BÖLÜM 3

INTRAKRANİYEL STENTLEMEDE ANTİTROMBOSİT VE ANTİAGREGAN TEDAVİ

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GİRİŞ

Son yıllarda anevrizma tedavisinde ve intrakranyal darlıkların tedavisinde artan stent kullanımı ile birlikte, bu tedaviler ile ilişkili oluşabilecek tromboembolik komplikasyonların önlenmesinde kullanılan antitrombotik tedavilerde oldukça önem kazanmıştır. Günümüzde nörogirişimsel işlemleri gerçekleştiren hekimlerin; periprosedürel tromboz mekanizmaları, antitrombotik ilaçların farmakolojik özellikleri, bu ilaçların endikasyonları ve bu ilaçların hasta bazlı etkinliğinin değerlendirilmesinde kullanılan direnç testleri konusunda bilgi sahibi olmaları tromboembolik ve hemorajik komplikasyonların önlenmesinde oldukça faydalıdır. Antitrombotik tedavi kullanımı yaklaşımlarında net bir görüş birliği bulunmamakta olup, farklı merkezlerde farklı medikasyon yaklaşımları benimsenebilmektedir. Bu bölümde; intrakranyal stent uygulamaları açısından uygun antitrombotik ilaçlar, bu ilaçların farmakolojik özellikleri, direnç testleri, endikasyonlar ve doğru hasta seçimi konusunda detaylı bilgiler yer almaktadır. Ayrıca hemostaz mekanizmalarına da kısaca değinilmiştir.

HEMOSTAZ

Primer Hemostaz

Zarar görmüş damar duvarındaki kolajene trombosit yüzey glikoproteinleri VI (GPVI) ve integrin α2β1 aracılığıyla ve trombosit yüzey glikoprotein Ib (GP1b)-IX-V kompleksine von Willebrand faktörü (vWF) bağlanmasıyla trombositlerin yaralanma bölgesine adezyonu gerçekleşir. GPVI, trombositlerin kolajenle uyarılmasında kilit bir rol oynayan 58 kD boyutunda bir trombosit membran glikoprotein reseptörüdür. Damar duvarının zarar görmesi ve subendotelyal matriksin kan akışına maruz kalması trombositlerin adezyonuna neden olur. Kolajen liflerinin kan damarlarında bulunan en yaygın formları olan tip I, III ve IV kolajenlerin yüksek trombojenik özelliği vardır. Başlangıç adezyonunun ardından trombosit-trombosit etkileşimi gerçekleşir. Adenozin difosfat (ADP) ve kolajen gibi farklı agonistler, adezyon sonrası trombosit aktivasyonunda rol oynar. Trombositler, trombosit yüzey membranında sunulan spesifik reseptörlere agonistlerin bağlanmasıyla aktive edilir. Trombosit aktivasyonu, plazmadan kalsiyum iyonlarının

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Semptomatik ve asemptomatik ICAD için risk faktörleri arasında yaş, Asya kökeni ve siyah ırk, hipertansiyon, diyabet, hiperlipidemi, metabolik sendrom, hareketsiz yaşam tarzı ve sigara içmek yer almaktadır (131). ICAD'deki inme mekanizmalarını; arterden artere embolizasyon, perforanların oklüzyonu ve bozulmuş distal perfüzyon oluşturmaktadır (130).

Antitrombotik tedavi ICAD hastalarında önemli bir tedavi yöntemidir. WASID çalışmasında tekrarlayan inme oranı warfarin ve ASA randomizasyon kolları arasında benzerlik gösterirken, ortalama 2 yıllık takipte warfarin kolunda aspirin koluyla karşılaştırıldığında istatistiksel anlamlı derecede daha yüksek ölüm oranı (%9,7-%4,3) ve majör kanama (%8,3 -%3,2) saptanmıştır (132). WASID çalışmasının ardından antitrombotikler ICAD için standart tedavi olmuştur, ancak daha yeni ve daha güvenli, yeni oral antikoagülanlar henüz ICAD hastalarında yeterince araştırılmamıştır (133).

ICAD hastalarında dual antitrombotik tedavi, risk faktörlerinin modifikasyonu ve hiperlipideminin tedavisi medikal tedavi yaklaşımını oluştururken, intrakranyal darlıkların stent ile tedavisi medikal tedavi ile kontrol altına alınamayan olgularda en önemli tedavi seçeneği olarak öne çıkmaktadır. İntrakranyal darlıklara yönelik stentleme öncesinde ve tedavi sırasında antitrombotiklerin kullanımı kanamamış anevrizmaların stent ile tedavisine benzer şekilde uygulanmaktadır. Antitrombotiklere yönelik direnç testleri gerçekleştirilerek, uygun antitrombotikler altında tedavinin gerçekleştirilmesini takiben, taburculuk sonrasında dual antitrombotik tedavi en az 6 ay süreyle sürdürülmektedir. Sonrasında hastanın aterosklerotik yükü göz önüne alınmalı, yaygın hastalıkta benzer tedavinin uzun süreli kullanımı ya da stent ile tedavi edilmiş fokal aterosklerotik hastalık varlığında yaşam boyu tekli antitrombotik tedavi yeterli olabilmektedir.

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