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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.

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

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

Sr. Director:

Exponemos un caso de nefropatía intersticial aguda (NIA) asociada a síndrome de Fanconi con resolución tras tratamiento precoz con corticoides.

Varón de 49 años. Hepatopancreatitis alcohólica previa. Hipertrigliceridemia e hipertensión arterial (HTA) que trata con gemfibrozilo y valsartán desde hace un año.

Dos meses antes, se realiza herniorrafia umbilical. En ese momento, la creatinina en plasma 0,79 mg/dl y el sistemático de orina, normal. Se trata mediante cefalosporina y analgesia con metamizol y paracetamol durante 3 semanas, con recuperación completa. Catorce días antes, reinicia metamizol a dosis de 575 mg/8 h vía oral por proceso gripal. Progresivamente desarrolla astenia, anorexia, dolor abdominal y náuseas. Disminuye la ingesta y empeora el control de TA, por lo que se aumenta las dosis de valsartán y metamizol, y se asocia paracetamol, ocasionalmente. Su estado general empeora, por lo que acude a urgencias donde se objetiva TA 135/85. Piel y mucosas secas, afebril, no lesiones en piel, ni otros hallazgos físicos. Diuresis elevada (3,5 l/día) macroscópicamente normal. Hemograma sin eosinofilia, acidosis metabólica con ácido láctico normal, CPK normal, deterioro de filtrado glomerular (FG), hipouricemia, proteinuria mixta de 1,4 g/24 h; sistemático de orina con pH alcalino, glucosuria con normoglucemia, microhematuria, isostenuria y presencia de cilindros hialino granulosos. Disminuida la reabsorción tubular de fosfatos, potasio, calcio, ácido úrico e hiperaminoaciduria generalizada. Ecografía de abdomen con riñones de 146 mm con diferenciación corticomedular conservada y sin signos de hidronefrosis. Vejiga y próstata sin alteraciones. Serología vírica y marcadores tumorales: negativos. Inmunología: normal, incluyendo complemento, ANA, ANCA, anti-MBG, proteinograma e inmunoglobulinas. Paratohormona intacta 72 pg/ml, 25-OH-vitamina D 6.0 ng/ml, ECA normal, Mantoux negativo y Rx de tórax normal (evolución analítica, tabla 1). Tras corregir los factores prerrenales sin mejoría de la FG, se realiza biopsia renal en la que visualizamos 13 glomérulos sin alteraciones, intersticio edematoso, intenso infiltrado linfoide acompañado de polimorfonucleares neutrófilos y eosinófilos, sin granulomas, necrosis tubular aguda, arterias y arteriolas sin alteraciones, inmunofluorescencia directa sin depósitos de inmunocomplejos ni complemento. Con el diagnóstico de fracaso renal agudo por necrosis tubular y nefritis intersticial aguda (NIA) secundaria a metamizol con síndrome de Fanconi se inicia tratamiento con metilprednisolona, 250 mg/día/3 días para pasar a prednisona a 1 mg/kg/día que se disminuye progresivamente hasta suspender 12 semanas después, tras las cuáles recupera su FG y las alteraciones tubulares.