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Case illustrated

Sloughing skin in intravenous drug user

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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 emphasise this rare association, prompt an early diagnosis and referral to prevent life threatening complications.

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 DIC. Her transthoracic echocardiogram (Fig. 3) showed a 5 cm vegetation on the tricuspid valve.

Her blood cultures revealed methicillin-sensitive *Staphylococcus aureus* and she was treated with cefazolin 2 g IV every 12h, clindamycin 600 mg IV every 8 h and intravenous gamma globulin 10% infusion 200 mg/kg for suspicion of toxic shock syndrome. Patient received total of 5 day therapy and was transferred to an outside burns unit. She was found to be positive for toxic shock syndrome toxin antibody however the test was designed to identify antibody negative individuals at risk for toxic shock syndrome and not for diagnostic purposes. It provided information regarding people who would be at risk of developing toxic shock syndrome if antibodies were negative.

This patient had Purpura fulminans due to *Staphylococcus aureus* infection. Also, she had manifestations of toxic shock syndrome which unfortunately could not be confirmed on laboratory testing due to diagnostic limitations.

Diagnosis: Methicillin sensitive *Staphylococcus aureus* tricuspid endocarditis, Purpura fulminans with Toxic shock syndrome and Disseminated intravascular coagulation.

Epilogue: Patient was transferred to a burns unit for further management of PF where she underwent multiple digit amputations for the gangrenous changes. For her valvular vegetation she underwent tricuspid valve replacement after which she developed complete heart block requiring a pacemaker. Her clinical course was also significant for multiple septic pulmonary emboli complicated by empyema requiring bilateral chest tube placement. During her prolonged hospital stay she was treated with oxacillin for methicillin-sensitive infective endocarditis and was discharged to an acute rehabilitation facility. Patient continued using intravenous drugs and was re-admitted with methicillin resistant prosthetic tricuspid valve and native aortic valve endocarditis. Patient also had early manifestations of PF which was caught and intervened upon in a timely fashion. No surgical interventions were done. Patient was discharged on intravenous antibiotics and had subsequent visit in the emergency department for fever with concerns for ventricular abscess. However patient did not want to be

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Fig. 1. Bullae formation with seepage of fluid.

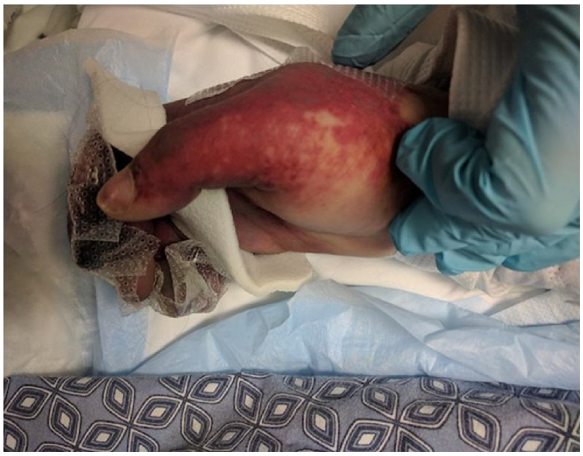

Fig. 2. Involvement of palms with gangreneous changes.

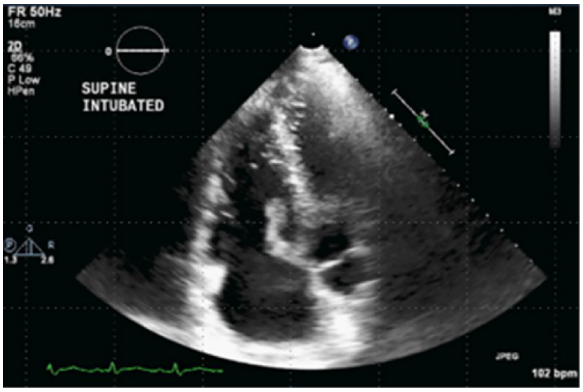

Fig. 3. Transthoracic echocardiogram showing a 5 cm vegetation.

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.