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

Narendra Bagri,[1] Abhijeet Saha,[1] Nandkishore K Dubey,[1] Ashish Rai,[2] Sameek Bhattacharya[2]

[1]Division of Pediatric Nephrology, Department of Pediatrics, Postgraduate Institute of Medical Education and Research and Associated Dr Ram Manohar Lohia Hospital, Baba Kharak Singh Marg, New Delhi, India
[2]Department of Burns and Plastic Surgery, Postgraduate Institute of Medical Education and Research and Associated Dr Ram Manohar Lohia Hospital, Baba Kharak Singh Marg, New Delhi, India

Necrotizing fasciitis is a rare complication of nephrotic syndrome in children, with a high mortality rate. We report a case with successful outcome with judicious intravenous antibiotics and skin grafting of the bilateral lower thighs.

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Case Report

## INTRODUCTION

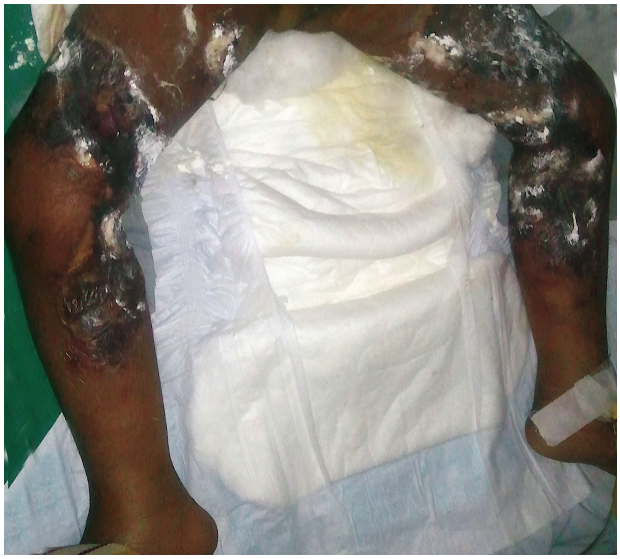
Infections like peritonitis, cellulitis, or sepsis due to *Streptococcus pneumoniae* and *Hemophilus influenzae* are important causes of morbidity and mortality in children with nephrotic syndrome.[1] Stretching and increased fragility of the skin predispose children with nephrotic syndrome to cellulitis. 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 erythmatous rashes over bilateral lower limbs. Within 10 hours of admission, the erythmatous 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, netilmycin, piperacillin-tazobactum, and cotrimoxazole. Subsequent urine cultures yielded *Candida*. Swab culture sensitivity from the lower limb lesions revealed *Pseudomonas aerigenosa*, which was sensitive to pipericillin-tazobactum and ciprofloxacin.

The child required prolonged hospitalization,



Lesions of necrotizing fasciitis involving subcutaneous tissue and muscles.

antibiotics, and antifungals according to culture and sensitivity reports. The patient underwent debridement of raw area over the bilateral lower limbs, and superficial skin grafting was done on the bilateral lower thighs under general anesthesia. Gradually, the patient's general condition improved and clinical signs of sepsis subsided and the child was started on steroids to achieve remission of FRNS. At 6 months follow-up, the child was in remission with good graft take.

## DISCUSSION

Hypogammaglobulinemia secondary to immunological dysregulation and urinary loss of factor B, I, and D leads to impairment of humoral defense system and alternative complement pathway in subjects of nephrotic syndrome.[4,5] Lymphocyte dysfunction due to spleenic hypofunction and loss of transferrin also contributes to impaired immunity in these children.[6] These factors, together with the use of corticosteroids and immunosuppressive treatments, increase the risk for infections in nephrotic patients. Cellulitis is common in children with anasarca and may get triggered by venipuncture, minor trauma, or spontaneous breach in continuity of edematous skin. Common inciting agents for cellulitis are beta-hemolytic *Treptococci, Hemophilus influenza*, and other gram-negative organisms.[7,8]

Necrotizing fasciitis is a rare invasive infection which involves necrosis of the fascia and subcutaneous tissue with relative sparing of the underlying muscle. Most patients with NF presents with signs of inflammation such as erythema, swelling, and pain at the affected site. With the progress of infection, the skin becomes increasingly tense and erythematous with indistinct margins and sequential color changes from a red-purple to a dusky blue before progressing to necrosis and formation of bullae and eventually becoming hemorrhagic. Our patient had classical evolution of signs and symptoms which helped us to make the diagnosis of NF. Most studies have shown that necrotizing fasciitis is polymicrobial in nature, with most cultures yielding a mixture of aerobic and anaerobic organisms.[9] Polymicrobial infection (type 1 NF) accounts for at least two third of all cases of NF. Infection with a single pathogen is reported in about 15% of cases, and overall streptococcus is the most common causative organism.[9] However, other aerobic and anaerobic pathogens, including *Bacteriodes*, *Clostridium*, *Proteus*, *Pseudomonas*, and *Klebsiella*, may be present.[3]

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.[10] 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.[11,12] 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.

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Correspondence to:
Abhijeet Saha, MBBS, MD
Division of Pediatric Nephrology, Room No 406; PGI Building;
PGIMER & Dr RML Hospital, New Delhi, India
E-mail: drabhijeetsaha@yahoo.com

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