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فارسی
Skin Grafting for Necrotizing Fasciitis in a Child With Nephrotic Syndrome

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INTRODUCTION

Infections like peritonitis, cellulitis, or sepsis due to Streptococcus pneumoniae and Hemophilus influenzae are important causes of morbidity and mortality in children with nephrotic syndrome.1 Stretching and increased fragility of the skin predispose children with nephrotic syndrome to cellulitis. However, deeper infection like necrotising fascitis (NF) is a rare complication of nephrotic syndrome.1,2 Only a few cases of NF complicating pediatric nephrotic syndrome have been reported.3 Although NF is a rare life-threatening infection, prompt diagnosis and expeditious surgical and medical intervention serve to decrease mortality. We report a case with successful outcome with judicious intravenous antibiotics and skin grafting of the bilateral lower thighs.

CASE REPORT

A 7-year-old boy, a known case of frequently relapsing nephrotic syndrome (FRNS), was admitted to our hospital with anasarca for 3 months, oliguria for 7 days, and fever for 4 days. General physical examination revealed hypertension, anasarca, and erythematous rashes over bilateral lower limbs. Within 10 hours of admission, the erythematous rashes over bilateral lower limbs progressed to purplish discoloration. On the next 5 days, the bilateral lower limb cellulitis and discoloration of the limbs progressed to dark-blue to purple necrotic patches with clear-cut borders and involved subcutaneous tissue and muscles and a diagnosis of NF was made (Figure).

Laboratory investigations revealed lymphopenia, toxic granules, and thrombocytopenia with evidence of disseminated intravascular coagulation. Ascitic tap revealed a total count of 2500 to 3000 leukocytes per milliliter with predominant polymorphonuclear cells. Urine examination showed 8 to 10 leukocytes per high-power field. Escherichia coli grew in the culture from both peritoneal fluid and urine, which was sensitive to amikacin, netilmicin, piperacillin-tazobactum, and cotrimoxazole. Subsequent urine cultures yielded Candida. Swab culture sensitivity from the lower limb lesions revealed Pseudomonas aerogenosa, which was sensitive to piperacillin-tazobactum and ciprofloxacin.

The child required prolonged hospitalization,
Lesions of necrotizing fasciitis involving subcutaneous tissue and muscles.

Causative agent in our case was *Pseudomonas aeruginosa* as it was isolated from swab culture taken from lesions of NF. *Pseudomonas aeruginosa* is an organism capable of producing collagenase, which may be responsible for the rapid spread of the necrotizing process in our patient. Collagen gives tissues their structure and strength; consequently, any process that result in its degradation is likely to have protean manifestations. In our patient, nephrotic syndrome, subcutaneous edema, stretching of the dermis, supplemented with previous immunosuppression with steroids might be the underlying risk factors for the development of NF. We presume NF in our patient to be blood borne, although the blood culture is sterile which may be due to prior use of antibiotics.

Necrotizing fasciitis has a rapid and severe clinical course with a mortality rate of 70% to 80% despite aggressive treatment. This high mortality is attributed to disseminated intravascular coagulation and widespread thrombosis that prevents antibiotic penetration into infected tissue. Our patient was managed inpatient for 3 months with antimicrobials, antifungal, blood transfusion, together with intensive surgical debridement and superficial skin grafting. This prompt and intensive treatment resulted in satisfactory end result.

In conclusion, although NF is a rare life-
threatening complication of nephrotic syndrome, prompt diagnosis and expeditious initial wide excision and debridement along with appropriate antibiotic coverage serve to decrease mortality.

CONFLICT OF INTEREST
None declared.

REFERENCES


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