Severe Anemia with Renal Failure as A Consequence of Homeopathic Medication for Nephrotic Syndrome: Case Management with Positive Outcome

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ABSTRACT

Nephrotic Syndrome is a glomerular disorder consisting of a combination of features such as marked proteinuria (more than 3 g of protein per day), hypoalbuminemia (less than 25 g/L), peripheral edema and at times hyperlipidemia. Nephrotic syndrome can lead to a number of complications either due to the disease process itself or as a result of drug treatment. Infections, thromboembolism, hypovolemic shock, renal compromise and anemia constitute the disease related complications of nephrotic syndrome. Here, we report a typical presentation of nephrotic syndrome where the patient had classic clinical and laboratory findings that supported the diagnosis of nephrotic syndrome which was complicated by the use of homeopathic medications leading to life threatening anemia and renal failure, which was successfully managed and patient shoed dramatic improvement with no ling term sequelae of disease or complications.

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INTRODUCTION

Nephrotic Syndrome is a glomerular disorder with marked proteinuria (more than 3 g of protein per day), hypoalbuminemia (less than 25 g/L), peripheral edema and hyperlipidemia.¹ The diagnosis is made based on clinical presentation, blood tests, renal imaging studies and renal biopsy. Worldwide, the incidence of Nephrotic syndrome has been reported to be 1.15 – 16.9 per 100,000 children.² A higher incidence of idiopathic nephrotic syndrome has been reported in Asian population.3 In adults, the incidence of nephrotic syndrome has been reported to be 3 new cases per 100,000 annually.4 Corticosteroids are the main stay of treatment for nephrotic syndrome. Studies have shown that in United States the incidence of steroid responsive nephrotic syndrome is found to be 2 - 2.7 cases/100,000 children with a cumulative prevalence of 16 cases/100,000 children.⁵ The occurrence of the condition varies by age group and gender. Research has revealed that nephrotic syndrome's male to female ratio in young children is 2:1 while in adolescents the male to female ratio is estimated to be 1:15.

Nephrotic syndrome can lead to a number of complications either due to the disease process itself or as a result of drug treatment. Infections, thromboembolism, hypovolemic shock, renal

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compromise and anemia constitute the disease related complications of nephrotic syndrome. Moreover, drugs used in nephrotic syndrome treatment can lead to numerous short and long term complications. Examples of such medications include corticosteroids, cyclophosphamide, cyclosporine and tacrolimus.⁶

CLINICAL CASE

A 55 years old female presented to the Nephrology Outpatient department (OPD) with significant proteinuria and peripheral edema in January 2021. Investigations revealed proteinuria of 6.6 g/day with hypoalbuminemia. Renal function tests showed a borderline creatinine level of 1.2 mg/dL. Her signs and symptoms were consistent with a diagnosis of nephrotic syndrome. Autoimmune screening and a renal biopsy was advised but patient refused. She was commenced on corticosteroid therapy (prednisolone @ 1mg/kg/day) but got lost to follow up. On 27th March 2021, patient presented to emergency department in a critical condition. On examination she had a systolic blood pressure of 60 mmHg. Anuria persisted for the next 24 hours. She gave a history of hematemesis and melena. She had been taking corticosteroids and homeopathic treatment for nephrotic syndrome since her last visit. Patient was started on intravenous fluids for resuscitation. A blood complete picture showed a dramatically low hemoglobin of 1.9 g/dl and an elevated creatinine level of 9.2 mg/dL. Hence, she was transfused 01 pint of whole fresh blood in the emergency department. A temporary dialysis catheter was passed and hemodialysis with a transfusion of 2 units of red cell concentrate (RCC) was done. For severe hypoalbuminemia and edema the patient was given IV salt poor albumin 5% plus IV diuretics once pressure blood stabilized. After stabilization, oesophagogastroduodenoscopy an (OGD) was done. It revealed the presence of diffuse gastritis for which she was commenced on intravenous proton pump inhibitor (PPI). Later, she was switched to oral proton pump inhibitor. A computed tomography (CT) scan showed the presence of a pseudo pancreatic cyst for which she was advised conservative therapy by gastroenterologist, repeat scan after 3 months showed complete resolution of the cyst. Patient underwent 08 hemodialysis sessions and 06 blood transfusions. On day 13 the patient was stable with a good urine output. Her serum creatinine showed a downward trend, temp dialysis catheter was removed and patient discharged to home.

She continued to follow up as an outpatient. Her serum creatinine during her follow up visits ranged between 2- 2.5 mg/dL. Urine analysis depicted a sub nephrotic range proteinuria. On 14th July 2022 renal biopsy was done. The biopsy was delayed as the patient was reluctant, however she agreed to get it done after adequate counselling and discussing the risks versus benefit ratio with the patient. At the time of biopsy, she had a proteinuria of 1.79 g/day and serum creatinine of 2.1 mg/dL. Renal biopsy result was suggestive of a diagnosis of Diffuse Proliferative Glomerulonephritis (DPGN) with 11/25 sclerosed glomeruli and 3/25 partially sclerosed glomeruli, no crescents, mild interstitial fibrosis and mild tubular atrophy. Sample of Immunofluorescence (IF) was unfortunately mishandled and due to delayed processing by the lab the sample was unfit for IF. As patient had stable renal functions over 4 months and sub-nephrotic proteinuria, she was started on antiproteinuric therapy only (candesartan @ 16mg/day) with plan for monitoring and addition of immunosuppressive therapy in case of disease progression.

In July 2022, more than a year after her initial presentation, she has a serum creatinine level of 0.9 mg/dL and urinary protein of 222 mg/day. Her hemoglobin has remained above 11 g/dl. She has now been off dialysis for 15 months and her most recent hemoglobin is 12 g/dl. The patient had a miraculous

recovery with a return of her renal function to the normal range.

DISCUSSION

Nephrotic syndrome has numerous causes that are classified as primary and secondary. The primary causes include minimal change nephropathy, focal glomerulosclerosis, segmental membranous nephropathy and inherited causes. Secondary contributors are infections e.g. Hepatitis B, hepatitis C and HIV, immunological causes such as lupus erythematosus. Amyloidosis and diabetes mellitus are also among the known causes of nephrotic syndrome.7 The pathogenesis of nephrotic syndrome may involve either a defect of the glomerular basement membrane, presence of circulating factors or abnormalities in the immune system.8

The key feature of nephrotic syndrome, as in this case, is massive proteinuria which is associated with the frothy appearance of urine. The prominent proteinuria consequently results in low circulating serum albumin levels. This hypoalbuminemia in turn contributes to low plasma oncotic pressure which ultimately leads to the development of peripheral edema. Similarly, the low plasma oncotic pressure stimulates hepatic lipoprotein synthesis that often leads to hyperlipidemia which appears in the form of fatty casts in urine.⁹

In this case, we were successfully able to manage the signs and symptoms of nephrotic syndrome such as peripheral edema by administration of intravenous albumin and starting the patient on intravenous furosemide. After renal biopsy, she was commenced on an angiotensin receptor blocker (ARB) i.e. candesartan. The patient was received in a hemodynamically unstable form with hypovolemic shock secondary to gastrointestinal bleeding as a consequence of possible use of homeopathic treatment.¹⁰ The ingredients used in the homeopathic remedies are unknown as they were taken by the patient in a packaging that came without any description of the ingredients. The gastrointestinal bleeding led to a sudden drop in the hemoglobin of the patient which was revived by repeated blood transfusions. The severe hypovolemia and bleeding led to acute renal failure. Hence, she was initiated on hemodialysis. This suggests that a comprehensive management of renal failure accompanied by hypovolemic shock secondary to the use of homeopathic medications might lead to the reversal of the decline in renal function. DPGN is

histopathological form of renal injury and cause is usually identified on IF. The etiology includes autoimmune diseases (systemic lupus erythematosus, rheumatoid arthritis), vasculitides (anti-GBM, ANCA associated vasculitis, IgA vasculitis), infection associated (endocarditis, viral hepatisis, schistosomiasis). The treatment is usually directed at the underlying disorder in the absence of crescents.¹²

Despite a successful management in this case, it is important to highlight the role and possible complications of homeopathic remedies to all patients. Homeopathic medications are known to cause platelet aggregation dysfunction, diarrhea and ulceration alongwith many other adverse effects.11 It is emphasized that we should investigate the ingredients of such homeopathic medications and educate the patients about their risks/benefits that have been proven in established researches and literature. Moreover, our management approach in this case was slightly limited due to the delay in performing a renal biopsy as the patient was initially reluctant to give consent for the procedure and further compounded by the loss of IF sample. The invasive nature of the diagnostic modality used in nephrotic syndrome is one of the major challenges in treating these patients. Another challenge is the lack of availability and high cost of the medications involved in its treatment. This may prompt patients to try to treat themselves using homeopathic remedies.

CONCLUSION

We have presented a case of worsening nephrotic syndrome (DPGN – proven on renal biopsy) secondary to gastrointestinal bleeding possibly resulting from the use of homeopathic medications. The patient was successfully managed with return of serum creatinine to near normal range after undergoing multiple blood transfusions and hemodialysis sessions. Further studies should be conducted to establish the risk of adverse effects of using homeopathic medications in patients with nephrotic syndrome.

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Authors' Contribution

Following authors have made substantial contributions to the manuscript as under:

W & KA: Data acquisition, data analysis, critical review, approval of the final version to be published.

ARQ & SAA: Study design, data interpretation, drafting the manuscript, critical review, approval of the final version to be published.

Authors agree to be accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved.

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