DOI: https://doi.org/10.30841/2307-5112.2-3.2021.240761
УДК 616.379-008.64: 616.1

Cardiovascular pathology – a factor of the adverse course of diabetic polyneuropathy

G.M. Chupryna[1], V.M. Dubynetska[1], K.A. Kachur[2]
[1] Shupik National Healthcare University of Ukraine, Kyiv
[2]Communal enterprise «Rivne region clinical referral hospital V. Polischuk» of Rivne region council

Diabetic polyneuropathy (DP) and angiopathy are interdependent processes, as disturbances in the microcirculatory system of peripheral nerves lead to increased axonal damage and is a kind of predictor of polyneuropathy progressing [6]. 80% of deaths from diabetes mellitus (DM) are associated with cardiovascular catastrophes, including coronary heart disease (CHD), stroke and peripheral artery disease [3].
The objective: **to analyze the most common cardiovascular pathology (CVP) and show its impact on the course of DP in type I and II DM.**
Materials and methods**. Was clinically examined 101 patient with DP. The examined patients were divided into groups: with DP on the background of type 1 DM (group I) (n=54) and with DP on the background of type II DM (group II) (n=47), and also were divided into subgroups: DP on the background of type I and II DM and existing CVP (including diabetic angiopathy) 82 (82%) (subgroup A) and with the DP on the background of DM type I and II without CVP – 19 (19%) (subgroup B). Patients were examined to determine the neurological status, were performed laboratory and instrumental methods of examination. Static calculation was performed in MS Excel 2003 and in the programme STATISTICA 10.**
Results**. Regarding to the patients of subgroup A and B we noted the natural predominance of trophic disorders, changes in the reflex sphere and sensitivity in subgroup A. Patients of group II more often than in group I had pathology of the cardiovascular system. Hypertension (HT) and CHD in both cases were registered with a high frequency. In subgroup A there was a combination of several nosologies: from the respiratory, urinary, gastroenterological system (1%), urinary and gastroenterological (3%), gastroenterological and endocrine (2%), urinary and endocrine (1%). In subgroup B diseases of urinary and gastroenterological pathology were found in (5%), gastroenterological (5%), endocrine (11%). The examined patients from group I and with the concomitant CVP have lower linear velocity of blood flow (LVBF) on both tibial arteries, patients in group II – have marginally higher LVBF. Analysis of the results of duplex scanning of lower extremity arteries showed a high incidence of stenosis, in particular the anterior tibial arteries (ATA) up to 30–40%, posterior tibial arteries (PTA) up to 40–50% and occlusion (PTA and femoral, popliteal, tibial segment) in individuals of group I.**
Conclusions**. In patients with DP on the background of type I and II DM and available CVP (subgroup A), the clinical manifestations of polyneuropathy were quite pronounced, especially in the field of trophic disorders, because CVP enhances the ischemia of the microsaceous channel of the peripheral nerves. In addition, persons with concomitant CVP have a wide range of another comorbid pathology, which accelerates the onset of DM complications.**
Keywords: *diabetic polyneuropathy, cardiovascular pathology, diabetic angiopathy, linear velocity of blood flow, transsyndromic comorbidity, echocardiography.*

Кардіоваскулярна патологія – чинник несприятливого перебігу діабетичної полінейропатії
Г.М. Чуприна, В.М. Дубинецька, К.А. Качур

Діабетична полінейропатія (ДП) та ангіопатія є взаємозалежними процесами, оскільки порушення у мікроциркуляторному руслі периферичних нервів веде до посиленого аксонального пошкодження та є певною мірою предиктором прогресування полінейропатії. Вісімдесят відсотків смертей при цукровому діабеті (ЦД) пов'язують із серцево-судинними катастрофами, серед яких ішемічна хвороба серця (ІХС), інсульт та захворювання периферичних артерій.
Мета дослідження: аналіз найпоширенішої кардіоваскулярної патології (КВП) та вивчення її впливу на перебіг ДП при ЦД 1-го та 2-го типу.
Матеріали та методи. Клінічно обстежено 101 пацієнта з наявністю ДП. Обстежених розподілено на групи: із ДП на тлі ЦД 1-го типу (група І) (n=54) та з ДП на тлі ЦД 2-го типу (група ІІ) (n=47), а також на підгрупи: ДП на фоні ЦД 1-го та 2-го типу і наявною КВП (зокрема з діабетичною ангіопатією) 82 (82%) (підгрупа А) та із ДП на фоні ЦД 1-го та 2-го типу без КВП 19 (19%) (підгрупа В). Пацієнтів оглянуто із визначенням неврологічного статусу, проведено лабораторні та інструментальні методи обстеження. Статичнне оброблення виконували у MS Excel 2003 та в програмі STATISTICA 10.
Результати. Щодо пацієнтів підгрупи А та В, відзначено закономірне переважання трофічних порушень, зміни в рефлекторній сфері та чутливості у підгрупі А. В осіб групи ІІ частіше, ніж в групі І діагностовано патологію серцево-судинної системи. Гіпертонічна хвороба (ГХ) та ІХС в обох випадках реєструвалися з високою частотою. У підгрупі А зустрічалася комбінація кількох нозологій: з боку дихальної, сечовидільної, гастроентерологічної системи (1%), сечовидільної та гастроентерологічної (3%), гастроентерологічної та ендокринної (2%), сечовидільної та ендокринної (1%). У підгрупі В хвороби сечовидільної та гастроентерологічної патології виявлено у 5%, гастроентерологічної – у 5%, ендокринної – в 11% пацієнток. Жінки І групи із супутньою КВП мають нижчу лінійну швидкість кровообігу (ЛШК) по обох гомілкових артеріях, пацієнтки ІІ групи – незначно вищі показники ЛШК. Аналіз результатів дуплексного сканування артерій нижніх кінцівок продемонстрував високу частоту стенозів, зокрема передніх великогомілкових артерій (ПВГА) до 30–40%, задніх великогомілкових артерій (ЗВГА) до 40–50% та оклюзії (ЗВГА та стегново-підколінно-гомілкового сегменту) в осіб групи І.
Заключення. У пацієнтів із ДП на фоні ЦД 1-го та 2-го типу і наявною КВП (підгрупа А) клінічні прояви полінейропатії досить виражені, особливо у сфері трофічних порушень, адже КВП посилює ішемізацію мікросудинного русла периферичних нервів. Окрім того, в осіб із супутньою КВП є широкий спектр іншої коморбідної патології, що пришвидшує настання ускладнень ЦД.
Ключові слова: *діабетична полінейропатія, кардіоваскулярна патологія, діабетична ангіопатія, лінійна швидкість кровообігу, транссиндромальна коморбідність, ехокардіоскопія.*

Кардиоваскулярная патология – фактор неблагоприятного течения диабетической полинейропатии
Г.Н. Чупрына, В.Н. Дубинецкая, Е.А. Качур

Диабетическая полинейропатия (ДП) и ангиопатия являются взаимозависимыми процессами, поскольку нарушения в микроциркуляторном русле периферических нервов ведут к усиленному аксональному повреждению и являются своего рода предиктором прогрессирования полинейропатии. Восемьдесят процентов смертей при сахарном диабете (СД) связывают с сердечно-сосудистыми катастрофами, в числе которых ишемическая болезнь сердца (ИБС), инсульт и заболевания периферических артерий.

Цель исследования: анализ наиболее распространенной кардиоваскулярной патологии (КВП) и изучение ее влияния на ход ДП при СД 1-го и 2-го типа.
Материалы и методы. Клинически обследован 101 пациент с наличием ДП. Обследованные разделены на группы: с ДП на фоне СД 1-го типа (группа I) (n=54) и с ДП на фоне СД 2-го типа (группа II) (n=47), а также на подгруппы: ДП на фоне СД 1-го и 2-го типа и имеющейся КВП (в том числе с диабетической ангиопатией) 82 (82%) (подгруппа А) и с ДП на фоне СД I и II типа без КВП 19 (19%) (подгруппа В). У пациентов зафиксирован неврологический статус, проведены лабораторные и инструментальные методы обследования. Статическую обработку выполняли в MS Excel 2003 и в программе STATISTICA 10.
Результаты. Относительно пациентов подгруппы А и В отмечаем закономерное преобладание трофических нарушений, изменений в рефлекторной сфере и чувствительности в подгруппе А. У лиц группы II чаще, чем в группе I встречалась патология сердечно-сосудистой системы. Гипертоническая болезнь (ГБ) и ИБС в обоих случаях регистрировались с высокой частотой. В подгруппе А встречалась комбинация нескольких нозологий: со стороны дыхательной, мочевыделительной, гастроэнтерологической системы (1%), мочевыделительной и гастроэнтерологической (3%), гастроэнтерологической и эндокринной (2%), мочевыделительной и эндокринной (1%). В подгруппе В болезни мочевыделительной и гастроэнтерологической патологий выявлено у 5%, гастроэнтерологической – у 5%, эндокринной – у 11% пациентов. Обследуемые из группы I с сопутствующей КВП имеют более низкую линейную скорость кровообращения (ЛСК) по обеим артериям голени, лица из группы II – незначительно более высокие показатели ЛСК. Анализ результатов дуплексного сканирования артерий нижних конечностей продемонстрировал высокую частоту стенозов, в частности передних большеберцовых артерий (ПББА) до 30–40%, задних большеберцовых артерий (ЗББА) до 40–50% и окклюзии (ЗББА и бедренно-подколенно-голенного сегмента) у пациентов группы I.
Заключение. У пациентов с ДП на фоне СД 1-го и 2-го типа и имеющейся КВП (подгруппа А) клинические проявления полинейропатии достаточно выражены, особенно в сфере трофических нарушений, ведь КВП усиливает ишемизацию микрососудистого русла периферических нервов. Кроме того, у лиц с сопутствующей КВП есть широкий спектр другой коморбидной патологии, что, несмотря на имеющуюся кардиоваскулярную коморбидность, ускоряет наступление осложнений СД.
Ключевые слова: *диабетическая полинейропатия, кардиоваскулярная патология, диабетическая ангиопатия, линейная скорость кровообращения, транссиндромальная коморбидность, эхокардиоскопия.*

While to 60% of patients with a long-standing history of diabetes mellitus (DM) have diabetic polyneuropathy (DP), 7–10% of people with the first diagnosed DM have verified DP [1, 17, 19]. The incidence of DP is higher in people with type 2 DM (6,100 per 100,000 people per year) than in people with type 1 DM (2,800 per 100,000 people per year) [2, 11, 14, 15]. Opposite, the prevalence of DP is almost the same as in type 2 DM (8–51% [7, 8, 13]) and type 1 DM (11–50% [4, 7, 18]). It is important that the prevalence of DP is even higher when asymptomatic (subclinical) neuropathy is included, 45% in patients with type 2 DM and 54% in patients with type 1 DM [7].

Damage to the blood vessels of the lower extremities in DM is the main cause of amputations of the lower extremities, unrelated to physical traumas or road accidents [10].

Diabetic foot is an important problem in economic terms, especially if amputation is the end of long hospital treatment with the patient's discharge home and the need to care for him. The cost of primary treatment is estimated at 7–10 thousand USD [12].

Increased lipid profile indicators in a patient with DM together with hypertension (HT), which predominates in middle-aged and elderly people, contributes to the formation of metabolic syndrome, which can lead to vascular accidents in the future [5].

DP and angiopathy are interdependent processes, as disturbances in the microcirculatory system of peripheral nerves lead to increased axonal damage, and the presence of trophic disorders in DP is accompanied by an inability of the vascular system to adequately deliver nutrients to nerve fibers, which contributes to chronic ischemia and is a kind of predictor of polyneuropathy progressing [6].

Diabetic angiopathies affect almost all organs due to impaired blood supply, and damage to various types of blood vessels leads to a significant deterioration in the course of the disease. The cardiovascular system is most affected. Today we are talking about an epidemic of atherosclerotic complications in patients with type II DM. 80% of deaths from DM are associated with cardiovascular catastrophes, including coronary heart disease (CHD), stroke and peripheral artery disease [3].

The objective: to analyze the most common cardiovascular pathology (CVP) and show its impact on the course of DP in type I and II DM.

MATERIALS AND METHODS

Was clinically examined 101 patient with DP, aged from 19 to 69 years (M±m; 50.94±1.34 years). Women predominated – 52 (52%) patients, men were 49 (49%). Type I DM was detected in 54 (54%), type II DM – in 47 (47%) patients.

The examined patients were divided into groups: with DP on the background of type 1 DM (group I) (n=54) and with DP on the background of type II DM (group II) (n=47). Depending on the presence of CVP, patients were divided into subgroups: DP on the background of type I and II DM and existing CVP (including diabetic angiopathy) 82 (82%) (subgroup A) and with the DP on the background of DM type I and II without CVP – 19 (19%) (subgroup B).

Patients were examined to determine the neurological status, were performed laboratory (general blood test, general urine test, biochemical blood test, glycated hemoglobin) and instrumental methods of examination (duplex scanning of the vessels of the lower extremities, electrocardiography (ECG), echocardiography (Echo), electroneuromyography (ENMG). Static calculation was performed in MS Excel 2003 and in the programme for statistical analysis STATISTICA 10.

RESEARCH RESULTS AND DISCUSSION

In subgroup A type I DM was verified in 42 (51%) patients, type II – in 40 (49%), in subgroup B type I DM – in 12 (63%), type II – in 7 (37%). The average data on the age category of patients are shown in Fig. 1.

Among 101 examined patient we observed changes in the reflex sphere of the lower extremities in 86 (86%), namely: decreased knee reflexes – 64 (74%), loss – 9 (10%), decreased Achilles reflexes – 49 (57%), loss – 36 (42%), decreased plantar reflexes in 40 (47%), loss in 45 (52%).

There were disturbances in the reflex sphere of the upper extremities in 42 (42%) examined patients: decreased carporadial reflex – 16 (38%), loss – 26 (62%), decreased biceps reflex – 26 (62%), loss – 16 (38%), decreased triceps reflex – 23 (55%), loss – 3 (7%).

Sensitivity disorders were found in 92 (92%) patients, of which 64 (70%) had hypoesthesia of the distal extremities, and 28 (30%) had hyperesthesia.

Regarding to the patients of subgroup A and B we noted the natural predominance of trophic disorders (Table 1), changes in the reflex sphere and sensitivity in subgroup A. Decrease in vibrational sensitivity is present in both subgroups, but in subgroup A the indicators are lower, in particular in the lower extremities (7.18±0,34 s).

In 1 patient of subgroup A was revealed a slight peripheral paresis of both hands, in subgroup B in 1 patient previous changes were combined with slight peripheral paresis of both feet. Manifestations

Table 1

Frequency of changes of the main parameters of the patients´ neurological status with DP subgroups A and B (absolute values)

Indicator		Subgroup A, n=82	Subgroup B, n=19
Trophic disorders of the lower extremities			
Hypohidrosis		32	11
Hyperhidrosis		27	2
Hypertrichosis		21	9
Hypotrichosis		32	3
White dermographism		49	10
Red dermographism		33	9
Hyperkeratosis		47	10
Foot fissure		42	6
Trophic changes of the nails		44	13
Dry skin		26	8
Reflexes			
Carpo radial	loss	25	1
	decreased	12	4
Biceps	loss	15	1
	decreased	22	4
Triceps	loss	3	0
	decreased	21	2
Knee	loss	8	1
	decreased	54	10
Achilles	loss	34	2
	decreased	38	11
Plantar	loss	38	7
	decreased	32	8
Sensitivity			
Hyperesthesia		23	5
Hypoesthesia		53	11
Average Indicator of vibrational sense on upper extremities, s		11.23±0,47	13±1.01
Average Indicator of vibrational sense on lower extremities, s		7.18±0.34	7.73±0.56

of «diabetic foot» were diagnosed in 12 (15%) patients of subgroup A, and in 6 (8%) amputation of fingers was performed, in subgroup B none of the patients had such complication of DM.

Patients of group II more often than in group I had pathology of the cardiovascular system (Fig. 2). HT and CHD in both cases were registered with a high frequency. In addition to CVP, disorders of the gastrointestinal tract in people with type II DM also dominate (Fig. 3). Gallstone disease (GD), chronic cholecystitis (CC), chronic pancreatitis (CP) and chronic hepatitis (CH) were more commonly diagnosed in both type I and type II DM.

In subgroup A there was a combination of several nosologies: from the respiratory, urinary, gastroenterological system (1%),

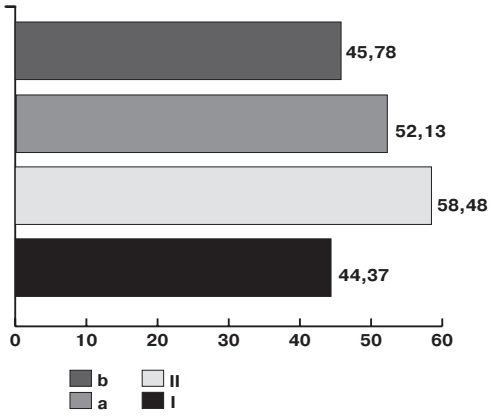

Fig. 1. The average age of the patients in each of the groups and subgroups

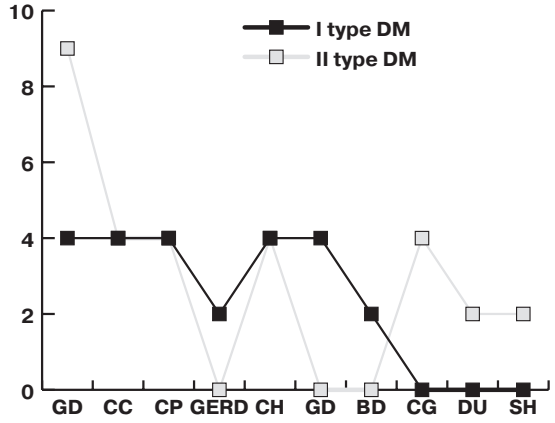

Fig. 2. Distribution of CVP in DP on the background of type I and II DM
*Note. HT – hypertension, CHD – coronary heart disease, AF – atrial fibrillation, CgHD – congenital heart disease.

ISSN 2307-5112 (Print) | ISSN 2412-8708 (Online)

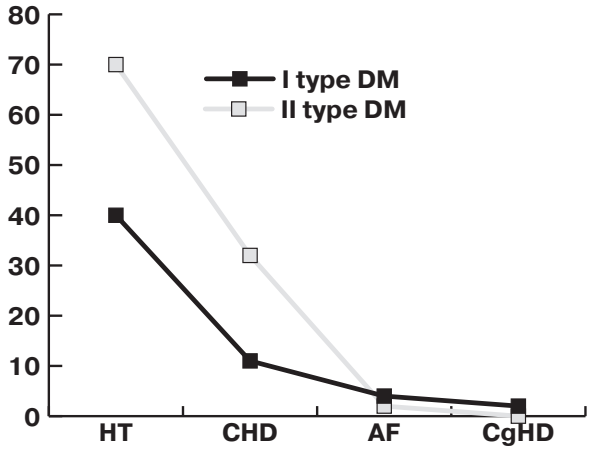

Fig.3. The spread of gastrointestinal pathology in DP on the background of type I and II DM

**Note.* GD – gallstone disease, CC – chronic cholecystitis, CP – chronic pancreatitis, GERD – gastroesophageal reflux disease, CH – chronic hepatitis, GD – gastroduodenitis, BD – biliary dyskinesia, CG – chronic gastritis, DU – duodenal ulcer, SH – steatohepatitis.

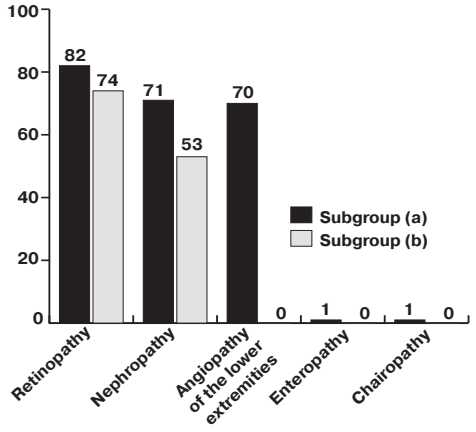

Fig. 4. Transsyndromic comorbidity in DP in the examined subgroups

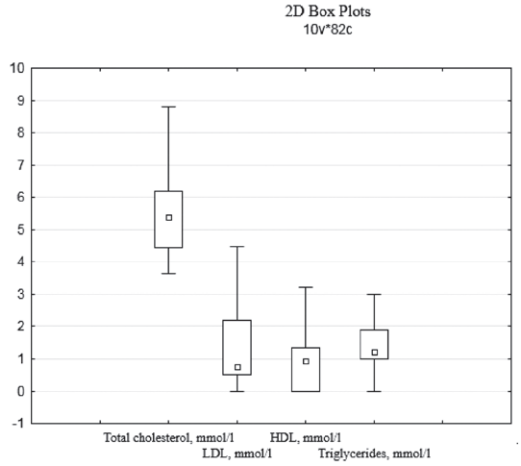

Fig. 5. Lipid profile of the examined subgroup A

(*Note.* LDL – low-density lipoprotein cholesterol, HDL – high-density lipoprotein cholesterol)

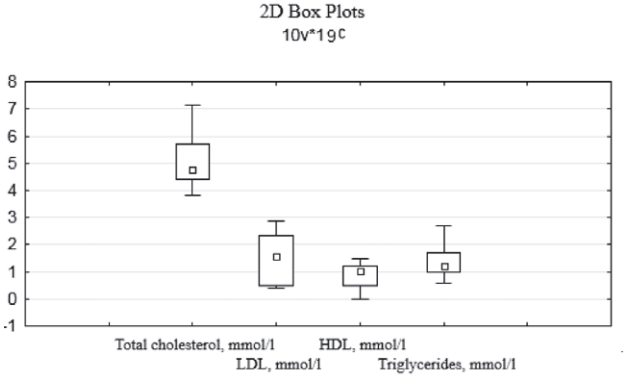

Fig.6. Lipid profile of the examined subgroup B

urinary and gastroenterological (3%), gastroenterological and endocrine (2%), urinary and endocrine (1%). Were diagnosed single lesions of the endocrine system (actually the thyroid gland) (15%), gastroenterological (5%), urinary (2%). Among this cohort of patients (5%) – varicoses of the lower extremities, in (1%) – suffered acute thrombosis of the veins of the left lower extremity. In subgroup B diseases of urinary and gastroenterological pathology were found in (5%), gastroenterological (5%), endocrine (11%).

Among transsyndromal comorbidity dominate retinopathy, nephropathy and angiopathy of the lower extremities dominate (Fig. 4), which are more often present in subgroup A.

Lipid profile indicators (Fig. 5, 6) in patients of subgroup A and B are within normal values. In subgroup A the quantity of the scope for all parameters of lipid metabolism is higher, in particular for total cholesterol.

The average rate of glycated hemoglobin (Fig. 7) in patients of group II is significantly higher than in group I, in subgroup B is slightly higher than in A.

According to the results of the recorded ECG in subgroup A in 14 (17%) blockade of the legs of His bundle, 12 (15%) sinus

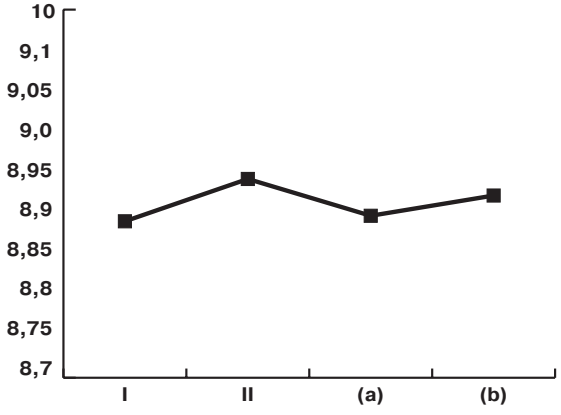

Fig. 7. The average value of glycated hemoglobin in the examined groups and subgroups

Table 2

Average rates of LVBF, sm/s in persons with DP

Rate	Normal rates	Group I		Group II	
		Generally in group, n=21	With CVP, n=11	Generally in group, n=13	With CVP, n=10
On the right ATA LVBF, sm/s	35–87 sm/s	55.95±4,50	54.54±4.69	58±4.97	62.7±5.53
PTA LVBF, sm/s	42–83 sm/s	54.52±4.08	50.45±5.53	46.53±4.19	51.9±3.59
On the left ATA LVBF, sm/s	35–87 sm/s	54.76±4.27	45.45±5.85	49.46±4.99	51.6±5.39
PTA LVBF, sm/s	42–83 sm/s	57.61±4.95	49.54±8.15	49.15±4.70	54.3±4.52

Table 3

Frecuency of violation of arterial circulation in DP

Degree of violation of arterial circulation	Group I, n=21	Group II, n=13
Stenosis 30–40% ATA	6 (29%)	2 (15%)
Stenosis 30–40% PTA	2 (10%)	3 (23%)
Stenosis 40–50% ATA	3 (14%)	2 (15%)
Stenosis 40–50% PTA	2 (10%)	0
Stenosis 50–60% PTA	1 (5%)	1 (8%)
Stenosis 60–70% ATA	0	2 (15%)
Stenosis 70–80% ATA	1 (5%)	0
Stenosis 70–80% PTA	1 (5%)	1 (8%)
Occlusion	2 (10%)	1 (8%)

tachycardia, 7 (9%) early ventricular repolarization syndrome, 3 (4%) sinus bradycardia, 1 (1%) Q-T prolongation, in subgroup B – 3 (16%) early repolarization syndrome, 2 (11%) blockade of the legs of His bundle, 2 (11%) sinus tachycardia.

The results of the Echo, which was carried out in 17 patients, in the subgroup A demonstrated the following changes: diastolic left ventricular (LV) dysfunction (7%), LV concentric hypertrophy (5%), left atrial (LA) dilatation (4%), fibrocalcific aortic valve (AV) (4%), prolapse of the anterior mitral valve (MV) (3%), additional atypical LV chord (1%), aortic dilatation (1%), AV stenosis (1%), pulmonary hypertension (1%), dilatation of both atrias and LV (1%), LV systolic dysfunction (1%), hypokinesis of the posterior basal and posterior diaphragmatic LV (1%). In subgroup B - additional chord of LV 1 (5%), diastolic dysfunction of LV 1 (5%), prolapse of MV 1 (5%).

In 34 (34%) patients (21 from group I and 13 from group II), was performed duplex vascular scans of the lower extremities.

Linear velocity of blood flow (LVBF) in the anterior (ATA) and posterior tibial (PTA) arteries was within normal range in all examined (table 2).

The examined patients from group I and with the concomitant CVP have lower LVBF on both tibial arteries. The LVBF on the left ATA is quite low – 45.45±5.85 sm/s. Regarding to the patients in group II, we observed marginally higher LVBF rates in patients with concomitant CVP. This phenomenon was associated with compensatory acceleration of blood flow in stenosed vessels during the initial stages of atherosclerosis and a wide intake of antiplatelet agents by patients of this sample.

Analysis of the results of duplex scanning of lower extremity arteries (table 3) showed a high incidence of stenosis, in particular ATA up to 30–40%, PTA up to 40–50% and occlusion (PTA and femoral, popliteal, tibial segment) in individuals of group I. According to the literature [9, 16], despite hyperlipidemia, it has been proved that in the absence of circulating insulin, such changes in lipid metabolism do not lead to the emergence of a vascular lesion, but the need to increase the dose of insulin is a sensitive indicator of the development of macroangiopathy.

CONCLUSIONS

1. In patients with DP on the background of type I and II DM and available CVP (subgroup A), the clinical manifestations of polyneuropathy were quite pronounced, especially in the field of trophic disorders. Hyperkeratosis, changes in the nail plate, cracks, hypotrichosis, hypohydrosis prevail, because CVP enhances the ischemia of the microsaceous channel of the peripheral nerves. The spread of white dermographism indicates initial lesions of the vegetative link of the nervous system.

2. In the examined patients with DP on the background of type I and II DM and the existing CVP (subgroup A), despite the lower level of glycated hemoglobin (8.88±0.18%) than in subgroup B (8.99±0.52%), was diagnosed a significant number of patients with a «diabetic foot». In addition, persons with concomitant CVP have a wide range of another comorbid pathology (from the gastroenterological, urinary, respiratory system), which accelerates the onset of DM complications.

3. Patients with DP and type I DM (group I) show with greater frequency the presence of lower extremity arterial stenoses of varying degrees, although the overall LVBF in the group is higher, with the exception of ATA on the right, than in the comparison group.

4. CVP creates an unfavorable background for restorative processes in the distal parts of nerve fibers, which are exposed to the devastating effects of hyperglycemia earliest and fastest.

Information about the author

Chupryna Gennadii M. – Shupik National Healthcare University of Ukraine, Kyiv, 04112, Kyiv, 9 Dorohozhytska Str; tel.: (067) 405-78-25. *E-mail: gen7chupryna@gmail.com*

Dubynetska Viktoriia M. – Shupik National Healthcare University of Ukraine, Kyiv, 04112, Kyiv, 9 Dorohozhytska Str; tel.: (096) 340-44-16. *E-mail: viktoria_md@ukr.net*

Kachur Kateryna A. – Communal enterprise «Rivne region clinical referral hospital V. Polischuk» of Rivne region council, 33000, Rivne, 36 July 16 Str.; tel.: (097) 751-50-19. E-*mail: katyakachur2012@gmail.com*

Відомості про авторів

Чуприна Геннадій Миколайович – Національний університет охорони здоров'я П.Л. Шупика, 04112, м. Киев, вул. Дорогожицька, 9; тел.: (067) 405-78-25. *E-mail: gen7chupryna@gmail.com*

Дубинецька Вікторія Миколаївна – Національний університет охорони здоров'я П.Л. Шупика, 04112, м. Киев, вул. Дорогожицька, 9; тел.: (096) 340-44-16. *E-mail: viktoria_md@ukr.net*

Качур Катерина Анатоліївна – Комунальне підприємство «Рівненський обласний клінічний лікувально-діагностичний центр ім. В. Поліщука» Рівненської обласної ради, 33000, м. Рівне, вул. 16 липня, 36; тел.: (097) 751-50-19. *E-mail: katyakachur2012@gmail.com*

Сведения об авторах

Чуприна Геннадий Николаевич – Национальный университет здравоохранения имени П.Л. Шупика, 04112, г. Киев, ул. Дорогожицкая, 9; тел.: (067) 405-78-25. *E-mail: gen7chupryna@gmail.com*

Дубинецкая Виктория Николаевна – Национальный университет здравоохранения имени имени П.Л. Шупика, 04112, г. Киев, ул. Дорогожицкая, 9; тел.: (096) 340-44-16. *E-mail: viktoria_md@ukr.net*

Качур Екатерина Анатольевна – Коммунальное предприятие «Ровенский областной клинический лечебно-диагностический центр им. В. Полищука» Ровенского областного совета, 33000, г. Ровно, ул. 16 июля, 36; тел.: (097) 751-50-19. *E-mail: katyakachur2012@gmail.com*

REFERENCES

1. Abbott A. et al. Prevalence and Characteristics of Painful Diabetic Neuropathy in a Large Community-Based Diabetic Population in the U.K. Diabetes Care, 2011, Oct. 34(10):2220-4.
2. Ang L., Jaiswal M., Martin C. & Pop-Busui R. Glucose control and diabetic neuropathy: lessons from recent large clinical trials. Curr. Diab. Rep. 14, 528(2014).
3. Beckman JA, Creager MA, Libby P. Diabetes and athero sclerosis: epidemiology, pathophysiology and management. JAMA. 2002; 287:2570–81.
4. Boulton A, Knight G, Drury J & Ward JD. The prevalence of symptomatic, diabetic neuropathy in an insulin-treated population. Diabetes Care, 1985; 8:125-8.
5. Chupryna GM, Dubynetska VM, Vashenyuk OL, Vashenyuk NO. Features of clinical and paraclinic parameters and comorbidity in patients with diabetic polyneuropathy. Health of Society, 2019;8(5):174-8.
6. Dubynetska VM., Chupryna GM. Diabetic polyneuropathy: a look at the problem through the prism of comorbidity. East European Jornal of Neurology, 2019;1(25):36-46.
7. Dyck PJ et al. The prevalence by staged severity of various types of diabetic neuropathy, retinopathy, and nephropathy in a population-based cohort: the Rochester Diabetic Neuropathy Study. Neurology, 1993; 43: 817-24.
8. Franklin GM, Kahn LB, Baxter J, Marshall JA & Hamman RF. Sensory neuropathy in non-insulindependent diabetes mellitus. The San Luis Valley Diabetes study. Am. J. Epidemiol; 1990;131:633-43.
9. Janka HU, Standl E. Hyperinsulinaemia as a possible risk factor of macrovascular disease in diabetes mellitus. An overview. Diabete Metab, 1987 Jul;13(3Pt 2):279-83.
10. Kaminskyi AV, Kovalenko AN. Diabetes mellitus and obesity: a clinical guide to diagnosis and treatment. K.: Lira; 2010. 256 p.
11. Martin CL, Albers JW & Pop-Busui R. Neuropathy and related findings in the diabetes control and complications trial/epidemiology of diabetes interventions and complications study. Diabetes Care; 2014;37:31-8.
12. Mota M, Vasile A, Mitroy N. Risk Factors Leading to Amputation in Patients with Diabetes Mellitus. Maria Mota, Andriana Vasile, Nikoletta Mitroy and others. International Journal of Endocrinology; 2005;01:9-24.
13. Partanen J. et al. Natural history of peripheral neuropathy in patients with non-insulin-dependent diabetes mellitus. N. Engl. J. Med; 1995: 333, 89-94.
14. Pop-Busui R. et al. Diabetic neuropathy: a position statement by the American Diabetes Association. Diabetes Care; 2017; 40:136-54.
15. Pop-Busui R et al. Impact of glycemic control strategies on the progression of diabetic peripheral neuropathy in the Bypass Angioplasty Revascularization Investigation 2 Diabetes (BARI 2D) Cohort. Diabetes Care;2013:36:3208-15.
16. Stout RW. Hormones and Atherosclerosis: M.: Medicine, 1985:240.
17. Tesfaye S, Andrew JM, Boulton, Peter J Dyck, Roy Freeman et al. Diabetic Neuropathies: Update on Definitions, Diagnostic Criteria, Estimation of Severity, and Treatments. Diabetes Care. 2010 Oct; 33(10):2285-93.
18. Tesfaye S et al. Vascular risk factors and diabetic neuropathy. N. Engl. J. Med.; 2005:352, 341-50.
19. Tracy JA et al. The Spectrum of Diabetic Neuropathies. Physical Medicine and Rehabilitation Clinics N Am. 2008 Feb; 19(1):1-26. 1-v.

Стаття надійшла до редакції 07.01.2021. – Дата першого рішення 15.01.2021. – Стаття подана до друку 26.02.2021