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O‘ZBEKISTON TERAPIYA AXBOROTNOMASI

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METABOLIK ASSOTSIRLANGAN JIGAR YOG' KASALLIGI: ZAMONAVIY MUAMMOLAR

DAMINOVA L.T., MUXIDDINOVA N.Z.

Toshkent davlat stomatologiya instituti

РЕЗЮМЕ

МЕТАБОЛИЧЕСКИ АССОЦИИРОВАННАЯ ЖИРОВАЯ БОЛЕЗНЬ ПЕЧЕНИ

Даминова Л.Т., Мухиддинова Н.З.

Ташкентский государственный стоматологический институт

Метаболически ассоциированная жировая болезнь печени (МАЖБП) – это широко распространенное хроническое заболевание, характе- ризующееся повышенной аккумуляцией жира в печени, в основе которого лежит дисфункция обмена веществ. Частота выявления МАЖБП в большинстве регионов мира значительно превышает 20% и имеет тенденцию к росту. Согласно современным представлениям этиология и патогенез МАЖБП рассматриваются в рамках концепции «множественных параллельных ударов». Согласно этой модели развитие и прогрессирование заболевания происходят в результате взаимодействия множественных генетических, средовых и адаптatsionных факторов, к которым относятся специфические генетические полиморфизмы (например, гена PNPLA3) и эпигенетические модификatsii, характер диеты (например, высокое потребление насыщенных жиров и фруктозы), гиподинамия, ожирение, инсулинорезистентность, дисрегуляция продукции адипокинов, липотоксичность, окислительный стресс, дисбиоз кишечной микробиоты (синдром избыточного бактериального роста в тонкой кишке). В качестве медикаментозных методов лечения patsientam с МАЖБП рекомендовано снижение массы тела (в случае наличия избыточной массы тела или ожирения), редукция потребления насыщенных жирных кислот и фруктозы, а также включение в patsion достаточного количества омега- 3-полиненасыщенных жирных кислот и пищевых волокон (псиллиума). Фармакотерапия МАЖБП должна быть направлена на коррекцию инсулинорезистентности, улучшение функции печени и снижение риска ассоциированных заболеваний.

Ключевые слова: метаболически ассоциированная жировая болезнь печени, неалкогольная жировая болезнь печени, неалкогольный стеатогепатит, стеатогепатит, стеатоз.

SUMMARY

METABOLICALLY ASSOCIATED FATTY LIVER DISEASE

Daminova L.T., Muhiddinova N.Z.

Tashkent state dental institute

Metabolically associated fatty liver disease (MAFLD) is a widespread chronic disease characterized by increased accumulation of fat in the liver, which is based on metabolic dysfunction. The incidence of MAFLD is well over 20% in most regions of the world and is on an increasing trend. Current thinking considers the etiology and pathogenesis of MAFLD under the concept of «multiple parallel blows». According to this model, the development and progression of the disease are due to the interaction of multiple genetic, environmental and adaptive factors, which include specific genetic polymorphisms (e.g., the PNPLA3 gene) and epigenetic modifications, dietary patterns (e.g. high saturated fat and fructose intake), sedentary activity, obesity, insulin resistance, dysregulation of adipokines, lipotoxicity, oxidative stress, and gut microbiota dysbiosis (small intestinal bacterial overgrowth syndrome). The basis for the diagnosis of MAFLD is the presence of proven hepatic steatosis in combination with one of the following criteria: overweight/obesity, presence of type 2 diabetes mellitus, signs of metabolic dysregulation. Nonmedicamental therapies recommended for patients with MAFLD include weight loss (if overweight or obese), reduction of saturated fatty acid and fructose intake, and inclusion of adequate amounts of omega-3 polyunsaturated fatty acids and dietary fibre (psyllium) in the diet. Pharmacotherapy of MAFLD should be aimed at correcting insulin resistance, improving liver function and reducing the risk of associated diseases.

Keywords: metabolically associated fatty liver disease, nonalcoholic fatty liver disease, nonalcoholic steatohepatitis, steatohepatitis, steatosis.

XULOSA

METABOLIK ASSOTSIRLANGAN JIGAR YOG' KASALLIGI: ZAMONAVIY MUAMMOLAR

Daminova L.T., Muxiddinova N.Z.

Toshkent davlat stomatologiya instituti

Metabolik assotsirlangan jigar yog‘ kasalligi (MAJYOK) metabolik disfunksiyaga asoslangan jigarda yog‘to‘planishining ko‘payishi bilan tavsiflangan keng tarqalgan surunkali kasallikdir. Dunyoning aksariyat mintaqalarida MAJYOKni aniqlash darajasi mintaqalar bo‘ylab 20% dan sezilarli darajada oshadi va doimiy o‘sish tendensiyasiga ega kasallik hisoblanadi. Zamonaviy tushunchalarga ko‘ra, MAJYOK etiologiyasi va patogenezi dunyo bo‘ylab «muammoning dolzarbligini, ko‘plab etiologik omillarning mavjudligini muhokama qilishga urinishlar» konsepsiyasi doirasida ko‘rib chiqilmoqda. Ushbu modelga ko‘ra, kasallikning rivojlanishi ko‘plab genetik, atrof-muhit va adaptiv omillarning o‘zaro ta’siri natijasida yuzaga keladi, ular orasida asosan parhezning tabiati. (masalan, to‘yingan yog‘lar va fruktozaning ko‘p iste’moli), jismoniy кam harakatlilik, semizlik, insulinga chidamlilikning rivojlanishi, adipokin ishlab chiqarish disregulatsiyasi, lipotoksiklik, oksidlovchi stress, ichak mikrobiota disbiyozi (ingichka ichak bakteriyalarining o‘sish sindromi va genetik polimorfizmlar (masalan, PNPLA3 geni) va epigenetik modifikatsiyalar) kabi etiologik omillar qayd etiladi. MAJYOK bilan og‘rigan bemorlarni dori vositalarsiz davolash usullari sifatida tana vaznini kamaytirish (ortiqcha vazn yoki semizlik bo‘lsa), to‘yingan yog‘ kislotalari va fruktoza iste’molini kamaytirish, shuningdek, yetarli miqdorda to‘yinmagan yog‘ kislotalarini kiritish tavsiya etiladi. MAJYOK uchun farmakoterapiya insuliga tolerantlikni davolashga, jigar faoliyatini yaxshilashga va ushbu patologiyaga bog‘liq kasalliklar xavfini kamaytirishga qaratilgan bo‘lishi kerak.

Kalit so‘zlar: metabolik assotsirlangan jigar yog‘ kasalligi, alkogolsiz jigar yog‘ kasalligi, alkogolsiz stetogepatit, steatogepatit, steatoz.

Metabolik bilan bog‘liq jigar yog‘ kasalligi (MAJYOK) metabolik disfunksiyaga asoslangan jigarda yog‘ to‘planishining ortib borishi bilan tavsiflangan keng tarqalgan surunkali kasallikdir [1–3]. Hozirgi vaqtda rivojlangan mamlakatlar aholisida MAJYOK bilan kasallanish yuqumli bo‘lmagan pandemiyaga aylanib bormoqda, uning o‘sish omillari semirish va qandli diabet bilan bog‘liq jarayondir [4, 12].

Zamonaviy tushunchalarga ko‘ra, konsepsiya doirasida MAJYOK etiologiyasi va patogenezi ko‘rib chiqiladi. [21]. Ushbu modelga ko‘ra, kasallikning rivojlanishida ko‘plab genetik, atrof-muhit va adaptiv omillarning o‘zaro ta’siri natijasida yuzaga keladi, ular orasida parhezning tabiati. (masalan, to‘yingan yog‘larni va fruktozani ko‘p iste’mol qilish), jismoniy kam harakatlilik, semizlik, insulinga chidamlilik, adipokin ishlab chiqarishni tartibga solishning buzilishi, lipotoksiklik, oksidlovchi stress, ichak mikrobiotasi disbiyozi (ingichka ichak patogen bakteriyalarining ko‘payishi sindromi) o‘ziga xos genetik polimorfizmlar (masalan, PNPLA3 geni) va epigenetik modifikatsiyalar [1, 18]. MAJYOK bilan og‘rigan har bir bemorda ushbu vositachilarning kombinatsiyasi kasallikning rivojlanishi davomida farq qilishi va dinamik ravishda o‘zgarishi mumkin [2, 3, 18]. Shu bilan birga, ushbu vositachilarning ta’siri ostida lipidlarning (triglitseridlar, erkin yog‘ kislotalari, keramidlar) dastlabki to‘planishi gepatotsitlarda sodir bo‘lib, jigar steatozining rivojlanishiga olib keladi, bu esa keyinchalik yallig‘lanish jarayoni bilan qo‘shiladi. Immunokompleks hujayralar tomonidan

organ parenximasining infiltratsiyasi natijasida kelib chiqadigan steatogepatit va keyinchalik fibrozning shakllanishi kuzatiladi. Shuni ta'kidlash kerakki, MAJYOK ko'p tizimli metabolik disfunksiyaning jigar ko'rinishi sifatida yuzaga keladi, bu nafaqat kasallikning jigar asoratlari (jigar sirzi va yoki gepatotsellyulyar karsinoma), balki o'limning asosiy sababi bo'lgan kardiometabolik hodisalarning rivojlanish xavfini oshiradi [5, 7, 8].

Ta'rif va tashxisot mezonlari rivojlanish tarixi «Yog'li jigar» atamasi birinchi marta 1836-yilda Nyukasl-apon Tayn (Buyuk Britaniya)dan Tomas Addison tomonidan spirtli ichimliklarni suiiste'mol qilgan bemorlarda jigar to'qimalarining yog'li degeneratsiyasining namoyon bo'lishini tasvirlash uchun ishlatilgan[6]. Ko'p o'tmay, 1839-yilda Vena (Avstriya) patologi Karl Rokitanskiy otopsiya namunalarini tahlil qilib, jigarda yog' to'planishi ushbu organ sirrozining rivojlanishiga sabab bo'lishi mumkin degan taxminni ilgari surdi [21]. 1884–1885-yillarda Yog'li jigar degeneratsiyasi va diabetes mellitus (DM), shuningdek, semirish o'rtasidagi bog'liqliklar birinchi marta tasvirlangan [9, 11]. Keyinchalik, 1950 va 1970-yillar oralig'ida bir nechta individual patologlar hisobot beradi. Qandli diabet bilan og'rigan ko'plab bemorlarda kuzatilgan alkogolsiz jigar yog' kasalligi va jigardagi gistopatologik o'zgarishlar o'rtasidagi o'xshashliklar tasvirlangan [10]. 1980-yilda J. Ludwig va hammualiflar tomonidan «alkogolsiz yog'li jigar kasalligi» (AYOJK) va alkogolsiz steatogepatit (ASG) atamasi taklif qilingan. Ular tomonidan Mayo klinikasida (AQSH) gistologik jihatdan alkogolli steatogepatitga o'xshash, ammo spirtli ichimliklarni suiiste'mol qilmagan bemorlarda kuzatilgan yog'li jigar kasalligining progressiv shakli tavsiflangan [16, 12]. O'shandan beri, 40 yil davomida kasallikning nomi «alkogolsiz» atamasini o'z ichiga oladi va bu alomatni asosiy differentsial diagnostika mezonlaridan biriga aylantiradi [5]. Bundan tashqari, aslida AYOJK surunkali jigar kasalliklarining boshqa etiologik variantlarini istisno qilishni talab qiladigan tashxis hisoblangan va hozirgacha qabul qilingan [13].

1-jadval

MAJYOK mezonlari

5		
Tomografiya tekshiruvi natijalari yordamida, qon biomarkerlari yoki jigarni gistologik tekshiruv natijalariga asoslanib qo'yilgan Jigar steatozi.		
Ortiqcha vazn yoki semirish (TVI≥25 kg/m^2 Bug'doy rangli bemorlarda; TVI≥23 kg/m^2 Osiyo aholisida)	Me'yoriy tana vazni (TVI < 25kg/m^2 Bug'doy rangli bemorlarda; TVI < 23 kg/m^2 Osiyo aholisida)	2-tip diabet (xalqaro mezonlarga muvofiq)

- Ikki yoki undan ortiq metabolik guruhlarning mavjudligi.
- Osiyolik erkaklar va ayollarda bel atrofi ≥102/88 cm (yoki osiyolik erkaklar va ayollarda ≥90/80 sm).
- Qon bosimi ≥130/85 mm Hg. Art. yoki maxsus dori terapiyasini qabul qilish.
- Plazma triglitseridlari darajasi ≥150 mg/dL (≥1,70 mmol/l) yoki ma'lum dori-darmonlarni qabul qilish terapiya.
- Erkaklar uchun HDL darajasi <40 mg/dL (<1,0 mmol/L) va ayollar uchun <50 mg/dL (<1,3 mmol/L) yoki maxsus dori-darmonlarni qabul qilish.
- Prediabet (ochlik glyukoza darajasi 100–125 mg/dL [5,6–6,9 mmol/l] yoki 2 soatlik glukoza darajasi ovqatdan keyin 140–199 mg/dL [7,8–11,0 mmol/L] yoki HbA darajasi 1s5,7–6,4% [39–47 mmol/mol]).
- Insulinga chidamlilik indeksi (HOMA) ≥22,5.
- C-reaktiv oqsil darajasi >2 mg/l.

So'nggi o'n yilliklarda AYOJK asosan metabolik sindrom bilan ifodalangan tizimli metabolik disfunksiya natijasi ekanligi haqida yetarlicha ishonchli dalillar to'plangan [5, 14, 18]. 2002-yildan 2017-yilgacha bo'lgan davrda ba'zi ekspertlar kasallikning nomini o'zgartirish va «alkogolsiz» atamasidan voz kechish zarurligi haqida so'zlab, «metabolik», «insulinga tolerantlik» kabi etiologik jihatdan to'g'ri variantlardan foydalanishni taklif qilishdi [15–16,1].

2020-yilda xalqaro konsensus bo'lib o'tdi Tadqiqotda 22 mamlakatdan 32 nafar ekspert ishtirok etdi, ularning maqsadi AYOJK joriy ta'rifining to'g'riligini va uni o'zgartirish zarurligini chuqur tahlil qilish edi. Ushbu konsensusning natijasi AYOJK o'rniga yangi MAJYOK atamasini kiritish bilan nomenklaturani o'zgartirish va metabolik omillarning ahamiyatini oshirish yo'nalishi bo'yicha ushbu kasallikning diagnostika mezonlarini qayta ko'rib chiqish taklifi edi [4].

2-jadval

MAJYOK tashisot mezonlari

Guruh	**Xavf omillari**
Jins va yosh omillari	MAJYOK bilan kasallanish katta yoshlilarda ortib boradi (ayniqsa, ayollarda).
Etnik kelib chiqishi	Chastotasi MAJYOK osoyoliklarda yuqori, qora tanlilarda pastroq.
Yondosh metabolik kasalliklar	Semizlik epidemiologik darajada MAJYOK uchun asosiy xavf omilidir. 2-tip diabet bilan metabolik sindrom MAJYOK bilan og‘rigan bemorlarning taxminan 1/2 qismida aniqlanadi va jigar fibrozining mustaqil darakchisi hisoblanadi.
Parhez omillari	Yuqori to‘yingan iste’moldagi yog‘lar, xolesterin va fruktoza MAJYOK xavfini oshiradi.
Genetik omillar	27% hollarda MAJYOK oilaviy klasterlash bilan tavsiflanadi. PLPLA3 genining (rs738409) yagona nukleotidli polimorfizmi MAJYOK xavfini oshiradi. APOC3 genining yagona nukleotidli polimorfizmlari (rs2854117 va rs2854116) MAJYOK xavfni oshiradi.
Ichak mikrobiotasi bilan bog‘liq o‘zgarishlar	Patogen bakterial mikrofloraning ortiqcha o‘sish sindromi MAJYOK bilan kasallangan odamlarda sezilarli darajada tez-tez uchraydi.

Yangi ta’rifga ko‘ra, MAJYOK diagnostikasi uchun asos quyidagi mezonlardan biri bilan birga tasdiqlangan jigar steatozining mavjudligi hisoblanadi: ortiqcha vazn/semizlik, insulingga tolerantlikning mavjudligi, metabolik disregulatsiya belgilarli mavjudligi [4]. Shu bilan birga, MAJYOK diagnostikasi uchun alkogolning gepatotoksik dozalarini suiiste’mol qilish faktini, shuningdek, surunkali jigar kasalliklarining boshqa etiologik variantlarini istisno qilish endi shart emas [17]. Bundan tashqari, mutaxassislar MAJYOKni yallig‘lanish faolligi darajasi va jigar fibrozining bosqichi bilan baholanadigan yagona patologik jarayon sifatida ko‘rib, klassik dixotomiyali tasnifdan (steatoz va steatogepatit) voz kechishni taklif qilishdi [2].

Epidemiologiyasi, xavf omillari va ular bilan bog‘liq kasalliklar. Hozirgi vaqtda MAJYOK eng keng tarqalgan jigar kasalliklari ro‘yxatida mustahkam o‘rin egallaydi [2, 29,30]. Yaqinda o‘tkazilgan tizimli tekshiruvga ko‘ra, dunyoning aksariyat mintaqalarida MAJYOK tarqalishi 20% dan ancha yuqori va o‘sish tendensiyasida [13]. Ushbu salbiy tendensiya diabet, semizlik, giperlipidemiya va metabolik sindrom kabi nozologiyalar bilan kasallanishning barqaror o‘sishiga asoslanadi, bu esa, o‘z navbatida, MAJYOKning darakchisi hisoblanadi [25, 30].

S. Lin va boshqalar tomonidan o‘tkazilgan tadqiqotga ko‘ra. (2020) MAJYOK bo‘yicha AJYOK diagnostikaning turli mezonlari tufayli epidemiologik tuzilishda aks ettirilgan [1]. NHANES III yagona ma’lumotlar bazasini tahlil qilishda bemorlarning 31,24% MAJYOK tashxisiga va 33,23% AJYOK tashxisiga to‘g‘ri keladi [14].

Z. Younossi va hammualiflarning dastlabki met-tahlililarga ko‘ra (2016) MAJYOKning eng yuqori tarqalish darajasi iqtisodiy rivojlangan mamlakatlarda kuzatilmoqda. M. Le va hammualliflar tomonidan yaqinda o‘tkazilgan met-tahlilda 245 ta tadqiqot natijalarini (5 milliondan ortiq kishi) jamlagan (2021) MAJYOKning umumiy global tarqalishi 29,8% (95% CI 28,6–31,1) ekanligini ko‘rsatdi. Global tendentsiyalar Rossiya Federatsiyasiga ham xosdir. Shunday qilib, 2007-yildan 2014-yilgacha bo‘lgan davrda MAJYOK bilan kasallanishning o‘sishi 10% dan ortiqni tashkil etdi (2007-yil – 27%, 2014-yil – 37,1%). Steatozning maksimal tarqalishi 70–80 yosh guruhida (34,26%), ASG – 50–59 yoshli bemorlarda (10,95%) qayd etilgan [8].

MAJYOK semizlik pandemiyasining natijasi bo‘lsa-da, bemorlarning taxminan 10–20% bu metabolik kasallikdan aziyat chekmaydi [23]. Shunday qilib, Y. Shi va boshqalarning so‘nggi met-tahliliga ko‘ra. (2020) semiz bo‘lmagan odamlarda MAJYOK tarqalishi 15,7% (95% CI 12,5–19,6%) [24] kuzatilgan.

Epidemiologik tadqiqotlar shuni ko‘rsatadiki, MAJYOK ushbu patologiyaning rivojlanishiga va prognozga moyilligiga ta’sir qiluvchi bir qator xavf omillari bilan tavsiflanadi (1-jadval) [1].Z. Younossi va hammualiflarning met-tahliliga ko‘ra. (2016) MAJYOK bilan bog‘liq eng keng tarqalgan metabolik kasalliklar semirish (51,34%; 95% CI 41,38–61,20), 2-tip diabet (22,51%; 95% CI 17,92–27,89), giperlipidemiya (69,16%);95% arterial gipertenziya – gipertenziya (39,34%; 95% va metabolik sindrom (42,54%; 95% CI 30,06–56,05) [28].

Semirib ketish epidemiologik darajada MAJYOK uchun asosiy xavf omilidir [20–21]. J. Liu va boshqa hammuliflar tomonidan met-tahlilda 116 ta tadqiqot natijalarini birlashtirgan (n = 2,667,052) (2021), ortiqcha vaznli va semirib ketgan kattalardagi MAJYOKning global tarqalishi 50,7% (95% CI 46,9–54,4) [37] ekanligini aniqladi.

Genetik xususiyatlar MAJYOKga moyillikda muhim rol o‘ynaydi [18,23]. Adiponutrin oqsilini kodlovchi PLPLA3 genining yagona nukleotidli polimorfizmi (SNP) rs738409 (I 148M) MAJYOK rivojlanish xavfining oshishi bilan bog‘liq. Met-tahlilga ko‘ra, ushbu SNP mavjudligi ASG (OR 3,26; 95% CI 2,14–4,95) va jigar fibrozisi (OR 3,25; 95% CI 2,86–3,70) rivojlanish xavfini oshiradi (Sookoians, 2011). G. Dai va boshqalarning so‘nggi met-tahlilida. 21 ta tadqiqot natijalarini (> 14 ming bemor) jamlagan (2019) PNPLA3 genining SNP (rs738409 – G

allelining mavjudligi) MAJYOK (OR 4,01; 95% CI) uchun mustaqil xavf omili ekanligini tasdiqladi (2,93–5,49) [42]. MAJYOK rivojlanishining boshqa genetik omillariga APOC3 genining SNPlari (rs2854117 va rs2854116), shuningdek, TM6SF2, MBOAT7 polimorfizmlari kiradi.

Genetik belgilarni faol o‘rganish kelajakda klinikadan oldingi bosqichda MAJYOK rivojlanish xavfi bo‘lgan guruhlarni aniqlashga va birlamchi profilaktikani o‘tkazishga imkon beradi. Shunday qilib, T. Mahfood Haddad va hammualliflar met-tahliliga ko‘ra (2017), MAJYOK yurak-qon tomir kasalliklari xususan, ishemik yurak kasalligi (OR 2,26; 95% CI 1,04–4,92 p < 0,001) va ishemik insult (OR 2,09; 95% CI 1,46–2,98; p < 0,001) [46]. (OR 1,77; 95% CI 1,26–2,48), rivojlanishi uchun xavf omilidir, Ushbu ma’lumotlar D. Kapuria va hammualiflar tomonidan ham met-tahlil bilan tasdiqlangan. (2018), MAJYOK ateroskleroz uchun xavf omili ekanligini ko‘rsatadigan 12 ta tadqiqot (>16 ming bemor) natijalarini jamlagan (OR 1,64; 95% CI 1,42–1,89) [6]. Yurak-qon tomir tizimi kasalliklariga qo‘shimcha ravishda, MAJYOK gepatobiliar tizim kasalliklarining rivojlanish ehtimolini oshiradi. Shunday qilib, V.Jaruvongvanich va hammualiflarning met-tahlilida (2016) ushbu patologiyasi bo‘lgan bemorlarda xolelitiaz (GSD) rivojlanish xavfining oshishini qayd etdi (OR 1,55; 95% CI 1,31–1,82). Xuddi shunday, MAJYOK ekstragepatik xolangiokarsinoma rivojlanishi uchun xavf omilidir (OR 2,24; 95% CI 1,58–3,17) [16]. J.Stine va hammualiflarning meta-tahliliga ko‘ra. (2018) sirrozdan oldingi bosqichda MAJYOK bo‘lgan bemorlarda HCK rivojlanish xavfi bir xil bosqichda jigar shikastlanishining boshqa etiologik variantlariga qaraganda sezilarli darajada yuqori (OR 2,61; 95% CI 1,27–5,35). L. Orci va boshqalar tomonidan yaqinda o‘tkazilgan met-tahlilda 18 ta tadqiqot natijalarini (*n* = 470,404) birlashtirgan (2022), sirrozdan oldingi MAJYOK bo‘lgan bemorlarda HCK bilan kasallanish 100 kishiga 0,03 (95% CI 0,01–0,07) ekanligini ko‘rsatdi.

Konservativ terapiya bo‘yicha yondashuvlar. Zamonaviy tavsiyalarga ko‘ra, parhez davo va turmush tarzini yaxshilash MAJYOK bilan og‘rigan bemorlarni davolashning asosiy taktikasi hisoblanadi. Bundan tashqari, Yevropa jigar kasalliklarini o‘rganish assotsiatsiyasi, Yevropa diabetni o‘rganish assotsiatsiyasi, semizlikni o‘rganish bo‘yicha Yevropa assotsiatsiyasi (YEASL, YEASD, YEASO) tavsiyalariga ko‘ra, 2016-yilda izolatsiya qilingan steatoz bilan og‘rigan bemorlarda, parhez terapiyasi va jismoniy faollikni oshirish asosiy terapevtik taktikadir, bu esa farmakoterapiyani talab qilmaydi.

Ko‘pgina tadqiqotlar shuni ko‘rsatdiki, vazn yo‘qotish MAJYOK jarayoniga ijobiy ta’sir ko‘rsatadi. 8 ta randomizatsiyalangan tadqiqot natijalarini birlashtirgan meta-tahlil shuni ko‘rsatdiki, ≥5% vazn yo‘qotish jigar steatozining regressiyasiga olib keladi va gistologik faollik indeksini (NAS) kamaytirish uchun ≥7% vazn yo‘qotish zarur. Yuqorida qayd etilgan tadqiqotlar natijalariga ko‘ra, Amerika jigar kasalliklarini o‘rganish assotsiatsiyasi (AASLD) steatozning regressiyasiga erishish va tana vaznining sezilarli darajada pasayishiga (7–10%) erishish uchun tana vaznini 3–5% ga kamaytirishni tavsiya qiladi) [27, 17].

C. Li va boshqalarning so‘nggi meta-tahlilida (2020), 22 ta randomizatsiyalangan nazorat ostida bo‘lgan sinovlar natijalarini (*n* = 1366) jamlagan, omega-3 ko‘p to‘yinmagan yog‘li kislotalardan foydalanish yog‘li jigar kasalligini sezilarli darajada kamaytirishini ko‘rsatdi (RR 1,52; 95% CI – 1,09–2,13) va shuningdek, etakchilik qiladi. triglitseridlar (standartlashtirilgan o‘rtacha farq – SMD 28,57; 95% CI – 40,81–16,33) va umumiy xolesterin (SMD –7,82);

Parhezli davodan tashqari, MAJYOK bilan og‘rigan bemorlar muntazam jismoniy faollikni oshirishlari kerak. Jismoniy harakatsizlik semizlik, insulinga tolerantlilik, metabolik sindrom va MAJYOK rivojlanishi uchun xavf omilidir [23, 30]. S. Vang va boshqalarning so‘nggi meta-tahlillaridan biri. (2020) MAJYOK bilan og‘rigan bemorlarda muntazam jismoniy faollik alaninaminotransferaza (ALT) darajasini kamaytirishga yordam berishini ko‘rsatdi (SRS – 0,17; 95% DI – 0,30–0,05), аспартатаминотрансферазы (CPC – 0,25; 95% ДИ – 0,38–0,13), gamma-glutamintranspeptidaza (SRS – 0,22; 95% DI – 0,36––0,08), umumiy xolesterin (SRS – 0,22; 95% DI – 0,34–0,09), triglitseridlar (SRA – 0,18; 95% DI – 0,31–0,06) va zichligi past lipoproteinlar (SRS – 0,26; 95% DI – 0,39–0,13) [26]. Yana shuni ta’kidlash joizki, MAJYOK uchun farmakoterapiya insulinga tolerantlikni muvofiqlashtirishga, jigar faoliyatini yaxshilashga va metobolik bog‘liq kasalliklar xavfini kamaytirishga qaratilgan bo‘lishi kerak.

XULOSA

Shunday qilib, MAJYOK keng tarqalgan surunkali kasallik bo‘lib, jigarda yog‘ ‘to‘planishining ko‘payishi bilan tavsiflanadi, bu metabolik disfunksiyaga asoslanadi. Dunyoning aksariyat mintaqalarida MAJYOKni klinik tahlili ko‘plab izlanishlar hali-hanuz davom etmoqda. Shu jihatdan bu kasallikninig bir necha metabolik modda almashinuvining buzilishlari bilan birgalikda uyg‘unlikda kechishi eng ahamiyatga ega jihatlaridan biridir. MAJYOK uchun farmakoterapiya insulinga tolerantlikni davolashga, jigar faoliyatini yaxshilashga va ushbu patologiya bilan bog‘liq kasalliklar xavfini kamaytirishga qaratilgan bo‘lishi kerak.

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