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A rare association: Acute postinfectious glomerulonephritis and thrombotic microangiopathy: About two cases

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Acute post infectious glomerulonephritis (APIGN) is defined as acute nephritic illness preceded by pharyngitis or pyoderma. It's morphologically characterized by intense endocapillary proliferation and massive accumulation of inflammatory cells. Less commonly, it appears as focal or diffuse crescentic GN. Atypical features like thrombotic microangiopathy (TMA) are rarely described in association with APIGN. We report simultaneous occurrence of postinfectious glomerulonephritis and TMA in renal biopsy specimens from tow patients.

The first case is a 6-year-old boy who presented acute nephritic syndrome occurred one week after having pharyngeal pain and diarrhea. He presented with edema and hypertension on admission. Laboratory evaluation showed hemolytic anemia with fragmentation, thrombocytopenia, elevated lactic dehydrogenase level, low complement C3 level, and elevated antistreptolysin-O titer. Serum creatinine level was 417 micromol/L, and urinalysis showed marked proteinuria, with protein of 1.25 g/d, and hematuria. Kidney biopsy confirmed postinfectious glomerulonephritis with diffuse endo and extra-capillary proliferation, humps with Complement C3 deposition along the capillary, and simultaneous subendothelial hyalin deposits and fibrinoid arteriolar and glomerular thrombi. The patient received

steroid and antihypertensive drugs. Renal function improved, hypertension was controlled and serum levels of C3 and C4 complement components returned to normal. TMA was also resolved as evidenced by normalization of serum LDH level, hemoglobin and platelet count.

The second patient is a 65-year-old man was admitted with acute renal failure, microscopic hematuria, edematous nephrotic syndrome and hypertension. This acute nephritic syndrome occurred two weeks after persistent infectious pneumonitis. Hemoglobin and platelet count were normal, serum creatinine level was 733 micromol/L and dialysis was indicated for hyperkalemia. The renal biopsy specimen was characteristic of APSGN with diffuse hypercellularity and humps, one segmental crescent and C3 deposition in immunofluorescence study. In addition there were glomerular microthrombi in one glomerulus. Patient was treated with intravenous antibiotics and intravenous diuretics. Renal function improved and serum creatinine level was 122µmol/l after one month.

Common factor between APIGN and TMA described so far is neuraminidase produced by streptococci. Neuraminidase injures vascular endothelium, initiating the cascade of thrombus formation.

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