Abstract Form

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Project Title:	Adult onset of minimal change glomerulopathy presenting as nephrotic syndrome

Research Category (please check one):

☐	**Original Research**	☒	**Clinical Vignette**	☐	**Quality Improvement**	☐	**Medical Education Innovation**

Introduction:
Minimal change disease accounts for 90% of nephrotic syndrome cases in children and 10% in adults. In those small percentage of adults, the age of incidence is largely 65 years old or older. This case describes a 31-year-old patient who presented with a constellation of nephrotic syndrome symptoms diagnosed with minimal change disease.

Case Report:
31 year old female with past medical history of right lower extremity deep vein thrombosis diagnosed 1 month prior on apixaban, asthma, bronchitis, and hypothyroidism was transferred from outside hospital, with initial chief complaint of shortness of breath and worsening swelling. She initially presented to outside hospital emergency room 1 month prior with pitting edema up to the thighs and was prescribed furosemide. She took furosemide for 5 days but felt that her swelling failed to subside while she became dehydrated. Upon presentation to our emergency room, bilateral lower extremity edema had progressively worsened, extending to abdomen, chest, and upper extremities. Initial vitals were largely unremarkable with temperature of 36.7, heart rate 88, respiratory rate 20, blood pressure 128/80 and sPO2 of 95% on room air. Labs also were largely unremarkable, including BUN 7, Creatinine 1.09 and eGFR 70. However total protein and albumin were low at 5.2 and 1.4, respectively. Lipid panel revealed dyslipidemia with elevated cholesterol 366, triglycerides 287, HDL 55, and LDL 268. Urinalysis showed 3+ blood, 4+ protein, 16-25 WBCs, >50 RBCs. Physical exam was remarkable for grade 4 abdominal wall pitting edema and bilateral lower extremity pitting edema. Chest radiograph revealed no enlarged cardiac silhouette, no pleural effusions, and no signs of pulmonary vascular congestion. Retroperitoneal ultrasound revealed normal sized bilateral kidneys with no evidence of hydronephrosis. Visualized portion of bilateral renal veins were patent. Bilateral lower extremity duplex showed no evidence of thromboembolism in bilateral common femoral, femoral, popliteal, and calf veins. Patient was switched from apixaban to unfractionated heparin in anticipation of renal biopsy and initiated on loop diuretic furosemide in conjunction with albumin for diuresis. 24-hour urine protein quantified was 34 g, well exceeding the cut-off of 3.5 g for definition of nephrotic range proteinuria. Serological workup for etiologies of nephrotic syndrome were sent including antinuclear antibodies, complements (C3/C4), serum protein electrophoresis and immunofixation, serum free light chains, syphilis serology, hepatitis B and C serologies, but failed to suggest a clear etiology. After washout period of apixaban, patient underwent the gold standard procedure to determine the cause of proteinuria – renal biopsy. Pathology results were significant for extensive podocyte foot process effacement, consistent with minimal change disease, mildly thickened glomerular capillary basement membranes, mild acute tubular injury, and mild arteriosclerosis. Subsequently patient was started on immunosuppression therapy with glucocorticoids – prednisone 80 mg daily and discharged. Upon post-hospitalization follow up, patient had complete remission with resolution of proteinuria by end of week 3 of glucocorticoid therapy.

Discussion:
Minimal change disease is classically thought of as common cause of nephrotic syndrome in children. However it is important to keep in mind as one of many etiologies of nephrotic syndrome in adults as minimal change has favorable outcome with treatment course of corticosteroids.