Case Report

Minimal Change Nephrotic Syndrome Presenting as Acute Renal Failure: An Unusual Seen Complication

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Abstract: Nephrotic syndrome is one of the commonly seen glomerular disease that affect pediatric age group. Majority of the patients with Minimal Change Nephrotic Syndrome have favourable outcomes and usually no complications are seen. Acute renal failure is a very rare and uncommon seen complication of minimal change nephrotic syndrome. We report a 9 year old boy who was admitted in our hospital with proteinuria and progressive azotemia. Haemodialysis was done to improve azotemia. Renal biopsy revealed minimal change disease with acute tubular necrosis. While on haemodialysis, steroids were started, which later allowed the patient to be weaned from haemodialysis and releaved him of 3 days duration. There was no history of vascular occlusion.

Keywords: Acute renal failure, acute tubular injury, Idiopathic nephrotic syndrome.

INTRODUCTION

Nephrotic syndrome is one of the the commonly seen glomerular disease that affect pediatric age group and also seen in OPD. Renal histopathology usually reveals minimal change nephrotic syndrome in more than 80% of these patients. Majority of patients with Minimal Change Nephrotic Syndrome have favorable outcomes without any serious complications. Renal failure is usually common in patients with nephritic syndrome and other glomerular disease but is very rare in in children with nephrotic syndrome, especially in minimal change type of nephrotic syndrome [1]. Smith and Hayslett have reported reversible acute renal failure in elderly patients with minimal change disease [2] but it is very rare entity in childhood [1]. Acute renal failure in patients of nephrotic syndrome is usually idiopathic or secondary to various causes like sepsis, interstitial nephritis, renal vein thrombosis [1].

We report a case of child with minimal change nephrotic syndrome who presented with oliguric acute renal failure caused by acute tubular injury requiring dialysis and it was found that childhood idiopathic nephrotic syndrome presented as reversible acute renal failure in our case. Our search of the published literature till date revealed only very few case reports with similar findings [3-5]. This case highlights the causative factors, histopathological findings and the outcome of acute renal failure with minimal change nephrotic syndrome.

CASE REPORT

A nine-year-old boy presented with oliguria and anasarca of 3 days duration. There was no history suggestive of haematuria, dysuria, sore throat, skin rash, fever, joint pain, flank pain, hypotension, and hypertension or nephrototoxic drug intake. Examination revealed anasarca and mild dehydration with blood pressure of 128/74 mm Hg. Other vitals were stable. There was no fever, pallor, icterus, lymphadenopathy, rash or signs of vascular thrombosis.

Investigations showed blood urea level of 134 mg/dL, creatinine 6.4 mg/dL, which increased further to 152 mg/dL and 8.8 mg/d respectively after one week. He also had metabolic acidosis (pH 7.18), bicarbonate 13 Eq/L. Spot urine examination showed 3+ proteinuria and 24-hr urine protein was 2.9 g. Serum total protein was 4.9 g/dL, albumin 2.0 g/dL, total cholesterol 296 mg/dL and triglyceride 438 mg/dL. Peripheral smear examination was suggestive of leucocytosis (32000/ microliter with neutrophilia 84%). The serum electrolytes and ionized calcium was within normal limits. Serum complement levels (C3, C4) were within normal limits. Serological examination for hepatitis B, C and HIV, antinuclear antibody (ANA) and anti-neutrophil cytoplasmic antibody (ANCA) was negative. Ultrasound of the abdomen showed normal sized kidneys with normal echogenicity without any evidence of urinary tract obstruction. Colour Doppler ultrasound scanning of the renal vessels did not show any evidence of vascular occlusion.
The patient was hospitalized and managed with adequate hydration. In view of anuria and azotemia; he was taken up for dialysis (peritoneal dialysis initially followed by hemodialysis). Even after two weeks of alternate day dialysis and supportive therapy, patient continued to be oliguric and dialysis dependent. A percutaneous kidney biopsy was performed to determine the cause of acute renal failure.

Light microscopy of kidney biopsy showed twenty four glomeruli, all of which were morphologically unremarkable. Tubulointerstitial compartment show patchy dilatation of tubules with presence of PAS positive casts (Fig. 1). These tubules with cast have flattened lining. The vascular compartment was unremarkable. Immunoflorescence was negative for IgG, IgM, IgA, C3 and C1q. Biopsy features were thus suggestive of minimal change disease with acute tubular injury.

In view of nephrotic range proteinuria, patient was treated with prednisolone according to the extended APN protocol [6]. The urine output gradually improved and renal failure recovered in four weeks. The patient achieved remission from proteinuria within four weeks of prednisolone therapy. He continued to be in remission at six months of follow up.

Fig. 1: Light microscopy of kidney biopsy showed twenty four glomeruli, all of which were morphologically unremarkable. Tubulointerstitial compartment show patchy dilatation of tubules with presence of PAS positive casts. These tubules with cast have flattened lining. The vascular compartment was unremarkable.

DISCUSSION

Nephrotic syndrome is a common renal disorder, characterized by alterations of permeability of the glomerular capillary wall, resulting in its inability to restrict the urinary loss of protein. It is characterised by proteinuria, hypoalbuminemia, edema and hyperlipidemia. The most common variety of nephrotic syndrome is minimal change disease which is seen in approximately in 80% cases and is characterized by normal renal histology on light microscopy [7].

The annual incidence of nephrotic syndrome varies from 2-7 per 100,000 children, and prevalence from 12-16 per 100,000 depending on the population and geographic area [4]. There is greater prevalence of nephrotic syndrome in children of south Asian region [8]. The condition is primary (idiopathic) in 95 per cent cases. Acute renal failure is an uncommon and rare complication of Idiopathic nephrotic syndrome in children. It can complicate with nephrotic syndrome following administration of NSAID’s, foscarnet, and interferon alfa therapy. Whereas it can also complicate preexisting nephrotic syndrome because of many causes including speedy deterioration of glomerulonephritis, hypotension, acute tubular necrosis, interstitial nephritis, renal vein thrombosis, intratubular obstruction by proteinous cast or interstitial edema [1].

The age at initial presentation is useful tool in assessing the underlying aetiology. The usual age of first attack of minimal change nephrotic syndrome is between 2-6 yr; however it may be seen in 30 per cent of the adolescents.

Differential diagnosis which were considered
- Acute Poststreptococcal Glomerulonephritis
- Acute Renal Failure
- Angioedema
- Crescentic Glomerulonephritis
- Finnish-type congenital nephrotic syndrome
- Focal Segmental Glomerulosclerosis
- Henoch-Schönlein Purpura
- HIV-Associated Nephropathy
- IgA Nephropathy
- Lupus Nephritis
- Malaria
- Membranoproliferative Glomerulonephritis
- Membranous Glomerulonephritis
- Malignant hypertension
- Membranous nephropathy
- Minimal change nephrotic syndrome
- Malignant hypertension
- Membranous nephropathy
- Malignant hypertension

Sakarcan et al. described in his case report four paediatric patients with reversible ARF due to mild acute tubular necrosis and all the four patients required dialysis, but had complete recovery of renal function with treatment [4]. Nagamani et al. reported 3 cases of acute renal failure in children with minimal change nephrotic syndrome, out of them one was post allergic interstitial nephritis while the other two were idiopathic [3]. Steele et al. reported 2 patients of acute renal failure of which one presented at the onset, while other patient developed acute renal failure at the time of relapse of the nephrotic syndrome. Both patients had complete recovery following dialysis and steroid therapy as seen in our case also [5]. Tawares et al. published a study of 149 patients with acute renal failure out of which 45% had either minimal change disease or focal glomerulosclerosis [9].

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In our patient, we excluded pre-renal azotemia as a contributing factor for acute renal failure by giving adequate hydration. The cause of renal failure was determined by kidney biopsy which showed minimal change disease with evidence of acute tubular injury, without evidence of interstitial nephritis, interstitial edema. The secondary causes of acute renal failure were ruled out by doing specific investigations.

Childhood idiopathic nephrotic syndrome can present as reversible acute renal failure, though very rare. The cause of renal failure may be difficult to determine and may require thorough investigations. These patients can be managed successfully with proper dialysis and supportive measures. Response to steroid therapy is satisfactory in such cases.

CONCLUSION

Nephrotic syndrome should be differentiated from other causes of renal failure as the treatment is very different and any misdiagnosis can lead to devastating effect on the patient. Biopsy should be done in condition when we are not able to diagnose the cause of acute renal failure. Acute renal failure secondary to minimal lesion nephrotic syndrome rarely leads to dialysis is an unexplained and rare complication in adults and children.

REFERENCES