Symmetrical peripheral gangrene (SPG) is a rare clinical entity, infective, and noninfective both types of etiologies are responsible. The basic underlying pathology in SPG is being disseminated intravascular coagulation and carries a high mortality. Here, we describe a 52-year-old male with dengue fever, who developed bilateral symmetrical dry gangrene of both hand and feet. His dengue IgM antibody was positive. All the peripheral pulses of the affected limbs were palpable. Color Doppler study of upper and lower limb vessels showed normal flow. The patient was managed with intravenous fluids, low molecular weight heparin, and fresh frozen plasma. His general condition was improved within 72 h with no further progression of gangrene. Clinician should suspect the possibility of SPG while dealing a case of dengue fever presenting as peripheral gangrene.

Key Words: Disseminated intravascular coagulation, symmetrical peripheral gangrene, dengue fever

INTRODUCTION

Symmetrical peripheral gangrene (SPG), is a rare clinical syndrome, characterized by symmetrical distal acrocyanosis, leading to gangrene of two or more sites in the absence of large vessel obstruction or vasculitis.\(^1\) It is a manifestation of numerous systemic diseases and thought to be cutaneous marker of disseminated intravascular coagulation (DIC).\(^2,3\) Uncommonly it is seen in infection, shock, drug, and toxins. Dengue infection is presented with a variety of symptoms. The hallmark of this infection is increased vascular permeability with coagulation disorders. These mechanisms can explain various systemic involvements.

CASE REPORT

A 52-year-old male presented at emergency with fever, severe myalgia, pain in small and large joints for 7 days with generalized maculopapular, erythematous itchy rash, that appeared on the 4th day of fever and gradually decreased over the next 3 days. He was complaining of severe pain along with the symmetric blackish discoloration of both hands and feet since last 2 days [Figure 1]. With the above complaints, the patient was admitted to our department. Fever was subsided on 8th day from the onset of fever.
On general examination, he was febrile (103°F), dehydrated with tachycardia (heart rate 108/min), tachypnoea (respiratory rate 26/min), and hypotension (blood pressure 90/60 mmHg) with normal jugular venous pressure. All the peripheral pulses of the affected limbs were palpable. Systemic examination was normal. Local examination revealed cold, dry, and wrinkled skin of both hands and feet, with a clear line of proximal demarcation, and incipient gangrenous changes [Figure 2]. There was no history of trauma, drug ingestion such as β-blockers, ergot, smoking, and Raynaud’s phenomenon. Intravenous line was secured, ringer lactate was started, and investigations were sent to the laboratory. Reports were as follows in Table 1. Color Doppler study of upper and lower limb vessel revealed a normal flow. Echocardiography did not reveal the evidence of vegetation or thrombus. Histopathological examination showed the area of dry gangrene with hyperkeratosis, hypergranulosis, rete ridge, and necrotic changes.\cite{4}

On the basis of clinical findings and investigation reports, a diagnosis of SPG was made. Patient was managed conservatively with intravenous fluid, low molecular weight heparin, broad-spectrum intravenous antibiotic, fresh frozen plasma was transfused, and warming of extremities were initiated. His general condition was improved within 72 h. Further progression of gangrene was stopped. Clear line of demarcation appeared on 5th day of admission [Figure 2], and surgical consultation was taken, and advised amputation but patient refused. At present, the patient is under follow-up treatment in medical outdoor [Figure 3].

**DISCUSSION**

SPG is characterized by ischemia followed by gangrene of two or more sites without the occlusion of large vessel.\cite{1} Important clinical features of fever followed by marked coldness, pallor, cyanosis, pain, and restricted mobility of extremity always arose with the suspicion

| Table 1: Investigations |
|-------------------------|
| Parameter               | Patients values | References values |
| Hemoglobin              | 10 g/dL         | 11–13 g/dL |
| Total leukocyte count   | 3400/mm³        | 4000–110,00/mm³ |
| Platelets               | 42,000/mm³      | 140,000–450,000/mm³ |
| Prothrombin time        | 16.5            | 11.5–15.5 |
| SGOT                    | 124.53          | 7–21 IU/L |
| SGPT                    | 125.39          | 8–32 IU/L |
| INR                     | 3.67            | <1.5 |
| D-dimer                 | 1.30            | 0–0.5 µg/ml |
| Random blood sugar      | 84 g/dL         | 70–125 mg/dL |
| Antinuclear factor,     | Normal          | |
| anti-CCP cANCA, pANCA   |                | |
| Antibody to protein C and S C3-C4 complement | Normal | |

SGOT: Serum glutamic oxaloacetic transaminase, SGPT: Serum glutamic-pyruvic transaminase, INR: International normalized ratio

![Figure 1: Showing dry wrinkled skin of both hand and feet with incipient gangrenous changes](image1.png)

![Figure 2: After 1-weeks of follow-up, changes in gangrenous area](image2.png)

![Figure 3: After 2 weeks of follow-up, necrotic changes with a clear line of proximal demarcation](image3.png)
of SPG.\(^1,5\) There are various causes responsible for SPG, but important are infective and noninfective causes. Common infective organisms responsible for SPG are \textit{Pneumococcus}, \textit{Staphylococcus}, \textit{Streptococcus}, \textit{Enterococcus}, \textit{Klebsiella}, \textit{Plasmodium falciparum}, \textit{Varicella zoster}. Various noninfective causes such as myocardial infarction, pulmonary embolism, cardiac failure, hypovolemic shock, decreased levels of protein C and protein S, antiphospholipid antibodies, cryoglobulinemia, acute lymphatic leukemia, and systemic lupus erythematosus may also cause SPG. Other important factors that decrease the resistance power of the body such as immuno compromised patients, diabetics, patient on chronic hemodialysis, and patient taking corticosteroid, are more prone to the development of SPG. The pathogenesis of SPG may include bacterial endotoxin release and development of DIC.\(^1\)

As per other studies, drugs such as adrenaline, noradrenaline, and dopamine can also cause SPG.\(^6,7\) Drugs such as ergotamine and vasopressin can cause vasospastic Raynaud’s phenomena and thus these drugs excluded from the etiology of SPG. The other aggravating factors such as asplenia, immunosupression, diabetes mellitus, and renal failure are also reported.\(^8\)

In our case by excluding other causes, a diagnosis of dengue fever with SPG was made. Dengue fever is the most important arthropod–borne arboviral infection.\(^9\) As per WHO estimation, there may be 50–100 million dengue infections worldwide every year.\(^10\) Various rare complication of dengue infection such as liver failure, DIC, encephalopathy, myocarditis, acute renal failure, and hemolytic uremic syndrome are reported that can be associated with SPG.\(^11\) DIC can be associated with SPG. In our case, SPG might be due to DIC as shown by increased fibrin degradation products, D-dimer assay, and increased prothrombin time. The upper and lower extremities, border of the ears, genitalia, tip of the nose, and scalp are more commonly affected because they are situated most peripherally.\(^11\) If leucopenia is present along with SPG, it predicts poor outcome.\(^34\) Identification and treatment of underlying etiological factors and treatment of DIC have remained the mainstay of management. Various other modalities of treatment like sympathetic blockade, intravenous nitroprusside therapy, local, or intravenous infusion of a beta-blocker (phenolamine, chlorpromazine) topical nitroglycerine ointment, and intravenous infusion of prostaglandin (epoprostenol) might be helpful to relieve the symptoms of the patient.\(^5\)

CONCLUSION

SPG carries a high morbidity and mortality. Identification and treatment of underlying etiological factors and treatment of DIC is the mainstay of management. A high index of suspicion and prompt management with usual measures may limit the progression of the disease and the damage caused by gangrene.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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Conflicts of interest

There are no conflicts of interest.

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