quite severe in adults, and the fact that more aggressive treatment strategies were recently questioned by the Pillebout group.

Conflicts of interest

The authors declare that they have no conflicts of interest related to the contents of this article.

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Development of Focal Segmental Glomerulosclerosis in a Patient with Polycythemia Vera: can Polycythemia Vera be a cause of Focal Segmental Glomerulosclerosis?

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Dear Editor,

Polycythemia vera (PV) is a myeloproliferative disorder of unknown etiology. This condition is characterized by the abnormal proliferation of erythroid and myeloid series cells in the bone marrow.[1] Focal segmental glomerulosclerosis (FSGS) is a glomerular disease characterized by the presence of nephrotic syndrome, hypertension, and the progressive deterioration of the renal function. Etiology is usually unknown, but it may be seen in secondary conditions.[2-3] PV in association with FSGS is rare.[4] As far as we know, only eight cases have been reported in the literature.[4-10] In the report, we have presented a patient who development of FSGS associated with PV.

A 46-year-old male patient diagnosed with PV six years earlier was referred to the nephrology clinic due to the detection of proteinuria on routine controls. No important features were found on his history except for his use of the azathioprine for a month. Through a 24-hour urine analysis, 4g/day proteinuria was detected in the patient. The patient was admitted to the clinic. In physical examination is normal without arterial blood pressure of 140/90 and the spleen was 5cm palpable. Renal size and parenchyma were normal in abdominal ultrasonography. Laboratory tests results and examinations of glomerular disease have been showed at Table 1. Hence, causes of nephrotic syndrome were excluded. Renal biopsy was performed. In light microscope were shown 29 glomeruli. Global sclerosis and hyalinization were shown in five glomerule. There was intensive segmental sclerosis in more small segments of the other two to three glomeruli. The also remaining some glomeruli have mesengial cell proliferation and expansion. In Bowmans capsule of one to two glomeruli presence of synechiae noted. Tubulointerstitial area has been examined, focal interstitial mononuclear cell infiltration has been observed. In particular areas of inflammation have attenuation of some tubules epithelium and in the presence of eosinophilic material in the lumen characterized by atrophic changes were

observed. In vascular stuctures were normal except for a slight thickening of the wall. Glomerulosclerosis, segmental sclerotic areas and slight thickening of the glomerular basement membrane have been detected through the use of Trichome stain. Furthermore, amiloid staining and immunofluorescence study showed a negative. All above these findings were indicative of FSGS. In arterial blood pressure monitoring, stage 1 hypertension was determined. Perindopril, azathioprine, and ASA were prescribed and the patient was discharged.

FSGS is a clinical and pathological disorder involving primarily the glomerulus.[2-3] Progressive glomerular scarring is the most important feature in this disease. Early in the disease process, glomerulosclerosis is both focal, and segmental in nature. Furhermore, in later stages of the disease diffuse and global glomerulosclerosis develops. The loss of filtration barrier, depletion of podocytes and genetic susceptibility are the culprit factors in pathogenesis of FSGS. The condition can be idiopathic or occur secondary to obesity, intrarenal hemodynamic alterations, conditions with glomerulomegaly, the reduced number of nephron, and renal atheroembolic disease.[2,3] The tendency to thromboses may occur in PV which one of the chronic myeloproliferative disorders.[1] It has been suggested that the increase level of red blood cells, elevation of the platelet count, increase in tissue factor, polymorphonuclear leukocytes, coagulation reactions related to the platelet surface and the presence of microparticles were culprit factors.[11]

In the light of these data, we hypotezised that PV may cause of FSGS via recurrent thrombosis in microvascular level. Furthermore, it is well known that atheroembolic disease is a cause of FSGS. Thus, our case is important for present to develop of FSGS in the patient with PV. In the existing literature, a small number of cases of FSGS that are thought to be due to PV have been reported.[4-10] In addition, 3.6 % (only two PV) incidence of FSGS has been reported in patients with myeloproliferative disease.[10] It has been expressed in these case reports that hyperviscosity from increased hematocrit, hypoperfusion, predisposition to thrombosis related to elevated platelet counts and the continuation of these conditions in recurrent attacks may have a role on the development of FSGS.[9] The emergence of FSGS has been reported average three to seven years after the diagnosis of PV.[4-10] In the case of the subject of our study, considering that FSGS has been diagnosed with PV six years later is consistent with the literature. The PV was thought to be the possible reason for FSGS. Additionally, FSGS may occur by occlusions due to the long term recurrent microvascular thrombosis and this also could disorder to glomerular hemodynamics.

Consequently, the co-existence of PV and FSGS seems to be a cause-effect relationship rather than a random combination. Further studies will be needed to demonstrate for a better understanding of this association.

Table 1. Haematological, chemistrical, serological and urinalysis findings of patient.

Haematology	Blood chemistry and Serology		Urine analysis
WBC (mm³): 11600	ALP: 88 U/L	Na: 142mmol/L	Glucose (-)
RBC (mm³):	AST: 23 U/L	K: 5.06 mmol/L	Protein (++)
Htc (%): 52	ALT: 29 U/L	Uric acid: 4.8 mg/dl	O.B. (-)
Hb (g/dl): 16.3	LDH: 230 U/L	Anti-HCV (-)	Microscopic
PLT: 562000	Total Cholesterole:	HBsAg (-)	Examination:
	107 mg/dl		WBC: 3
	TG: 201 mg/dl	Anti-HIV (-)	RBC: 2
	TP: 6.7 g/dl	ANA (-)	24 hour urine
	Albumin: 4,2 g/dl	C3:1.2 g/L	protein: 4 gr/day
	Glucose: 88 mg/dl	C4:0.3 g/L	
	BUN: 18 mg/dl	RF (-)	
	Crea: 1.2 mg/dl	PTH: 64 pg/ml	

ALP: alkaline phosphatase; BUN: blood urea nitrogen; O.B.: occult blood; TP: total protein.

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Conflict of interest

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Tubulointerstitial nephritis and sclerosing cholangitis associated with autoimmune pancreatitis

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To the Editor:

Autoimmune pancreatitis (AIP) is a form of chronic pancreatitis caused by an autoimmune inflammatory process with lymphocyte infiltration and fibrosis that lead to organ dysfunction,[1] related to high levels of IgG4 and anti-carbonic anhydrase II antibodies.2,3 This disease frequently produces extra-pancreatic manifestations as well, such as sclerosing cholangitis and tubulointerstitial nephritis.[4]

Sclerosing cholangitis associated with AIP produces imaging test results and a clinical presentation similar to that of primary sclerosing cholangitis (PSC), but has a dramatic response to steroid treatment.[5]

Here, we describe the case of a patient with repeated episodes of pancreatitis and cholangitis who was managed as a case of PSC with no response, and who developed tubulointerstitial nephritis with renal biopsy findings suggestive of an autoimmune process, with resolution of gastrointestinal and renal manifestations through the administration of steroids.

CASE REPORT

Our patient was a 37-year old male who sought treatment in March 2006 for jaundice, fever, and abdominal pain; we first suspected an episode of cholangitis, but an endoscopic retrograde cholangiopancreatography and p-ANCA tests due to suspected PSC were negative, leading to the suspicion of microlithiasis.

In May of 2006, we performed an endoscopic sphincterotomy. Eight days later, the patient showed another episode of cholangitis. We considered the possibility of acalculous gallbladder disease as the cause for the recurring cholangitis; a cholecystokinin scintigraphy was compatible with this diagnosis, and we performed a laparoscopic cholecystectomy, but 15 days later the patient returned with yet another episode of cholangitis.

We returned to the suspected diagnosis of PSC, and performed a liver biopsy that revealed acute cholangitis with minimal foci of fibrosis. In early 2007, we administered a magnetic resonance cholangiography that revealed constrictions that were compatible with the diagnosis of PSC, with no possibility of performing a surgical intervention.

We managed the patient as a case of PSC, administering ursodeoxycholic acid and low doses of antibiotics (ciprofloxacin), and yet the patient continued to suffer repeated episodes of cholangitis.

In October 2007, the patient sought treatment for fever and abdominal pain; we started treatment with ciprofloxacin and requested an abdominal contrast tomography based on the patient's creatinine value of 8.7mg/dl. In May 2007, the patient's creatinine value was 1.2mg/dl.

The patient was evaluated in nephrology, and the only finding was paleness.

Laboratory analyses revealed creatinine: 7.6mg/dl, blood urea nitrogen (BUN): 46, normal sodium and potassium levels, pH: 7.32, bicarbonate: 16, Hb: 9.7g/dl, urinalysis with glycosuria (50mg/dl) and no hyperglycaemia.

A renal ultrasound revealed normally sized kidneys with increased bilateral echogenicity.

The patient was diagnosed with acute renal failure secondary to tubulointerstitial nephritis due to the consumption of quinolones.

After antibiotic treatment was removed and the patient was hydrated on the following day, creatinine decreased to 5.5mg/dl and BUN to 36mg/dl. Serum complement was normal, anti-nuclear antibodies (ANA) and serological tests for syphilis (VDRL) and human immunodeficiency virus (HIB) were negative; 24-hour proteinuria was 580mg. The patient was discharged with a creatinine value of 2.2mg/dl.

Twenty days later, the patient returned again for treatment for fever, diarrhoea, and oedema. Upon hospitalisation the patient had a creatinine value of 15mg/dl, potassium at 5.8mEq/l, and urine cytochemistry revealed leukocyturia, proteinuria (25mg/dl), glycosuria (50mg/dl), and haematuria (erythrocytes: 6 per field). A physical examination revealed no pathological findings. We considered this to be an exacerbation of the previous case of renal failure; due to the suspicion of tubulointerstitial nephritis, we started treatment with prednisone and took a renal biopsy.

The renal biopsy revealed: acute tubulointerstitial nephritis; immunofluorescence revealed: IgG ++ (interstitial), IgA and IgM +++ (interstitial), k and lambda chains: absent, C3: +++ peripheral, M and Bowman's