A CASE REPORT ON POST INFECTIOUS PURPURA FULMINANS IN INFANT

P.V.S.N.Eswari1*, V.Madhavi1 and T.S.Durga Prasad2

1Pharm.D Intern, Department of Pharmacy Practice, Sri Padmavathi School of Pharmacy, Tiruchanoor, Tirupati, Andhra Pradesh, India.
2Associate Professor, Department of Pharmacy Practice, Sri Padmavathi School of Pharmacy, Tiruchanoor, Tirupati, Andhra Pradesh, India.

*Corresponding Author: P.V.S.N.Eswari
Pharm.D Intern, Department of Pharmacy Practice, Sri Padmavathi School of Pharmacy, Tiruchanoor, Tirupati, Andhra Pradesh, India.

ABSTRACT

Purpura Fulminans (PF) is a life-threatening thrombotic disorder of acute onset which is characterized by cutaneous hemorrhage due to severe bacterial, viral infections or may be idiopathic. We present a case report of 11 month old male child presented with purpura fulminans with symptoms of skin lesions over dorsal aspects of both arms, both lower limbs and ulcerative lesions developed over scrotum. Patient had chicken pox 1 week prior to the presentation. Results: Staphylococcus aureus was isolated from the swab culture but the bacterium was not isolated from the blood culture. He received antibiotic therapy, fresh frozen plasma, soframycin ointment. The skin lesions began to implement a remission process. Conclusion: Purpura fulminans due to Staphylococcus aureus is a newly and emerging disease commonly associated with superantigen production.

KEYWORDS: Purpura Fulminans (PF), superantigen production.

INTRODUCTION

Purpura Fulminans (PF) is a life-threatening disorder of acute onset which is characterized by cutaneous haemorrhage. It was first described in 1884 by Guelliot.[1] Later PF has been found to be associated with gram-positive and gram-negative bacteria (meningococcal) and viral infections.[2] It is a hemorrhagic condition featured by hypotension, DIC and purpura leading to tissue necrosis. Whereas gram negative organism remains the commonest cause of acute infectious type, S. aureus infection has only been rarely complicated by purpura fulminans.[5] PF has a characteristic erythema with central areas of a blue-black haemorrhagic gangrenous necrosis which has a surrounding erythematous border. The necrosis may extend to the muscle and the bone, contributing to a late mortality and morbidity. Its healing leads to scarring, and auto-amputation of the digits.[6] The pathogenesis of PF is not known but may involve acute transient decrease in protein C (PC), protein S (PS) or anti-thrombin III. Varicella-associated PF is a rare syndrome with substantial morbidity and mortality. Onset is often sudden, usually 7–10 days after the onset of common chickenpox.[3] Here with we are reporting purpura fulminans to a child after 7 days of varicella infection which was treated and cured.

CASE REPORT

An 11 months old male child presented with a history of fever 20 days back and varicella 1 week back. Now presented with complaints of ulcerative lesions over dorsal aspects of both limbs and scrotum. Patient had chicken pox 1 week prior to the presentation. PLT:1.24 lakhs, Hb:8.3g/dL, Staphylococcus aureus isolated in the swab culture but the bacterium was not isolated from the blood culture. He received antibiotic therapy, fresh frozen plasma, soframycin ointment. The skin lesions began to implement a remission process. The child was treated and the lesions were started to recure."Fig.3,4".

Investigations shows PLT:1.24lakhs,Hb:8.3g/dL, Staphylococcus aureus isolated in the swab culture, CRP: Negative, PT:13.9sec, APTT:35.2 sec. The routine urine test, the liver function tests, the renal function tests and the serum electrolytes were normal. Due to financial constraints and the case having no clinical manifestations, the workup for other hyperocoaguble states was not done. The child was treated and the lesions were started to recure"Fig.3,4".
DISCUSSION

PF has characteristic central areas of a blue-black hemorrhagic gangrenous necrosis which has a surrounding erythematous border. If there is a delay in treatment, the necrosis may extend to the muscle and bone contributing to a late mortality and morbidity. It heals with scarring and auto-amputation of the digits. Though the inherited and the acquired abnormalities of the protein C and the protein S anticoagulant pathways are responsible for a majority of the cases of Purpura Fulminans, the present case was post infectious disease which was caused after occurrence of varicella. The mortality rate infectious variety of PF and the idiopathic variety with secondary infection have decreased considerably, as a result of the treatment of the secondary infections, a better supportive care and the use of other therapies. The treatment given to the patient was IV fluids, antibiotics acting against gram positive bacteria to treat staphylococcus infection, soframycin ointment thrice daily, vancomycin injection 140 mg IV TID.

REFERENCES