Bölüm
27

YOĞUN BAKIM ÜNİTESİNDE RENAL YETMEZLİĞE YAKLAŞIM

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GİRİŞ

Akut renal yetmezlik günümüzde akut renal hasar (AKI) terimi ile ifade edilir (1). Böbrek boşaltım fonksiyonunda hızlı (saatler-günler içerisinde) azalma sonucunda kreatinin, üre ve diğer klinik olarak ölçülememiş atık ürünler gibi azot metabolizması ürünlerinin birikmesi ile karakterize klinik bir sendromu ifade eder.

Akut böbrek hasarı (AKI), hem dahili hem de cerrahi yoğun bakım ünitelerinde (ICU) yatan kritik hastalarda karşılaşılan önemli morbiditelerden biridir (2). Yoğun bakımdaki kritik AKI' lı hastaların yaklaşık %6' sı yoğun bakım ünitesinde (ICU) yatışları sırasında bir tür renal replasman tedavisi (RRT) ile tedavi edilir (3). Yoğun bakım ünitesinde çalışan her hekimin AKI' nın önlenmesi ve tedavisi için önemli stratejiler hakkında temel bilgiye sahip olması gerekir.

AKI EPİDEMİYOLOJİSİ

AKI insidansı yapılan çalışmaların hasta popülasyonuna bağlı olarak farklılık gösterir. Amerika Birleşik Devletleri'ndeki hastane yatışlarının %1'ini oluşturur (4,5). Yoğun bakımda yatan hastalarda pek çok neden AKI' ya neden olmakta, en sık neden ise akut tübüler nekroz (ATN)' dur. Buna karşın süreç sepsis, nefrotoksik ilaçlar, kontrast ajanlar ve cerrahi sonrası dahil olmak üzere multifaktöryeldir (5). ICU hastalarının yaklaşık %5 ila %20' sinde AKI gelişir (2,3). ICU ile ilişkili AKI insidansı son yıllarda artmıştır ve bunun nedeni büyük olasılıkla sepsis ile ilişkili hastane yatış sıklığının artmasıdır.

AKI PATOFİZYOLOJİSİ

A-) Böbreğe ait nedenler: AKI patogenezi vasküler, tübüler ve inflamatuar faktörlerin birlikte rol aldığı karmaşık bir durumdur (3,6). Septik şoka seconder AKI' nın ağırlıklı olarak sistemik arteryel vazodilatasyon ve eşlik eden intrarenal vazokonstriksiyon sonrasında renal kan akışında bir azalmaya bağlı olarak geliştiği düşünülür. Bu durum böbrek hipoperfüzyonu ve iskemi ile sonuçlanır. Böbreklerin, vücuttaki herhangi bir organla karşılaştırıldığında birim kütle başına düşen kan akımı daha yüksektir, ancak gerçekte böbreğin aldığı oksijen fraksiyonu ise daha azdır. Bu, böbreği hipoperfüzyon koşullarına çok duyarlı hale getirir. İskemi ve toksinlerin artışı; vazokonstriksiyon, endotel hasarı ve doğuştan-edinilmiş inflamatuar immün yanıtların aktivasyonu ile sonuçlanır (3,6). Renal dokulara oksijen temini, travmadan kaynaklanan akut kan kaybı veya septik şok nedeniyle büyük miktarlarda kristaloid resüsitasyonu ile akut hemodilüsyon neticesinde bozulabilir (7).

Sonuçta ortaya çıkan AKI, hem lokal hem de sistemik olarak bir dizi enflamatuar süreci tetikler. Bu sistemik inflamatuar yanıt sendromu (SIRS) başlangıçta proinflamatuar sitokinlerin sistemik olarak salınması, ardından antienflamatuar sitokinlerin salınımı ile karakterizedir. Bu anti-enflamatuar yanıt, enflamatuar süreci kontrol etmeyi ve sınırlamayı amaçlar (8). AKI ortamındaki üremi bu doğal olay dizisini bozabilir ve bunun multiorgan yetmezliğinin patogenezinde anahtar rol oynadığı düşünülmektedir.

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