

SYMMETRICAL PERIPHERAL GANGRENE: MULTIFACTORIAL ASSOCIATION. A CASE REPORT

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ABSTRACT

We describe a case of a young woman, operated for perforation peritonitis who was admitted postoperatively in ICU for deranged consciousness level, hypotension and inadequate respiration. She developed symmetrical peripheral gangrene (SPG) supposedly due to multifactorial association of shock, septicemia, DIC and Dopamine. Poor understanding of aetiopathogenesis and treatment lead to evolution of gangrene and autoamputation of two toes of both the legs.

KEY WORDS: Symmetrical peripheral gangrene (SPG), Multifactorial

Symmetrical peripheral gangrene (SPG), a rare clinical entity known for decades was first described by Hutchison (1891) in a 37 years old male who developed gangrene in fingers, toes and ear lobules after shock.¹ Later, SPG has been associated with several conditions and it has been proposed to be a cutaneous marker of DIC.² SPG is a rare clinical condition, which manifests as acral ischemic damage in two or more extremities without any evidence of obstruction or vasculitis of the relevant artery. We report this entity in association with a combination of clinical conditions such as peritonitis, septicemia, DIC, shock and dopamine infusion.

CASE REPORT

A 32 years old female without any premorbidities presented with altered behavior, inability to pass feces and flatus, pain in abdomen, generalized tenderness, rebound tenderness and guarding. Roentgenogram showed gas under diaphragm and she was found to have perforation peritonitis. She was attended by her family physician in the village who had given I.V. fluids, antibiotics and analgesics without any relief. Laparotomy, peritoneal lavage and closure of the illeal perforation were done under general anesthesia. Preoperatively pulse was 130/min and BP 80/60 mmHg. Dopamine infusion (5-6

µgm/kg/min) could maintain BP to that range only and further fall could be avoided. All the peripheral pulses were palpable. Routine laboratory investigations were unremarkable. Prothrombin time was elevated with platelet count of $1.5 \times 10^9/\text{mm}^3$ and total leucocyte count of $5000/\text{mm}^3$. Blood was positive for Fibrin degradation products and D-dimers. Echocardiography did not reveal any evidence of thrombus or vegetation. Venous blood lactate was 42mg/dl (lab ref. 6-16 mg/dl). Post operatively she was on elective mechanical ventilation and received intensive medical care in the ICU. Peritoneal-wash culture grew commensals and non-lactose fermenters sensitive to Ceftriaxone and piperacillin-tazabactam. Parenteral nutrition, I.V. fluids Ceftriaxone and advanced nursing were provided to treat her. She developed brownish discoloration (and later gangrene) symmetrically in both the feet leading ultimately to auto amputation of 4th and 5th toes bilaterally. Skin biopsy from the gangrenous site showed non-specific changes. Later a revision amputation and debridement was done. (Figure 1. Showing symmetrical peripheral gangrene in two toes of both the feet)

DISCUSSION

SPG has been reported in a multitude of medical conditions³ such as DIC, infections, myocardial infarction, congestive cardiac failure, dog bite, shock, hypertension, coma, pulmonary embolism, paroxysmal ventricular tachycardia, appendicitis, Hodgkins disease, polymyalgia rheumatica, extracorporeal shock wave lithotripsy, viral gastroenteritis, suprapubic proctectomy, use of vasopressor (dopamine, epinephrine, norepinephrine), SLE, small cell lung cancer, ergotism, metastasising carcinoma of the colon, acquired hemolytic anemia, reaction to drugs (sulphamezathine and penicillin), pulmonary embolism, pneumonia etc. Among the infections meningococcal, streptococcal, E coli, pseudomonas, S paratyphi, Kleibsellia, proteus vulgaris, P mirabilis, pasturella multocida, D F₂ gram negative bacillus, viral gastroenteritis, varicella, rubella and disseminated tuberculosis are reported to cause it. The published reports of this type of gangrene indicate that it may result from: (1) Vasospastic conditions (2) Small vessel obstruction or (3) Conditions producing very low cardiac output. Palpable distal pulses and absence of vasculitis and microemboli makes the pathogenic mechanism behind its causation as difficult to explain with the current understanding of pathology. Hemodynamic instability has been tried to explain the condition but lots of cases with stable hemodynamic status have also been reported. Elevations in the concentrations of FDP reflecting the ongoing fibrinolysis have been documented in association with SPG and malaria.⁴ An analysis of the previously reported cases and our case show that this case presents

acutely and most commonly affects fingers and toes, rarely nose upper lip ear lobules and genitalia. Vasospastic effect may be more intense in digital vascular beds than larger vessels due to vasopressors such as dopamine, noradrenaline and adrenaline. Occlusion of small blood vessels when the intraluminal pressure falls below a certain critical value⁵ (36-60 mm Hg) has been shown. But cutaneous manifestation may be hallmark of several such sites within the body. On the other hand there are case reports of SPG without any fall of blood pressure and use of vasopressors. Associations of medium and large dose dopamine and SPG has been assigned.⁶ Literature is deficient in suggesting any prevention or treatment of this condition and generally its too late once its recognized, since it develops to a variable extent when amputation and debridement becomes inevitable. Our patient had received dopamine, had septicemia, shock and DIC which all individually have been incriminated to cause SPG. The clinical entity is very rare. Possible genetic predisposition cannot be ruled out. General conditions of the patients are very poor at the time of its development as they are non-ambulatory and bed ridden. Treatment with epoprostenol, tissue plasminogen activator⁷, aspirin, vasodilators and sympathetic blockade have been suggested. But such modalities are generally unsatisfactory. Supposedly aspirin may help by antiaggregatory mechanism. Evolution and natural history of the condition has many similarities with severe cold injury. In both the conditions there is dry gangrene, mummification and absence of infection. Hence keeping the affected part warm, inter digital padding and protections from trauma are suggested. Amputation should be deferred till clear demarcation of the healthy and diseased part takes place; otherwise viable tissue may be sacrificed. Development of the condition may be irreversible, but further progression can be prevented. Preservation of joint mobility and range of motion is achieved by early physiotherapy. We believe that the association of this clinical entity with a wide variety of pathogenic conditions negates the possibility of single pathogenic mechanism behind it. Common analysis of all the cases reveal that it develops in critically ill patients due to entirely different primary diseases. Controlled studies on its aetiology and management are lacking. Surprising symmetry of the condition remains unanswered on the basis of microvascular aetiology alone. But the analysis of the previous case reports reveal that only one anatomical location (eg. Ear/fingers/fingers/feet) is generally effected initially. Further, external manifestation may only be visible part of several such sites within the body. The prognosis depends on the underlying disease, local progression and extent of SPG. Uncontrolled local progression can have grave consequences.

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Figure-1

