Pericardial Tamponade in Steroid Resistant Nephrotic Syndrome  
– A Rare Complication

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ABSTRACT

A 5 year old female child with steroid resistant nephrotic syndrome (SRNS) developed pericardial tamponade which is a rare complication during the course of illness. She had immediate pericardiocentesis treatment. A very small number of cases of pericardial tamponade in children with nephrotic syndrome have been documented in the literature. To prevent fatalities in children with nephrotic syndrome, it is important to recognise this uncommon but dangerous and life-threatening condition.

Keywords: Pericardial tamponade, Steroid resistant Nephrotic syndrome, pericardiocentesis,

I. INTRODUCTION

Nephrotic syndrome is a common childhood disorder characterised by Oedema with protein excretion >40 mg/m² per hour or urine protein:creatinine ratio ≥2000 mg/g (≥200 mg/mmol) or ≥3-4+ proteinuria on dipstick with serum albumin <2.5 gm/dl and hypercholesterolemia. Idiopathic NS can be classified on the basis of response to steroid therapy, pattern of relapse, histopathology, or by genetic mutations. Most simply, NS is categorised on the basis of clinical response to steroid therapy, as steroid sensitive (SS) or steroid resistant (SR). The majority of children respond well to steroids within 4 weeks (steroid-sensitive NS [SSNS]); however, most will relapse, with approximately half becoming frequent relapse or steroid dependent. In contrast, SRNS is associated with increased risk of progression to end-stage renal disease (ESRD) and also are more prone to develop complications of nephrotic syndrome.

Owing to the heterogeneity of SRNS, only 50% are at risk of progressing to ESRD in 5 years; typically those children who do not achieve complete or partial remission on multiple immunosuppressants. Increased urinary losses of protein and protein-bound molecules contribute to several complications in patients with nephrotic syndrome. We report a case of steroid resistant nephrotic syndrome child initially presenting with spontaneous bacterial peritonitis. She developed series of complication during her hospital stay including cardiac tamponade, which was particularly difficult to diagnose owing to the signs being masked by underlying condition.

II. CASE REPORT

We are hereby reporting a case of 5 year old female child who is a known case of SRNS, minimal change disease on biopsy done 3 years back and no pathological gene involvement detected on whole exome sequencing. Her proteinuria was not responsive to cyclophosphamide, cyclosporine and she had received 3 courses of Rituximab to which she maintained partial remission. Her last Rituximab was received 6 months back since then she was on oral prednisolone and supportive medication. She was brought with increase in oedema since 14 days.

On admission, she had anasarca with reduced urine output and mild respiratory distress for which various diuretics were started (IV frusemide, metolazone, Aldactone, torsemide), with dose adjustment was done with potassium correction with thorough monitoring. She also received two transfusion of albumin with frusemide. She also had concomitant SBP during the course, she was started on injectable antibiotics (ceftriaxone) which was stepped up to inj meropenem and ceftazidime. Her urine output was low and she developed AKI with rising creatinine max to 1.1 (eGFR - 57). She was started on inj methylprednisolone. She also developed right upper limb vasculopathy for which inj clexane and oral pentoxyphylline added. On day 22 of admission, she suddenly developed tachypnoea which was increasing on lying down with tachycardia. She developed cardiac tamponade detected on 2DECHO for which pericardiocentesis was done.

The fluid suggestive of transudate so albumin transfusion given. During this, femoral central line was inserted following which she developed right leg Deep Vein Thrombosis diagnosed by ultrasonography doppler study, aspirin was added. Later she developed right leg cellulitis for which ampicillin plus cloxacillin started and it got resolved. After draining of cardiac tamponade there was significant improvement in cardiac output and oedema reduced and there was significant weight loss, she was discharged on oral prednisolone, diuretics, antihypertensives, Antithrombotic and supportive therapy on biweekly follow up. Her next cycle of Rituximab has been planned.

III. DISCUSSION

Fluid retention, venous thromboembolism, infections, and hyperlipidaemia are the most common complications in patients with NS specially SRNS. Fluid retention may lead to peripheral oedema, ascites, and pleural effusion. This child’s oedema complicated into SBP, which caused AKI complicating oedema management. Even with resolution of AKI, the underlying pericardial effusion worsened to tamponade. The resulting in decrease in cardiac output further worsened the glomerular filtration causing water retention and vicious cycle. The early signs of cardiac tamponade of raised jugular venous pressure and tender hepatomegaly were difficult to diagnose due to severe oedema. In a child with difficult nephrotic syndrome, there should be a high suspicion of significant pericardial effusion which needs to be drained if it causes tamponade. Large pericardial effusion and pericardial tamponade is very rare in childhood idiopathic NS, with only a few documented cases in the literature. Cardiac tamponade is a life-threatening condition that results from rapid accumulation of pericardial fluid, thus impairing cardiac filling.

IV. CONCLUSION

With this report we would like to remind clinicians that pericardial tamponade is an uncommon but serious complication of NS. When a child with nephrotic syndrome exhibits chest pain, tachycardia, tachypnoea, dyspnoea, and cardiomegaly, the doctor should perform a thorough cardiac assessment. When the clinical findings suggest cardiac tamponade, pericardiocentesis is a life-saving operation. (5)
REFERENCES


