операционного периода с акцентированием внимания на необходимости рентгенологического контроля и измерения расстояния миграции корней после оперативного вмешательства. Возникновение сенсорных нарушений после коронектомии 3НМ наблюдалось при С2-С3 положении по классификации G.Pell, B.Gregory (1933) и при мезиоангулярному и вертикальном положении по классификации G.Winter (1926). Среднее арифметическое значение расстояния миграции корней в группе наблюдения составило 3,6±1,69 мм за 12 месяцев послеоперационного периода. Оптимальным сроком повторного оперативного вмешательства с целью удаления корней является интервал от 12 до 24 месяцев после коронектомии ОНР.

Ключевые слова: нижние третьи моляры, коронектомия, рентгенодиагностика, миграция корней, послеоперационный период.

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period features, focusing on the need for radiological monitoring and measuring the distance of the roots migration after surgery. The occurrence of sensory impairments after coronectomy of 3NM was observed at the C2-C3 position according to the G.Pell, B.Gregory classification (1933) and at the mesio-angular and vertical position according to the G.Winter classification (1926). The arithmetic mean of the distance of migration of roots in the observation group was 3.6 ± 1.69 mm over 12 months of the postoperative period. The optimal period of reoperation for root removal is the interval from 12 to 24 months after the coronectomy of the OHP.

Key words: lower third molars, coronectomy, X-ray, root migration, postoperative period.

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N.I. Tokarchuk, Y.V. Vyzhga, V.T. Tokarchuk
National Pirogov Memorial Medical University, Vinnytsia

ROLE OF THE FIBROTIC MARKERS FOR PYELONEPHRITIS IN INFANTS

e-mail: nadia_tokarchuk@ukr.net

The study was aimed at estimation of the transforming growth factor ß1 and monocytic chemo attractant protein type I in infants with pyelonephritis. Pyelonephritis against the background of vesicoureteral reflux is followed by high activity of the inflammatory process with increased amount of MCP-1 in serum that by 2.5 times exceeds the results of the kids with primary pyelonephritis. During the study we evaluated high results of the profibrotic marker TGF B1 against the background of vesicoureteral reflux in infants that is by 2.8 times higher than results of the kids with primary pyelonephritis. The article is dedicated to the study of fibrotic markers, polymorphic variants of gene TGF- ß1 in positions -509CT and +869CT in pyelonephritis against the background of vesicoueretral reflux in infants. Children that found to be a careers of genotype C-509C and T+869T gene of transforming growth factor B1 have more severe course of the disease and hyperproduction of serum TGF-B1 than heterozygote and homozygote T-509T and C+869C.

Key words: pyelonephritis, transforming growth factor B1, monocytic chemo attractive protein-1, gene polymorphism of transforming growth factor B1.

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A lot of studies present that reasons of the pyelonephritis development in infants had already changed [4]. We should admit, that development of the pyelonephritis in infants is common at the background of urinary tract malformations, one of the most important is vesicoureteral reflux (VUR) that influence on development of fibrotic changes in kidney structure [10]. The highest risk of the kidney sclerosis formation is observed during the first year of life is 40% [1].

It's known that pathogenesis of kidney parenchyma injury in case of vesicoureteral reflux is complicated and has plural sides. Rising of the blood pressure in kidney pelvis leads to activation of channels cells and vascular endothelium, influence on hyperproduction of inflammatory mediators, that supply monocytes and leucocytes migration into injured region with formation of the inflammatory changed infiltrate tissue. Together with early cytokines (interleukin 1, 6, 8) proliferative cytokines are synthesized with origination of transforming growth factor ß1 (TGF-ß1). The latest study data shows its role on formation and progress of the fibrotic changes of the tissues [9]. TGF- β, as a fibrogenic cytokine, stimulates changes of the kidney parenchyma and its remodeling [14].

Nowadays it is known that diseases develop in case of unfavorable combination of the polymorphic genes. Highly illuminated the latest updates concerning connection between vesicoureteral reflux and gene TGF-ß1 polymorphism (genotype -509 CC and +869 TT), that allows estimate aptitude and improve diagnostic of vesicoureteral reflux, especially in infants [8].

As well important problem devoted to optimization of the diagnostic of inflammatory activity in case of pyelonephritis in infants, cause standart inflammatory markers are negative, or doubtful in results and meanings [15]. Due to all presented it's a perspective to search for markers that may in a short period of time at the debut of the disease contribute inflammatory activity. Though one of the most perspective of them is monocytic chemo attractive protein of the 1 type (MCP-1) that presents the highest chemotaxic

effect to monocytes and T-lymphocytes. With an overproduction of the substance connect such pathophysiological conditions as interleukin synthesis increase, adhesive molecules and prostaglandins by mesangial cells, thickening of the basal membrane of glomerular system and damage of the glomerular hemodynamic [5]. MCP- 1 is not just chemo attractant that provides migration of the mononuclear cells into the pull of the inflammation, but inflammatory mediator by itself [7].

As well MCP-1 potentiate fibrotic changes in kidney parenchyma [15]. According to literature data that presents role of MCP-1 as marker initiator of inflammatory response in kidneys and following supporter of the process, its actual to contribute further investigations of it in infants with pyelonephritis.

The purpose of the study was estimation of the transforming growth factor ß1 and monocytic chemo attractant protein type I in infants with pyelonephritis.

Material and methods. We have carried out comprehensive clinical and laboratory-instrumental studies of 150 children at the age from 1 month up to 3 years: 50 of them with pyelonephritis at the background of vesicoureteral reflux (main group), 50 of them without signs of reflux (comparative group). Inclusion criteria were: presence of the pyelonephritis, vesicoureteral reflux, age from 1 month to 3 years. Exclusion criteria were: presence of genetic or chromosomal pathology, congenital defects except kidney pathology, presence of the chronic kidney disease, kidney failure, surgical correction of the vesicoureteral reflux.

We used random method and description methods of the study. Randomization was provided in blocks for 4 patients to achieve adequate contribution of the patients in a groups.

Group of control was presented by 50 healthy children. Laboratory investigations were presented by general clinical studies: complete blood count, complete urine test, bacteriological urine study, vaginal swap for girls, feces study, some of biochemical studies – creatinine, urea, and instrumental methods – USS of abdomen, kidney scan, cystography , excretory urography.

As well we used special methods – estimation of the TGF-ß1 (immune enzyme method (ELISA) with lab kits «TGF- ß1» (Biosourse, EuropeS.A.), monocytic chemo attractant protein-1 (immune enzyme method with lab kits «Human MCP-1» (PlatinumELISA; BMS 281, BengerMedSystems, Austria). Molecular-genetic study was done by genotype checking for polymorphism C>T in position -509 and position +869 T>C of transforming growth factor gene B1 (C-508T and T+869C) by polymerase link reaction in a real time with the next analyze of the length of restrictive fragments during their separation with electrophoresis in 2,5% agar gel.

Evaluation of the results: results were statistically proceeded with computer programs «STATISTICA» forWindows 8.0.0. (SPSSI.N.C.; 1989-1997), «STATISTICAV.10.0» (StatSoftInc; 1984-1996), «MicrosoftExcel».

Results of the study and their discussion. We analyzed influence of VUR on stage of activity of inflammatory response in case of pyelonephritis. So, we found that infants with VUR and higher possibility to receive III stage of the inflammatory activity (40%) to compare with I (28%) and II (32%) stages of the activity (OR=4.8, S=0.4, 95% CI 1.89–12.37), $p<0.05$, that indicates more severe currency of the disease in this group. Though in primary pyelonephritis is more often to meet I stage of activity (OR= 5.7, S=0.46, 95% CI 2.32 – 14.43) and II (OR= 2.07, S=0.4, 95% CI 1.63– 4.61).

We should admit that standard markers of inflammatory activity are low specific for both primary and secondary pyelonephritis. Specific of leucocytosis in secondary pyelonephritis Sp=0.4 (LR+1.05,+PV=51%,-PV=49%) and in primary it was - Sp=0,56 (LR+1.12,+PV=40%, -PV=44%, index Kappa = -0.02). Specific of erythrocyte sedimentation rate was as well low in children of the main group (Sp=0.58; LR+1.32,+PV=44%, -PV=46%, index Kappa =-0.08), as well comparative group (Sp=0.6; LR+1.52,+PV=55%, -PV=53%, index Kappa = 0.08). Specific of C-reactive protein was higher a little – main group – Sp=0.66; LR+1.72, +PV=51%, -PV=50%, index Kappa = 0.02 and comparative group – Sp=0.64; LR+1.52, +PV=25%, -PV=42%, index Kappa = -0.024), $p>0.05$.

All the written above influence further evaluation of the more adequate diagnostic methods of the inflammatory activity in case of pyelonephritis. That is why we checked amount of monocytic chemo attractant protein-1 in all children. We figured out that in children of the main group amount of MCP-1 in serum was (474.6±114.37 pg/ml) [95% CI, 426.45-503.15], that was significantly higher than in children from the comparative group (384.51±106.78 pg/ml) [95% CI, 223.45 – 468.52] and in a healthy children (121.1±35.09 pg/мml) [95% CI, 89.87-151.52], ($p<0.001$).

We found that with rising of the inflammatory activity in case of pyelonephritis content of MCP-1 in serum was higher. The same changes were common for primary pyelonephritis as well, but with significantly lower amount to compare with values of the main group of children, $p<0.05$.

As well we estimated high sensitivity (85%) and specific (78%) prognostic value of the positive (+PV) 0.72 and negative tests (-PV) 0.36 of MCP-1 in main group of the children.

Correlation that was provided through the results confirmed pathogenic connection between signs of activity of inflammatory response in infants with pyelonephritis. So, we figured out moderate and severe positive correlation between MCP-1 and CRP (r_{xy}= 0.66; p<0.01) in infants with pyelonephritis.

Taking into account profibrotic characteristics of the inflammatory response in case of pyelonephritis we evaluated checking of the TGF-ß1, as a marker of fibrotic predictor in kidneys. Results presented significantly higher content of TGF-ß1 in children with pyelonephritis at the background of VUR (8.72±0.94 ng/ml) [95% CI, 7.16-10.28] to compare with children that had primary pyelonephritis (5.67± 0.65 ng/ml) [95% CI, 3.42-7.92], p<0.05. At the same time through all the kids TGF- ß1 was significantly lower (1.51± 0.82 ng/ml) [95% CI, 0.65-2.37] to compare with results in primary and secondary pyelonephritis cases, p<0.01.

The results we got confirm that rising of the inflammatory activity in kidneys is followed by higher signs of fibrosis. So, in case of III of inflammatory activity of pyelonephritis we got significantly higher amount of TGF- ß1 (9.23±1.48ng/ml, [CI, 6.83-11.38]), to compare with II (8.22±1.26 ng/ml [95% CI, 7.03-9.78]) and I stage of activity (4.63±0.49 ng/ml [95% CI, 2.83-6.45]), p<0.05.

We should admit that children of the main group with III stage of the activity had TGF- ß1 highest sensitivity (89%) and specific (81%), prognostic positive value ((+PV) 0.84) and negative ((-PV) 0.35) results, diagnostic meaning (0.92) and index Cohen's kappa (K) 0.90). Such results present agreement between studied marker in case of inflammatory response and prove necessity of its estimation in children with severe pyelonephritis to estimate fibrotic processes.

Such results as well indicate increased influence of the profibrotic marker with the duration of the disease. So, children that had inflammatory process more than 6 months got TGF- ß1 (9.02±2.04 ng/ml) [95% CI, 6.98-11.06] significantly higher than kids with the duration of the disease less than 3 months, (p<0.05).

The lowest result of the studied marker was at the beginning of the disease (TGF- ß1 - (4.01±1.03 ng/ml) [95% CI, 2.98-5.04], (p<0.01).

As well severe correlation between TGF-B1 and duration of the inflammation process in case of secondary pyelonephritis (r_{xy}= 0.78) [95% CI, 0.63-0.88], p <0,001. Direct severe correlation was found between TGF- ß1 and MCP-1 (r_{xy}= 0.86), [95% CI, 0.78-0.94], p <0.001.

To evaluate genetic factors that influence on development of the irreversible changes of the kidneys we estimated polymorphism of TGF-ß1 gene in position -509 and +869 in our patients.

Characteristic of the genotypes of polymorphic variant -509 gene TGF – B1, in patients with pyelonephritis confirmed that in a structure of the disease homozygote of the mutant allele C were dominant and their part was significantly higher that frequency of the homozygote T allele (p<0.05).

To compare frequency of the allele between children of the main group and group of comparison we found that children with primary pyelonephritis, capacity of minor allele T was higher than in cohort of children with secondary pyelonephritis (64% against 25%, p<0.05). Frequency of major allele C was higher in a main group than in comparative group (74% and 35%, p<0.05).

Analysis of the frequent spreading of the allele in children of the main and comparative groups showed that patients with secondary pyelonephritis had lower frequency of the minor allele C to compare with cohort of primary pyelonephritis (32% and 62%, p<0.05). At the same time, frequency of the major allele T was higher in a main group to compare with comparative group (67 % against 38%, p<0.05).

Presence of the genotype C-509C TGF – B1 gene polymorphism in patients with secondary pyelonephritis significantly increases risk of the disease development in 5.56 times to compare with genotype CT (OR=5.56, S=0.69, 95% CI:1.92 – 13.23). We found that genotype CC significantly increases risk of the pyelonephritis development by 2.52 times compared to TT genotype (OR=2.52, S=0.66, 95% CI: 0.98 – 10.6). Presence of the TT-509 genotype in secondary pyelonephritis significantly increases risk of the disease development – by 1.84 times (OR=1.84, S=0.61, 95% CI: 0.53 – 5.8). Figured out that careers of T+869T genotype of gene TGF – B1 polymorphism are in a risk for the development of the inflammatory process by 3.82 (OR=3.82, S=0.6995% CI: 1.43 – 12.01) times higher than children heterozygote C/T+869. Presence of CC+869 genotype significantly increases possibility of pyelonephritis development by 1.03 times (OR=1.03, S=0.58, 95% CI: 0.46 – 5.5).

We analyzed influence of the allele variants of polymorphic marker -509 gene TGF – B1 on the level of transforming growth factor B1. The majority of the kids of main group – homozygote of mutant allele C ((68%) 34/50 cases) – had significantly higher amount of TGF – B1 (11.15±2.24 ng/ml), than heterozygote ((14%) 7/50), that got amount of TGF – B1 8.16±1.98 ng/ml, p<0.05 and homozygote careers ((18%) (9/50)) of main allele T with amount of TGF – B1 (4.56±1.03 ng/ml, p<0.01). At the same time significantly lower meanings of TGF – B1 (3.12±0.98 ng/ml) were found through ((40%) (20/50)) of TT genotype careers of

comparative group kids than in homocareers allele C ((14%) (7/50)) that got amount TGF – B1 – 5.58±1.76 ng/ml ($p<0.05$), and in heterozygote (46%) (23/50) amount TGF – B1 – 4.56±1.56 ng/ml)**.**

The analyze of the average meanings of TGF-ß1 according to allele variants of polymorphic gene TGF – B1 marker in position +869 presented that careers of mutant allele T (58%) (29/50 persons) had significantly higher level of TGF-ß1 (8.62±2.31 ng/ml), than (26%) (13/50) of heterozygote (amount TGF – B1 was 7.03±1.92 ng/ml, $p<0.05$) and (16%) (8/50) of homozygote careers allele C (TGF – B1 – 5.74±1.71 ng/ml), $p<0.05$. Presented results shows ability of fibrotic damage in kidneys of the children that are C-509C and T+869T genotypes careers.

According to the last epidemiological issues, done by European authors, frequency of pyelonephritis in pediatric population various from 0.4 to 5.4% [7]. As clinical studies demonstrate special attention should be paid to infants, due to complicated diagnostic of the disease and difficulties with complete treatment [6].

Scientists try to solve the problem from the different positions – by finding pathogenic mechanisms of development, progression and becoming chronic pyelonephritis. A lot of modern scientists concern pyelonephritis to be immune pathological process [5]. As well, they evaluate role of a separate reasons from the organism such as functional and organic obstruction of the urinary tract, metabolic disorders, risk factors, etc [11].

It is already approved that pyelonephritis in children has mainly secondary character (95-96%), and can be a complication of different clinical variants of urine transportation disorders, that can cause its chronic character with frequent episodes of relapsing inflammatory processes and long treatment periods. The most common reason of the secondary pyelonephritis is vesicoureteral reflux. Combination of the inflammatory process and vesicouretheral reflux can influence on fibrotic transformation of kidney parenchyma in infants with pyelonephritis [12]. But discussions concerning pathogenesis of the sclerotic changes of kidney parenchyma in children with vesicouretheral reflux are still ongoing. From one side, its doubtless that bacterial pyelonephritis plays a leading role in fibrosis, but yet, acute inflammatory response leads to nephrosclerosis not in all clinical cases.

Presence of the vesicouretheral reflux and intensity of the morphological changes correlates strictly. As well it's a correlation between interstitional fibrosis and vesicouretheral reflux and urinary tract infection: with a higher stage of reflux, would be the highest impact of the urinary tract infection on nephrosclerosis. Persistent vesicouretheral reflux leads to destruction and wrinkling of the kidney. Its known that nephrosclerosis is mainly common for children before 5 years old, but for elder group is more common antireflux mechanism, connected with prolongation of the intramural part of the urethra, that lead to decreased sensitivity of the kidney parenchyma towards plural infection agents [13].

Scientists insist on statement, that urinary tract infection is one of the main reasons that can lead to infiltration of kidney parenchyma with inflammatory cells with the further production of inflammatory mediators and fibrogenesis. Children with relapsing pyelonephritis have increased production of the inflammatory cytokines. This fact influences on development of more intense morphological and functional tubular and parenchyma changes in kidneys. All the next episodes of the active disease spread zone of nephrosclerosis with fibrotic transformation [15].

Literature data indicates on important role of the chemokines in development and support of the inflammatory process in kidneys. Pathogenesis of the tubular and parenchyma changes lay on couple of the mechanisms such as proteinuria, tubular ischemia, hypoxia, influence of protein and enzyme factors, cytokines, growth factors, etc. Any episode of the parenchyma damage leads to secretion of inflammatory mediators [12].

Biosynthesis of the MCP-1 in patients with kidney pathology increases under the influence of inflammatory cytokines, that leads to monocytic infiltration of glomerules. The latest data confirm that using of the MCP-1 as objective criteria of inflammatory process activity in patients with kidney diseases is reasonable [14].

Plural scientific studies demonstrate that synthesis of the proliferative cytokines, such as transforming growth factor B1, due to increased pressure at the background of vesicouretheral reflux. TGF-ß1 – polyfunctional cytokine that plays role in cell proliferation mechanisms, their differentiation, migration, apoptosis, as well as separate metabolic reactions in target cells. Cells demonstrate specific receptors towards this cytokines, that usually stays in a latent form. TGF-ß1 contributes towards nephrosclerosis progression. Its known that TGF-ß1 takes part in remodeling of kidney parenchyma due to activation of smooth muscle proliferation in kidney arteries. Increased level of TGF-ß1 in serum is a marker of the kidney fibrosis. TGF- β as a fibrotic cytokine stimulates changes of the kidney structure [13].

The same important nowadays single studies dedicated estimation of the correlation between vesicouretheral reflux and gene TGF-ß1 polymorphism (genotype -509 CC and +869 TT), that allows

evaluate predisposition and improve diagnostic of the pathology in infants. Study demonstrates that variability of TGF-ß1 gene is a marker of vesicouretheral reflux and influence kidney pathology development, course of the disease.

So, literature data indicates necessity of the complex patients investigation in case of pyelonephritis. As well dynamic observance should be provided for children with vesicouretheral reflux with evaluation of the profibrotic markers.

Conclusions

1. Pyelonephritis at the background of vesicoureteral reflux is followed by high activity of the inflammatory process with increasing of the amount of monocytic chemo attractive protein-1 in serum by 1.2 times higher than amount of the kids with primary pyelonephritis. Significant correlation between monocytic chemo attractive protein-1 and amount of CRP (rxy=0.66, p<0.01) in infants with pyelonephritis at the background of vesicoureteral reflux.

2. Pyelonephritis at the background of vesicoureteral reflux in infants is followe by rising up of transforming growth factor B1 in serum, that is by 1.53 times higher than results of the children with primary pyelonephritis. Profibrotic marker increased with worsening of the inflammatory activity of the disease.

3. Infants with pyelonephritis at the background of vesicoureteral reflux in case of C-509C та T+869T genotypes had high transforming growth factor B1 (65.02±6.74%) as well as high serum level of TGF-B,1 was estimated had increased possibility of the disease development by 4.48 times compared to genotype T-509T and C+869C by 3.03 times compared to genotype C-509T and T+869C.

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Реферати

РОЛЬ ФИБРОТИЧЕСКИХ МАРКЕРОВ ПРИ ПИЕЛОНЕФРИТЕ У ДЕТЕЙ РАННЕГО ВОЗРАСТА

Токарчук Н.И., Вижга Ю.В., Токарчук В.Т.

Целью работы было определение показателей трансформирующего фактора роста ß1 и моноцитарного хемоатрактантного протеина 1-го типа у детей раннего возраста, больных пиелонефритом. Пиелонефрит на фоне пузырно-мочеточникового рефлюкса сопровождается активностью воспалительного процесса с повышением уровня МХП – 1 в сироватке крови больных, что в 2,5 раза вище в сравнении с показателем детей с первичным пиелонефритом. При выполнении работы выявлено высокие

РОЛЬ ФІБРОТИЧНИХ МАРКЕРІВ ПРИ ПІЄЛОНЕФРИТІ У ДІТЕЙ РАННЬОГО ВІКУ

Токарчук Н.І., Вижга Ю.В., Токарчук В.Т.

Метою роботи було визначення показників трансформуючого фактора росту ß1 і моноцитарного хемоатрактантного протеїну 1-го типу у дітей раннього віку, хворих на пієлонефрит. Пієлонефрит на тлі міхурово-сечовідного рефлюксу супроводжується активністю запального процесу з підвищенням рівня МХП - 1 в сироватці крові хворих, що в 2,5 рази вище в порівнянні з показником дітей з первинним пієлонефритом. При виконанні роботи виявлено високі показники

показатели профибротического маркера TGF B1 при пузырно-мочеточниковом рефлюксе у детей, что в 2,8 раза выше в сравнении с показателем детей с первичным пиелонефритом. Статья посвящена исследованию маркеров фиброзообразования полиморфных вариантов гена TGF- ß1 у позициях -509CT и +869CT при пиелонефрите на фоне пузырно-мочеточникового рефлюкса у детей раннего возраста. Дети-носители генотипа C-509C и T+869T гена трансформирующего фактора роста B1 имеют тяжелее течение заболевания и гиперпродукцию сывороткового TGF-B1 чем гетерозиготы и гомозиготы T-509T та C+869C.

Ключевые слова: пиелонефрит, трансформирующий фактор роста B1, моноцитарный хемоаттрактантный протеин 1, полиморфизм гена трансформирующего фактора роста B1.

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профібротичного маркера TGF B1 при міхурово-сечовідному рефлюксі у дітей, що в 2,8 рази вище в порівнянні з показником у дітей з первинним пієлонефритом. Стаття присвячена дослідженню маркерів фіброзоутворення поліморфних варіантів гена TGF- ß1 у позиціях -509CT і + 869CT при пієлонефриті на тлі міхурово-сечовідного рефлюкса у дітей раннього віку. Діти-носії генотипу C-509C і T + 869T гена трансформуючого фактора росту B1 мають важчий перебіг захворювання і гиперпродукцию сироваткового TGF-B1 ніж гетерозиготи і гомозиготи T-509T та C + 869C.

Ключові слова: пієлонефрит, трансформуючий фактор росту B1, моноцитарний хемоаттрактантний протеїн 1, поліморфізм гена трансформуючого фактора росту B1.

Рецензент Крючко Т.О.

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М.А. Тріщинська, О.А. Галушко, В.О. Свистільник
Національна медична академія післядипломної освіти імені П.Л.Шупика, Київ

ОСОБЛИВОСТІ ПОКАЗНИКІВ ДОБОВОГО МОНІТОРИНГУ АРТЕРІАЛЬНОГО ТИСКУ У ОСІБ З ПОЧАТКОВИМИ ПРОЯВАМИ ХРОНІЧНОЇ ІШЕМІЇ ГОЛОВНОГО МОЗКУ

E-mail: docmarina99@gmail.com

Артеріальна гіпертензія (АГ) є одним із основних факторів ризику цереброваскулярних захворювань. Ураження церебральних судин відбувається протягом деякого часу, що залежить від ряду характеристик артеріального тиску (АТ), які не можливо виявити при звичайному вимірювання АТ. Метою нашого дослідження було вивчення характеристик АТ протягом доби (за даними ДМАТ) та їх прогностичне значення у хворих з ранніми проявами хронічної ішемії мозку (ХІМ). Нами було обстежено 116 осіб з початковими проявами хронічної ішемії мозку (ХІМ). Всім пацієнтам було проведене загально-клінічне, клініко-неврологічне, клініко-лабораторне, нейровізуалізаційне обстеження та добове моніторування АТ (ДМАТ). Була виявлена тенденція до відмінності між пацієнтами груп 1 та 2 (А і Б) за показниками добового індексу (ДІ) систолічного та діастолічного АТ — у осіб із структурними змінами на МРТ головного мозку судинного ґенезу (група 2) спостерігалося зменшення ДІ — недостатнє зниження АТ вночі. У 58,9 % пацієнтів, які були обстежені, швидкість наростання $АТ_{сист.}$ вранці була збільшена, величина ранкового підйому $АТ_{сист.}$ перевищувала 55 мм рт. ст. у 18,1 % осіб, величина ранкового підйому $АТ_{діаст.}$ перевищувала 35 мм рт. ст. у 34,3 %. Рівень глюкози мав зворотній помірної сили зв'язок із рівнем та навантаженням систолічним та середнім АТ. Рівень С-реактивного білку (С-РБ) статистично значущо корелював із індексом часу (ІЧ) $АТ_{сист.}$ вдень ($r=0,497$, $p=0,042$). По мірі підвищення навантаження $АТ_{сист.}$ спостерігалася тенденція до підвищення рівня неспецифічних маркерів запалення, до яких відносять фібриноген та С-РБ. Наявність дрібних судинних вогнищ у білій речовині великих півкуль головного мозку асоціювалося із добовим ритмом АТ по типу нон-діппер ($X^2_{(1)} = 5,22$; V Крамера = 0,225, $p=0,022$; точний критерій Фішера=0,038). З показником ІЧ $АТ_{сист.}$ вночі асоціювалися наявність дрібних вогнищ, судинного ґенезу ($X^2_{(1)} = 6,1$; V Крамера = 0,241, $p=0,014$) та наявність церебральної атрофії ($X^2_{(1)} = 5,4$; V Крамера = 0,228, $p=0,02$; точний критерій Фішера=0,025). Пацієнти з різним ступенем ураження головного мозку судинного ґенезу статистично значущо не відрізнялися за середньодобовими показниками ДМАТ, за величиною ранкового підйому АТ (як систолічного, так і діастолічного), за швидкістю ранкового підйому $АТ_{сист.}$ та за розповсюдженістю певних добових кривих АТ. У осіб із структурними змінами на МРТ головного мозку судинного ґенезу спостерігалося недостатнє зниження АТ вночі у порівнянні з пацієнтами групи 1. У більшості (58,9 %) пацієнтів, швидкість наростання $АТ_{сист.}$ вранці була збільшена, що свідчило про ймовірність негативного впливу на судинну систему за відсутності явних ознак навантаження АТ навіть на фоні антигіпертензивної терапії у пацієнтів із початковими проявами ХІМ. Збільшене навантаження систолічним артеріальним тиском вночі значущо впливало на ризик структурного ураження головного мозку.

Ключові слова: добовий моніторинг артеріального тиску, хронічна ішемія головного мозку, початкові прояви.

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Цереброваскулярні захворювання (ЦВЗ) займають третє місце в Україні за поширеністю серед хвороб системи кровообігу і мають найбільшу питому вагу серед причин первинної інвалідності населення працездатного віку внаслідок нервових хвороб — 44,2 % [13]. ЦВЗ мають ряд особливостей як за складом факторів ризику, так і відповіддю на терапію на етапі первинної і вторинної профілактики. В Україні протягом останніх 10 років кількість хворих із ЦВЗ збільшилася у 1,5 рази [13]. В основі такої негативної динаміки лежить суттєве збільшення поширеності основних судинних факторів ризику (СФР) серед населення країни — артеріальної гіпертензії (АГ), дисліпідемії, малорухомого способу життя, ожиріння, цукрового діабету, тютюнопаління, тощо, їх