Conclusions. Although traditionally fever is a considered a hallmark of FMF, with the discovery of genetic mutations, we can confirm a greater variety of clinical presentation, not all cases presenting with all classical symptoms. The described family presents with mainly peritoneal symptoms and all siblings display the same mutations FMF-V726A heterozygote and FMF-E148Q heterozygote

Key words: Familial Mediterranean Fever, serositis, genetic testing

42. A CASE OF IGA NEPHROPATHY AND AMYLOIDOSIS IN PATIENT WITH ANKYLOSING SPONDYLITIS

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Background. IgA nephropathy is considered the most common cause of glomerulonephritis. Traditionally it presents with gross hematuria after an upper airway infection. However, there is a considerable population presenting asymptomatic microscopic hematuria. Patients with SpA are believed to be more affected by IgA nephropathy than the general population, as the two conditions share common etiopathogenic pathways. This mechanism might involve the decreased expression of the receptor responsible for the clearance of IgA 1 and its immune complexes on the surface of monocytes and neutrophils. Another frequent association for patients with systemic inflammatory diseases is renal amyloidosis.

Case report. Male patient B, 49 y.o., was admitted to the Republican Clinical Hospital in Apr 2017 with hypotension (75/50 mmHg), profuse edema of lower limbs up to inguinal area and confusional state. Patient was known with a history of Ankylosing Spondylitis since the age of 14, with IV x-ray stage of sacroiliitis, coxofemural and spine involvement. Since 1991 the patient followed regularly NSAIDs and intermittently corticosteroids in small doses. For a period of 6 years intermittent microscopic hematuria and mild proteinuria were noticed. The patient repeatedly tested with increased levels of serum IgAs, however refused kidney biopsy. In December 2016 he was admitted with fever, myalgia and arthralgia and HTA to a local intensive care unit. Upon that admittance the patient displayed oliguria, microscopic hematuria, mild proteinuria, and accelerated ESR, with a creatinine of 249 mcmol/L. Musculoskeletal complaints prompted increased doses of NSAIDs and corticosteroids (Prednisone 40 mg, and Aceclofenac 100mg x 2 /day), considering his main disease, despite the modified pattern of myalgia and peripheral arthralgia. A week after he was discharged he developed profuse edema that consequently led to his admittance to the republican hospital. Hematology revealed severe anemia, leucocytosis and accelerated ESR. Urinalysis showed normal SD, with leucocyturia up to 27 HPF, microscopic hematuria up to 80 RBCs HPF, with a proteinuria of 30 g/24h. Serum chemistry showed hypoproteinemia (32 g/L) and hypoalbuminemia (8.6 g/L), and elevated creatinine – 409 mcmol/L. Kidney biopsy was performed revealing moderate amyloid deposits. Despite initiated hemodyalisis, the patient died within 1 month from multiorgan insufficiency.

Conclusions. long standing AS favored the development of IgA nephropathy in the given patient; most likely the co-occurrence of newly depicted high levels of creatinine, with hematuria and modified pattern on musculoskeletal complaints spoke about acute tubulo-

interstitial nephritis due to use extensive use of NSAIDs precipitating loss of kidney function particularly considering pre-existing amyloid deposits.
Key words: IgA nephropathy, renal amyloidosis, ankylosing spondylitis

43. A CASE OF DIFFERENTIAL DIAGNOSIS IN A PATIENT WITH HAND OA

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Background: Hand osteoarthritis is mainly a primary osteoarthritis, involving genetic predisposition. Although clinical diagnostic criteria were developed and many cases can be diagnosed without additional diagnostic procedures, some patients need a comprehensive assessment to exclude other possible arthritis.
Case report: Female patient A., 61 years old, presented with pain both at rest and during motion in wrists, first CMC (carpometacarpal), 2-3rd MCP (metacarpophalangeal) and first to Vth PIPs (proximal interphalangeal), as well as 2-3rd DIPs (distal interphalangeal) joints. Being asked the patient reported morning stiffness more than 30 minutes but less than one hour. She reported the symptoms having a gradual onset for the last year, however the complaints worsened in the last 2 months and as she reports the MCPs got swollen in the last months. Physical examination revealed no tenderness in the wrists, yet significant tenderness in both first CMCs, mildly tender MCPs on squeeze test, as well as tenderness and mild swelling in II-III PIPs. At this moment considering morning stiffness, the reported joint swelling and the pattern of joint involvement, 3 main diagnoses should be considered: early onset RA, osteoarthritis of the hand and calcium pyrophosphate deposition disease. Laboratory assessments: Uric acid: 402 μmol/l; ALT:26.1 U/L; Anti HBcor sum (Anti HBcor sum:7.27 S/CO, Anti HBcor sum: Reactive); Anti HCV (Anti HCV:0.08 S/CO, Anti HCV: Nonreactive); ASL-O (Antistreptolizina-O):91 IU/ml; AST:31.7 U/L; Direct Billirubin:6.4 μmol/l; Total Billirubin:17.0 μmol/l; Calcium:2.68 mmol/l; Creatinine:58.0 μmol/l; Rheumatoid factor: 124.0 IU/ml; HBs Ag (HBs Ag:0.31 S/CO, HBs Ag :Nonreactive); C-reactive protein: 5.56 mg/l; Uree:8.1 μmol/l; Fibrinogen: 4.0 g/l; anti CCP < 10 U/ml. X-ray revealed diffuse moderately expressed osteoporosis, signs of osteophyte formation in the PIPs, and asymmetric narrowing of the joint space, and subchondral bone sclerosis, advanced disarthrosis, capsular densifications on the capsule of the II and III MCPs.
Conclusions: The final diagnosis was Hand osteoarthritis based on specific radiological findings and a clinical picture pleading more for a degenerative condition. Initially, before the definite development of Heberden's and Bouchard's nodes patients go through a stage of inflammation with mild joint swelling, which poses certain question in the initial diagnosis of hand osteoarthritis. The confounding laboratory data such as presence of Rheumatoid Factor and a mildly increased C-reactive protein may be explained by the depiction of positive Anti-Hbcore sum). Additionally the patient did not have anti-CCP antibodies which are more specific for RA.
Key words: hand osteoarthritis, rheumatoid arthritis, calcium pyrophosphate deposition disease, osteophytes, joint space narrowing