ABSTRACT

A 32 year old female, an active intravenous drug user, was admitted for fever, myalgias and an erythematous macular rash on her distal extremities. She quickly decompensated and developed septic shock. Her examination was significant for a progressive rash which within two days developed bullae and necrosis with progression to a confluent rash involving her palms and soles (Figs. 1 and 2). Her rash involved nearly one third of her body with what was equivalent to a third degree burn. Her labs were significant for leukocytosis with bandemia, elevated liver function tests with worsening thrombocytopenia and fibrinogen levels consistent with disseminated intravascular coagulation (DIC). Her transthoracic echocardiogram (Fig. 3) showed a 5 cm vegetation on the tricuspid valve. Her blood cultures were positive for methicillin-sensitive Staphylococcus aureus. She was meeting the clinical criteria for toxic shock syndrome (TSS) and subsequent testing for toxic shock syndrome toxin antibody was positive. She was treated with antibiotics and intravenous gamma globulin (IVIG). Due to her worsening rash she was transferred to a burns unit. She was diagnosed with Purpura fulminans (PF) which is a skin manifestation of DIC and has a rare association with Staphylococcus aureus infection. The main focus of this case report is to emphasize this rare association, prompt an early diagnosis and referral to prevent life threatening complications.

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Case illustrated

Sloughing skin in intravenous drug user

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admitted and left against medical advise without subsequent follow up.

Discussion

Purpura Fulminans is a skin manifestation of disseminated intravascular coagulation. Pathogenesis involves thrombosis of small dermal vessels causing necrosis of the skin. It can be broadly classified into three categories. Inherited or acquired abnormalities of the protein C or coagulation system (generally seen in children), acute infectious and idiopathic causes. The most common acute infection with which PF is associated is meningococcemia. The endotoxin produced by the organism causes an imbalance between procoagulant and anticoagulant pathway producing the skin changes of PF. In our case, the causative organism was *Staphylococcus aureus* which has a rare association with PF per literature review. It is possible that reaction to super antigen production by *Staphylococcus aureus* is the basis for PF. It’s clinical significance is that it acts similar to a third degree burn which may require multiple debridements and possibly amputations. Treatment involves administering protein C infusion, corticosteroids and possibly IVIG along with routine sepsis care.

*Staphylococcus aureus* infection is a very common infection. The main focus of this case report is to emphasize this rare association with PF and to guide clinicians for prompt identification and referral to avoid life threatening complications.

Author’s contribution

Data collection and compilation, literature review.

Conflict of interest

None.