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Case Report

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A Case of Symmetrical Peripheral Gangrene after Sepsis treatment due to Disseminated Intravascular Coagulation and use of Vasopressors

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Abstract

A 58-year-old woman, known case of COPD controlled on bronchodilators and low dose inhaled corticosteroids presents to the hospital emergency with fever, cough and increasing breathlessness for two days. She is admitted as a case of acute exacerbation of COPD. Preliminary examination and investigations reveal left lower lobe pneumonia. Quite soon the patient develops gram negative bacterial septicemia. In a matter of two weeks things turn sinister as we save the patient from sepsis and septic shock, but she develops the rare and most dreaded complication of inotropes.

Keywords: Gram negative Sepsis, Septic Shock, DIC, MODS, Immune Complex Vasculitis, Septic embolism, SPG.

INTRODUCTION

As Internists we regularly come across COPD cases; Patients who are not compliant to medications or are unable to quit smoking are the ones that frequently land up in exacerbations. Infection and increasing air pollution also contribute to most of such cases. Inhaled and intravenous corticosteroids, Antimicrobials as per the sensitivity pattern in this area, bronchodilators including aminophyllines and home oxygen therapy provide good results in most of the cases. Some patients with immunocompromised status pose a challenge as they run the risk of sepsis with multi organ failure and drug resistance.

CASE REPORT

A 58-year-old woman was brought in a wheelchair to the hospital casualty by her family complaining of fever, increasing breathlessness and cough for two days. Her past medical records suggested that she was a known case of COPD (due to exposure to bio-mass fuel for more than 30 years) and was on Salmeterol-Fluticasone combination inhaler, anticholinergic inhaler and a theophylline bronchodilator. She did not suffer from any other chronic illness. Her BMI was 21. She did not have any substance addiction.

She was conscious, oriented, dyspneic and tachypneic. After a quick initial physical examination and laboratory investigations, the patient was started on oxygen to keep her saturation around 92% and started on intravenous antibiotics and nebulized salbutamol and budenoside. She was hypotensive. Brisk hydration and anti-microbials did not improve her vitals. Her x-ray revealed a left lower lobe pneumonic consolidation.

Her blood culture and sputum report revealed gram negative (Enterobacter) growth. Her antibiotic was changed as per the sensitivity report. Despite these measures, she had already developed Septic shock and Multiorgan Dysfunction Syndrome (MODS). Her creatinine and Liver function tests worsened. She was put on nor adrenaline infusion at 1µg/kg/min and was intubated and mechanically ventilated for three days. Her D-Dimer levels were high, Serum fibrinogen level was 13.5 mg/dL (N-150 to 400 mg/dL), and INR 2.1. All features suggested of Disseminated Intravascular Coagulation (DIC) in the patient.

With proper intensive and hydro-electrolytic care over the next few days she survived the sepsis and DIC. In the next three to four days her Renal and Liver function tests improved. Her urine output and Blood pressure improved. She was weaned off the ventilator and ionotropic support but soon after she

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complained of severe pain in her both hands and feet. All peripheral pulses were felt equally. But her hands and feet had turned dark and cold. Her all four distal extremities had developed acro-cyanosis. Although Low molecular weight heparin was started, in a matter of next two days her fingers and toes and distal part of feet had turned completely gangrenous.

A color arterial Doppler done in our hospital revealed no intra-arterial/intra luminal flow obstruction.

As vascular surgery consult was immediately necessary and amputation seemed the only option left to save the life of the patient, we transferred the patient to a higher facility where there was access to vascular surgery and plastic surgery departments.

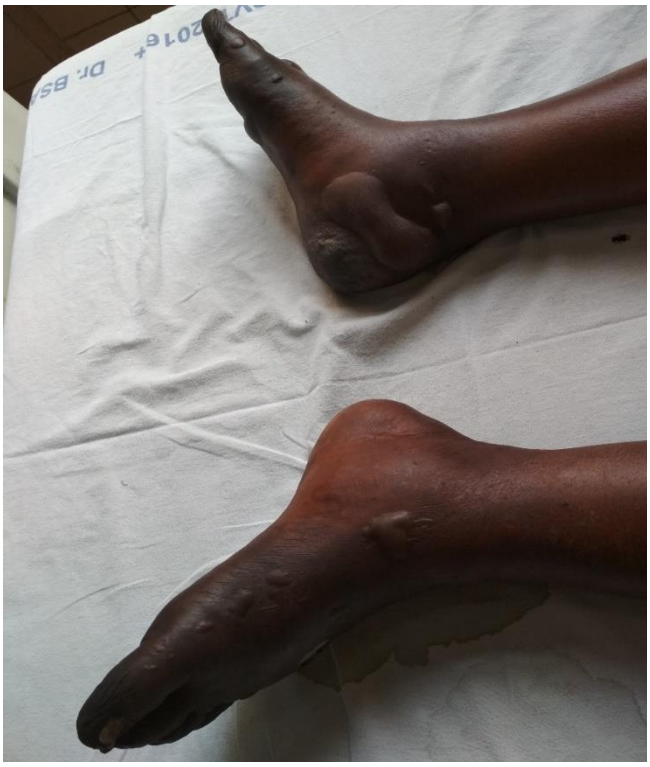

Figure 1: Image of her feet showing gangrenous changes

Figure 2: Gangrenous changes in the Right hand due to SPG

Figure 3: Image showing her fingers that developed gangrene uptill Proximal interphalangeal joints

DISCUSSION

Gangrene refers to death of macroscopic portions of tissue with putrefaction which turn black because of the breakdown of hemoglobin and the formation of iron sulphide. It usually affects most distal parts of the limb because of arterial obstruction due to thrombosis, embolus or arteritis [1]. Dry gangrene is desiccated, mummified tissue with a clear line of demarcation. Wet gangrene is due to both arterial and venous block along with superadded infection. It spreads proximally faster and there is no clear line of demarcation [2]. Attempts of amputation of the local tissue in the presence of poor circulation or infection usually fail, and the gangrene usually reappears in the wound edges [1].

Symmetrical peripheral gangrene as described by Hutchinson is a gangrenous lesion of two or more extremities in the absence of major vascular obstructive disease [3]. The peripheral pulses are preserved and palpable. The pathophysiology that led to SPG in this patient was decreased blood supply to the extremities due to hypotension due to septic shock which redirected the blood flow from peripheries to important organ systems in the body and the use of vasopressors that further reduced blood supply to the peripheral regions of the limbs. Disseminated intravascular coagulation is associated with most cases of SPG due to activation of coagulation pathway [4]. The common causes of SPG are Myocardial infarction, heart failure, pulmonary thromboembolism, pro coagulant conditions like leukemias, Para neoplastic syndromes, connective tissue disorders, deficiency of protein C and S, hyperosmolar coma, hypernatremia or severe dehydration, cryoglobulinemia, sickle cell anemia [5]. Infective causes include Streptococcus pneumoniae, Staphylococcus aureus, Neisseria meningitis, Streptococcus pyogenes, Klebsiella pneumoniae, Salmonella paratyphi, Proteus vulgaris, Proteus mirabilis. Viral and parasitic causes of SPG include viral gastroenteritis, Varicella zoster, Rubeola, Plasmodium species especially falciparum [5]. A rare cause of SPG that has been mentioned in literature is infection with Capnocytophaga canimorsus due to a dog bite [6].

SPG with DIC has a high mortality rate with survivors most often requiring amputation of at least one limb. The following diseases can mimic SPG: Purpura fulminans, senile gangrene, diabetic microangiopathy leading to gangrene, Buerger's disease, Raynaud's phenomenon/disease, ligation or tourniquet, frost bite, vascular

occlusion, rheumatologic disorders, arteriopathies like Takayasu's disease, radiation injury, trauma and scleroderma.

Treatment options for SPG include aspirin, heparin, hyperbaric oxygen, streptokinase, tissue plasminogen activator, prostaglandins, plasmapheresis, antimicrobials to prevent or treat infection, and sometimes even intravenous nitroglycerine and nitroprusside. We are not sure how effective these therapeutic options are in preventing or reversing the progression of SPG.

CONCLUSION

Peripheral symmetrical gangrene is a rare but dreadful complication of sepsis with intravascular coagulation and the use of vasopressors. Patients on inotropes should be properly monitored, and their use reduced/stopped with earliest signs of improvement in the hemodynamic status of the patient. Efforts to identify earliest signs of peripheral small vessel vascular occlusion should take foremost importance in the prevention of SPG.

Conflicts of interest

None declared.

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