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Case Report

A rare but formidable complication of sepsis: symmetrical peripheral gangrene

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ABSTRACT

Symmetrical peripheral gangrene (SPG) is a rare clinical entity. It was first described in late 19th century and since then has been reported with array of medical conditions mainly those complicated with shock, sepsis, and disseminated intravascular coagulation (DIC). Here in, we describe a patient with sepsis and SPG. Clinicians should be aware of this entity as early recognition can help in reducing morbidity and mortality.

Keywords: Disseminated intravascular coagulation, Sepsis, Symmetrical peripheral gangrene

INTRODUCTION

Symmetric peripheral gangrene (SPG) is defined as symmetrical distal ischemic damage in two or more sites in the absence of a major vascular occlusive disease. It carries a high mortality rate with a very high frequency of multiple limb amputations in the survivors. A more or less stereotyped clinical picture of SPG in spite of the ever-widening etiological spectrum is suggestive of disseminated intravascular coagulation (DIC) as the final common pathway of its pathogenesis. It is proposed to be a cutaneous marker of the same.[1]

CASE HISTORY

Twenty one-year-old, unmarried female was admitted with chief complaints of high grade fever, cough with sputum production, and progressive dyspnea for 14 days. There was no history of chest pain and palpitation. There were no neurological complaints. Urinary and bowel habits were normal. There was no history of jaundice, hematuria, Raynaud's phenomenon, joint pains, taking ergot, β-blockers etc. At admission, patient was febrile, had blood pressure of 100/60 mm of Hg, pulse rate of 110 beats/min, and respiratory rate was 30/min. All peripheral pulses were palpable, regular, and of normal volume. On systemic examination, there were bilateral crepitation in chest, cardiac examination was unremarkable. Investigations revealed hemoglobin 9.8 g% and total leukocyte count 35600/cumm. Peripheral blood smear revealed normocytic hypochromic picture with neutrophila and shift to left. Biochemistry revealed following parameters: random blood glucose-110 mg%, total bilirubin-1 mg%, SGOT-140 IU/L, SGPT-82 IU/L, alkaline phosphatase-60 IU/L, albumin-3.4 g%, urea-80 mg%, creatinine-1.5mg%, uric acid-3.6 mg%, calcium-9 mg%, and phosphorous-4.6 mg%. Electrolytes and lipid profile were normal. Urine showed albumin 1+ on dipstick and was negative for pus cells, red blood cells cast, and active sediment. Sputum, urine and blood cultures were sterile. HBsAg, anti-HCVIgG, VDRL, ELISA for HIV, and antinuclear antibody were negative. Chest X-ray revealed bilateral infiltrations.

Electrocardiogram showed sinus tachycardia. Doppler study for upper and lower limb arteries was normal. After examination and investigation, clinical possibility of septicemia with focus of lower respiratory tract infection suspected. On the third day, cyanosis was observed in the both toe tips. With due course of two days toes of both fingers show blackening but radial and dorsalis pedis pulsations were well felt in all limbs. An immediate suspicion of DIC with prompt addition of broad spectrum antibiotics for possible gram positive infection, warming of extremities and fresh frozen plasma infusion done. Further investigations like prothrombin time 30 s (International normalized ratio 2.9), platelets 80000/cmm, positive fibrin degradation products, hematuria, oliguria and acute renal failure confirmed DIC. Over next two days, patient's renal function test and liver function test were normalized, gangrenous changes in fingers improved, and dry gangrene became confined to bilateral toes. The patient's general condition stabilized over the next 72 h. The cyanosis in the toes of the bilateral foot receded. The condition of the left toes improved - color and sensation returning to them. Line of demarcation of the gangrenous area appeared to involve finger tips and right toes. On follow up at 4 weeks, toes were dry, shriveled with features of auto amputation of only middle toe of right foot.

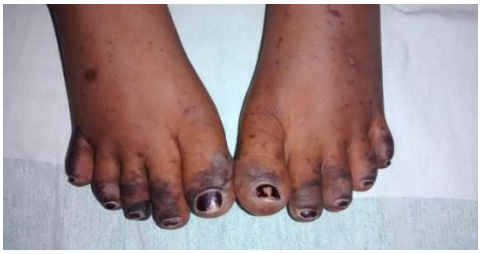

Figure 1: Showing symmetrical peripheral gangrene of toes.

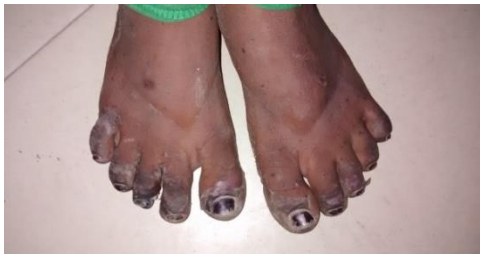

Figure 2: Showing partial improvement in toes.

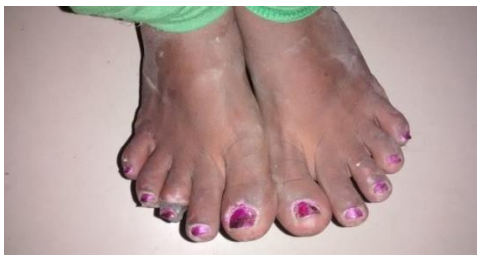

Figure 3: Show improvement at 4th week in follow up.

DISCUSSION

SPG may manifest unpredictably in conditions associated with sepsis, low output states, vasospastic conditions, myeloproliferative disorders and hyper viscosity syndrome. It has previously been associated with viral gastroenteritis and falciparum malaria.[2,3] Multiple factors like viral gastroenteritis, DIC, dehydration, hypotension and noradrenaline infusion may be implicated. Although gram positive and, less commonly, gram negative bacteria are associated with SPG.[4] In our case, blood, sputum and urine cultures were all negative. Prior antibiotic usage may be an explanation. Recent literature points to a 100% association with DIC, high mortality rate of up to 35%, rates of amputation ranging from 70 to 90% and a possible association with the winter season.[4,5] The condition is aggravated by asplenism, hypothermia, vasopressor infusion, immunosuppression, diabetes mellitus and renal failure. It may occur as a complication of malignancy, ergot poisoning and increased sympathetic tone states.

The pathogenesis of SPG may include the Schwartzman reaction, bacterial endotoxin release and platelet plugging in peripheral arterioles due to vascular collapse and DIC. Larger vessels are spared and peripheral pulses are generally palpable. Low flow states exacerbate the situation.

The ischemic changes usually begin distally and may progress proximally to involve the entire extremity. These changes are not generally preceded by vascular occlusion. SPG should be suspected at the first signs of coldness, pallor, cyanosis, or pain in the extremity, as the condition can rapidly progress to frank gangrene. The exact pathogenesis of SPG is not well understood but hallmark is microcirculatory failure. The hypercoaguable state, DIC, and vasospasm invariably coexist. SPG has resemblance to cold injury with features of mummification, absence of infection, and dry gangrene common to both. Examination of the amputated specimens often reveals thrombi concentrated in the small vessels with sparing of large vessels.

No treatment is universally effective. It should be individualized according to the underlying disease and patient's general condition. Early recognition of the SPG and immediate discontinuation or reduction (if possible) of the vasopressors may prevent its further progression. Patient should be treated with appropriate antibiotics for sepsis. If there is evidence of DIC, then heparinization may be effective,[6] beside these aggravating factors like cold, renal failure, diabetes mellitus, and immunosuppression should be identified early and managed accordingly. Intravenous prostaglandins, nitroprusside, topical nitroglycerine, papavarine, reserpine, streptokinase, dextran, hyperbaric oxygen, and sympathetic blockade have all been tried with variable and unequivocal success. The affected limb should be protected from trauma, cold, and secondary infection.

Amputation of the gangrenous area may be required.[7] Patient should be continuously watched for gangrene to become demarcated and only then amputation should to be attempted. This initial nonsurgical approach helps in avoiding loss of viable tissue and gives time for patient's condition to stabilize.

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