Splenic Infarction as Starting Symptom in Minimal Change Nephropathy

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To the Editor: A 17-year-old Chinese male patient without any medical history presented to The First Affiliated Hospital of Nanchang University. His initial symptoms were abdominal pain and progressive edema. The pain presented initially around umbilical area and then progressed to left upper quadrant of the abdomen. His physical examination revealed that he had mild edema of the face and legs, direct tenderness of the left upper abdomen, without rebound tenderness. His blood pressure level was 110/75 mmHg. His heart and lungs were normal. Except spleen, no organs or lymph node enlargement was found. His laboratory investigations were as follows: urinary protein, 3+; hematuria, −; 24-h urinary protein, 4.8 g/day; liver and renal function were normal; albumin, 14.9 g/L; total cholesterol, 6.21 mmol/L; triglycerides, 3.77 mmol/L; hemoglobin, 154 g/L; white blood cell count, 7.8×10^9/L; red blood cell count, 5.15×10^{12}/L; platelets, 565×10^9/L; fibrinogen, 6.2 g/L, antinuclear antibody-negative; anti-double-stranded DNA antibody-negative; thyroid function was normal; hepatitis B surface antigen-negative; and anti-hepatitis C virus-negative. The abdominal computed tomography scan disclosed that spleen was enlargement, uneven density. Abundant hypodensity shadow was found in spleen [Figure 1], which indicated splenic infarction. The infarction area was over 80% of the whole spleen. After 1 week of anticoagulation therapy, he was discharged with complete resolution of abdominal pain. At that time, a percutaneous renal biopsy was performed for the patient. The glomeruli showed an almost normal appearance under light microscopy. The mesangium was mild proliferated. The basement membranes were extensive vacuolar degeneration. Renal tubular epithelial cells were vacuolar and granular degeneration. Renal interstitium and vessels were unremarkable. Immunofluorescence microscopy revealed an absence of deposits of immunoglobulin, complements, and fibrinogen. By electron microscopy, we found that foot processes of podocyte were diffuse fusion, and no electron deposits were observed. With a diagnosis of minimal change disease, an "adequacy" dose steroid regimen (prednisolone 60 mg/d) was administered orally. After the treatment of prednisone, the patient's edema remitted slowly. By the 17th day of therapy, his serum albumin increased to 40.5 g/L, proteinuria turned to negative. The dosage of oral prednisone was tapered gradually over the next months. After continuous treatment, his nephritic syndrome resolved completely and no signs of relapse were noted during 18 months of follow-up period.

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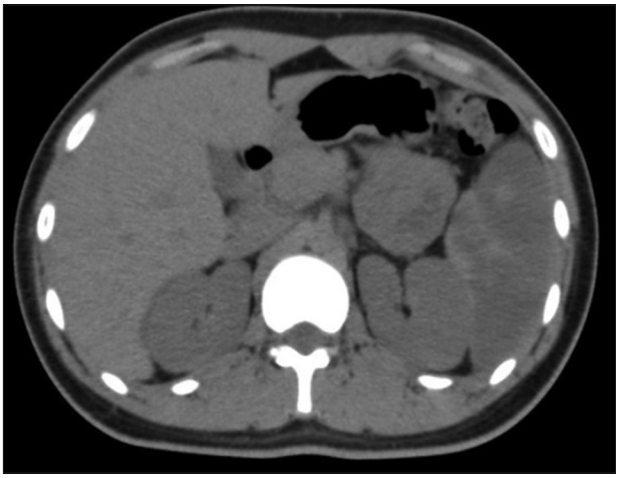

Figure 1: Abdominal computed tomography showed that spleen was enlargement, uneven density. Abundant hypodensity shadow was found. The infarction area was over 80% of whole spleen.

The hypercoagulable state is supported by several factors in nephrotic syndrome. The main factor is the coagulation factor leak, notably because of the antithrombin III.[1-3] Arterial thrombosis and their clinical complications are less common than venous. Mesangial proliferative glomerulonephritis combined with splenic infarction has been reported.[4] As splenic artery is a terminal artery without communicating branch, splenic infarction has a tendency to spontaneous healing. In addition to few cases need to apply the anticoagulation and thrombolysis drugs, most patients do not need special treatment. Our experience is that nephrotic syndrome patients should be paid more attention to thromboembolic complications, regardless of age, pathological type, medical history, and medication management.

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