TÜRK KARDİYOLOJİ DERNEĞİ

TÜRK KARDİYOLOJİ DERNEĞİ
Kardiyak Görüntüleme Çalışma Grubu

TÜRK KARDİYOLOJİ DERNEĞİ
Kalp Kapak Hastalıkları Çalışma Grubu

Kardiyak Görüntüleme ve Kapak Hastalıkları Toplantısı

10-12 Ekim 2025 Crowne Plaza, ANKARA

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BİLİMSEL PROGRAM

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İçerenköy Mah Harman Sk. Kılıç Apt. No: 2/A
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E posta : KGKH2025@simurgevent.com
www.simurgevent.com

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BİLİMSEL SEKRETERYA
Nish İstanbul A Blok Kat: 8 No: 47-48
Çobançeşme, Bahçelievler 34196 İstanbul / Turkey
T: +90 212 221 17 30/38 F: +90 212 221 17 54
E-Posta: tkd@tkd.org.tr www.tkd.org.tr

KURULLAR

TÜRK KARDİYOLOJİ DERNEĞİ YÖNETİM KURULU

Başkan	Muzaffer Değertekin
Başkan Yardımcısı	Ertuğrul Okuyan
Başkan Yardımcısı	Dursun Aras
Genel Sekreter	Bülent Mutlu
Genel Sekreter Yardımcısı	Mehmet Ertürk
Sayman	Eralp Tutar
Üye	Fahriye Vatansever Ağca
Üye	Serdar Sevimli
Üye	Can Yücel Karabay

KALP KAPAK HASTALIKLARI ÇALIŞMA GRUPLARI

Başkan	İbrahim Halil Kurt
Üye	Cem Doğan
Üye	Bedri Caner Kaya
Üye	Kadir Uğur Mert
Üye	Mehmet Levent Şahiner
Üye	Hatice Tolunay

KARDİYAK GÖRÜNTÜLEME ÇALIŞMA GRUBU

Başkan	Gamze Babur Güler
Üye	Suzan Hatipoğlu
Üye	Mustafa Ozan Gürsoy
Üye	Dilay Karabulut
Üye	Türkan Seda Tan Kürklü
Üye	Pelin Kacara Özer

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10 EKİM 2025, CUMA | A SALONU

08:00-09:00 Bir Kardiyolog'a Sadece Ekokardiyografinin Yetmediği Vakalar

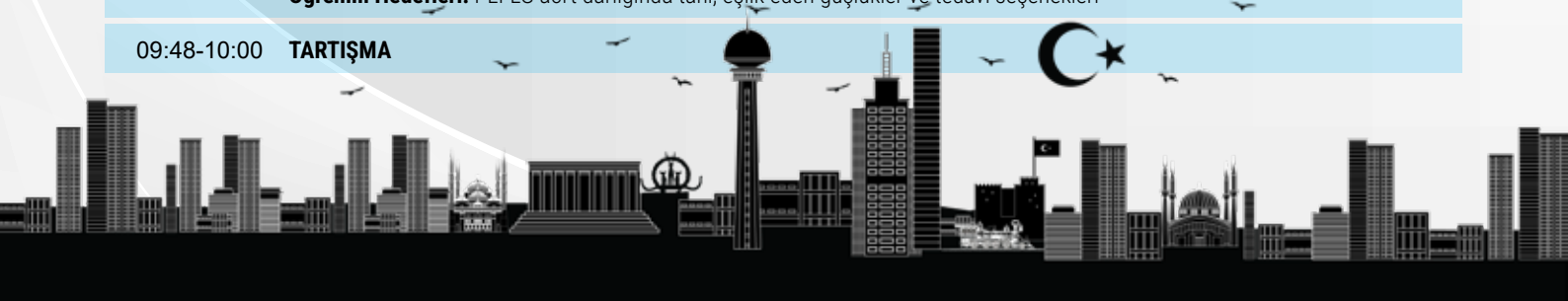
Oturum Başkanı : Dr. Cemil İzgi, Dr. Dilek Yeşilbursa
Panel: Dr. Armağan Acele, Dr. Remziye Doğan

08:00-08:12	**Hipertrofik Ventrikülde Etyolojiyi Anlamakta Kardiyak MRI Katkısı Nedir?** **Öğrenim Hedefi:** Hipertrofik ventrikül etyolojisinde kardiyak MRI'nın katkısı	**Dr. İbrahim Altun**
08:12-08:24	**Mitral ve Aort Kapak Kaçaklarını Kardiyak MRI ile Takip Etmem mi Gerekiyor?** **Öğrenim Hedefi:** Aort ve mitral kapak kaçaklarının cerrahi kararında ve takiplerinde kardiyak MRI'nın katkısı nedir?	**Dr. Ahmet Demirkıran**
08:24-08:36	**Dilate Kardiyomiyopati Etyolojisine Kardiyak MRI Nasıl Bir Katkı Sağlar?** **Öğrenim Hedefi:** Dilate KMP'de kardiyak MRI vaka örnekleri ile etyolojinin aydınlatılması	**Dr. Ahmet Barutçu**
08:36-08:48	**Aritmojenik KMP'de Segmentleri Kardiyak MRI Ekokardiyografiden Daha iyi Değerlendirir mi? Tanı Başka Ne Gibi Katkılar Sunar?** **Öğrenim Hedefi:** Aritmojenik kardiyomiyopatide segmenter değerlendirme, LGE tutulum paterni, t anı kriterlerinin değerlendirilmesi.	**Dr. Begüm Uygur**
08:48-09:00	**TARTIŞMA**	

09:00-10:00 İleri Aort Darlığı Hastasında Ya Dert Bir Değilse…

Oturum Başkanı : Dr. Levent Şahiner, Dr. Ertan Vuruşkan
Panel: Dr. Özgür Ulaş Özcan, Dr. Oğuzhan Birdal

09:00-09:12	**Ciddi Sol Ventrikül Sistolik Disfonksiyonunun Yönetimi** **Öğrenim Hedefleri:** LV EF ciddi disfonksiyone iken TAVI şansımızın olduğu hastanın yönetimi, LV EF ciddi disfonksiyone iken genç konjenital kapak hastalığının yönetimi	**Dr. Ali Rıza Akyüz**
09:12-09:24	**İleri Mitral Yetersizliği Olan Bir Vakanın Yönetimi** **Öğrenim Hedefleri:** İleri mitral yetersizliğinin izole aort müdahalesi ile azalma şansı var mı? Eşlik eden primer mitral patolojilerin yönetimi	**Dr. Derya Tok**
09:24-09:36	**Dar Aort Kökünün Cerrahi ve Transkatater Çözümleri ama Devam Eden Sorunları** **Öğrenim Hedefleri:** Cerrahi aort kapak ve TAVI'de hasta protez uyumsuzluğu nedir? Önlemler nelerdir? Tanı ve tedavisi ne olmalıdır?	**Dr. Adnan Burak Akçay**
09:36-09:48	**Paradoksik Düşük Akım Düşük Gradiyent Aort Darlığında Tanısal Zorluklar, Tedavi Kararında Güçlükler** **Öğrenim Hedefleri:** PLFLG aort darlığında tanı, eşlik eden güçlükler ve tedavi seçenekleri	**Dr. Sibel Turhan**
09:48-10:00	**TARTIŞMA**	



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10:00-10:30	**KAHVE MOLASI**	
10:30-11:30	**Protez Kapak Patolojilerine Vaka Örnekleriyle Işık Tutalım**	
	Oturum Başkanı : Dr. Mehmet Özkan, Dr. Saide Aytekin **Panel:** Dr. Belma Yaman, Dr. Zehra Güven Çetin	
10:30-10:42	**Aort Kapakta Duydum da Mitral Kapakta da Hasta Protez Uyumsuzluğu Olur mu? Bu İkisini Nasıl Tanıyacağım?** **Öğrenim Hedefleri:** Vakalarla aort ve mitral kapakta hasta protez uyumsuzluğundan ne zaman şüphelenilir? Tanısı nasıl koyulur?	**Dr. Dilek Çicek Yılmaz**
10:42-10:54	**Cerrahi ve Transkatater Biyoprotez Kapak Trombüsü; Tanı ve Yönetimi** **Öğrenim Hedefleri:** Cerrahi ve TAVI kapak trombüsünden ne zaman şüphe edilir? Tanısı? Güncel kılavuzlar eşliğinde yönetimi?	**Dr. İrem Yılmaz**
10:54-11:06	**Yıl 2025, Hala Pannus ve Trombüsü Mekanik Kapakta Karıştırıyor muyuz?** **Öğrenim Hedefleri:** Mekanik kapakta pannus nedir? Nasıl tanınır? Trombüsle karışır mı?	**Dr. Sabahattin Gündüz**
11:06-11:18	**Biyoprotez Kapak Dejenerasyonu: Nedenleri, Tanısı, Transvalvüler Kaçaklar?** **Öğrenim Hedefleri:** Biyoprotez dejenerasyonunun nedenleri? Tanısı? Transvalvüler kaçakların değerlendirilmesi	**Dr. Mehmet Ali Astarcıoğlu**
11:18-11:30	**TARTIŞMA**	
11:30-12:30	**Aort Yetersizliği ve Aortopatiler: Güncel Kılavuzlarla Artık Sınırlar Daha Net!**	
	Oturum Başkanı : Dr. Oben Baysan, Dr. İbrahim Halil Kurt **Panel:** Dr. Nihal Tefik	
11:30-11:42	**Aortopatiyi Tanımak: Morfolojik Belirteçler, Genetik Değerlendirme ve Görüntüleme** **Öğrenim Hedefleri:** Aort hastalıklarında morfolojik bulgulardan genetik tanıya giden vaka örnekleri	**Dr. Ebru Özpelit**
11:42-11:54	**Aort Yetmezliğinde Etyoloji, Derecelendirme: Kaçak Santral Değilse?** **Öğrenim Hedefleri:** AY etyolojileri, AY derecelendirilmesi, eksantrik jetlerde zorluklar ve çözümleri	**Dr. Kumral Çağlı**
11:54-12:06	**Aortopati ve Aort Yetmezliğinde Müdahale Kararları: Ne Zaman, Kime, Nasıl?** **Öğrenim Hedefleri:** Cerrahi ve transkateter yaklaşımlar, endikasyonlar ve güncel kılavuz önerileri	**Dr. Murat Çelik**
12:06-12:18	**Pür Aort Yetersizliğine Transkatater Girişim: Kime? Nasıl?** **Öğrenim Hedefleri:** Sadece aort yetersizliği olan hastada TAVI uygulaması; hasta seçim kriterleri, işlem ve sonuçları	**Dr. Uygar Çağdaş Yüksel**
12:18-12:30	**TARTIŞMA**	

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12:30-13:00	**Açılış Töreni - Dr. Muzaffer Değertekin**
13:00-14:00	**ÖĞLE YEMEĞİ**

14:00-15:00 Live in Box: Perkutan Mitral Balon Valvuloplasti

Oturum Başkanı : Dr. Mustafa Demirtaş, Dr. Faruk Ertaş
Uzman Panel: Dr. Ramazan Topsakal , Dr. Yüksel Kaya, Dr. Serdar Sevimli

14:00-14:15	**Romatizmal Mitral Darlığını Değerlendirme, Balon Valvuloplastiye Uygunluk** **Öğrenim Hedefleri:** Romatizmal mitral darlığında ciddiyet ve balon valvuloplastiye uygunluk değerlendirilmesi	**Dr. Asuman Biçer**
14:15-14:30	**Transeptal Ponksiyonda Detaylar** **Öğrenim Hedefleri:** Transseptal ponksiyonun floroskopik ve ekokardiyografik füzyon eşliğinde değerlendirilmesi	**Dr. Serkan Kahraman**
14:30-14:45	**Live in Box: Mitral Balon Valvuloplasti** **Öğrenim Hedefleri:** Live in box - Mitral balon valvuloplasti işleminin gösterilmesi	**Dr. Kadir Uğur Mert**
14:45-15:00	**TARTIŞMA**	

15:00-16:00 Barlow Hastalığına Dair Her Şey

Oturum Başkanı : Dr. Çetin Erol, Dr. Hatice Tolunay
Panel: Dr. Mehmet Şah Topcuoğlu, Dr. Levent Pay

15:00-15:10	**Barlow Hastalığı: Fibroelastik Yetersizlik ve Forma Fruste Mitral Kapak'tan Farkı Nedir? Barlow'da Mitral Yetersizliği Derecelendirmede Zorluklar ve Çözümler…** **Öğrenim Hedefleri:** Temel tanımlar nelerdir? Sınıflandırma nasıl olmalıdır? İzole geç sistolik ve multiple jet ağırlıklı Barlow hastalığında derecelendirme nasıl olmalıdır?	**Dr. Sena Sert Şekerci**
15:10-15:20	**Mitral Anüler Disjunction ve Psödo MAD Nedir?** **Öğrenim Hedefleri:** MAD ve Psödo MAD'ın görüntüleme farkları nelerdir? AKÖ ile ilişkisi nedir?	**Dr. Elif Ayduk**
15:20-15:30	**Barlow Hastalığında Ani Kardiyak Ölümü Nasıl Öngöreceğiz?** **Öğrenim Hedefleri:** MAD dışı EKG, klinik ve ekokardiyografik parametreler nelerdir?	**Dr. Kutay Vurgun**
15:30-15:40	**Barlow Hastasında Cerrahiye Ne Zaman Veriyoruz? Onarım Şansı Var Mı? Durabilite Nedir?** **Öğrenim Hedefleri:** Barlow hastalığında cerrahi zamanlama diğer mitral yetersizliği etyolojileri ile aynı mıdır? Onarılırsa durabilite nedir?	**Dr. Cengiz Köksal**
15:40-16:00	**TARTIŞMA**	

16:00-16:30	**KAHVE MOLASI**

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16:30-17:30	**Sarkomerik Hipertrofik Kardiyomiyopatide Tartışmalı Başlıklar**	
	Oturum Başkanı : Dr. Tuğrul Okay, Dr. Zehra Gölbaşı **Panel:** Dr. Gizem Yüksel, Dr. Ajar Koçak	
16:30-16:40	**Sarkomerik Hipertrofik Kardiyomiyopatide Sınıflama Nasıl Olmalıdır? LVOT Gradiyentini Doğru Değerlendirmenin Yöntemleri?** **Öğrenim Hedefi:** Obstrüktif, apikal, mid ventriküler tipler ne demek? Gruplar arasında epidemiyolojik farklılıklar? Obstrüktif tipleri ayırmada doğru LVOT gradiyent ölçümü nasıl olmalıdır?	**Dr. İrem Dinçer**
16:40-16:50	**Nonobstrüktif Sarkomerik Hipertrofik Kardiyomiyopatinin Prognozu Nasıldır? Sorunları Nelerdir?** **Öğrenim Hedefi:** Nonobstrüktif tip HKMP'lerin prognostik verileri, beklenen komplikasyonları (Apikal HKMP dahil)	**Dr. Arda Güler**
16:50-17:00	**Obstrüktif HKMP'de Farmakolojik Tedavi Seçenekleri** **Öğrenim Hedefi:** Obstrüktif HKMP'de medikal tedavi seçenekleri nelerdir? Hangi basamakta neyi kullanıyoruz? Yan etki ve kontrendikasyonlar nelerdir?	**Dr. Selcen Yakar Tülüce**
17:00-17:10	**Miyektomi Kime? Ne Zaman? Nasıl Yapılmalıdır?** **Öğrenim Hedefi:** Miyektomi için hasta seçim kriterleri nelerdir? Nasıl yapılmalıdır? Avantaj ve komplikasyonları nelerdir?	**Dr. Tarık Kızıltan**
17:10-17:30	**TARTIŞMA**	
17:30-18:30	**Multimodalite Görüntülemenin Fark Yarattığı Vakalar Kardiyoloji Asistanlarımız Anlatıyor...**	
	Oturum Başkanı : Dr. Necla Özer, Dr. Abdurrahman Oğuzhan **Panel:** Dr. Tuğba Kayhan Altunel, Dr. Ayça Arslan	
17:30-17:36	**Vaka 1 - Dr. Bekir Akmaz**	
17:36-17:38	**Tartışma**	
17:38-17:44	**Vaka 2 - Dr. Merve Aydın**	
17:44-17:46	**Tartışma**	
17:46-17:52	**Vaka 3 - Dr. Fahriye Ismayilova**	
17:52-17:54	**Tartışma**	
17:54-18:00	**Vaka 4 - Dr. Gözde Cansu Yılmaz**	
18:00-18:02	**Tartışma**	
18:02-18:08	**Vaka 5 - Dr. İsmet Enes Erkoç**	
18:08-18:10	**Tartışma**	
18:18-18:24	**Vaka 6 - Dr. Alperen Peksoy**	
18:24-18:26	**Tartışma**	
18:26-18:32	**Vaka 7 - Dr. Hazal Ünlügenç**	
18:32-18:34	**Tartışma**	

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11 EKİM 2025, CUMARTESİ | A SALONU

08:00-09:00 İleri Mitral Yetersizliği Hastasında Ya Dert Bir Değilse…

Oturum Başkanı : Dr. Aylin Yıldırır, Dr. Mustafa Kılıçkap
Panel: Dr. Songül Üstündağ, Dr. Örsan Deniz Urgun

08:00-08:12 **Ciddi Sol Ventrikül Sistolik Disfonksiyonunun Yönetimi** — **Dr. Betül Balaban Koçaş**
Öğrenim Hedefleri: Nereye kadar mitral yetersizliğine cerrahi yapabiliriz? Düşük EF'de düzelme beklenir mi?

08:12-08:24 **Mitral Yetersizliği Cerrahisi Olacak Genç Hastada Cerrahi AF Ablasyonu/LAA Ligasyonu Önerelim Mi?** — **Dr. Muhammet Gürdoğan**
Öğrenim Hedefleri: Mitral kapak cerrahisinde AF ablasyonu ve/veya LAA ligasyonu rutin olarak cerrahiye eklenmeli midir? Yaş bir faktör müdür? Başka klinik ve görüntüleme parametreleri var mıdır?

08:24-08:36 **Ciddi Yüksek Pulmoner Vasküler Rezistans Eşlik Ediyorsa** — **Dr. Özgül Uçar Elalmış**
Öğrenim Hedefleri: Mitral yetersizlik hastasında cerrahi kontrendikasyon oluşturabilecek PVR üst sınırı var mıdır? Peroperatif alınacak önlemler var mıdır?

08:36-08:48 **Mitral Yetersizliğine Atriyal Fibrilasyon Sebep Oluyorsa, Tedavi Alternatifleri Nelerdir? Sadece Ablasyon Seçeneğini Nereye Kadar Kullanabiliriz?** — **Dr. Özgür Sürgit**
Öğrenim Hedefleri: Atriyal mitral yetersizliğinde tedavi seçenekleri nelerdir? Ne zaman ablasyon? Ne zaman cerrahi? Ne zaman perkütan girişim?

08:48-09:00 **TARTIŞMA**

09:00-10:00 Live in Box: Alkol Septal Ablasyon Kursu

Oturum Başkanı : Dr. Tuğrul Okay, Dr. İbrahim Halil Kurt
Uzman Panel: Dr. Tarık Kızıltan

09:00-09:10 **Alkol Septal Ablasyon için Hasta Seçim Kriterleri, İşlem Hazırlığı, İşlem Sırasında Kayıtlar** — **Dr. Mehmet Ertürk**

09:10-09:35 **Vaka 1 (İşlem Sırasında Ekokardiyografik, Floroskopik Görüntüleme, Kayıtlar)** — **Dr. Emre Sezici**

09:35-10:00 **Vaka 2 (İşlem Sırasında Ekokardiyografik, Floroskopik Görüntüleme, Kayıtlar)** — **Dr. Kamil Tülüce**

10:00-10:30 **KAHVE MOLASI**

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10:30-11:30	**Pulmoner Hipertansiyon: Görüntüleme Penceresinden Tanısı ve Ayırıcı Tanısı**	
	Oturum Başkanı : Dr. Bahri Akdeniz, Dr. Gülten Taçoy **Panel: Dr. Emine Altuntaş, Dr. Zeynep Ulutaş**	
10:30-10:42	**Sağ Ventrikül Volüm Yükü mü? Basınç Yükü mü? Pulmoner Arter Basıncını Ekokardiyografik Olarak Nasıl Değerlendireceğiz?** **Öğrenim Hedefi:** D septum nedir? Sistolik ve diyastolik D septum ayrımı, pulmoner arter basıncının ekokardiyografik hesaplanması nasıl olmalıdır?	**Dr. Ayşe Çolak**
10:30-10:42	**Pulmoner Hipertansiyon Oturumunda Noninvaziv Hemodinamik Değerlendirme** **Öğrenim Hedefi:** Pulmoner arteriyel hipertansiyonda noninvaziv hemodinamik değerlendirilebilecek parametreler; PVR, TAPSE/PAP, PAPI, Eplar, Vexus vs.	**Dr. Volkan Kozluca**
10:54-11:06	**Grup 1 Dışı Pulmoner Hipertansiyonda Hastaları Ayırmada Ekokardiyografik İpuçları** **Öğrenim Hedefi:** Grup 1 dışı pulmoner hipertansiyonda ekokardiyografik ipuçları	**Dr. Nihan Kahya Eren**
11:06-11:18	**Dilate Sağ Kalpte Pulmoner Hipertansiyon Ayırıcı Tanı Vaka Örnekleri** **Öğrenim Hedefi:** Vaka örnekleri ile dilate sağ ventrikül hastalarında konjenital vakaları, kardiyomiyopatileri, primer triküspit kapak hastalıklarını değerlendirmek	**Dr. Gamze Babur Güler**
11:18-11:30	**TARTIŞMA**	
11:30-12:30	**Triküspit Kapak Hastalıklarında Güncel Yönetim**	
	Oturum Başkanı : Dr. Eralp Tutar , Dr. Ali Oto **Panel: Dr. Mustafa Yılmaz, Dr. Begüm Yetiş Sayın**	
11:30-11:42	**Triküspit Regürjitasyonunun Etyoloji Değerlendirilmesi: 3B Ekokardiyografinin Fark Yaratabileceği Durumlar** **Öğrenme Hedefleri:** Triküspit regürjitasyonunun değerlendirilmesinde 3B ekokardiyografinin sağladığı ek değer	**Dr. Andrzej Gackowski**
11:42-11:54	**Hangi Görüntüleme Teknikleri ve Kestirim Değerleri Sağ Ventrikül Yetmezliğinin Yakın Olduğunu Gösterir?** **Öğrenme Hedefleri:** Triküspit cerrahisi endikasyonu için sağ ventrikül yetmezliği/genişlemesi eşik değerleri. Sağ ventrikül fonksiyon ve boyutunun multimodal görüntüleme ile nasıl takip edileceği	**Dr. Fahriye Vatansever Ağca**
11:54-12:06	**Perkütan Triküspit Kapak Girişimleri** **Öğrenme Hedefleri:** Triküspit kapak için perkütan girişim seçenekleri, hasta seçim kriterleri ve sonuçlar	**Dr. Betül Eker Dayı**
12:06-12:18	**Cerrahi Sonrası Triküspit Kapak: Komplikasyonlar Nasıl Tanınır ve Yönetilir?** **Öğrenme Hedefleri:** Triküspit cerrahisinin erken ve geç dönem komplikasyonları, bu komplikasyonların nasıl yönetileceği	**Dr. Gamze Babur Güler**
12:18-12:30	**TARTIŞMA**	
12:30-14:00	**ÖĞLE YEMEĞİ**	

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14:00-15:00 Live in Box: Valve in Valve Oturumu

Oturum Başkanı : Dr. Muzaffer Değertekin, Dr. Cem Barçın
Uzman Panel: Dr. Ender Örnek, Dr. Atila Bitigen, Dr. Seyfettin Gürbüz

14:00-14:10 **Valve in Valve Endikasyonları ve Kapak Seçim Kriterleri**
Öğrenim Hedefleri: Valve in Valve endikasyonları, kapağın bilindiği ve bilinmediği koşullarda yeni kapak seçimi
Dr. Veysel Özgür Barış

14:10-14:35 **Live in Box: Aort Dışı Poziyonda Valve in Valve**
Öğrenim Hedefleri: Mitral veya Triküspit Biyoprotez kapaklarda Valve in Valve vakası
Dr. Levent Korkmaz

14:35-15:00 **Live in Box: Aort Kapak İçerisinde Valve in Valve**
Öğrenim Hedefleri: Aort Kapak için Valve in Valve Vakası
Dr. Can Yücel Karabay

15:00-15:45 **Kararın Fark Yaratsın: Forziga Tedavisi - UYDU SEMPOZYUMU** AstraZeneca

Panel: Dr. Lale Tokgözoğlu, Dr. Yüksel Çavuşoğlu, Dr. Gamze Babur Güler

15:45-16:30 **KAHVE MOLASI**

16:30-17:30 **Transkatater Mitral Girişimlerinde Güncelleme**

Oturum Başkanı : Dr. Erdoğan İlkay, Dr. Fahriye Vatansever
Panel: Dr. Murat Tulmaç , Dr. Mert Evlice

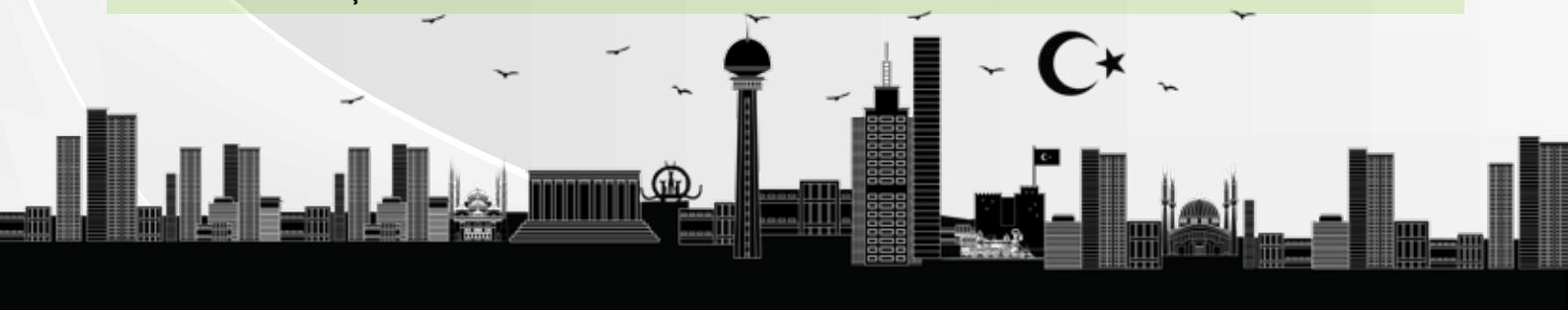
16:30-16:40 **Güncel Çalışmalara Yetişmek Zor Mu? Güzel Bir Özet Dinliyoruz…**
Öğrenim Hedefi: Mitral perkütan girişimlerinde güncel çalışma sonuçları ve kılavuzlardaki son değişiklikler
Dr. Uğur Nadir Karakulak

16:40-16:50 **Mitral Uç Uca Tamir İşleminde Hasta Seçimi, İşlem Yönetimi, Son Kontroller**
Öğrenim Hedefi: Mitral TEER Tedavisinde güncel hasta seçim kriterleri, işlem takibi ve cihaz sonrası değerlendirmeler
Dr. Ekrem Güler

16:50-17:00 **Katater Laboratuvarını Kabusa Dönüştüren Vaka Örnekleri**
Öğrenim Hedefi: Mitral TEER uygulanan komplike vaka örnekleri
Dr. Andrzej Gackowski

17:00-17:10 **Mitral TEER Dışı Perkütan Girişim Alternatifleri**
Öğrenim Hedefi : TEER dışı perkütan tedavi alternatifleri ve örnek vakalar
Dr. Hüseyin Bozbaş

17:10-17:30 **TARTIŞMA**



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Kardiyak Görüntüleme ve Kapak Hastalıkları Toplantısı

10-12 Ekim 2025 Crowne Plaza, ANKARA

17:30-18:30	**Kardiyak Kitlelerde Multimodalite Görüntüleme (Vaka Oturumu)**
	Oturum Başkanı : Dr. Mustafa Kemal Batur, Dr. Sümeyye Güllülü **Panel: Dr. Sevim Türkday Derebey, Dr. Aslıhan Merve Toprak Su**
17:30-17:36	**Vaka 1 - Dr. Mükremin Çoşkun**
17:36-17:38	**Tartışma**
17:38-17:44	**Vaka 2 - Dr. Yunus Emre Yavuz**
17:44-17:50	**Tartışma**
17:50-17:56	**Vaka 3 - Dr. Aylin Şafak**
17:56-17:58	**Tartışma**
17:58-18:04	**Vaka 4 - Dr. İlayda Güngör Bostancı Alp**
18:04-18:06	**Tartışma**
18:06-18:12	**Vaka 5 - Dr. Tuğba Çetin**
18:12-18:14	**Tartışma**
18:14-18:20	**Vaka 6 - Dr. Ece Çelebi Çoşkun**
18:20-18:22	**Tartışma**
18:22-18:28	**Vaka 7 - Dr. Çağlar Kaya**
18:28-18:30	**Tartışma**

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Kardiyak Görüntüleme ve Kapak Hastalıkları Toplantısı

10-12 Ekim 2025 Crowne Plaza, ANKARA

12 EKİM 2025, PAZAR | A SALONU

08:00-09:00	**Mekanik Sirkülasyon Desteğinde Ekokardiyografi**
	Oturum Başkanı : Dr. Ahmet Çelik, Dr. Ali Kemal Kalkan, Dr. Rüçhan Akar **Panel:** Dr. Aslı Sönmez, Dr. Çağrı Zorlu, Dr. Serhat Sığırcı
08:00-08:12	**Kime? Ne Zaman? Uygun Hasta Seçimi Nasıl Olmalı?** **Öğrenim Hedefi:** Hangi hasta mekanik sirkülasyon desteğine ihtiyaç duyar? Hangi cihaz, ne zaman seçilmeli? LVAD/BIAD/ECMO/İmpella... **Dr. Hatem Soliman Aboumarie**
08:12-08:24	**İmplantasyon Öncesinde Değerlendirme** **Öğrenim Hedefi:** Sol ve Sağ Ventrikül fonksiyonlarının değerlendirilmesi; Aort kapak fonksiyonları, Trombüs, PFO ve Pulmoner Hipertansiyon varlığının değerlendirilmesi **Dr. Bilge Karaduman**
08:24-08:36	**Yerleştirme Sırasında Değerlendirme, Akım Hızının Düzenlenmesi** **Öğrenim Hedefi:** İşlem sırasında görüntüleme desteği, akım hızını ayarlamada kılavuzluk **Dr. Zübeyde Bayram**
08:36-08:48	**Komplikasyonları Tanıma** **Öğrenim Hedefi:** Hematom, İntrakardiyak Trombüs, Enfeksiyon, Kondüit yetersizliği vs. **Dr. Rezzan Deniz Acar**
08:48-09:00	**TARTIŞMA**
09:00-10:30	**Ultrason Eşliğinde Konjesyon Değerlendirmesi**
	Oturum Başkanı : Dr. Özlem Yıldırımtürk, Dr. Tamer Sayın **Konuşmacı:** Dr. Hatem Soliman Aboumarie
10:30-11:00	**KAHVE MOLASI**
11:00-12:00	**İleri Görüntüleme Yöntemleri ile Tanı ve Prognozda Bir Adım Öndeyiz**
	Oturum Başkanı : Dr. Hülya Çiçekçioğlu, Dr. Bahar Pirat **Panel:** Dr. Füsun Helvacı, Dr. Fatma Özge Salkın
11:00-11:12	**Anderson Fabry Hastalığında Kardiyak Tutulumun Değerlendirilmesi** **Öğrenim Hedefleri:** Fabry hastalıklarının multimodalite değerlendirilmesi, görüntüleme yöntemlerinin prognoza katkısının öğrenilmesi **Dr. Fatma Nihan Çağlar**
11:12-11:24	**Otoimmun, Otoinflamatuar Hastalıklara Bağlı Kardiyak Tutulumda Görüntüleme** **Öğrenim Hedefleri:** Sistemik otoimmun hastalıkların tanı ve prognozunda görüntüleme seçeneklerimiz **Dr. Çetin Alak**
11:24-11:36	**Dilate Olmayan Sol Ventrikül Kardiyomiyopati Nedir?** **Öğrenim Hedefleri:** Nondilate sol ventrikül kardiyomiyopati nedir? Tanı ve yönetimi nasıl olmalıdır? **Dr. Betül Cengiz Elçioğlu**
11:36-11:48	**Kardiyak Amiloidozda İleri Görüntüleme** **Öğrenim Hedefleri:** Kardiyak amiloidozda strain görüntüleme, kardiyak MRI'nın katkısı **Dr. Selda Murat**
11:48-12:00	**TARTIŞMA**

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Kardiyak Görüntüleme ve Kapak Hastalıkları Toplantısı

10-12 Ekim 2025 Crowne Plaza, ANKARA

12:00-13:00	**Perikard Hastalıkları Güncellemesi**	
	Oturum Başkanı : Dr. Yücel Balbay, Dr. Elif Eroğlu **Panel:** Dr. Emre Paçacı, Dr. Canan Elif Yıldız	
12:00-12:12	**Konstriktif Perikarditten Ne Zaman Şüphelenelim? Ekokardiyografik Tanıda Neler Önemlidir?** **Öğrenim Hedefleri:** Konstriktif perikardinin patofizyolojisi, kliniği ve ekokardiyografisi	**Dr. Esra Dönmez**
12:12-12:24	**Radyoterapiye Bağlı Perikard Hastalıklarında Tanı ve Tedavi Güçlükleri** **Öğrenim Hedefleri:** Radyoterapi ilişkili perikard hastalıklarının değerlendirilmesi, yönetiminde güçlükler	**Dr. Ayça Türer Cabbar**
12:24-12:36	**Cerrahi Sonrası Geçici ve Kalıcı Konstriktif Perikardit Tanıları Nasıl Koyulur? Tedavileri Nasıl Olmalıdır?** **Öğrenim Hedefleri:** Cerrahi sonrası geçici ve kalıcı konstriktif perikardit tanısında bulgular, görüntüleme nasıldır? Tedavi alternatifleri nelerdir?	**Dr. Ayşe Saatcı Yaşar**
12:36-12:48	**Konstriktif Perikarditin Cerrahi Tedavisi ve Postoperatif Takibi: Tüm Kardiyologların Bilmesi Gereken Ne Varsa…** **Öğrenim Hedefleri:** Konstriktif perikardit cerrahisi nasıl yapılır? Postoperatif değerlendirmede başarılı bir cerrahinin kriterleri nelerdir?	**Dr. Ruçhan Akar**
12:48-13:00	**TARTIŞMA**	
13:00	**Poster Yarışması**	**Hakem Kurulu: Dr. Hülya Gamze Çelik, Dr. Meryem Aktöz, Dr. Bilgin Timuralp, Dr. İrem Dinçer, Dr. Hasan Boğa, Dr. Selcen Yakar Tülüce, Dr. Gönül Açıksarı**

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Kardiyak Görüntüleme ve Kapak Hastalıkları Toplantısı

10-12 Ekim 2025 Crowne Plaza, ANKARA

KURSLAR

10 EKİM 2025, CUMA | KURS SİMULATÖR ODASI

9:00-10:30	**Fokus Ekokardiyografi** Akut Aortik Sendromlar, Pulmoner Emboli, Akut Valvüler Patolojiler, Akut Miyokard İnfarktüsü Komplikasyonları	Dr. Mustafa Ozan Gürsoy, Dr. Pelin Karaca Özer
11:00-12:30	**Aort Kapak Patolojileri Kursu** (2B/3B Aort Kapak Değerlendirilmesi, Temel Patolojilerin İncelenmesi: Romatizmal, Konjenital, Senil Dejeneratif)	Oturum Başkanı: Dr. Omaç Tüfekçioğlu Konuşmacı: Dr. Serkan Ünlü
14:00-15:30	**TEE Kursu: MitraClip ve TriClip için Ekokardiyografi Kursu (TEE Simülatörü ile)**	Dr. Andrzej Gackowski
16:00-17:30	**Temel Ekokardiyografi Kursu** (Pencereler, Ejeksiyon Fraksiyonu Hesaplama, Kapak Fonksiyonlarının Değerlendirilmesi)	Dr. Dilay Karabulut, Dr. Emir Derviş

10 EKİM 2025, CUMA | KURS WORKSTATION ODASI

08:00-09:30	**Minimalist TAVI Kursu**	Oturum Başkanı: Dr. İbrahim Halil Kurt Konuşmacı: Dr. Abdullah Yıldırım
10:00-11:30	**Kardiyak Acillerde BT ile Değerlendirme**	Dr. İbrahim Altun
12:00-13:00	**BT Anjiografi ile Koroner Damarların Değerlendirilmesi**	Dr. Emre Özpelit
13:30-15:00	**Kapak Hastalıklarında Kardiyak MRI**	Dr.Ahmet Barutçu, Dr. Begüm Uygur
15:00-16:30	**Perkütan Pulmoner Kapak İmplantasyon Kursu**	Dr. Tevfik Karagöz, Dr.Ahmet Göktuğ Ertem

11 EKİM 2025, CUMARTESİ | KURS SİMULATÖR ODASI

09:00-10:30	**TEE Kursu: MitraClip ve TriClip için Ekokardiyografi Kursu (TEE Simülatörü ile)**	Oturum Başkanı: Dr. Gökhan Kahveci, Konuşmacı: Dr. Betül Eker Dayı
11:00-12:30	**Stres Ekokardiyografi Kursu (Koroner Arter Hastalığında Stres Ekokardiyografi, Aort Darlığında, Mitral Darlığı ve Yetersizliğinde Stres Ekokardiyografi Ne Zaman, Kime Yapalım?)**	Oturum Başkanı: Dr.Özlem Esen Konuşmacı: Dr. Duygu İnan
14:00-15:30	**Kardiyologlar için Temel Nükleer Tıp kursu**	Oturum Başkanı: Dr. Feyza Çağlıyan Konuşmacı: Dr.Hülya Peker, Dr. Semra İnce
16:00-17:30	**Mitral Kapak Patolojileri Kursu** (2B/3B Mitral Kapak Değerlendirilmesi, Temel Patolojilerin Değerlendirilmesi; Romatizmal, Dejeneratif, AF ile İlişkili)	Oturum Başkanı: Dr. İrem Dinçer Konuşmacı: Dr. İrem Müge Akbulut

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Kardiyak Görüntüleme ve Kapak Hastalıkları Toplantısı

10-12 Ekim 2025 Crowne Plaza, ANKARA

KURSLAR

11 EKİM 2025, CUMARTESİ | KURS WORKSTATION ODASI

09:00-10:30	**Kardiyomiyopatiler için Kardiyak MRI**	**Oturum Başkanı: Dr. Cemil İzgi Konuşmacı: Dr. Seda Tan Kürklü**
	GE HealthCare **Hands-On Kursu**	
11:00-12:30	**Kardiyomiyopatilerde Strain Ekokardiyografi Kursu (Görüntü Alma, Analiz ve Veri Yorumlama, Sol Ventrikül Değerlendirme)**	**Dr. Elif Eroğlu, Dr. Menekşe Gerede Uludağ**
	PHILIPS **Hands-On Kursu**	
14:00-15:30	**3B Ekokardiyografi İle Paravalvuler Kaçak Boyutu, Mitral Kapak Alanı Ve Atriyal Septal Defekt Boyutu Değerlendirilmesi**	**Oturum Başkanı: Dr. Fahriye Vatansever Ağca, Konuşmacı: Dr. Ayşe İrem Demirtola**
15:30-17:00	**Özel Başlıklarda Kardiyak MRI (Konstriktif Perikardit, Kardiyak ve Ekstra Kardiyak Kitlelerin Değerlendirilmesi)**	**Dr. Özge Özden, Dr. Elif Ayduk**

TÜRK KARDİYOLOJİ DERNEĞİ

TÜRK KARDİYOLOJİ DERNEĞİ
Kardiyak Görüntüleme Çalışma Grubu

TÜRK KARDİYOLOJİ DERNEĞİ
Kalp Kapak Hastalıkları Çalışma Grubu

Kardiyak Görüntüleme ve Kapak Hastalıkları Toplantısı

10-12 Ekim 2025 Crowne Plaza, ANKARA

BİLDİRİ KİTAPÇIĞI

PS-01

DELAYED TRAUMATIC AORTIC VALVE AVULSION TEAR AFTER BLUNT CHEST TRAUMA

Koray DEMİRCİ[1], Zübeyde BAYRAM[1], Tanıl ÖZER[1]

Department of Cardiology, Kosuyolu High Specialization Education and Research Hospital, Istanbul, TURKEY

Introduction

Aortic valve damage is a rare complication of blunt chest trauma. We present a case involving a dilated left ventricle and severe aortic regurgitation originating from two slit-like defects that turned out to be an aortic avulsion tear beneath the right coronary cusp (RCC), following a fall three years prior.

Case Report

A 53-year-old male construction worker presented with exertional dyspnea during routine screening. TTE revealed severe eccentric aortic regurgitation, LV dilation (ESD: 6.0 cm), and EF of 45%. TEE revealed two slit-like defects located at the base of the RCC causing a severe aortic regurgitation. The valve leaflets and aorta appeared intact, with no evidence of vegetation, calcification, or impaired leaflet mobility. A comprehensive medical history revealed that the patient had experienced a fall from height three years earlier. Intraoperative findings confirmed RCC avulsion with NCC extension, requiring valve replacement. Postoperative recovery was uneventful. At one-month follow-up, EF improved to 55%, and LV ESD decreased to 4.5 cm.

Discussion & Conclusion

Aortic valve injury from blunt chest trauma is rare but can lead to delayed aortic regurgitation (AR). While acute AR is more common, gradual valve dysfunction may occur over time. Post-traumatic aortic valve injury is more frequently described in the literature as valve perforation, prolapse, or secondary to aortic dissection. However, a delayed aortic valve avulsion in the base of the RCC after chest trauma has been reported first time in the literature with this case. Since delayed severe AR can be caused by a blunt chest trauma, it is necessary to investigate if there is a history of trauma in patients presenting severe AR of undetermined etiology.

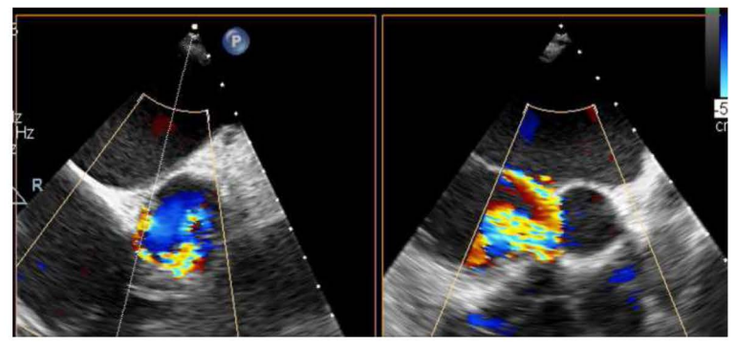


Figure 1. Severe AR from the base of the RCC

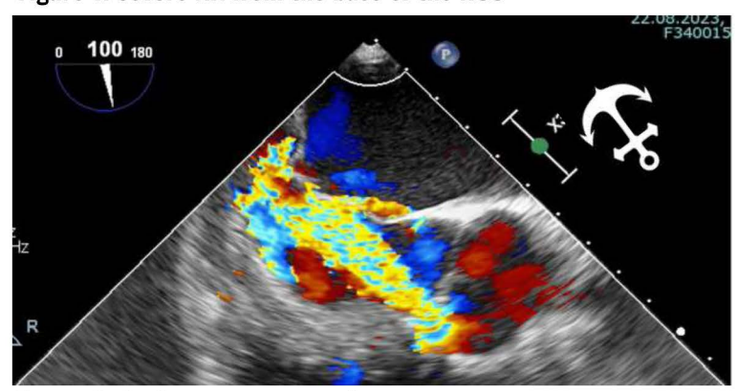


Figure 2. Anchor sign from avulsion tear

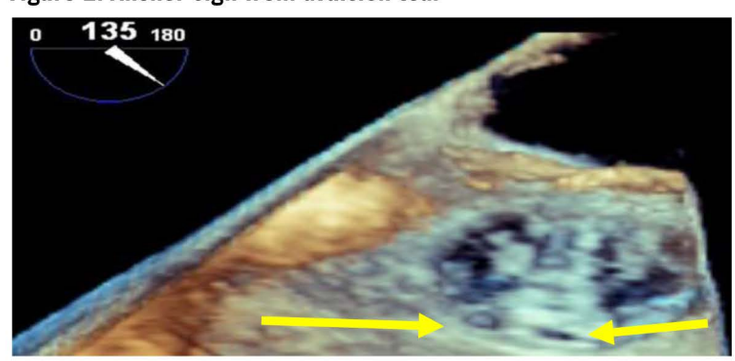


Figure 3. 3D image of the defects

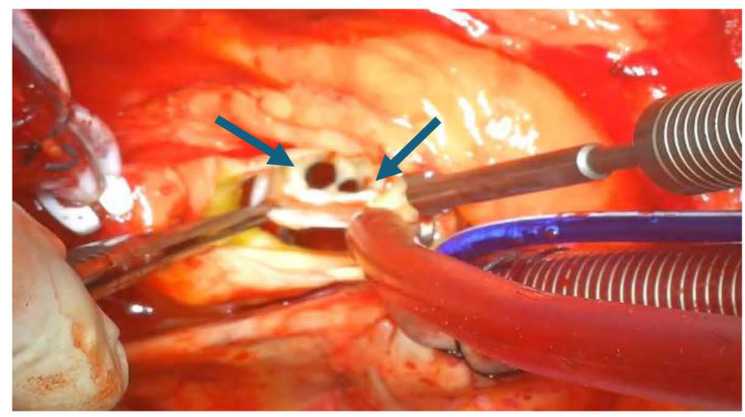


Figure 4. Surgical view of the avulsion tear defects

PS-02

PHEOCHROMOCYTOMA INDUCED TAKOTSUBO CARDIOMYOPATHY PRESENTING AS ACUTE CORONARY SYNDROME IN A YOUNG HYPERTENSIVE MALE: A CASE REPORT

Bilal Mete Ulker, Ayse Dilara Baliymez, Omer Furkan Demir, Fahriye Vatansever Agca

University of Health Sciences, Bursa Yuksek Ihtisas Training and Research Hospital

Abstract

A patient presenting with ACS-like symptoms was diagnosed with Takotsubo Cardiomyopathy secondary to pheochromocytoma. After medical stabilization with alpha- and beta-blockers, surgical resection confirmed the diagnosis. This case highlights the importance of considering secondary hypertension in young patients with cardiogenic presentations.

Case Presentation

A 35-year-old male presented to the emergency department with acute-onset chest pain, headache, diaphoresis, and dyspnea that began one hour prior. His medical history included hypertension treated with amlodipine 10 mg/day; he denied smoking, alcohol, or illicit drug use. On physical examination, he was hypertensive (BP 165/95 mmHg), tachycardic (HR 95 bpm), tachypneic, and had cold, clammy skin. Auscultation revealed bilateral rales and a diastolic murmur in the aortic area. Routine laboratory tests were obtained (Table1), and a 12-lead ECG was performed (Figure1). Based on the findings, the patient was transferred to the catheterization laboratory for coronary angiography (Figure2).

Parameter	Result	Reference Range
Creatinine	1.53 mg/dL	0.72 – 1.25 mg/dL
GFR	53.87 mL/min	70 – 180 mL/min
Sodium	136 mmol/L	135 – 145 mmol/L
Potassium	3.9 mmol/L	3.5 – 5.1 mmol/L
Hemoglobin	12.4 g/dL	13.5 – 17.5 g/dL
Troponin I	76,048 ng/L	0 – 34.6 ng/L
CK-MB	121.4 ng/mL	0 – 7.2 ng/mL

Table 1. Initial laboratory results on admission demonstrating elevated cardiac biomarkers and mild renal dysfunction.

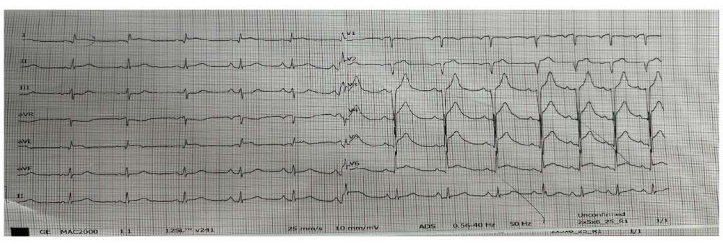


Figure 1. ECG on admission showing sinus rhythm with ST-segment elevations in V1–V6, DI, and aVL.

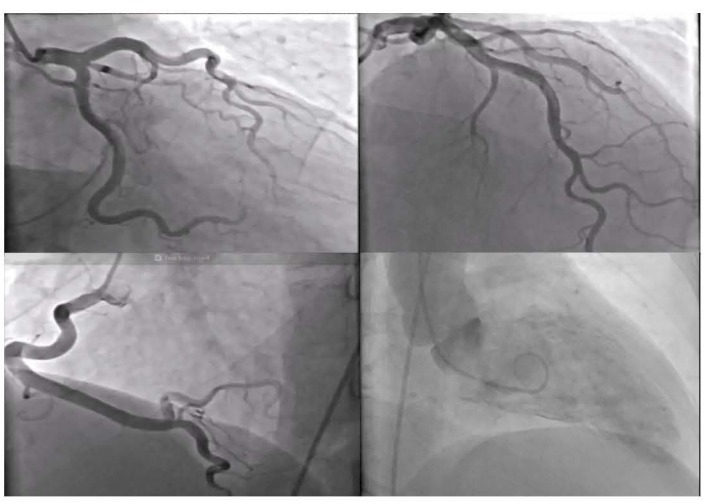


Figure 2. Coronary angiography showing normal coronary arteries; ventriculography revealed an apical aneurysm.

Transthoracic echocardiography showed a left ventricular ejection fraction (LVEF) of 35%, with an aneurysmal and hypokinetic apical region, left ventricular hypertrophy, moderate aortic regurgitation, mild mitral and tricuspid regurgitation, and normal ascending aorta and right heart chambers. Medical therapy was initiated with a preliminary diagnosis of Takotsubo cardiomyopathy (T-CMP).

Due to clinical deterioration five hours after CICU admission marked by worsening dyspnea, tachycardia (HR 145 bpm), and elevated BP (195/110 mmHg) thoracoabdominal CT angiography was performed to exclude aortic dissection. No dissection was identified. Incidentally, a left adrenal mass compressing the kidney was detected. With suspected pheochromocytoma-induced T-CMP, the patient was referred to endocrinology.

Blood and urine catecholamines were obtained (Table2), and medical therapy was revised to include carvedilol 12.5 mg twice daily, doxazosin 4 mg once daily, perindopril 10 mg one daily, ivabradine 5 mg twice daily, spironolactone 25 mg once daily, furosemide 40 mg twice daily, acetylsalicylic acid 100 mg one daily, and clopidogrel 75 mg once daily. The hypertensive episode was successfully managed. Imaging studies (Figure3) and biochemical analysis confirmed the diagnosis of pheochromocytoma.

Parameter	Result	Reference Range
Plasma Dopamine	133.7 kU/L	< 50 kU/L
Plasma Normetanephrine	6203.1 pg/mL	< 200 pg/mL
Plasma Noradrenaline	773.7 pg/mL	< 420 pg/mL
Urinary Normetanephrine	8350.5 µg/24h	< 419 µg/24h

Table 2. Catecholamine levels suggestive of pheochromocytoma.

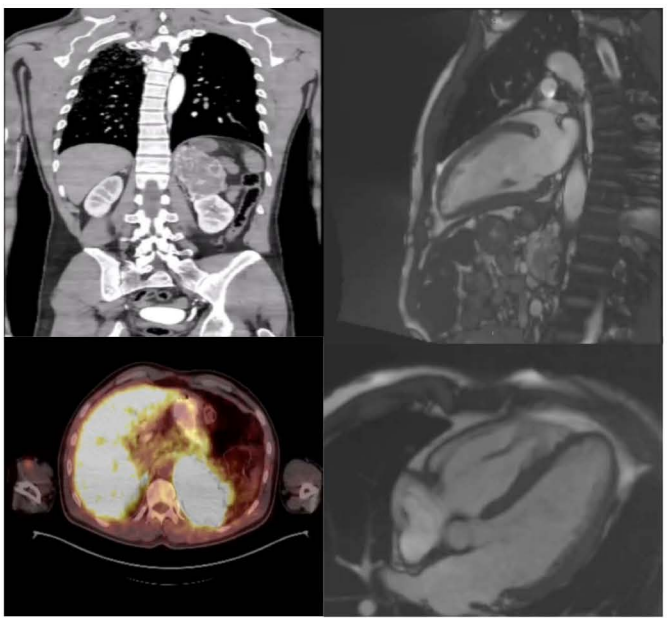


Figure 3. Incidentally, a left adrenal mass compressing the kidney was detected on CT. Cardiac MRI revealed an apical aneurysm, while PET-CT demonstrated a 10 × 6.5 cm adrenal lesion with central necrosis and intense DOTATATE uptake (SUVmax: 75.5), consistent with pheochromocytoma.

The endocrine surgery council recommended resection. Repeat echocardiography at two weeks showed LVEF improvement to 50% and resolution of the apical aneurysm. A left-sided nephrectomy was performed for complete surgical excision, and the specimens were sent for histopathological analysis (Figure4).

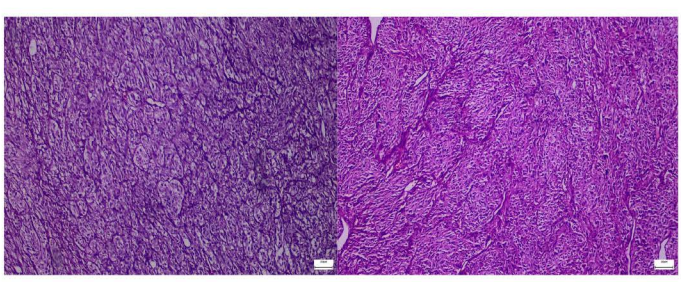


Figure 4. Histology showing Zellballen pattern characteristic of pheochromocytoma.

Results

Postoperatively, hypertensive symptoms resolved, and the patient was discharged in stable condition without complications.

Conclusion:

Pheochromocytoma is a rare but reversible cause of Takotsubo Cardiomyopathy triggered by catecholamine excess. Timely diagnosis, preoperative alpha- and beta-blockade, and surgical resection are critical for favorable outcomes. A multidisciplinary approach ensured hemodynamic stabilization and recovery. Secondary hypertension should be considered in young patients with hypertensive crises and ACS-like presentations.

PS-03

MANTLE CELL LYMPHOMA MIMICKING AORTIC INTRAMURAL HEMATOMA

Fulya Avcı Demir[1], Mehmet Beşir Akpınar[2], Akif Arslan[3], Bülent Eser[4]
[1] Department of Cardiology, İstinye University, İstanbul
[2] Department of Cardiovascular Surgery, Antalya Medicalpark Hospital, Antalya
[3] Department of Cardiology, Antalya Medicalpark Hospital, Antalya
[4] Department of Hematology, Antalya Medicalpark Hospital, Antalya

Introduction: Mantle Cell lymphoma (MCL) is an aggressive, rare form of (4-6%) non-Hodgkin lymphomas (NHL). Aortic involvement has been reported in rare cases of diffuse B-cell lymphoma, the most common type of NHL. However, according to our knowledge there have been no cases of MCL in literature presenting with acute aortic syndrome. Here, we present a case of MCL diagnosed with the help of intraoperative and pathological findings, in which an intramural hematoma (IMH) was suspected based on computed tomography (CT).

Case Presentation

45-year-old male patient with no known chronic disease was referred to the cardiology clinic due to aortic dilation detected on a chest X-ray. The patient was completely asymptomatic. His ECG showed a normal sinus rhythm. With the exception of a dilated ascending aorta(55 mm), the findings of transthoracic echocardiography were normal. Aiming to evaluate the whole aorta, a CT scan was planned

CT scan revealed the presence of an intramural haematoma measuring 16 mm in thickness around the aorta. The patient was then referred to the cardiovascular surgery.

The cardiovascular surgery team planned operation with greft implantation to the ascending aorta. During the operation due to the exclusion of an IMH based on the presence of a mass around the aorta, the excised mass was sent for pathology (Figure 1).

According to the pathology report he was diagnosed with MCL and referred immediately to the haematology outpatient clinic.A positron emission tomography scan was requested for staging at the hematology clinic.
Hypermetabolic lymph nodes, the largest 19 mm, were detected in the anterior mediastinal paratracheal area, adjacent to the aorta.Chemotherapy treatment has been scheduled. During the course of the chemotherapy regimen, the patient was planned to closely monitored for both cardiotoxicity and recurrence of the disease

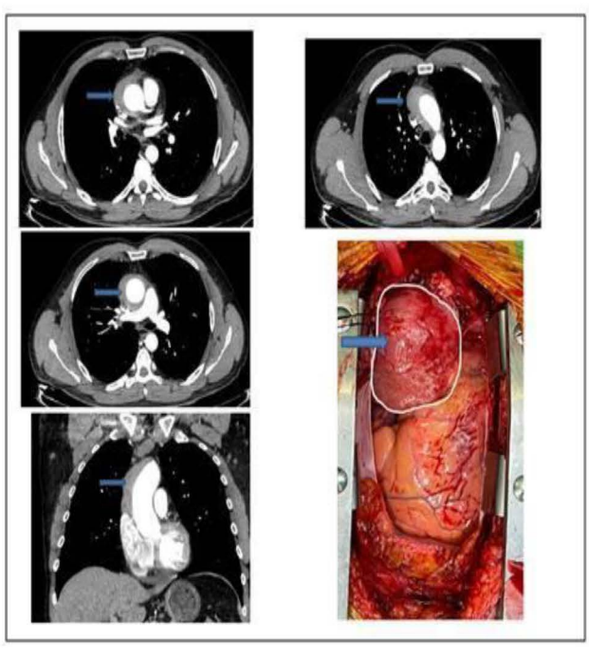


FIGURE: Axial and intraoperative viewS of periaortic lymphoma mimicking intramural hematoma

Discussion: We present a case of a patient with MCL involving the thoracal aorta, who is totally asymptomatic and whose radiological findings mimicked intramural hematoma, an acute aortic syndrome. Acute aortic syndromes include acute aortic dissection, intramural hematoma and penetrating aortic ulcer. They are life-threatening conditions that require rapid and accurate diagnosis. Typically, patients present with severe, sudden-onset pain in the chest, back, abdomen, and/or lower extremities depending on the location and extent of the aortic disease.

MHL, a rare form of NHL, typically occurs in lymph nodes and commonly involves extranodal sites, particularly the gastrointestinal (GI) tract, where it can present as a single polyp, multiple polyps, submucosal masses, ulcerative lesions or diffuse mucosal infiltration The involvement of the extrapleural space is a rare feature of lymphoma. The fat in this space can be displaced or invaded by proliferative tissue. The descending thoracic aorta is an extrapleural structure. This means lymphoma has the potential to invade the soft tissue surrounding the aorta, which could compromise the entire vessel wall. Tumours in proximity to the descending thoracic aorta are uncommon. They can be misdiagnosed. This can be as aortic disease, particularly aneurysm, dissection and intramural hematoma.

Conclusion: Periaortic involvement in lymphoma should be considered in patients with imaging findings and symptoms like night sweats and unexplained weight loss, along with chest pain. Even in asymptomatic patients, as in our case, we must consider the possibility of that. This is important because once diagnosed, immediate chemotherapy is vital for this aggressive disease.

PS-04

MALIGNANT PERIPHERAL NERVE SHEATH TUMOUR (MPNST) PRESENTING WITH CARDIAC METASTASIS TO THE INTERVENTRICULAR SEPTUM: A RARE CASE REPORT

AUTHORS
Etkin Elifoğlu, Ali Sezgin, Veysel Ozan Tanık

AFFILIATIONS
Ankara Etlik City Hospital, Cardiology Department, Ankara

Introduction

Malignant peripheral nerve sheath tumours (MPNSTs) are rare soft tissue sarcomas that are known to be aggressive. Cardiac metastasis is an extremely rare occurrence, and the extant literature on the subject is limited. Although the majority of reported cases involve ventricular cavities, the occurrence of interventricular septal metastasis is an exceptional condition. This report presents a case of lower extremity MPNST with cardiac spread to the interventricular septum, evaluated using multimodal imaging.

Figure 1. Plain radiograph of the lower extremity demonstrating a soft tissue mass in the left gluteal region without evident bone involvement.

Discussion

Cardiac metastasis from MPNST is exceedingly rare. In our case, although systemic metastatic spread was evident on PET-CT, the intracardiac lesion—located in the interventricular septum—was further evaluated by cardiac MRI. The presence of widespread extra-cardiac metastases, along with the lesion's location, morphology, and imaging features, supported its metastatic rather than primary nature.

Multimodal imaging, particularly TTE and high-resolution cardiac MRI, plays a crucial role in evaluating lesion structure, localisation, and invasiveness. The preserved right heart chambers and valvular function aligned with an asymptomatic presentation.

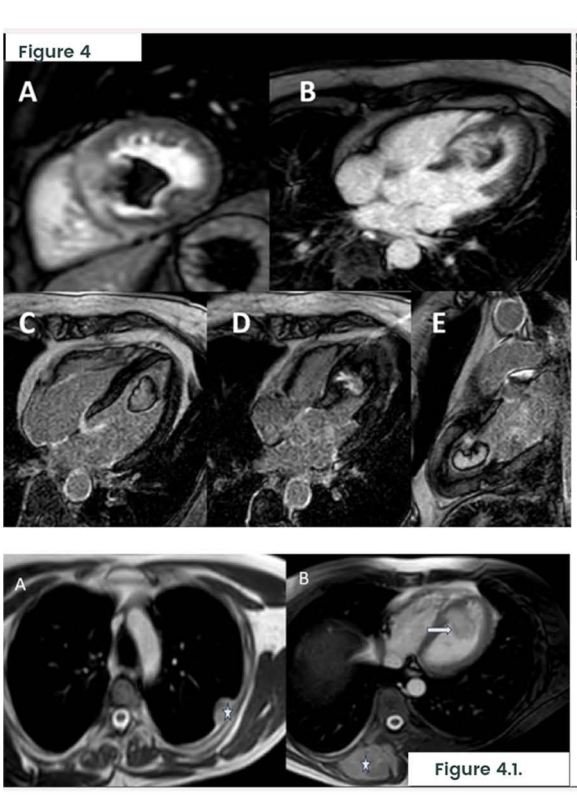
Conclusion

This case highlights the utility of multimodal imaging in characterising a metastatic MPNST lesion located in the interventricular septum. The combined use of TTE, PET-CT, and especially cardiac MRI played a pivotal role in diagnosis and multidisciplinary management. In line with similar cases in the literature, the intracardiac mass was considered part of systemic dissemination. Multimodal imaging not only improves diagnostic accuracy but also contributes significantly to treatment planning and prognostic assessment.

Case Presentation

A 44-year-old male patient with no known systemic disease presented with a necrotic, erythematous, and warm mass in the left gluteal region that had been progressively enlarging over the past four months (Figure 1). Superficial soft tissue ultrasonography revealed a hypoechoic lesion with internal vascularity. Although the tru-cut biopsy initially suggested a dedifferentiated liposarcoma, other mesenchymal neoplasms such as MPNST could not be excluded. Following wide excision, histopathological analysis confirmed high-grade malignant peripheral nerve sheath tumour (MPNST). Staging PET-CT demonstrated metastatic lesions in both lungs and the costovertebral junction (Figure 2). Postoperative adjuvant chemotherapy was planned.

Pre-chemotherapy transthoracic echocardiography (TTE) revealed a 24×17 mm, homogenous, hyperechogenic mass located in the mid-interventricular septum, extending into the left ventricular cavity. (Figure 3) Cardiac MRI revealed a lesion measuring approximately 30×41 mm protruding from the anteroseptal wall into the lumen at the mid level of the left ventricle, causing thickening of the IVS and extending slightly towards the right ventricle. On the T2 STIR sequence, there was a marked increase in intensity in the mass and anteroseptal wall, with a slight increase in intensity in the inferoseptal wall. T1-T2 mapping values showed a marked increase in the anteroseptal wall and mass, while a mild increase was observed in the inferoseptal region. At the same level, late contrast enhancement was observed only in the anteroseptal wall and mass, along with early contrast enhancement. The findings were interpreted as suggestive of metastasis. (Figure 4 ,Figure 4.1) The patient was scheduled for multidisciplinary evaluation involving cardiology, oncology, and cardiovascular surgery teams and was placed under close clinical follow-up.



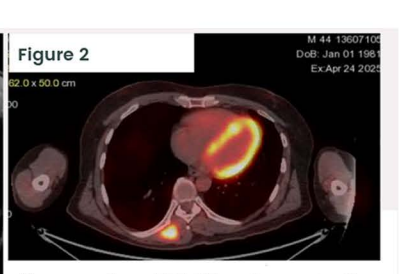


Figure 2. PET-CT demonstrating metastatic lesions in both lungs and the costovertebral junction.

Figure 4. Early-phase contrast-enhanced images (A, B) show the neck portion of the mass in contact with the interventricular septum, while late-phase contrast-enhanced images (C, D, E) demonstrate progressive contrast enhancement, including the protruding portion of the mass into the ventricular lumen.

Figure 4.1. On T1-weighted morphological images, metastatic masses are observed (A) based on the left lateral costal pleura and (B) within the right posterior paravertebral muscle planes, along with a metastatic lesion protruding into the left ventricle.

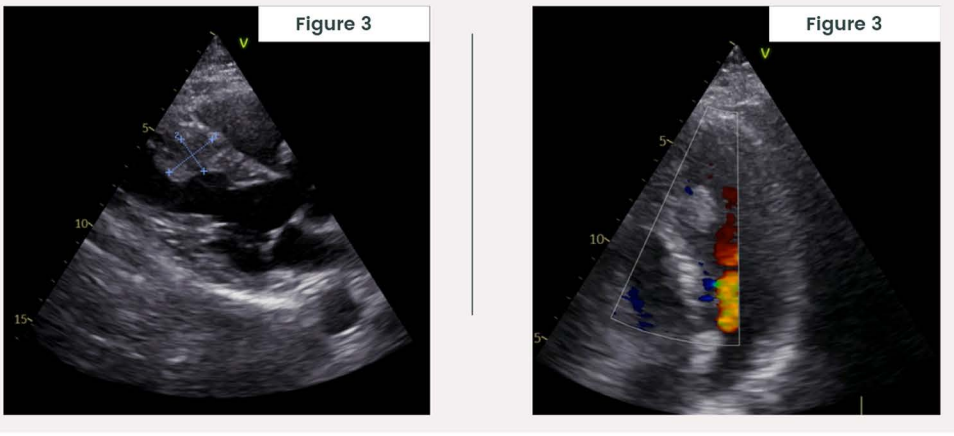


Figure 3. Appearance of the mass on modified apical four-chamber and parasternal long-axis views.

PS-05

WHAT SEEMS A CLOT MAY NOT BE A CLOT! A LEFT ATRIAL APPENDAGE MASS RESISTANT TO ANTICOAGULATION

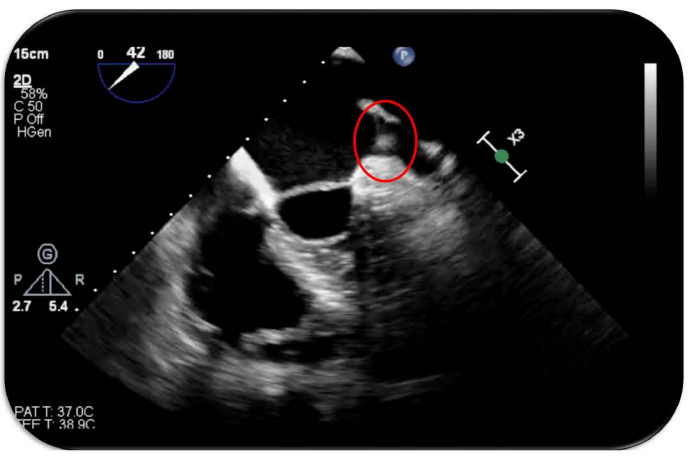
Serhat KESRIKLIOGLU[1], Yunus Emre YAVUZ[1], Nuraiym MOLOSHOVA[1], Hakan AKILLI[1]
[1] Department of Cardiology, Necmettin Erbakan University Faculty of Medicine, Konya, Türkiye

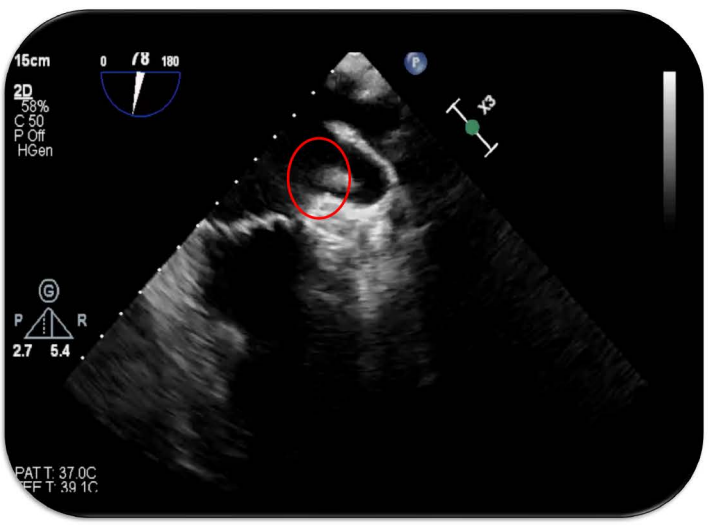
Introduction

Hypertrophic cardiomyopathy (HCM) is an inherited cardiomyopathy associated with increased sudden cardiac death (SCD) risk. Posterior HCM differs from classic septal involvement, presenting distinct clinical and imaging features. Recent evidence identifies posterior-apical HCM as a separate phenotype with unique genetics, higher fibrosis burden, and greater SCD risk. Cardiac magnetic resonance (CMR) is crucial for phenotyping and detecting concomitant myocarditis.

Case

A male patient with a history of smoking, diabetes mellitus, hypertension, and newly diagnosed atrial fibrillation presented with transient visual disturbance and palpitations several months ago. TEE revealed a 10 mm left atrial appendage (LAA) mass (red circles) interpreted as thrombus, and anticoagulation with a direct oral anticoagulant was initiated. Follow-up imaging after several months showed persistence of the mass (9 mm), prompting a switch to vitamin K antagonist therapy. Despite adequate anticoagulation, repeat TEE revealed minimal size change (8 mm) and no new symptoms. Given the lack of response, a non-thrombotic mass was suspected. Cardiac MR demonstrated imaging features consistent with myxoma. Cardioversion was performed, restoring sinus rhythm, and the patient was followed





Imaging Modality	Myxoma	Thrombus
TTE / TEE	Usually a round or oval mass, most often located in the left atrium; sessile or pedunculated; commonly attached near the interatrial septum.	May occur in any cardiac chamber; typically mobile or mural associated with regional wall motion abnormality, or attached to intracardiac devices/catheters. Spontaneous echo contrast may be present.
CMR – T1W/T2W signal	T1: Isointense or mildly hypointense; T2: Hyperintense.	Usually hypointense on both T1 and T2 (acute thrombus may appear hyperintense).
Late Gadolinium Enhancement (LGE)	Heterogeneous contrast uptake.	No enhancement.

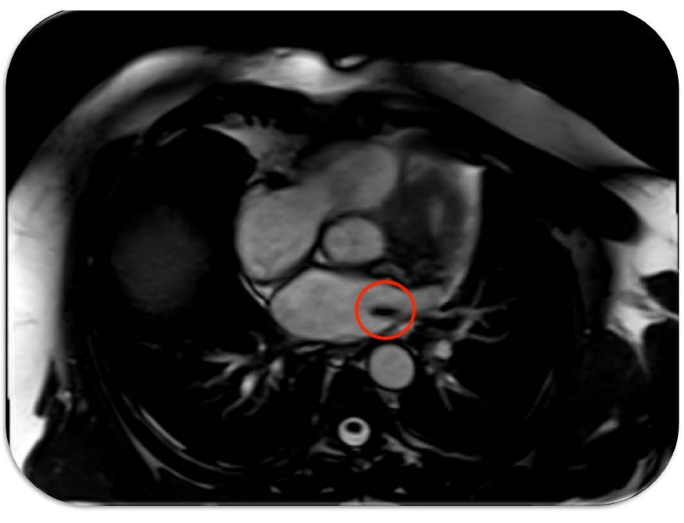
Discussion

LAA thrombus is a common complication of atrial fibrillation and a key risk factor for embolic events. However, persistence despite adequate anticoagulation should raise suspicion for alternative etiologies, including benign tumors such as myxomas. Myxomas account for ~50% of benign primary cardiac tumors, with 75% occurring in the left atrium. Surface thrombus formation is not uncommon, further complicating differentiation from pure thrombus.

Echocardiography is the first-line imaging modality for cardiac masses, offering high sensitivity for detection but limited specificity for tissue characterization. Both myxomas and thrombi may appear homogeneous or heterogeneous depending on internal hemorrhage, necrosis, or calcification. Mobility, attachment site, and morphology are often overlapping, making definitive diagnosis difficult.

Cardiac MR (CMR) provides incremental value through superior soft tissue characterization, perfusion assessment, and evaluation of LGE. In retrospective series, CMR distinguished tumor from thrombus with diagnostic accuracy exceeding 94–97%.

Thrombi typically lack contrast enhancement, while myxomas often demonstrate heterogeneous LGE reflecting their mixed histology. This imaging precision informs surgical planning and avoids unnecessary delays in definitive management.



Highlights

- Persistent LAA masses despite optimal anticoagulation should trigger immediate consideration of non-thrombotic etiologies, including myxoma.
- LAA should be assessed from multiple TEE views; single-view imaging may miss key features.
- Cardiac MRI provides superior tissue characterization, with nearly 97% accuracy in distinguishing tumor from thrombus.

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Communication : Dr. Serhat Kesriklioglu. drserhatk@gmail.com

PS-06

ATYPICAL MORPHOLOGICAL PRESENTATION OF HCM WITH SUSTAINED MONOMORPHIC VT: ASYMMETRIC POSTERIOR FREE WALL HYPERTROPHY

AUTHORS
Betül Akıncıoğlu,Nazlı Turan Şerifler, Hatice Kübra Özdemir ,Belma Kalaycı

AFFILIATIONS
Ankara Etlik City Hospital,Cardiology Department, Ankara
Ankara Etlik City Hospital,Radiology Department,Ankara

Introduction

Hypertrophic cardiomyopathy (HCM) is an inherited cardiomyopathy associated with increased sudden cardiac death (SCD) risk. Posterior HCM differs from classic septal involvement, presenting distinct clinical and imaging features. Recent evidence identifies posterior-apical HCM as a separate phenotype with unique genetics, higher fibrosis burden, and greater SCD risk. Cardiac magnetic resonance (CMR) is crucial for phenotyping and detecting concomitant myocarditis.

Figure1

Discussion

Posterior HCM is rare and poses diagnostic challenges. CMR findings, including hypertrophy, crypts, and fibrosis, have prognostic significance. Coexistent myocarditis, rarely reported, may accelerate hypertrophy and increase arrhythmic risk. ICD implantation and genetic testing were conducted per current guidelines.

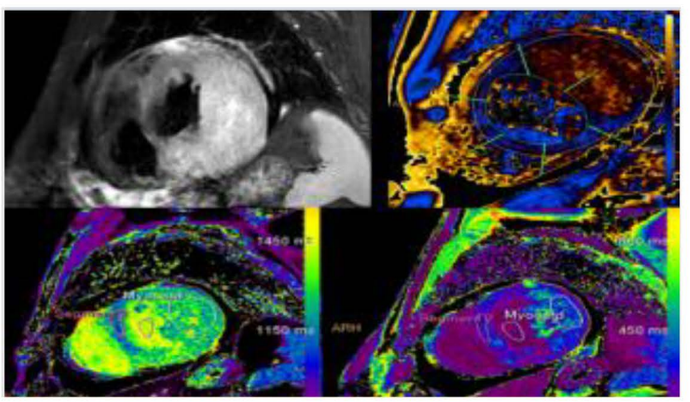


Figure 3 :At the mid-ventricular level, T2-weighted STIR sequence (A) demonstrates hyperintensity within the myocardium, more prominent in the free wall, suggestive of edema or inflammation. Corresponding T2 (B) and T1 (C) mapping values at the same level show increased values, again more marked in the free wall

Case Presentation

A 37-year-old male with a history of neuroendocrine carcinoma in remission was referred after PET-CT revealed heterogeneous metabolic activity in the left ventricle (LV). He reported exertional dyspnea and orthopnea, with a family history of sudden cardiac death and pacemaker implantation.
On examination, basal crackles and minimal pretibial edema were present. ECG showed wide QRS ventricular tachycardia with a pulse. Echocardiography revealed marked posterior wall hypertrophy (50 mm)(Figure 1) moderate–severe mitral regurgitation, mild tricuspid regurgitation, pulmonary pressure of 46 mmHg, and pleural/pericardial effusion. CMR demonstrated massive posterior wall hypertrophy (66 mm), myocardial crypts, increased T2 STIR signal, elevated T1/T2 values, mild extracellular volume expansion, and late gadolinium enhancement (fibrosis 3%), suggesting concomitant myocarditis.(Figure 2,3) Sustained VT was inducible on electrophysiology study, and an ICD was implanted for secondary preventionThe patient was discharged in stable condition with early follow-up arranged

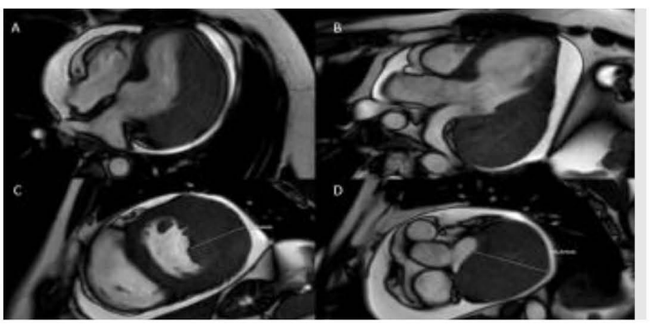


Figure 2 :Four-chamber (A) and LVOT (B) SSFP cine images demonstrate asymmetric, marked thickening of the left ventricular free wall, causing a reduction in ventricular cavity size. Short-axis (C, D) SSFP cine images reveal free wall thickening measuring up to 50 mm at the mid-ventricular level and 66 mm at the basal level, while the interventricular septum measures up to 16 mm. The myocardial signal intensity is relatively hyperintense, more pronounced in the free wall. Pericardial effusion is also present

Conclusion:

Posterior massive HCM is a rare phenotype with high SCD risk. CMR is pivotal for morphology and fibrosis assessment, while myocarditis may worsen prognosis. Genetic evaluation supports risk stratification and family screening. This case underscores the value of multimodal imaging and personalized management.

PS-07

MULTIMODAL CARDIAC IMAGING AND ENDOSCOPIC ULTRASOUND-GUIDED FINE NEEDLE ASPIRATION BIOPSY FOR ACCURATE DIAGNOSIS AND MANAGEMENT OF GIANT PRIMARY PERICARDIAL SCHWANNOMA

Ali Hakan Konuş[1], Münir Demirci[2]

[1] Department of Cardiology, Bingöl State Hospital, Bingöl, Turkey
[2] Department of Nuclear Medicine, Erzurum City Hospital, Erzurum, Turkey

Background: Primary pericardial schwannoma is a highly unusual tumor and only a few cases have been reported in the literature. We report a case of giant primary pericardial schwannoma that was accurately diagnosed and managed by multimodal cardiac imaging and endoscopic ultrasound-guided fine needle aspiration (EUS-FNA).

Case: A 47-year-old female patient presented with complaints of exertional dyspnea and non-anginal chest pain. The patient's New York Heart Association (NYHA) functional capacity score was 2-3 and N-terminus pro-B type natriuretic peptide (NT-proBNP) was elevated at 684 pg/ml. Transthoracic echocardiography (TTE) and transesophageal echocardiography (TEE) revealed a well-circumscribed intrapericardial mass containing a cystic lesion, and compressing the left atrium (LA) and inferior vena cava (IVC) (Figures 1A, 1B and 2). Contrast-enhanced computed tomography (CT) showed that the lesion was located in the posterior mediastinum, measured 10.1x8.1x5.2 cm, had regular borders, and was compressing the esophagus (Figures 3A, 3B and 3C). Magnetic resonance imaging (MRI) showed a well circumscribed, T1-isointense and T2-hyperintense pericardial mass. The mass was observed to have homogeneous signal intensity on T1 and T2-weighted images (Figures 3D, 3E and 3F). CT and MRI showed that the mass did not cause myocardial involvement, however, the mass could not be clearly demarcated from the LA and IVC. Whole-body 18 F-fluorodeoxyglucose (18 F-FDG) positron emission tomography -CT images revealed that the mass was primary, had no metastasis, and had mild to moderate 18 F-FDG avidity (Figure 4). Immunohistochemical evaluation with EUS-FNA revealed diffuse positive staining with S100 of the spindle cell proliferation foci constituting Antoni A and Antoni B areas which is consistent for schwannoma, and the Ki-67 index was less than 1%. The mass was completely removed after the pericardium was opened by performing a median sternotomy approach with del Nido cardioplegia. LA and IVC were repaired. No residual mass was detected in the patient's follow-up (Figures 1C and 1D). At 6-month and 1-year follow-up, there were no symptoms, NYHA score was 1, and NT-proBNP was normal.

Conclusion: Primary pericardial schwannomas are exceedingly rare. A comprehensive evaluation of the mass with TTE, TEE, CT, and MRI may help improve the prognosis of patients. According to imaging findings, a well-circumscribed solid pericardial mass with cystic degeneration located in the posterior mediastinum should bring schwannoma into consideration in the differential diagnosis. This case shows that EUS-FNA can be an effective and reliable method for the diagnosis of cardiac schwannomas. To our knowledge, this is the first case in which EUS-FNA has been used for the diagnosis of cardiac schwannoma. The integrative approach, including comprehensive cardiac imaging and EUS-FNA in deciding on invasive or less invasive surgical approaches and optimal treatment strategies, yielded a successful outcome in this case.

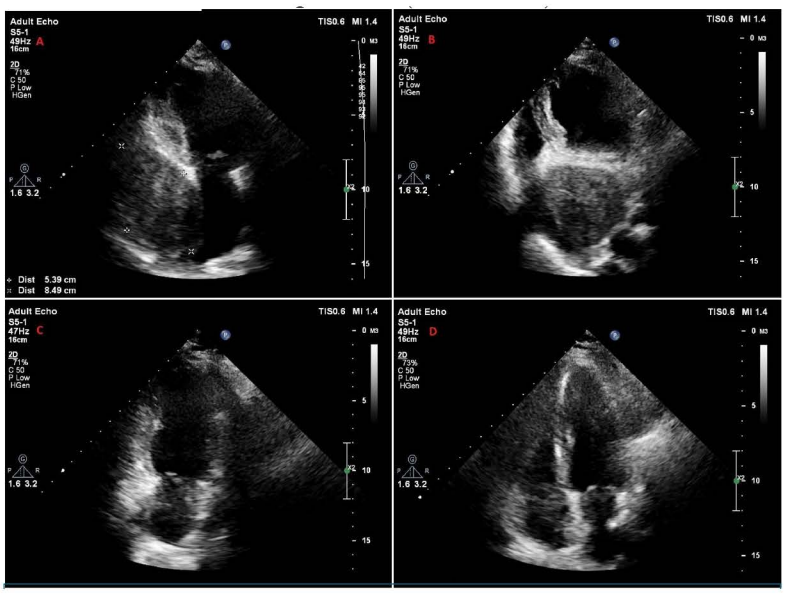


Figure 1

TTE images of the patient. A, B: Apical 2-chamber (A) and apical 4-chamber (B) views of an isoechoic mass located posterior to the left atrium and left ventricle, appearing to be intrapericardial, and compressing the left atrium. C, D: Apical 2-chamber (C) and apical 4-chamber (D) views of the patient at one year follow-up, showing that the mass was successfully removed completely.

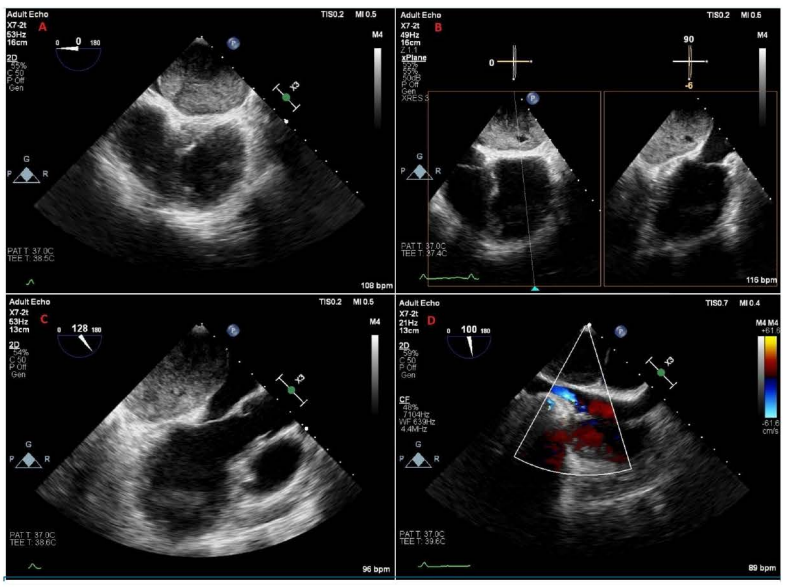


Figure 2

Transesophageal images of the mass. A: Midesophageal 4-chamber view. B: Biplane view showing that the mass contains an area of cystic degeneration. C: Midesophageal long axis view showing that the mass is compressing the left atrium, mitral annulus and partially the left ventricle. D: Midesophageal bicaval view showing the mass creating mild compression on the inferior vena cava.

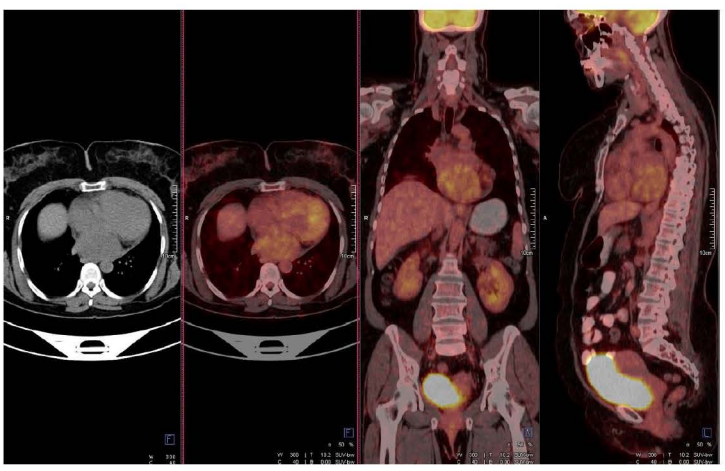


Figure 4

18F-fluorodeoxyglucose positron emission tomography shows FDG uptake in the mass. The standard uptake value was ~5.2.

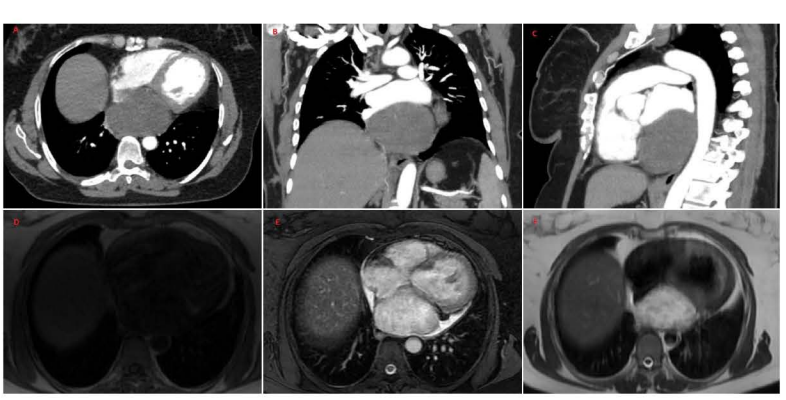


Figure 3

CT (A, B, C) and MRI (D, E, F) images of the patient. CT and MRI demonstrated no evidence of the invasion of the inferior vena cava, left atrium, left ventricle and pulmonary veins. T1-isointense (D) and T2-hyperintense (E, F) pericardial mass.

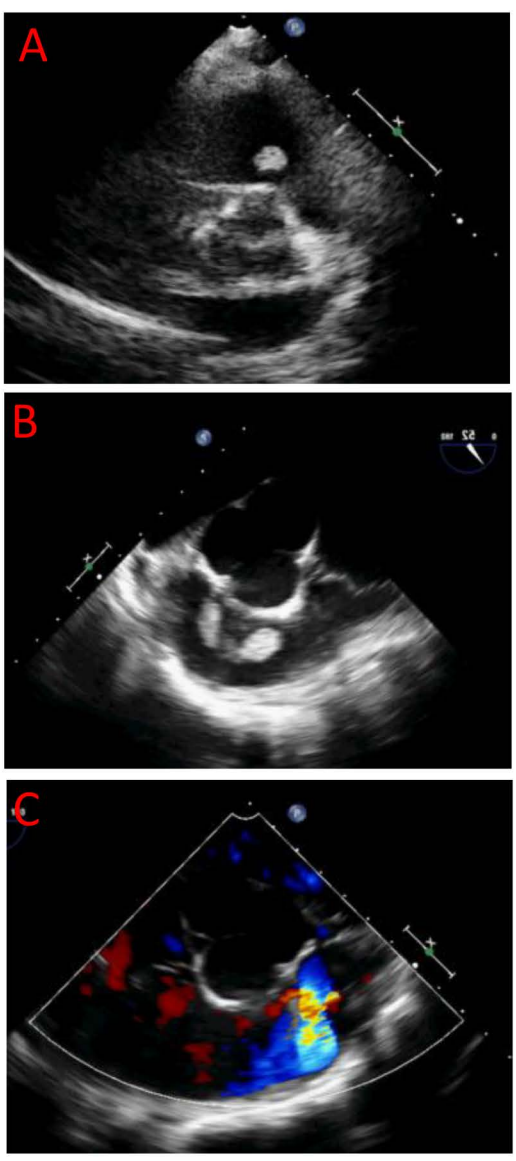
PS-08

A CASE OF TRANSTHYRETIN AMYLOIDOSIS WITH VAL30MET MUTATION MANIFESTED ISOLATED PULMONARY VALVE INFILTRATION

Emre YALÇIN[1], İbrahim KAMACI[2], Hacer Durmuş TEKÇE[2], Fatma Yeşim PARMAN[2], Cafer ZORKUN[1], Berrin UMMAN[1], Derya BAYKIZ[1]

[1]Department of Cardiology, Istanbul University, Istanbul, Turkey
[2]Department of Neurology, Istanbul University, Istanbul, Turkey

Introduction: Misfolded proteins, driven by genetic and environmental factors form extracellular amyloid fibrils that induce cytotoxicity. These deposits target multiple organs particularly the heart with nine proteins identified as causative agents in cardiac amyloidosis(1–4).



Case: A patient known mutant TTR amyloidosis with Val 30Met mutation with familial polyneuropathy presented with gastrointestinal bleeding. Endoscopy revealed a forrest 3 ulcer in the corpus, a forrest 2c ulcer in the antrum, and erosive pangastritis. While being monitored in the intensive care unit, MSSA bacteremia and candiduria developed. Transthoracic echocardiography (TTE) revealed a 2.1 × 1.1 cm pedunculated mass with regular borders intermittently protruding from the pulmonary valve into the pulmonary artery (Figure A). Additionally, a second mobile mass measuring 0.9 × 0.6 cm was observed at the tip of the primary mass (Figure A). The patient was started on cefazolin for presumptive infective endocarditis (IE) and fluconazole for possible candidal urinary infection. Transesophageal echocardiography (TEE) was performed for detailed evaluation, revealing a mobile, pedunculated mass protruding into the right ventricular outflow tract (RVOT) and pulmonary artery (Figure B,D). The mass measured up to 3.0 cm in length, with its most prominent hyperechoic and mobile portion (measuring 1.7 × 1.2 cm) located at the tip (Figure B,D). The mass exhibited regular borders and soft tissue density. Grade 3 pulmonary regurgitation was observed in this region (Figure C). Given the differential diagnoses of vegetation, thrombus or mass with unknown origin, further evaluation was planned. FDG-PET revealed no pathological hypermetabolic activity suggestive of IE. Cardiac MRI demonstrated a highly mobile 1.8 cm lesion on the atrial aspect of the pulmonary valve, which exhibited contrast enhancement (Figure E). The presence of contrast uptake ruled out thrombus, leading to consideration of IE-related vegetation and papillary fibroelastoma in the differential diagnosis. Given the lesion's big size, vegetation was deemed more likely than papillary fibroelastoma. The patient underwent surgical excision of the mass and was performed bioprosthetic pulmonary valve replacement after multidisciplinary evaluation. Surgical pathology demonstrated amyloid deposition painted by congo red. Postoperative course was uncomplicated with normal prosthetic valve function (Figure F). The patient was discharged on tafamidis and anticoagulant therapy.

Discussion: Although mTTR amyloidosis represents a rare form of amyloidosis, certain mutations demonstrate higher prevalence in specific geographic regions and populations. In European countries, the estimated incidence is generally 1:100,000 (5). The most common mutations detected in mTTR amyloidosis are Thr60Ala, Val30Met and Val122Ile. While Thr60Ala and Val122Ile mutations predominantly manifest with cardiac involvement, the Val30Met mutation typically presents with neuropathic features as the primary clinical manifestation (6). In patients with suspected cardiac amyloidosis (CA), key diagnostic clues include: severe aortic stenosis, autonomic dysfunction, peripheral neuropathy, proteinuria, atrioventricular conduction abnormalities, chronic troponin elevation, apical sparing with reduced longitudinal strain, and disproportionately low QRS amplitudes on ECG (7). This case of neuropathic mTTR amyloidosis demonstrates isolated pulmonary valve infiltration, a remarkable presentation lacking typical cardiac amyloidosis findings.

Conclusion: mTTR amyloidosis is increasingly recognized with improved awareness. It may present with various cardiac and extra cardiac manifestations. Early diagnosis is critical to reduce cardiac morbidity and mortality. The importance of multimodality cardiac imaging has become more evident in the diagnosis and treatment. To our knowledge, this represents the first documented case of mTTR amyloidosis presenting with isolated pulmonary valve involvement, without characteristic myocardial infiltration.

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PS-09

PERIKARTTA HIPEREKOJENITE... SESSIZ DISEKSIYON IPUCU OLABILIR MI?

Mücahit Yetim[1], Ceren Yağmur Doğru Yılmaz[1], Osman Karaaslan[1]

1.Hitit Üniversitesi Çorum Erol Olçok Eğitim Araştırma Hastanesi, Kardiyoloji A.B.D

Özet

Akut aortik sendromlar yüksek mortalite ve morbidite oranı sebebi ile dikkatle ele alınması gereken bir kliniktir. Yaş ilerledikçe sıklığı artar ve akut vakalarda özellikle tip A akut aortik sendromlarda acil cerrahi müdahale gerekir. Gecikmiş müdahale mortalite ve morbiditeyi arttırır. Bu sebeple özellikle risk faktörü olan hastalarda şüphe duymak ve görüntülemede küçük ipuçlarını bile gözden kaçırmamak kıymet taşır. Bu yazımızda göğüs ağrısı şikâyeti olmayan, kontrolsüz hipertansiyon ve kısa süreli hafıza kaybı ile başvuran hastada perikartta hiperekojen görünüm ve eşlik eden sıvana tarzı effüzyondan şüphelenerek tespit edilen aort diseksiyonu ve intramural trombüs vakasını sunduk.

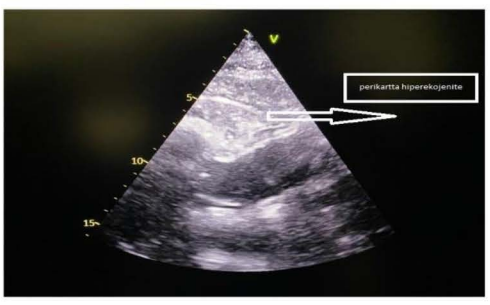
Vaka Sunumu

76 yaşında erkek hasta tansiyon düzensizliği sebebi ile polikliniğe başvurdu. Özgeçmişinde mesane tümörü sebebi ile operasyon, 60 paket/yıl sigara tüketimi, hipertansiyon ve kronik böbrek yetmezliği öyküsü mevcuttu. Göğüs ağrısı, nefes darlığı gibi kardiyak yakınma tarif etmedi. Evdeki tansiyon takipleri ortalaması 150/100 mmHg bandındaydı. Hasta detaylı sorgulandığında 1 hafta önce unutkanlık ile seyreden geçici iskemik atak geçirdiği öğrenildi. Hastanın elektrokardiyografisi sinüs ritminde olup sol ventrikül hipertrofisi ile uyumluydu. Nabız 110 atım/dakika idi. Yapılan transtorasik ekokardiyografide (TTE) 1.derece mitral yetmezlik, eser derece aort yetmezliği görüldü. Nabız 110 atım/dakika idi. Yapılan transtorasik ekokardiyografide (TTE) 1.derece mitral yetmezlik, eser derece aort yetmezliği görüldü. İnterventriküler septum 1,5 cm, sol ventrikül arka duvarı 1,4 cm, asendan aort 3,5 cm ölçüldü. Asendan aort içinde şüpheli bir flap görüntüsü olmakla beraber ekojenite yeterli değildi. Hastanın parasternal uzun aks görüntülemesinde perikart boşluğunda yaklaşık 1 cm kalınlığında diffüz hiperekojen görünüm ve hiperekojenite etrafında minimal perikardiyal effüzyon görüldü(Resim 1). Hastaya torakoabdominal bilgisayarlı tomografi anjiyografi(BTA) çekildi. Çekilen tomografide tübüler aorta, abdominal aortada ve sağ A.iliaca externa'da diseksiyon flebi, asendan aortta itibaren desendan aortaya devam eden yaygın plak ve intramural hematom saptandı(Resim 2).Mevcut durum BTA ile açıklanamaması üzerine hastaya TEE yapıldı. TEE'de aterosklerotik penetran ülser ve bunlara eşlik eden intramural hematom ve diseksiyon flebi izlendi(Resim 3-4). Hasta kalp damar cerrahiye danışıldı. Kalp damar cerrahi medikal tedavi kararı verdi Hastaya antitrombotik tedavi başlandı. Yoğun statin tedavisi ile takip edilmesine karar verildi. Takiplerinde stabil seyrettiği görüldü.

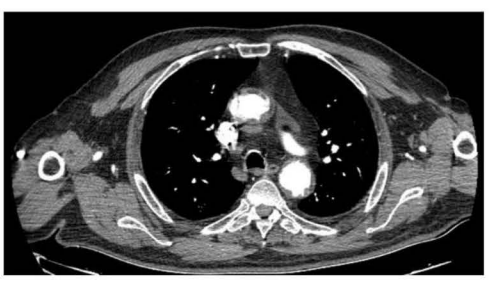
Tartışma

Akut aortik sendromlar aort duvar bütünlüğünün bozulması ile ortaya çıkar; rüptür ve uç organ perfüzyonunun bozulması ile sonuçlanabilir(1). Yüksek mortalite oranına sahiptir. Akut aortik sendromlar penetran aterosklerotik ülser veya intramural hematom şeklinde başlayıp diseksiyon şeklinde ilerleyebilir. Klinik tablolar birbiri ile iç içe olabilir. Erken tanı ve şüphe mortalitenin önüne geçmek için ilk basamaktır. Sıklığı yıllık yaklaşık 3,5-6/100.000'dir(2). Pek çok sebebi olmakla beraber altta yatan patogenez sıklıkla hipertansiyon ve aterosklerozdur. Genetik yatkınlık, vaskülitler ve bağ doku hastalıkları da akut aortik sendromlar ile karşımıza çıkabilirler. Yaşla beraber sıklığı artar. Genellikle yırtıcı, bıçak saplar tarzda göğüs ağrısı ile prezente olur ancak ağrısız veya atipik ağrı ile karşımıza çıkabilirler. A. Brachiocephalica'ya ilerlediyse nörolojik semptomlar ya da üst ekstremite semptomları, A. İliaca'ya ilerlediyse alt ekstremite ağrısı olabilir (3). Fizik muayenede aort kapakta üfürüm duyulabilir, ekstremiteler arasında nabız farkı olabilir. Elektrokardiyogramda ST segment elevasyonu saptanabilir. TTE tanı koymada ilk basamak tetkiktir ancak sensitivite ve spesifitesi düşüktür. Bu nedenle mutlaka BTA ve TEE gibi görüntüleme yöntemlerinin kullanılması önerilmektedir. Asendan aort diseksiyonu koroner arter diseksiyonu, tamponad, akut kapak disfonksiyonu ile seyredebilir ve TTE ile tamponad, kapak disfonksiyonu, asendan aort içinde diseksiyon flebi görülebilir. Ancak ideal görüntüleme bilgisayarlı tomografidir(4). Manyetik rezonans görüntüleme de tanı koymada faydalanılabilecek tetkikler arasındadır.

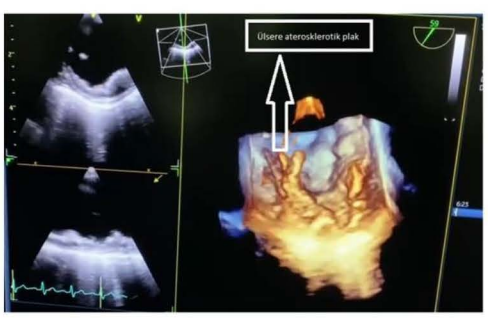
Tanımlama yaparken akut aortik sendromun meydana geldiği anatomik bölgeye göre temelde 2 tip sınıflama kullanılır(5, 6). DeBakey sınıflaması tip I-III olarak 3 gruptur, Stanford sınıflaması tip A ve B olarak iki gruptur. Asendan ve desendan aortu içeren patolojiler DeBakey tip-I, sadece asendan aortu ilgilendiren patolojiler DeBakey tip-II, sadece desendan aortu ilgilendiren patolojiler DeBakey tip-III olarak tanımlanır. Stanford sınıflamasına göre asendan aortu içeren tüm patolojiler tip A iken asendan aortu içermeyen desendan aortu ile sınırlı kalan patolojiler tip B olarak tanımlanır. Tip A akut aortik sendromlarda acil cerrahi girişim gerekirken tip B akut aortik sendromlarda endovasküler tedavi ön plandadır.



Resim-1:Parasternal uzun aksta perikartta hiperekojen görünüm



Resim-2: BTA'da intramural hematom ve diseksiyon flebi görüntüsü



Resim-3:TEE'de aterosklerotik plak

Resim-4 : TEE'de penetran aterosklerotik ülser ve intramural hematom

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PS-10

DIAGNOSIS AND SUCCESSFUL TREATMENT OF ADVANCED-STAGE LUNG CANCER PRESENTING WITH CARDIAC METASTASIS AND SUPERIOR VENA CAVA SYNDROME FOLLOWING A MULTIMODALITY APPROACH: A CASE REPORT

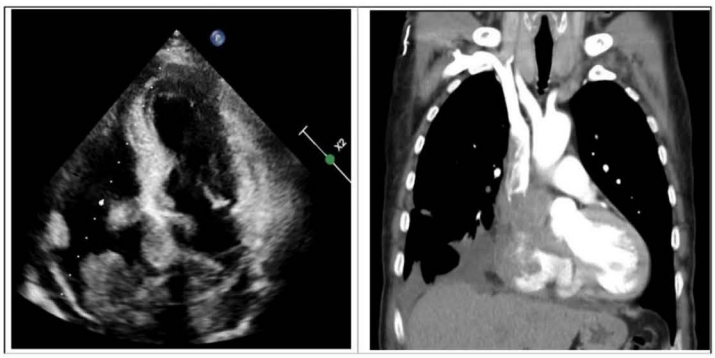
Tezel Kovancı, Emrah Bayam, Sedat Kalkan

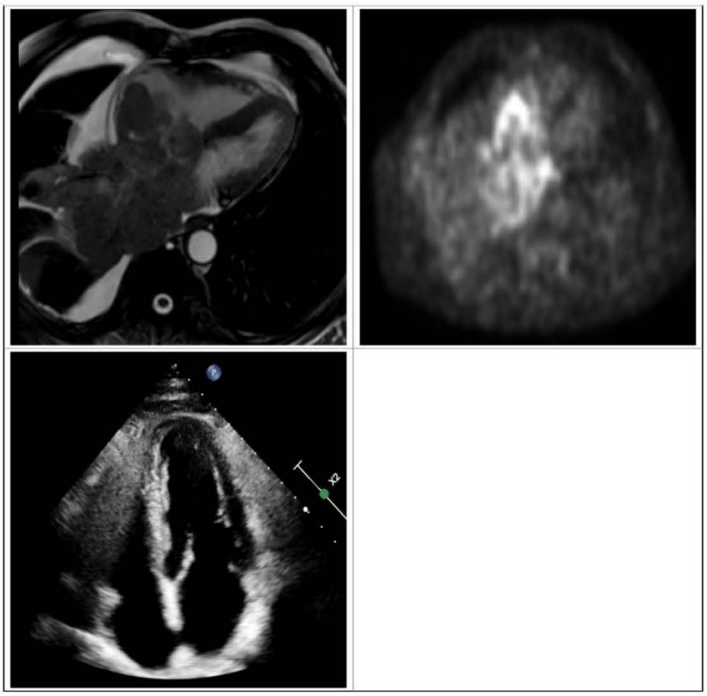
Introduction

Primary cardiac tumors are exceedingly rare in clinical practice. Metastatic involvement of the heart is 20–40 times more frequent, most often from lung and breast carcinomas. Solid metastases within the myocardium or cardiac chambers are rare and often discovered late. We present a case of small cell lung carcinoma with a giant cardiac metastasis manifesting as SVC syndrome and emphasize the role of multimodal imaging.

Case Presentation

A 63-year-old male presented with progressive facial flushing, dyspnea, and weight loss. Imaging revealed a large biatrial mass extending from the lung, encasing the right pulmonary artery, and causing SVC narrowing. Multimodal imaging (TTE, CT, MRI, PET-CT) confirmed small cell lung carcinoma with direct cardiac invasion. Urgent combined chemoradiotherapy resulted in complete resolution of the cardiac mass and significant reduction in the lung lesion within one year.





Discussion

Cardiac metastases, though more common than primary tumors, remain rare clinically. Multimodal imaging is essential for accurate diagnosis, treatment planning, and monitoring response. In SCLC with SVC syndrome, early systemic therapy can yield excellent outcomes, as in this patient.

Conclusion

This case illustrates a rare giant cardiac metastasis from SCLC presenting with SVC syndrome. Early recognition and multimodal imaging enabled successful treatment with chemoradiotherapy, resulting in complete cardiac mass resolution.

PS-11

SUCCESSFUL TRANSCATHETER TREATMENT OF CARDIAC ELECTRONIC IMPLANTABLE DEVICE (CEID)-RELATED MASSIVE TRICUSPID REGURGITATION USING EDGE-TO-EDGE REPAIR – TKD ABSTRACT

Ramazan Astam, MD[1]; Ersin Sarıcam, MD[2]; Melike Polat, MD[2]; Zeynettin Kaya, MD[2]; Erdogan Ilkay, MD[2]

[1]Department of Cardiology, Batman Training and Research Hospital, Batman, Turkey.
E-mail: drastan80@gmail.com
. ORCID: 0000-0001-7519-5197

[2]Medicana International Ankara Hospital, Cardiology Clinic, Ankara, Turkey

[3]Mediterranean Health Foundation Life Hospital, Cardiology Clinic, Ankara, Turkey

Background

Severe symptomatic tricuspid regurgitation (TR) with right heart failure is associated with significant morbidity and mortality. There are three types of TR, each with different etiologies: primary, secondary, and cardiac electric implantable device-related (CEID).

CEID-related TR has proposed some mechanism, including lead-induced mechanical disruption of the tricuspid valvular or subvalvular apparatus and pacing-induced electrical dyssynchrony.

We presented symptomatic a patient with a case CEID associated TR. We performed successful transcatheter treatment by using edge-to-edge repair.

Case Presentation

A 78-year-old woman patient admitted cardiology clinic because of dyspnea, abdominal swelling for 6 months. She underwent mitral valve replacement 8 years ago. Last year, she had a pacemaker (PPM) implantation due to sick sinus syndrome.

On examination, pulsating jugular venous distention with coincident liver pulsations, 3/6 holosystolic murmur at left lower sternal border were understood. The electrocardiogram showed sinus rhythm. Transthoracic echocardiography revealed normal left ventricular size and function, and massive TR. Systolic pulmonary arterial pressure were 35 mmHg, tricuspid annular size 44 mm (TV annular dilation). After then, we made transesophageal echocardiography and three-dimensional echocardiography for identifying the etiology of TR and whether lead position contributes to TR.

Although the main cause of progressive RV failure is pulmonary hypertension induced by left side disease, this patient's clinical deterioration coincides with recent pacemaker implantation and echocardiographic findings were compatible with CEID related TR. We did not see direct mechanical interference or damage of the TV apparatus (lead impingement on a TV leaflet, leaflet perforation, distortion of the valvular apparatus). However, we thought that secondary mechanism pacing-induced RV dysfunction/dilatation. Hence, CEID-associated TR should be considered.

In CEID-TR therapy, firstly, loop diuretics and aldosterone antagonists can improve the RV loading conditions and reduce the TR severity by reducing annular dilatation. Secondly, transvenous lead extraction could be done. But this patient had an old person and sinus node dysfunction (PPM dependence - 100% pacing burden). Because of refractory medical therapy, we decided on transcatheter treatment for massive TR because of high surgical risk.

Successful edge-to-edge repair of TR was achieved this patient edge to edge repair, 2 clips in the anteroseptal commissure, 1 clip in the posteroseptal commissure (TriClip device (Abbott Vascular). In procedure, we took care of pacemaker lead position through a pig tail catheter (Picture-1)

In TR after clipping, we saw a reduction of massive TR to second grade (picture 2, mild level TR). The clipping of the TV led to a reduction in vena cava inferior diameter from 38 mm to 21 mm. Mean tricuspid gradient was 2 mmHg. After 1 month following, the patient had an increase in functional capacity. The mild TR was stable. Vena cava inferior diameter was 19 mm.

Discussion

CIED-related tricuspid regurgitation (TR) represents a challenging clinical entity, often encountered in elderly patients with significant comorbidities who are not suitable candidates for surgery. In patients who remain symptomatic despite optimal medical therapy, are pacemaker-dependent, and have prohibitive surgical risk, transcatheter edge-to-edge repair (TEER) has emerged as a promising alternative in recent years. In our case, TEER with the TriClip device resulted in a substantial reduction in TR severity and significant clinical improvement. These findings support the role of TEER as a safe and effective therapeutic option for selected high-risk patients with suitable anatomy.

Conclusions

Edge-to-edge repair of the CEID-TV is feasible with a promising reduction in TR, which could result in clinical improvement.

Picture-1

PS-12

NATIVE VALVE ENDOCARDITIS AFTER CABG WITHOUT CLASSIC RISK FACTORS: A CASE CAUSED BY ENTEROCOCCUS FAECALIS

Muhammed Tekbil[1], Tolga Koşmaz[1], Selim Kul[1], Ali Rıza Akyüz[1]
[1]Ahi Evren Thoracic and Cardiovascular Surgery Training and Research Hospital

Introduction

Infective endocarditis (IE) is a life-threatening infection of the endocardial surface, typically involving heart valves. Although traditionally linked to predisposing factors such as prosthetic valves, congenital heart disease, or intravenous drug use, IE can rarely develop in native valves without such risk factors. Enterococcus faecalis, responsible for 10–15% of IE cases, is the predominant enterococcal species and is increasingly associated with healthcare-related infections.(1) Common sources include the genitourinary tract, abdominal infections, or gastrointestinal translocation.(2) Recognition of E. faecalis as a significant postoperative pathogen is crucial, particularly in atypical presentations.

Case Report

A 60-year-old male with a history of coronary artery disease, multiple percutaneous coronary interventions, and recent coronary artery bypass grafting (CABG) presented on postoperative day 60 with persistent lower back and chest pain. He was afebrile but had an early diastolic decrescendo murmur at the left lower sternal border. Laboratory results showed elevated C-reactive protein and minimal troponin elevation. Contrast-enhanced computed tomography revealed a splenic infarction, and abdominal ultrasonography confirmed splenomegaly.

Transthoracic echocardiography demonstrated mobile vegetation on the native aortic valve with severe aortic regurgitation; transesophageal echocardiography confirmed large, mobile vegetations. Blood cultures grew Enterococcus faecalis. The patient was admitted to the coronary care unit, initiated on targeted antibiotic therapy, and referred to cardiac surgery.

•Intraoperative findings revealed friable vegetations on all cusps of the native aortic valve. The vegetations were excised, and a mechanical prosthetic valve was implanted. Postoperative recovery was uneventful, and the patient completed a prolonged course of intravenous antibiotics.

Figures

Figure 1: Axial examination reveals a subcapsular hypodense area (infarction) in the anterior superior pole of the spleen, separated from the zebra pattern.

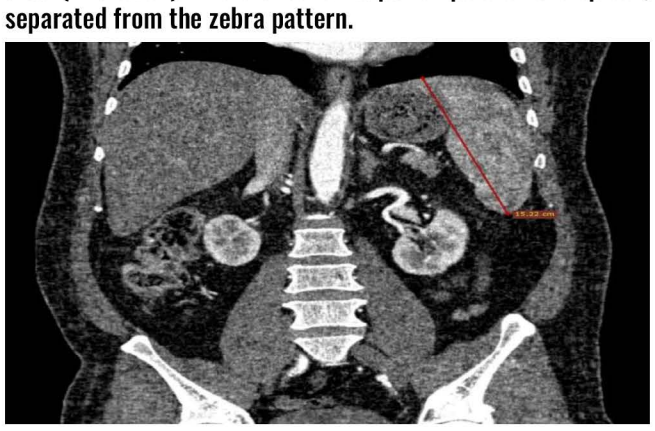


Figure 2: Splenomegaly in coronal sections

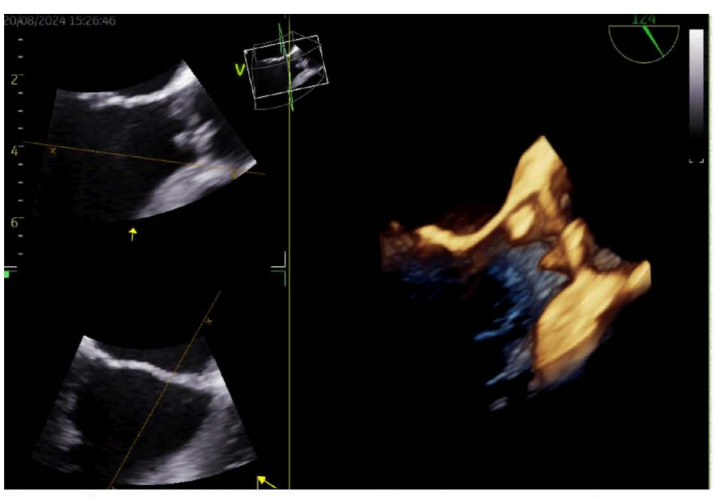


Figure 3: Image of vegetation on the native aortic valve in 3-dimensional transesophageal echocardiography (TEE)

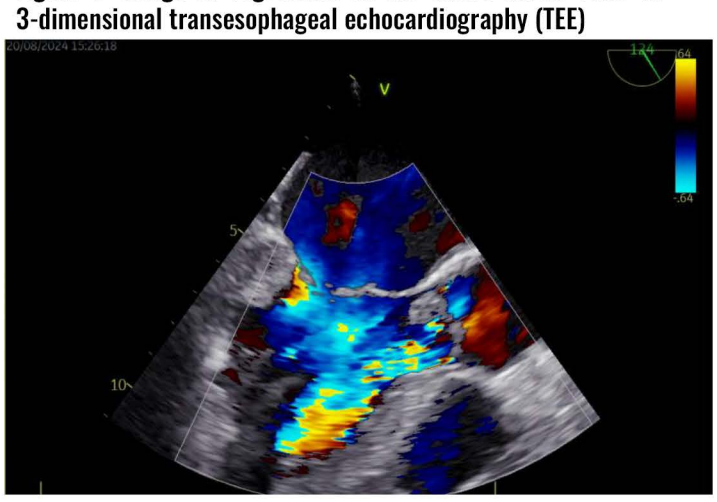


Figure 4: 2D TEE aortic insufficiency jet

Discussion

Enterococcus faecalis IE has undergone significant epidemiological shifts, with a rising incidence of healthcare-associated cases. It is more common in elderly patients with comorbidities, and while genitourinary sources are frequent, gastrointestinal translocation is also recognized. Postoperative E. faecalis IE on native valves without prosthetic involvement is rare.

This case is notable for several features: absence of classical risk factors, early postoperative onset following CABG, and an atypical presentation dominated by back pain rather than fever or constitutional symptoms. The splenic infarction served as a key diagnostic clue, prompting echocardiographic assessment and subsequent diagnosis. (3-4-5)

The literature highlights that E. faecalis IE carries a high risk of embolic complications, including splenic infarction. Prompt imaging and microbiological confirmation are essential for early diagnosis. Surgical intervention is warranted in cases with severe valvular dysfunction, large vegetations, or recurrent embolization, as demonstrated here.(6)

Conclusions

This case underscores the need for heightened clinical suspicion for IE in postoperative patients presenting with embolic events or unexplained systemic symptoms, even in the absence of classical risk factors. Enterococcus faecalis should be recognized as an important cause of both community-acquired and healthcare-associated IE, capable of affecting native valves shortly after cardiac surgery. Timely echocardiographic evaluation, blood cultures, and early surgical involvement are critical for favorable outcomes.

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TÜRK KARDİYOLOJİ DERNEĞİ

BİLİMSEL SEKRETERYA
Nish İstanbul A Blok Kat: 8 No: 47-48
Çobançeşme, Bahçelievler 34196 İstanbul / Turkey
T: +90 212 221 17 30/38 F: +90 212 221 17 54
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