

Case Report

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A case of varicella induced remission of nephrotic syndrome

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ABSTRACT

Here authors report a case of a 7-year old girl diagnosed with nephrotic syndrome with urinary tract co-infection who during treatment with steroids developed a Varicella Zoster infection. After cessation of steroid therapy and commencement with antiviral drugs, the patient showed significant reduction in nephrotic features and complete resolution of nephrotic syndrome as with the VZV super-infection.

Keywords: Nephrotic syndrome, Remission, UTI, Varicella zoster

INTRODUCTION

Varicella Zoster Virus (VZV) primary or secondary infections in an immunocompromised state such as AIDS, chronic steroid use, and nephrotic syndrome is well documented to present with numerous dreaded complications such as visceral dissemination, leading to pneumonia, hepatitis, encephalitis, and disseminated intravascular coagulopathy.^{1,2} Here, however authors present a case of a 7-year-old girl who presented with first episode nephrotic syndrome and complicated urinary tract infection whose nephrotic syndrome went into remission after VZV infection.

CASE REPORT

A 7-year-old girl presented to us with a history of periorbital oedema for 1 month and fever for 3 days, intermittent in nature, and not associated with chills/rigor/rash. The child did not have any history of recent illness or haematuria. The child's prenatal, natal, postnatal, and developmental history were unremarkable. The patient had been immunized for measles, mumps, rubella, diphtheria, tetanus, hepatitis B, HiB and polio with no prior vaccination for varicella zoster. The child

weighed in at 20 kg and height was 110 cm. Physical examination findings were significant for fever and anasarca. Abdominal examination revealed a distended and tense abdomen with no organomegaly or visible scars or dilated veins. Upper respiratory, lower respiratory, cardiovascular, and nervous system examinations were unremarkable. Laboratory investigations were significant for proteinuria 3+, hypoalbuminemia (0.86 g/dl), hyperlipidemia (LDL=304.9 mg/dl), and TLC count was 33.4/mm³. Protein creatinine ratio was found to be markedly elevated to 5.94 mg/g. Urine creatinine was 0.4 mg/dl and urea was 27.82 mg/dl. The patient was negative for hepatitis B virus surface antigen, hepatitis C virus antibodies, and human immunodeficiency virus antibodies. Chest radiography was normal. A diagnosis of nephrotic syndrome, first episode with co-infection was proposed. IV empirical antibiotic therapy with Inj Ceftriaxone 690 mg was started whilst urine and blood culture and sensitivity were sent. Urine culture was positive for Klebsiella growth with bacterial load of 10⁵ CFU/ml. IV Inj Ceftriaxone was continued for a total of 7 days and repeat cultures sent post antibiotic therapy were negative for bacterial growth however urine analysis was still significant for proteinuria 3+. Daily steroid therapy with Tab Prednisolone was thence commenced at

60mg/kg/m². The patient's abdominal girth, weight and urine protein measured by dipstick analysis however showed no significant improvement. On day 10 of steroid therapy, the patient developed a vesicular rash over her hand and patient was diagnosed with VZV infection. Steroid therapy was promptly stopped, and patient was started on Inj Acyclovir (20 mg/kg/dose) IV for 7 days followed by Tab Acyclovir for 5 days for a total of 12 days of antiviral treatment. The vesicular rash gradually subsided over the course of 7 days of antiviral treatment. During VZV infection we noticed a significant decline in patient's oedema. The patient's abdominal girth, which was recorded to be 58 cm prior varicella infection, was now recorded to be 46 cm and weight of the patient which was 20.02 kg prior to infection was now 14.4 kg. Urine analysis was negative for protein and urine protein creatinine ratio was now 0.82 mg/g. A complete remission of nephrotic features was observed. Subsequent out-patient visits at 1 month and 6 months were insignificant for recurrence of oedema and proteinuria.

DISCUSSION

In this unusual case of nephrotic syndrome with urinary tract co-infection complicated with VZV superinfection, we were able to successfully manage the urinary tract infection and prevent the dreaded complications associated with varicella zoster infection with prompt clinical diagnosis seconded by laboratory confirmations and treatment with timely administration of antibiotics, prednisolone and acyclovir. Nephrotic syndrome, a state characterized by hypoalbuminemia, accompanied with steroid therapy, leaves the patients at increased risk of bacterial and viral super infections. Primary infection with varicella-zoster virus and reactivation of VZV (shingles) are common in immunocompromised hosts and are associated with significant morbidity.² VZV infections have also been proved to cause relapses of steroid sensitive nephrotic syndrome.³ Literature involving VZV induced remission of nephrotic syndrome, however, is scarce. A similar case of varicella zoster induced remission of nephrotic syndrome was reported by Saeed MA et al.⁴

It is well proven that measles infection can induce the remission of nephrotic syndrome. In studies by Ching-Yuang Lin and Janeway et al.^{3,5} this phenomenon of remission was attributed to the increased production of anti-inflammatory steroid, and prolonged depression of cell-mediated immunity marked by a moderate fall in CD4 T-cells and a severe fall in CD8 T-cells during the spontaneous remission of nephrotic syndrome following acute measles.^{3,5,6} The prevalence of CD4 T-cells, primarily, TH2 cells in minimal change disease explains the increase in IL-4, IL-5, IL-9, IL-10, IL13. IL-13 has shown to induce effacement of foot processes and

proteinuria in experimental set ups.⁷ The role varicella zoster infection can play during its interaction with TH-2 cells and its produced cytokines especially IL-13 is not well defined. Despite timely management, treatment and successful outcome, our approach in this case was limited because we were unable to perform a kidney biopsy, CD4 and CD8 levels and immunoglobulin assays due to financial constraints. A further evaluation of cytokine analysis would have aided in determining the pathogenesis of remission of nephrotic syndrome in this case. Contrary to belief of varicella causing fatal outcomes in patients with nephrotic syndrome, authors present a case of varicella induced remission. Further immunological based testing including cytokine analysis, CD4, CD8 TH1 and TH2 cell interaction studies, are required to ascertain the pathogenesis of varicella induced remission of nephrotic syndrome.²

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