Symmetrical peripheral gangrene associated with peripartum cardiomyopathy

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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 parturient with peripartum cardiomyopathy (PPCM) and SPG. Clinicians should be aware of this entity as early recognition can help in reducing morbidity and mortality.

Key words: Disseminated intravascular coagulation, peripartum cardiomyopathy, symmetrical peripheral gangrene

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INTRODUCTION

Symmetrical peripheral gangrene (SPG) is a rare clinical condition. It was first described by Hutchison in 1891 in a 37-year-old male who developed gangrene of fingers, toes, and ear lobules after shock.[1] Afterwards, it has been described with several conditions and has been proposed to be a cutaneous marker of disseminated intravascular coagulation (DIC).[2] Here in, we describe a case where SPG occurred in a setting of peripartum cardiomyopathy (PPCM).

CASE REPORT

Thirty-year-old, para 4, seven days post-partum with unbooked pregnancy, home conducted delivery with alive infant was admitted with chief complaints of progressive dyspnoea for 4 days, orthopnea for 1 day, cough with pink frothy sputum, decreased urine output, and swelling feet for 1 day. There was no history of chest pain, palpitation, and fever. There were no neurological complaints. Lochia, urinary, and bowel habits were normal. At admission, patient was afebrile, had blood pressure of 96/60 mm of Hg, pulse rate of 102/min, and respiratory rate was 28/min. Jugular venous pressure was raised up to 6 cm above angle of Louis. Pedal edema was present. There was bluish discoloration of fingers of both hands. Beside this dorsal aspect of bilateral feet showed edema, lividity, and cyanotic hue on plantar aspect. Bilateral toes showed symmetrical gangrenous changes with eschar formation [Figures 1-3]. All peripheral pulses were palpable, regular, and of normal volume. On systemic examination, there were bilateral basal crepitations in chest, tender hepatomegaly, and S_3 gallop. Investigations revealed hemoglobin 9.8 g% and total leukocyte count 15,600/cumm. Peripheral blood smear revealed normocytic hypochromic picture with neutrophilia and shift to left. Biochemistry revealed following parameters: Random blood glucose-110 mg%, total bilirubin-1 mg%, SGOT-110 IU/L, SGPT-102 IU/L, alkaline phosphatase-90 IU/L, albumin-3.0 g%, urea-120 mg%, creatinine-1.5mg%, uric acid-3.6 mg%, calcium-9 mg%, and phosphorous-4.6 mg%. Electrolytes, lipid profile, and coagulogram

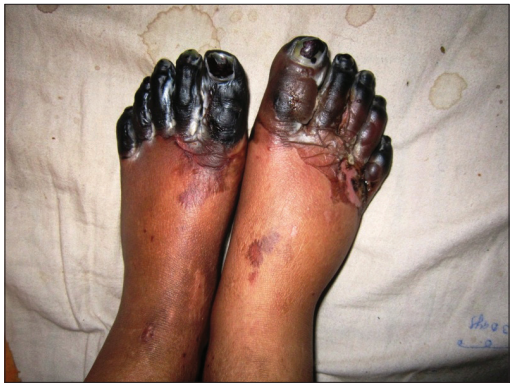

Figure 1: Image showing symmetrical peripheral gangrene of toes

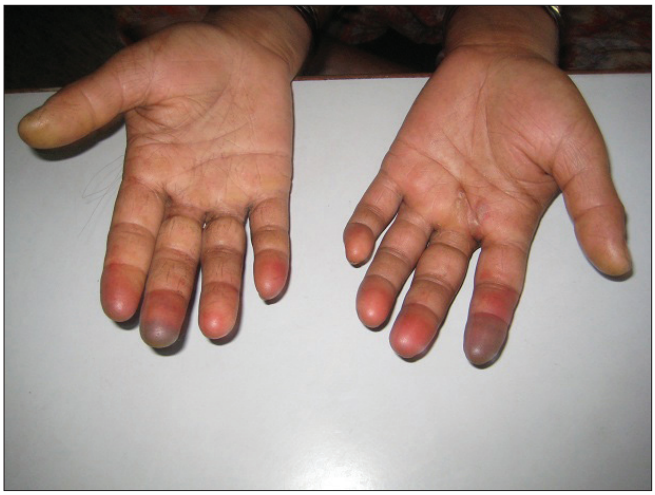

Figure 2: Image showing cyanotic changes in fingers

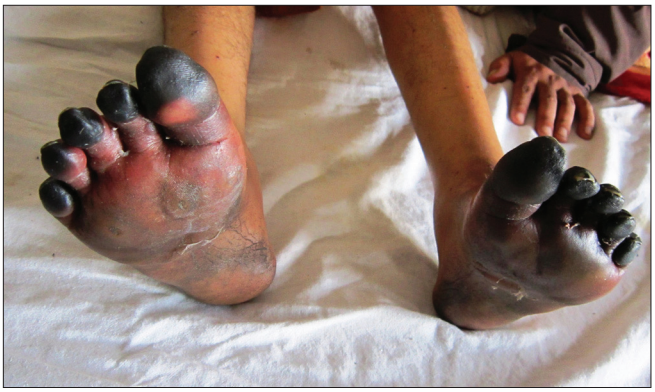

Figure 3: Image showing cyanotic hue on plantar aspect of feet along with peripheral gangrene of toes

were normal. Urine showed albumin 1+ on dipstick and was negative for pus cells, red blood cells cast, active sediment. Urine and blood cultures were sterile. HBsAg, anti-HCVIgG, VDRL, ELISA for HIV, and antinuclear antibody were negative. Chest X-ray revealed bat wing appearance with cardiomegaly. Electrocardiogram showed sinus tachycardia. 2D echocardiogram revealed global hypokinesia with left ventricular ejection fraction (LVEF) of 40%. Doppler study for upper and lower limb arteries was normal. After examination and investigation, clinical possibility of PPCM with SPG was kept. Patient was treated with back rest, oxygen inhalation, enoxaparin (low molecular weight heparin), aspirin, diuretics, and low dose angiotensin-converting enzyme (ACE) inhibitors. After 5 days of in hospital treatment, patient's blood pressure normalized to 110/80 mm of Hg. Pedal edema and raised jugular venous pressure resolved. Over next few 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. Patient was ambulatory at discharge, normotensive, and advised follow up in general surgery for management of dry gangrene of toes. On follow up at 4 weeks, 2D echo showed LVEF of 52% and bilateral toes were dry, shriveled with features of auto amputation.

DISCUSSION

SPG is a clinical entity that manifests as acral ischemic damage in two or more extremities without any evidence of obstruction or vasculitis. The ischemic changes usually occur in fingers or toes but can also involve lips, ear lobules, nose, and external genitalia. The most common causes of SPG are bacterial infections like pneumococcus, staphylococcus, meningococcus, and streptococcus.[3] Other established causes are falciparum malaria,[4] viral gastroenteritis, paraneoplastic syndromes, malignancies like Hodgkin's lymphoma, ergotism, vasopressors, shock, and decreased level of protein C. It is a rare complication of septicemia, with a high mortality and about half of the patients who survive the episode require amputation of the affected limb.[5] It has also been described with conditions leading to low peripheral blood flow like cardiogenic and hypovolumic shock. Our patient had experienced hypotension in pre hospitalization period due to left ventricular dysfunction in the setting of PPCM and developed SPG.

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.[5,6] Beside this aggravating factors like cold, renal failure, diabetes mellitus, and immunosuppression should be identified early and managed accordingly. Intravenous (i.v.) prostaglandins like epoprostenol, 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.

CONCLUSION

In conclusion, we think that appreciation of early features of SPG is important as relevant arteries usually remain palpable during the course of illness. The arteries are palpable as the disease mainly affects the microvasculature and it helps in differentiating from other causes of gangrene like atherosclerosis, embolism or Buerger's disease. Once it develops, the affected part should be protected from cold, trauma, and secondary infection. Inter digital padding may be helpful as it prevents damage to opposing surfaces of digits. Joint movement should be maintained by appropriate physiotherapy to prevent contracture. Heparin has been used by various authors especially when there is underlying DIC. Surgery should be considered only when the gangrenous part becomes well demarcated. Beside this the therapy of underlying condition and correction of aggravating factors is the mainstay of the treatment.

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