CHARACTERISTICS OF ARTICULAR SYNDROME IN SYSTEMIC VASCULITIS

[1]Syniachenko O., [2]Khaniukov O., [2]Yehudina Ye., [1]Taktashov H., [2]Gashynova K.

[1]Donetsk National Medical University of Health Ministry of Ukraine, Lyman;
[2]State Institution "Dnipropetrovsk Medical Academy of the Ministry of Health of Ukraine", Ukraine

The prevalence of systemic vasculitis (SV) is steadily rising in the world, accounting 2-3 cases per 10 thousand population [8,14]. Currently, the study of SV refers to the most dynamically developing areas of rheumatology, while emphasizing both the clinical and pathogenetic commonality of these diseases, and certain differences of separate nosological forms [1,2,10]. Differential diagnosis is rather complicated, because the clinical symptoms of SV are characterized by the presence of "crossed syndromes" [3].

The articular syndrome is one of the main signs of Takayasu's arteritis (TAA) [5], microscopic polyangiitis (MPA) [20], Wegener's granulomatosis with polyangiitis (GPA) [16,17], cryoglobulinemic vasculitis (CGV) [12,19]. Joint damage in the form of arthritis or arthralgia is observed in 65-70% of the patient number with eosinophilic granulomatosis with polyangiitis, Churg-Strauss syndrome (EPA) [9], in 40-50% of CGV [18], in 45-65% of Henoch-Shonlein purpura (HSP) [4,6,15], the knee and ankle joints are involved in the pathological process most often [7]. In MPA, the number of patients with musculoskeletal changes increases within the first 5 years after debut of the disease, when, among other things, the decrease in periarticular bone mineral density begins to develop [11].

It should be noted that in the contemporary literature, only the frequency of arthropathy in patients with SV is noted. The purpose and objectives of this study are the research of separate joint lesion in TAA, MPA, GPA, EPA, HSP, CGV and polyarteriitis nodosa (PAN), their X-ray sonographic characteristics, the relationship between severity of articular syndrome and extraarticular manifestations of diseases, and the aspects of the arthritis pathogenesis in such category of patients.

Material and methods. Under supervision were 525 patients with SV, the characteristics of which are presented in Table. 1. The ratio of the patients' number with HSP, MPA, CGV, PAN, TAA, GPA and EPA was 7: 4: 3: 1: 1: 1: 1. According to the generally recognized Chapel Hill nomenclature (USA, 2012), TAA refers to SV of large-vessels, PAN-medium. Small vessel vasculitis includes GPA, EPA and HSP, which is characterized by IgA-dominant immune deposits in the small blood vessels, and CGV, in which cryoglobulin immune deposits are responsible for small blood vessel vasculitis What is more, MPA, GPA and EPA are associated with antineutrophil cytoplasmic antibodies, HSP and CGV are immune complex SV [13]. Women tend to prevail in TAA, men – in PAN, at a ratio of 3: 1 and 2: 1 respectively, younger age patients were with HSP, and older - with CGV. It should be emphasized that the clinical characteristics of patients are presented at the time of their examination. So, at the previous stages, all examined with MPA, HSP and CGV had skin changes, 93% with HSP - joint syndrome. In cases of PAN, MPA and CGV, peripheral nervous system lesions (mono- and polyneuropathy, radiculopathy, and motron metatarsalgia) were exceeded, and the central nervous system lesions (discirculatory encephalopathy, cerebral circulation violation, corticoneural and pseudobulbar syndromes) prevailed in patients with TAA and GPA. 55% of TAA patients had aortic heart disease, 17% had myocardial infarction, 21% - a cerebral stroke. 36% patients with PAN were carriers of viral hepatitis B antigen (HBV), and 29% patients with CGV - hepatitis C (HCV). Cryoglobulins were detected in 97% of the patients with CGV at the time of the examination, increased level of immunoglobulin (Ig) A - in 72% of the patients with HSP, antineutrophil cytoplasmic antibodies - in 75% with GPA, in 71% with MPA and 57% with EPA (the ratioes of antibodies to myeloperoxidase and proteinase-3 were 1: 6, 2: 1 and 8: 1 correspondingly). The level of eosinophils in blood of the patients with EPA was 15.1±2.12% of the number of white blood cells or 2.1±0.82 10^9/l.

X-ray examination of peripheral joints, sacroiliac joints, spine and lungs was performed on the "Multix-Compact-Siemens" apparatus (Germany), ultrasound examination of joints and internal organs - on "Envisor-Philips" (Netherlands), electrocardiographic - on «MIDAC-EK1T» (Ukraine) and «Bioset-8000» (Germany), echocardiographic - on «HD-11-XE-Philips» (Netherlands), spirographic - on «Master -Scope-Jaeger »(Germany), study of the alveolar-capillary membrane – on «Master-Screen-Body-Jaeger »(Germany). "BS-200" and "Olympus-AU640" analyzers (Japan), "PR2100-Sanofi diagnostic Pasteur" reader (France), immunoblot "Euroline-Euroimmun" (Germany), computer tensiometers «ADSA-Toronto» (Germany-Kana-da) and «PAT2-Sinterface» (Germany) were used to evaluate the laboratory indices.

The number of painful joints (NPJ), the Lunsbury indices (iL), iDAS and iDAS28 were estimated, the integrated severity of arthropathy index (iSA) were calculated by the formula:

$iSA = \sqrt{iL} \bullet DAS.$

The statistical processing of the obtained research results was carried out using a computer variational, nonparametric, correlation, one (ANOVA) and multivariate (ANOVA / MANOVA) dispersion analysis (programmes "Microsoft Excel and Statistica-Stat-Soft», USA). Mean values (M), their standard errors (m), standard deviations

Table 1. Characteristics of the examined patients with SV

Indices	Group of patients with SV (n=525)						
	TAA	PAN	MPA	GPA	EPA	HSP	CGV
Number of patients in groups	29	39	116	28	27	193	93
Men/women %	24/76	62/38	41/59	57/43	44/56	51/49	41/59
Age (M±m), years	44,2±2,48	44,2±1,88	44,8±1,23	46,0±2,41	40,4±2,59	26,2±0,72	52,3±1,28
Duration of the disease (M±m, years)	10,4±1,98	8,5±1,30	6,4±0,70	4,3±0,83	10,7±2,05	9,0±0,60	4,6±0,62
II-III stage of activity, %	86	79	85	93	85	72	92
Acute course, %	–	15	28	56	–	19	43
Signs of lesion, %							
skin	17	31	82	21	52	64	85
skeletal muscle	48	54	38	36	37	15	26
heart	76	80	51	50	48	31	61
liver	28	56	56	46	33	22	62
spleen	4	13	8	7	19	7	25
nervous system	21	90	48	46	37	18	40
kidney	62	62	67	64	56	67	68
Kidney failure, %	35	26	35	29	26	18	32
Glomerular filtration rate, ml/min	107,1 ±4,78	104,4 ±4,18	101,2 ±2,51	108,2 ±3,99	110,5 ±4,34	110,3 ±1,92	103,3 ±2,68

(SD), correlation coefficients, dispersion criteria, Student, Wilcoxon-Rao, McNamara-Fischer and reliability of statistical indicators were estimated.

Results and their discussions. Such joint lesions as arthritis or arthralgia, are noted in 32% of patients with GPA, in 41% patients with TAA, 47% - with HSP, 52% - with PAN, 63% - with MPA and EPA, 67% - CGV, and monoaligoarthritis - in 33; 50; 54; 25; 58; 18 and 50% of observations respectively. According to the one-factor dispersion analysis, the development of articular syndrome in MPA is influenced by changes in skeletal muscles and lungs, in GPA – by the duration of the disease, changes in the skin, heart and liver, in HSP – by the age of the patients, degree of the pathological process activity, skin lesions and kidneys, in EPA - only by skin syndrome, in CGV – by myositis/myalgia, pneumopathy and nephropathy. The occurrence of arthropathy depends on the severity of extra-articular manifestations of PAN, MPA, GPA, HSP, CGV.

Taking into account the frequencies of arthropathy formation in MPA and CGV, we conducted an additional analysis. It turned out that the articular syndrome in patients with MPA is reliably affected by the indicators of the ratio between systolic pressure in the pulmonary artery to the peripheral arterial pressure and diffusion capacity of the lungs, and in cases of CGV - parameters of pulmonary vascular resistance and end diastolic volume of the right ventricle.

As demonstrated by the performed ANOVA, the character of articular syndrome in TAA depends on the parameters of fibrinogen in blood, in PAN - on the concentration of circulating immune complexes and antibodies to native deoxyribonucleic acid (aDNA), in MPA - on the level of antibodies to proteinase-3, in GPA - on the content of the latter and the values of fibronectin in blood, in EPA - on C-reactive protein indications in blood and the presence of antibodies to myeloperoxidase, in HSP - on parameters of IgA, indices of platelet and red blood cells aggregation, thromboxane A2, prostacyclin and prostaglandin E2, in CGV - on tumor necrosis factor-γ, endothelin-1 and prostaglandin F_{2a}. It is necessary to emphasize that in PAN there is a significant influence HBV-carrier on the development of joint lesions. In such patients, the joint syndrome is detected in all cases, whereas it is only in 48% of cases without HBV (the differences are high-significant).

The frequency of the separate joint involvement in the pathological process and the presence of X-ray sonography signs of arthropathy are represented in generalized form in table. 2,3 at that features of the articular syndrome in patients with different variants of SV have been revealed. For EPA, there were typical lesions of vertex and sternoclavicular joints, for AAT - proximal interphalangeal joints of hand, humeral and knee articulations, for PAN - ulnar and metatarsophalangeal, for MPA - wrist and ankle, for CGV - sacroiliac and vertebra. In turn, the changes in the sternoclavicular junctions refer to the "joints-exclusion" in AAT, GPA and CGV, ankle, sacroiliac and vertebrae – in GPA, elbow – in TAA, shoulder –in EPA and CGV.

There are typical changes in meniscus horns and the presence of intra-articular bodies of Pellagri-Shtaydi and Hoff for TAA, and Baker cysts - for PAN. Tendovaginitis

Table 2. Typical and atypical signs of arthropathy in patients with SV

	TAA	PAN	MPA	GPA	EPA	HSP	CGV
1					⊕		⊗
2	⊗			⊗	⊕		⊗
3	⊕						
4							
5			⊕			⊕	
6	⊗	⊕				⊕	
7	⊕				⊗		⊗
8							
9		⊕					
10			⊕	⊗			
11	⊕						
12							
13				⊗			⊕
14	⊗			⊗			⊕

Joints: 1 - maxillary; 2 - sternoclavicular; 3 - proximal interphalangeal of hands; 4 - metacarpophalangeal; 5 - wrist; 6 - ulnar; 7 - humeral; 8 - proximal interphalangeal of foot; 9 - metatarsophalangeal; 10 - ankle; 11 - knee; 12 - hip; 13 - sacroiliac; 14 - vertebrae. Å - typical signs; Ä - absence of signs

Table 3 Typical and atypical symptoms of musculoskeletal system lesions in patients with SV

	TAA	PAN	MPA	GPA	EPA	HSP	CGV
I	⊗	⊕	⊗				⊕
II	⊗		⊗	⊗			
III							
IV		⊕		⊗	⊕		
V		⊕			⊕		⊗
VI	⊗			⊗	⊗		⊗
VII			⊗	⊗			
VIII			⊗	⊗			⊗
IX			⊗	⊗			
X	⊕	⊕		⊗			
XI	⊗			⊗			
XII	⊕			⊗			
XIII				⊗			
XIV	⊕						

Symptoms: I - tendovaginitis; II - enthesopathy; III - epiphyseal osteoporosis; IV - subchondral sclerosis; V - osteocystis; VI - osteousuras; VII - ligamentosis; VIII - aseptic necrosis; IX - arthrocalcinates; X - changes in the meniscus; XI - Baker's cysts; XII - bodies of Pellagri-Shtaydi; XIII - chondromic bodies; XIV - the bodies of Hoff; Å - a typical symptom; Ä - absence of a symptom

can be considered typical for PAN and CGV, subchondral sclerosis and osteocystosis - for patients with GPA and EPA. It is noted the absence of cases of tendovaginitis and entesopathy in TAA and MPA, and on the whole a scanty variety of "x-ray sonographic landscape of articular syndrome" in cases of GPA.

According to the multifactorial analysis of Wilcoxon-Rao, the character of the arthropathy course in patients with MPA depends on the lung and heart lesions, in the case of HSP - on the involvement of lung and liver, in CGV - on renal pathology and integral gravity of extraarticular signs of the disease. As ANOVA / MANOVA testifies, the integral X-ray and sonographic signs of joint changes in patients with PAN depend on the disease ac-

Table 4. Indices of joint syndrome in patients with SV (M±m)

SV	Index				
	NPJ, abs	IL, balls	iDAS, r.u.	iDAS 28, r.u.	iWA, r.u.
TAA	10,6±3,10	78,7±14,38	2,4±0,50	3,4±0,51	22,7±6,10
PAN	11,9±2,68	60,6±9,30	2,2±0,32	2,9±0,32	19,3±4,25
MPA	8,5±1,38	58,7±4,85	1,7±0,19	2,7±0,20	15,0±2,29
GPA	14,4±4,79	48,0±17,07	2,3±0,62	3,3±0,67	18,8±8,33
EPA	13,1±2,91	60,8±12,12	2,0±0,27	2,9±0,29	16,7±4,11
HSP	7,7±0,87	60,8±4,54	1,5±0,18	2,5±0,19	13,9±2,10
CGV	7,3±1,32	53,8±4,37	1,2±0,09	2,2±0,10	9,5±1,26

tivity degree and the severity of extraarticular manifestations, with MPA - on the nature of nephropathy, and with HSP - pneumopathy.

As performed by ANOVA, the age of patients with TAA, PAN, MPA and EPA affects the frequency of the maxillary articulation lesions. The changes in proximal interphalangeal joints of brushes closely related with the age of the patients with TAA, knee joints - with PAN, sternoclavicular, wrist and proximal interphalangeal feet - with MPA, wrist and shoulder - with HSP. The presence of cardiopathy (disturbance of myocardial excitability, electrical conduction of the heart, changes in the myocardium, endocardium and cardiac valves, diastolic and systolic dysfunction of the left ventricle) have a dispersive effect on the lesions of proximal interphalangeal joints of the fingers, wrist and ankle in patients with TAA, but only on the wrist - with CGV. Metatarsophalangeal articulation lesion in TAA depend on the nature of nephropathy (urinary or nephrotic syndrome, the condition of kidney function), in the case of PAN - wrist, CGV - proximal interphalangeal foot and hip.

The origin of tendovaginitis in PAN and EPA is closely related to hepatic pathology, entesopathy in MPA and HSP - to renal disease. The appearance of intraarticular bodies (chondromic, Pellagri-Shtaydi, Hoff) occurs in parallel with an increase of the disease duration in TAA patients, and they are determined by the severity of the skin-muscular syndrome in PAN patients. Bone-destructive signs of the musculoskeletal system disorders depend on extraarticular manifestations of SV. It should be noted that Baker's cysts in MPA and lesions of the maxillary joints in HSP are observed exclusively in men, whereas the incidence of lesions of proximal interphalangeal joints of brushes and metacarpophalangeal joints in patients with HSP significantly prevails in the female group, respectively in 3,7 and 3.2 times.

As can be seen from Table. 4, the greatest number of painful joints (NPJ) is peculiar to patients with GPA, and the greater integral severity of the joint syndrome is typical for patients with TAA. According to the dispersion analysis, parameters of NPJ in PAN are closely related to the severity of skin lesions, in MPA - to acute course of the disease, in HSP - to changes in the nervous system and in CGV - to severity of hepatic pathology.

The degree of severity of articular syndrome assessed by iSA, in PAN and HSP depends on the functional state of the kidneys, in MPA and HSP - on the integral severity of the extraarticular signs of the disease. In addition, iSA is affected by the overall disease activity in the group of patients with HSP. Whereas the severity of arthropathy grows with an increase in the duration of MPA course, then in patients with HSP its decreases (Fig. 1). In turn, in both groups of SV patients, iSA grows, according to the increase in the integrated severity of extrarenal manifestations of diseases (Fig. 2).

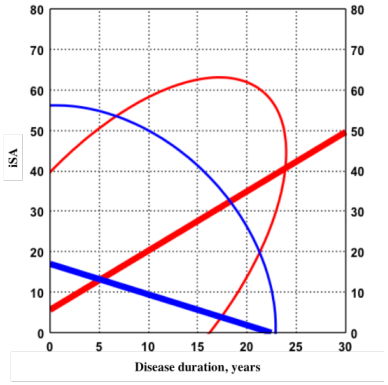

Fig. 1. The relationship between iSA indicators with MPA duration (red curves) and HSP (blue)

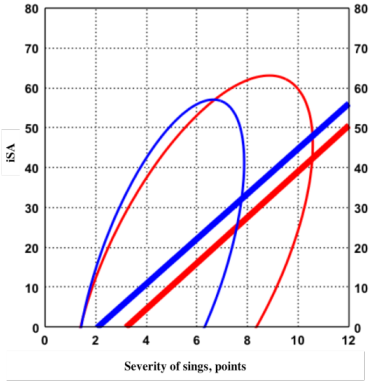

Fig. 2. The relationship between iSA indicators and the severity of MPA symptoms (red curves) and HSP (blue)

The prevalence of articular syndrome in patients with TAA is significantly influenced by the indices of circu-

lating immune complexes in blood and antibodies to cardiolipin, with MPA – by levels of rheumatoid factor (RF), fibrinogen, aDNA, endothelin-1, homocysteine and module of viscoelasticity of blood serum, with GPA – by concentration of RF, antibodies to myeloperoxidase, IgG, cyclic guanosine monophosphate and surface viscosity of blood, with HSP - aDNA, anti-cardiolipin and tumor necrosis factor-α, with CGV - RF and IgM. The severity of the joint syndrome course in cases of AAT, PAN and MPA is associated with the seropositivity of the diseases by RF, GPA - with the presence of antibodies to proteinase-3, EPA - with the content of fibronectin, HSP - with levels of IgA, b2-microglobulin and RF, CGV - with bulk viscosity, surface elasticity, relaxation with thromboxane A2. According to the correlation analysis, iSA index directly relates to RF parameters in blood in patients with MPA, and vice versa - with its viscoelasticity, and in patients with HSP - to IgA concentration, with CGV - to the level of thromboxanemia.

Taking into account the statistical processing of the obtained results of studies, relevant conclusions having a certain practical importance have been made: 1) prognostic factors with respect to the frequency of development and course of joint pathology in patients with PAN are HBV-carrier, and in MPA, HSP and CGV - severity of extraarticular signs of diseases; 2) RF parameters in blood in TAA, PAN, MPA, HSP and CGV > 20 IU / ml (> M±SD patients with SV) are considered risk factors for the severe course of arthropathy.

Conclusion. Such joint damage as arthritis or arthralgia is revealed in 32% of patients with GPA, 41% - with AAT, 47% - with HSP, 52% - PAN, 63% - MPA and EPA, 67% - CGV, which has gender differences, depends on the duration of the disease, the degree of activity of the pathological process, the severity of extra-articular signs, lung function and the state of hemodynamics in the lesser circulation, the frequency of lesions of individual bone articulations, tendovaginitis, enthesopathy and X-ray sonography signs of the joint syndrome. There has been its own dimorphism in different nosologies, in patients with PAN it is associated with the carriage of HBV, and in the pathogenetic constructs of arthropathy in SV there are disorders of the immunity system (immuno-inflammatory proteins, cytokines, various antibodies), rheological properties of blood and endothelial functions of blood vessels. And the high RF parameters in blood are risk factors for the severe course of joint damage in cases of TAA, PAN, MPA, HSP and CGV.

REFERENCES

1. Дядык А.И. (ред.) Системные васкулиты в современной клинической практике. Издатель Заславский, Донецк, 248 с. 2013
2. Кузьміна А.П. Клінічна схожість симптомів систем-них васкулітів при різному походженні. Укр. ревматол. журн., 2013, 53(3): 133–134.
3. Шилкина Н.П., Дряженкова И.В. Системные васкулиты: этапы диагностики. Терапевт. арх., 2013, 85(4): 39–42.
4. Belli A.A., Dervis E. The correlation between cutaneous IgM deposition and renal involvement in adult patients with Henoch-Schönlein purpura. Eur. J. Dermatol., 2014, 24(1): 81–84.
5. Borchers A.T., Gershwin M.E. Giant cell arteritis: a review of classification, pathophysiology, geoepidemiology and treatment. Autoimmun. Rev., 2012, 11(6–7): 544–554.
6. Calvo-Rio V., Loricera J., Mata C. et al. Henoch-Schönlein purpura in northern Spain: clinical spectrum of the disease in 417 patients from a single center. Medicine, 2014, 93(2): 106–113.
7. Chen O., Zhu X.B., Ren P. et al. Henoch Schonlein purpura in children: clinical analysis of 120 cases. Afr. Health Sci., 2013, 13(1): 94–99.
8. Della Rossa A, Cioffi E, Elefante E, Ferro F, Parma A, Vagelli R, Talarico R., Mohammad A.J. et al. Systemic vasculitis: an annual critical digest of the most recent literature. Clin Exp Rheumatol. 2014 May-Jun;32(3 Suppl 82):S98-105.
9. Gendelman S, Zeft A, Spalding SJ. Childhood-onset eosinophilic granulomatosis with polyangiitis (formerly Churg-Strauss syndrome): a contemporary single-center cohort. J. Rheumatol., 2013,40 (6): 929-935.
10. Gibelin A, Maldini C, Mahr A. Epidemiology and etiology of wegener granulomatosis, microscopic polyangiitis, churg-strauss syndrome and goodpasture syndrome: vasculitides with frequent lung involvement. Semin. Respir. Crit. Care Med., 2011,32(3): 264–273.
11. Itabashi M, Takei T, Moriyama T et al. Long-term damage assessment in patients with microscopic polyangiitis and renal-limited vasculitis using the Vasculitis Damage Index. Mod. Rheumatol., 2014, 24(1): 112–119.
12. Lamprecht P. Cryoglobulinaemic vasculitis: new aspects. Clin. Exp. Rheumatol., 2012,30(1): 3–5.
13. Luqmani RA, Suppiah R, Grayson PC, Merkel PA and Watts R. Nomenclature and classification of vasculitis – update on the ACR/EULAR Diagnosis and Classification of Vasculitis Study (DCVAS)., Clin Exp Immunol. 2011 May; 164 (Suppl 1): 11–13.
14. Mohammad AJ, Jacobsson LT, Westman KW et al. Incidence and survival rates in Wegener's granulomatosis, microscopic polyangiitis, Churg-Strauss syndrome and polyarteritis nodosa. Rheumatology, 2009, 48(12): 1560–1565.
15. Poterucha TJ, Wetter DA, Gibson LE et al. Histopathology and correlates of systemic disease in adult Henoch-Schönlein purpura: a retrospective study of microscopic and clinical findings in 68 patients at Mayo Clinic. J. Am. Acad. Dermatol., 2013,68(3): 420–424.
16. Salazar-Exaire D., Ramos-Gordillo M., Vela-Ojeda J. et al. Silent ischemic heart disease in a patient with necrotizing glomerulonephritis due to Wegener's granulomatosis. Cardiorenal. Med. 2012; 2(3): 218–224.
17. Soriano A., Lo Vullo M., Casale M. et al. Meningeal involvement in Wegener granulomatosis: case report and review of the literature. Int. J. Immunopathol. Pharmacol., 2012, 25(4): 1137–1141.
18. Terrier B., Krastinova E., Marie I. et al. Management of noninfectious mixed cryoglobulinemia vasculitis: data from 242 cases included in the CryoVas survey. Blood, 2012, 119(25): 5996–6004.
19. Terrier B., Cacoub P. Cryoglobulinemia vasculitis: an update. Curr. Opin Rheumatol., 2013, 25(1): 10–18.
20. Tsuchiya N. Genetics of ANCA-associated vasculitis in Japan: a role for HLA-DRB1*09:01 haplotype. Clin. Exp. Nephrol., 2012, 23(11): 132–136.

SUMMARY

CHARACTERISTICS OF ARTICULAR SYNDROME IN SYSTEMIC VASCULITIS

[1]Syniachenko O., [2]Khaniukov O., [2]Yehudina Ye., [1]Taktashov H., [2]Gashynova K.

[1]Donetsk National Medical University of Health Ministry of Ukraine, Lyman; [2]State Institution "Dnipropetrovsk Medical Academy of the Ministry of Health of Ukraine", Ukraine

The purpose of the study – investigation the separate joint lesion in systemic vasculitis, their X-ray sonographic characteristics, the correlation of the articular syndrome severity with extra-articular manifestations of the diseases, as well as aspects of the arthritis pathogenesis in this category of patients.

The study included 525 patients in the ratio of the examined with Henoch-Schonlen purpura, microscopic polyangiitis, cryoglobulinemic vasculitis, polyarteritis nodosa, Takayasu's arteritis, Wegener's granulomatosis with polyangiitis and eosinophilic granulomatosis with polyangiitis Churg-Strauss as a 7:4:3:1:1:1:1. Joint's damage in the form of arthritis or arthralgia observed in 32-67% different groups of patients, that depending on the disease duration, the degree of the pathological process's activity, extraarticular signs severity, lung parenchyma involving and hemodynamic status in the pulmonary circulation. The frequency of the certain bone lesions, existence of tenosynovitis and enthesopathies, X-ray sonographic signs of articular syndrome in different kind of vasculitis has its own gender dimorphism. The immune system malfunction, the rheological properties of blood and endothelial function of vessels collaborate in pathogenetic constructions of arthropathy. What is more, the high value of rheumatoid factor in blood associates with severe course of joint damage. Joint syndrome at different variants of systemic vasculitis is progressing in 1/3-2/3 of cases, this syndrome has definite features of clinical course and pathogenesis.

Keywords: vasculitis systemic, joints, clinic, pathogenesis.

РЕЗЮМЕ

ХАРАКТЕРИСТИКА СУСТАВНОГО СИНДРОМА ПРИ СИСТЕМНЫХ ВАСКУЛИТАХ

[1]Синяченко О.В., [2] Ханюков А.А., [2]Егудина Е.Д., [1]Такташов Г.С., [2]Гашинова Е.Ю.

[1]Донецкий национальний медицинский университет МЗ Украины, Лиман; [2]ГУ «Днепропетровская медицинская академия МОЗ Украины», Днепр, Украина

Целью исследования явилось изучение поражения отдельных суставов при системных васкулитах, их рентгеносонографических особенностей, взаимосвязи тяжести артикулярного синдрома с внесуставными проявлениями заболеваний, а также аспектов патогенеза артритов у данной категории больных. Под наблюдением находилось 525 больных. Соотношение числа обследованных с васкулитом Шенлайна-Геноха, микроскопическим полиангиитом, криоглобулинемическим васкулитом, узелковым полиартериитом, аортоартериитом Такаясу, гранулематозом с полиангиитом Вегенера и эозинофильным гранулематозным полиангиитом Черджа-Стросса составило 7:4:3:1:1:1:1. Поражение суставов в виде артрита или артралгий наблюдалось у 32-67% больных в разных группах. Тип поражения суставов при выше перечисленных заболеваниях зависел от длительности заболеваний, степени активности патологического процесса, тяжести экстраартикулярных признаков, вовлечения в процесс легочной паренхимы и состояния гемодинамики в малом круге кровообращения. Частота поражений отдельных костных сочленений, наличие тендовагинитов, энтезопатий и рентгеносонографических признаков суставного синдрома при разных васкулитах имеет свой гендерный диморфизм. В патогенетической этиологии артропатии участвуют нарушения иммунной системы, реологических свойств крови и эндотелиальной функции сосудов. Высокие уровни ревматоидного фактора в крови ассоциированы с риском развития тяжелого течения суставного синдрома.

Суставной синдром при разных вариантах системных васкулитов развивается в 1/3-2/3 случаев, имеет свои особенности патогенеза и клинического течения.

რეზიუმე

სახსროვანი სინდრომის დახასიათება სისტემური ვასკულიტების დროს

[1]ო. სინიაჩენკო, [2]ა. ხანიუკოვი, [2]ე. ეგუდინა, [1]გ. ტაკტაშოვი, [2]ე. გაშინოვა

[1]უკრაინის ჯანმრთელობის დაცვის სამინისტროს დონეცკის ეროვნული სამედიცინო უნივერსიტეტი, ლიმანი; [2]უკრაინის ჯანმრთელობის დაცვის სამინისტროს დნეპროპეტროვსკის სამედიცინო აკადემია, დნეპრი, უკრაინა

კვლევის მიზანს შეადგენდა სისტემური ვასკულიტების დროს ცალკეული სახსრის დაზიანების, მათი რენტგენოსონოგრაფიული თავისებურებების, არტიკულურ სინდრომსა და დაავადებების არასახსროვან გამოვლინებებს შორის ურთიერთკავშირის, ასევე, ამ კატეგორიის პაციენტებში ართრიტის პათოგენეზის ასპექტების შესწავლა. დაკვირვების ქვეშ იმყოფებოდა 525 პაციენტი.

გამოკვლეულ პაციენტთა თანაფარდობა შენ-

ლაინ-ჰენოხის ვასკულიტით, მიკროსკოპიული პოლიანგიიტით, კრიოგლობულინემიური ვასკულიტით, კვანძოვანი პოლიარტეტიიტით, ტაკაიასუს აორტოარტერიიტით, გრანულომატოზით ვეგენერის პოლიანგიიტით და ჩერჯ-სტროსის გრანულომატოზური პოლიანგიიტით შეადგენდა 7:4:3:1:1:1:1–ს. სახსრების დაზიანება ართრიტის, ან ართრალგიის სახით პაციენტთა სხვადასხვა ჯგუფში აღინიშნა შემთხვევათა 32-67%-ში. სახსრების დაზიანების ტიპი ზემოჩამოთვლილი დაავადებების დროს დამოკიდებული იყო დაავადების ხანგრძლივობაზე, პათოლოგიური პროცესის აქტივობის ხარისხზე, სახსარგარე ნიშნების სიმძიმეზე, პროცესში ფილტვის პარენქიმის ჩართულობასა და ჰემოდინამიკის მდგომარეობაზე სისხლის მიმოქცევის მცირე წრეში. ცალკეულ ძვალთშესახსრების დაზიანები სიხშირეს, ტენდოვაგინიტების, ენტეზოპათიების და სახსროვანი სინდრომის ნიშნების არსებობას სხვადასხვა ტიპის ვასკულიტის დროს ახასიათებს გენდერული დიმორფიზმი. ართროპათიის პათოგენეზურ ეტიოლოგიაში მონაწილეობს დარღვევები იმუნურ სისტემაში, სისხლის რეოლოგიურ თვისებებსა და სისხლძარღვების ენდოთელურ ფუნქციაში. სისხლში რევმატოიდული ფაქტორის მაღალი მაჩვენებლები ასოცირებულია სახსროვანი სინდრომის მძიმე მიმდინარეობის განვითარების რისკთან.

სახსროვანი სინდრომი სისტემური ვასკულიტის სხვადასხვა ვარიანტის დროს ვითარდება შემთხვევათა 1/3-2/3-ში, აქვს პათოგენეზის და კლინიკური მიმდინარეობის თავისებურებანი.

ASSESSMENT OF NEURODEVELOPMENTAL OUTCOMES IN INFANTS 6-12 MONTHS OF AGE ACCORDING TO IMPACT OF PERINATAL RISK FACTORS

[2]Tskimanauri N., [1]Khachapuridze N., [3]Imnadze P., [2]Chanadiri T., [1]Bakhtadze S.

[1]Tbilisi State Medical University; [2]Neurological Clinic LTD "Valeo", Tbilisi;
[3]I. Javakhishvili Tbilisi State University, Georgia

Infants exposed to some perinatal risk factors are at increased risk of neurodevelopmental problems throughout early childhood. Multiple risk factors are associated with increased risks of perinatal morbidity and mortality, however long term neurodevelopmental outcome of survivors is poorly described. According to literature data infants were significantly more likely to have neurodevelopmental impairment compared to healthy control infants, when assessed at 6-12 months and 2-3 years of age [1,12].

Studies have suggested that some early-childhood neurosensory and developmental outcomes for some groups of infants, who were born with some neonatal pathologies, such as extremely prematurity [4], neonatal infections and early-onset sepsis [3], hypoxic-ischemic encephalopathy, CNS malformation, intracranial hemorrhage [17] and others, thereafter were become critically ill during the neonatal period and were treated, but stayed at risk for adverse neurodevelopmental outcomes, have improved over the last decade. Even so it is not clear whether this trend applies to population of infants, who born healthy, but had been impacted by pathologies of pregnancy/delivery or other perinatal risks.

Authors have usually presented a single-center analysis, which have shown influence of separate factors identified as important risk factors. Major neurologic abnormalities, cognitive delays and impairs psychomotor development during the first year of life are presented in all cases of studied population [17]. There are identified significant correlations between these single risk factors, related to neonatal pathologies, and neurodevelopmental adversities. However, there are no large, recent analyses to examine whether neurodevelopmental outcomes improved, worsened, or remained the same for those vulnerable infants born healthy, but impacted by combination of factors and pathological conditions related to their development before birth.

Literature was reviewed the associations between some maternal pathological conditions exposure during pregnancy and labor with child neurodevelopment, which were stronger at neonatal period than at older ages. At the same time, information about the persistence of this association at later ages is limited.

The purpose of this research was: a. assessment of risk predictors for adverse neurodevelopmental outcome at age of 6 month and 12 month in divided groups of infants, partition of which had implemented by birth as healthy or with neonatal pathologies and b. to report developmental follow-up data from a case-control prospective study of infants exposed to separate and combination impact of risk factors.

Material and methods. Between January 2015 and December 2016, we prospectively enrolled 1018 live-born infants, information about which we had received from the medical reports of the participating clinics in Tbilisi (capital of Republic of Georgia) and Mtskheta, Dusheti (districts of Georgia), and included them in the study.

At the first stage of research it was conducted descriptive population-based prospective pilot study, as a result of which: