Symmetrical Peripheral Gangrene due to Viral Gastroenteritis


Abstract
A case of multifactorial symmetrical peripheral gangrene due to viral gastroenteritis, shock, dopamine infusion, exposure to low temperature and nonlactose fermenters sepsicaemia is presented for its rarity and devastating consequences.

INTRODUCTION
Symmetrical peripheral gangrene (SPG) is an uncommon clinical entity. It was first described in 1891 by Hutchison in a 37 years male who developed gangrene of fingers, toes and ear lobules after shock.1 It is a manifestation of many systemic disorders. Disseminated intravascular coagulation (DIC) is a commonly found in SPG and it is proposed to be a cutaneous marker of DIC.2,3 Uncommonly it is seen with infections, shock, drugs and malignancy. We describe a case where infection, shock, dopamine and prolonged exposure to low environmental temperature contributed in the development of symmetrical peripheral gangrene.

CASE REPORT
A 65 years old, previously healthy female presented with drowsiness and altered behavior. Prior to this she had diarrhoea and vomiting for three days. She did not have toilet in her house and was going out to pass stools in open air. The frequency of stool was so much that she preferred staying outside the house in the cold. The night temperature was quite low. She was taking oral rehydration solution and antibiotics without any relief. As her symptoms were not controlled she came to our institution. On examination, she was stuporose, resenting examination and responding inappropriately and inadequately to painful stimuli. She had cold and clammy extremities with a bluish tinge, dry axillae, tongue and sunken eyes. Her pulse was 130 per minute with a systolic BP of 80 mm of Hg. All peripheral pulses were palpable. She had oedema of lower limbs up to mid-leg. Abdominal examination revealed generalized tenderness and decreased intensity of intestinal sounds. Rest of her physical examination was normal. Her investigations showed haemoglobin - 9.5 gm/dl, leukocyte count - 11300/mm³ with polymorphs - 78%, lymphocytes - 22%, sedimentation rate 5 mm in 1st hour; peripheral blood smear showed anisocytes, microcytes, macrocytes and hypochromia. Neutrophils showed toxic granules. Platelet count was 1.48 lacs/mm³. Chest X-ray was unremarkable. Her coagulation profile was normal. Blood urea was 122 mg/dl, serum creatinine 2.8 mg/dl, plasma protein 6.8 gm/dl, albumin 3.6 gm/dl, bilirubin - 3.4 mg/dl, SGOT - 86 IU/L, SGPT - 92 IU/L, alkaline phosphatase - 16 KAU. Stool culture grew commensals, blood culture grew non-lactose fermenters (NLF) sensitive to ceftriaxone and urine was sterile. She was treated with IV fluids, IV ceftriaxone, and dopamine at 5.0 microgm/kg/min for one day. The patient’s condition improved steadily and dopamine was discontinued but she developed blue-black discoloration of the tips of fingers of both hands and feet. Later, she developed gangrene of the distal and middle phalanges of all the fingers of right hand, distal and middle phalanges of index, middle and ring fingers of left hand, great toe, distal phalanges of 2nd and 3rd toes of right foot and distal two-third of left foot. There were clear fluid-filled bullae over the left ankle which ruptured spontaneously (Fig. 1). Skin biopsy from gangrenous site revealed non-specific changes not compatible with vasculitis.

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or micro-thrombi. She was managed conservatively till the clear line of demarcation appeared before she could be undertaken for surgery. Her attendants took her to another institution where she underwent amputation of gangrenous parts along with skin grafting.

**DISCUSSION**

SPG is defined as symmetrical distal ischaemic damage in two or more sites in the absence of a major vascular occlusive disease. The distal ischaemic damage occurs in the fingers and toes, and rarely nose, upper lip, ear lobules or genitalia. It usually presents acutely. It is seen commonly in DIC. Other less common causes are infections, myocardial infarction, congestive cardiac failure, dog-bite, shock, hypertension, hyperosmolar coma, pulmonary embolism, paroxysmal ventricular tachycardia, appendicitis, Hodgkin’s disease, polymyalgia rheumatica and extra-corporeal shock wave lithotripsy. Among the infections meningococcaemia, streptococcal, pneumococcal, staphylococcal, *E. coli*, *Pseudomonas*, *S. paratyphi*, *Klebsiella*, *Proteus vulgaris*, *P. mirabilis*, *Pasteurella multocida*, DF-2 Gram negative bacillus, viral gastroenteritis, varicella and rubeola are known to cause it. It has also been reported in *Plasmodium falciparum* in a series of infection studies from India. Drugs like adrenaline, nor-adrenaline and dopamine are known to cause SPG whereas drugs like ergotamine, vasopressin etc. can cause vasospastic phenomena and are excluded from the aetiology of SPG.

SPG in our patient started with cyanosis of fingers and toes, followed by gangrene. She also had fluid-filled bullae over the right ankle which ruptured spontaneously. There were no skin manifestations of DIC in the affected parts. Clear-cut demarcation of gangrene was detected on the 7th day. Multiple factors like viral gastroenteritis, shock, prolonged exposure to cold, dopamine and NLF organism induced septicaemia were operating in our patient. SPG has been successfully treated with epoprostenol and tissue plaminogen activators. The natural history of SPG has many similarities with severe cold injury. In both the conditions there is dry gangrene, mummification and absence of infection. It has therapeutic bearing. The peripheral tissue injury in both should be treated with inter-digital padding and protected from trauma. Early surgery should be resisted as the initial assessment of tissue damage may not be correct and viable tissue may be sacrificed during amputation. Preservation of joint mobility and range of motion is achieved by early physiotherapy.

**REFERENCES**