Bölüm

7

Hipertansiyon, İmmunite ve İnflamasyon

Emine YAĞCI [1]
Cansu ÖZBAYER [2]
Hülyam KURT [3]

GİRİŞ

Hipertansiyon en yaygın kronik hastalıklardan biri olmakla birlikte kalp yetmezliği, felç, kronik böbrek hastalığı ve ölümlerin başlıca nedenidir. Diğer hastalıklar ile benzer şekilde, idiyopatik hipertansiyon, kesin nedeni ve patofizyolojisi bilinmediğinde temel (esansiyel) veya birincil (primer) olarak adlandırılır. Hipertansiyonun bilinen, doğrudan nedenleri tüm vakaların yalnızca % 5-10'unda tespit edilebilir ve altta yatan kesin bir patofizyolojik mekanizma nedeniyle "ikincil" olarak adlandırılır [(1)].

Esansiyel hipertansiyon, kan akışına karşı artmış periferik vasküler direnç ile karakterizedir. Endotel, vasküler tonusun çok önemli bir düzenleyicisidir. Hipertansiyonlu hastalarda endotel fonksiyonu bozulmuştur, buna azalmış vazodilatasyon, proinflamatuar ve protrombotik durumla ilişkili olarak artmış vasküler ton eşlik eder. Bu nedenle, vasküler dokuda lokalize olan düşük dereceli inflamasyon, hipertansiyonun patofizyolojisine, aterosklerozun başlamasına ve ilerlemesine ve ayrıca kardiyovasküler hastalıkların gelişimi için önemli bir risk olarak kabul edilmektedir [(2, 3)].

Düşük dereceli inflamasyonun, kan basıncı yükselmesinin başlatılmasında ve sürdürülmesinde önemli bir aracı olduğu ve kronik inflamatuar hastalıklarla bağlantılı olarak kan basıncı yükselmesini tetikleyebileceği bildirilmektedir. Adezyon molekülleri ve kemokin ekspresyonu, immün hücre aktivasyonu ve infiltrasyonu, sitokin salınımı ve oksidatif stres gibi tüm inflamatuar mekanizmalar hipertansiyonda artar [(4)].

Bağışıklık sistemi, inflamasyon ve hipertansiyon birbiriyle ilişkilidir. Doğuştan gelen ve kazanılmış bağışıklık sistemi, kan basıncını yükseltebilir ve organ hasa-

[1] Arş. Gör., Eskişehir Osmangazi Üniversitesi, eminetsci@gmail.com
[2] Doç.Dr., Kütahya Sağlık Bilimleri Üniversitesi, cansu.ozbayer@ksbu.edu.tr
[3] Prof. Dr., Eskişehir Osmangazi Üniversitesi, hkurtayda@gmail.com

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