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Case Report

Purpura fulminans: MRSA sepsis compounded by diclofenac

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Abstract Purpura fulminans is a rare, albeit a life-threatening emergency which has been reportedly triggered by infections, congenital disorders of protein C or S deficiencies, as well as drugs. Here we report a case of a previously healthy 17 year old male who developed purpura fulminans after fever, sore throat and then getting injection diclofenac. Blood cultures grew *Staph. aureus.* This case is the first to report two causative etiologies in a single patient.

Key words
Purpura fulminans; MRSA, Diclofenac.

Introduction

Purpura fulminans is a life-threatening hematological emergency characterized by progressive hemorrhagic necrosis of the skin due to cutaneous vascular thrombosis, and disseminated intravascular coagulation.[1,2] It is usually accompanied with circulatory collapse. The usual causes are congenital, idiopathic, and infectious.[2,3] Here we present a case of a young boy who had both infectious and drug-related etiology.

Case report

A 17 year old male developed the complaint of high grade fever for 2 days, along with sore throat, myalgias, prostration, and cough. At a local dispensary, he received injection Lincomycin, inj. Hydrocortisone, inj. Dicofenac IM, and inj. Omeprazole. Within 2 hours, he complained of tingling and pain in his fingers and toes which rapidly, within 6-8 hours, developed edema and eruption of ecchymotic and purpuric patches and plaques all over the body, but mostly on the extremities, along with excruciating pain. His digits became blue and cold. He was taken to a local DHQ where he was found to be in shock and after stabilization, brought to our hospital after a delay of 1 day. On admission, his vitals were BP 80/50 mmHg, PR 126 bpm, Temp. 103^0F. Multiple purpuric plaques were seen, mostly on extremities, distal digits were cyanosed, cold and edematous. Peripheral pulses were intact with no signs of meningeal irritation and his GCS was 15/15. No prior significant personal or family history was elicited.

His labs on admission showed: Hb: 10.3g/dl WBC: 16.5* 10^3/uL (91.5% neutrophils) Platelets: 42 * 10^3/uL (confirmed on peripheral smear) CRP: 15.78 mg/dl (N<0.5) ESR: 85 FDP's: >1600 (N<250) INR 1.7 CPK: 1731 U/L LDH: 732 U/L ALT: 61 IU AST: 98 IU ALP: 226 IU Bilirubin: 1.0 Albumin: 2.3 g/dl Total protein: 4.8g/dl S/Urea: 43mg/dl S/Creatinine: 0.7mg/dl Urine C/E: Pus cells: 6-7/hpf RBC's: 10-11/hpf

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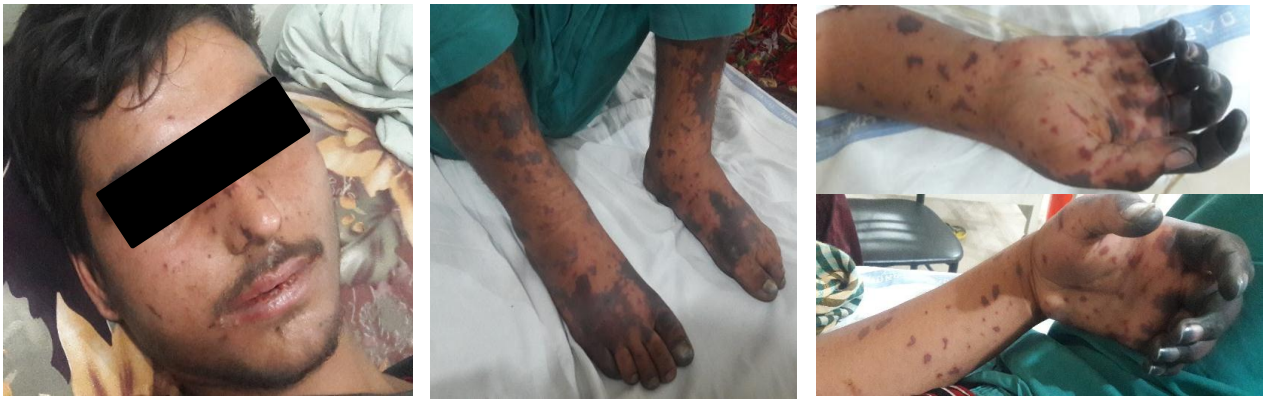

Figure 1 On the day of admission- multiple ecchymotic patches and plaques with digital cyanosis.

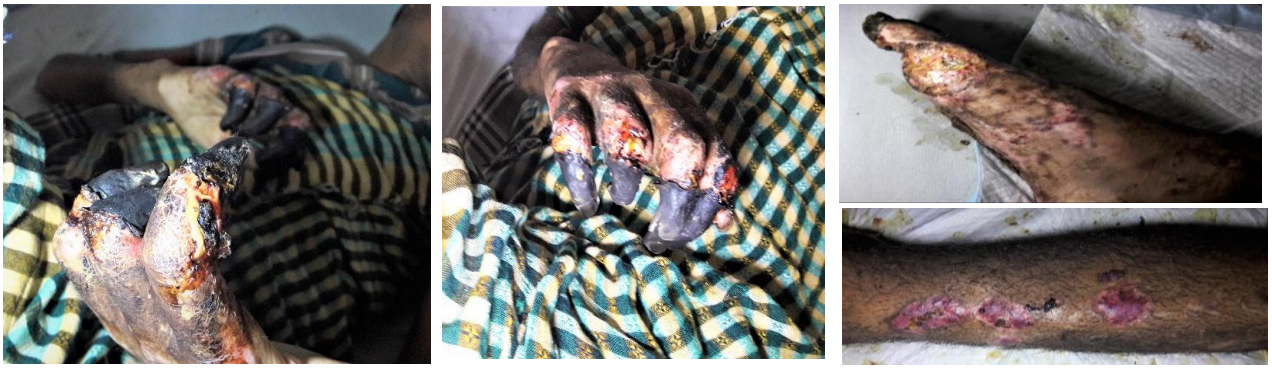

Figure 2 After development of necrotic plaques, before amputation of fingers of both hands.

Blood cultures: *Staphylococcus aureus* (MRSA) ASOT: 65.9; HBsAg and Anti-HCV non-reactive, Dengue IgM and MP slide negative

He was given 3 FFP infusions and 1 whole blood, along with saline infusions to maintain his blood pressure and urinary output. He was started on vancomycin, heparin, methylprednisolone, nifedipine (while monitoring BP), and aspirin. His vitals improved and his lab parameters afterwards were:

Hb: 11.7g/dl WBC: 7.1*10^3/ul Platelets: 362*10^3/ul Albumin: 3.1g/dl INR: 1.3 Protein C, S, Anti-thrombin III and Factor V Leiden were all within normal limits. D-dimers also became normal.

Skin biopsy revealed multiple thrombi occluding the cutaneous vessels along with frank hemorrhage, supporting the clinical diagnosis of purpura fulminans.

The necrotic plaques ulcerated, and healed with post-inflammatory pigmentary changes. Unfortunately, the fingers of both hands developed distal dry gangrene, and after the line of demarcation had formed, in consultation with the surgical department, amputation of fingers of both hands was done, sparing the thumbs. The patient was referred to rehabilitation centre for prosthesis fitting, and extensive psychological support and therapy.

Discussion

Acute infectious purpura fulminans is usually caused by *Meningococcus* sp., *Staphylococcal* sp. and *Pneumococcal* sp.[3] In addition, rare reports also cite *Hemophilus influenza, Capnocytophaga canimorsus, E.coli,*[3] and *Klebsiella pneumoniae.*[4] Rickettsial disease and malaria may also present as purpura fulminans. Viral infections like measles, varicella, HHV-6 and Covid-19 have also been reported to be a

causative agent.[5] Apart from infectious etiology, the neonatal type is usually due to congenital protein C and S deficiency.[6,7] A few rare cases have been described as having factor V Leiden mutation, with normal protein C and S levels. SLE and vasculitides have also been cited as triggering factors of purpura fulminans.

Drug as a causative agent has been frequently reported in the literature, with non-steroidal anti-inflammatory drugs diclofenac and ketorolac, phenytoin and quinidine.[8] Diclofenac has also been associated with rhabdomyolysis, as in our patient.[9] No case reports have been reported so far in the literature regarding the association of injection lincomycin or omeprazole with triggering the purpura fulminans. In our patient, the previous history of tonsillitis was significant as infections can decrease the serum concentration of protein S.[10] Bacterial endotoxins can create a temporary hypercoagulable state. In addition to the already present infection, he received an additional dose of IM diclofenac which resulted in the resulting devastating complications and loss of his digits with marked psychosocial consequences for the young man. Urgent medical attention, with restoration of circulation, infusion of fresh frozen plasma, along with antibiotics is the mainstay of management.[1] Time is critical as our patient presented late while the digital gangrene had already set in and thus could not be saved. This case report highlights the need for medical practitioners to familiarize themselves with the possible complications of widely used drugs as well as infections to minimize the adverse effects on the patient's entire life with prompt treatment.

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