

Symmetric peripheral gangrene secondary to Sepsis

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Abstract

Symmetric peripheral gangrene is a well-documented but rare clinical syndrome characterized by symmetrical distal ischemic damage leading to gangrene of two or more sites in the absence of large vessel obstruction or vasculitis. We report here one such case associated with sepsis.

Keywords: Sepsis, gangrene, symmetric

1. Introduction

Symmetric peripheral gangrene is a well-documented but rare clinical syndrome characterized by symmetrical distal ischemic damage leading to gangrene of two or more sites in the absence of large vessel obstruction or vasculitis.

2. Case Study

A 40 year old gentleman presented to our institute with history of breathlessness, coughs with expectoration, pain abdomen, and swelling of lower limbs, decreased urine output since 7 days and altered sensorium since 2 days. Patient was chronic alcoholic, non-smoker with no comorbidities. On the day of admission patient had tachypnea (RR-26cpm), tachycardia (PR-116bpm), raised JVP and bilateral pedal edema. Systemic examination showed bilateral coarse crepitation and hepatomegaly.

Investigations on day of admission revealed high leukocyte count(15,300cells/cumm),normal liver function tests and acute kidney injury(blood urea 62mg/dl, s. creatinine 3.2mg/dl), HIV and HBsAg were negative, Urine routine shows 4 to 6 pus cells, CXR showed right pneumonic consolidation with pleural effusion (Figure 1).

Figure 1: Chest X Ray showing consolidation, pleural effusion.



ECG showed ST depression in V3-V6 leads with P pulmonale, 2D echocardiography showed ischemic heart disease, globally hypokinetic left ventricle, mild pulmonary artery hypertension and ejection fraction of 32%.

For the above complaints patient was treated with antibiotics, diuretics, antiplatelets and anticoagulants. After 2days of admission patient developed gum bleeding, which subsided after stopping antiplatelets and anticoagulants? Coagulation parameters showed aPTT 31.8 sec (control 28sec), PT 17.6sec (control 14sec) and INR 1.2. Over the next 4 days, patient developed icterus, symmetrical gangrene of all four limbs and on examination all peripheral pulses were palpable (figure 2 and 3).

Fig 2: Distal symmetric gangrene of toes.



Figure 3: Distal symmetric gangrene of hands.



Further Investigations showed elevated leukocyte count (29,800cells/cumm), thrombocytopenia (60,000 cells/cumm), deranged LFT(i.e. total proteins 5.2g/dl, serum albumin 2.3mg/dl, total bilirubin 3.0mg/dl, direct bilirubin 1.7mg/dl, AST 305U/L, ALT 401U/L and ALP 78U/L.), deranged RFT (blood urea 194mg/dl and serum creatinine 12.6 mg/dl); antinuclear antibodies, rheumatoid factor and anti HCV were negative, ultrasonography of abdomen showed congested hepatomegaly and kidneys size and echo texture were normal, blood culture and urine culture showed no growth.

Finally patient went against medical advice after 5days as patient was not willing for dialysis and further treatment.

3. Discussion

Symmetrical peripheral gangrene (SPG) is a well-documented but rare clinical syndrome characterized by symmetrical distal ischemic damage leading to gangrene of two or more sites in the absence of large vessel obstruction or vasculitis.[1] Although first described more than a century ago by Hutchison [2], most of the cases of SPG have been documented as single case reports.

The pathogenesis of SPG is not well understood. A low-flow state is commonly present in association with a vasospastic, hypercoagulable state leading to microcirculatory occlusion. These ischemic changes begin distally and may advance proximally to involve a whole extremity. The pathogenesis of SPG may involve the Schwartzman reaction, bacterial endotoxin release, and platelet plugging in peripheral arterioles due to vascular collapse and DIC. [3] DIC might be associated with 85 to 100% of cases of SPG.[4] DIC most commonly occurs due to sepsis and pneumococcus is the most common organism responsible. Other organisms like staphylococcus, streptococcus and gram-negative organism have also been implicated. In case of septicemia, activation of neutrophils and release of vasoactive substances also play a role.[4]

Symmetric peripheral gangrene can also occur as a complication of malignant disease (paraneoplastic syndrome), ergotism or protein C deficiency. Aggravating factors include asplenia, immunosuppression, previous cold injury to extremities, diabetes mellitus, renal failure, increased sympathetic tone, use of vasopressors and winter season [5].

SPG should be suspected at the first sign of marked coldness, pain, cyanosis or pallor in the extremity, as the condition can progress rapidly to acrocyanosis and, if not reversed, frank gangrene. The ischemic changes begin distally and may progress proximally to involve the entire extremity. These changes are not ordinarily preceded by demonstrable peripheral vascular occlusive disease on angiography and may be associated in the early stages with intact distal pulses because the large vessels are often spared. The low-flow state results in occlusion of the microcirculation of the affected parts.[5]

SBP had high mortality rate of up to 35%, rates of amputation ranging from 70 to 90%.³ No treatment is universally effective. Early recognition

and immediate discontinuation of vasopressor, if possible and vigorous therapy of sepsis and DIC with intravenous antibiotic therapy and heparinization (if feasible) are essential components of SPG management. Other measures that might be helpful are sympathetic blockade, intravenous nitropruside therapy, topical nitroglycerine ointment, local or intravenous infusion of an α -blocker (phentolamine, chlorpromazine) and intravenous infusion of prostaglandin (epoprostenol).

4. Conclusion

SPG carries a high morbidity and mortality. A high index of suspicion and prompt management with usual measures may limit the progression of gangrene, limb saving and life saving. Here we present a case to SPG occurring in patients with sepsis.

References

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