Kalp ve Damar Cerrahisi : Teori, Metodoloji ve Uygulama

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İÇİNDEKİLER


BÖLÜM 1

Kardiyak Yaralanmalar

Sedat Ozan Karakişi[1] & Doğuş Hemşinli[2] & İbrahim Yel[3]

KARDİYAK YARALANMALAR

1-PENETRAN KALP YARALANMALARI

Kalp yaralanmaları eski çağlardan beri hekimlerin ilgisini çeken milattan sonra 19. Yüzyılın sonlarına kadar kesin ölümle sonuçlandığı düşünülen bir durumdur. 1987'de Rehn penetran kalp yaralanmasında ilk başarılı tamiri yayınlamıştır. 1986 yılında Calhoon künt travmaya bağlı kardiyak rüptür hastalarında %70 survey bildirmiştir. (Goldstein AL et al. 2017.) İyi hazırlanmış travma merkezlerinde bile künt kardiyak travmanın mortalitesi yüksektir.

Penetran kardiyak yaralanmalar kesici delici alet (bıçak, şarapnel vs.), kırılan kemikler nedeniyle oluşabilir. En sık sağ ventrikül sonra sırasıyla sol ventrikül, sağ atriyum ve sol atriyuım yaralanması görülür. Nadiren papiler kaslarda, ventrikül septumunda ve kalp kapaklarında yaralanma görülebilir. Koroner arter yaralanmaları nadir görülmekle birlikte genellikle distal tip olup ligasyon yeterli olmaktadır. (Goldstein AL et al. 2017)(Asensio et al. 1996)

Temelde penetran kardiyak yaralanmanın iki sonucu vardır, kanama ve tamponad. Bu klinik tablo minimal olabildiği gibi ağır bir şekilde de olabilir. Bıçak gibi yaralanmaların çoğunda tamponad olan ya da olmayan şok tablosu hakimdir. Ateşli silah yaralanmalarında ise acil servise ulaştıklarında ölüm ya da ölüme yakın bir klinik tablo görülür.

Kesici alet yaralanmalarında kardiyak yaralanmaların büyük çoğunluğunda perikardiyal ve kardiyak kesi küçüktür. Oluşan trombüs ve etraf yağ dokusu yaralanan kardiyak bölgeyi kapatabilir. Özellikle sağ ventrikül yaralanmalarında kanama bu şekilde durarak tamponad kliniği hâkim olur. Oluşan tamponadın surveyi artırdığı tespit edilmiştir. Oluşan pıhtının intraperikardiyal bası oluşturması ile sağ atriyum ve sağ ventrikül yaralanmalarında kanama durabilir. (Ivatury and Rohman 1992)(Gosavi, Tyroch, and Mukherjee 2016)

Göğüse penetran travması olan tüm hastalarda kalp yaralanması olabilir. Ekokardiyografi şüphesi olan tüm hastalara yapılabilir. İntraperikardiyal küçük effüzyonlar stabil hastalarda ekokardiyografi ile takip edilebilir. Subksifoid

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perikardiyal pencere şok, tamponad bulguları olmayan hastalarda ciddi bir travma sebep olmadan tanı koydurur. (Stassen 2021)

Acil serviste kalp yaralanması düşünülen hastada şok durumunda bile acil olarak resüsitasyon torakotomisi yapılmalıdır. Penetran kalp yaralanması olan bir hastanın, yaralanma ve cerrahiye alınma arasında yarım saatten fazla zaman geçmemesi gerekir. Ateşli silah yaralanmalarında mortalite kesici delici alet yaralanmalarına göre daha fazladır. Künt travma sonucu tamponad buğuları gösteren hastada ameliyat endikasyonu acildir ve mortalisi %85ten fazladır. Ameliyata alınmayan hastalar mortal seyreder.

Tamponad gelişmiş ise hemoperikarda ponksiyon yapılır ve hemen sternotomi yapılır. Stentomi ile kardiyak muayene yapılabildiği gibi gerektiğinde torakal bölgelerde değerlendirilebilir. Ateşli silah yaralanmalarda balistik etki ile yarlanma oluşabilir. Sternotomi sonrası intraplevral ciddi bir yaralanma varsa torakotomi de ilave edilerek kanama kontrol altına alınabilir. Yaralanma vetrikülde ve küçük bir yuva varsa parmak basılarak geçici hemostaz sağlanabilir. Yaralanma atriyumda ise lateral klemp konulabilir. Yaralanma önemli ve parmakla kontrol altına alınamıyorsa yara orifisine foley sonda sokulup, balonu dışarı çıkmayacak kadar şişirilip kanama kontrol altına alınabilir.

Kalp kavitelerinde laserasyon, multipl yaralanma görüldüğünde ekstrakorporeal sirkülasyona geçilmesi gerekir. Özellikle ateşli silah yaralanmalarında kurşunun trasesine göre multipl yaralanma görülebilir.

Ventrikül yaralanmaları parmak ile basılarak hemostaz sağlanmışsa 3/0 teflon veya perikardiyal destekli "U" sütür ile onarım yapılabilir. İşlem sırasında ventriküler kasların yırtılmamasına dikkat edilmelidir. Dikiş geçilirken koroner arterlerin içine alınmaması gerekir.

Ventrikül duvarında büyük defektler varsa onarım yapmak için pompaya girmek gerekir ancak bu durum nadirdir. Defektin onarımı için omentum grefti gibi yama gerekebilir. Atriyum lezyonları klempin altından geçilen sütürlerin bağlanmasıyla tamir edilir. Septum ya da kapaklardaki lezyonlar hemodinamiyi bozmuyorsa ikinci bir ameliyat sırasında değerlendirilir.

Koroner arter yarlanmaları genelde minör dallara bağlı oluşur. Ancak özellikle sol anterior desending ve koroner arterlerin orta veya proksimal kesimlerinde olan yaralanmalarda bypass grefti gerekir.

Büyük damarların intraperikardiyal yaralanması nadir bir olaydır.

Vena Cava İnferior yaralanması genelde parsiyel bir yaralanma şeklinde görülür ve klep konulduktan sonra lateral sütür konularak kontrol altına alınabilir.

Vena Cava Superior yaralanması nadirdir ve genelde mortaldir. Yaklaşım genellikle sternotomi ve sağ anterior torakotomi ile olur. En ideal yaklaşım posterolateral torakotomi olmasına rağmen acil şartlarda pek mümkün değildir. Kelmp konulduktan sonra direkt sütür veya perikardiyal yama ile onarım yapılabilir.

Aort yaralanması nadiren acile canlı olarak giriş yapar. Yaklaşıma eksplorasyon sırasında karar verilir. Asendan aort, arcus aorta proksimal kesimi, brakiosefalik arter ve sol karotis arterin proskimal kesimlerinin ekslplrasyonu için sternotomi yeterli olurken sol subklavian arter için sol anterior torakotomi de yapmak gerekebilir. Yaralanan bölgenin önce parmakla hemostazı sonrası direk sütür veya greft interpozisyonu ile onarım yapılabilir.

Pulmoner Arter yaralanmaları masif hemo-pnömotoraks kliniği ile ortaya çıkar. Pulmoner arter sıklıkla hilus bölgesinden yaralanır. Hemostaz sıklıkla hilustaki damarların kontrol altına alınması ile sağlanır.

Sağ Pulmoner Arter kanamalarında en basit kanama kontrolü parmak ile komprasyon yapılmasıdır. Arterin frajil olması ve kolayca diseke olabileceğinden klemlplenme önerilmez. Sağ pulmoner artere ulaşmanın en kolay yolu aorto-kaval aralıktan girilerek pulmoner arterin askıya alınmasıdır. Vena cava superior askıya alınıp dışa doğru çekildikten sonra pulmoner arterdeki yaralı kısım sütüre edilir ya da tam kat kesi durumunda uç-uca anastomoz yapılır.

Sol Pulmoner Arter yaralanmasında genel olarak intraperikardiyal yaklaşımla hemostaz sağlanabilir.

Kalp yaralanmalarında hastanın geliş durumu çok önemlidir. Acil servise geldiğinde yaşam bulguları olmayan hastaların kliniği mortal seyreder. Kardiyak yaralanmaların yarısı acil servise canlı ulaşamaz. Kesici delici alet yaralanmalarının mortalitesi ateşli silah yaralanmalarına göre daha azdır. Kardiyak yaralanmaların uzun dönem sonuçları çok iyidir ancak hastalarda kardiyak arrest bağlı hipoksik beyin, ventriküler septum ve kapak yaralanması gibi etkenler morbidite sebebidir.

2-KÜNT KARDİYAK YARALANMALAR

Künt kardiyak travma en sık motorlu araç kazalarında, yüksekten düşme, crash yaralanma gibi mekanizmalarla meydana gelir.

Direkt kuvvetler prekordiyal ekti oluşturarak yaralanma yapabilir, ani artan intratorasik basınç direkt olarak kalbe yansır. İntraabdominal kuvvetler indirekttir, artan venöz dönüş, diyafragmanın yükselmesi ve kalp boşluklarında over distansiyon oluşur. Kompresif kuvvetler ise kalbin sternum ve vertebra arasında sıkışmasıyla oluşur. Büyük damarlarda asılı şekilde duran kalpte deselerasyon kuvvetleri sarkaç benzeri etki oluşturur. Sonuç olarak direkt,

indirekt, kopressif ve deselerasyon kuvvetlerinin kombinasyonu ile de künt travma olabilir. (Janson et al. 2003)(Marcolini and Keegan 2015)

Künt travma sonucu en sık miyokardiyal kontüzyon oluşmaktadır. Yaralanma; subendokardiyal, miyokardiyal hemoraji, sellüler düzeyde ödem ya da miyokardı tam kat tutan enfarktüse kadar değişebilir. Oluşan hasarın derecesi travmanın şiddetine bağlıdır.

Mikroskopik olarak hemoraji, lökosit infiltasyonu, ödem, nekroz görülebilir. Kontüze miyokardiyal dokusu ile normal doku arasında keskin bir sınır vardır ve inotrop destek verildiğinde koroner arter hastalığında olduğu gibi hasarlanan alanda genişleme görülmez. Yaralanma derecesine göre miyokard fonksiyonunda azalma olur, nekroz gelişmişse, koroner arter hastalığındakinden ayırt edilemeyen, skar dokusu gelişir.

Miyorkardiyal kontüzyon olan hastalarda göğüs ağrısı sık olarak görülür ancak, bunun kardiyak, yumuşak doku ya da kemik kaynaklı mı olduğunun ayrımını yapmak pek mümkün değildir.

Normal EKG konyüzyonu ekarte ettirmez ancak yeni olan bir Q dalgası diyagnostiktir. CK-MB miyokardiyal hasar için sensitiftir ancak yaygın doku hasarında diğer dokulara bağlı da yükseleceği için sensitivitesi azalır. Troponın I EKG ile birlikte değerlendirildiğinde sensitiftir. Ekokardiyografide bölgesel ya da global hipokinezi görülebilir.

Miyokardiyal kontüzyonu olan hastalarda aritmi, miyokardiyal-ventriküler rüptür kardiyak yetmezlik gelişebilir. Eğer hasta stabil ve ek yaralanmaların tedavisi ile stabil edilmişse önemli kontüzyon yoktur ve prognoz iyidir. Eğer önemli kontüzyondan şüpheleniliyorsa ya da tanı konulmuşsa yakın takip ve monitörizasyon gerekir. Hastalar aritmi yönünden takip edilmelidir. Potasyum ve magnezyum seviyeleri normale getirilmelidir.

Ventriküler disfonksiyondan dolayı kardiyak outputu düşmüş olan hastalarda inotrop destek ve intraaortik balon pompası desteğine ihtiyacı olabilir. Miyokardiyal kontüzyonlar genelde kalıcı sekel bırakmadan iyileşir nadiren şiddetli kontüzyona bağlı miyokardiyal fibrozise bağlı progresif kalp yetmezliği gelişebilir. Bu durumda transplantasyon gerekir. (Lindstaedt et al. 2002)(Salim et al. 2001)

Kardiyak rüptür göğüs travmalarının en sık ölüm sebeplerinden biridir. Rüptür travmayı takiben ya da 2 hf sonra gelişir. Laserasyon genelde periyadiyak-epikardiyal yüzeydedir, belirgin hemodinamik bozukluk yapmaz. Ancak laserasyon miyokardın derinlene doğru ilerlerse veya tam kat olursa hastada tamponad kliniği gelişebilir.

Ventriküler septum yaralanmaları penetran ve non onetran göğüs travmlarında görülebilir. En sık müsküler bölümde apekse yakın lokalizasyonda olur. Septum yaralanmalarında ilk anda palpabl thrill alınabilir. Künt travmalarından sonra oluşan ventriküler septal yaralanma birkaç gün sonra nekroz geliştikten sonra ortaya çıkar. Sol ventrikül geç diyastol veya erken sistolinde olan künt travmalarda septal yaralanmalar daha sıktır. Operasyon sırasında kalpte thrill alınabilir ya da sağ atriyum-pulmoner arterden alınan kan örnekleri ile tanı konulabilir. Alternatif ekokardiyografi ve kateterizasyonla da tanı konulabilir. Septal laserasyonlar genellikle akut dönemde onarım gerektirmezler. Bu lezyonların spontan kapanabileceği görülmüştür. Erken dönem oluşan hemorajik ve ekimotik kas dokusunun tamirinden sonra nüks olabilmektedir. 2-3 ay sonra oluşan fibröz doku daha kesin bir kapanma sağlar. Erken dönem tamirde hastanın kardiyopulmoner bypassa girmesi gerekeceği için heparine bağlı başka yüksekse, kardiyak dilatasyon görülüyorsa ya da akciğer konjesyon bulguları varsa eken dönem tamiri gerekebilir. (Stamm et al. 2002)

Valvüler yaralanmalar daha çok künt travmlarda ortaya çıkar. Ani yükselen basınca bağlı liflerde ya da komissurlerde yırtılmalar olur.

Aort kapak yaralanması künt travmada en sık ölüm sebebidir. Aort kapak yaralanması sol ventirikül diyastolünde iken ani basınç artışına bağlı olarak oluşur. Aort kapak yetmezliğine bağlı klinik bulgular oluşur. Diyastolik basınçta azalma, geniş nabız basıncı, taşikardi görülür, diyastolik üfürüm duyulabilir. Tedavi edilmezse kalp yetmezliği progresif olarak ilerler. Klinik aort kapak yetmezliği düzeyine bağlıdır, bazı hastalarda acil kapak replasmanı ihtiyacı olabilir. Bazı hastalarda aort diseksiyonu ile birliktedir.

Mitral kapak yaralanması aort kapak yaralanmalarına göre daha az görülür. Geç diyastol veya erken sistolde oluşan travmayla meydana gelirler. En sık papiller adele rüptürü vardır, korda tendinea rüptürü ve leaflet yırtılmaları azdır. Kalp yetmezliği semptomları vardır. Transtorasik ekokardiyografi ile tanı konulabilir. Klinik bulgulara göre operasyon zamanına karar verilir. Şiddetli yetmezlik başlangıçta intraaortik balon pompasından fayda görür. (Pathi, Jones, and Davidson 1996)

Triküspit kapak yaralanması, kapağın anterior yerleşimine bağlı olarak, mitral kapağa göre daha sık görülür. Klinik olarak diğer kapaklara göre daha iyi progresiflidir. Sağ kalp yetmezliği bulguları görülür. Ekokardiyografi ile tanı konulur. Onarım mümkünse erken cerrahi önerilir. (Chiu et al. 1996)

Künt travmalarda koroner arter yaralanmaları nadirdir, en sık sol anterior desenden arter sonra sırasıyla sağ koroner ve circumflex arter gelir. Eğer distal arter uçlarında laserasyon varsa ligate edilir. Miyokard enfaktüsündeki gibi

medikal tedavi verilir. Arteriyo-venöz fistül gelişmişse fistülün proskimal ve distaline ligasyon ve distal bypass yapılır.

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BÖLÜM 2

Periferik Damar Yaralanmaları

Mahmut Fırat Kaynak[1]

1. Giriş

Periferik damar yaralanmaları (PDY), özellikle penetran ve yüksek enerjili travmalar sonucu acil servis başvurularında karşılaşılan, morbidite ve mortalite riski yüksek durumlar arasında yer alır. Gecikmiş tanı ya da yetersiz müdahale, ampütasyon oranlarında artışa neden olabilir (Xu et al., 2019).

PDY'ler, travma nedeniyle başvuran hastalarda %1–4 oranında görülmekle birlikte, büyük damarların tutulduğu olgularda bu oran hayati risk taşır (Feliciano et al., 2011). Bu bağlamda acil tıp hekimlerinin PDY hakkında bilgi sahibi olmaları, mortalite ve ampütasyon oranlarını doğrudan etkiler. (Kobayashi et al., 2020).

2. Epidemiyoloji

PDY genellikle genç, aktif bireylerde, trafik kazaları, ateşli silah yaralanmaları, kesici-delici alet travmaları ve endüstriyel kazalar gibi yüksek enerjili travmalar sonucu oluşur. En sık alt ekstremite (özellikle femoral ve popliteal arterler) ve üst ekstremite brakiyal arterler etkilenmektedir (Halvorson et al., 2011).

Mirdamadi ve arkadaşlarının (2022) retrospektif verilerine göre, PDY'lerin %60'tan fazlası penetran travmalardan kaynaklanmaktadır. Vakaların %80'i erkeklerde görülmekte olup, ortalama yaş 30 civarındadır (Mirdamadi et al., 2022).

Yaralanma Mekanizmalarına Göre Sıklık:

- **Penetran travmalar:** %60–70 (ateşli silah, bıçak yaralanmaları)
- **Künt travmalar:** %20–30 (düşme, trafik kazaları)
- **İatrojenik nedenler:** %5–10 (kateterizasyon, girişimsel prosedürler)

Ayrıca savaş ve afet alanlarındaki veriler, ekstremite damar yaralanmalarının bu tür koşullarda oldukça sık görüldüğünü ortaya koymaktadır (Xu et al., 2019).

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3. Klinik Değerlendirme ve Fizik Muayene Bulguları

PDY acil serviste değerlendirilmesi hızlı, sistematik ve hedefe yönelik olmalıdır. Erken tanı, ekstremitenin korunması açısından hayati öneme sahiptir.

3.1 Sert ve Yumuşak Vasküler Bulgular

Klinik olarak vasküler yaralanmalar, "sert (hard)" ve "yumuşak (soft)" bulgulara göre sınıflandırılır. Bu sınıflama, cerrahi gereksinim açısından yönlendirici olup, tanı algoritmalarında temel rol oynar (Kobayashi et al., 2020).

Sert Bulgular (hard sign) (Cerrahi endikasyon kabul edilir):

- Devam eden eksternal hemoraji
- Nabızsız ekstremite
- Genişleyen pulsatif hematom
- Thrill sesi alınması
- Distal iskemik bulgular (soğukluk, solukluk, motor/duyu kayıp)

Yumuşak Bulgular (soft sign):

- Yakın anatomik bölgede yara
- Küçük hematom
- Nörolojik defisit
- Hafif kanama
- Geçici nabızsızlık (şok sonrası vs.)

3.2 Periferik Nabız Muayenesi

Periferik nabızların bilateral palpasyonu önemlidir. Kuşkulu olgularda el Doppleri ile değerlendirme yapılmalıdır. (Halvorson et al., 2011).

3.3 Klinik Sınıflama ve Karar Algoritmaları

Western Trauma Association tarafından önerilen algoritmaya göre, 'hard sign' varlığında direkt cerrahi girişim veya acil BT anjiyografi önerilir. Yumuşak bulgular varsa, görüntüleme sonrası karar verilir (Feliciano et al., 2011).

4. Görüntüleme Yöntemleri ve Tanı Yaklaşımı

PDY tanı koymanın temel amacı, hayatı tehdit eden kanamaları veya ekstremiteyi tehdit eden iskemiyi hızla ortaya koymaktır. Klinik bulguların yetersiz olduğu durumlarda görüntüleme yöntemleri devreye girer. Bu yöntemler,

tanıyı netleştirmekle kalmaz; aynı zamanda müdahale planlamasında da yol göstericidir.

4.1 Doppler Ultrasonografi (USG)

Renkli Doppler USG, acil serviste hızlı uygulanabilirliği ve noninvaziv doğası sayesinde ilk basamak görüntüleme yöntemi olarak öne çıkar. Özellikle yumuşak vasküler bulguların olduğu olgularda yararlıdır. Ancak yöntemin kullanıcıya bağımlı olması, derin veya travmaya bağlı ödemli dokularda tanısal duyarlılığını sınırlayabilir (Halvorson et al., 2011).

4.2 BT Anjiyografi (CTA)

BT anjiyografi, PDY tanısal doğruluğu en yüksek yöntemlerden biridir. Kontrast madde ile yapılan bu tarama, damar lümeni, trombüs, aktif kanama, pseudoanevrizma gibi patolojilerin hızlı bir şekilde değerlendirilmesini sağlar. Günümüzde, özellikle stabil hastalarda cerrahiye karar verme sürecinde sıklıkla tercih edilmektedir (Kobayashi et al., 2020).

4.3 Anjiyografi (DSA)

Klasik dijital subtraksiyon anjiyografi (DSA), hem tanı hem de tedavi amacıyla kullanılabilen bir yöntemdir. Ancak zaman ve kaynak gerektirdiğinden, genellikle girişimsel radyoloji olanaklarının mevcut olduğu merkezlerde uygulanır. Endovasküler müdahale planlanan olgularda vazgeçilmezdir (Feliciano et al., 2011).

4.4 ABI (Ankle-Brachial Index)

ABI ölçümü, klinik şüphe taşıyan ancak sert bulguları olmayan hastalarda tarama amacıyla kullanılabilir. Brachial ve ayak bileği arteriyel basınçları karşılaştırılarak elde edilen bu indeksin <0.9 olması, vasküler yaralanma açısından ileri görüntüleme gerekliliğini ortaya koyar (Modrall et al., 1998).

5. Yaralanma Tipleri ve Damar Patolojileri

PDY tipi, hem klinik tablonun ağırlığını hem de müdahale yöntemini belirler. Yaralanmanın mekanizmasına bağlı olarak arterde veya vende oluşan hasar farklı formlarda karşımıza çıkar. Bu farklılıklar, tanı stratejilerinin yanı sıra cerrahi ya da endovasküler tedavi planlamasında da belirleyici rol oynar.

5.1 Transeksiyon

Damarın tam kalınlıkta kesilmesi anlamına gelen transeksiyon, genellikle penetran travmalar sonucu ortaya çıkar ve çoğu zaman yoğun kanama ile

seyreder. Bu durum, acil cerrahi onarım gerektiren bir tablodur. Damar uçları genellikle retrakte olur ve distal perfüzyon hızla bozulur (Kobayashi et al., 2020).

5.2 Intimal Yaralanma

Damarın iç tabakasında meydana gelen bu tip hasar, çoğunlukla künt travmalarla ilişkilidir. İntimal diseksiyon veya flap oluşumu, zamanla tromboz gelişmesine neden olabilir. Bu tür yaralanmalar, başlangıçta belirgin nabız değişikliği olmadan da seyredebilir; bu nedenle şüpheli durumlarda görüntüleme kaçınılmazdır (Halvorson et al., 2011).

5.3 Tromboz

Travmatik endotel hasarı sonrasında damar içinde pıhtı oluşabilir. Bu durum, perfüzyon kaybı ile kendini gösterir ve genellikle distal nabızların kaybolmasıyla birlikte değerlendirilir. Tromboz hem arteriyel hem venöz sistemde görülebilir ve klinik olarak iskemik bulgularla seyreder (Feliciano et al., 2011).

5.4 Arteriovenöz Fistül

Penetran travmalarda arter ve venin birlikte hasar görmesi durumunda, aralarında anormal bir bağlantı oluşabilir. Bu durum arteriyel kanın venöz sisteme doğrudan geçmesine neden olur ve palpabl thrill ya da duyulan brui ile tanınabilir. Tedavi edilmediği takdirde kalp debisinde artış ve distal iskemi gibi sorunlara yol açabilir.

5.5 Pseudoanevrizma

Damar duvarında kısmi yırtılma sonucu, dışa doğru genişleyen ancak endotel ile kaplı olmayan bir balonlaşma meydana gelir. Genellikle gecikmiş olarak ortaya çıkar ve enfekte olabilir. Ultrasonografi ve BT anjiyografi ile kolayca tanınır (Xu et al., 2019).

6. Acil Müdahale ve Cerrahi Olmayan Yaklaşımlar

PDY tedavi süreci, olay yerinden başlayarak hastane içi müdahalelere kadar uzanan çok aşamalı bir zinciri içerir. Acil servisteki ilk değerlendirme, sadece hayat kurtarıcı olmakla kalmaz; aynı zamanda ekstremitenin fonksiyonunu korumada belirleyici rol oynar. Cerrahi dışı ilk yaklaşım adımları, özellikle masif kanamaların kontrolü, hipovoleminin önlenmesi ve iskemik sürecin geciktirilmesine odaklanır.

6.1 Basınçlı Kompresyon- Turnike uygulaması

Aktif hemorajisi olan hastalarda doğrudan basınç uygulanması, en temel ve etkili yöntemlerden biridir. Temiz bir gazlı bezle yapılan basınç uygulaması,

arteriyel kanamaların geçici kontrolünde oldukça etkilidir. Özellikle geçici stabilizasyon sağlanıncaya kadar cerrahiye zaman kazandırır (Feliciano et al., 2011). Turnike uygulaması ise periferik perfüzyonu keserek iskemiye yol açabileceğinden, süresi 1–2 saatle sınırlandırılmalı ve mümkün olan en erken zamanda çözülmelidir. (Xu et al., 2019).

6.2 Ekstremitenin Pozisyonlanması ve Splintleme

Travmaya uğrayan ekstremitenin uygun pozisyonda immobilize edilmesi hem kanamanın kontrolüne yardımcı olur hem de mevcut damar yaralanmasının daha da kötüleşmesini engeller. Bu, özellikle eşlik eden kemik kırıklarının bulunduğu olgularda önemlidir.

6.3 Sıvı Resüsitasyonu ve Transfüzyon

Masif hemorajisi olan hastalarda hipovolemik şokun önlenmesi için erken sıvı replasmanı ve gerekirse kan transfüzyonu yapılmalıdır. Ancak son yıllarda travma resüsitasyonundaki eğilim, "hedefe yönelik sıvı tedavisi" yaklaşımı ile minimal sıvı verilerek yeterli perfüzyon sağlamak yönündedir (Kobayashi et al., 2020).

6.4 Antibiyotik ve Tetanus Profilaksisi

Açık yaralanmalarda enfeksiyon riski yüksek olduğundan, geniş spektrumlu antibiyotiklerle erken profilaksi uygulanmalıdır. Ayrıca, tetanoz immünizasyon durumu sorgulanmalı ve gerekirse profilaksi sağlanmalıdır.

7. Cerrahi Yaklaşımlar ve Endovasküler Müdahaleler

PDY cerrahi tedavi, damar bütünlüğünün yeniden sağlanmasını ve ekstremite perfüzyonunun korunmasını amaçlar. Müdahale kararı, yaralanmanın tipi, yeri ve eşlik eden yapısal hasarlara göre şekillenir.

7.1 Cerrahi Teknikler

Açık cerrahi, özellikle aktif kanama, iskemik ekstremite ya da damar transeksiyonu gibi durumlarda tercih edilir. Temel cerrahi işlemler arasında:

- **Primer tamir** (uç uca anastomoz),
- **Vasküler greftle rekonstrüksiyon** (özellikle vena saphena kullanımı),
- **Ligasyon** (venöz yaralanmalarda)
- **Endovasküler Girişimler**

(Kobayashi et al., 2020).

8.1 Erken Dönem Komplikasyonları

🔴 Masif Kanama ve Hemorajik Şok

Aktif arteriyel kanamalar kısa sürede hipovolemik şoka yol açabilir. Özellikle gözle görünmeyen retroperitoneal veya kas içi hematomlarda kan kaybı gözden kaçabilir. Acil serviste nabız, kapiller dolum ve mental durum takibiyle perfüzyonun dinamik olarak izlenmesi esastır (Feliciano et al., 2011).

⚠️ Akut Ekstremite İskemi

Distal nabzın kaybı, soğukluk, solukluk ve motor-duyu kaybı ile kendini gösterir. Gecikmiş tanı ampütasyonla sonuçlanabilir. Bu nedenle, özellikle nabzı olmayan ama kanaması olmayan hastalarda iskemik bulgular titizlikle aranmalıdır.

💥 Kompartman Sendromu

Arteriyel rekanalizasyon sonrası ya da damar tamirine eşlik eden ödemde, kapalı fasyal alanlarda basınç artışı gelişebilir. Ağrı, pasif ekstansiyonla artış, duyu kaybı ve kas zayıflığı ilk belirtilerdir. Hekim, saatler içinde gelişebilecek bu tabloya karşı tetikte olmalıdır (Kobayashi et al., 2020).

🦠 Enfeksiyon

Özellikle kontamine yaralanmalarda ya da gecikmiş müdahalede yara enfeksiyonu, fasiit, greft enfeksiyonu gelişebilir.

8.2 Geç Dönem Komplikasyonları

🧱 Vasküler Oklüzyon / Tromboz

Damar tamirinin ardından reoklüzyon riski vardır. Semptomlar günler veya haftalar sonra ortaya çıkabilir. Uzun dönem takipte distal nabızlar ve ABI ölçümü ile perfüzyon değerlendirilmelidir.

📈 Pseudoanevrizma

İlk müdahale sırasında fark edilmemiş damar duvar hasarları zamanla balonlaşmaya neden olabilir. Genellikle pulsatif kitle ve brui ile ortaya çıkar. Doppler USG veya BT anjiyografi ile tanı konur (Xu et al., 2019).

🔁 Arteriovenöz Fistül

Bazı olgularda arter ve ven arasındaki bağlantılar geç dönemde belirti verir. Klinik olarak sürekli brui, genişlemiş damarlar ve venöz hipertansiyon bulguları görülebilir.

♿ Nörolojik Sekeller

Damar hasarına eşlik eden sinir yaralanmaları uzun dönem parezi, parestezi ve fonksiyon kaybına yol açabilir.

9. Özel Durumlar

9.1 Pediatrik Hastalar

Çocuklarda damar çapı daha küçük olduğundan hem tanısal görüntüleme hem de cerrahi müdahaleler teknik olarak daha zordur. Ek olarak, kan kaybı toleransları düşük olduğundan, az miktarda hemoraji bile hızlı dekompansasyona yol açabilir. Bu nedenle, nabız takibi ve kapiller dolum zamanı gibi klinik bulgulara daha fazla güvenilirlik atfedilir (Kobayashi et al., 2020).

9.2 Antikoagülan Kullanan Hastalar

Travmadan önce antikoagülan veya antitrombosit ajan kullanan hastalarda hemorajik komplikasyon riski belirgin şekilde artar. Klinik olarak kanama daha geç fark edilebilir ve hematomlar hızla büyüyebilir. İlk değerlendirmede fark edilemeyecek gizli kanama odakları olabilir.

9.3 Afet/Savaş Alanı ve Kısıtlı Kaynak Ortamları

Afet bölgelerinde veya askeri operasyon sahalarında, tanı ve tedavi imkânları sınırlı olabilir. Bu gibi ortamlarda turnike uygulaması, basit kompresyon ve hasar kontrol cerrahisi gibi yöntemler öne çıkar. Erken triyaj ve tahliye stratejileri hayati önem taşır (Xu et al., 2019).

10. Sonuç ve Klinik Öneriler

PDY, erken tanı ve uygun müdahale ile yüksek oranda başarıyla yönetilebilen ancak zamanla yarış gerektiren klinik tablolardır. Acil serviste görev yapan hekimler için bu tür olgular, hızlı karar verme ve disiplinler arası iş birliği becerisi gerektirir.

🔑 Klinik Uygulama İçin Temel Öneriler

- **Fizik muayene her şeyin başlangıcıdır.** Ciddi bulguların varlığında, ileri görüntüleme beklenmeden cerrahi değerlendirme yapılmalıdır.
- **ABI ölçümü (< 0.9)**, şüpheli olgularda basit ama etkili bir tanı aracıdır.
- **BT anjiyografi**, stabil hastada tanı doğruluğu yüksek, hızlı ve erişilebilir bir yöntemdir.

- **Turnike uygulaması**, ön hastane koşullarında hayat kurtarır ancak hastane ortamında süresi mutlaka takip edilmelidir.
- **Kompartman sendromu**, rekanalizasyon sonrası dahi gelişebilir; takipte bu olasılık akılda tutulmalıdır.
- **Antibiyotik ve Tetanus profilaksisi**, açık ve kontamine yaralanmalarda geç kalınmadan başlanmalıdır.

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BÖLÜM 3

Kardiyak Miksomalar

İbrahim Yel[1] &Sedat Ozan Karakişi[2] & Doğuş Hemşinli[3]

1-GİRİŞ

Miksomalar kalbin primer benign tümörleridir. Kalbin primer tümörlerinin %80'i benigndir.(Reynen 1995) Erişkinlerde bening kardiyak tümerlerin %50'sini miksomalar oluşturur.(Sánchez Sotelo et al. 2023) Pediatrik benign tümörlerinin %10-15'ini oluşturmaktadır. Miksoma saptanan kişilerin üçte ikisi 30-60 yaş aralığındadır.(McAllister, Hall, and Cooley 1999) İnsidental saptanabildiği gibi kalbin bulunduğu bölgesine göre nefes darlığı, emboli gibi klinik bulgular da verebilir. Tipik sporadik miksomaların aksine ailesel geçişli miksomalar daha erken yaşlarda görülebilir ve cerrahi sonrası rekkürens daha fazla görülür. Histolojik olarak sporadik miksomalarla aynıdır.(Van Gelder et al. 1992)

2-PATOLOJİ-HİSTOPATOLOJİ

Miksomalar çoğunlukla aynı makroskobik ve mikroskobik özelliklere sahip olsalar da bazen farklılıklar olabilmektedir. Miksomalar çoğunlukla subendokardiyal dokunun mezenkim hücrelerinden kaynaklanan jelatinimsi, yumuşak, polipoid, pediküllü frajil bir yapıya sahiptir. Miksomaların %75'i sol atriyuma %18'i sağ atriyuma ve %4-6 ventriküllerde yerleşir.(McAllister HA 1978)

Mikroskobik olarak miksomalar çok kenarlı hücreler ve asit mukopolisakkritlerrden zengin miksoid matirksten oluşur. (Left et al. 2024) Matriks içinde yer alan hücreler genellikle tek tek ya da küçük kümeler halinde dağılmıştır. Düşük düzeyde mitotik aktivite gözlenirken, matriks bileşenleri arasında kollajen ve elastin lifleriyle birlikte düz kas hücreleri ve retikülositler yer alır. Ayrıca, kistik yapılar ve zaman zaman hemorajik alanlara da rastlanabilir.(Reynen 1995) Miksomaların subendokardiyal mezenkim hücrelerinden kökenli olabileceği düşünülmektedir. Bazı bölgelerde mikroskobik kalsiyum birikimlerine, metastatik kemik oluşumlarına ve glandüler yapılara rastlanabilir. Miksomaların taban kısmında subendokardiyuma doğru uzanarak

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oraya tutunan arter ve venler mevcuttur; bu vasküler yapılar subendokardiyal bölgede yüzeysel bir seyir izler. Ferrans ve Roberts, miksoma hücrelerinin histopatolojik özelliklerinin tümörün kökenine ilişkin kesin bilgi sunmadığını belirtmişlerdir. Ayrıca Kutanöz leiomyomatozis ile birlikte görülebildiği gibi ateş, hipergamaglobulinemi ve kilo kaybı gibi sistemik bulguların, nöronal kökenli mekanizmalarla ilişkili olabileceği bildirilmektedir. (Ferrans and Roberts 1973) Miksomaların boyutları 1 cm ile 15 cm arasında değişiklik göstermekle birlikte, çoğunlukla yaklaşık 5–6 cm çapındadır. Ağırlıkları genellikle 50–60 gram arasında olup, bazı olgularda 175 grama kadar ulaşan miksomalar da rapor edilmiştir Tipik olarak, tümörler orijin aldıkları bölgeye bir sap aracılığıyla tutunmakta ve bulunduğu kardiyak kaviteye doğru polipoid, düzgün yüzeyli bir yapı şeklinde projekte olmaktadır. Jelatinöz ve mukoid kıvamlarıyla karakterize edilirler. Hareketlilikleri ise, sapın uzunluğu, yapının tutunduğu yer ve içerdikleri kollajen miktarına bağlı olarak değişkenlik gösterir. Çoğunlukla saplı ve mobil özellikte olmakla birlikte, geniş tabanla çevre dokulara tutunmuş, dolayısıyla hareket yeteneği kısıtlı miksoma tipleri de mevcuttur. Makroskopik olarak beyaz, sarı ya da kahverengi tonlarında olabilirler; iç yapılarında zaman zaman hemorajik alanlar ile trombüs formasyonları gözlenebilir.(McAllister HA 1978) Miksomalar dış görünüşlerine göre solid ve papiller olmak üzere ikiye ayrılmaktadır. Papiller yapıdaki miksomalar kolayca dağılabilir ve düzgün sınırlı değildir, bu durum muhtemel embolilerden sorumludur. Solid miksomalar düzgün yüzeyli ve bir kapsülle çevrilidir, çoğunlukla cerrahi olarak tek parça olarak kardiyak kaviteden eksize edilir. (Garatti et al. 2012) Atriyal Miksomalar çoğunlukla fossa ovalisten köken almakla birlikte, atriyal duvardan, mitral ve triküspit kapaklardan, koroner sinüsten, pulmoner ven girişlerinden Eustachian Valve gibi bölgelerden kaynaklanabilir. Sol atriyumda oluşan kitlelerde akciğer maligniteleri, mezotelyoma, karsinom ve sarkomlarda düşünülmelidir. Sağ atriyum tümörlerinde böbrek ve uterus tümörlerin vena kava içerisindeki uzanımları unutmamalıdır. Atriyal miksomalar çoğunlukla bir atriyuma bir noktadan tutunsada aynı atriyum içerisinde multifokal tutulum gösteren ve biatriyal tuturulm gösteren miksomalarda azınlıkta olmakla birlikte bildirilmiştir. Multifokal tutulum çoğunlukla ailesel ve sendromik olanlardır. (Irani et al. 2008) Ventriküler miksomalar kadın ve çocuklarda daha fazla görülür ve mutlifokal eğilimlerdi daha fazladır, daha çok sağ ventrikülde bulunurlar. (McAllister HA 1978) Çoğu serbest duvar ya da interventriküler septumdan kaynaklanır. Sol ventrikül miksomaları nadirdir ve genellikle posterior papiller bölgeden kaynaklanır.

3-KLİNİK VE PATOFİZYOLOJİ

Kardiyak miksomaların klinik bulguları net olmaduğu için tanıdan miksomadan şüphelenilmesi önemlidir. Miksomalar asemptomatik olduğu gibi 3 ana klinik gruba ayrılabilir. Sistemik bir hastalığı andıran konstitüsyonel bulgular, embolik bulgular, mital ya da triküspit kapakların tıkanmasını taklit eden bulgular.(Silverman 1980) Miksomalı hastaların büyük çoğunluğunda kilo kaybı, ateş, yorgunluk, anemi (hemolitik), sedimantasyon yüksekliği ve immunglobulin yüksekliği gibi sistemik belirtilere rastlanır. Serum immunglobulin artışının, tümör dejenreasyonun ve tümör tarafından üretilen aşırı interlökin 6'ya yanıt olarak ortaya çıkabilir. Daha az olarak saptanan bulgular ise polisitemi, lökositoz, trombositopeni, çomak parmak, artralji, Raynaud fenomeni, çomak parmak ve meme fibroadenomlarıdır. (Endo et al. 2002) Miksomalı hastaların yaklaşık üçte birinde sistemik emboli görülür. Emboli durumu tümörün frajil olması ve kalp boşluklarında yer olmasından dolayı sıktır. Emboli sıklıkla ekstremite arterlerine, serebral dolaşıma, renal arterlere ve koroner arterlere olur. Nadiren sol atriyumdaki dev miksoma abdominal aorta bifurkasyon total oklüzyonuna neden olabilir. (McAllister HA 1978)(Buch and Soni 2025) Özellikle genç sinüs ritmindeki hastalarda, enfektif endokardit yokluğunda, tekrarlayan embolilerde kardiyak miksoma akla gelmelidir. Sağ atriyal miksomalar pulmoner emboliye neden olabilir ayrıca trombolitik tedaviye rağmen tamamen düzelmez ve pulmoner hipertanisyona neden olabilir. Hastada atrial septal defekt varlığında paradoks emboliler görülebilir. Sol atiryumda yerleşen miksomalar pulmoner venlerde oklüzyona, pulmoner hipertansiyona ve sağ kalp yetmezliğine neden olabilir. Semptomlar arasında hemoptizi, öküsürük, efor dispnesi, ortopne, göğüs ağrısı, çarpıntı ve perifeik ödem görülebilir. Özellikle yatınca dipnenin azalması gibi hastanın pozisyonundaki değişikliğinin semptomun şiddetinde belirgin etki yapması miksomayı akla getirir. (A Rare Cause of Dyspnea: Left Atrial Myxoma Mimicking Pulmonary Embolism n.d.) Sağ atriyum miksomaları genelde daha büyüktür ve triküspit kapak darlığını taklit edebilir ya da kapak dejenerasyonuna bağlı olarak triküspit kapak yetmezliğine neden olabilir. (Guhathakurta and Riordan 2000)

4- SEMPTOM VE BULGULAR

Sol atriyal miksomalar sıklıkla mitral darlığı taklit ederler. En sık çarpıntı ve dispne görülür. Semptomlar genelde kısa sürelidir. Sağ atriyal miksomalar sağ kalp yetmezliğini taklit edebilir. Perfierik ödem, hepatomegali, abdominal distansiyon görülebilir. Sistemik embolilerde periferlerde soğukluk, nörolojik defisit, koroner anjina, pulmoner emboliye bağlı dispne görülebilir. Hsataların çoğunda kardiyak oskültasyonda apikal diyastolik veya sistolik üfürüm ya da her ikisi birden duyulabilir. Sağ kalp yetmezliği ya da pulmoner hipertansiyon görülen hastalarda triküspit yetmezliği üfürümü duyulabilir.

5-TANI

Noninvazin yöntemlerden transtorasik ekokardiyografi kardiyak tümör tanısında yüksek sensitivite ve spesifiteye sahiptir. Ekokardiyografide, kardiyak boşluklar, kapak yapıları, vena kavalar ayrıntılı bir şekilde değerlendilebilir. Miksomanın tutunduğu bölge, etkilenen kapak yapıları, ventrikül fonksiyonlarına etkisi gösterilebilir. Neredeyse %100 yakın tanı sadece trans torasik ekokardiyografi ile konulabilir. Transözefageal eko (TEE) miksomaların morfolojisini değerlendirmede transtorasik ekoya göre daha üstündür. (Munirathinam, Kumar, and Singh 2022) Miksoma tanısında şüphe varsa kardiyak bilgisayarlı tomografi (BT) ya da manyetik rezonans görüntüleme (MRG) tümör invazyonu, boyutu, yüzey dokusu hakkında bilgi verebilir. BT ve MRG sıklıkla kalbin malign tümörlerinin tanısında kullanılır. Kardiyak kateterizasyon miksoma tanısından çok anjinayı araştırmak için yapılır. Elektrokardiyografi (EKG) taşikardi görülebilir, çoğunlukla sinüs ritmindedir. Telekardiyografide kardiyomegali, sol atriyal büyüme, pulmoner konjesyon görülebilir ancak hiç birisi miksoma için spesifik değildir.

6-TEDAVİ

Miksomaların tedavisinde şu anda kabul görmüş en etkili yöntem cerrahi olarak eksize edilmesidir. Cerrahi yapılırken temel yaklaşım mediyan sternetomidir. Alternatif olarak sağ anterolateral torakotomi ile de yaklaşım yapılabilir. Kardiyopulmoner bypass altında kalp boşluğu açılarak direk görüş ile cerrahisi yapılır. Cerrahi sırasında embolik olaylara neden olmamak için aort klemp konulana kalbe majör manipülasyonlardan kaçınılmalıdır. Sağ atriyal miksomaya yaklaşım için sağ atriyotomi yapılır, sol atriyal miksoma için sol atriyotomi ya da sağ atriyum içerisinden transeptal olarak ulaşılabilir. Ventrikül içerisindeki miksomalar için sağ ya da sol atriyotomi ile ulaşım sağlanmaya çalışılır, eğer yeterli olmazsa ventrikilotomi yapılabilir. (Kamiya et al. 2001)(Guhathakurta and Riordan 2000)(Sánchez Sotelo et al. 2023)

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BÖLÜM 4

Postoperative Bleeding and Cardiac Tamponade in Cardiovascular Surgery

Mümtaz Murat Yardımcı[1] & Cengiz Güven[2]

1.Giriş

Postoperative bleeding, although reported with a revision rate of 1–5%, remains one of the most significant complications associated with open-heart surgery. Cardiopulmonary bypass (CPB) itself and systemic heparinization expose each patient to varying degrees of bleeding risk. Ensuring normal hemostatic mechanisms preoperatively, managing the effects of anticoagulants, and diagnosing and treating perioperative hemostatic disorders are essential for successful surgical outcomes (1).

2. Preoperative Evaluation

The most crucial step in identifying patients with coagulation defects is taking a detailed history. Patients with mild coagulation disorders often present with normal routine screening tests. Most bleeding disorders are acquired and usually result from primary liver disease, uremia, or the use of medications that interfere with hemostasis. However, congenital bleeding disorders, though rarer, can also be present.

Key preoperative questions to assess bleeding risk include:

a. Presence of abnormal bleeding tendency or bruising

b. Whether the bleeding is generalized or localized

c. Whether it occurs spontaneously

d. Association with trauma

e. Severity of trauma or surgical procedure

f. History of epistaxis or prior surgeries

g. History of transfusions

h. Current medications

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Physical examination is generally less helpful in preoperative assessment of bleeding disorders, but findings such as petechiae, ecchymosis, and spider angiomas warrant further investigation (1,2).

3. Laboratory Screening Tests

Initial evaluation typically includes prothrombin time (PT), activated partial thromboplastin time (aPTT), platelet count (especially in patients with a history of bleeding or recent antiplatelet use), and bleeding time.

*Prothrombin Time (PT): Evaluates abnormalities in the extrinsic pathway. Prolongation may indicate deficiencies in factor VII, factor V, prothrombin, or fibrinogen, or the presence of circulating anticoagulants like heparin.

*Partial Thromboplastin Time (PTT): Assesses the intrinsic pathway. Prolongation suggests deficiencies in factors XII, XI, IX, VIII, V, II, or I, or the presence of circulating anticoagulants.

*Activated Partial Thromboplastin Time (aPTT): Identifies functional deficiencies in the intrinsic and common pathways. It is prolonged in the presence of specific factor deficiencies or inhibitors, DIC, liver disease, massive transfusion, or heparin contamination.

*Thrombin Time (TT): Used when PT and aPTT are normal, but bleeding is suspected. Prolongation may be due to fibrinolysis, fibrin degradation products, or heparin.

*Platelet Count: Should exceed 100,000/mm^3. However, this does not assess platelet function.

*Bleeding Time: Prolonged (>6 minutes) in thrombocytopenia, aspirin use, or functional platelet defects.

*The combination of history, PT, aPTT, and platelet count provides effective screening for postoperative bleeding risk (4,5).

4. **Hereditary Bleeding Disorders**

Hereditary coagulation disorders usually result from deficiency of a specific factor. Classic hemophilia A and B result from deficiencies in factors VIII and IX, respectively—both are X-linked recessive disorders. Mild forms may remain asymptomatic except during major hemostatic challenges such as surgery. These syndromes typically show prolonged aPTT, with normal PT and platelet function.

Von Willebrand Disease (vWD) is the most common hereditary bleeding disorder, characterized by easy bruising, mucosal bleeding, prolonged bleeding time, and abnormal ristocetin-induced platelet aggregation. aPTT may be prolonged. Diagnosis allows for safe surgery with appropriate factor replacement.

Factor VIII concentrates are used for hemophilia A, and factor IX or prothrombin complex for hemophilia B. In emergencies, fresh frozen plasma or cryoprecipitate can be used. Rarely, acquired hemophilia may develop due to antibodies against factor VIII in non-hemophilic individuals (5,6).

5.Acquired bleeding disorders

5.1. ***Platelet Dysfunction from Diseased Heart Valves or Assist Devices***

Turbulent flow across diseased valves or intra-aortic balloon pumps chronically activates and damages platelets, leading to both depletion and dysfunction. Use of antiplatelet agents may worsen this dysfunction. Bleeding time is an important screening test in this group. If thrombocytopenia (<100,000/μL) is present, it may be due to consumption. Platelet transfusions provide only temporary benefit until the source of activation is corrected. Optimal timing is after cardiopulmonary bypass has been terminated (7,8).

5.2. **Congenital Cyanotic Heart Disease**

Children with congenital heart defects often exhibit bleeding disorders. Platelet aggregation may be impaired. Thrombocytopenia, shortened platelet lifespan, and dysfunction are associated with arterial hypoxemia and hemoconcentration. Severe hypoxemia may impair hepatic synthesis of clotting factors. Compensatory hemoconcentration leads to relative plasma deficiency and impaired clot formation. Recent studies suggest decreased von Willebrand factor receptors on platelets in cyanotic patients. Preoperative phlebotomy and hemodilution to a hematocrit of 50–60% may enhance both platelet count and function (9).

5.3. **Drug-Induced Hemostatic Defects**

Medications are the most frequent cause of impaired hemostasis before cardiac surgery. While cessation of all hemostasis-affecting drugs is rarely feasible, many cause only mild, subclinical effects. Therapy may need to continue until the time of surgery due to necessity or urgency (10).

6.**Anticoagulants**

If therapeutic anticoagulation cannot be withheld before surgery, several strategies are available. Warfarin may be discontinued 1–2 days prior, allowing some residual anticoagulant effect. Vitamin K or fresh frozen plasma (FFP) may be administered intraoperatively. Heparin and protamine are routinely used during CPB. In emergencies, IV heparin infusions may continue preoperatively but require careful intraoperative monitoring due to variability in heparin response (11).

7. Antiplatelet Agents

Aspirin should ideally be discontinued 5–7 days preoperatively, as its use correlates with increased postoperative bleeding. If platelet function is impaired despite a normal platelet count, bleeding time testing is useful. In the absence of circulating aspirin, prolonged bleeding time may be corrected with 8–12 units of fresh platelet suspension. If bleeding time is normal despite aspirin use, surgery may proceed safely. However, prolonged bleeding time may necessitate postoperative platelet transfusion. Some cardiac and antibiotic medications may induce thrombocytopenia. In idiosyncratic reactions (e.g., quinidine), stopping the drug often restores normal platelet counts. Heparin-induced thrombocytopenia (HIT), a more severe immune-mediated reaction, can occur in ~5% of patients with repeated heparin exposure (11).

8. Fibrinolytic Agents

Tissue plasminogen activator (tPA), urokinase, and streptokinase promote endogenous fibrinolytic enzyme plasmin formation. Excessive fibrinolysis reduces plasma fibrinogen levels below safe thresholds (<100 mg/dL), resulting in fibrin(ogen) degradation products that interfere with platelet function and the coagulation cascade. This hemorrhagic state may be exacerbated by concurrent heparin administration. The effects of fibrinolytic agents are short-lived; systemic fibrinolytic activity and its secondary effects decline within hours after drug discontinuation. In cases of severe bleeding accompanied by low plasma fibrinogen, heparin should be discontinued and cryoprecipitate administered. Antagonists such as protamine and epsilon-aminocaproic acid may be used but carry a risk of thrombosis (12).

9.Renal and Hepatic Failure and Disseminated Intravascular Coagulation (DIC)

Bleeding tendency in uremia primarily results from platelet dysfunction and is associated with anemia due to renal failure and accumulation of plasma factors inadequately cleared by the kidneys. Interaction between platelets and von Willebrand factor, as well as platelet mechanisms, are impaired. The problem originates from uremic plasma, rendering platelet transfusions ineffective. Dialysis and correction of anemia are necessary preoperatively. In uremic patients, cryoprecipitate to increase plasma levels of von Willebrand factor or desmopressin acetate (DDAVP) may improve hemostasis.

Major hepatic failure presents with complex factor deficiencies and DIC. Synthesis of clotting factors, particularly vitamin K-dependent factors II, VII, IX, and X, is impaired. Fibrinolytic products increase due to decreased clearance, accompanied by consumption-related decreases in fibrinogen and platelets. If PT is prolonged, parenteral vitamin K (10 mg) may be administered preoperatively.

If PT does not normalize or aPTT/platelet counts remain abnormal, further detailed investigations (including evaluation for DIC and pathological fibrinolysis) are warranted. FFP and platelet transfusions may be required to normalize coagulation parameters. DIC may develop in patients with septic endocarditis, mediastinitis, or malignancy, characterized by elevated fibrin degradation products, thrombocytopenia, and prolonged PT and aPTT. Treatment focuses on eliminating the stimulus provoking coagulation and may include FFP and platelet administration according to laboratory results. Antithrombin III concentrates may be effective. Aortic aneurysms or chronic/acute dissections can trigger consumption coagulopathy (13–15).

10. Evaluation of Postoperative Bleeding and Stabilization in Intensive Care

Currently, fewer than 3% of cardiac surgery patients require re-exploration, mostly for bleeding or tamponade. Uncomplicated cardiac surgery patients generally require 1–3 units of packed red blood cells, whereas more complex procedures such as valve replacements, endocardial resections, or redo surgeries may require 3–5 units or more. Acceptable blood loss in the first 24 hours postoperatively is generally 800–1200 mL. Early postoperative bleeding exceeding 100 mL/hour sustained over several hours is considered significant. Non-surgical bleeding causes post-protamine administration often include inadequate heparin neutralization, preoperative use of heparin, aspirin, NSAIDs, or thrombotic agents, and prior sternotomy. Postoperative bleeding is closely related to blood pressure control, metabolic stability, maintenance of normothermia, effects of surgery and cardiopulmonary bypass, and meticulous medical care. These factors are common to all patients and must be managed upon ICU admission (16).

Surgical causes of bleeding are frequently identified upon reoperation, but hemostatic defects remain a significant problem in cardiac surgery patients. Distinguishing between purely mechanical and hematologic bleeding sources is often challenging. Excessive bleeding from arteriotomy closure sites or prolonged bleeding from skin and subcutaneous tissues despite heparin neutralization suggests abnormal hemostasis. In such cases, intraoperative platelet count, PT, and aPTT should be checked. Upon ICU admission, these tests should be repeated from a new venous access. They serve as screening for gross coagulation pathway abnormalities or thrombocytopenia. Mild PT or aPTT elevations are common immediately after bypass, with individual coagulation factor levels usually exceeding requirements for hemostasis. Minor abnormalities without active bleeding can be monitored as they typically resolve within 4–6 hours post-bypass (16).

Significant prolongations of coagulation times usually stem from circulating anticoagulants such as heparin or fibrin degradation products. Initial treatment includes normalizing activated clotting time (ACT) with protamine sulfate (25–50 mg IV) and increasing positive end-expiratory pressure (PEEP) up to 10 cm H_2O, provided cardiac output and systolic blood pressure are unaffected. Rarely, congenital coagulation defects or consumption-related factor deficiencies may underlie abnormal bleeding. If major abnormalities are present or bleeding is excessive, further testing is mandatory, focusing on:

*Excess heparin

*Integrity of the coagulation cascade

*Platelet function

Desmopressin use has been recently reported in uremic and secondary platelet dysfunction patients, as well as in cardiac patients undergoing reoperation. However, its routine use in cardiac surgery remains unclear. Desmopressin acetate, a vasopressin analogue, temporarily increases plasma levels of factor VIII and von Willebrand factor by promoting release from endogenous stores. Studies in high-risk bleeding patients show significant blood loss reduction, though results lack confirmation in larger and low-risk groups. Desmopressin is likely beneficial in patients with significant von Willebrand factor-dependent hemostatic defects and is not routinely indicated in low-risk patients (17).

Aprotinin is used prophylactically in patients undergoing reoperation to reduce perioperative bleeding and transfusions after cardiopulmonary bypass, particularly in high-risk or transfusion-limited cases. Derived from bovine lung, aprotinin is a natural protease inhibitor. Although its mechanism is not fully understood, it reduces bleeding after cardiac surgery by inhibiting kallikrein, thus preventing contact activation of the coagulation cascade. Additionally, aprotinin inhibits plasminogen conversion to plasmin (antifibrinolytic effect) and platelet adhesion and aggregation, mitigating hemostatic defects associated with CPB. Its use reduces perioperative blood loss, chest tube drainage, and transfusion requirements. However, it may cause anaphylaxis and nephrotoxicity. Its prothrombotic potential and impact on perioperative myocardial infarction risk remain debated. Aprotinin markedly elevates ACT, especially when measured by the Hemochron system, which may mask inadequate heparin anticoagulation. The role of PEEP in reducing postoperative bleeding is controversial but is applied at baseline (5 cm H_2O) to enhance oxygenation in all patients. Factor concentrates are rarely preferred due to high hepatitis risk (18,19).

If PT or aPTT is prolonged, 2–4 units of fresh frozen plasma (FFP) should be administered. Platelet transfusion is indicated if platelet count falls below 100,000/mm^3 in patients with ongoing bleeding. Platelets are dosed

approximately 1 unit per 10 kg body weight. When platelet count, PT, and aPTT are all abnormal, platelets are transfused first, followed by FFP. Post-CPB platelet dysfunction may warrant platelet transfusion even if platelet counts appear normal, as platelets are an initial coagulation product. Persistent bleeding despite initial products requires repeating coagulation tests, bleeding time, fibrinogen, and fibrin degradation product measurements (20,21).

If fibrinogen levels fall below 100 mg/dL, cryoprecipitate (1 unit per 10 kg) should be administered. Rarely, fibrinolysis with low fibrinogen and increased fibrin degradation products may occur. In the presence of significant fibrinolysis—characterized by elevated fibrin degradation products, low fibrinogen, and reduced clot lysis time—epsilon-aminocaproic acid is indicated. This agent competitively inhibits plasminogen activation. If bleeding is noted at suture lines or prosthetic graft sites following initial clot formation, fibrinolysis should be suspected. Initial dosing is 25 mg/kg IV over 1 hour, followed by 1–2 g/hour for 4.5 hours. Though indications are rare, it may be beneficial when standard treatments fail (22).

11. Indications for Mediastinal Re-exploration

*Bleeding >200 mL/hour for 4–6 hours

*Total bleeding >1500 mL within 12 hours

*Sudden increase in chest tube drainage

*Signs of pericardial tamponade

Active heparin presence in early postoperative blood commonly causes ACT or aPTT prolongation. Alternatively, high concentrations of FFP (due to excessive fibrinolysis or autotransfusion) may act as anticoagulants. Administering 25–50 mg additional protamine over minutes is a simple clinical approach; if heparin excess is the cause, ACT and aPTT will normalize. Protamine and thrombin time testing may further clarify the problem. Heparin prolongs thrombin time, which improves with small amounts of protamine in vitro. Conversely, thrombin time prolongation due to FFP does not correct with protamine. Residual heparin effects may persist despite FFP (21).

If thrombocytopenia and bleeding coexist, 8–12 units of fresh platelet suspension may be given. Sometimes, patients display generalized hemorrhagic tendencies without abnormal coagulation tests or thrombocytopenia, often due to significant platelet function defects. This constitutes an indication for desmopressin infusion (0.3 mcg/kg IV). Cardiopulmonary bypass invariably causes some degree of platelet dysfunction. Bleeding time is unreliable in active bleeding due to peripheral vasoconstriction and hypoperfusion. Platelets should be transfused even if platelet counts do not meet standard thresholds because

thrombopathy often coexists. Routine prophylactic platelet transfusion is not indicated as it does not reduce transfusion requirements (23).

Excessive fibrinolysis ranges from mild anticoagulation to DIC with thrombocytopenia and hypofibrinogenemia. Inadequate suppression of clotting during extracorporeal circulation, sepsis, transfusion reactions, and large volumes of recirculated blood can cause laboratory abnormalities. Intraoperatively, this syndrome is identifiable when clots form but lyse immediately. Postoperatively, severe pathological fibrinolysis manifests as generalized bleeding and deranged laboratory parameters. Treatment should commence without waiting for laboratory results and includes antifibrinolytics such as epsilon-aminocaproic acid and aprotinin, along with transfusions of FFP, platelets, and red cells to compensate hemorrhagic losses. Cryoprecipitate contains supra-normal concentrations of fibrinogen, von Willebrand factor, and factor VIII and is preferred over FFP only in cases of severe hypofibrinogenemia (<100 mg/dL) with bleeding (20,24,25).

Massive transfusion complicated by bleeding problems is rare. Transfusion volumes exceeding 1 to 1.5 times blood volume cause significant plasma protein dilution. Platelet deficiency is the most common abnormality as banked blood contains low platelet counts. Factors V and VIII are labile in stored blood but deficiency is less common due to extravascular stores and synthesis. These patients typically have prolonged bypass times, high volumes of recirculated blood, and preexisting acquired coagulation defects (24,25).

Hemodynamic stability and blood loss volume determine the need for reoperation. If clinical status mandates re-exploration, the patient should return to the operating room without delay for coagulation test results. Emergency cardiac surgery patients may be receiving anticoagulants, fibrinolytic agents, or platelet inhibitors. Cardiac decompensation may coexist with hepatic or renal dysfunction or mechanical cardiac assist devices, where pronounced platelet dysfunction and drug effects are expected. Redo surgery and prolonged pump times confer higher postoperative bleeding risk. Massive transfusion after major bleeding further complicates the clinical picture and impedes determination of bleeding etiology (26).

12.Cardiac Tamponade

To prevent tamponade, many surgeons create a wide opening in the pericardium to ensure adequate drainage of the mediastinum and place a tube at the portion of the pericardial space adjacent to the left ventricle. However, these measures do not reliably prevent pericardial tamponade. Cardiac tamponade is a life-threatening condition. Although opening a pleural space related to the pericardium may be beneficial, it is not a definitive method to prevent tamponade.

Tamponade can occur whether the pericardium is open or closed. Even a strategically located small thrombus on the posterior pericardium affecting the left atrium or pulmonary veins can cause tamponade symptoms. A sudden decrease in chest tube drainage should raise suspicion of tamponade. This complication should especially be considered in patients undergoing open heart surgery who develop low cardiac output early postoperatively. Late tamponade may develop days or weeks after surgery. When arterial pressure tracings are monitored in the surgical ICU, a respiratory-synchronous oscillation in systolic pressure can be observed. All intrathoracic organs are subjected to pressure changes related to respiration/ventilation. Pressure gradients develop at the entry and exit points of veins or arteries into the thorax. During spontaneous inspiration, negative intrathoracic pressure directs venous return into the thorax, increasing right atrial and right ventricular filling. This negative intrathoracic pressure is transmitted to the left ventricle, enhancing the ventricular force generated to maintain systemic arterial pressure. Both systolic pressure and pulse pressure decrease (27).

Right ventricular distension shifts the interventricular septum toward the left, impairing left ventricular diastolic filling. Decreased left ventricular preload results in reduced stroke volume and consequently lowers systolic and pulse pressures. These two mechanisms reduce blood pressure during negative-pressure inhalation, and this phenomenon is exaggerated in the presence of increased pericardial pressure (tamponade). During cardiac tamponade, inspiratory right ventricular filling increases while the interventricular septum shifts leftward, diminishing septal curvature. Inspiration significantly reduces left ventricular end-diastolic volume. Increased right ventricular size occurs at the expense of left ventricular filling. The reduction in left ventricular volume correlates with pulsus paradoxus and decreased cardiac output. Elevated pericardial pressure lowers systolic blood pressure.

Pericardial effusion refers to an accumulation of pericardial fluid that does not cause hemodynamic changes. Tamponade is the accumulation of fluid that decreases left ventricular filling and cardiac output and leads to pulsus paradoxus. This distinction is particularly important in surgical patients. There is no linear relationship between pericardial fluid volume and pericardial pressure. If the patient has enough time to increase intravascular volume and allow pericardial space expansion, even large effusions may cause only minor hemodynamic changes. However, when the elastic limits of the pericardial space are acutely exceeded, tamponade develops, obstructing forward flow (27).

Clinically, blood pressure is low, and pulse pressure is narrow. A drop in systolic pressure indicates severe tamponade and typically appears in advanced hemodynamic compromise. Filling pressures are elevated except in cases of

severe hypovolemia. Marked pulsus paradoxus may be present. However, diagnosis can be difficult due to low systemic blood pressure and signs of heart failure combined with elevated right heart pressures. Additionally, assessing pulsus paradoxus is challenging in mechanically ventilated patients. Portable chest X-rays may show cardiomegaly, and echocardiography reveals increased right ventricular diastolic pressures with inspiration and simultaneous reduction in left ventricular dimensions. Atrial and ventricular diastolic pressures equalize (27,28).

Treatment of pericardial tamponade includes maintaining blood pressure with volume resuscitation and inotropes, and if hemodynamics are severely compromised, urgent thoracotomy in the ICU or operating room. If immediate transfer to the OR is not possible, the lower end of the sternotomy incision can be opened in the ICU to drain blood from the substernal space. Hemodynamic improvement occurs rapidly. Exploration of the pericardial space in the ICU to control bleeding prevents tamponade and fatal outcomes (27).

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BÖLÜM 5

Aortic Dissections in Cardiovascular Surgery

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Introduction

Aortic dissection is a catastrophic event characterized by the accumulation of blood between the intima and media layers due to a tear in the media of the aorta (Bossone and Eagle, 2021). This event is not associated with the presence of an aneurysm. Therefore, the term 'acute aortic dissection' is more appropriate than 'dissecting aortic aneurysm.' Following an acute event, re-entry tears can form with progressive aneurysmal dilation of the false lumen, resulting in a double-barreled aorta (Clothier and Kobsa, 2025).

The incidence of acute aortic dissection averages 5.2 per million, occurring twice as frequently as ruptured abdominal aortic aneurysm. While it can occur in all age groups, it is rare in young and very elderly individuals. Seventy-five percent of cases are concentrated between the 4th and 7th decades, with peak incidence between 50-60 years of age. The male-to-female ratio is 2:3 (Isselbacher et al., 2022).

Etiology

While not entirely clear, several predisposing conditions exist. The most important factor is arterial hypertension. The reported incidence of hypertension in acute dissections is 75%, and it is more common in cases of Type B dissection. Hypertension is thought to accelerate degenerative changes in the aging aorta, creating a suitable environment for dissection. Although a clear relationship between hypertension and the onset of the dissection process is not established, hypertension facilitates the progression of the event (Mutailifu et al., 2024; Guo et al., 2011).

The role of heredity in aortic dissections in the general population is unclear, except for those with connective tissue disorders such as Ehlers-Danlos Syndrome or Marfan Syndrome. Familial dissection and cases of annuloaortic ectasia linked to a mutation in the type 3 procollagen gene have been reported in the literature. Marfan Syndrome exhibits autosomal dominant inheritance. At one end of the clinical spectrum is the typical presentation characterized by abnormalities of the skin, skeletal, ocular, and cardiovascular systems, while in some cases, only cardiovascular involvement is observed. Genetic studies have

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identified a missense mutation in the fibrillin gene on chromosome 15 (Mah et al., 2018).

The most common cardiovascular manifestation of the syndrome is the formation of an ascending aortic aneurysm, which may occasionally be accompanied by aortic insufficiency. The primary cause of death in Marfan Syndrome is complications related to aortic involvement. Approximately 33% of patients with Marfan Syndrome develop aortic dissection. The incidence of Marfan Syndrome in patients who develop acute aortic dissection ranges from 4-12%.

Especially in young patients, congenital aortic stenosis, aortic coarctation, and bicuspid aortic valve are associated with a high risk of dissection. The risk of dissection is nine times higher in individuals with bicuspid aortic valve. The incidence of bicuspid aortic valve in cases of dissection ranges from 7-13%. Bicuspid valve-related dissections typically originate in the ascending aorta. There is a perioperative risk of dissection in cases undergoing aortic valve replacement due to a bicuspid aortic valve. The perioperative risk of dissection in patients who have undergone aortic valve replacement has been found to be 0.07%. The association of dissection with other congenital heart diseases is rare (Wojnarski et al.,2015).

Cardiac catheterization, coronary angiography, and surgical procedures such as cannulation during cardiopulmonary bypass (CPB) (especially femoral cannulation) and cross-clamp injury can increase the risk of dissection (Elefteriades et al., 2016).

The primary cause of acute dissection during pregnancy is hemodynamic stress. Approximately 50% of acute dissections in women under 40 years of age occur during pregnancy. The majority of these cases involve congenital malformations of the aorta or connective tissue disease (Braverman et al., 2021).

Localized dissections involving the descending aorta may be associated with intimal disruption in an atherosclerotic plaque region. Dissection associated with atherosclerotic changes in the ascending aorta is rare. Additionally, aortic dissection may uncommonly be seen in patients with syphilitic aortitis, endocarditis, mycotic infections of the aorta, giant cell aortitis, polyarteritis nodosa, and SLE, in Cushing Syndrome, pheochromocytoma, and following thyroidectomy. Other rare associations include familial hypercholesterolemia, cystinuria, and Turner Syndrome (Mutailifu et al., 2024).

Anatomical and Clinical Classification

Clinically, dissections detected within the first two weeks from symptom onset are considered acute, and those detected after this period are considered

chronic. Mortality during the critical initial two-week period ranges from 57-89%. Acute and chronic dissections differ in their pathology and physiological bases, as well as in surgical treatment, response to therapy, and prognosis. DeBakey's original dissection classification has been modified to three main types (Ramesh et al., 2024; Rosenberger et al., 2012):

- **Type 1:** Dissection begins in the ascending aorta and involves the entire aorta.
- **Type 2:** Dissection is localized to the ascending aorta.
- **Type 3:** Dissection originates distal to the subclavian artery but does not involve the aortic arch or ascending aorta. DeBakey Type 2 dissection is typically seen in chronic dissections with aneurysmal dilation of the ascending aorta (Rosenberger et al., 2012).

The simpler Stanford classification considers localizations that create differences in treatment approach (Rosenberger et al., 2012):

- **Stanford Type A dissections (proximal dissections):** Include all dissections involving the ascending aorta, regardless of the location of the initiating intimal tear.
- **Stanford Type B dissections (distal dissections):** Refer to dissections that do not involve the ascending aorta and originate distal to the left subclavian artery, extending along the aorta. These two types have different clinical characteristics and treatment methods.

Pathology

The characteristic pathology of acute dissections is the progressive separation of the intima-media layers by a column of blood. The connection between the true lumen of the aorta and the separated 'false lumen' of the media layer is provided by a 'primary or entry intimal tear.' The intimal tear is a prominent feature of acute dissection. However, it is debated whether the intimal tear is the primary event leading to layer separation in all dissections, or a secondary event to initial layer separation. Only 2-4% of dissections develop without an intimal tear, and most of these are confined to the ascending aorta. In the majority of dissections that develop without an intimal tear, the lumen is completely thrombosed (Sherk et al., 2021; Sorber et al., 2022). This condition is known as 'aortic intramural hematoma.' With the routine use of TEE and CT, these cases have become more frequently identified. It is difficult to determine whether this pathology is a separate condition from dissection. There is evidence that intramural hematoma, reflecting an early stage of typical dissection, leads to the formation of an intimal tear in the hematoma-filled false lumen. These observations support an older theory of pathogenesis, hypothesizing that rupture

of the vasa vasorum and hematoma in the media layer are the triggering events in the dissection process. Intimal rupture then progresses along the aorta at points of highest hydraulic stress, typically along fixation points. These points correspond to the regions where intimal tears are observed in the sinotubular junction and aortic isthmus. The vast majority of intimal tears are localized in the first few centimeters of the ascending aorta. The second most common localization is the initial portion of the descending aorta. Only 10-15% of intimal tears are located in the aortic arch. The prognosis for arch tears is poor, and their surgical importance is therefore significant. A very small proportion of primary tears are distal to the thoracic aorta. A primary intimal tear is generally transverse and usually does not exceed half the diameter of the aorta. Rarely, a circumferential intimal tear with complete loss of intimal continuity, known as 'intimointimal intussusception,' may be observed. Once initiated, the dissection process rapidly progresses along the length of the aorta, usually through the outer one-third of the media. The dynamic forces driving the progression of dissection have been studied in detail. Two key factors identified by Wheat and Palmer are ejection velocity and the rate of myocardial fiber shortening (dp/dt). The aim of medical treatment in dissection is to reduce these forces. Longitudinal dissection and the false lumen involve the right anterior part of the ascending aorta, and the medial one-third of the ascending aorta is generally intact. In the arch, the false lumen typically extends along the greater curvature and often spreads into the innominate, left carotid, and left subclavian arteries. In the descending aorta, the false lumen follows the anterolateral wall of the aorta and continues along the anterolateral wall as it extends into the abdomen, often involving the left renal artery. There are several important pathological and clinical points in the progression of the dissecting process within the false lumen. Dissection can interrupt blood flow to the major branches of the aorta either by external compression or by causing the branches to separate from the true lumen. Both mechanisms ultimately lead to a condition known as hypoperfusion syndrome, resulting in distal organ or extremity ischemia and associated functional impairment. Proximal extension of the dissection to the aortic root results in separation of the aortic commissures, leaflet prolapse, and acute aortic insufficiency. Proximal dissection can also lead to dissection of the coronary arteries, especially the right coronary artery, and myocardial ischemia or infarction. Due to high-pressure formation, the false lumen can frequently rupture into the pericardial or pleural spaces from regions near the primary intimal tear. Rupture into the pericardium is the most frequent cause of death within the first two weeks. Rupture of the thinner outer wall of the dissection sac into the pleural or pericardial spaces is more likely than a re-entry tear or internal rupture that would occur in the opposite direction of flow towards the true lumen. Re-entry tears most commonly occur in the inner tubular portion, in areas where side

branches have been pruned, particularly along the intercostal arteries in the descending aorta. The endpoint of the dissecting process in the aorta is determined by scarring in the media, typically of atherosclerotic origin. A natural barrier, such as coarctation, can also limit dissection. In surviving patients, after an acute episode, the false lumen either thromboses and heals, or remains patent and continues to expand, forming an aneurysmal dilation. The presence of a re-entry tear prevents thrombosis of the false lumen. In the chronic phase, persistence of the false lumen indicates a poor prognosis and is associated with a high risk of aneurysmal dilation. The false lumen then organizes into fibrous tissue, a large part of which is collagen. Over time, the wall of the false lumen becomes thicker than the aortic wall, but due to the structural weakness of the fibrous tissue, it continues to expand or rupture (Verma et al., 2023; Shen et al., 2020; Chung, 2023).

Histology

The histopathological basis of aortic dissection involves changes best described by the term "medial degeneration," referred to as "cystic medial necrosis," which in fact contains no necrosis or cysts (17). These degenerative changes include fragmentation of elastic fibers, loss of smooth muscle cells, and an increase in connective tissue and collagen. This condition leads to structural weakness of the aortic media. Especially in dissections in young patients or those associated with conditions like Marfan Syndrome, significant loss of elastic tissue and accumulation of connective tissue are more common (Akutsu, 2024).

Clinical

Common clinical findings are present in all aortic dissections. In cases of sudden death, diagnosis can only be made post-mortem. Pain is the most dramatic symptom in aortic dissections. Unless there is sensory loss due to neurological involvement, pain is present in all patients with dissection and is severe. It is typically described by the patient as 'tearing' and 'ripping' and is often accompanied by a fear of death. In most Type A dissections, the pain is substernal, while in distal dissections, it is more commonly located in the back. Pain is usually accompanied by pallor, coldness, sweating, nausea, and vomiting, reminiscent of a shock state despite hypertensive blood pressure. Except for conditions like Marfan Syndrome, dissection can rarely be painless and may go unnoticed until chronic aneurysmal dilation develops in the ascending aorta (Reed, 2024).

Syncope is the second most common initial symptom associated with dissection. It is usually transient and is due either to the intensity of the pain or to transient cerebral ischemia. Other neurological findings such as transient blindness, hemi/paraparesis or plegia, and even deep coma may be present.

Dyspnea and hemoptysis can be seen in acute dissections. These are more frequently observed in chronic dissections and indicate congestive heart failure symptoms due to aortic insufficiency or bronchial erosion due to the expanding false lumen (Morello et al., 2021).

Initial physical examination typically points to a patient in shock despite normal or elevated blood pressure. At presentation, blood pressure is normal or elevated in approximately 80% of patients. Severe, uncontrolled hypertension may be present. The severe hypertensive response in dissections can be related to renal ischemia, dysfunction of baroreceptor mechanisms in the aortic arch, or sometimes 'pseudo-coarctation' due to severe narrowing of the lumen. The presence of hypotension should suggest cardiac tamponade due to hemopericardium or hypovolemia due to rupture into the pleural space. Another cause of hypoperfusion can be myocardial ischemia and infarction due to impaired coronary circulation (Morello et al., 2021).

The most important clinical finding in acute dissections is pulse loss or difference between pulses, accompanied by varying degrees of extremity ischemia. It is present in approximately 40% of patients at presentation. The appearance of a murmur indicative of acute aortic insufficiency is an important finding for Type A dissections and is present in half of the patients. Depending on the degree of aortic valve insufficiency, congestive heart failure may be present in the acute phase. Other clinical findings include bilateral neck vein distension in tamponade, innominate vein occlusion or superior vena cava compression due to mediastinal hematoma, appearance of pulsatile neck masses due to dissection progression into the carotid arteries, and ecchymoses extending to the suprasternal area. Five percent of patients have a pericardial friction rub. Paralytic ileus may be observed. Flank pain accompanied by hematuria and oliguria indicates renal involvement. Hematemesis and melena can be present in relation to intestinal ischemia and are indicators of poor prognosis. Five to six percent of cases present with stroke, and the incidence of neurological findings is 42%. Localized neurological findings are the result of central infarction, extremity ischemia, or nerve compression due to hematoma (Reed, 2024; Morello et al., 2021).

Diagnosis

Diagnosis begins with suspicion of dissection. In many patients, the diagnosis can be inferred from the clinical picture and findings. Sudden onset of severe pain in a hypertensive patient, a newly emerged cardiac murmur, and pulse discrepancies should suggest dissection (Paneitz et al., 2022).

Laboratory findings in acute dissections are non-specific and largely serve to rule out other possible diagnoses. Commonly observed findings in aortic

dissections include hematuria and leukocytosis with a left shift in the blood count. Febrile reactions may accompany some cases. Elevated BUN and creatinine levels may be observed due to hypertensive renal disease or hypoperfusion of both kidneys. Anemia in the acute phase usually indicates blood loss into the left pleural space. Abnormalities seen in blood biochemistry are due to accompanying organ dysfunction. SGOT is generally normal and is useful in the differential diagnosis of MI and dissection. LDH elevation is routinely found and indicates excessive red blood cell hemolysis in the false lumen. Uncommonly, elevated amylase values may be found, usually indicating severe renal impairment or bowel ischemia. In acute dissections, changes in coagulation parameters such as prolonged aPTT and increased fibrin degradation products may occur. This may be accompanied by a decrease in platelet count. Rarely, a complete DIC picture may be observed. The underlying mechanism for these changes is the consumption of coagulation factors in the dissection hematoma (Paneitz et al., 2022).

The most common ECG finding is left ventricular hypertrophy and strain associated with pre-existing hypertension. The primary role of ECG is in differential diagnosis. MI-like findings may be present in 10-20% of patients. Consequently, an appearance suggesting concomitant MI does not rule out the possibility of dissection. However, the absence of such changes brings the diagnosis closer to dissection. Approximately 10% of patients show signs of pericardial involvement. Rarely, heart block may occur if the dissecting hematoma affects the interatrial and membranous septum (Paneitz et al., 2022).

Important points for the surgeon include:

a) Demonstration of ascending aortic involvement.

b) Localization of the intimal tear.

c) Demonstration of aortic root width and presence of aortic insufficiency.

d) Knowledge of the perfusion status of the main aortic branches.

e) Visualization of the presence of a re-entry tear.

f) Width and patency of the false lumen.

g) Level of coronary circulation.

Radiological findings detected on plain radiographs in acute dissections are related to distortion caused by the false lumen and fluid leakage into the pleural space. In over 80% of cases, there is mediastinal widening and obliteration or blurring of the aortic knob. Intimal calcification more than 6mm away from the

aortic contour is a very important finding favoring dissection. Another common and suspicious finding is the presence of left pleural effusion (Erbel et al.,2014).

Transthoracic echocardiography (TTE) has become the most important tool in diagnosis, intraoperative treatment planning, and postoperative follow-up. Its ease of performance at the bedside, coupled with its high sensitivity and specificity (99% and 98% sequentially), makes TTE advantageous as a diagnostic method in acute aortic dissections. Transesophageal echocardiography (TEE) can identify the presence and extent of the intimal flap, true and false lumens, primary and secondary intimal tears, periadventitial leaks, aortic insufficiency, aortic root dimensions, and coronary or brachiocephalic artery dissection, all of which are crucial in acute dissection. The observation of a mobile 'intimal flap' in the aortic lumen and detection of flow with Doppler on both sides (in true and false lumens) is specific. Two potential problems are associated with TEE. A hypertensive response can occur during this procedure, which is dangerous in acute dissection. Sedation and topical or general anesthesia may be required. The main technical limitation of TEE is the formation of an air interface due to the interposition of the trachea between the probe and the distal ascending aorta and aortic arch. However, the inability to obtain information from these limited areas rarely affects clinical approach (Kosmalska et al., 2021).

Using intravenous contrast infusion, computed tomography (CT) can accurately demonstrate the dimensions of the dissection, false lumen, intimal flap, and displacement within the true lumen. Dynamic CT allows for separate contrast flows in the true and false lumens and further definition of the intimal flap. CT is also useful in the postoperative follow-up of surgically treated patients. Magnetic resonance imaging (MRI) is useful in evaluating aortic pathology and provides clear information regarding dissection. The lack of need for contrast for imaging in MRI is an advantage. However, the difficulty of applying MRI restricts its use, especially in acute cases. Nevertheless, it is useful in stable patients, chronic dissections, and in the follow-up of treated patients (Kosmalska et al., 2021).

With the development of other non-invasive diagnostic methods, angiography has become secondary in the diagnosis of acute aortic dissections. However, aortography remains indispensable for determining the perfusion status of many aortic branches and for planning operative strategies in chronic dissections. The most specific angiographic finding is the demonstration of two separate lumens in the aorta. Due to flow differences, the clearance of contrast medium within the lumens and the degree of opacification of the lumens also differ. Both lumens cannot be filled with a single injection from a single point of the aorta. The status of the main branches of the aorta is related to the condition of the intimal flap at the time of injection. Determining the perfusion characteristics of the main

branches is very important, especially in chronic dissections where the false lumen persists and serves as the sole perfusion source for visceral branches. False-negative aortograms related to aortic intramural hematoma may occur. In such cases, TEE and CT are more effective in demonstrating intramural hematoma. The diagnostic and therapeutic value of coronary arteriography in acute dissections is low (Hallinan and Anil, 2014).

Treatment

Significant reductions in mortality have been achieved in the treatment of aortic dissections due to advancements in diagnostic tools, operative techniques, and pharmacological approaches.

Acute Dissections

Medical Treatment

Medical treatment is initiated as soon as dissection is suspected clinically and continues throughout the diagnostic workup. Medical treatment should focus on pain relief, blood pressure control, and halting the dissecting process. Initially, the patient should be monitored in the intensive care unit with arterial, central venous or pulmonary artery catheters, and a urinary catheter. Despite adequate blood pressure control, changes in CNS or spinal cord function, peripheral pulses, aortic valve sounds, or changes in the abdomen or extremities indicate progression of the dissection. The principles of medical treatment for acute dissections were established by Wheat in the 1960s, based on the premise that dissection progresses in experimental studies in relation to both mean arterial pressure and the rate of increase in arterial pulse (dp/dt). Anti-impulse therapy aims to reduce mean arterial pressure and myocardial contractility. Beta-blockade with propranolol (2-5 mg IV q 4-6 hours) should be performed to the extent tolerated. Labetalol has a combined selective alpha-1 adrenergic blockade and non-selective beta-blockade effect and reduces peripheral resistance without significantly affecting heart rate or cardiac output. It should be used in patients who cannot tolerate or do not respond to propranolol. Esmolol is a safe, intravenous, cardioselective beta-blocker and can be used in initial treatment. This drug's ultra-short half-life allows for immediate stable blood levels and its effect can be easily titrated (25-300 mcg/kg/min). Reduction of blood pressure to achieve adequate peripheral perfusion is provided by afterload-reducing agents, primarily sodium nitroprusside (0.5-5.0 mcg/kg/min IV). Since SNP alone will increase dp/dt, it should only be started after sufficient beta-blockade has been achieved. The second choice for blood pressure control is trimethaphan (0.2-6.0 mg/kg/min IV). One of trimethaphan's effects is the reduction of dp/dt, and it can be used without prior beta-blockade. Trimethaphan may be preferred in patients who cannot tolerate beta-blockers (Silaschi et al., 2017; Malaisrie et al., 2021).

Surgical Treatment:

Emergency surgery is indicated in acute Type A dissections and in cases where medical treatment fails, pain or hypertension cannot be controlled, organ hypoperfusion is observed with progressive dissection, or there is a high risk of rupture. The primary cause of early mortality, aortic rupture, occurs proximal to the primary intimal tear. In acute Type A dissections, it is in the ascending aorta, and in acute Type B dissections, it is proximal to the descending aorta. Surgical treatment aims to prevent proximal and distal progression of the dissection, excise the intimal tear, and replace the portion of the aorta closest to rupture (Murphy et al., 2020). In many cases, replacement of the aortic segment containing the intimal tear (ascending aorta in Type A and the initial portion of the descending aorta in Type B) achieves these goals. Ideally, resection of the affected aortic segment and the intimal tear redirects blood flow to the true lumen and, if no distal tear is present, converts the false lumen into a hematoma-filled cavity, allowing for complete healing of the dissection. Clinical studies show that resection of the primary tear area, including arch tears, reduces late complications related to dissection.

The most significant technical difficulty in the surgical treatment of acute dissections is inadequate hemostasis. The weakness of the dissected aortic wall and hypothermic perfusion further exacerbate hemostatic disorders associated with cardiopulmonary bypass. In the past, many primary repair techniques were developed to improve hemostasis between the graft and the aortic suture line. However, these repair techniques have unacceptably high failure rates. The graft's aortic suture lines can be reinforced with Teflon felt, fibrin glue, or gelatin-resorbed formalin biological glue (GRF). Routine open aortic anastomosis during hypothermic circulatory arrest for distal anastomosis and excision of the aortic segment traumatized by aortic cross-clamping are highly beneficial for hemostasis at the suture line. Inspection of the aortic arch is effective in identifying unpredictable arch tears and effectively eliminating primary tears in many cases. Many intimal tears in the aortic arch occur near the orifice of the innominate artery and allow for arch reconstruction with a single suture line. In some cases, due to the location of the arch tear or impairment of the arch, replacement of the entire arch with reimplantation of the arch vessels is required (Bachet et al., 1987; Kazui et al., 2001; Gott et al., 2001).

Type A Dissections

Type A dissections are approached via median sternotomy, and the femoral artery with the strongest pulsation is cannulated for right atrial-femoral bypass. If the artery is dissected, cannulation of the true lumen is crucial for safe perfusion. After initiating CPB, the aorta is dissected from the pulmonary artery, a bloodless field is obtained by ensuring venous return to the oxygenator via the

pump, and the aorta is clamped at the base of the innominate artery. The maximum risk for malperfusion occurs during the initial period of bypass, as circulation shifts from an antegrade to a retrograde (femoral artery) path. Continuous monitoring with TEE can determine flow characteristics in the descending aorta, thereby preventing unrecognized malperfusion complications. Systemic hypothermia (20 degrees Celsius) with hemodilution allows for low flow and low pressure, reducing the risk of aortic trauma during perfusion. The aorta is explored with a longitudinal aortotomy to identify the location of the intimal tear, and the coronary ostia are perfused with cold cardioplegic solution to provide myocardial protection until local hypothermia is achieved. The aortic valve is inspected for insufficiency, and if present, commissural dissection is corrected by resuspending the commissures. Then, the aorta is transversely opened just above its commissures and reinforced with Teflon felt to obliterate the proximal false lumen and create a suture ring for the proximal anastomosis. If the tissues are fragile, autologous pericardium is used on the intimal side in addition to Teflon strips on the adventitial surface. Alternatively, Teflon felt is placed between the dissected layers to close this space, and the aortic root is reconstructed. The graft is then anastomosed to the proximal aortic stump, leaving the graft inside the aorta and effectively sandwiching the dissected layers between the graft and the external Teflon felt. This creates a secure and hemostatic suture line. During the period of hypothermic circulatory arrest (16-18 degrees Celsius), the aortic cross-clamp is removed, and the distal aorta is trimmed from the base of the innominate artery, resecting the cross-clamp area and all proximal tear areas. A reinforced distal cuff is prepared, and the graft is anastomosed to obliterate the distal false lumen. To reinforce the suture line, 3cm of Teflon felt is sutured in a way that compresses the distal false lumen. Then, CPB is performed antegrade via cannulation from the ascending aortic replacement, thereby preventing the false lumen from being exposed to retrograde pressure from the femoral cannula and preventing distal malperfusion during separation from extracorporeal circulation (Kazui et al., 2001; Gott et al., 2001).

Alternatively, an intraluminal graft can be used to prevent leaks from the suture lines into the false lumen. However, many intimal tears are localized very close to the coronary ostia, which poses a danger for placing the proximal graft ring in this area. Therefore, an intraluminal graft should be used in distal anastomoses during a short period of circulatory arrest, positioned at the base of the innominate artery. The intraluminal prosthesis is connected to the proximal graft with a simple graft-to-graft anastomosis. Fibrosis is ensured by wrapping the aorta over the intraluminal graft and reinforcing it with Teflon felt (Kazui et al., 2001).

In acute Type A dissections, preservation of the native valve is possible in 70-80% of cases. Aortic valve competency is achieved by simple resuspension of the commissures and reinforcement with Teflon material, or by valve-sparing root replacement. Preservation of the aortic valve generally does not affect short-term mortality or long-term survival in many cases. Postoperatively, valve replacement is not required in 80-90% of cases during a 10-year follow-up. However, valve preservation in dilated roots leads to insufficiency and is a major cause of reoperations in Type A dissections. In patients with dilated aortic roots, annuloaortic ectasia, or Marfan Syndrome, a composite valve graft is required rather than resuspension or aortic valve repair. Outcomes are worse with separate valve and graft replacement in patients with medial degenerative disease and aortic dissection. If the valve cannot be preserved, a composite valve graft is placed from the aortic root, and the coronary ostia are anastomosed to the graft (Bentall procedure). If the aortic root is dilated more than 36mm, a composite graft is indicated. Direct extension of the dissection into the coronary ostia creates a technical problem, and root repair can be difficult. In this situation, the safest approach is the Bentall procedure with the Cabrol modification, involving composite replacement of the root and aortic valve. The dissected coronary ostia are removed as buttons on aortic tissue reinforced with a Teflon strip frame, and coronary perfusion is provided with a separate tubular graft (Bachet et al., 1987; Kazui et al., 2001; Gott et al., 2001).

Type B Dissections

These are approached via left lateral thoracotomy. The aorta is cross-clamped with proximal and distal control. Resection of the intimal tear-containing segment and the dilated portion of the descending aorta, followed by graft interposition, obliterates the distal and proximal false lumens and redirects flow to the true lumen. As in Type A dissections, suture lines are reinforced with Teflon strips. Intraluminal grafts can be used for distal anastomoses, or for proximal anastomoses if sufficient true lumen exists between the intimal tear and the left subclavian artery. Ensuring distal perfusion is critical for safe operation, as prolonged clamping of the dissected descending aorta can increase the risk of paraplegia. Distal perfusion techniques, such as heparin-bonded shunts and atriofemoral left heart bypass, can provide adequate distal perfusion and left heart decompression during cross-clamping. Perfusion from the left atrium or left ventricular apex to the femoral artery is achieved with a centrifugal pump. This method offers the advantages of low-dose heparinization and rapid volume infusion. Retrograde progression of the dissection into the aortic arch can render proximal clamping hazardous. Hypothermic circulatory arrest is employed to replace affected portions of the aortic arch and distal aorta (Gott et al., 2001; Estrera et al., 2006).

Management of Complications

Prevention and Treatment of Intraoperative Malperfusion Syndrome

Following aortic rupture, malperfusion syndrome is the second most common cause of mortality in acute dissection. The incidence of major malperfusion in clinical series is 33%, with a mortality rate of 38-50% (Lansman et al., 2005). Malperfusion can occur during surgical treatment, similar to patients presenting with cerebral symptoms and ischemic extremity findings. Intraoperative malperfusion, observed in 13% of cases, can be detected by TEE. Throughout all phases of the operation, monitoring right radial artery pressure indicates the adequacy of innominate artery perfusion and prevents the development of cerebral malperfusion. Perfusion induction should be performed slowly to prevent abrupt displacement of the intimal flap due to retrograde perfusion from the femoral cannula. TEE assessment of flow patterns in the true and false lumens is the most effective method for detecting intraoperative malperfusion. At the end of the repair, perfusion is re-established antegrade, through the ascending graft, towards the true lumen, and femoral retrograde perfusion is not used for the remainder of the operation. This prevents the formation of residual secondary tears due to retrograde pressure and intraoperative and major neurological damage due to compression of the descending aorta or aortic root. During a short period of circulatory arrest, an arterial cannula at the end of a pre-prepared Y-extension on the arterial pump line is placed into the completely transected ascending aorta and secured with an umbilical tape. This soft cannula should then remain within the resected aortic segment. It is important to prevent selective cannulation of the arch branches by advancing this soft cannula into the true lumen beyond the arch vessels into the proximal descending aorta. This maneuver can be performed quickly and is also the only effective method in cases of circumferential ascending aortic tear and intimointimal intussusception of the inner tubular portion into the aortic arch. Under circulatory arrest, the prolapsed inner tube is withdrawn into the ascending aorta, and a cannula is placed into the true lumen and secured around the ascending aorta. Some utilize cannulation from the left ventricular apex or excision of the membrane separating the two lumens in the arch to ensure flow to the arch branches. Both methods have disadvantages. Continuous perfusion from a left ventricular cannula is impractical, especially when the aortic valve is incompetent. Forceful fenestration in the arch leaves only a fragile adventitial layer, itself posing a risk of dissection.

Fenestration and Management of Malperfusion

In patients presenting with malperfusion syndrome, emergency proximal repair and redirection of flow to the true lumen are effective in preventing malperfusion in over 90% of cases. Persistent peripheral malperfusion situations

should be evaluated individually for each patient. In the simplest cases, lower extremity ischemia can be treated with a crossover graft. More severe malperfusion, such as total obstruction of the aortic bifurcation or renal or visceral ischemia, is effectively treated with abdominal aortic fenestration (window). Fenestration is performed in the infrarenal aorta by isolating the aorta up to its bifurcation with clamps placed distal to the renal arteries. The aorta is opened longitudinally through the dissected segment, and the dissected membrane in the isolated aortic segment is completely excised, with the aortotomy closed by primary suture. Alternatively, the aorta is completely transected near the distal clamp site, the wide portion of the membrane up to the proximal clamp is excised, and after the distal false lumen is obliterated with a separate suture, the aorta is repaired with an end-to-end anastomosis. This method exposes a portion of the anastomotic suture line to the adventitial layer of the dissected aorta. Due to this potential problem, abdominal fenestration provides better long-term outcomes. Surgical fenestration in the abdomen is a simple and well-tolerated procedure. This approach should also be considered in Type B dissections and cases with malperfusion in distal aortic branches that require intervention before proximal repair of the descending aorta. Radiological catheter techniques provide an alternative to surgical fenestration by creating controlled re-entry tears to restore flow to compressed aortic branches (Kouchoukos and Masetti, 2005).

Stroke and Paraplegia

Approximately 5-6% of patients with acute dissection present with stroke symptoms. When the tear is localized in the arch, the incidence of neurological symptoms rises to 42%. Most are associated with Type A dissection, and 85% of established strokes accompany carotid occlusion (Morello et al., 2021).

Stroke in acute aortic dissection presents a therapeutic dilemma. Ischemic cerebral infarction may transform into hemorrhagic infarction during CPB, leading to catastrophic neurological outcomes. In the past, extra-anatomic bypass of the carotid arteries was employed. However, recent consensus favors direct repair of the ascending aorta, as in most cases, complete prevention of cerebral malperfusion leads to improvement in neurological function. Although outcomes are worse and postoperative mortality risk increases in cases of stroke, the presence of neurological deficit at presentation in acute dissection does not constitute a contraindication for surgery. Stroke-related mortality in acute dissections is 14%. In Stanford series, only 43% of cases showed neurological improvement and satisfactory long-term outcomes.

The incidence of paraplegia in acute dissections ranges from 2-6%, with most paraplegias occurring in acute Type A dissections. In most patients presenting with paraplegia, postoperative neurological improvement is poor. Nevertheless,

emergency operation is indicated to prevent other lethal complications of dissection (Borghese et al., 2023).

Pulse Deficits and Extremity Ischemia

Pulse loss is present in 30-50% of acute Type A dissections. Local peripheral vascular operations are not appropriate as they increase acute mortality and delay definitive treatment of the dissection. Persistent extremity ischemia following aortic repair can be treated with catheter fenestration techniques and surgical fenestration, or rarely, a bypass graft may be required (Lansman et al., 2005).

Renal Failure

Dissection involves the renal arteries in 60% of patients. Clinically significant renal dysfunction occurs in 5-8% of cases but carries a high mortality risk (50%). Appropriate proximal repair of the dissection ensures renal perfusion in many cases. Postoperatively, renal perfusion should be assessed with a renal perfusion scan to determine if local treatment, such as catheter fenestration, is needed. In many patients, renal function improves, and the need for dialysis in survivors is rare (Norton et al., 2021).

Visceral Ischemia

Evidence of visceral malperfusion is observed in 3-5% of patients and is associated with high mortality (43%). If severe bowel necrosis is not present, emergency treatment should focus on the proximal aorta. Awareness of the possibility of visceral ischemia and early diagnosis is the best preventive measure, as all strategies are doomed to fail once significant necrosis has developed. If there is doubt about the viability of abdominal contents after proximal repair, further investigation with a mini-laparotomy should be performed before leaving the operating room. Assessment of visceral malperfusion with the aid of local Doppler examination can determine the necessity of more advanced local treatment methods such as catheter or surgical fenestration or vascular bypass (Norton et al., 2021).

True Lumen Compression and Pseudo-Coarctation

Rarely, coarctation-like obstruction of the descending aorta can occur due to true lumen compression, creating a pressure gradient in the narrowed aortic segment. This situation most commonly arises in the distal descending aorta and can lead to uncontrolled proximal hypertension. Treatment involves replacement of the affected aortic segment. Generally, due to its distal descending aortic localization, the risk of postoperative paraplegia increases. In high-risk patients with Type B dissection, extra-anatomic bypass from the ascending aorta to the infrarenal aorta may be performed (Papakonstantinou et al., 2022).

There is consensus among many experienced surgeons that the best short- and long-term outcomes are achieved through complete resection of the dissected aorta and full-thickness graft anastomosis to the distal aorta. When inclusion techniques are used in acute aortic dissections, the incidence of residual leak is high, and outcomes are poor. In some series, the inclusion technique is identified as a predictor of operative mortality. Similarly, the reoperation rate is higher in series employing the inclusion technique (40% at 10 years) compared to series where total excision of the dissected ascending aorta was performed (23% at 10 years). Some surgeons, based on this observation, have gone a step further and advocated for prophylactic total aortic replacement at the onset of the event. However, the magnitude and risk of this operation, combined with long-term outcomes (25% mortality, 19% reoperation), do not make such an approach rational in the acute phase (Papakonstantinou et al., 2022).

Arch Dissection

The presence of an arch tear indicates a poor prognosis. Nevertheless, the treatment of acute arch dissection (intimal tear in the aortic arch) is controversial due to the technical difficulties of direct arch replacement. It is clear that non-surgical conservative treatments or arch-wrapping procedures (in the presence of a primary tear) are inadequate and associated with high mortality. Similarly, limited surgical treatments for acute Type A dissection involving restricted resection of the ascending aorta, or leaving intimal tears in the arch or proximal descending aorta, lead to poor short- and long-term outcomes and perpetuate the risk of dissection and aortic rupture. In light of recent experience, the arch in acute dissections can be replaced with an acceptable surgical risk, and replacement should be performed in all cases (Norton et al., 2021, Papakonstantinou et al., 2022).

Marfan Syndrome

There is consensus that root replacement is the optimal treatment for acute aortic dissections in patients with Marfan syndrome. Failure to perform this consistently leads to long-term complications involving the aortic root and is a leading cause of morbidity and even mortality in these patients. The incidence of late reoperation is significantly higher (50%) in cases where composite graft replacement was not performed during the initial operation (Papakonstantinou et al., 2022).

Flow Redirection and Thromboexclusion

These methods have been proposed as alternatives to resection and graft replacement in Type B dissections. They involve extra-anatomic bypass from the ascending aorta to the infrarenal abdominal aorta and closure of the proximal descending aorta with specially designed permanent clamps. The aim is to

promote clotting in the descending aorta and reverse flow direction to ensure perfusion of the lower intercostal arteries, thereby reducing the risk of paraplegia. However, complications associated with clamp use exist. Visceral embolization, bowel infarction, and pancreatitis have occurred. Additionally, paraplegia has not been entirely prevented. This approach should only be considered in high-risk patients with Type B dissection, particularly those with severe lung disease, and staplers should be used in the descending aorta instead of permanent clamps (Kazimierczak et al., 2019).

Chronic Dissections

Surgical Indications

The need for surgical treatment in chronic dissections typically arises from symptoms related to the distal aorta (primarily pain and other symptoms of enlargement), expansion of the distal false lumen and late aneurysm formation, or problems associated with the aortic root and chronic aortic insufficiency. In asymptomatic patients, the most important indication for surgery is the size and rate of expansion of the distal aorta. In chronic dissections, 88% of rupture cases occur at diameters below 10cm, and 23% at diameters below 6cm. The most significant predictor of late mortality in medically managed cases is an aortic dissection diameter not exceeding 5cm. Another surgical indication in chronic ascending aortic dissections includes persistent or new cardiac complications and complications from previous aortic or cardiac operations. In some cases, localized ballooning in areas of weakness within the aneurysmal wall indicates the need for early surgery. When recommending elective operation for the descending aorta in asymptomatic patients, factors such as the patient's age, coexisting pulmonary dysfunction, other medical conditions, and the length of the resected segment should be considered. The risk of postoperative paraplegia must be carefully evaluated (Coady et al., 2002; Elefteriades,2003).

Surgical Treatment

Chronic dissections generally necessitate extensive aortic resection and reconstruction. In many chronic Type A dissections, composite graft replacement of the aortic valve and root is required. In some chronic Type A dissections, valve-sparing root replacement may be possible. Rarely, significant dilation may be present in the ascending arch and descending portions of the aorta, necessitating their replacement. This is typically achieved using the staged replacement technique known as the elephant trunk procedure. In many cases of chronic Type B dissection, replacement of the entire descending aorta and, in some, the thoracoabdominal aorta may be required. In chronic dissections, perfusion of vital structures may depend on the presence of the false lumen, requiring flow to both the true and false lumens. This is achieved by creating a

fenestration between the true and false lumens and performing a distal anastomosis to the outer wall of the chronically dissected aorta. Tissue fragility in chronic dissections is not an issue; however, operative morbidity increases proportionally with the extent of aortic resection (Kouchoukos et al., 2000; De Paulis et al., 2001).

Postoperative Follow-up and Late Complications

Patients who have undergone surgery for acute aortic dissection are at risk for the development of aortic-related complications. Complications in the proximal repair region are generally associated with surgical choices or techniques used in aortic root reconstruction. Various reasons may include the use of the inclusion technique or primary repair of the dissected aorta, failure to perform root replacement in the presence of Marfan syndrome or a dilated aortic root, or the use of intraluminal grafts in proximal anastomoses. Complications in the distal aorta are linked to the persistence of the distal false lumen and constitute the primary cause of late problems in these patients. The reported rate of persistent false lumen in the literature ranges from 70-100%. The continued presence of a distal false lumen is known to be associated with aneurysmal dilation of the aorta, late reoperation, and death due to rupture. With technical advancements, the rate of persistent false lumen has been reduced to below 50%, leading to improvements in survival and a reduction in late complication rates. Patient follow-up commences upon discharge, with baseline postoperative angiographic, tomographic, and magnetic resonance imaging studies establishing a regular non-invasive surveillance schedule. These studies should be performed annually in cases where stable repair has been achieved, and more frequently in other situations to ensure optimal timing for elective surgical treatment in the chronic phase (Elsayed et al., 2017; Kölbel et al., 2014; Nienaber and Eagle,2002).

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