Clinical and Immunogenetic Features of Liver Fibrosis Development in Chronic Hepatitis of Various Etiologies: A Literature Review

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Abstract. Chronic hepatitis of various etiologies remains one of the major causes of progressive liver damage worldwide. Persistent inflammatory processes in the liver can lead to fibrosis, cirrhosis, and ultimately liver failure. The development and progression of liver fibrosis are determined not only by the etiological factor but also by complex interactions among immune, genetic, and environmental mechanisms.

This literature review examines current scientific data on the clinical and immunogenetic characteristics of liver fibrosis development in patients with chronic hepatitis of different origins. Particular attention is given to the role of cytokines, immune response pathways, genetic polymorphisms, and molecular markers involved in fibrogenesis. The review also discusses the prognostic significance of immunogenetic factors and their potential application in early diagnosis, risk stratification, and personalized therapeutic approaches.

Keywords: chronic hepatitis, liver fibrosis, immunogenetics, cytokines, genetic polymorphism, fibrogenesis, chronic inflammation, liver cirrhosis, molecular markers, personalized medicine.

TURLI ETIOLOGIYALI SURUNKALI GEPATITLARDA JIGAR FIBROZI RIVOJLANISHINING KLINIK-IMMUNOGENETIK XUSUSIYATLARI

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Annotatsiya

Surunkali gepatitlar turli etiologik omillar — virusli gepatit B va C, autoimmun jarayonlar, metabolik buzilishlar hamda toksik ta’sirlar natijasida rivojlanib, jigar fibrozi, sirroz va gepatotsellyulyar karsinoma bilan asoratlanishi mumkin bo‘lgan dolzarb tibbiy muammo hisoblanadi. Jahon sog‘liqni saqlash tashkiloti ma’lumotlariga ko‘ra, surunkali gepatit B va C global miqyosda millionlab bemorlarda uchrab, jigar sirrozi va jigar saratoni bilan bog‘liq o‘limlarning muhim sabablaridan biri bo‘lib qolmoqda [1].

Jigar fibrozi rivojlanishida nafaqat etiologik omilning davomiyligi va virus yuklamasi, balki bemorning immun javobi, yallig‘lanish mediatorlari, fibrogenezga aloqador sitokinlar va genetik moyillik ham muhim ahamiyatga ega. Ayniqsa IL-6, IL-17A, IL-17F, IL-23R, TGF-β1 va COL1A1 kabi immun-yallig‘lanish hamda fibrozlanish yo‘llariga aloqador markerlar surunkali gepatitlarda fibroprogressiyani baholash uchun istiqbolli ilmiy yo‘nalish hisoblanadi [2–6].

Kalit so‘zlar: surunkali gepatit, jigar fibrozi, immunogenetika, IL-6, IL-17A, IL-17F, IL-23R, TGF-β1, COL1A1, fibrogenez.

Kirish

Jigar fibrozi surunkali shikastlanishga javoban rivojlanadigan reparativ, ammo nazoratsiz davom etganda patologik tus oladigan jarayondir. Gepatotsitlar zararlanishi, Kupffer hujayralari faollashuvi, yallig‘lanish sitokinlari ajralishi va yulduzsimon hujayralarning miofibroblastlarga aylanishi natijasida kollagen va boshqa ekstrasellyulyar matriks komponentlari ortiqcha to‘planadi. EASL tavsiyalarida

surunkali jigar kasalliklarida fibroz darajasini baholash uchun elastografiya, noinvaziv indekslar va klinik-laborator ko‘rsatkichlarni birgalikda qo‘llash zarurligi ta’kidlangan [2; 659–689-b.].

Turli etiologiyali surunkali gepatitlarda fibroz rivojlanish tezligi bir xil emas. Virusli gepatitlarda virus replikatsiyasi, yallig‘lanish faolligi va immun javobning xususiyati muhim bo‘lsa, autoimmun gepatitlarda immun agressiya, metabolik zararlanishlarda esa insulinrezistentlik va oksidlovchi stress fibrogenezni kuchaytiradi. Shunga qaramay, ushbu etiologiyalarning umumiy yakuniy patogenetik bo‘g‘ini — surunkali yallig‘lanish, yulduzsimon hujayralar faollashuvi va kollagen sintezining ortishidir.

Immunogenetik jihatdan Th17/IL-17 yo‘li jigar fibrozining muhim mexanizmlaridan biri sifatida qaraladi. IL-17 yo‘li yulduzsimon hujayralar faollashuvi, TGF-β ekspressiyasi, kollagen sintezi va yallig‘lanish hujayralari migratsiyasini kuchaytirishi mumkin. Surunkali B va C gepatitlarda Th17, IL-17 o‘qi fibrogenez va sirrozga o‘tish jarayonida immunopatogenetik va prognostik ahamiyatga ega ekani ko‘rsatilgan [3].

IL-23R, IL-17A va IL-17F genlaridagi polimorfizmlar immun javobning davomiyligi, yallig‘lanish intensivligi va fibrozlanish xavfiga ta’sir qilishi mumkin. Ayrim tadqiqotlarda IL-17A rs2275913, IL-17F rs763780 va IL-23R rs10889677 polimorfizmlari gepatit B infeksiyasi oqibati, sirroz va gepatotsellyulyar karsinoma xavfi bilan bog‘liq bo‘lishi mumkinligi qayd etilgan [4].

IL-6 surunkali gepatitlarda yallig‘lanish faolligi, gepatotsit shikastlanishi va fibroprogressiya bilan bog‘liq sitokinlardan biridir. IL-6 genidagi ayrim polimorfizmlar yallig‘lanish mediatorlari ishlab chiqarilishiga ta’sir qilib, kasallikning surunkalashuvi, fibrozlanish darajasi va klinik og‘irligini belgilashi mumkin. Yallig‘lanish genlari bo‘yicha meta-tahlillarda TGF-β1, IL-10, IL-18 va IFN-γ polimorfizmlari jigar sirroziga moyillik bilan bog‘liq bo‘lishi mumkinligi ko‘rsatilgan [5].

COL1A1 geni esa fibrogenezning struktur komponenti bilan bog‘liq muhim nomzod genlardan biridir. Ushbu gen I-tip kollagen sintezida ishtirok etadi va jigar to‘qimasida ekstrasellyulyar matriks to‘planishiga bevosita aloqador. COL1A1 polimorfizmlari HBV bilan bog‘liq jigar sirrozi va fibrozlanish jarayonlari bilan bog‘liq bo‘lishi mumkinligi haqidagi ma’lumotlar ushbu genni klinik-immunogenetik tadqiqotlar uchun muhim marker sifatida ko‘rib chiqishga asos beradi [6].

Shu nuqtai nazardan, turli etiologiyali surunkali gepatitlarda jigar fibrozini baholashda faqat klinik, biokimyoviy va instrumental ko‘rsatkichlar bilan cheklanish yetarli emas. IL-6, IL-17A, IL-17F, IL-23R, TGF-β1 va COL1A1 kabi markerlarni kompleks o‘rganish fibroz rivojlanish xavfini erta aniqlash, yuqori xavf guruhlarini ajratish va individual prognostik model yaratish imkonini beradi.

Xulosa

Turli etiologiyali surunkali gepatitlarda jigar fibrozi ko‘p omilli jarayon bo‘lib, uning rivojlanishida etiologik omil, yallig‘lanish faolligi, immun javob turi va genetik moyillik o‘zaro bog‘liq holda ishtirok etadi. IL-17/IL-23 o‘qi, IL-6 vositachiligidagi yallig‘lanish javobi, TGF-β1 bilan bog‘liq fibrogenez va COL1A1 orqali kollagen sintezining faollashuvi jigar fibrozining asosiy immunogenetik bo‘g‘inlari sifatida qaraladi. Mazkur markerlarni klinik, laborator va elastografik ko‘rsatkichlar bilan integratsiyalash surunkali gepatitlarda fibrozlanish xavfini erta baholash va individual prognozlash tizimini ishlab chiqish uchun muhim ilmiy-amaliy asos yaratadi.

Foydalanilgan adabiyotlar

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