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NERVOUS SYSTEM LESION IN ANKYLOSING SPONDYLITIS, IN THE DISEASE BEGINNING IN CHILDHOOD AND ADULTHOOD

Summary. The incidence of ankylosing spondylitis (AS) in population amounts to 0.3 %, which is significantly more likely to develop at the age of 20–30 years. There are two forms of AS – juvenile and adults, depending on the age of the disease debut. The problem of juvenile AS (JAS) diagnostics is one of the most relevant in pediatric rheumatology, and the evolution of this disease remains unexplored in adulthood.

The aim of the study – to learn the frequency and nature of separate clinical signs of CNS and PNS lesions in patients with AS, and to evaluate their special features in the disease beginning in childhood and adulthood.

Materials and Methods. 217 patients with AS (193 males and 24 females) with an average age of 38 years were examined. The rapidly progression course is detected in 21 % of patients, moderate and high activity rate – in 79 %, stage II–III – in 82 %, polyarthritis – in 65 %. JAS was noted in 16 % of cases (all boys), in which stage III took place twice as often than other patients.

Results and Discussion. Changes in PNS are observed in 4.9 times more often among patients with JAS, and CNS is 2 times less often than in cases of the disease debut in adulthood, moreover among the patients of the 1st group, the severity of CNS disturbance is associated with the involvement of the cervical spine and the prevalence of spondylopathy, PNS is associated with the availability of tendovaginitis, arthritis of the "root joints" (shoulder, hip) and changes in the thoracic spine, while in the 2nd group it is associated with the parameters of the integral index of arthritis activity and the x-ray stage of the disease, with disturbance of "root" and sacroiliac joints, wherein the age of the disease onset affects the development of asthenic vegetative and corticonuclear syndrome, the emergence of radiculopathy, cervicocranialgia and metacarpal canal syndrome, and in the pathogenetic architectonics of the CNS pathology the level of immunoglobulin-A plays a greater role, and in PNS the serum interleukin 1-β contents, which, in addition, in cases of JAC determine the occurrence of cervicocranialgia, and in the remaining observations of AS – the Morton's metatarsalgia.

Conclusions. AS with different age of the disease debut is a risk factor for the development of certain symptoms of the CNS and PNS disturbances, which in these groups have their pathogenetic features.

Key words: ankylosing spondylitis; children; adults; nervous system; lesion; course.

INTRODUCTION The prevalence of ankylosing spondylitis (AS) reaches 0.3 % among the population [1, 2], and one of the most common manifestations of the disease is the central and peripheral nervous system lesions (CNS, PNS) [3, 4], which is primarily caused by the pathology of the spine [5]. The main causes of severe neuropathy in patients with AS are subluxation and micro fracture of the vertebrae [6, 7, 8], as well as demyelinating pathology [9]. It should be noted that for such patients there are violations of the nervous system vegetative link [10], which, first of all, affect its parasympathetic component [11]. Almost always AS is accompanied by psychosomatic disorders [12, 13], which, eventually, further exacerbates the poor quality of patients' life [14, 15]. Two forms of AS are identified – juvenile onset (JAC) and adult onset (AAC), depending on the age of the disease debut [16]. R. Conway and F. D. O'Shea [17] consider that these variants of the disease differ "like apples from oranges". An increasing research interest in the disease over recent years has led to a number of significant insights in AS. The differences between juvenile and adult-onset AS represents an ongoing controversy. It is still uncertain whether these 2 entities are distinct disease processes or different manifestations of the same disease modulated by age of onset. A number of groups have looked at this aspect of the disease using a variety of methodologies and publishing a number of contrasting findings [18].

The aim of the study – to learn the frequency and nature of separate clinical signs of CNS and PNS lesions in patients with AS, to assess their special features in disease that began in childhood and adulthood.

MATERIALS AND METHODS 217 AS patients aged 16 to 57 years old (an average of (37.7±0.64 years) were under observation, 88.9 % of men and 11.1 % of women among them. The disease duration averaged (10.5±0.39) years. I degree of AS activity was established in 21.2 % of cases, II – in 56.7 % and III – in 22.1 %, I stage was noted in 18.4 % of the surveyed patients', II – in 50.7% and III – in 30.9 %. Slowly progressive disease course occurred in 79.3 % of cases, fast-progressing – in 20.7 %. The so-called "central form" of AS was diagnosed in 33.6 % of cases, "rhizomelic" – in 2.8 %, "peripheral" – in 5.1 %, "undifferentiated" – 58.5 %. Ophthalmopathy (uveitis) and visceral changes are established in ¾ of the patients' number.

Peripheral arthritis was determined in 66.4 % of the patients, sarcoileitis – in 97.2 %, manifested spondylopathy – in 94.9 %. The parameters of the articular syndrome incidence were (5.4±0.25) r.u., its activity (DAS) – (4.0±0.10) r.u., the Lansbury index – (117.1±3.89) points, the index of arthritis progression – (1.3±0.15) r.u. The Barnett-Nordin meta-carpal index was (0.43±0.004) r.u., the bone mineral density index – (-1.49±0.055) SD. Enthesitis were noted in 38.7 % of the surveyed, tendovaginitis – in 28.6 %.

All patients were divided into two groups: the first (main) amounted to 35 (16.1 %) patients with JAS (all males with onset of the disease under the age of 18), and the others 182 (83.9 %), with AAS were included in the 2nd (control) group. The age of the main group representatives in the disease debut was (14.3±0.52) years, and at the time of the examination – (24.9±0.83) years, while in the control group it was (29.6±0.45) years and (40.2±0.58) years respectively. The duration of the disease in the 1st and 2nd groups did not differ. Both groups were approximately equal in the disease degree activity, but rapid progressive course of the disease in AAS was 2.7 times more common.

Patients underwent X-ray ("Multix-Compact-Sièmens", Germany) and ultrasound ("Envisor-Philips", the Netherlands and "ATL3500-Siemens", Germany) with a study of peripheral arthritis, spondylopathy and sacroileitis, and performed

dual-energy X-ray absortiometry of the proximal femur ("QDR-4500-Delphi-Hologic", USA), echocardiography (Acuson-Aspen-Siemens, Germany, Envisor-C-Philips, the Netherlands) and ultrasound doppler examination of vessels (angiographer "Aplia-XG-Toshiba", Japan). In the part of patients, electroneuromiography (Neuro-MVP-4, Russia) was performed with the estimation of the pulse velocity and potential amplitude, computerized (Somazom-Emotion-6-Siemens, Germany) and magnetic resonance ("Gygoscan-Intera-Philips", The Netherlands) tomography of the sacroiliac joints, spine and brain. The serum levels of antibodies to cyclic citrullinated peptide (aCCP), the concentration of interleukin (IL) 1β and tumor necrosis factor (TNF) α were studied using the enzyme immunoassay (PR2100 Sanofi diagnostic pasteur, France), and the immunobiochemical analyzer "Olympus-AU-640 "(Japan), concentrations of C-reactive protein (CRP), fibrinogen (FG), circulating immune complexes (CIC), immunoglobulins (Ig) A, G and M were determined in blood serum. The aCCP values in serum of the examined AS patients were 22.2±8.41 ± 0.57 U/ml, CRP – 12.0 ± 5.89 ± 0.40 mg/l, FG – 7.4 ± 2.99 ± 0.20 g/l, IgA – 2.3 ± 0.58 ± 0.04 mmol / L, IgG – 15.7 ± 3.23 ± 0.22 mmol/l, IgM – 2.0 ± 0.62 ± 0.04 mmol/l, CIC – 104.5 ± 61.08 ± 4.15 r.u., IL1β – 88.5 ± 100.17 ± 6.80 pg/ml, TNFα-153.5 ± 220.12 ± 14.94 pg/ml.

Statistical analysis of the research results was carried out by computer variations, nonparametric, correlation, regression, one (ANOVA) and multivariate (ANOVA/ MANOVA) variance analysis (Microsoft Excel and Statistica-Stat-Soft, USA). The mean values (M), their standard deviations (SD) and standard errors (m), Pearson parametric correlation coefficients (r) and nonparametric Kendall (τ), Brown-Forsythe dispersion criteria (BF), Wilcoxon-Rao (WR), the multiple regression (R), Student (t) and McNamara-Fisher, the reliability of statistical parameters (p).

RESULTS AND DISCUSSION The CNS lesion was diagnosed in 74.7 % of the patients with AS, PNS – 33.2 %, in patients with AAS twice as likely, and changes in PNS is 5 times less. It should be noted that PNS lesion occurred in all JAS patients. Among the patients with AS, dyscirculatory encephalopathy was detected in 54.4 % of cases, asthenic vegetative syndrome – in 39.2 %, corticonuclear syndrome – in 22.6 %, cortical ataxia – 19.4 %, peripheral polyneuropathy and pyramidal syndrome –18.0 %, cerebellar ataxia – 17.5 %, radiculopathy – 16.6 %, pseudobulbar syndrome – 14.3 %, cervicocranialgia – 13.8 %, Morton's metatarsalgia – 12.9 % , peripheral mononeuropathy – 10.6 %, metacarpal canal syndrome – 9.2 %, transient cerebrovascular disorders – 8.3 %, epileptiform syndrome – 7.8 %, neuralgia of the trigeminal nerve – 4.6 %, facial nerve neuritis – 3.7 %.

The pathology of the central nervous system (CNS) in the case of AAS is manifested with dyscirculatory encephalopathy in 72.8% of cases, with asthenic vegetative syndrome – in 52.5 %, with corticonuclear syndrome – in 30.3 %, with cortical ataxia – in 25.9 %, with pyramidal syndrome – in 24.1 %, with cerebellar ataxia – in 23.5 %, with pseudobulbar syndrome – in 19.1 %, with transient ischemic attack – in 11.1 %, with epileptiform syndrome – in 10.5 %, and the PNS lesion is revealed in 54.2 % of observations like peripheral polyneuropathy, in 50.0 % – radiculopathy, in 41.7 % – cervicocranialgia, in 38.9 % – Moroton's metatarsalgy, in 32.0 % – peripheral mononeuropathy, in 27.8 % – metacarpal canal syndrome, in 13.9 % – neuralgia of the trigeminal nerve, in 11.1 % – facial nerve neuritis.

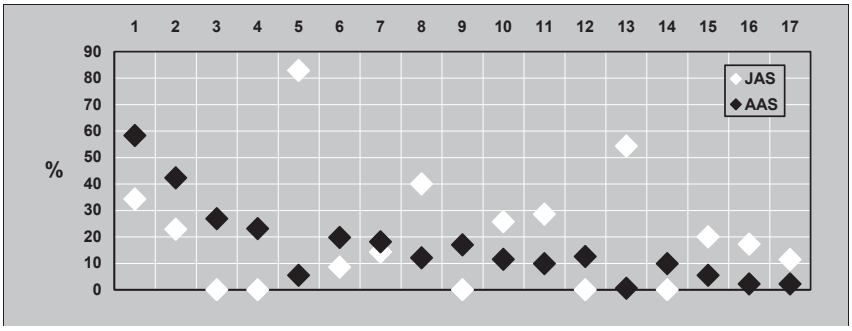


Fig. 1. The incidence of individual clinical signs of nervous system lesion in patients with JAS and AAS

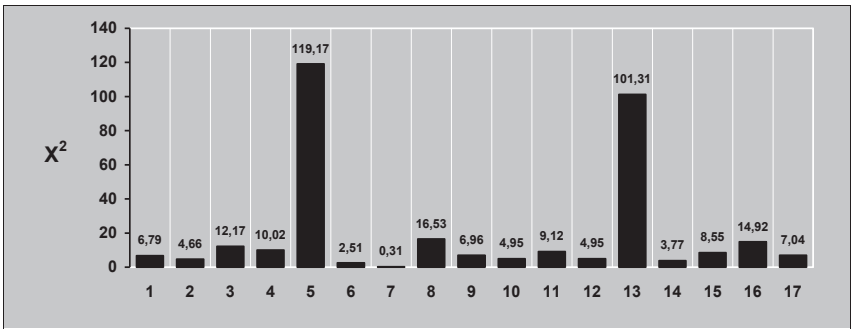


Fig. 2. Difference in frequency of individual signs of nervous system lesion in patients with JAS and AAS

1 – dyscirculatory encephalopathy, 2 – asthenic vegetative syndrome, 3 – corticonuclear syndrome, 4 – cortical ataxia 5 – peripheral polyneuropathy, 6 – pyramidal syndrome, 7 – cerebellar ataxia, 8 – radiculopathy, 9 – pseudobulbar syndrome, 10 – cervicocranialgia, 11 – Morton's metatarsalgia, 12 – peripheral mononeuropathy, 13- metacarpal canal syndrome, 14 – transient cerebrovascular disorders, 15 – epileptiform syndrome, 16 - neuralgia of the trigeminal nerve, 17 – facial nerve neuritis

Such signs of nervous system lesion as peripheral mononeuropathy, corticonuclear syndrome, cortical ataxia, pseudobulbar syndrome, and transient ischemic attack were not observed in JAS, and dyscirculatory encephalopathy was observed significantly less frequently by 70 %, and asthenic vegetative syndrome – by 85 %. Meanwhile, disease beginning in childhood is marked by peripheral polyneuropathy, radiculopathy, cervicocranialgia, Morton's metatarsalgia, metacarpal canal syndrome, neuralgia of the trigeminal nerve, facial nerve neuritis, and epileptiform syndrome, which were observed more often than in AAS, respectively, in 15,1; 3,3; 2,2; 2,9; 90,5; 7,8; 5,2 and 3,6 times, as shown by McNemar-Fisher's analysis and reflected in Fig. 1 and 2.

According to Wilcoxon-Rao's multivariate dispersion analysis, the age of patients in the disease debut is influenced on integral signs of CNS changes. We selected those signs of AS clinical course that simultaneously had Brown-Forsythe dispersion relations and Kendall's correlation with the severity of the nervous system lesion. It turned out to have existed a direct relationship between the severity of the cervical spine lesion and the prevalence of spondylopathy in cases of JAS, and with the DAS parameter and the involvement of the "root" (shoulder, hip) joints in cases of AAS.

According to Wilcoxon-Rao analysis, the age of AS patients at the onset of the disease affects the integral clinical signs of PNS. In the main group, there is a direct correlation between the severity of changes in PNS with tendovaginitis and involvement the thoracic spine, and in the control group – with the AS stage, the presence of the coxitis and the severity of sacroileitis.

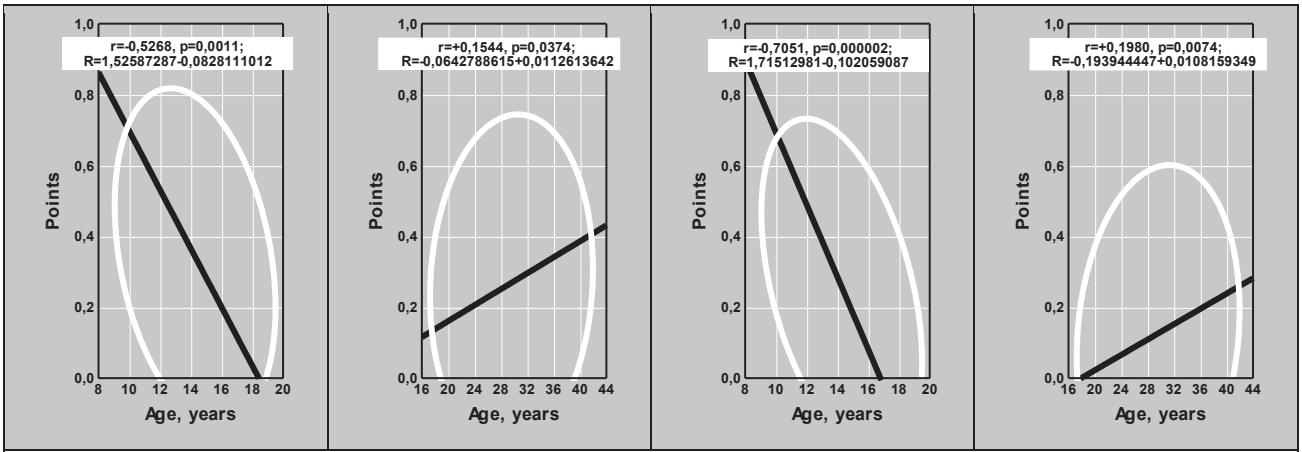
The age of the disease debut in the main group correlates inversely with the severity of the dyscirculatory encephalopathy, asthenic vegetative and epileptiform syndrome, and in the control one – directly correlates with the corticonuclear syndrome and has negative correlation relations with the epileptiform syndrome and cerebellar ataxia, as shown by the Kendall and Pearson correlation analyzes (Fig. 3–6).

Both in the first and in the second patients' groups with AS there are direct correlations the severity of radiculopathy with the age of patients, and the inverse ones – with cervicocranialgia. In addition, there are multi-directional reliable relationships in JAS patients with the facial nerve neuritis (direct relation) and metacarpal canal syndrome (inverse relation). Based on the survey findings, we made conclusions with a definite practical orientation: the debut of the disease under the age of 11 (<M-SD in the patients of the main group) is a risk factor for the development of asthenic vegetative syndrome, cervicocranialgia and metacarpal canal syndrome, and the onset of the disease is above 35 years of age (> M + SD in the control group) – a prognostic negative sign in the occurrence of corticonuclear syndrome and radiculopathy.

As shown by Brown-Forsyth's dispersion analysis, in the case of JAS, the beginning of asthenic vegetative syndrome is influenced by the DAS parameters, the pyramidal syndrome – by the prevalence of spondylopathy, cerebellar ataxia – by the stage of the pathological process, peripheral polyneuropathy – by the presence of tendovaginitis in patients, cervicocranialgia – by the cervical spine lesion, metacarpal canal syndrome – by the degree of disease activity. In cases of AAS, the development of dyscirculatory encephalopathy is closely related to enthesitis, pseudobulbar syndrome – with the "root" joints lesion, pyramidal syndrome – with changes in the cervical spine, cortical ataxia and metacarpal canal syndrome – with DAS indices, radiculopathy – with AS stage.

Regardless of the AS variant and the nature of the nervous system lesion, there is a dispersion relation between the severity of the CNS, PNS pathology and IgA, CIC levels in blood. In addition, the severity of the central nervous system lesion in the patients of the main and control groups is closely related to aCCP serum level, and in the case of AAS, as is also to fibrinogenemia level. The severity of CNS lesion in patients with JAS is directly correlated with level of IgA and TNFα in blood, and PNS – with IL1β. In cases of AAS, there are inverse correlations between the severity of CNS changes and immunoglobulinemia, CIC and IL1β parameters, and it is observed a direct correlation with TNFα values. It should be emphasized that the severity of PNS lesion in patients with AAS is inverse correlated with the concentration of CIC in blood and directly from IL1β.

Taking into account the performed statistical data results of the survey, the prognostic positive sign in regard to the severity of the CNS damage is the level of IgA <2 mmol / L



Correlation-regression Pearson's relations of patients' age in the disease debut with clinical manifestations of the nervous system lesion.

CNS		PNS	
Fig.3.Relation with asthenic vegetative syndrome in JAS	Fig. 4. Relation with corticonuclear syndrome in AAS	Fig. 5. Relation with cervicocranialgia in JAS	Fig. 6. Relation with radiculopathy in AAS

(<M-SD of this main group) in patients with JAS and the serum level of IgA> 3 mmol /l in AAS (> M + SD control group).

The next stage of our research was an assessment of the immune disorders role in the pathogenetic constructions of CNS and PNS lesion in patients with JAS and AAS. For this purpose, we selected those indicators that simultaneously had significant Brown-Forsythe dispersion relations and Kendall correlation. It turned out that the development of dyscirculatory encephalopathy directly depends on the level of CIC, pyramidal syndrome – on CRP, peripheral polyneuropathy – on IL1β, cervicocranialgia – on IgA, the metacarpal canal syndrome – on TNFα, Morton's metatarsalgia – on FG. There were no such dependencies in patients with AAS, but there were completely different ones. Thus, corticonuclear syndrome and the development of mono- and polyneuropathy were associated with the level of CIC, pseudobulbar syndrome – with IgM, the severity of radiculopathy – with CRP, Morton's metatarsalgia – with IL1β. These data once again point to the completely polysemantic manifestations of the AS with the debut of the disease in childhood and adulthood.

CONCLUSIONS Changes in PNS are observed 4.9 times more often in patients with JAS, and CNS lesion is 2 times less likely than in cases of the disease debut in adulthood, while in patients with JAS the severity of CNS damage is associated with the involvement of the cervical spine and the abundance of spondylopathy, PNS – with the presence of tendovaginitis, arthritis of the "root joints" (shoulder, pelvic) and changes in the thoracic spine, whereas in the case of AAS, PNS lesion depends on DAS parameters, the X-ray stage of the disease, the involvement of the sacroiliac joints, and the age of the disease onset affects the development of asthenic vegetative and corticonuclear syndromes, the occurrence of radiculopathy, cervicocranialgia and metacarpal canal syndrome. And IgA level is more involved in the pathogenetic constructions of the CNS pathology, and the level of IL1β in blood – in PNS, moreover, IL1β and IgA levels determine the occurrence of cervicocranialgia and Morton's metatarsalgia in other observations of the AS.

Conflict of interest The authors declare that there is no conflict of interest, while the authors did not receive from the individuals and organizations financial support for research, fees, and other forms of rewards.

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УРАЖЕННЯ НЕРВОВОЇ СИСТЕМИ ПРИ АНКІЛОЗИВНОМУ СПОНДИЛІТІ, ЩО ПОЧАВСЯ У ДИТЯЧОМУ ТА ДОРОСЛОМУ ВІЦІ

Резюме. Поширеність анкілозивного спондиліту (АС) серед населення сягає 0,3 %, що значно частіше розвивається у віці 20–30 років. Виділяють дві форми АС – дитячу і дорослу, залежно від віку дебюту захворювання. Проблема діагностики ювенільного АС (ЮАС) належить до найактуальніших у педіатричній ревматології, при цьому еволюція такого захворювання в дорослому віці залишається не вивченою. Існує віковий диморфізм уражень центральної нервової системи (ЦНС) і периферійної (ПНС), але у хворих на ЮАС та в решти випадків він не з'ясований.
Мета дослідження – вивчити у хворих на АС частоту й характер окремих клінічних ознак уражень ЦНС та ПНС, оцінити їх особливості при захворюванні, що почалося в дитячому й дорослому віці.
Матеріали і методи. Обстежено 217 хворих на АС (193 чоловіки і 24 жінки) середнього віку – 38 років. Швидкопрогресуючий перебіг констатовано в 21 % спостережень, помірний і високий ступені активності – в 79 %, II–III стадії – у 82 %, поліартрит – в 65 %. ЮАС діагностовано в 16 % випадків (усі хлопчики), в яких III стадія мала місце удвічі частіше, ніж в інших пацієнтів.
Результати досліджень та їх обговорення. Зміни ПНС спостерігають у 4,9 раза частіше у хворих на ЮАС, а ЦНС – в 2 рази рідше, ніж у випадках дебюту захворювання в дорослому віці. При цьому в пацієнтів першої групи тяжкість ураження ЦНС пов'язана із залученням до процесу шийного відділу хребта і з поширенням спондилопатії, ПНС – з наявністю тендовагінітів, артриту кореневих суглобів (плечових, кульшових) і змін грудного відділу хребта, тоді як в другій групі – з параметрами інтегрального індексу активності артриту та рентгенологічної стадії хвороби, з ураженням кореневих й крижоздухвинних зчленувань, причому вік початку захворювання впливає на розвиток астеновегетативного і кортиконуклеарного синдромів, виникнення радикулопатії, цервікокраніалгії та синдрому метакарпального каналу, а в патогенетичних побудовах патології ЦНС більшою мірою бере участь рівень у крові імуноглобуліну-А, ПНС – вміст сироваткового інтерлейкіну-1β, який, окрім того, при ЮАС визначає появу цервікокраніалгії, а в інших спостереженнях АС – мортонівської метатарзалгії.
Висновки. АС з різним віковим дебютом захворювання є чинником ризику розвитку окремих ознак ураження ЦНС та ПНС, які в цих групах мають свої патогенетичні особливості.

Ключові слова: спондиліт анкілозивний; діти; дорослі; нервова система; ураження; перебіг.

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ПОРАЖЕНИЕ НЕРВНОЙ СИСТЕМЫ ПРИ АНКИЛОЗИРУЮЩЕМ СПОНДИЛИТЕ, НАЧАВШЕМСЯ В ДЕТСКОМ И ВЗРОСЛОМ ВОЗРАСТЕ

Резюме. Распространенность анкилозирующего спондилита (АС) среди населения достигает 0,3 %, что значительно чаще развивается в возрасте 20–30 лет. Выделяют две формы АС – детскую и взрослую, в зависимости от возраста дебюта заболевания. Проблема диагностики ювенильного АС (ЮАС) относится к наиболее актуальным в педиатрической ревматологии, при этом эволюция такого заболевания во взрослом возрасте остается не изученной. Существует возрастной диморфизм поражений центральной нервной системы (ЦНС) и периферической (ПНС), но у больных ЮАС и в остальных случаях он не выяснен.
Цель исследования – изучить у больных АС частоту и характер отдельных клинических признаков поражений ЦНС и ПНС, оценить их особенности при заболевании, начавшемся в детском и взрослом возрасте.
Материалы и методы. Обследованы 217 больных АС (193 мужчины и 24 женщины) среднего возраста – 38 лет. Быстропрогрессирующее течение констатировано в 21 % наблюдений, умеренная и высокая степень активности – в 79 %, II–III стадии – в 82 %, полиартрит – в 65 %. ЮАС отмечен в 16 % случаев (все мальчики), у которых III стадия имела место вдвое чаще, чем в остальных пациентов.
Результаты исследований и их обсуждение. Изменения ПНС наблюдаются в 4,9 раза чаще у больных ЮАС, а ЦНС – в 2 раза реже, чем в случаях дебюта заболевания во взрослом возрасте, при этом у пациентов первой группы тяжесть поражения ЦНС связана с вовлечением в процесс шейного отдела позвоночника и с распространенностью спондилопатии, ПНС – с наличием тендовагинитов, артрита корневых суставов (плечевых, тазобедренных) и изменений грудного отдела позвоночника, тогда как во второй группе – с параметрами интегрального индекса активности артрита и рентгенологической стадии болезни, с поражением корневых и крестцовоподвздошных сочленений, причем возраст начала заболевания влияет на развитие астеновегетативного и кортиконуклеарного синдромов, возникновение радикулопатии, цервикокраниалгии и синдрома метакарпального канала, а в патогенетических построениях патологии ЦНС в большей степени участвует уровень в крови иммуноглобулина-А, ПНС – содержание сывороточного интерлейкина-1β, которые, кроме того, при ЮАС определяют появление цервикокраниалгии, а в остальных наблюдениях АС – мортоновской метатарзалгии.
Выводы. АС с разным возрастным дебютом заболевания является фактором риска развития отдельных признаков поражения ЦНС и ПНС, которые в этих группах имеют свои патогенетические особенности.

Ключевые слова: спондилит анкилозирующий; дети; взрослые; нервная система; поражение; течение.