KARBONMONOKSİT ZEHİRLENMELERİ ve GEÇ NÖROPSİKİYATRİK SEKELLER

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Giriş

Karbonmonoksit (CO) zehirlenmeleri dünyada en sık görülen zehirlenme türlerinden biri olup, hücresel oksijende azalmaya neden olarak doku hasarına yol açan önemli bir sağlık sorunudur[1-3]. CO beyin, cilt, kalp, böbrek, iskelet kası ve periferal sinirler gibi farklı dokuları etkileyerek zehirlenmelerde birçok klinik bulgunun, sistemik komplikasyonun ve uzun dönem sekel bulgularının ortaya çıkmasına neden olur[1, 4].

Geç Nöropsikiyatrik Sekeller

CO zehirlenmelerinde nörolojik bulgular monofazik veya bifazik özellik gösterebilir ve zehirlenen bireylerde asemptomatik sessiz dönemi takiben geç nöropsikiyatrik bulgular saptanabilir[3, 5-7]. Akut CO zehirlenmeleri ardından iyileşen hastaların yaklaşık %10-30'u geç nöropsikiyatrik sekel bulguları ile yeniden hastaneye başvurmaktadır. Bu bulgular CO zehirlenmelerine bağlı gelişen morbiditenin önemli bir nedenini oluşturmaktadır[8, 9].

CO zehirlenmelerinde geç nöropsikiyatrik sekel oluşumunun kesin nedeni bilinmemekle birlikte yapılan patofizyolojik çalışmalar sonucunda birkaç hipotez öne sürülmüştür[1, 6, 8]. CO maruziyeti ile birlikte CO'nun hemoglobine kompetatif bağlanması gerçekleşir. Bu durum hemoglobinin oksijen transport kapasitesinde azalmaya yol açarak oksijen hemoglobin dağılım eğrisinde sola kaymaya ve doku düzeyinde oksijen salınımının etkilenerek hücresel düzeyde hipoksiye neden olur[5, 10]. Bir diğer mekanizma CO nedenli doku hipoksisini takiben gelişen reoksijenizasyonun santral sinir sisteminde hasarlanmaya yol açmasıdır. Hiperoksijenizasyon serbest oksijen radikallerinin oluşumunu artırarak protein ve nükleik asitlerin oksidizasyonuna neden olur. Ek olarak CO maruziyetinin; lipid peroksidasyonuna yol açarak desature yağ asitlerinin yıkımı ve santral sinir sistemi lipidlerinin geri dönüşümlü demyelinizasyonuna ve inflamasyona neden olarak geçici beyaz cevher hasarı oluşumuna katkıda bulunduğu öne sürülmüştür[3,

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