BÖLÜM 3

COVID-19 VE OTOİMMÜNİTE

Emrah SALMAN[1]

GİRİŞ

Otoimmün hastalıkların etiyolojisi henüz tam olarak bilinmemektedir, fakat genetik predispozisyon, bakteriyel, viral, fungal, parazitik enfeksiyonlar, hormonal faktörler ve immün sistem disregülasyonu gibi çeşitli faktörlerin katkıda bulunduğu düşünülmektedir. Otoimmün hastalıklara en sık yol açtığı düşünülen virüsler Parvovirüs B19 (PV-B19), Epstein Barr virüsü (EBV), Sitomegalovirüs (CMV), Herpesvirüs-6 (HHV-6), İnsan T-hücre lenfotropik virüs Tip I (HTLV-1), Hepatit A (HAV) ve Hepatit C (HCV) ve Rubella virüsü'dür (1). Bu virüslerin romatoid artrit, sistemik lupus eritematozus, Sjögren sendromu, primer biliyer kolanjit, multipl skleroz, polimiyozit, üveit, Henoch Schönlein purpurası, sistemik juvenil idiyopatik artrit, sistemik skleroz, Hashimoto tiroiditi ve otoimmün hepatit gibi kronik inflamatuvar veya otoimmün hastalıklarda rol alabilecekleri öne sürülmüştür (2, 3). Otoimmüniteye yol açan mekanizmalar arasında moleküler benzerlik, konak doku ve hücreleri hasar gördüğünde korunmuş gizli antijenlerin salınımı, konak hücre proteinlerinin değişimi ve poliklonal aktivasyon gibi çeşitli mekanizmalar yer almaktadır (4).

Hiperinflamatuvar hastalıklar ve COVID-19 arasında ortak patojenik mekanizmalar ve klinik-radyolojik durumlar olduğu düşünülmüştür ve genetik olarak predispoze kişilerde SARS-CoV-2'nin hızlı bir otoimmün ve/veya otoinflamatuvar disregülasyon gelişimine neden olarak ağır interstisyel pnömoniyi tetiklediği öne sürülmüştür (5). COVID-19'lu kişilerde otoantikorların varlığı çeşitli çalışmalarda farklı sıklıklarda bildirilmiştir: Anti-nükleer antikorlar (ANA) %35.6, anti-Sjögren sendromu antikoru (anti-SSA) %25, romatoid faktör %19, lupus antikoagülanı %11 ve interferona karşı gelişen antikorlar %10 oranındadır (6-9).

[1] Uzm. Dr., Ankara Bilkent Şehir Hastanesi, Tıbbi Mikrobiyoloji Bölümü emrahsalman85@hotmail.com

inme sıklıkla yaşlı hastalarda kaydedilmiştir. COVID-19'un vasküler bulguları 50 yaş altında venöz veya arteriyel tromboz veya inme şeklindedir ayrıca bazen antifosfolipid antikorları ile ilişkili katastrofik klinik tablo oluşabilmektedir (62). Sistemik lupus eritematozus gibi sistemik otoimmün hastalıklara sahip kişilerde antifosfolipid antikorları tromboz ve gebelik morbiditesinin temel nedenidir. Antifosfolipid sendromunun (APS) ağır formları (katastrofik APS) mikroanjiyopatinin histopatolojik bulgusu ile hızlı şekilde ortaya çıkan çoklu organ trombotik hasarı ile karakterizedir (63). Katastrofik APS genellikle vakaların çoğunda enfeksiyon gibi presipite edici bir faktör tarafından meydana gelmektedir (64). Enfeksiyon ve antifosfolipid sendromu arasındaki ilişki farklı otoimmün durumlarda gözlenmiştir ve kardiyolipini içeren fosfolipidlerin bir karışımı olarak sifilizin serolojik test pozitifliğinde görülmüştür (65). Antifosfolipid sendromunun saptanması antikardiyolipin ve anti-beta 2-glikoprotein immünassay ile lupus antikoagülanıyla yapılmaktadır (66). Geçici antifosfolipid antikor yüksekliği HIV, varisella zoster (VZV), HCV, CMV, EBV, adenovirüs (ADV), PV-B19 gibi çeşitli viral enfeksiyonlarca oluşabilmektedir (67). Bu gözlemler beta 2-glikoprotein molekülü ve çeşitli virüslerin membran proteinleri arasındaki moleküler benzerliği önermektedir (68).

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