Tubercular meningitis presenting as Gangrene in all four limbs in an elderly patients: an empathetic relationship

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Abstract
Gangrene is ischemic necrosis of limbs or its parts, without involvement of proximal arterial obstruction or vasculitis, often associated with disseminated intravascular coagulation and septic shock. This rare clinical syndrome are usually caused by infectious or non infectious causes, among them tuberculosis as a cause is rarely associated. We here report a case of 68 year old lady, who initially was diagnosed as tubercular meningitis and later developed peripheral gangrene in all four limbs.

Keywords: Tubercular meningitis, Gangrene, Limbs, Elderly.

Introduction
Peripheral gangrene of the limbs is distal ischemic damage without large vessel obstruction or vasculitis.1 This rare clinical entity is an emergency condition generally seen with disseminated intravascular coagulation and sepsis due to infective and non infective etiological factors. Most of the cases have been documented as rare case reports.2-3 The exact mechanism is not known but low-flow state along with disseminated intravascular coagulation has been suggested.4-5 This condition can lead to significant morbidity and mortality due to limb amputations in the survivors. Here we report a case of 68 year old lady, who initially was diagnosed as tubercular meningitis, developed gangrene in all four limbs and later succumbed to her illness. Thus, its early recognition and its etiologies can have profound impact on the management of the condition and its final outcome.

Case Report
A 68 year old female presented to the department of medicine with the chief complaints of low grade fever off and on since two weeks, headache and altered sensorium since 5 days. There was no history of any chronic illness like pulmonary tuberculosis in past. She denied any history of alcohol consumption, smoking, and use of any medications like corticosteroids or ayurvedic drugs. There was no history of hypertension, diabetes mellitus and ischemic heart disease.

On examination, the patient was drowsy and febrile. Pulse rate was 116/min and blood pressure was 110/60 mm of Hg. Neck stiffness was present and Kernig’s sign was positive. Other systemic examination was normal. On laboratory investigations, hemoglobin was 10.8 g/dl and the white cell count was 14,200 with neutrophils 62% and lymphocytes 28%. Liver function and kidney function tests were normal. Routine examination of Urine was normal. Her ESR was 112 mm in first hour. Chest X-Ray showed no obvious abnormality. Blood cultures showed no growth of organisms. In view of headache and altered sensorium lumber puncture was done and cerebro spinal fluid (CSF) examination revealed 800 cells among them 90% were lymphocytes, raised protein (98mg/dl) and low glucose (15mg/dl). CSF ADA was in strong suspect range for TB meningitis. MRI brain showed features of tubercular meningitis. She had 1 episode of generalized seizure episode during the hospital stay. She was started on anti tubercular drugs, corticosteroids, anti epileptics and other supportive management like antibiotics and intravenous fluids. On 2nd day patient’s blood pressure started dropping for which patient was stared on ionotropic supports. Repeat blood investigations showed thrombocytopenia with platelet count of 57,000 and PT/INR was 1.98 with a PTT of 45 sec. No bleeding manifestations were seen. Patient also developed acute renal failure with serum urea of 271 and serum creatinine of 4.52. Slow and low efficient hemodialysis was started in view of hypotension. The presentation was suggestive of septic shock with DIC secondary to tuberculosis. On 3rd day patient started developing acrocyanosis in all 4 limbs with cold extremities. Repeat blood cultures showed no bacterial growth. Blood reports were negative for dengue fever, malarial fever, scrub typhus. All peripheral pulses were well felt. Doppler study of all 4 limbs was normal. Her ANA and ANCA levels were within normal limits. The cyanosis progressed to gangrenous changes within 2 days. Patients platelet counts dropped to 18,000/mm3 and PT/INR increased to 2.8 and aPTT of 56 sec. Patient was continued with treatment for TBM and was transfused with platelets and fresh frozen plasma. Pentoxyphyllin tablets were given with topical nitroglycerine ointment. On11th day a line of demarcation of the gangrene was established in all 4 limbs (Fig. 1). No surgical intervention was done in view of the poor general condition of the patient. During further hospital stay her general condition kept deteriorating. Patient eventually succumbed to the disease on 14th day of hospitalization.
Discussion
Proposed mechanisms of this gangrene in all four limbs may be the Shwartzman reaction, bacterial endotoxin release, arteriolar platelet plugging in peripheral arterioles due to vascular collapse and disseminated intravascular coagulation (DIC). The DIC being the final common pathway of its pathogenesis as its association are seen in 85-100% of cases.

Most of the time patient presents with fever and painful extremities. Coagulopathy, shock and necrotic areas over extremities are common and usually progress to acrocyanosis and dry gangrene. Involvement is generally symmetrical and large vessels are characteristically not involved. Apart from the extremities, other acral areas like tip of the nose, the ears, genitalia, and the scalp may be involved.

Differential diagnosis of this condition may be atherosclerosis, Buerger’s disease, thromboembolic gangrene, Raynaud’s phenomenon, frostbite, ergotism, calciphylaxis, postoperative thrombotic thrombocytopenic purpura, myeloproliferative disorders, and vasculitis. Gangrene resulting from tuberculosis is uncommon, supported by few case reports. Possible mechanism may be embolization of arterioles by the tubercular bacilli and vasculitis associated with it.

Conclusion
The purpose of this case report is to highlight this uncommon presentation of tubercular meningitis, which is a common condition in our country.

Conflicts of Interest: None.

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How to cite this article: Beheti A, Kumar S, Husain A, Godhiwala P, Raisinghani N. Tubercular meningitis presenting as Gangrene in all four limbs in an elderly patients: an empathetic relationship. Ann Geriatrics Educ Med Sci 2019;6(1):9-10.