An association was observed between both methods and variables of remission or disease activity.

Disclosure of Interests: None declared


THE EFFECT OF DRUG THERAPY IN JUVENILE IDIOPATHIC ARTHRITIS ON THE LEVEL OF CYSTATIN C AS A MARKER OF RENAL FUNCTION

S. Samsonenko1, T. Borysova1. 1Dnipro State Medical University, Department of Pediatrics, Dnipro, Ukraine

Background: Juvenile idiopathic arthritis (JIA) is a chronic disease requiring years of therapy with non-steroidal anti-inflammatory drugs (NSAIDs), immunosuppressant’s, cytostatics, immunobiological agents. The aforementioned drugs, namely NSAIDs and cytostatics are potentially nephrotoxic [1]. The above drugs, namely NSAIDs and cytostatics, are potentially nephrotoxic. About 8% of children with JIA have kidney damage, which develops on average 5 years after the onset of the disease. It has been established that the main risk factor for the development of kidney damage is the long-term exposure to NSAIDs and methotrexate in children with active forms of JIA [2]. Early diagnosis of kidney damage will allow timely correction in the dosage of drugs and avoid their nephrotoxic effects [3].

Objectives: To determine the effect of drug therapy in children with JIA on eGFR by using the Cystatin C-based equation and the Hoek formula based on the serum cystatin C study.

Methods: 80 children with JIA participated in the study. The age of subjects was 10.4±4.41 (10.6-15.0) years. All children received methotrexate as a base drug. At the moment of examination 22 children received NSAIDs, 25 children received immunobiological preparations. Serum cystatin C content was determined by enzyme immunoassay. The Cystatin C-based equation and Hoek formulas were used to set the GFR by serum cystatin C levels.

Results: Non-steroidal anti-inflammatory drugs led to a decrease in GFR as found by both the Cystatin C-based equation 2012 and the Hoek formula. The incidence of GFR reduction in patients treated with NSAIDs using the Cystatin C-based equation 2012 was 100%, and using the Hoek formula was 81.8%. The use of NSAIDs in children with JIA is a risk factor for the development of reduced GFR calculated by the Hoek formula. The incidence of reduced GFR in children with NSAID use was 54.5%, 6.7 times greater than in those without NSAIDs (OR = 12.9; CI: 3.76-44.25, p<0.001). There was a low chance of a Hoek formula decrease in GFR in children with JIA who received immunobiological therapy 9.1% vs 46.8% (OR = 0.11; CI: 0.03-0.42; p<0.001).

Conclusion: Use of NSAIDs in children with JIA was more often associated with a reduction in GFR by the Cystatin C-based equation 2012. However, the manifestations of SS that were present at the time of pSS verification were multidisciplinary: constitutional abnormalities – 33.3%, nonpolyserous polyarthritides – 64.3%, polyarthritis – 26.7%, lymphadenopathy – 73.3%, cutaneous involvement – 53.3% (2 – xerosis, 2 – annular erythema, 1 – erythema nodosum – 2, 2 – Raynaud phenomenon, 2 – nonspecific ratty rashes, 1 – hemorragic rash). At the time of diagnosis 7 pts (46.7%) had isolated involvement of salivary glands in 8 pts (53.3%) – combined with involvement of lacrimal glands. The decrease in salivary gland function was recorded in 80% of cases, hypolacrimia – in 46.7%, 1 patient had isolated hypolacrimia. Recurrent parotitis was present in 6 pts (40.0%). At time of diagnosis pulmonary involvement had 20.0% of pts, 1 patient had renal tubular acidosis. 8 pts (53.3%) had various hematological disorders: anemia – in 3 pts (20.0%), leukopenia – in 6 (40.0%). ARA Hep-2 were detected in 100% pts (in titer 1/80 – 4; 1/1200 – 7, 1/2560 – 3, 1/120000 – 1, with mixed patterns in all pts: speckled + homogenous – 9 pts, speckled + homogeneous-cytoplasmic – 6 pts), anti-Ro - in 12 pts (80.0%), anti-La – in 18 pts (53.3%). RF – in 9 pts (60.0%), 6 pts (40.0%) had polyclonal hypergammaglobulinemia, max 42%. 2 pts (13.3%) had concomitant autoimmune non-rheumatic disease; 1 – cutaneous psoriasis, 1 – autoimmune thyroiditis. The treatment of each patient was justified by the main individual manifestations: 93.3% received glucocorticoids, 26.7% - methotrexate, 33.3% - hydroxychloroquine, 6.7% - myophenolate mofeti. Treatment with biotherapy (B) was received by 13 (33.3%) pts (7 – rituximab (RTM), 6 – abatacept (ABA)) with a good response in 10 pts, including improvement in the function of the salivary and lacrimal glands in 7 pts, 1 patient received 2B - RTM and ABA sequentially due to the development of MAS 7 days after 1st RTM infusion. B was discontinued in 3 pts: 1 due to development of hemorraghic vasculitis 2 days after the 1st RTM infusion, 1 – COVID-19 with lung involvement (CT 3-4) 2 weeks after the 1st RTM infusion, 1 – inefficiency of ABA during 15 months.

Disclosure of Interests: None declared


Prospective Evaluation of Cognitive Function in Patients with Juvenile Idiopathic Arthritis

E. Ortiz-Martínez1, C. Padilla-Leiva2, P. Cabezudo-Garcia2, G. Díaz-Cordobes1, L. Muñoz-Becerra1, N. Mena-Vázquez1, E. Instituto de Investigación Biomédica de Málaga (IBIMA), Hospital Regional Universitario de Málaga, UGC de Reumatología, Málaga, Spain; 2Universidad de Málaga, Departamento de Medicina, Málaga, Spain; 3Instituto de Investigación Biomédica de Málaga (IBIMA), Hospital Regional Universitario de Málaga, UGC de Neurociencias, Málaga, Spain

Objectives: Prospectively evaluate changes in the cognitive function of patients with juvenile idiopathic arthritis (JIA) and associated factors.

Methods: Design and protocol: We performed a prospective cohort study with JIA patients that participated in a previous cross-sectional study (2019) to evaluate cognitive function. After 24 months, the patients were administered the same test battery previously used through an established protocol, and data was collected from their clinical histories. The neuropsychological tests were corrected by a neuropsychologist and neuropsychologist. Study population: Inclusion criteria: Patients aged ≥16 years with JIA classified according to the criteria of ILAR 2001. Patients with inflammatory or rheumatic diseases other than JIA, previous neurological disease not associated with the course of JIA, and patients with scores lower than the normal in the manual skill test were excluded. Outcomes: The main variable was cognitive impairment, defined as worsening of ≥2 scaled points after 24 months (V24) in any of the subtests used to evaluate each cognitive area in the Wechsler Adult Intelligence Scale (WAIS). The evaluated cognitive domains and their respective subtests were: Attention/concentration (Digit Span); verbal function (Vocabulary); organization (Block Design); working memory (Letter-Number Sequencing); problem-solving (Similarities). Depression was evaluated by The Beck Depression Inventory-II (BDI-II): minimal (0-13), mild (14-19), moderate (20-28), and severe (29-63). Other variables: Clinical-epidemiological characteristics; treatments; and inflammatory activity evaluated as the C-reactive protein average standard deviation. Statistical analysis: Descriptive analysis, followed by χ2 and paired T-Test. Multivariate analysis to identify independent variables associated with impairment of cognitive function in JIA.