A 30 years old chronic alcoholic, non-smoker, non-diabetic male was admitted with moderate to high grade fever along with cough and expectoration of three days duration. He also had history of multiple episodes of loose motions, vomittings for one day and one episode of haematemesis prior to admission. On examination he was pale, conscious, oriented but drowsy with signs of severe dehydration. Peripheral pulses were not palpable and blood pressure was not recordable. Respiratory system examination revealed inspiratory crepitations in right lower interscapedular, infrascapular as well as infraaxillary areas. Investigations revealed hemoglobin of 10.2 gm%, total leucocyte count 27800/mm³, differential leucocyte count P₇,L₂,Eₓ,M₁, platelet count 19000/mm³, total serum bilirubin 1.3 mg/dl, serum alanine aminotransferase 145 U/l, serum alkaline phosphatase 273 U/l, total serum protein 5.3 gm/dl (albumin 2.4 gm/dl), prothrombin time 26.2 sec (control - 13.8), activrivated partial thromboplastin time 53.6 sec (control - 28.2), random blood sugar 50 mg%, blood urea 120 mg%, serum creatinine 2.1 mg% and serum electrolytes (sodium / potassium) 140/4.5 mEq/L. Peripherial smear for malarial parasite and malaria antigen were negative. X-ray chest revealed right lower zone pneumonia. Ultrasound abdomen was normal. Blood, stool and urine cultures were sterile. Viral markers for hepatitis B, C and antinuclear antibody were negative. Enzyme linked immunosorbent assay for human immunodeficiency virus and venereal disease research laboratories test were non-reactive. A probable diagnosis of symmtric peripheral gangrene (SPG) due to right lower lobe pneumonia with septicemic shock and multisystem failure was kept. He was put on intravenous (IV) fluids, antibiotics, and four units of platelets were transfused. Despite fluid correction he continued to be in shock and responded to inotropic support. On third day, patient developed swelling over both upper and lower limb along with bluish black discoloration with definitive level upto 10 cm above the ankle in lower limb (Fig. 1) and 5 cm above the joint in upper limb with blackening of fingertips (Fig. 2). Arterial colour doppler of both upper and lower limbs revealed thickened shaggy wall, peak systolic velocity was decreased with increased reverse components, sluggish flow in the proximal arteries with no flow in distal arteries bilaterally. He was started on low molecular weight heparin in addition to IV fluids and antibiotics. Despite of all conservative treatment, he showed progressive deterioration with blackening of extremities. Amputation with skin grafting was planned but unfortunately he left against medical advise.

SPG is defined as symmetrical distal ischemic damage at 2 or more sites in the absence of large vessel obstruction, sometimes used synonymously as purpura fulminans. Disseminated intravascular coagulation (DIC) and hemorrhagic infarction of skin with uninvolved proximal arteries are hallmark of this condition. Studies indicate that upto 85% of patients with SPG have associated DIC. The most common cause of SPG in clinical setting is bacterial infection, especially pneumococcus, staphylococcus, streptococcus pneumoniae and meningococccemia but gram negative organisms have also been implicated. Other established causes are faciparum malaria, viral gastroenteritis, paraneoplastic syndrome, ergotism and decreased level of protein C. Aggravating factors like asplenia, immunosuppression, diabetes mellitus, renal failure, cold injury and use of vasopressors should be identified and managed accordingly.

Treatment plan should be individualized depending on the aggressiveness of disease and type of complications as no modality of treatment is universally accepted in managing SPG. However, early intervention with IV fluids, antibiotics and heparinization to treat sepsis and DIC are essential components of SPG management. IV nitroprusside, IV prostaglandins (e.g. epoprostenol), topical nitroglycerine ointment, papavarine, reserpine, streptokinase, dextran, hyperbaric oxygen and sympathetic blockers are tried with little success. Amputation may be required depending upon non-viability and line of demarcation of distal parts.³

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