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CASE REPORT

PURPURA FULMINANS IN INFANTS - CASE REPORT

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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. Disseminated intravascular coagulation (DIC), antiphospholipid antibodies and acquired or congenital C and S protein deficiency play a role in its pathogenesis. Here we report two cases of purpura fulminans in infants due to viral infections (dengue hemorrhagic fever and disseminated varicella) complicated by DIC.

Keywords:

Purpura fulminans,

Viral infections,

Infants.

INTRODUCTION

Purpura fulminans (PF) is a rare fatal condition that may occur during or after severe bacterial or viral infections. (Canpolat and Bakir, 2002) It was first described by Guelliot in a patient with chickenpox in 1884 as gangrenous skin lesions. (Alexander *et al.*, 2003) Later PF has been found to be associated with gram-positive and gram-negative bacteria(meningococcal) and viral infections. (Campanelli *et al.*, 2004; Gurgey *et al.*, 2005) Disseminated intravascular coagulation (DIC), antiphospholipid (APL) antibodies and antibodies to protein C and S is thought to play a role in its pathogenesis. (Gurgey *et al.*, 2005; Asherson and Cervera, 2003)

Case 1 (Figures 1 a, b, c, d, e)

A 7 months old infant presented with high grade fever (>103 °C) of 20 days duration, which was not responding to antipyretics. After 10th day of fever, asymptomatic, blanchable erythematous maculopapular rash developed on the palms, face and trunk spreading to other parts of the body. Within 2 to 3 days the rash progressed to large well defined non blanchable ecchymotic patches with angulated margins on the lower limbs, buttocks, soles and external ears. There was no history of upper respiratory and urinary tract infections. The hemoglobin level was 12.2gm/dl, WBC count was elevated (12,000/cmm), the platelet count was decreased (40,000/cmm), elevated hematocrit levels and the CRP was positive (12 mg/L). The dengue serology for IgM and IgG was positive. PCR test for dengue virus was not done due to unavailability. CUE, CXR and U/S abdomen were normal.

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A diagnosis of Purpura fulminans was made probably due to dengue hemorrhagic fever.



Fig. 1(a)



Fig. 1(b)



Fig. 1(c)



Fig. (d)



Fig. 1(e)

The infant was admitted in PICU and treated with antipyretics, IV fluids, electrolytes, fresh frozen plasma and IV antibiotics along with good nursing care. The infant recovered completely after three weeks without any sequele.

Case 2 (Figures 2 a and b)

A 9 months old infant presented with multiple,painful non blanching, ecchymotic papules and plaques ranging from 0.5 to 1 cm over the trunk and limbs with hemorrhagic gangrenous necrosis over the face of 10 days duration. Occasional vesicles

were present over the back. It started as umbilicated vesicular lesions on the face, trunk and limbs which has been treated with systemic steroids by a private practitioner. Baby was unconscious and not responding to painful stimuli and was admitted in PICU.



Fig. 2(a)



Fig. 2(b)

There was history of varicella infection in their family. Routine investigations like CBP, CUE, and CXR were within normal limits. CRP was positive (24 gms/L), HSV Ig M serology was negative.Tzanck test showed multinucleated giant cells.VZV serology, APLA, protein C and protein S was not done due to poor resources. A clinical diagnosis of Purpura fulminans was considered probably due to disseminated varicella. Baby was treated with ET intubation, nasogastric feeding, IV fluids, electrolytes, IV acyclovir 10 mg/kg/dose three times a day, IV antibiotics along with nursing care. In spite of supportive and symptomatic therapy the baby succumbed to complications.

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. PF is caused by infections or it may be idiopathic. The infectious variety is caused by severe gram negative bacterial or viral infections. The idiopathic variety is uncommon and it mostly involves the skin. The differential diagnosis includes thrombotic thrombocytopenic purpura, Henoch-Schönlein purpura and post-infectious thrombocytopenic purpura which differ by the degree of severity of the skin involvement.

The condition mostly affects the fingers and toes in children. (Gurgey *et al.*, 2005) In our cases there was also involvement of limbs, trunk and face in addition to fingers and toes. In our first case the diagnosis and treatment of dengue fever was delayed, as a result of which the baby developed dengue hemorrhagic fever as a complication. The second case was also misdiagnosed and treated with systemic steroids by a private practitioner which further progressed to purpura fulminans probably due to immunosuppressive steroids. PF reported in children with severe bacterial and viral infections such as varicella may be associated with underlying metabolic disorders. An acquired protein S deficiency is responsible for most cases of purpura fulminans occurring after varicella. The concomitant presence of antiphospholipid antibodies may also play a role. (Alexander *et al.*, 2003; Gurgey *et al.*, 2005) The transient occurrence of APL antibodies, auto antibodies to protein C or protein S has been described in both viral and bacterial infections and has been implicated in the pathogenesis of infection-associated thrombosis. (Gurgey *et al.*, 2005; Asherson and Cervera, 2003)

Conclusion

Though PF is uncommon, early diagnosis and effective treatment of viral infections will prevent complications like Purpura Fulminans which is a rapidly progressive disease leading to morbidity and mortality.

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