fasciotomy alone, or in conjunction with thrombectomy or thrombolysis, helps in reducing compartment pressures. If all efforts fail and amputation is required, the procedure should be delayed for as long as possible.[9] Despite all treatment modalities, venous gangrene remains a life threatening and limb threatening condition with overall mortality of 20- 40% and amputation rate of 12-50%.[8]

Epidemiological evidences have shown patients with autoimmune disorders to have an increased prevalence of DVT. [10] Our patient was suffering from rheumatoid arthritis which probably was the etiology of the venous thrombosis. The onset and progression of venous gangrene was sudden and rapid. She was planned for thrombectomy. Alternatively we could have administered urgent thrombolytic therapy using streptokinase in the emergency itself which may have led to a better outcome. Venous gangrene is a rare condition and it would be prudent for emergency physicians to be more proactive to use alternative treatment options like thrombolysis for the sick patient.

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