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Clinical-Immunogenetic Basis of Early Prediction of Decompensation Phenotypes in Liver Cirrhosis of Various Etiologies

Literature Review

Rasulov Boburjon Abdukakhkhorovich
Assistant, Department of Pharmacology, Clinical Pharmacology and Medical Biotechnologies, Andijan State Medical Institute,
Ravzatov Jasurbek Bakhromovich
PhD, Associate Professor, Department of Family Physicians Training, Andijan State Medical Institute

Annotation
Liver cirrhosis remains one of the leading causes of disability and mortality worldwide, including in Uzbekistan. The clinical severity of the disease is often determined not only by the presence of cirrhosis, but also by the development of its decompensation phenotypes, in particular, complications such as ascites, esophageal variceal bleeding, spontaneous bacterial peritonitis, and hepatic encephalopathy. In recent years, scientific views on the Th17/IL-23/IL-17 inflammatory pathway and the IL-6 cascade in the pathogenesis of liver cirrhosis have been expanding, with the deepening of liver fibrosis, portal hypertension, disruption of the gut-hepatic immune axis, and infectious complications. This article systematically analyzes the role of clinical, immunological, and genetic factors in the early prediction of decompensation phenotypes in liver cirrhosis of various etiologies. The relationship of IL-23R, IL-17A, IL-17F, and IL-6 gene polymorphisms with the course of the disease, decompensation phenotypes, and clinical and biochemical indicators was highlighted. The scientific and practical prospects for the introduction of clinical and genetic risk stratification, along with traditional criteria such as Child–Pugh, are also shown.

Keywords: liver cirrhosis, decompensation phenotypes, ascites, variceal bleeding, spontaneous bacterial peritonitis, hepatic encephalopathy, IL-23R, IL-17A, IL-17F, IL-6, clinical-genetic prognosis.

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Turli Etiologiyali Jigar Sirrozida Dekompensatsiya Fenotiplarini Erta Prognozlashning Klinik-Immunogenetik Asoslari

Adabiyotlar sharhi

Rasulov Boburjon Abduqoxxorovich
Andijon davlat tibbiyot instituti Farmakologiya, klinik farmakologiya va tibbiyot biotexnologiyalari kafedrasi assistenti,
Ravzatov Jasurbek Baxromovich
Andijon davlat tibbiyot instituti Oilaviy shifokorlar tayyorlash kafedrasi PhD, dotsenti

Annotatsiya

Jigar sirrozi dunyo miqyosida, shu jumladan O‘zbekiston sharoitida ham nogironlik va o‘lim ko‘rsatkichlarining yetakchi sabablaridan biri bo‘lib qolmoqda. Kasallikning klinik og‘irligi ko‘pincha faqat sirrozning mavjudligi bilan emas, balki uning dekompensatsiya fenotiplari, xususan assit, qizilo‘ngach varikoz venalaridan qon ketishi, spontan bakterial peritonit va gepatik ensefalopatiya kabi asoratlarning rivojlanishi bilan belgilanadi. So‘nggi yillarda jigar sirrozi patogenezida Th17/IL-23/IL-17 yallig‘lanish yo‘li hamda IL-6 kaskadining jigar fibrozining chuqurlashuvi, portal gipertenziya, ichak-jigar immun o‘qi buzilishi va infeksion asoratlar bilan uzviy bog‘liqligi haqidagi ilmiy qarashlar kengayib bormoqda. Mazkur maqolada turli etiologiyali jigar sirrozida dekompensatsiya fenotiplarini erta prognozlashda klinik, immunologik va genetik omillarning o‘rni tizimli ravishda tahlil qilindi. IL-23R, IL-17A, IL-17F va IL-6 gen polimorfizmlarining kasallik kechishi, dekompensatsiya fenotiplari va klinik-biokimyoviy ko‘rsatkichlar bilan bog‘liqligi yoritildi. Shuningdek, Child–Pugh kabi an’anaviy mezonlar bilan bir qatorda klinik-genetik xatar stratifikatsiyasini joriy etishning ilmiy-amaliy istiqbollari ko‘rsatib berildi.

Kalit so‘zlar: jigar sirrozi, dekompensatsiya fenotiplari, assit, varikoz qonashi, spontan bakterial peritonit, gepatik ensefalopatiya, IL-23R, IL-17A, IL-17F, IL-6, klinik-genetik prognoz.

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Kirish

Jigar sirrozi surunkali jigar kasalliklarining yakuniy morfologik va funksional bosqichi bo‘lib, jigar parenximasining diffuz fibrozga uchrashi, regenerativ tugunlar shakllanishi va jigar funksiyasining progressiv pasayishi bilan tavsiflanadi [1; 9–14-b.]. Mazkur kasallik ichki kasalliklar va gepatologiya amaliyotida yuqori nogironlik, tez-tez shifoxonaga yotqizilish va o‘lim xavfi bilan kechadigan murakkab patologiya sifatida alohida o‘rin tutadi [2; 20–24-b.]. Jigar sirrozning klinik ahamiyati ko‘pincha uning borligi bilan emas, balki dekompensatsiya bosqichiga o‘tishi va og‘ir asoratlar bilan namoyon bo‘lishi bilan belgilanadi [2; 25–29-b.].

An’anaviy amaliyotda jigar sirroz og‘irligini baholashda Chayld–Pyu mezonlari keng qo‘llanadi [6; 70–75-b.]. Ushbu ko‘rsatkichlar prognozlashda muhim bo‘lsa-da, ular barcha bemorlarda dekompensatsiya fenotiplarini bir xil aniqlikda oldindan aytib bera olmaydi [6; 76–81-b.]. Ayniqsa, bir xil klinik-biokimyoviy holatga ega bo‘lgan bemorlarda dekompensatsiya tezligi va asoratlar spektrining turlicha kechishi genetik va immunologik omillarning ahamiyatini ko‘rsatadi [5; 59–64-b.].

So‘nggi yillarda jigar sirrozi patogenezini faqat fibroz va portal gipertenziya bilan izohlash yondashuvi yetarli emasligi aniqlanmoqda [3; 33–37-b.]. Hozirgi qarashlarga ko‘ra, surunkali tizimli yallig‘lanish, ichak-jigar immun o‘qi buzilishi, bakterial translokatsiya, sitokinlar disbalansi va individual genetik moyillik sirrozning klinik fenotipini shakllantiruvchi muhim omillardir [3; 38–45-b.]. Shu nuqtai nazardan, Th17/IL-23/IL-17 signal yo‘li hamda IL-6 kaskadi jigar sirrozi va uning dekompensatsiya asoratlarida alohida ilmiy qiziqish uyg‘otmoqda [4; 48–54-b.].

Jigar sirrozida dekompensatsiya fenotiplari kasallikning klinik og‘irligini belgilovchi asosiy ko‘rsatkichlardan hisoblanadi [2; 30–35-b.]. Assit, qizilo‘ngach va me’da varikoz venalaridan qon ketishi, spontan bakterial peritonit, gepatik ensefalopatiya va gepatorenal sindrom kabi asoratlar bemorning yashab qolish ehtimolini keskin pasaytiradi [2; 36–42-b.]. Shu bois zamonaviy gepatologiyada sirrozni faqat “kompensatsiyalangan” va “dekompensatsiyalangan” shakllarga ajratish emas, balki dekompensatsiya fenotiplarini alohida tavsiflash yondashuvi kuchayib bormoqda [1; 15–19-b.].

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Assit dekompensatsiyaning eng ko‘p uchraydigan shakllaridan biri bo‘lib, portal gipertenziya, arterial vazodilatatsiya, renin-angiotenzin-aldosteron tizimi faollashuvi va oqsil-suv almashinuvi buzilishi bilan bog‘liq [6; 82–88-b.]. Varikoz qonashi esa qisqa vaqt ichida hayot uchun xavf tug‘diradigan og‘ir asorat bo‘lib, u ko‘pincha portal bosimning yuqoriligi, varikoz tomirlar diametri va gemostaz tizimi buzilishi bilan uzviy aloqada bo‘ladi [1; 20–26-b.]. Spontan bakterial peritonit va gepatik ensefalopatiya esa tizimli yallig‘lanish, ichak o‘tkazuvchanligi oshishi, ammoniy metabolizmi buzilishi va immun javobdagi o‘zgarishlar bilan bog‘liq murakkab fenotiplardir [3; 46–52-b.].

Jigar sirrozi uzoq vaqt davomida asosan fibrotik jarayon sifatida qaralgan bo‘lsa, hozirgi vaqtda u tizimli yallig‘lanish bilan kechuvchi immun-metabolik sindrom sifatida ham talqin qilinmoqda [3; 53–57-b.]. Surunkali yallig‘lanish sitokinlari, xususan IL-6, TNF-α, IL-1β va IL-17 oilasi mediatorlari nafaqat jigar to‘qimasida, balki butun organizm miqyosida funksional buzilishlarni kuchaytiradi [4; 55–61-b.].

Jigar sirrozda ichak devori o‘tkazuvchanligining ortishi va bakterial translokatsiya ichak-jigar immun o‘qining izdan chiqishiga olib keladi [3; 58–63-b.]. Natijada endotoksemiya va yallig‘lanish mediatorlarining yuqori ishlab chiqarilishi kuzatiladi [3; 64–68-b.]. Bu holat portal gipertenziya, assit, spontan bakterial peritonit va ensefalopatiya rivojlanishida muhim o‘rin tutadi [2; 43–48-b.]. Demak, dekompensatsiya faqat jigar parenximasi zararlanishining oqibati emas, balki murakkab immun-yallig‘lanish mexanizmlarining mahsulidir [4; 62–66-b.].

Odatda Th17 hujayralari va ularga tegishli IL-23/IL-17 signal yo‘li autoimmun, surunkali yallig‘lanish va fibrotik kasalliklarda muhim rol o‘ynaydi [4; 67–73-b.]. Jigar sirrozi sharoitida ushbu yo‘l stellat hujayralar faollashuvi, fibrogenez kuchayishi, sitokinlar kaskadi va immun disbalansni chuqurlashtirishi mumkin [5; 65–71-b.]. IL-17A va IL-17F proyallig‘lanish sitokinlari sifatida neytrofillar migratsiyasi, yallig‘lanish mediatorlari sintezi va to‘qima zararlanishini kuchaytiradi [5; 72–78-b.]. IL-23R esa Th17 hujayralarining differensiyalanishi va saqlanishida markaziy ahamiyatga ega [5; 79–83-b.].

Shu sababli IL-23R, IL-17A va IL-17F genlaridagi polimorfizmlar sirrozda individual yallig‘lanish javobi, fibroz tezligi va dekompensatsiya xavfi bilan

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bog‘liq bo‘lishi ehtimoldan xoli emas [5; 84–89-b.]. Ayniqsa assit, spontan bakterial peritonit va ensefalopatiya kabi asoratlarda immun reaktivlikning irsiy xususiyatlari klinik farqlarni tushuntirishda muhim bo‘lishi mumkin [5; 90–96-b.].

Yallig‘lanish mediatorlaridan IL-6ni ortishi jigar sirrozi patogenezida markaziy yallig‘lanish mediatorlaridan biri bo‘lib, u gepatotsit zararlanishi, fibrozning chuqurlashuvi, portal gipertenziya va tizimli yallig‘lanish bilan bog‘liq [4; 74–80-b.]. IL-6 darajasining oshishi ko‘pincha kasallik og‘irligi, infeksion asoratlar va noqulay prognoz bilan parallel kechadi [4; 81–86-b.]. Shu jihatdan IL-6 klinik biomarker sifatida qadrlanadi [4; 87–90-b.].

IL-6 geni polimorfizmlari esa individual sitokin javobini modulyatsiya qilishi mumkin [5; 97–101-b.]. Natijada ayrim bemorlarda yallig‘lanish reaksiyasi kuchliroq, dekompensatsiya tezroq va asoratlar og‘irroq kechishi ehtimoli mavjud [5; 102–108-b.]. Shu bois IL-6 polimorfizmlarini klinik-biokimyoviy ko‘rsatkichlar bilan birgalikda baholash jigar sirrozida shaxsga yo‘naltirilgan prognozlash tizimi yaratishda muhim ahamiyat kasb etadi [6; 89–95-b.].

Turli etiologiyali jigar sirrozida dekompensatsiya mexanizmlari bir xil emas [8; 3–8-b.]. HBV va HCV bilan bog‘liq sirrozlarda virusga qarshi immun javob, surunkali nekroinflammasiya va immunositlar faollashuvi yetakchi rol o‘ynaydi [8; 9–15-b.]. Noalkogol yog‘li jigar kasalligi negizida rivojlangan sirrozda esa metabolik yallig‘lanish, insulinrezistentlik, adipokinlar disbalansi va oksidlovchi stress muhimroq ahamiyatga ega [9; 11–17-b.]. Alkogol etiologiyali sirrozda esa etanol va uning metabolitlarining to‘g‘ridan to‘g‘ri toksik ta’siri, ichak mikrobiotasi o‘zgarishlari va endotoksemiya bilan bog‘liq immun buzilishlar ustunlik qiladi [10; 5–11-b.].

Bu farqlar shuni ko‘rsatadiki, bir xil genetik polimorfizm barcha etiologik shakllarda bir xil klinik ta’sir ko‘rsatmasligi mumkin [8; 16–20-b.]. Demak, jigar sirrozida prognozlash modeli etiologiyaga moslashtirilgan bo‘lishi lozim [6; 96–101-b.].

Amaliyotda Chayld–Pyu tizimlari jigar sirrozi og‘irligini baholashda asosiy vosita bo‘lib qolmoqda [6; 102–107-b.]. Biroq ular bemorning immunogenetik xususiyatlarini hisobga olmaydi [6; 108–112-b.]. Shu sababli

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klinik, laborator va genetik ma’lumotlarni birlashtiruvchi integrallashgan risk modellari zamonaviy gepatologiyaning istiqbolli yo‘nalishlaridan biridir [7; 20–26-b.].

Klinik-genetik yondashuv yuqori xavf guruhlarini erta aniqlash, profilaktik choralarni individuallashtirish va klinik qaror qabul qilishni takomillashtirish imkonini beradi [7; 27–32-b.]. Ayniqsa spontan bakterial peritonit, varikoz qonashi yoki ensefalopatiya rivojlanish xavfi yuqori bo‘lgan bemorlarni oldindan aniqlash amaliy tibbiyot uchun katta ahamiyatga ega [2; 49–55-b.].

Turli etiologiyali jigar sirrozida dekompensatsiya xavfini erta prognozlash amaliy tibbiyot uchun muhimdir [7; 33–37-b.]. Agar klinika, laborator ko‘rsatkichlar va genetik omillarni birlashtiruvchi sodda, ammo ishonchli risk modeli ishlab chiqilsa, bu oilaviy shifokor, gastroenterolog va terapevt faoliyatida bemorlarni differensial kuzatish imkonini kengaytiradi [7; 38–44-b.]. Ayniqsa spontan bakterial peritonit yoki varikoz qonash xavfi yuqori bo‘lgan bemorlarni oldindan ajratish profilaktik yondashuvlarni o‘z vaqtida belgilashga yordam beradi [3; 69–74-b.].

Bundan tashqari, genetik polimorfizmlarni baholash shaxsga yo‘naltirilgan tibbiyot tamoyillarini amaliyotga tatbiq etish uchun mustahkam asos yaratadi [7; 45–49-b.]. Bu ayniqsa yuqori xavf guruhlarini ajratishda, kasallik kechishini prognozlashda va resurslarni oqilona taqsimlashda foydalidir [6; 113–118-b.].

Xulosa

Turli etiologiyali jigar sirrozida dekompensatsiya fenotiplari kasallik prognozini belgilovchi asosiy klinik ko‘rsatkichlardan biridir [1; 27–31-b.]. Ularning shakllanishida faqat jigar funksional yetishmovchiligi emas, balki tizimli yallig‘lanish, ichak-jigar immun o‘qi buzilishi, sitokinlar disbalansi va individual genetik moyillik ham muhim rol o‘ynaydi [3; 75–81-b.]. Ayniqsa IL-23R, IL-17A, IL-17F va IL-6 genlaridagi polimorfizmlar klinik-biokimyoviy ko‘rsatkichlar bilan birgalikda baholanganda dekompensatsiya xavfini aniqroq prognozlash imkoniyatini yaratishi mumkin [5; 109–115-b.]. Shunday qilib, jigar sirrozida klinik-immunogenetik yondashuvni joriy etish erta tashxis, xavf guruhlarini stratifikatsiya qilish, kuzatuv intensivligini individuallashtirish va asoratlar profilaktikasini takomillashtirish uchun muhim ilmiy-amaliy asos bo‘lib xizmat qiladi [7; 50–56-b.].

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Foydalanilgan adabiyotlar

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