

Bölüm

6

Hipertansiyonun Fetal Programlanması

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GİRİŞ

Pek çok erişkin hastalığının kökeninin fetal ve doğum sonrası erken yaşama kadar uzanabileceği kavramı, fetal (gelişimsel) programlama ya da Barker hipotezi olarak bilinmektedir. Bu hipoteze göre, erken gelişimin kritik evrelerinde, özellikle intrauterin yaşamdaki yetersiz beslenme ve glukokortikoidlere maruziyet gibi olumsuz koşullar, fizyoloji ve metabolizmada kalıcı değişiklikler yaratarak yaşamın sonraki dönemlerinde kardiyovasküler, metabolik ve endokrin hastalıklara yakalanma riskinin artmasına neden olabilmektedir.

Hipertansiyonun bilinen risk faktörleri arasında bulunan fetal programlama, erişkinlikte bu hastalığa yatkınlığın belirlenmesinde potansiyel bir rol oynar. Epidemiyolojik çalışmalar, olumsuz fetal çevre koşullarının bir belirteci olan düşük doğum ağırlığı (DDA) ile hipertansiyon arasında ters bir ilişki olduğunu kanıtlamaktadır. Doğum ağırlığı kendi başına tetikleyici bir etken olmamakla birlikte organ yapı ve/veya işlevlerinin, dolayısı ile hastalığa yatkınlık derecesinin değişmesine neden olan bir faktördür. DDA ile hipertansiyonu birbirine bağlayan mekanizmalar birçok bileşeni kapsamaktadır ve normal düzenleyici sistemlerdeki ve arteriyel basıncın uzun süreli kontrolünde görev alan böbrek fonksiyonlarındaki değişiklikleri içermektedir. Kitabın bu bölümünde, hipertansiyonun fetal programlamasına etki eden faktörler ve bunlara aracılık eden mekanizmalar gözden geçirilecektir.

FETAL PROGRAMLAMA ÇALIŞMALARI VE DENEYSSEL MODELLER

Çok sayıdaki epidemiyolojik çalışma, hipertansiyonun fetal programlamasının araştırılması için temel oluşturmuş, bazı klinik çalışmalar ise DDA ile hipertansiyon arasındaki bağlantıya aracılık eden mekanizmaları ele almıştır. Bunun yanında, hipertansiyonun fetal programlamasını incelemek için kullanılan, çoğunlukla

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