9. Bergmann C, Senderek J, Windelen E, Küpper F, Middeldorf I, Schneider F, et al. Clinical consequences of PKHD1 mutations in 164 patients with autosomal-recessive polycystic kidney disease (ARPKD). Kidney Int. 2005;67:829.

Víctor Martínez[a,*], Cristina Trasancos[b], Fernanda Ramos[a], Concepción Alcázar[a], Juan B. Cabezuelo[a], Miguel A. García-González[c]

[a] *Servicio de Nefrología, Hospital Reína Sofía, Murcia, Spain*
[b] *Servicio de Medicina Interna, Hospital Morales Meseguer, Murcia, Spain*
[c] *Unidad de Nefro-genética, Hospital Clínico Universitario de Santiago, Santiago de Compostela, A Coruña, Spain*

[*] *Corresponding author.*
E-mail address: victormj80@gmail.com (V. Martínez).

http://dx.doi.org/10.1016/j.nefroe.2016.06.002

A rare cause of diarrhea in patients with focal segmental glomerulosclerosis

Una causa poco frecuente de diarrea en pacientes con glomeruloesclerosis focal y segmentaria

Dear Editor,

Focal segmental glomerulosclerosis (FSGS) symbolizes a common histologic pattern of glomerular injury associated with numerous disease mechanisms. Ulcerative colitis (UC) represents one of the types of inflammatory bowel disease, which occurs in genetically predisposed individuals. The coexistence of these two diseases is an unexpected condition. Lately, case reports have been published documenting the development of nephropathy after treatment of ulcerative colitis with mesalamine or sulphasalazine. In cases in the literature, this coexistence has been identified as associated with 5-ASA therapy.[1–5] In this case, we report the ulcerative colitis occurring in a patient with focal segmental glomerulosclerosis not affiliated with 5-ASA therapy.

A 66-year-old man, with a 3-year history of focal segmental glomerulosclerosis, was admitted with bloody and mucoid diarrhea which had been for lasting for 10 days. There was no fever, nausea, vomiting or infection. There was no feature in the patient's history except diarrhea. Physical examination also was normal. Laboratory investigation demonstrated impaired renal function and proteinuria due to focal segmental glomerulosclerosis. Renal function test which is similar to the old values showed serum creatinine level of 3.15 mg/dl (0.8–1.3), BUN level of 89 mg/dl (17–43) and 24-h urine protein level of 1876 mg/day (<200). In addition, his erythrocyte sedimentation rate (ESR) was 79 mm/h (<20) and C-reactive protein (CRP) was 6.66 mg/dl (<0.4). A large amount of leukocytes and erythrocytes was seen in the stool microscopy. Stool cultures were detected negative twice. Colonoscopy revealed that there were to exudates of millimetric ulcers descending colon, sigmoid colon and rectum. The colon biopsy confirmed the diagnosis of ulcerative colitis.

The patient was started on mesalamine. His symptoms showed marked improvements after starting mesalamine treatment. After treatment, laboratory investigation demonstrated; creatinine level of 3.7 mg/dl (0.8–1.3), BUN level of 116 mg/dl (17–43), ESR level of 38 mm/h (<20), CRP level of 0.319 mg/dl (<0.4), and 24-h urine protein level of 2099 mg/day (<200). There were no abnormalities suggestive of nephrotoxicity in patients due to mesalamine, while acute phase reactants declined. The decline in ESR and CRP levels is thought to be in favor of improving ulcerative colitis activation.

A focal segmental glomerulosclerosis after ulcerative colitis treatment with mesalamine and sulfasalazine has been reported in the literature.[1] Additionally in the literature, there have been several minimal changes in the disease following the treatment of inflammatory bowel disease with mesalamine or sulfasalazine.[2–6] A case report has been published of nephrotic syndrome due to Crohn's disease with mesalamine treatment.[7]

In this case, we discuss about the developed ulcerative colitis in a patient who was followed for focal segmental glomerulosclerosis. Unlike previously reported cases, mesalamine and sulfasalazine have no effect on the togetherness of the two diseases. Although primary and secondary FSGS forms are defined based on the underlying cause, the podocyte damage is a common result eventually. Some genetic factors affect the inflammation which is the main cause of development of the ulcerative colitis. An unknown cause such as genetic, environmental or infections except drugs may be factors in the etiology of these two diseases. Furthermore, an unknown cause can facilitate the development of nephrotoxicity after mesalamine and/or sulfasalazine treatment. In our case, the patient's renal function did not change significantly after mesalamine treatment.

The coexistence of ulcerative colitis and focal segmental glomerulosclerosis is a rare condition. Mesalamine and/or sulfasalazine which have been used in ulcerative colitis treatment may be nephrotoxic. In our case, we have detected togetherness between ulcerative colitis and non-drug-induced focal segmental glomerulosclerosis. It should be kept in mind that the two diseases may be caused by an unknown factor such as genetic, environmental or infections except drugs.

REFERENCES

1. Fofi C, Nicoletti MC, Onetti Muda A, Giulio S. Focal segmental glomerulosclerosis with IgA deposits in a patient with ulcerative colitis. G Ital Nefrol. 2003;20:641–4.
2. Molnar T, Farkas K, Nagy F, Ivanyi B, Wittmann T. Sulfasalazine-induced nephrotic syndrome in a patient with ulcerative colitis. Inflamm Bowel Dis. 2010;16:552–3.
3. Skhiri H, Knebelmann B, Martin-Lefevre L, Grunfeld JP. Nephrotic syndrome associated with inflammatory bowel disease treated by mesalazine. Nephron. 1998;79:236.
4. Fornaciari G, Maccari S, Borgatti PP, Rustichelli R, Amelio N, Lattuada I, et al. Nephrotic syndrome from 5-ASA for ulcerative colitis? Complicated by carcinoma of the colon and sclerosing cholangitis. J Clin Gastroenterol. 1997;24:37–9.
5. Barbour VM, Williams PF. Nephrotic syndrome associated with sulphasalazine. Br Med J. 1990;301:818.
6. Novis BH, Korzets Z, Chen P, Bernheim J. Nephrotic syndrome after treatment with 5-aminosalicylic acid. Br Med J (Clin Res Ed). 1988;296:1442.
7. Firwana BM, Hasan R, Chalhoub W, Ferwana M, Kang JY, Aron J, et al. Nephrotic syndrome after treatment of Crohn's disease with mesalamine. Avicenna J Med. 2012;2:9–11.

Osman Saglam[a,*], Selman Unverdi[b], Murat Duranay[b]

[a] *Department of Internal Medicine, Ankara Training and Research Hospital, Ankara, Turkey*
[b] *Department of Nephrology, Ankara Training and Research Hospital, Ankara, Turkey*

[*] *Corresponding author.*
E-mail address: ossag03@gmail.com (O. Saglam).

http://dx.doi.org/10.1016/j.nefroe.2016.04.002

Acute renal failure secondary to interstitial acute nephritis and Fanconi syndrome due to metamizole and gemfibrozil☆

Fracaso renal agudo por nefritis intersticial aguda con síndrome de Fanconi en relación con metamizol y gemfibrozilo

To the Editor:

We present a case of acute interstitial nephropathy (AIN) associated with Fanconi syndrome, resolving after early treatment with steroids.

A 49-year-old man with a past medical history of alcoholic pancreatitis and liver disease, hypertriglyceridemia, and hypertension (HTN) treated with gemfibrozil and valsartan for over one year.

Two months ago, he had undergone an umbilical hernia repair. At that time, plasma creatinine was 0.79 mg/dl and urinalysis was normal. He received a cephalosporin, and 3 weeks of analgesia in the form of metamizole and paracetamol, with complete recovery. Two weeks later, metamizole was restarted at a dose of 575 mg/8 h orally for flu-like symptoms. He developed progressive weakness, abdominal pain, and nausea. Oral intake was reduced and BP control worsened, therefore the doses of valsartan and metamizole were increased, with additional paracetamol as required. His general condition worsened, therefore he attended the emergency department where BP was 135/85. Skin and mucous membranes were dry, he was afebrile, with no skin lesions, and no other physical findings. He had a high urine output (3.5 L/day), which was macroscopically normal. Blood tests showed a full blood count with no eosinophilia, metabolic acidosis with normal lactic acid, normal CK, reduced glomerular filtration rate (GFR), hypouricemia, and a mixed proteinuria of 1.4 g/24 h. Urinalysis revealed an alkaline pH, glycosuria with normoglycaemia, microhaematuria, isosthenuria, and the presence of granular hyaline casts.[4,5] There was reduced tubular reabsorption of phosphate, potassium, calcium, and uric acid, and generalised hyperaminoaciduria. Abdominal ultrasound showed kidneys measuring 146 mm with preserved corticomedullary differentiation and no signs of hydronephrosis. There were no abnormalities of the bladder or prostate. Viral serology and tumour markers were negative. Immunology was normal,

☆ Please cite this article as: Martín-Navarro JA, Petkov-Stoyanov V, Gutiérrez-Sánchez MJ, Pedraza-Cezón L. Fracaso renal agudo por nefritis intersticial aguda con síndrome de Fanconi en relación con metamizol y gemfibrozilo. Nefrologia. 2016;36:321–323.