22. Bölüm

KALP YETERSİZLİĞİNDE VENTRİKÜLER TAŞİKARDİLER

Ayhan COŞGUN[1]

Önemli tıbbi ve cerrahi ilerlemelere rağmen, kalp yetersizliğinin (KY) tedavisi ve yönetimi, önemli bir morbidite ve mortalite nedeni ile birlikte, ekonomik yük olarak 21. yy da karşımıza halen bütün heybetiyle çıkmaktadır. KY ilk tanısı konulduktan sonra ilk 5 yıl içerisinde, %50 mortalite oranına sahiptir.(1) KY, sistolik ve diyastolik kalp yetmezliği şeklinde klasifiye edilirken, aynı zamanda kronik stabil KY'den akut dekompanse KY' ne kadar klinik şekli ile de klasifiye edilebilmektedir. Bu şekilde olanlar, akut miyokard enfarktüsüne bağlı olarak yeni ve akut başlangıçlı olabilirken, alkole bağlı dilate kardiyomiyopati gibi subakut şekilde de ortaya çıkabilmektedir.(2)

Ventriküler aritmiler (VA) KY'de sık görülür ve ani kardiyak ölüm riskini artırır.(3) Çok sayıda çalışma, KY'deki VA' in sadece bağımsız bir risk faktörü mü, yoksa kalbin başarısız fenotipik bir tezahürü mü olduğunu araştırmıştır.

KALP YETERİSİZLİĞİNDEKİ VENTRİKÜLER ARİTMİLERİN OLASI MEKANİZMALARI

Miyokardiyal Hipertrofinin ve Gerilmenin Etkisi

Yetersizlikli ventrikülde, akut gerilmenin yanı sıra kronik yüklenme de, ventriküler miyokardda elektrofizyolojik heterojenite ve gerilmeyle aktive olan membran kanalları yoluyla VA'lere neden olabilir.(4) Akut yükün, etkili refrakter periyodu (ERP) kısalttığı gösterilmiştir ve uygulanan miyokardiyal esnemenin zamanlamasına bağlı olarak aksiyon potansiyelini (AP) uzatabilir veya kısaltabilir.(5) Artan ön yük, kardiyak döngü boyunca mekanik gerilmeye yol açar. Kronik, sürekli mekanik gerilme aynı zamanda, ERP'yi ve ortalama aksiyon potansiyel süresini (APD) kısaltırken, gerilmeye bağlı ventriküler aritmojenisite için odak görevi görebilen aktivasyon sürelerini uzatır.(6)

Akut İskemi

Akut iskemi sırasında ventriküler aritmilerin iki farklı fazı vardır: faz 1A (koroner akışın durmasından 2 ila 10 dakika sonra) ve faz IB (kabaca 15 ila 30 dakika arasında). Faz IA'daki aritmiler, büyük ölçüde hipoksi, hücre dışı potasyum birikimi ve asidozun kombine etkilerinden kaynaklanan, iskemik miyokardın depresif uyarılabilirliği ile ilgilidir, bu da iletimin yavaşlamasına ve heterojen uyarılabilirliğin geri kazanılmasına yol açar.(7) Faz 1a aritmileri genellikle iyi huyludur ve sadece kısa ventriküler taşikardi (VT) dönemleri olarak kendini gösterir.(8) Bu aritmilere sınır bölgelerdeki re-entri halkaları sebep olur ve bu anor-

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olumsuz yönde etkileyebilir.[31,64] Diğer antiaritmik ilaçlardan kaçınılmalıdır.[65]

Aritmojenik merkezin transkateter radyofrekans modifikasyonu, uygun ICD deşarjlarının sayısını azaltabilir ve KY ve sık, tekrarlayan ventriküler taşiaritmili hastalarda aritmik fırtınayı sonlandırmak için kullanılabilir, bu nedenle bu hastalarda düşünülmelidir. Dirençli ventriküler aritmisi olan hastalarda elektrofizyoloji uzmanlığı ile KY Ekibi üyelerinin tavsiyesine başvurulması önerilir. [66]

Tablo 1. Kalp yetmezliğinde ventriküler taşiaritmilerin tedavisi için ESC önerileri [2]

Öneriler	Sınıf	Seviye
Ventriküler taşikardiyi agreve eden durumlar (Örnek olarak düşük potasyum seviyesi, düşük magnezyum seviyesi, devam eden iskemi) düzeltilmelidir.	IIa	C
Beta-blkoer, MRA, Sakubitril/vaslsartan verilmesi kesinlikle önerilir. Çünkü bunların AKÖ riskini azalttığı kanıtlanmıştır.	I	A
Düşük EF'li KY olan seçilmiş hastalarda ICD ve ya CRT-D takılması kesinlikle önerilir.	I	A
ICD' si olan ya da takılması kontrendike olan semptomatik VT' si olan hastalarda risk faktörlerine dikkat edilmesi, en uygun medikal tedavi, amiodaron, katater ablasyon ve CRT dahil olmak üzere çeşitli stratejiler VT' yi azaltmak için düşünülmelidir.	IIa	C
Güvenlik gereği, asemptomatik KY olan hastalarda antiaritmik ajanların kullanılması önerilmez (KY' nin kötüleşmesi, proaritmik etki ve AKÖ).	III	A

Sonuç

Sonuç olarak, KY'de ventriküler taşikardi, değişen nörohormonal sinyalizasyon, yapısal remodelling ve elektrofizyolojik değişiklikler gibi çok sayıda karmaşık mekanizmanın fenotipik bir ekspresyonunu temsil eder. Teknolojik gelişmeler karmaşık yapıları daha iyi anlamamıza izin verdiğinden, KY nin patofizyolojisini ve VT için bir tetikleyici olarak rolünü daha iyi anlayabiliriz.

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