GİRİŞ

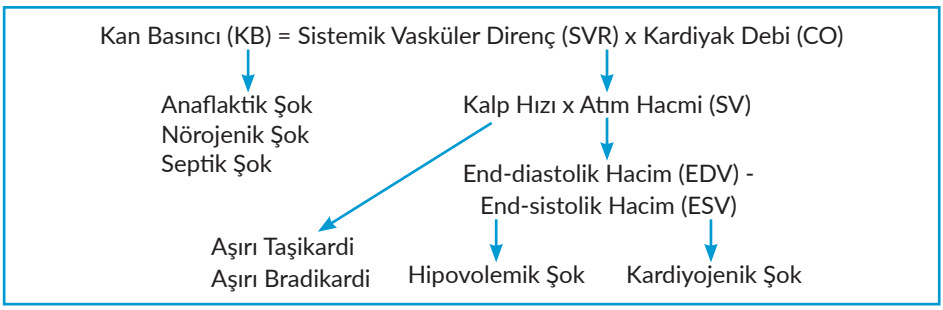
Doku oksijenasyonunun yeterliliği, oksijen sunumu (DO_2) ve oksijen tüketimi arasındaki dengeye (VO_2) bağlıdır. Şok, hücrelerin oksijen ihtiyacı ile temini arasında oluşan uyumsuzluk sonucu organların ve dokuların yetersiz ve bozulmuş oksijen kullanımı ile seyreden; hedef organ hasarı ile sonuçlanan yüksek morbidite ve mortalite ile ilişkili akut dolaşım yetmezliği ve kritik doku hipoperfüzyonudur (1).

Yetersiz oksijen tüketiminin yaygın olması tüm organları etkileyerek hücresel hipoksiye neden olur. Şokun erken döneminde, aerobik metabolizmadan anaerobik metabolizmaya geçiş, geçici olarak kompanse edilebilir, ancak laktat anaerobik metabolizmanın sonucu olarak birikmeye devam eder. Takiben hipoperfüzyon ile hücresel hipoksi, bozulan mikrosirkülasyon veya mitokondriyal disfonksiyonla süratle hücre ölümü ve doku nekrozuyla birden fazla organ yetmezliği oluşturur. Şok resüsitasyonunun amacı, yetersiz organ perfüzyonu ve doku hipoksisi oluşturan etyolojiyi hızla tanımlarken, sıvı infüzyonu ve vazoaktif ajanların uygulanması yoluyla hemodinamik stabilizasyon sağlayarak, şokun kötüleşmesini önlemek ve dolaşım yetersizliğini vücudun doku oksijen gereksinimlerini karşılayan bir düzeye getirerek organ hasarı devam etmeden ve geri dönüşümü olmayan döneme girmeden, hızla tersine çevirerek ölümü önlemek ve eş zamanlı olarak saptanan şok nedeninin düzeltilmesini içermelidir (2,3,4).

Şokun Patofizyolojik Özellikleri

Doku oksijenasyonunda oksijen sunumunun (DO_2) komponentleri, kardiyak debi (*cardiac output* (CO)) ve arteryal oksijen konsantrasyonudur.

Kardiyak debi aynı zamanda doku perfüzyonu ve sistemik kan basıncının (KB) başlıca fizyolojik belirleyicileri arasındadır (Şekil 1) (5).



Şekil 1. Şok patofizyolojisinde kan basıncının sistemik vasküler direnç ve kardiyak debi ile ilişkisi

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