Symmetrical Peripheral Gangrene : An Unusual Presentation of Dengue Fever

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Abstract
Symmetric peripheral gangrene (SPG) can be associated with a variety of infective and non-infective etiologies. SPG occurs because of disseminated intravascular coagulation. It has a high mortality rates. Here, we report a case of 30-year-old female with dengue fever and rash with development of bilateral symmetric dry gangrene of hands. The peripheral pulses of the affected limbs were palpable equally bilaterally. Her dengue IgM antibody was positive. Patient was managed with intravenous fluids, low molecular weight heparin and fresh frozen plasma etc. Her general condition improved within 48-72 hours. Thus it is important to investigate patient on the lines of dengue fever specially when patient present with high grade fever, myalgias and symmetrical peripheral gangrene.

Key Words
Symmetric Peripheral Gangrene, Dengue Fever, Disseminated Intravascular Coagulation; Low molecular Weight Heparin

Introduction
Symmetrical peripheral gangrene (SPG) is a rare clinical entity. It was first described by Hutchinson in 1891. (1) SPG is manifestation of numerous systemic diseases. SPG is thought to be a coetaneous marker of DIC, because underlying mechanism for developing SPG is DIC. (2, 3) Uncommonly it is seen in infection, shock, drug and toxins. Here we report a case of 30 year old female presented with SPG of hands as a complication due to dengue fever.

Case Report
A 30-years-old female was presented at emergency of Guru Nanak Dev Hospital, Amritsar with chief complaints of fever, muscular pains, and pain in small and large joints for six days. She also gave history of maculopapular and erythematous itchy rash, which appeared on fourth day of fever and gradually decreased over next 3 days. She was complaining of severe pain along with symmetric blackish discoloration both of her hands. Local examination revealed cold, dry, wrinkled skin of both hands with a clear line of proximal demarcation and incipient gangrenous changes (Fig 1). All the peripheral pulses of the affected limbs were palpable. She became afebrile on seventh day. Her hemoglobin was 10 gm%, total leucocyte count 2800/mm3, platelet 68,000/mm 3, SGPT-46 IU/ml, SGOT-32/ml, alkaline phosphatase-157 IU/ml. Serum IgM dengue antibody were positive. There was no history of taking ergot or beta blockers. Fibrin degradation products and D dimers were positive in serum. Prothrombin time was 25 Sec, with INR of 2.7. Anti-nuclear factor, anti- cyclic citrullinated peptides antibody, anti-phospholipid antibody, antibodies to protein C and S were negative.

Colour Doppler of upper limb vessels was done, which indicated normal flow. Her echocardiography was with in normal limits. Histo-pathological examination showed area of dry gangrene with necrotic debris.

On the basis of clinical and pathological findings, a diagnosis of symmetrical peripheral gangrene was made.
Patient was managed conservatively and started low molecular weight heparin, fresh frozen plasma and intravenous fluids and broad spectrum IV antibiotics. Disease dis not progressed further. Orthopedic consultation was taken and advised amputation but patient refused.

Discussion

Symmetric peripheral gangrene is characterized by symmetrical distal ischemic damage, leading to gangrene of two or more sites in the absence of major vaso-occlusive disease. A physician should always suspect SPG when there is fever followed by marked coldness, pallor, cyanosis, pain, and restricted mobility of extremity. (4,5) Numerous causes are described for SPG. The common infective pathogens are Pneumococcus, Staphylococcus, Streptococcus, Klebsiella, Neisseria, Enterococcus, Plasmodium falciparum, Varicella zoster etc., in the non infective etiology there is Myocardial infarction, pulmonary embolism, cardiac failure, hypovolemic shock, systemic lupus erythematosus. Immuno deficient patients like diabetic, patient on chronic hemodialysis, patient taking corticosteroid, are more prone to development of SPG. The pathogenesis of SPG may include bacterial endotoxin release and DIC. (4)

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. (6) Severe complications of dengue infections such as liver failure, disseminated intravascular coagulation, encephalopathy, myocarditis, acute renal failure, and hemolytic uremic syndrome are rare but can be associated with it. (7) DIC can be associated with SPG. In our case DIC is the cause of SPG as shown by increased FDP, D-dimer assay, and increased PTI. The upper and lower extremities, borders of the ears, genitalia, tip of the nose and scalp are the areas which are more commonly affected because they are situated most peripherally. (4) If leucopenia is associated with SPG, it is predictor of poor outcome. (3) Identification and treatment of underlying etiological factors and treatment of DIC have remained the mainstays of management. Other measures that might be helpful are sympathetic blockade, i.v.nitroprusside therapy, local or intravenous infusion of a beta-blocker (phenolamine, chlorpromazine) topical nitroglycerine ointment and i.v. infusion of prostaglandin (epoprostenol). (5)

Conclusion

SPG can be associated with various systemic diseases. The basic underlying pathology is being DIC. SPG should be thought of as a possibility while dealing a case of dengue fever presenting as bilateral peripheral gangrene.

References

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