**Elasticheskie svoystva sosudov, pokazateli funktsional'nogo sostoyaniya pochek i pochechnogo krovotoka u bol'nykh serdechno-sosudistymi zabolevaniyami, assotsiirovannymi s sakharnym diabetom 2 tipa**

*Oskola E.V., Shubina A.T., Zairova A.R., Andreevskaya M.V., Bogieva R.M., Pogorelova O.A., Bolotina M.G., Balakhonova T.V., Rogoza A.N., Karpov Yu.A.*

Institut klinicheskoy kardiologii FGBU «Rossiyskogo kardiologicheskogo nauchno-proizvodstvennogo kompleksa» MZ RF, Moskva

(direktor – akademik RAN i RAMN E.I. Chazov)

***Tsel'.*** *Izuchit' vzaimosvyaz' izmeneniy pokazateley zhestkosti arteriy razlichnogo tipa, pul'satsionnosti gemodinamiki, funktsii pochek i pochechnogo krovotoka u bol'nykh osnovnymi serdechno-sosudistymi zabolevaniyami (SSZ) pri nalichii ili otsutstvii sakharnogo diabeta 2 tipa (SD-2).*

 ***Materialy i metody.*** *V issledovanie bylo vklyucheno 96 patsientov s ishemicheskoy bolezn'yu serdtsa i arterial'noy gipertoniey, v tom chisle 54 patsienta s SD-2 i 42 patsienta bez SD-2. Opredelyali pokazateli uglevodnogo i lipidnogo obmenov, funktsii pochek, a takzhe pokazateli zhestkosti arteriy razlichnogo tipa, parametry pul'satsionnosti gemodinamiki i pochechnogo krovotoka.*

***Rezul'taty.*** *Pri nalichii SD-2 otmecheno povyshenie pokazateley zhestkosti arteriy razlichnykh tipov: skorosti pul'sovoy volny v aorte na karotidno-femoral'nom uchastke (sosud elasticheskogo tipa) na 16% (r<0,001), β obshchey sonnoy arterii (sosud myshechno-elasticheskogo tipa) na 7,6% (r<0,05), β plechevoy arterii (sosud myshechnogo tipa) na 22% (r<0,05). Odnovremenno otmecheno uvelichenie pokazatelya mikroal'buminurii v 5 raz (r<0,05) i rezistivnogo indeksa pochechnogo krovotoka na 12,5% (r<0,05). V obeikh gruppakh patsientov, kak s SD-2, tak i bez SD-2, vyyavleny vzaimosvyazi pokazateley zhestkosti aorty i parametrov pul'satsionnosti (pul'sovogo arterial'nogo davleniya (PAD), izmerennogo na plechevoy arterii, tsentral'nogo PAD, indeksa augmentatsii) s funktsional'nym sostoyaniem pochek i pochechnogo krovotoka.*

***Zaklyuchenie.*** *Poluchennye rezul'taty mogut svidetel'stvovat' ob obshchnosti i vzaimosvyazi patogeneticheskikh mekhanizmov, lezhashchikh v osnove razvitiya povyshennoy zhestkosti magistral'nykh sosudov i disfunktsii pochek u bol'nykh SSZ, assotsiirovannymi s SD-2.*

***Klyuchevye slova:*** *sakharnyy diabet tipa 2, zhestkost' sosudov, pochechnyy krovotok, disfunktsiya pochek.*

***Arterial stiffness, renal function and renal blood flow in patients with cardiovascular disease and type 2 diabetes mellitus***

*Oskola E.V., Shubina A.T., Zairova A.R., Andreevskaya M.V., Bogieva R.M., Pogorelova O.A., Bolotina M.G., Balahonova T.V., Rogoza A.N., Karpov Yu.A.*

*Cardiology Research Complex, Moscow, Russian Federation*

***Aim.*** *To investigate the interrelationship of changes in indicators of arterial stiffness of various types vessels, hemodynamics pulsatility, renal function and renal blood flow in patients with underlying cardiovascular disease (CVD) in the presence or absence of type 2 diabetes mellitus (DM-2).*

***Materials and Methods****. The study included 96 patients with coronary artery disease and arterial hypertension, among them 54 subjects with DM-2 and 42- without DM-2. Sarbohydrate and lipid metabolism, renal function, stiffness of various types of arteries, parameters of hemodynamics pulsatility and renal blood flow were investigated.*

***Results.*** *Arterial stiffness of various types vessels was increased in type 2 diabetes group: carotid- femoral pulse wave velocity as a marker of stiffness of aorta (vessel of elastic type) was increased by 16% (p <0,001), index β of common carotid artery (vessel of musculo - elastic type ) - by 7,6% (p < 0,05) and the index β of brachial artery (vessel of muscular type) - by 22% (p < 0,05). The level of microalbuminuria was 5-fold higher (p <0,05) and renal resistive index was 12,5 % higher (p < 0,05) in diabetics group. Significant correlations were found between stiffness of aorta, parameters of hemodynamics pulsatility (pulse pressure, measured at the brachial artery, the central pulse pressure, augmentation index) and renal function and renal blood flow in both groups of patients.*

***Conclusion.*** *The results may indicate the general pathogenetic mechanisms and interrelationship of development of increased stiffness of aorta and renal dysfunction in patients with cardiovascular disease and DM-2.*

***Keywords:*** *type 2 diabetes mellitus, vascular stiffness, renal blood flow, renal dysfunction.*

Zhestkost' aorty obladaet nezavisimym prognosticheskim znacheniem v otnoshenii fatal'nykh i nefatal'nykh serdechno-sosudistykh sobytiy (SSS) u bol'nykh s arterial'noy gipertoniey (AG) [1], sakharnym diabetom 2 tipa (SD-2) [2] i ishemicheskoy bolezn'yu serdtsa (IBS) [1,3]. Pri etom sposobnost' pokazateley zhestkosti aorty predskazyvat' neblagopriyatnye sobytiya (serdechno-sosudistye oslozhneniya i letal'nost') vyshe u patsientov s iskhodno vysokim serdechno-sosudistym riskom (s IBS, AG, SD-2, zabolevaniyami pochek), chem v obshchey populyatsii [1]. V ryade klinicheskikh issledovaniy byla takzhe prodemonstrirovana prognosticheskaya znachimost' pokazateley zhestkosti obshchikh sonnykh arteriy (OSA) v razlichnykh gruppakh patsientov, v tom chisle s SD-2 [4], s khronicheskoy bolezn'yu pochek (KhBP) [5]. V issledovanii SMART bylo otmecheno, chto uvelichenie pokazateley zhestkosti i tolshchiny intima-media (TIM) OSA yavlyaetsya markerom serdechno-sosudistogo riska (SSR) kak u patsientov s faktorami riska serdechno-sosudistykh zabolevaniy (SSZ) (v tom chisle s SD-2), tak i u patsientov s uzhe imeyushchimisya SSZ (ateroskleroticheskim porazheniem perifericheskikh arteriy, AG) [6]. U bol'nykh CD-2 vyyavlyayutsya bolee vysokie pokazateli zhestkosti aorty [2,7], OSA [4], arteriy myshechnogo tipa [7] po sravneniyu s litsami bez SD-2, sopostavimymi po vozrastu i urovnyu arterial'nogo davleniya (AD).

SD-2 porazhaet sosudy kak makro-, tak i mikrotsirkulyatornogo rusla. Vsledstvie makroangiopatii razvivayutsya ateroskleroz i ego oslozhneniya, s razvitiem mikroangiopatii svyazany retinopatiya i nefropatiya. Bolee poloviny patsientov s SD-2 umirayut ot serdechno-sosudistykh oslozhneniy (SSO) [8]. Diabeticheskaya nefropatiya stoit na vtorom meste posle serdechno-sosudistykh zabolevaniy (SSZ) sredi prichin smertnosti bol'nykh SD-2 [9]. Krome togo, KhBP kak umerennoy, tak i tyazheloy stadiy uvelichivaet riski SSO, serdechno-sosudistoy smertnosti (SSS) i smertnosti ot vsekh prichin [10].

Povyshennaya zhestkost' magistral'nykh arteriy assotsiiruetsya s razvitiem kak makro-, tak i mikrososudistykh oslozhneniy pri SD-2 [7]. V svoyu ochered', dazhe pri nachal'nykh stadiyakh nefropatii otmechaetsya uvelichenie zhestkosti sosudov [11]. Izvestno o vzaimosvyazi pokazateley zhestkosti aorty i obshchikh sonnykh arteriy s mikroal'buminuriey (MAU) i snizheniem skorosti klubochkovoy fil'tratsii (SKF) u bol'nykh SD-2 [12,13]. Vzaimosvyaz' pokazateley zhestkosti magistral'nykh arteriy i funktsional'nogo sostoyaniya pochek u patsientov s SSZ i SD-2 mozhet byt' obuslovlena nalichiem obshchikh patogeneticheskikh mekhanizmov, uchastvuyushchikh v razvitii izmeneniy v stenke sosudov i klubochkakh pochek: aktivatsiya renin-angiotenzin-al'dosteronovoy sistemy (RAAS), endotelial'naya disfunktsiya, okislitel'nyy stress, protsessy glikirovaniya i vospaleniya [14]. Znachimyy vklad v izmenenie struktury sosudistoy stenki i klubochkov pochek pri SD-2 vnosyat protsessy glikirovaniya strukturnykh belkov (kollagen, elastin) [15]. Krome togo, pod vliyaniem konechnykh produktov glikirovaniya (KPG), nakaplivayushchikhsya v usloviyakh aktivatsii okislitel'nogo stressa pri SD-2, proiskhodit uvelichenie produktsii kollagena, laminina i fibronektina v stenke sosudov s izmeneniem ikh svoystv i uvelicheniem zhestkosti [16]. Eti zhe mekhanizmy igrayut vazhnuyu rol' v patogeneze glomeruloskleroza i tubulointerstitsial'nogo fibroza pri nefropatii [17]. Krome togo, pri uvelichenii zhestkosti magistral'nykh arteriy povyshaetsya pul'satsionnost' krovotoka na periferii, chto mozhet okazyvat' povrezhdayushchee deystvie na organy-misheni, v tom chisle pochki.

Tsel'yu nastoyashchego issledovaniya bylo izuchenie pokazateley zhestkosti arteriy razlichnogo tipa, parametrov pul'satsionnosti i pochechnogo krovotoka, pokazateley funktsii pochek i ikh vzaimosvyaz' u bol'nykh serdechno-sosudistymi zabolevaniyami s SD-2.

**Material i metody**

***Kriterii vklyucheniya patsientov v issledovanie***

Kriteriyami vklyucheniya v issledovanie bylo nalichie u patsientov stabil'noy IBS i AG, diagnoz kotorykh byl ustanovlen v sootvetstvii s rekomendatsiyami Rossiyskogo Kardiologicheskogo Obshchestva. Gruppu SD-2 sostavili patsienty s ustanovlennym endokrinologom diagnozom SD-2 (soglasno kriteriyam VOZ), libo patsienty, nakhodyashchiesya na terapii sakharosnizhayushchimi preparatami po povodu diagnostirovannogo ranee SD-2. V gruppu sravneniya (bez SD-2) byli vklyucheny patsienty, u kotorykh otsutstvovalo narushenie uglevodnogo obmena po dannym peroral'nogo testa tolerantnosti k glyukoze (75g) (po kriteriyam VOZ, 2006g).

V issledovanie ne vklyuchali patsientov s perenesennymi v techenie 3 mes. do vklyucheniya v issledovanie serdechno-sosudistymi oslozhneniyami (insul't, infarkt miokarda, nestabil'naya stenokardiya), patsientov s klinikoy peremezhayushcheysya khromoty, ukazaniyami na revaskulyarizatsiyu brakhiotsefal'nykh arteriy i arteriy nizhnikh konechnostey v anamneze, s gemodinamicheski znachimymi stenozami pochechnykh arteriy, s nedostatochnost'yu krovoobrashcheniya bolee 2 funktsional'nogo klassa, KhBP bolee 2 stadii, narusheniem funktsii pecheni, anemiey, s postoyannoy formoy mertsatel'noy aritmii, s ostrymi vospalitel'nymi i sistemnymi zabolevaniyami, s nedavnimi (do 6 mes.) khirurgicheskimi vmeshatel'stvami.

***Laboratornye metody issledovaniya***

Peroral'nyy test tolerantnosti k glyukoze (GTT) provodili patsientam bez ukazaniy v anamneze na diagnostirovannyy ranee SD i pri urovne glyukozy v syvorotke krovi natoshchak < 7,0 mmol'/l. Kontsentratsiyu glyukozy v tsel'noy kapillyarnoy krovi opredelyali glyukozooksidaznym metodom na analizatore Biosen firmy EKF Diagnostic (Germaniya) natoshchak i cherez 2 chasa posle priema vnutr' 75g glyukozy, rastvorennoy v 250 ml vody.

Pokazateli lipidnogo i uglevodnogo obmenov (obshchiy kholesterin, kholesterin lipoproteinov nizkoy plotnosti, kholesterin lipoproteinov vysokoy plotnosti, triatsilglitseridy, glyukoza), uroven' kreatinina v syvorotke krovi opredelyali s pomoshch'yu test-naborov firmy Abbott (SShA) na analizatore «ARCHITECT S- 8000 Abbott Diagnostics» (SShA).

Velichinu glikirovannogo gemoglobina (HbA1c) v tsel'noy krovi (EDTA) opredelyali metodom immunoturbodimetrii s pomoshch'yu test-naborov firmy Abbott (SShA) na analizatore «ARCHITECT S- 8000 Abbott Diagnostics» (SShA).

Raschet klirensa kreatinina provodili po formule Kokrofta – Golta: Klirens kreatinina, ml/min = 88 × (140 - vozrast, gody) x massa tela, kg / 72 x kreatinin syvorotki, mkmol'/l; dlya zhenshchin rezul'tat umnozhali na 0,85.

Kontsentratsiyu tsistatina C v syvorotke krovi opredelyali s pomoshch'yu nabora reaktivov firmy BioVendor (Cheshskaya respublika) na vertikal'nom mikroplanshetnom fotometre SUNRISE firmy TECAN (Avstriya), ispol'zovali promyvateli (washers) dlya mikroplanshetov firmy TECAN Columbus (Avstriya). Raschet SKF po tsistatinu C (SKFts) provodili po formule Macisaac R.J. i soavt. (2006): SKFts (ml/min/1,73m2) = (84,6/tsistatin C) - 3,2.

Opredelenie mikroal'buminurii (MAU) v razovoy portsii mochi provodili s ispol'zovaniem test-naborov firmy «Roche» (Frantsiya) na analizatore «Hitachi 912 Roche» (Frantsiya). Za mikroal'buminuriyu v sootvetstvii s normativami Mezhdunarodnoy Diabeticheskoy Federatsii bylo prinyato znachenie otnosheniya al'bumin/kreatinin bolee 2,5 mg na mmol' kreatinina u muzhchin i bolee 3,5 mg na mmol' kreatinina u zhenshchin [18].

***Instrumental'nye metody issledovaniya***

V issledovanii izuchali pokazateli zhestkosti arteriy razlichnykh tipov. Provodili otsenku zhestkosti aorty (sosud elasticheskogo tipa), obshchey sonnoy arterii (sosud myshechno-elasticheskogo tipa), plechevoy i luchevoy arteriy (sosudy myshechnogo tipa).

Regional'nuyu zhestkost' aorty (sosud elasticheskogo tipa) otsenivali po pokazatelyu skorosti pul'sovoy volny (SPV) s ispol'zovaniem razlichnykh instrumental'nykh metodik.

Metodom applanatsionnoy tonometrii s pomoshch'yu pribora SphygmoSor (AtCor, Sidney, Avstraliya) opredelyali SPV na karotidno-femoral'nom uchastke (SPVkf). Dlya opredeleniya otrazhennoy volny v aorte s pomoshch'yu applanatsionnogo tonometra provodili tonometriyu luchevoy arterii (SphygmoSor, AtCor, Sidney, Avstraliya). V nashey rabote iz pokazateley tsentral'noy gemodinamiki, avtomaticheski vydavaemykh priborom, udelyaetsya vnimanie pokazatelyu tsentral'nogo pul'sovogo i sistolicheskogo arterial'nogo davleniya (tsPAD i tsSAD, sootvetstvenno) i pokazatelyu indeksa augmentatsii otrazhennoy volny (Aix) i indeksa augmentatsii otrazhennoy volny, skorrektirovannyy po chastote serdechnykh sokrashcheniy (Aix75) [3]. Pri nalichii mezhgruppovykh razlichiy po chastote serdechnykh sokrashcheniy (ChSS), analiz rezul'tatov provoditsya po pokazatelyu Aix75.

Dlya otsenki regional'noy zhestkosti aorty primenyalsya takzhe ul'trazvukovoy metod (UZ-metod), pozvolyayushchiy opredelit' SPV v niskhodyashchem otdele aorty (grudnoy i bryushnoy otdely) [19].

Zhestkost' magistral'nykh arteriy otsenivali takzhe s pomoshch'yu ob"emnoy sfigmografii na pribore VaseraVS-1000 (Fukuda Denshi, Yaponiya). Skorost' rasprostraneniya pul'sovoy volny opredelyali «pleche-lodyzhechnym sposobom» (SPVpl) [20]. Pokazatel' otrazhaet skorost' rasprostraneniya pul'sovoy volny preimushchestvenno po arteriyam elasticheskogo i, chastichno, myshechno-elasticheskogo tipov. Pribor avtomaticheski rasschityvaet eshche odin pokazatel' arterial'noy zhestkosti - kardio-lodyzhechnyy sosudistyy indeks (cardio-ankle vascular index - CAVI) [21]. Indeks CAVI yavlyaetsya proizvodnym ot SPV na aorto-lodyzhechnom segmente i pozvolyaet sudit' ob istinnoy zhestkosti arteriy, pri minimal'nom vliyanii AD. V nashey rabote uchityvali srednie (pravaya i levaya storona) znacheniya pokazateley SPVpl i CAVI.

 Pokazateli lokal'noy zhestkosti obshchikh sonnykh arteriy (OSA), pravoy plechevoy arterii (PA) i pravoy luchevoy (LA) arterii (indeks zhestkosti , modul' uprugosti Er) opredelyali s primeneniem tekhnologii ekho-treking na ul'trazvukovom apparate «AlocaProSound7» [22]. Znacheniya indeksa , v otlichie ot drugikh pokazateley lokal'noy zhestkosti, ne zavisyat ot kolebaniy AD. V rabote uchityvali srednie (pravaya i levaya storona) znacheniya pokazateley lokal'noy zhestkosti.

Pokazateli pochechnogo krovotoka otsenivali s pomoshch'yu UZ-metoda. Dupleksnoe skanirovanie pochechnykh arteriy provodili po standartnoy metodike na UZ sisteme EnVisor (Philips) s ispol'zovaniem mul'tichastotnogo konveksnogo datchika (3-5MGts) [23]. Kolichestvennyy analiz spektra dopplerovskogo sdviga chastot vklyuchal raschet sleduyushchikh pokazateley: indeksa rezistivnosti (RI) i pul'satsionnogo indeksa (PI).

***Statisticheskiy analiz***

Statisticheskuyu obrabotku poluchennykh rezul'tatov provodili s pomoshch'yu paketa programm Statistica 10. Rezul'taty predstavleny v vide mediany, 25-go i 75-go protsentiley. Analiz mezhgruppovykh razlichiy po kolichestvennym priznakam provodili s pomoshch'yu U-kriteriya Manna–Uitni; dlya sravneniya grupp po kachestvennym priznakam ispol'zovali tochnyy kriteriy Fishera. Korrelyatsionnyy analiz pokazateley provodili s pomoshch'yu metoda rangovoy korrelyatsii Spirmena. Razlichiya schitali dostovernymi pri r<0,05.

**Rezul'taty issledovaniya**

***Klinicheskaya kharakteristika patsientov***

 V issledovanie bylo vklyucheno 96 patsientov s IBS i AG, v tom chisle 54 patsienta s SD-2 i 42 patsienta bez SD-2. V pervuyu gruppu byli vklyucheny patsienty kak s vpervye vyyavlennym SD-2, tak i s dlitel'nym techeniem zabolevaniya (do 40 let), pri sredney prodolzhitel'nosti SD-2 - 8,2 goda.

Gruppy patsientov ne razlichalis' po vozrastu, polu i urovnyu AD, izmerennomu na plechevoy arterii (tabl. 1). V gruppe SD-2 po sravneniyu s gruppoy bez SD-2 otmechalas' bolee vysokaya ChSS: 60 (56-64) ud/min protiv 56 (51-62) ud/min (r<0,01). Gruppy patsientov byli sopostavimy po provodimoy gipotenzivnoy, antianginal'noy, lipidsnizhayushchey i antiagregantnoy terapii. Bol'shinstvo vklyuchennykh v issledovanie bol'nykh s SD-2 po naznacheniyu endokrinologa poluchali mono- ili kombinirovannuyu sakharosnizhayushchuyu terapiyu, v tom chisle metformin, preparaty sul'fonilmocheviny, ingibitory dipeptidilpeptidazy-4, insulin; 5 patsientov nakhodilos' na dietoterapii.

Gruppy patsientov dostoverno razlichalis' po urovnyu glyukozy venoznoy krovi natoshchak i urovnyu HbA1c, kotoryy v gruppe bol'nykh SD-2 sostavil 7,3 (6,9-8,0)%, a v gruppe bez SD-2 - 5,9 (5,7-6,1)% (r<0,001) (tabl. 1).

Gruppy ne razlichalis' po urovnyu obshchego kholesterina (OKhS), kholesterina lipoproteidov nizkoy (KhS LPNP) i vysokoy plotnosti (KhS LPVP) v syvorotke krovi. Uroven' triatsilglitseridov (TAG) byl dostoverno vyshe v gruppe bol'nykh s SD-2 (tabl. 1).

**Tablitsa 1 Klinicheskaya kharakteristika patsientov v gruppakh sravneniya**

|  |  |  |  |
| --- | --- | --- | --- |
| **Pokazatel'** | **Patsienty s SD-2 (n=54)** | **Patsienty bez SD-2 (n=42)** | **р** |
| Vozrast, let | 63 (56-69) | 63 (56-69) | nd |
| Pol, muzh/zhen | 32/22 | 28/14 | nd |
| SAD, mm rt.st | 136 (130-154) | 130 (126-144) | nd |
| DAD, mm rt.st.  | 80 (76-86) | 82 (76-88) | nd |
| ChSS, ud/min | 60 (56-64) | 56 (51-62) | р<0,05 |
| Glyukoza venoznoy krovi natoshchak, mmol'/l | 8,5 (6,7-9,6) | 5,4 (5,0-5,8) | р<0,001 |
| Glikirovannyy gemoglobin, % | 7,3 (6,9-8,0) | 5,9 (5,7-6,1) | р<0,001 |
| OKhS, mmol'/l  | 4,7 (4,0-6,1) | 4,7 (4,0-5,2) | nd |
| KhS LPNP, mmol'/l | 2,7 (2,3-4,1) | 2,8 (2,3-3,4) | nd |
| KhS LPVP, mmol'/l | 1,1 (1,0-1,2) | 1,1 (1,0-1,3) | nd |
| TAG, mmol'/l  | 1,7 (1,4-2,5) | 1,4 (1,1-1,6) | р<0,001 |

***Primechanie:*** Dannye predstavleny v vide: mediana (25-y i 75-y protsentili). SAD i DAD - sistolicheskoe i diastolicheskoe arterial'noe davlenie, sootvetstvenno; ChSS-chastota serdechnykh sokrashcheniy; OKhS - obshchiy kholesterin, KhS LPNP – kholesterin lipoproteinov nizkoy plotnosti, KhS LPVP- kholesterin lipoproteinov vysokoy plotnosti, TAG- triatsilglitseridy.

***Pokazateli regional'noy i lokal'noy zhestkosti sosudov u bol'nykh IBS i AG s SD-2***

 Povyshenie regional'noy zhestkosti aorty (sosud elasticheskogo tipa) u bol'nykh s SD-2 po sravneniyu s gruppoy bez SD-2 bylo otmecheno pri ispol'zovanii razlichnykh metodov otsenki skorosti rasprostraneniya pul'sovoy volny (SPV) v aorte (tabl. 2, ris. 1). Karotidno-femoral'naya SPV (SPVkf) u bol'nykh SD-2 byla na 16% vyshe, chem v gruppe sravneniya: 11,9 (10,6-14,0) m/s protiv 10,0 (9,3-11,3) m/s, sootvetstvenno (r<0,001). SPV, izmerennaya s pomoshch'yu UZ-metoda (SPVAo) u bol'nykh s SD-2 byla na 26% vyshe, chem v gruppe bol'nykh bez SD-2: 9,5 (8,0-10,9) m/s i 7,0 (5,9-8,7) m/s, sootvetstvenno (r<0,001). Po rezul'tatam ob"emnoy sfigmografii otmecheno dostovernoe povyshenie pleche-lodyzhechnoy SPV (SPVpl) u bol'nykh s SD-2 na 8,2% (r<0,05) po sravneniyu s gruppoy bez SD-2. Mediana CAVI byla neskol'ko vyshe v gruppe bol'nykh s SD-2 po sravneniyu s gruppoy bez SD-2, odnako, statisticheski znachimykh mezhgruppovykh razlichiy ne vyyavleno (r=0,13) (tabl. 2, ris.1).

 Pokazateli lokal'noy zhestkosti obshchikh sonnykh arteriy (sosudy myshechno-elasticheskogo tipa) byli vyshe v gruppe SD-2, po sravneniyu s gruppoy bez SD-2: indeks zhestkosti β byl vyshe na 7,6% (r<0,05), a pokazatel' Er - na 12% (r<0,05) (tabl. 2, ris.1).

Pokazateli lokal'noy zhestkosti plechevoy arterii (sosud myshechnogo tipa) takzhe byli dostoverno vyshe v gruppe bol'nykh SD-2: indeks zhestkosti β - na 22% (r<0,01), Er - na 14 % (r<0,01) (tabl. 2, ris.1). Po pokazatelyam zhestkosti luchevoy arterii (indeksu zhestkosti β i pokazatelyu Er) razlichiy vyyavleno ne bylo (r=0,3 i 0,2 sootvetstvenno) (tabl. 2, ris.1).

Takim obrazom, v gruppe bol'nykh s SD-2 nablyudalos' dostovernoe povyshenie zhestkosti sosudov elasticheskogo (aorta), myshechno-elasticheskogo (OSA) i myshechnogo (PA) tipov po sravneniyu s gruppoy bez SD-2 (tabl. 2, ris.1).

**Tablitsa 2 Zhestkost' sosudov, pokazateli pul'satsionnosti, funktsional'nogo sostoyaniya pochek i pochechnogo krovotoka u bol'nykh SSZ s SD-2 i bez SD-2**

|  |  |  |  |
| --- | --- | --- | --- |
| **Pokazatel'** | **Patsienty s SD-2 (n=54)** | **Patsienty bez SD-2 (n=42)** | **р** |
| **Pokazateli zhestkosti sosudistoy stenki** |
| SPVkf (m/s) | 11,9 (10,6-14,0) | 10,0 (9,3-11,3) | <0,001 |
| SPVAo (m/s)  | 9,5 (8,0-10,9) | 7,0 (5,9-8,7) | <0,001 |
| SPVpl (m/s)  | 14,6 (12,9-16,2) | 13,4 (11,9-15,3) | <0,05 |
| CAVI  | 8,9 (7,9-9,5) | 8,4 (7,9-9,1) | nd |
| Indeks β OSA  | 10,5 (8,9-13,6) | 9,7 (8,3-11,5) | <0,05 |
| Ep OSA | 149,2 (121,8-195,5) | 131,5 (111,3-157,7) | <0,05 |
|  Indeks β PA  | 14,1 (11,4-19,6) | 11 (7,8-15,4) | <0,01 |
| Ep PA | 180 (147-255,5) | 155 (108-204) | <0,05 |
| Indeks β LA | 22,6 (18,7-32,2) | 22,1 (16,2-29,9) | nd |
| Ep LA | 316,8 (244,5-454,8) | 308 (204-391) | nd |
| **Pokazateli pul'satsionnosti tsentral'noy gemodinamiki i PAD, izmerennogo na plechevoy arterii** |
| tsPAD, mm rt.st. | 49 (42-58) | 43 (39-50) | <0,05 |
| Aix75 | 24 (20-29) | 24,5 (19-29) | nd |
| pPAD, mm rt.st. | 56 (48-66) | 52 (46-58) | <0,05 |
| **Laboratornye pokazateli funktsii pochek** |
| Kreatinin (mkmol'/l) | 74,2 (69,6-83,4) | 78,2 (69,0-85,6) | nd |
| Klirens kreatinina (ml/min) | 110,5 (90,9-137,7) | 101,9 (88,7-125,9) | nd |
| Otnoshenie al'bumin/kreatinin (mg/mol' Cr) | 0,5 (0,01-1,7) | 0,1 (0-0,6) | <0,05 |
| Tsistatin C (mg/l) | 1,34 (1,25-1,53)  | 1,34 (1,27-1,49) | nd |
| SKFts (ml/min/1,73m2 ) | 60 (52-64,7) | 59,5 (52,2-63,5) | nd |
| **Dannye UZ-issledovaniya pochechnogo krovotoka** |
| RI | 0,8 (0,7-0,8) | 0,7 (0,7-0,8) | <0,01 |
| PI | 1,7 (1,4-1,9) | 1,4 (1,2-1,5) | <0,001 |

***Primechanie:*** n- kolichestvo patsientov. Rezul'taty predstavleny v vide mediany, 25-go i 75-go protsentiley. SPV kf – skorost' pul'sovoy volny na karotidno-femoral'nom uchastke; SPV Ao – skorost' pul'sovoy volny v niskhodyashchey aorte; SPV pl – skorost' pul'sovoy volny na pleche-lodyzhechnom segmente; CAVI – kardio-lodyzhechnyy sosudistyy indeks zhestkosti; OSA- obshchaya sonnaya arteriya; PA – plechevaya arteriya; LA- luchevaya arteriya; β -indeks zhestkosti; Ep -modul' uprugosti; pPAD – pul'sovoe arterial'noe davlenie (PAD), izmerennoe na plechevoy arterii; tsPAD – tsentral'noe PAD; Aix75- indeks augmentatsii, skorrektirovannyy po ChSS; SKFts - skorost' klubochkovoy fil'tratsii, rasschitannaya po tsistatinu-C; PI-pul'satsionnyy indeks; RI - rezistivnyy indeks.

V gruppe SD-2 otmecheno dostovernoe povyshenie pokazatelya tsentral'nogo SAD po sravneniyu s gruppoy bez SD-2: 123 (113-135) mm rt.st. protiv 114 (106-128) mm rt.st., sootvetstvenno (r<0,05).

V gruppe bez SD-2 otmechena korrelyatsionnaya vzaimosvyaz' SPVkf s urovnem perifericheskogo SAD: rs=0,42; r<0,05.

V gruppe bez SD-2 obnaruzhena korrelyatsionnaya vzaimosvyaz' vozrasta s indeksom zhestkosti CAVI (rs=0,66; r<0,001) i s pokazatelem SPVpl (rs=0,62; r<0,001).

Sredi vsekh obsledovannykh bol'nykh s IBS i AG (96 patsientov) otmechena vzaimosvyaz' zhestkosti sosudov razlichnykh tipov s pokazatelyami uglevodnogo obmena: urovnya glikemii natoshchak – s pokazatelyami regional'noy zhestkosti aorty SPVkf i SPVAo (rs=0,28; r<0,01 i rs=0,20; r<0,05, sootvetstvenno). Takzhe otmechena vzaimosvyaz' urovnya glikemii s pokazatelyami zhestkosti arteriy myshechno-elasticheskogo i myshechnogo tipov: s indeksom zhestkosti β OSA (rs=0,22; r <0,05), s modulem uprugosti Er OSA (rs=0,23; r<0,05), s indeksom zhestkosti β PA (rs=0,24; r <0,05) i modulem uprugosti Er PA (rs =0,22; r <0,05). Uroven' HbA1c korreliroval s pokazatelyami kak regional'noy zhestkosti aorty, tak i lokal'noy zhestkosti arteriy razlichnykh tipov: s SPVkf (rs=0,30; r<0,01), s modulem uprugosti Er OSA (rs=0,22; r<0,05), s indeksom zhestkosti β PA (rs=0,28; r <0,01), modulem uprugosti Er PA (rs =0,26; r <0,05) i modulem uprugosti Er LA (rs=0,23; r <0,05).

Sredi bol'nykh s SD-2 otmechena korrelyatsionnaya vzaimosvyaz' prodolzhitel'nosti zabolevaniya SD-2 s SPVpl (rs=0,36; r<0,05) i s pokazatelyami lokal'noy zhestkosti OSA: indeksom β (rs=0,29; r<0,05) i Er OSA (rs=0,27; r<0,05). Krome togo, v gruppe bol'nykh SD-2 vyyavlena vzaimosvyaz' urovnya HbA1c s pokazatelem lokal'noy zhestkosti luchevoy arterii: indeksom zhestkosti β LA (rs=0,28; r<0,05).

V gruppe sravneniya (bez SD-2) vzaimosvyazi pokazateley uglevodnogo obmena s zhestkost'yu sosudov ne otmecheno.

Pokazateli pul'satsionnosti tsentral'noy gemodinamiki i PAD, izmerennogo na plechevoy arterii

Po dannym applanatsionnoy tonometrii s ispol'zovaniem pribora SphygmoCor pokazateli tsentral'nogo PAD (tsPAD) byli vyshe v gruppe bol'nykh s SD-2 na 12%, po sravneniyu s gruppoy bez SD-2 (r<0,05) (tabl. 2). Dostovernykh razlichiy po pokazatelyu indeksa augmentatsii (Aix75) ne otmecheno (r=0,93) (tabl. 2).

Pokazateli PAD, izmerennogo na plechevoy arterii, byli vyshe v gruppe SD-2 na 7% po sravneniyu s gruppoy bez SD-2 (r<0,05) (tabl. 2).

Pokazateli funktsii pochek i pochechnogo krovotoka u obsledovannykh patsientov

Gruppy patsientov ne razlichalis' po pokazatelyam fil'tratsionnoy funktsii pochek: urovnyu kreatinina i tsistatina S v syvorotke krovi, klirensu kreatinina i velichine SKF, rasschitannoy po tsistatinu S (SKFts) (tabl. 2). V obeikh gruppakh (s SD-2 i bez SD-2) otmechalas' otritsatel'naya korrelyatsionnaya vzaimosvyaz' klirensa kreatinina s vozrastom patsientov (rs=-0,65; r<0,001 i rs=-0,56; r<0,001, sootvetstvenno). Krome togo, v gruppe bol'nykh s SD-2 proslezhivalas' otritsatel'naya vzaimosvyaz' klirensa kreatinina s prodolzhitel'nost'yu zabolevaniya SD-2: rs=-0,27; r<0,05.

V gruppe bol'nykh s SD-2 u 17% patsientov vyyavlena MAU, togda kak v gruppe bol'nykh bez SD-2 MAU ne obnaruzhena (r<0,01). Velichina MAU v gruppe bol'nykh s SD-2 byla v 5 raz vyshe, chem u bol'nykh bez SD-2: 0,5 (0,01 – 1,7) protiv 0,1 (0 – 0,6) mg na mmol' kreatinina (r<0,05).

Pri issledovanii pokazateley pochechnogo krovotoka (RI, PI) pri nalichii SD-2 bylo otmecheno uvelichenie RI na 12,5% (r<0,01) i PI na 17,7% (r<0,001) po sravneniyu s gruppoy bez SD-2 (tabl. 2).

 Krome togo, v gruppe bol'nykh s SD-2 otmechena vzaimosvyaz' pokazateley RI i PI s prodolzhitel'nost'yu zabolevaniya SD-2: rs=0,37; r<0,01 i rs=0,30; r<0,05, sootvetstvenno.

V gruppe sravneniya (u bol'nykh bez SD-2) otmechena vzaimosvyaz' pokazateley RI i PI s vozrastom patsientov: rs=0,47; r<0,01 i rs=0,41; r<0,01, sootvetstvenno.

Pri analize dannykh vsekh vklyuchennykh v issledovanie patsientov (96 chelovek) obnaruzhena vzaimosvyaz' velichiny PI s pokazatelyami uglevodnogo obmena: urovnem glyukozy venoznoy krovi natoshchak i s urovnem HbA1c (rs=0,32; r<0,01 i rs=0,23; r<0,05, sootvetstvenno). V to zhe vremya, vzaimosvyaz' pokazatelya RI otmechena tol'ko s urovnem glyukozy venoznoy krovi (rs=0,25; r<0,05), a s urovnem HbA1c dostovernoy vzaimosvyazi vyyavleno ne bylo (rs=0,16; r=0,13).

Vzaimosvyaz' pokazateley zhestkosti magistral'nykh arteriy, funktsii pochek i pochechnogo krovotoka u obsledovannykh patsientov

V obeikh gruppakh patsientov, kak s SD-2, tak i bez SD-2, vyyavleny vzaimosvyazi pokazateley zhestkosti magistral'nykh arteriy s pokazatelyami funktsional'nogo sostoyaniya pochek i pochechnogo krovotoka.

V gruppe bol'nykh s SD-2 pokazateli regional'noy zhestkosti aorty (sosud elasticheskogo tipa) byli vzaimosvyazany s pokazatelyami kak fil'tratsionnoy funktsii pochek, tak i pochechnogo krovotoka. Otmechalas' vzaimosvyaz' kontsentratsii kreatinina, tsistatina-S v syvorotke krovi, SKFts s pokazatelyami zhestkosti aorty: SPVkf, SPVpl i pokazatelem CAVI. Pokazateli pochechnogo krovotoka (RI, PI) takzhe byli vzaimosvyazany s zhestkost'yu aorty: s SPVAo, SPVpl i pokazatelem CAVI (tabl. 3). Sosudy myshechno-elasticheskogo i myshechnogo tipov takikh vzaimosvyazey ne prodemonstrirovali.

**Tablitsa 3 Korrelyatsionnye vzaimosvyazi pokazateley funktsional'nogo sostoyaniya pochek i pochechnogo krovotoka s regional'noy zhestkost'yu aorty i pokazatelyami pul'satsionnosti u bol'nykh IBS i AG s SD-2**

|  |  |  |  |  |  |  |
| --- | --- | --- | --- | --- | --- | --- |
| **Pokazateli** | **SPVAo** | **SPVpl**  | **CAVI** | **SPVkf**  | **pPAD**  | **tsPAD** |
| **Kreatinin** |  | rs=0,37р<0,05 |  |  |  |  |
| **Tsistatin C** |  | rs=0,48р<0,05 | rs=0,5р<0,05 | rs=0,44р<0,05 |  |  |
| **SKFts** |  | rs=-0,48р<0,05 | rs=-0,47р<0,05 | rs=-0,44р<0,05 |  |  |
| RI | rs=0,34р<0,01 | rs=0,38р<0,05 | rs=0,37р<0,05 |  | rs=0,28р<0,05 | rs=0,32р<0,05 |
| PI | rs=0,28р<0,05 | rs=0,40р<0,05 | rs=0,37р<0,05 |  | rs=0,35р<0,01 | rs=0,43р<0,01 |

***Primechanie:*** SKFts - skorost' klubochkovoy fil'tratsii, rasschitannaya po tsistatinu-C; RI - rezistivnyy indeks; PI-pul'satsionnyy indeks; SPV kf – skorost' pul'sovoy volny na karotidno-femoral'nom uchastke; SPV Ao – skorost' pul'sovoy volny v niskhodyashchey aorte; SPV pl – skorost' pul'sovoy volny na pleche-lodyzhechnom segmente; CAVI – kardio-lodyzhechnyy sosudistyy indeks zhestkosti; pPAD – pul'sovoe arterial'noe davlenie (PAD), izmerennoe na plechevoy arterii; tsPAD – tsentral'noe PAD.

V gruppe bol'nykh bez SD-2 otmechena vzaimosvyaz' indeksa zhestkosti β OSA s urovnem tsistatina S (rs=0,49; r<0,05).

Vzaimosvyaz' pokazateley pul'satsionnosti tsentral'noy gemodinamiki i PAD, izmerennogo na plechevoy arterii, s pokazatelyami funktsii pochek i pochechnogo krovotoka u obsledovannykh patsientov

Pri nalichii SD-2 otmechena vzaimosvyaz' parametrov pochechnogo krovotoka (RI i PI) s pokazatelyami PAD, izmerennogo na plechevoy arterii i tsentral'nogo PAD (tabl. 3).

V gruppe sravneniya (bez SD-2) takzhe proslezhivalas' vzaimosvyaz' pokazatelya pochechnogo krovotoka (RI) s indeksom augmentatsii (Aix75) (rs=0,45; r<0,05). Krome togo, otmechena vzaimosvyaz' velichiny MAU s urovnem tsentral'nogo PAD (rs=0,54; r<0,01).

 **Obsuzhdenie**

 V nashem issledovanii izuchalis' pokazateli zhestkosti sosudov razlichnykh tipov: regional'naya zhestkost' aorty (sosud elasticheskogo tipa), lokal'naya zhestkost' OSA (sosud myshechno-elasticheskogo tipa), plechevoy i luchevoy arteriy (sosudy myshechnogo tipa). Pri SD-2 nablyudalos' uvelichenie zhestkosti sosudov vsekh tipov: aorty, OSA i PA (tabl. 2, ris.1), chto ranee otmechalos' i drugimi issledovatelyami [2,4,7,24]. Znachimykh mezhgruppovykh razlichiy po pokazatelyu zhestkosti LA vyyavleno ne bylo. Znachitel'noe povyshenie zhestkosti PA u bol'nykh SD-2 i otsutstvie znachimykh mezhgruppovykh razlichiy po velichine zhestkosti LA mozhet byt' ob"yasneno tem, chto v silu strukturnykh osobennostey LA (bolee razvityy gladkomyshechnyy sloy) funktsional'nyy komponent formirovaniya sosudistoy zhestkosti (izmenenie tonusa arterii) bolee vyrazhen u LA, poetomu velichina lokal'noy zhestkosti LA mozhet byt' podverzhena znachitel'nym kolebaniyam.

Poluchennye nami rezul'taty o vzaimosvyazi vozrasta patsientov s pokazatelyami zhestkosti magistral'nykh arteriy (SPVpl, CAVI) v gruppe bez SD-2 i otsutstvie takovoy vzaimosvyazi v gruppe SD-2, soglasuyutsya s dannymi literatury [24]. Eta osobennost' ob"yasnyaetsya tem, chto vliyanie vozrasta na pokazateli zhestkosti sosudov elasticheskogo tipa pri nalichii SD-2 skazyvaetsya tol'ko v otnositel'no molodom vozraste, a s godami niveliruetsya, ustupaya mesto vliyaniyu povrezhdayushchikh faktorov, svyazannykh s SD-2, v to vremya kak v gruppe bol'nykh bez SD-2 vliyanie vozrasta na pokazateli zhestkosti sosudistoy stenki sokhranyaetsya.

Regional'nuyu zhestkost' aorty izuchali s ispol'zovaniem neskol'kikh diagnosticheskikh metodov. Maksimal'nye mezhgruppovye razlichiya po velichine regional'noy zhestkosti aorty byli polucheny po rezul'tatam UZ-metoda: u bol'nykh s SD-2 SPVAo byla vyshe na 26%, togda kak SPVkf byla vyshe na 16%, a naimen'shie mezhgruppovye razlichiya nablyudalis' po rezul'tatam ob"emnoy sfigmografii: velichina SPVpl byla vyshe v gruppe bol'nykh s SD-2 na 8,2%. Ne bylo otmecheno mezhgruppovykh razlichiy po pokazatelyu CAVI. UZ-metod pozvolyaet registrirovat' SPV neposredstvenno v niskhodyashchem otdele aorty, togda kak pri ispol'zovanii drugikh metodov sushchestvennyy vklad v poluchennye rezul'taty vnosyat drugie uchastki sosudistogo rusla: v sluchae otsenki SPVkf – zhestkost' naruzhnoy sonnoy i bedrennoy arteriy, a pri otsenke SPV metodom ob"emnoy sfigmografii (SPVpl) okhvatyvaetsya naibolee protyazhennyy uchastok sosudistogo rusla: ot plechevoy arterii do arteriy goleni. Poluchennye rezul'taty pozvolyayut vyskazat' predpolozhenie o tom, chto vklad SD-2 v razvitie povyshennoy arterial'noy zhestkosti u bol'nykh s SSZ (AG i IBS) yavlyaetsya maksimal'nym na urovne aorty (sosud elasticheskogo tipa).

Pri ispol'zovanii lyubogo metoda opredeleniya sosudistoy zhestkosti velichina SPV zavisit ot urovnya AD. Uroven' perifericheskogo AD v gruppe bol'nykh s SD-2 znachimo ne otlichalsya ot gruppy sravneniya, odnako, mediana SAD u bol'nykh s SD-2 vse zhe byla na 6 mm rt.st. vyshe, chem v gruppe bol'nykh bez SD-2. Krome togo, po pokazatelyam tsentral'nogo AD otmecheno dostovernoe povyshenie SAD v gruppe s SD-2 po sravneniyu s gruppoy bez SD-2 (na 9 mm rt.st.). Takim obrazom, nel'zya isklyuchit' vklad urovnya AD v povyshenie SPV v gruppe bol'nykh s SD-2.

CAVI yavlyaetsya raschetnym pokazatelem, pozvolyayushchim nivelirovat' vliyanie AD v moment issledovaniya na velichinu sosudistoy zhestkosti. V nashem issledovanii mediana znacheniy CAVI byla neskol'ko vyshe v gruppe bol'nykh s SD-2 (8,9 protiv 8,4), khotya statisticheski znachimykh mezhgruppovykh razlichiy ne vyyavleno. Vmeste s tem, velichina CAVI, kak i SPVpl, opredelyaetsya ne tol'ko zhestkost'yu aorty, no i zhestkost'yu drugikh krupnykh sosudov, togda kak u bol'nykh SSZ s SD-2 maksimal'nye razlichiya po velichine sosudistoy zhestkosti, po-vidimomu, nablyudayutsya na urovne aorty.

Uroven' AD v moment issledovaniya yavlyaetsya vazhnoy determinantoy velichiny arterial'noy zhestkosti (SPV). Odnako, s drugoy storony, progressiruyushchee uvelichenie zhestkosti aorty, snizhenie ee dempfiruyushchey funktsii privodit k povysheniyu urovnya sistolicheskogo i pul'sovogo AD, chto lezhit v osnove mekhanizma razvitiya izolirovannoy sistolicheskoy gipertenzii [25]. V nashem issledovanii gruppy patsientov znachimo ne razlichalis' po urovnyu perifericheskogo SAD na plechevoy arterii, odnako, po urovnyu perifericheskogo PAD bylo otmecheno dostovernoe razlichie s povysheniem ukazannogo pokazatelya v gruppe SD-2 po sravneniyu s gruppoy bez SD-2. V svoyu ochered', pokazateli tsentral'nogo SAD i PAD (tsSAD i tsPAD) byli takzhe znachimo vyshe u bol'nykh s SD-2 po sravneniyu s gruppoy bez SD-2, chto otmechali ranee i drugie issledovateli [26]. V rezul'tate povysheniya zhestkosti aorty uvelichivaetsya skorost' provedeniya pul'sovykh voln na periferiyu s posleduyushchim vozvratom otrazhennykh voln i uvelicheniem (augmentatsiey) tsentral'nogo AD. Tsentral'noe PAD kosvenno ukazyvaet na povyshenie pul'satsionnosti krovotoka. Izvestno, chto pokazatel' tsentral'nogo PAD yavlyaetsya prediktorom neblagopriyatnykh serdechno-sosudistykh iskhodov u bol'nykh SD-2 [26].

Po dannym Hashimoto J. i Ito S. tsPAD i tsSAD yavlyalis' prediktorami razvitiya al'buminurii u bol'nykh s AG, 20% iz kotorykh imeli SD-2 [27]. V svoyu ochered', v nashem issledovanii byla otmechena vzaimosvyaz' velichiny MAU s urovnem tsentral'nogo PAD v gruppe bol'nykh bez SD-2. Krome togo, v obeikh gruppakh patsientov proslezhivalas' vzaimosvyaz' pokazateley pul'satsionnosti tsentral'noy gemodinamiki (tsentral'nogo PAD, Aix75) i PAD, izmerennogo na plechevoy arterii, s pokazatelyami pochechnogo krovotoka: indeksom rezistivnosti (RI) i pul'satsionnym indeksom (PI). Predpolagayut, chto PI kharakterizuet stepen' povrezhdayushchego vozdeystviya pul'sovykh voln na sosudy pochek, a povyshenie RI schitaetsya naibolee rannim markerom porazheniya pochek i vyyavlyaetsya u bol'nykh s nedavno diagnostirovannymi AG ili SD-2 eshche do poyavleniya MAU [28,29]. V gruppe bol'nykh s SD-2 PI byl vyshe na 17,7%, a RI – na 12,5% po sravneniyu s bol'nymi bez SD-2.

Kak u bol'nykh s SD-2, tak i u bol'nykh bez SD-2, otmechena vzaimosvyaz' pokazateley zhestkosti magistral'nykh arteriy s funktsional'nym sostoyaniem pochek. V gruppe bol'nykh s SD-2 uvelichenie zhestkosti aorty assotsiirovalos' s bolee nizkimi pokazatelyami fil'tratsionnoy funktsii pochek (po urovnyu kreatinina krovi, tsistatina S, SKFts) i s izmeneniem kharakteristik pochechnogo krovotoka (povysheniem RI i PI), chto soglasuetsya s rezul'tatami drugikh issledovaniy [27,29]. Dannye o nalichii vzaimosvyazi mezhdu zhestkost'yu aorty i funktsional'nym sostoyaniem pochek byli polucheny i drugimi issledovatelyami. Bouchi R. i soavtory otmetili vzaimosvyaz' zhestkosti aorty (po pokazatelyu SPVkf) s klirensom kreatinina i MAU u bol'nykh s SD-2 [30]. V rabote Hamano K. i soavtorov nablyudalas' vzaimosvyaz' RI pochechnogo krovotoka s SPVpl u patsientov s SD-2 bez priznakov gemodinamicheski znachimogo stenozirovaniya pochechnykh arteriy [31]. Po dannym issledovaniya J-TOPP povyshenie SPVpl bylo nezavisimym faktorom riska razvitiya MAU u bol'nykh AG bez SD-2 [32].

Vmeste s tem, v ryade issledovaniy byla otmechena vzaimosvyaz' funktsional'nogo sostoyaniya pochek s zhestkost'yu ne tol'ko aorty, no i drugikh arteriy [33,34]. Zhan W.W. i soavtory otmetili vzaimosvyaz' pokazateley zhestkosti OSA s MAU u patsientov s SD-2 [34]. V issledovanii Hoorn u patsientov s SD-2 otmechena vzaimosvyaz' pokazateley funktsii pochek (klirensa kreatinina i MAU) s zhestkost'yu arteriy razlichnykh tipov: aorty, OSA, PA. Pri snizhenii klirensa kreatinina otmechalos' umen'shenie pokazateley rastyazhimosti OSA i PA, a uvelichenie MAU assotsiirovalos' s umen'sheniem rastyazhimosti OSA i s umen'sheniem vremeni rasprostraneniya pul'sovoy volny na karotidno-femoral'nom uchastke [11]. Po rezul'tatam nashego issledovaniya vzaimosvyaz' funktsional'nogo sostoyaniya pochek i pochechnogo krovotoka v gruppe bol'nykh SD-2 nablyudalas' tol'ko s pokazatelyami zhestkosti aorty. V gruppe bol'nykh bez SD-2, krome togo, byla vyyavlena vzaimosvyaz' indeksa zhestkosti β OSA s urovnem tsistatina S. Vzaimosvyazi funktsional'nogo sostoyaniya pochek i pokazateley zhestkosti arteriy myshechnogo tipa v nashem issledovanii vyyavleno ne bylo. V svyazi s etim sleduet otmetit', chto v nashem issledovanii, soglasno kriteriyam vklyucheniya, uchastvovali tol'ko patsienty s uzhe imeyushchimisya klinicheskimi proyavleniyami ateroskleroza (IBS). Ranee Hashimoto J. i soavtory ne otmetili vzaimosvyazi pokazateley funktsii pochek i pochechnogo krovotoka s zhestkost'yu arteriy myshechno-elasticheskogo i myshechnogo tipov u patsientov s SSZ bez SD-2 [27].

Takim obrazom, po rezul'tatam nashego issledovaniya mozhno sdelat' vyvod, chto vzaimosvyaz' zhestkosti aorty, OSA s funktsional'nym sostoyaniem pochek i pochechnogo krovotoka proslezhivaetsya i u bol'nykh s uzhe imeyushchimisya klinicheskimi proyavleniyami ateroskleroza, a vzaimosvyazi disfunktsii pochek s zhestkost'yu arteriy myshechnogo tipa ne nablyudaetsya.

Nablyudaemye nami i drugimi avtorami vzaimosvyazi podtverzhdayut nalichie obshchikh mekhanizmov razvitiya povysheniya zhestkosti arteriy razlichnykh tipov i izmeneniy funktsional'nogo sostoyaniya pochek.

Pomimo gemodinamicheskikh faktorov (povrezhdayushchee deystvie pul'sovoy volny), v osnove nablyudayushchikhsya vzaimosvyazey funktsional'nogo sostoyaniya pochek i pochechnogo krovotoka s zhestkost'yu arteriy i urovnem tsentral'nogo AD mogut lezhat' i obshchie strukturnye izmeneniya sosudistoy stenki i klubochkov pochek. Ne sluchayno nalichie priznakov dazhe nachal'nykh stadiy KhBP svyazano s povyshennym riskom razvitiya SSO [10,14]. Izmeneniya v pochkakh, kak i izmeneniya v sosudakh, u bol'nykh s AG i SD-2 proiskhodyat parallel'no pod vliyaniem agressivnykh faktorov, odnovremenno vozdeystvuyushchikh i na sosudy, i na klubochki pochek (aktivatsiya protsessov glikirovaniya, perekisnogo okisleniya, faktory vospaleniya). Pri etom intensivnost' vozdeystviya etikh faktorov u bol'nykh s SD-2, ochevidno, vyshe, chem u bol'nykh bez SD-2.

Nami, takzhe kak i drugimi avtorami, otmechena vzaimosvyaz' pokazateley zhestkosti arteriy razlichnykh tipov s pokazatelyami uglevodnogo obmena [35,36]. Obrashchaet na sebya vnimanie tot fakt, chto v kachestve gruppy sravneniya v issledovanii uchastvovali bol'nye IBS i AG bez priznakov narusheniya uglevodnogo obmena po rezul'tatam GTT (75g). Odnako, velichina HbA1 v etoy gruppe patsientov costavila 5,9 (5,7-6,1)% (mediana, 25-y i 75-y protsentili), togda kak uroven' HbA1c > 5,6% uzhe ukazyvaet na nalichie narusheniy uglevodnogo obmena (prediabeta) [37]. Takim obrazom, nesmotrya na normal'nye rezul'taty GTT(75g), bolee 75% patsientov iz gruppy sravneniya imeli skrytye narusheniya uglevodnogo obmena. Etot fakt podtverzhdaet vysokuyu rasprostranennost' narusheniy uglevodnogo obmena sredi bol'nykh SSZ, otmechennuyu v drugikh issledovaniyakh [38].

Pri otsenke vzaimosvyazi pokazateley zhestkosti sosudov razlichnykh tipov s urovnem HbA1c byli vyyavleny sleduyushchie osobennosti. V gruppe bol'nykh s SD-2, srednyaya prodolzhitel'nost' zabolevaniya diabetom u kotorykh sostavlyala 8 let, bol'shinstvo iz kotorykh uzhe poluchali sakharosnizhayushchuyu terapiyu, vzaimosvyazi zhestkosti aorty, obshchikh sonnykh arteriy s urovnem HbA1c na moment obsledovaniya vyyavleno ne bylo. Odnako, nablyudalas' vzaimosvyaz' pokazateley zhestkosti aorty i OSA s prodolzhitel'nost'yu zabolevaniya SD-2. Uroven' HbA1c u bol'nykh s SD-2 izmenyaetsya pod vliyaniem lecheniya i otrazhaet stepen' kompensatsii uglevodnogo obmena tol'ko za predshestvuyushchie 3 mesyatsa. V to zhe vremya, izmenenie zhestkosti sosudov elasticheskogo i myshechno-elasticheskogo tipov (aorta, OSA) pri SD-2 proiskhodit postepenno na protyazhenii vsego zabolevaniya, zavisit ot stepeni kompensatsii diabeta v razlichnye ego periody i imeet «nakopitel'nyy» kharakter. Vmeste s tem, v gruppe bol'nykh s SD-2 otmechalas' vzaimosvyaz' velichiny HbA1c s pokazatelyami zhestkosti LA (sosud myshechnogo tipa), chto, po-vidimomu, yavlyaetsya otrazheniem dinamichnogo izmeneniya funktsional'nogo komponenta sosudistoy zhestkosti arteriy myshechnogo tipa. Esli povyshenie zhestkosti aorty yavlyaetsya sledstviem strukturnykh izmeneniy ee stenki, to zhestkost' sosudov myshechno-elasticheskogo i myshechnogo tipov dopolnitel'no opredelyaetsya vazomotornym komponentom, na kotoryy, v svoyu ochered', okazyvayut vliyanie sostoyanie funktsii endoteliya, aktivnost' simpaticheskoy, renin-angiotenzinovoy sistem i uroven' drugikh aktivnykh mediatorov v krovotoke. Izmenenie zhestkosti sosudov razlichnykh tipov pod vliyaniem kompensatsii SD-2 trebuet dal'neyshego izucheniya. Po-vidimomu, bystree i v bol'shey stepeni pri dostizhenii kompensatsii uglevodnogo obmena mozhno ozhidat' izmeneniya pokazateley zhestkosti arteriy myshechnogo tipa.

Takim obrazom, v nashem issledovanii pri nalichii SD-2 otmecheno povyshenie pokazateley zhestkosti sosudov razlichnykh tipov, s maksimal'nym povysheniem zhestkosti na urovne aorty. U bol'nykh SD-2 nablyudalos' povyshenie pokazateley pul'satsionnosti tsentral'noy gemodinamiki i ikh vzaimosvyaz' s pokazatelyami pochechnogo krovotoka. Zhestkost' aorty i obshchikh sonnykh arteriy assotsiirovana s nachal'nymi priznakami disfunktsii pochek, chto, po-vidimomu, ob"yasnyaetsya obshchnost'yu patogeneticheskikh mekhanizmov, lezhashchikh v osnove razvitiya kak makro-, tak i mikroangiopatii.

*Avtory deklariruyut otsutstvie konflikta (dvoystvennosti) interesov pri napisanii dannoy rukopisi.*

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***Svedeniya ob avtorakh***

Oskola Elena Vasil'evna - aspirant otd. angiologii Instituta klinicheskoy kardiologii im. A. L. Myasnikova. E-mail: oelen2010@yandex.ru

Shubina Anna Timofeevna – kand. med. nauk, nauch. sotr. otd. angiologii Instituta klinicheskoy kardiologii im. A. L. Myasnikova.

Zairova Alsu Rafkhatovna – kand. med. nauk, nauch. sotr. otdela novykh metodov diagnostiki Instituta klinicheskoy kardiologii im. A. L. Myasnikova.

Andreevskaya Marina Vladimirovna – ml. nauch. sotr. lab. ul'trazvukovykh metodov issledovaniya otdela novykh metodov diagnostiki Instituta klinicheskoy kardiologii im. A. L. Myasnikova.

Bogieva Roksana Mirabovna – kand. med. nauk, ml. nauch. sotr. lab. ul'trazvukovykh metodov issledovaniya otdela novykh metodov diagnostiki Instituta klinicheskoy kardiologii im. A. L. Myasnikova.

Pogorelova Ol'ga Aleksandrovna – kand. med. nauk, st. nauch. sotr. otdela novykh metodov diagnostiki Instituta klinicheskoy kardiologii im. A. L. Myasnikova.

Bolotina Marina Grigor'evna – vrach-endokrinolog Instituta klinicheskoy kardiologii im. A. L. Myasnikova.

Balakhonova Tat'yana Valentinovna – d-r med. nauk, prof. otdela novykh metodov diagnostiki Instituta klinicheskoy kardiologii im. A. L. Myasnikova.

Rogoza Anatoliy Nikolaevich – d-r biol. nauk, prof., rukovoditel' otd. novykh metodov diagnostiki Instituta klinicheskoy kardiologii im. A. L. Myasnikova.

Karpov Yuriy Aleksandrovich – d-r med. nauk, prof., rukovoditel' otd. angiologii Instituta klinicheskoy kardiologii im. A. L. Myasnikova, zam. General'nogo Direktora Rossiyskogo kardiologicheskogo nauchno-proizvodstvennogo kompleksa MZ RF.