Kocaeli Üniversitesi Tıp Fakültesi
Çocuk Sağlığı ve Hastalıkları
Anabilim Dalı

Kardiyoloji Bilim Dalı
Olgu Sunumu

31 Ocak 2017 Salı

Yandal Ar. Gör. Uzm. Dr. Okan Tuğral

ÇOCUK SAĞLIĞI VE HASTALIKLARI AD
SABAH TOPLANTISI SUNUMU

PEDİATRİK KARDİYOLOJİ BD

31/01/2017 - Salı

Dr. Okan Tuğral

OLGU

L.T.
15 yaş
Kız

Yakınma

- ✓ Çabuk yorulma
- ✓ Halsizlik
- ✓ Göğüs ağrısı
- ✓ Nefessiz kalma

Öykü

- ✓ Son 2-3 aydır olan **merdiven çıkarken yolda tempolu yürürken çabuk yorulma ve nefessiz kalma**
- ✓ **Göğüs ağrısı** 3 aydır ara ara olmakta

  -Daha çok efor sırasında kısa süreli 1-2 dk

  -İğne batar, bıçak saplanır tarzda

  -Yayılım göstermemekte

  -Kendiliğinden sonlanmakta

  -Bulantı ve karın ağrısı eşlik etmemekte
- ✓ Çarpıntı ve senkop öyküsü yok

Özgeçmiş-Soygeçmiş

- ✓ Prenatal-Natal-Posnatal dönemde herhangi bir sorun yaşamamış.
- ✓ Ailede 40 yaş öncesi ani ölüm öyküsü yok.
- ✓ **Babaannede, babada ve bir kuzeninde kalp hastalığı öyküsü mevcut.** Sonuçlarıyla ilgili rapor yok.
- ✓ Anne-Baba arasında akrabalık yok.

Fizik Bakı

- ✓ Vücut sıcaklığı: 36,5^{0}C Nabız: 82/dk
- ✓ SS: 18/dk SO_2: %99
- ✓KB: üst ekstremite: 120-125/70-75 mmHg

  alt ekstremite: 130-135/75-80 mmHg
- ✓ Ağırlık: 69 kg(90-97p) , Boy:159 cm (25-50p)
- ✓ GD iyi, Bilinç açık. Koopere-Oryante.
- ✓S1 - S2 N. AFN +/+.
- ✓**Sternum sol 2-3. İKA 2/6 sistolik ejeksiyon üfürümü**
- ✓Trill yok.
- ✓Diğer sistem bakıları normal.

Laboratuar

Hemogram

BK: 7200/mm3

MNS: 3100/mm3

Hgb: 11,4 gr/dl

Hct: %35

MCV: 71 fl

RDW: 16,2

Trombosit: 243000/mm3

AFR

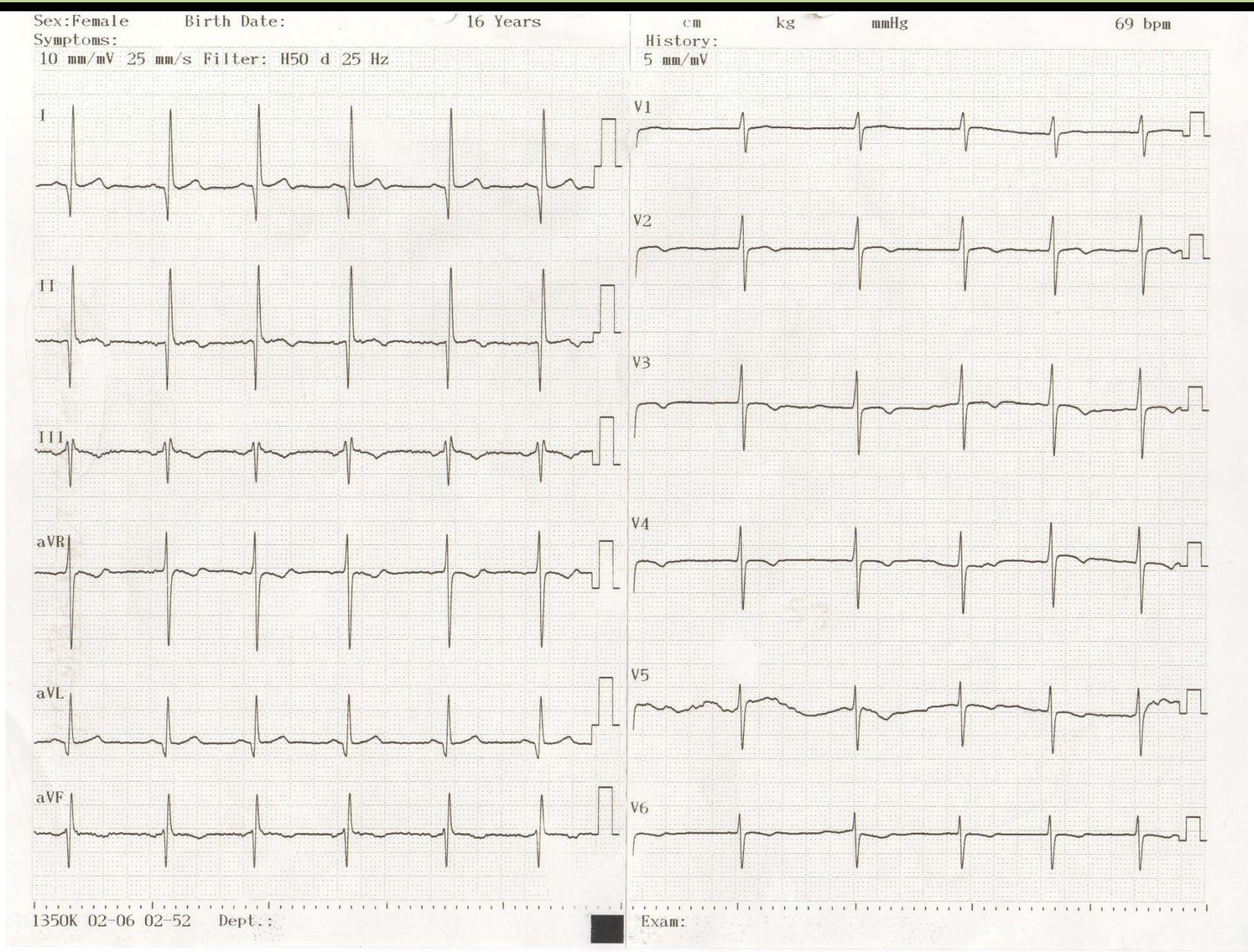
CRP: 0,6 mg/dl

Sedimentasyon: 22 mm/saat

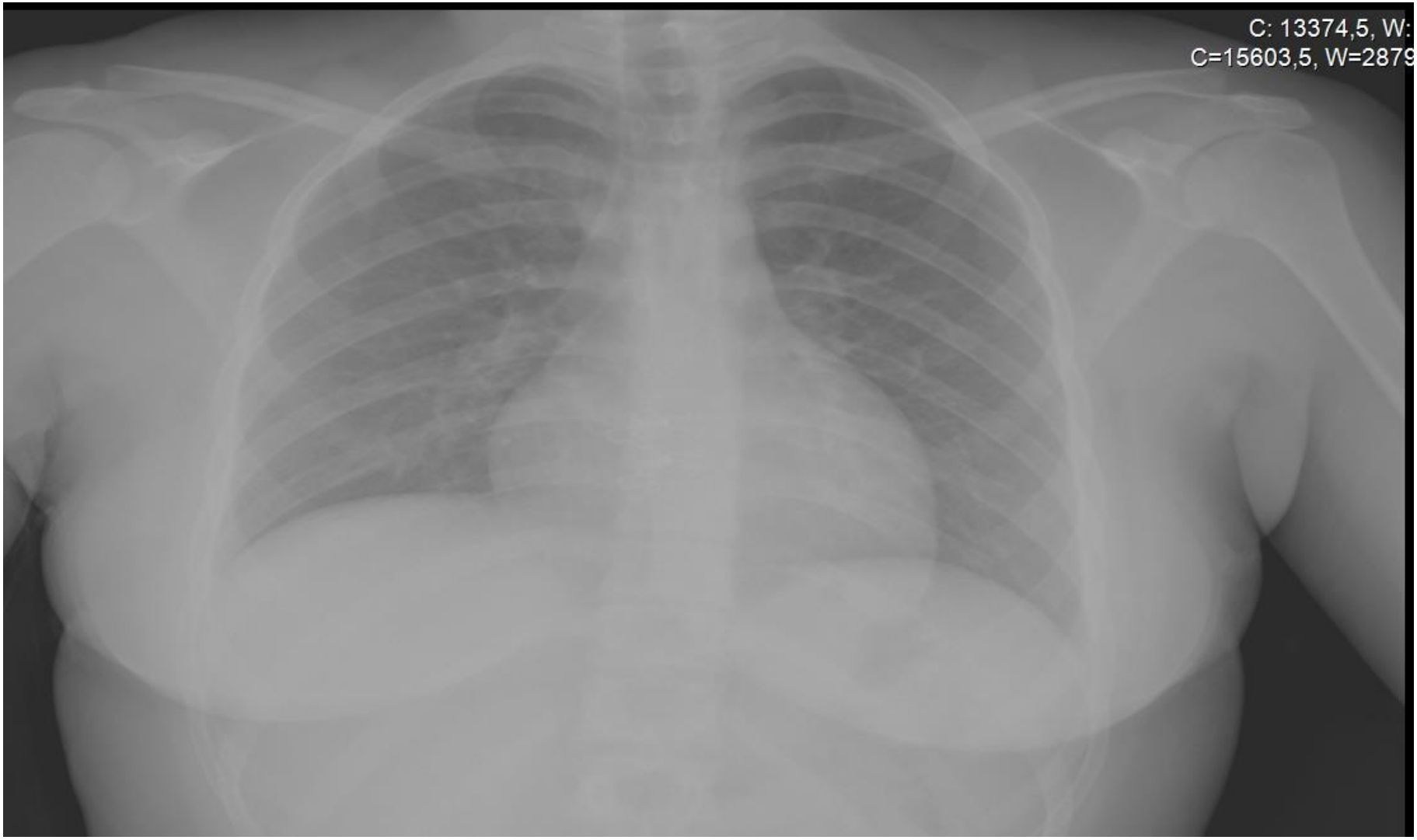
Laboratuar

Biyokimya	Kardiyak Biyobelirteçler
Kan şekeri: 88 mg/dl	CK-MB: 3,8 ng/ml (N:2,0-7,2)
Üre: 26 mg/dl	Troponin I: 0,017 ng/ml (N:0,010-0,023)
Kreatinin: 0,68 mg/dl	Troponin T: 0,014 ng/ml (N:0,010-0,017)
T. Protein: 7,2 gr/dl	Miyoglobin: 32 ng/ml (N:20-112)
Albumin: 4,1 gr/dl	Pro-BNP: 302 pg/ml
AST: 25U/L	
ALT: 17 U/L	
CK: 25 U/L	
T.Bil: 0,5 mg/dl	
İyonlar: normal	

EKG


Sex:Female Birth Date: 16 Years
Symptoms:
10 mm/mV 25 mm/s Filter: H50 d 25 Hz
cm kg mmHg 69 bpm
History:
5 mm/mV
I
II
III
aVR
aVL
aVF
V1
V2
V3
V4
V5
V6
1350K 02-06 02-52 Dept.:
Exam:

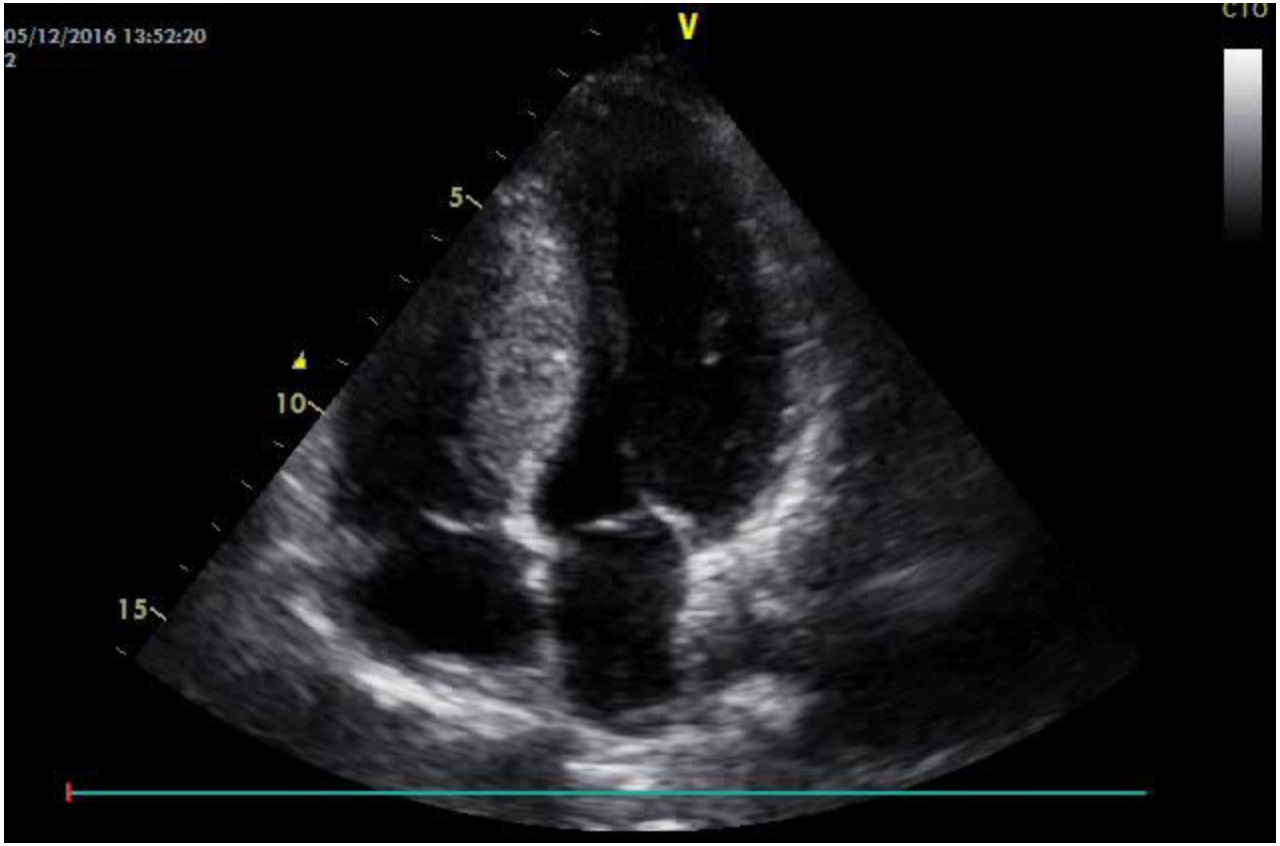
Telekardiyografi



OLASI TANI(LAR)?

İLERİ TETKİK???

EKOKARDİYOGRAFİ



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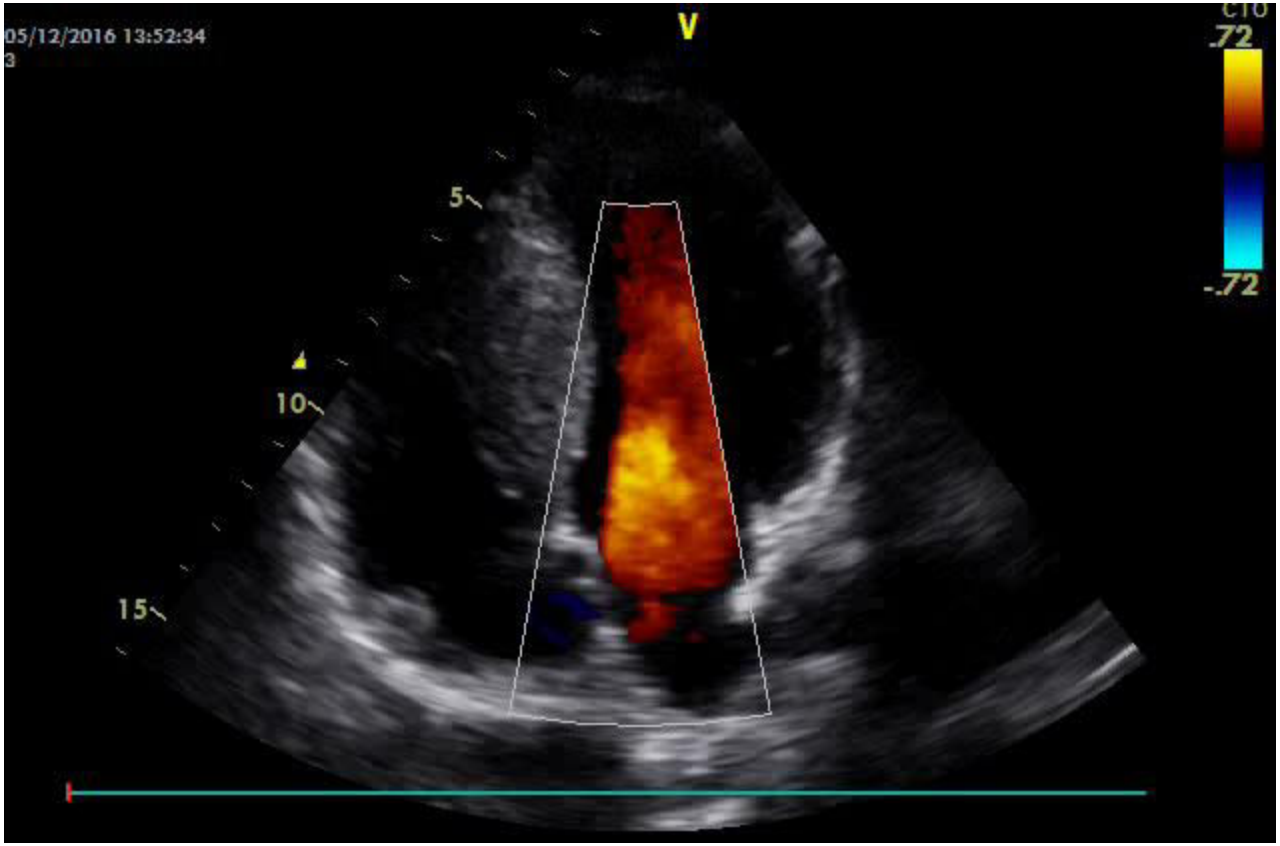

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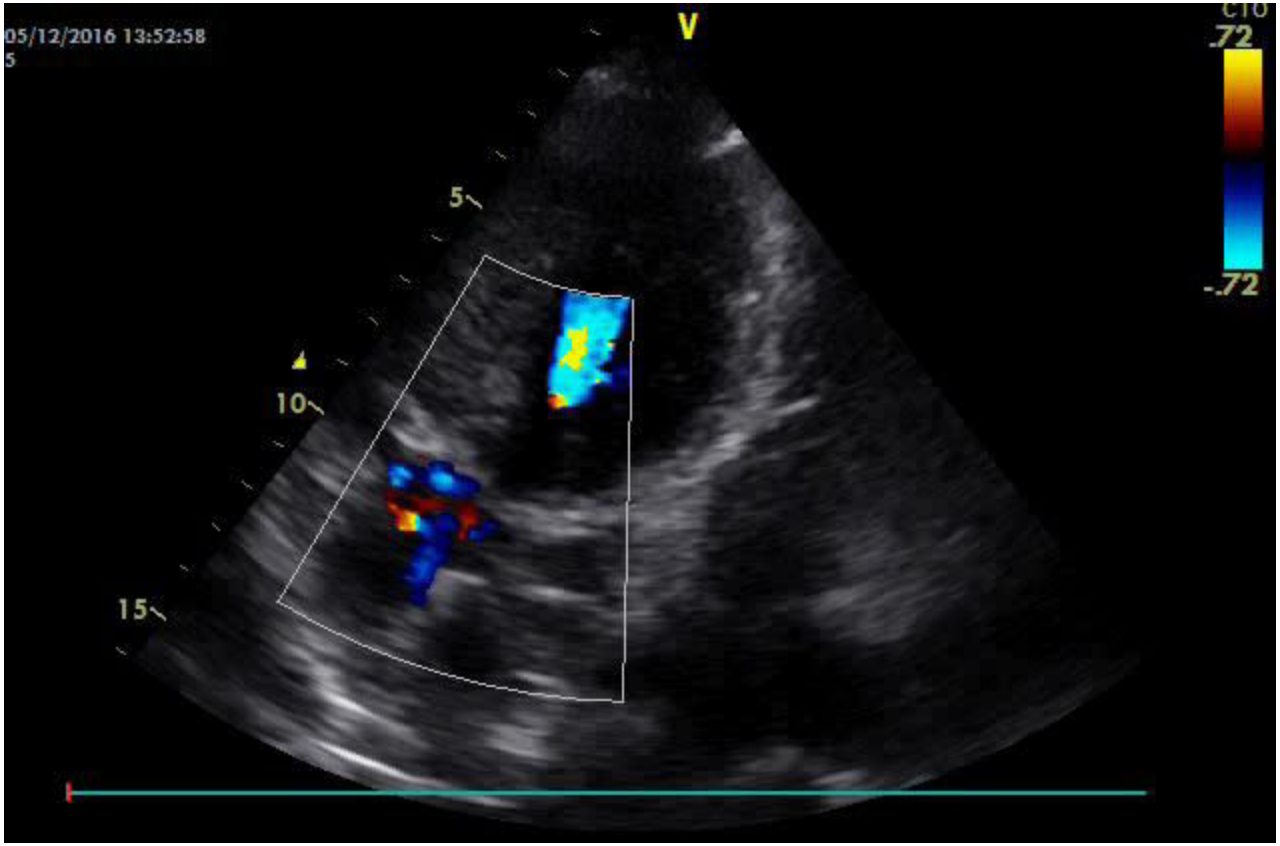

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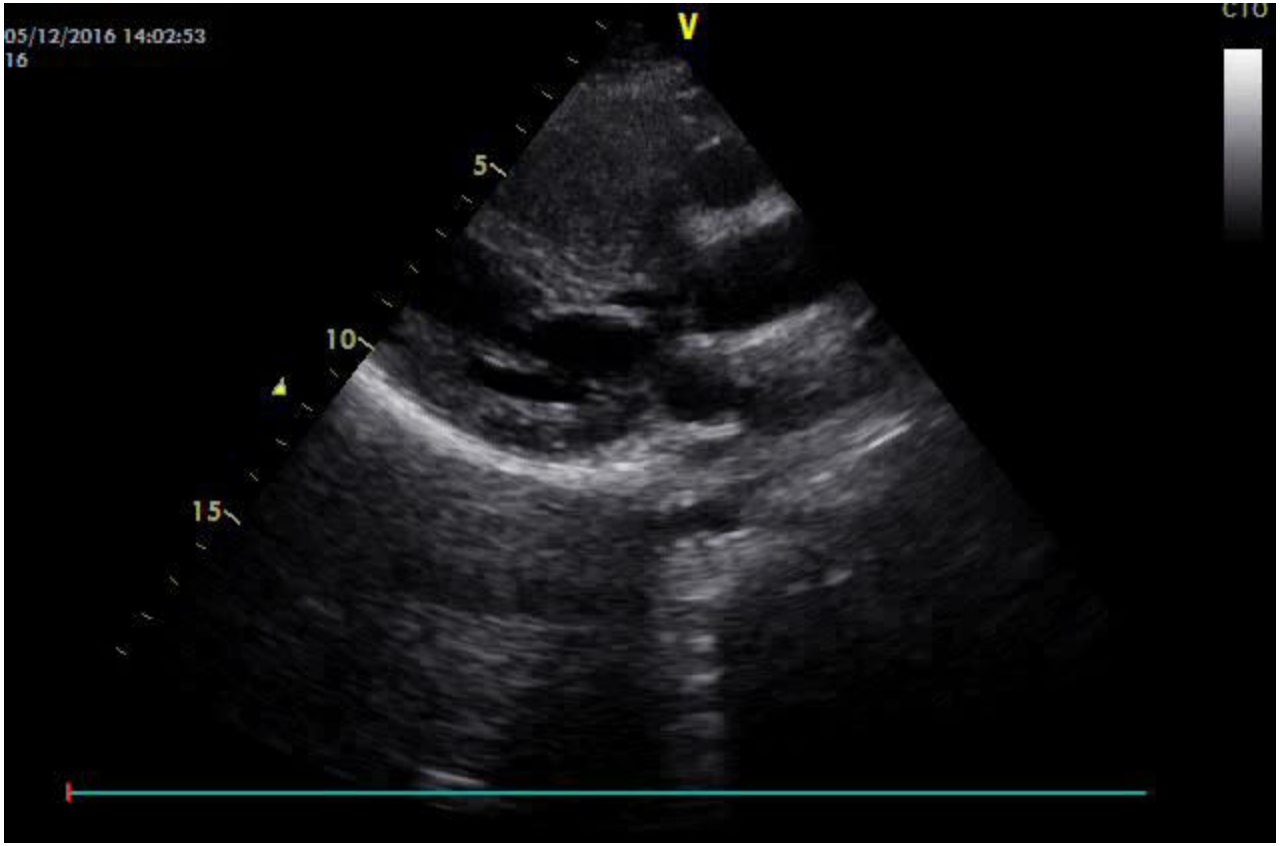

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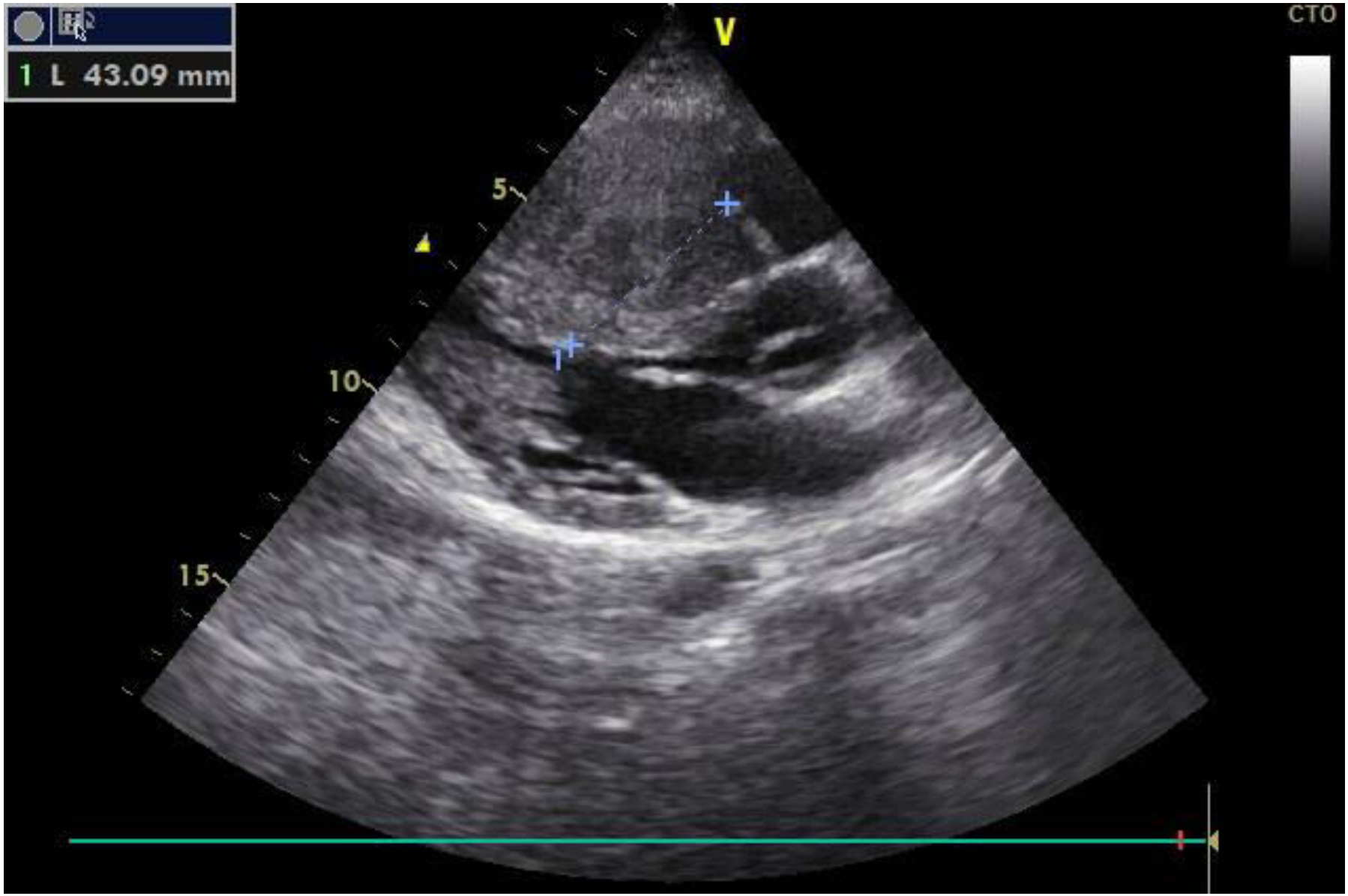

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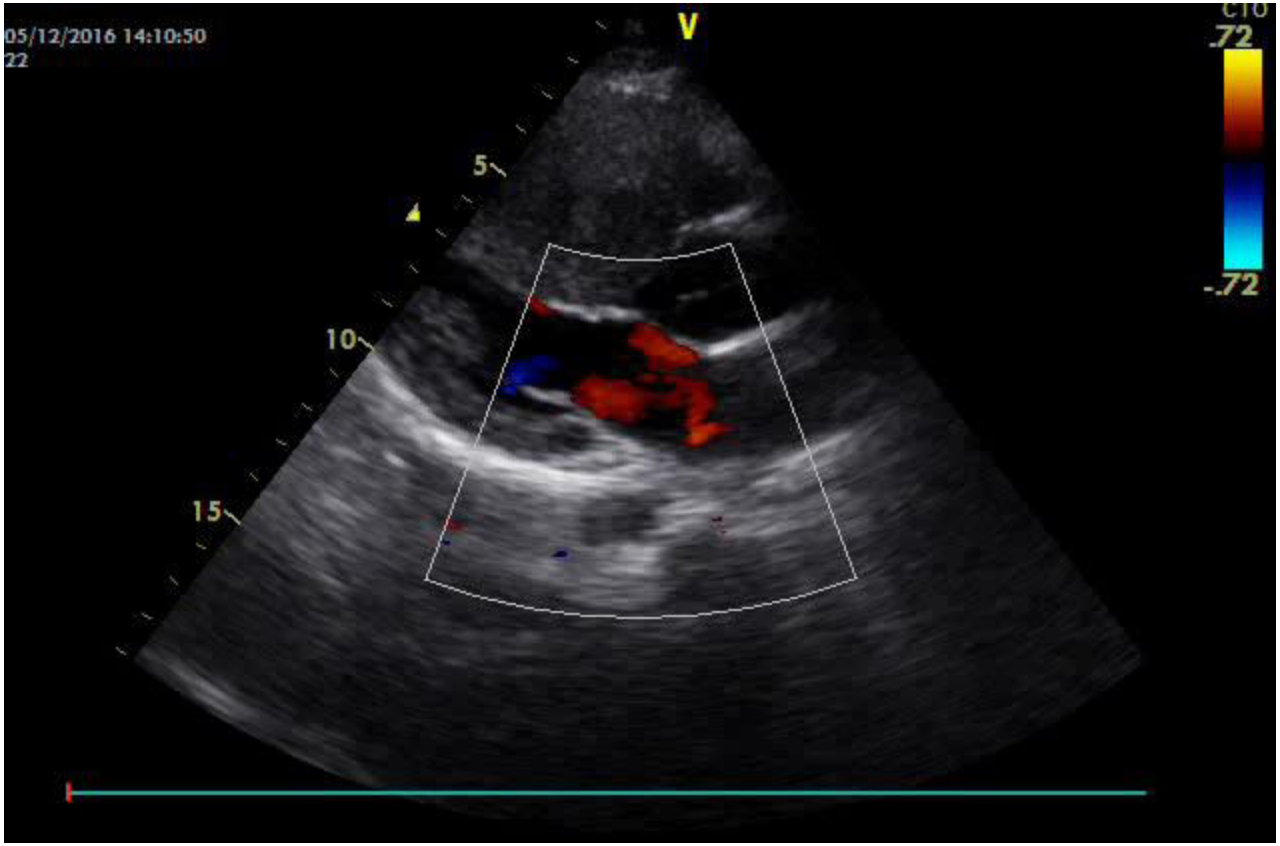

Klinik Seyir

- ✓Olgu **Hipertrofik Kardiyomiyopati** tanısı ile izleme alındı.
- ✓Aile bilgilendirildi.
- ✓Aile taraması yapıldı.

  ***-Babaannede, babada ve bir kuzeninde Hipertrofik KMP saptandı.

- ✓Hipertrofik KMP etyolojisini saptamak amacıyla metabolik tarama tetkikleri yapıldı.

  -Patolojik bulgu saptanmadı.

Cardiomyopathy Screen - Investigations

Name:

Unit Number:

DOB:

Biochemistry Lab:

Test (units)	Date	Result	Comments
Urea		(mmol/L)	Lithium Heparin (LH)
Creatinine		(mmol/L)	LH
Sodium		(mmol/L)	LH
Potassium		(mmol/L)	LH
Bicarbonate		(mmol/L)	LH (to calculate anion gap)
Chloride		(mmol/L)	LH (to calculate anion gap)
Total Protein		(g/L)	LH
Albumin		(g/L)	LH
ALP		(IU/L)	LH
AST		(IU/L)	LH
Bilirubin		(µmol/L)	LH
Calcium		(mmol/L)	LH
Phosphorus		(mmol/L)	LH
CRP		(mg/L)	LH
CK		(IU/L)	LH
Cholesterol			LH
Triglyceride			LH
Uric acid		(mmol/L)	LH
Troponin		(µg/L)	LH
Ammonia		(µmol/L)	LH 0.5 ml (ice sample –send within 10 min)
Carnitine Total and Free		(µmol/L) (µmol/L)	LH or white Guthrie card
Acyl Carnitine Profile		(µmol/L)	LH or white Guthrie card
Plasma Amino acids		(µmol/L)	LH (0.5 ml)
Blood gas			Capillary

Anion gap			Calculate make sure requesting HCO_3 and Chloride in Biochemistry sample
Glucose		(mmol/L)	BM Capillary or oxalate(yellow top)1/2ml
Lactate		(mmol/L)	BM capillary or oxalate
Thyroid Function tests		TFT Free T4 Free T3	LH
Immunology			
Auto antibodies			Plain top 1 ml

Haematology Lab

WCC			EDTA
Hb			EDTA
Platelet			EDTA
Blood Film			EDTA
ESR			EDTA 4mls
APTT			Citrate (Green top)
PT			Citrate (Green top)
Fibrinogen			Citrate (Green top)
Ferritin			Plain top 1 ml to Haematology

Urine tests

Urine organic acids			
Urine amino acids			
Urine Glucose			
Urine Protein			
Urine Ketones			
Urine-Mucopolysaccharide screen			
Urine-VMA			

Other

Standard Virology assay			Plain top 1 ml
Coxsackie *Viral titres and Enteroviruses**			*Plain top and discuss with virology for coxsackie ** for enterovirus send with stool sample
ECG			Extn 3325
Chest Radiograph			Extn 3027
ECHO			Viv – 3920

Klinik Seyir

✓Supraventriküler ve ventriküler aritmiler açısından 24 saatlik Holter ile monitorize edildi.

-Aritmi saptanmamakla birlikte 6 ayda bir holter EKG planlandı.

-Beta bloker tedavi başlandı.

✓ICD implantasyonu ve cerrahi açısından Pediatrik kardiyoloji-Pediatrik KVC konseyinde tartışıldı.

-Risk analizi yapılarak ICD implantasyonu kararı alındı.

✓Yakın klinik, ekokardiyografik ve holter EKG izlemine alındı.

HİPERTROFİK KARDİYOMİYOPATİ

Hipertrofik Kardiyomiyopati (HKMP)

- Sol ventrikül (LV) hipertrofisi ile karakterize olup sağ ventrikül hipertrofisi de eşlik edebilir.
- HKMP en sık genetik geçiş gösteren kalp hastalığı
- Olguların %50'si OD kalıtımla geçiş göstermekte
- Sarkomerin protein yapılarını kodlayan 13 genden birisindeki mutasyon sonucu oluşmakta
- Yıllık insidansı 100.000'de 0,3-0,5.
- Sıklığı 2/1000

Hipertrofik KMP

- En karakteristik bulgusu LV dilatasyonu olmadan LV hipertrofisi
- LV kavitesi normal ya da küçük
- Sağ ventrikül hipertrofisi eşlik edebilir
- LV hipertrofisini açıklayacak diğer doğumsal veya kazanılmış kalp hastalıkları olmamalıdır.
- Mitral kapak ön yaprakçığın sistolde olan öne hareketi (SAM) subaortik obstrüksiyona yol açabilir.

Hipertrofik Kardiyomiyopati (HKMP)

Familial	Non-familial
Familial, unknown gene	Obesity
▶ Sarcomeric protein disease	Infants of diabetic mothers
– β-myosin heavy chain	
– Cardiac myosin binding protein C	
– Cardiac troponin I	
– Troponin T	
– α-tropomyosin	
– Essential myosin light chain	
– Regulatory myosin light chain	
– Cardiac actin	
– α-myosin heavy chain	
– Titin	
– Troponin C	
▶ Z-disk protein disease	Amyloid (AL/pre-albumin)
– Myozenin 2	
– Telethonin	
– Muscle LIM protein	
– Vinculin/metavinculin	
– LIM binding domain 3	
– α-actinin 2	
– Cardiac ankyrin repeat protein	

Circulation

Epidemiology and Cause-Specific Outcome of Hypertrophic Cardiomyopathy in Children: Findings From the Pediatric Cardiomyopathy Registry

Steven D. Colan, Steven E. Lipshultz, April M. Lowe, Lynn A. Sleeper, Jane Messere, Gerald F. Cox, Paul R. Lurie, E. John Orav and Jeffrey A. Towbin

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Epidemiology and Cause-Specific Outcome of Hypertrophic Cardiomyopathy in Children

Findings From the Pediatric Cardiomyopathy Registry

Steven D. Colan, MD; Steven E. Lipshultz, MD; April M. Lowe, MS; Lynn A. Sleeper, ScD; Jane Messere, RN; Gerald F. Cox, MD, PhD; Paul R. Lurie, MD; E. John Orav, PhD; Jeffrey A. Towbin, MD

Background—Current information on the epidemiology and outcomes of hypertrophic cardiomyopathy (HCM) in children is limited by disease diversity and small case series.

Methods and Results—The Pediatric Cardiomyopathy Registry has collected prospective and retrospective data on children diagnosed with HCM since 1990. We identified the various causes of HCM in childhood and determined the relationship between outcomes, cause, and age at presentation. Of 855 patients <18 years of age with HCM, 8.7% (n=74) had inborn errors of metabolism, 9.0% (n=77) had malformation syndromes, 7.5% (n=64) had neuromuscular disorders, and 74.2% (n=634) had idiopathic HCM. Children with HCM associated with inborn errors of metabolism and malformation syndromes have significantly worse survival than the other 2 groups. Patients with idiopathic HCM diagnosed before 1 year of age (n=227) had worse survival from the time of diagnosis than those diagnosed after 1 year of age (n=407). Patients with idiopathic HCM who survived to at least 1 year of age, however, had an annual mortality rate of 1% that was similar regardless of whether they were diagnosed before or after 1 year of age.

Conclusions—In children, HCM is a diverse disorder with outcomes that depend largely on cause and age. Patients presenting before 1 year of age have the broadest spectrum of causes and the poorest outcome. In those children with idiopathic HCM who survive beyond age 1, however, survival is independent of age at diagnosis, with an annual mortality rate (1%) that is much lower than previously reported in children and is not different from has been found in population-based studies in adults. (***Circulation*. 2007;115:773-781.**)

Genetic syndromes, metabolic disorders, and neuromuscular diseases associated with hypertrophic cardiomyopathy in infants and children

Multiple congenital anomaly syndromes
Noonan syndrome
LEOPARD syndrome
Beckwith-Wiedemann syndrome
Cardiofacialcutaneous syndrome
Rubinstein-Taybi syndrome
Costello syndrome
Inborn errors of metabolism
Glycogen storage diseases:
Pompe disease (glycogen storage disease type II)
Forbes disease (glycogen storage disease type III, also known as Cori disease)
Phosphorylase kinase deficiency (glycogen storage disease type IX)
Carnitine deficiency:
Carnitine palmitoyltransferase type II deficiency
Carnitine-acylcarnitine translocase deficiency
Mucopolysaccharidoses:
Hurler syndrome (mucopolysaccharidosis type I)
Hunter syndrome (mucopolysaccharidosis type II)
Morquio syndrome
Scheie syndrome
Other lysosomal diseases:
Danon disease
Fucosidosis
I-cell disease
Fabry disease
Mannosidosis
Organic acidurias:
Methylmalonic aciduria
Barth syndrome (3-methylglutaconic aciduria, type II)
Glycosylation disorders (eg, phosphomannomutase 2 deficiency)
Congenital generalized lipodystrophy
Mitochondrial disorders
MELAS syndrome
Leigh syndrome
Complex I deficiency
Sengers syndrome
Defects of beta-oxidation enzymes:
Very long chain acyl-CoA dehydrogenase deficiency
Long chain 3-hydroxyacyl-CoA dehydrogenase deficiency
Multiple acyl-COA dehydrogenase deficiency
Combined respiratory chain deficiencies
Neuromuscular disorders
Friedreich ataxia
Myotonic dystrophy
Minicore (multicore) myopathy

Prepared with data from:

1. *Colan DC. Cardiomyopathies. In: Nadas' Pediatric Cardiology, 2nd ed, Keane JF, Lock JE, Fyler DC. (Eds), Saunders, Philadelphia 2006. p.432.*
2. *Colan SD, Lipshultz SE, Lowe AM, et al. Epidemiology and cause-specific outcome of hypertrophic cardiomyopathy in children: findings from the Pediatric Cardiomyopathy Registry. Circulation 2007; 115:773.*

UpToDate®

Hipertrofik KMP-Klinik

- Çeşitli klinik tablolara neden olabilir
  - Göğüs ağrısı (atipik/anjinal)
  - Presenkop/senkop
  - Çarpıntı
  - Kalp yetersizliği ile ilgili bulgular
    - Takipne
    - Beslenme güçlüğü
    - Büyüme geriliği
    - Çabuk yorulma, terleme
  - Aritmi (supraventriküler veya **ventriküler**)
    - ****Ani Kardiyak Ölüm****

Hipertrofik KMP-Klinik

Sol Ventrikül Hipertrofisi

↓

Diyastolik Disfonksiyon

↓

Sol Atriyal Dilatasyon

↓

Pulmoner Venöz Konjesyon

↓

Konjestif Semptomlar

Hipertrofik KMP-Klinik

- **Genç, atletik ve sağlıklı görünen bireylerde gelişen ani kardiyak ölümlerin 1/3 nedenidir.**
- İnfant ve adölesan dönemde ani kardiyak ölüm sıklığı artmaktadır.
- Ani kardiyak ölümler daha çok VT/VF ile ilişkili

Hipertrofik KMP-Klinik

- Genellikle asemptomatik veya minor semptomlara neden olmaktadır.
- Hastalar daha çok
  - Üfürüm (%53)
  - Aile taraması (%15)
  - Altta yatan sendromik bulgular (%6)
  - Konjestif kalp yetersizliği bulguları (%8)
  - Aritmik semptomlar (%2)
  - Özellikli olmayan semptomlar (%16) sonucu yapılan kardiyak değerlendirme sonucu tanı almaktadır.

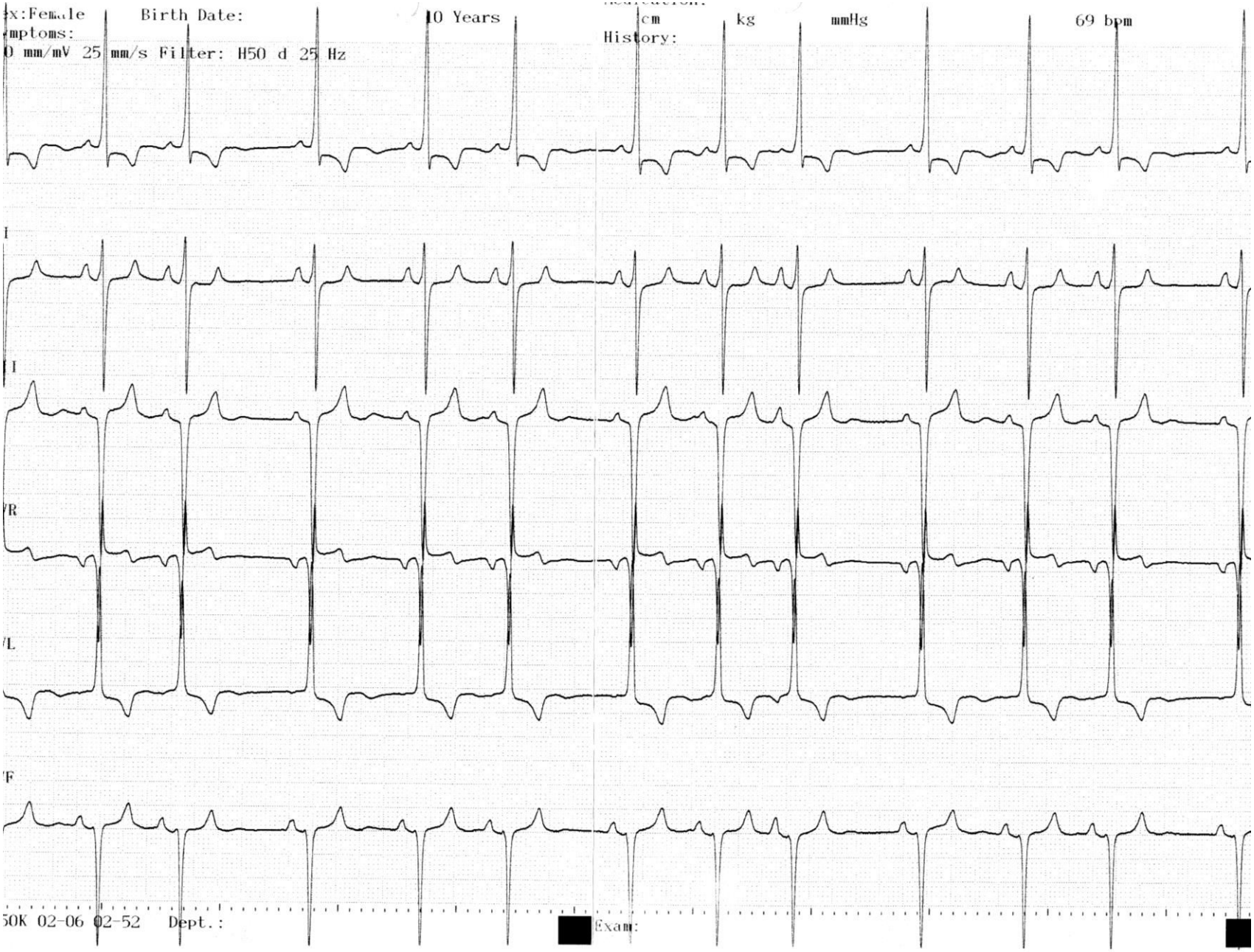
Hipertrofik KMP-Fizik Bakı

- Üfürüm
  - LVOT obstrüksiyon
    - Sol sternal kenarda 2-3. İKA'ta sistolik ejeksiyon
  - Mitral yetersizlik
    - Apekste orta-geç sistolik veya pansistolik
- Palpasyonla yaygın LV apikal vuru
- Konjestif kalp yetersizliği bulguları
- Dismorfik, sendromik bulgular

HKMP-Tanı

- HKMP açısından ayrıntılı aile öyküsü ve 3. kuşağı içeren aile ağacı
- Elektrokardiyografi
- Ekokardiyografi
- Metabolik hastalıklar açısından tarama
- Genetik hastalıklar açısından değerlendirme
- 24 saatlik Holter EKG
- Kardiyak MR görüntüleme
- Kardiyak kateterizasyon ve biyopsi

EKG


Birth Date:
10 Years
cm
kg
mmHg
69 bpm
History:
0 mm/mV 25 mm/s Filter: H50 d 25 Hz
I
II
VR
VL
VF
50K 02-06 02-52
Dept.:
Exam:

EKG

Sex:Female
Birth Date:
10 Years
cm
kg
mmHg
66 bpm
Symptoms:
History:
10 mm/mV 25 mm/s Filter: H50 d 25 Hz
V1
V2
V3
V4
V5
V6
1350K 02-06 02-52
Dept.:
Exam:

HKMP-İzlem ve Tedavi

- Tedavinin hedefleri
  - Semptomları azaltmak
  - LVOT obstrüksiyonunu azaltmak
  - LV fonksiyonlarını (diastolik/sistolik) korumak
  - Yaşam süresini uzatmak
  - ***Ani kardiyak ölümden korumak****

HKMP-İzlem ve Tedavi

- Medikal Tedavi
  - Beta blokerler –ilk tercih-
    - Propranolol, metoprolol, atenolol
  - Kalsiyum kanal blokerleri (nondihidropiridin)
    - Verapamil (***1 yaş altında kaçınılmalı)
    - Disopramid
- Cerrahi tedavi
  - Miyektomi
- Kalp transplantasyonu
- ICD yerleştirilmesi

European Heart Journal (2014) **35**, 2733–2779
doi:10.1093/eurheartj/ehu284

ESC GUIDELINES

2014 ESC Guidelines on diagnosis and management of hypertrophic cardiomyopathy

The Task Force for the Diagnosis and Management of Hypertrophic Cardiomyopathy of the European Society of Cardiology (ESC)

Table 7 **Major clinical features associated with an increased risk of sudden cardiac death in adults**

Risk Factor	Comment
Age	• The effect of age on SCD has been examined in a number of studies[73,82,99,208,244,372–374] and two have shown a significant association, with an increased risk of SCD in younger patients.[73,99] • Some risk factors appear to be more important in younger patients, most notably, NSVT,[69] severe LVH[375] and unexplained syncope.[99]
Non-sustained ventricular tachycardia	• NSVT (defined as ≥3 consecutive ventricular beats at ≥120 BPM lasting <30 seconds) occurs in 20–30% of patients during ambulatory ECG monitoring and is an independent predictor of SCD.[69,73,83,246,248,374] • There is no evidence that the frequency, duration or rate of NSVT influences the risk of SCD.[69,376]
Maximum left ventricular wall thickness	• The severity and extent of LVH measured by TTE are associated with the risk of SCD.[69,120,121,373] • Several studies have shown the greatest risk of SCD in patients with a maximum wall thickness of ≥30 mm but there are few data in patients with extreme hypertrophy (≥35 mm).[69,73,120,247,248,373,377,378]
Family history of sudden cardiac death at a young age	• While definitions vary,[73,120,372,377] a family history of SCD is usually considered clinically significant when one or more first-degree relatives have died suddenly aged <40 years with or without a diagnosis of HCM, or when SCD has occurred in a first-degree relative at any age with an established diagnosis of HCM.
Syncope	• Syncope is common in patients with HCM but is challenging to assess as it has multiple causes.[379] • Non-neurocardiogenic syncope for which there is no explanation after investigation is associated with increased risk of SCD.[73,83,99,244,246–248] • Episodes within 6 months of evaluation may be more predictive of SCD.[99]
Left atrial diameter	• Two studies have reported a positive association between LA size and SCD.[73,99] There are no data on the association between SCD and LA area and volume. Measurement of LA size is also important in assessing the risk of AF (see section 9.4).
Left ventricular outflow tract obstruction	• A number of studies have reported a significant association with LVOTO and SCD.[73,82,83,246,372,380] Several unanswered questions remain, including the prognostic importance of provocable LVOTO and the impact of treatment (medical or invasive) on SCD.
Exercise blood pressure response	• Approximately one third of adult patients with HCM have an abnormal systolic blood pressure response to exercise characterised by progressive hypotension or a failure to augment the systolic blood pressure that is caused by an inappropriate drop in systemic vascular resistance and a low cardiac output reserve.[241,381] • Various definitions for abnormal blood pressure response in patients with HCM have been reported[69,83,246,377]; for the purposes of this guideline an abnormal blood pressure response is defined as a failure to increase systolic pressure by at least 20 mm Hg from rest to peak exercise or a fall of >20 mm Hg from peak pressure.[237] • Abnormal exercise blood pressure response is associated with a higher risk of SCD in patients aged ≤40 years,[237] but its prognostic significance in patients >40 years of age is unknown.

HCM = hypertrophic cardiomyopathy; LA = left atrium; LVH = left ventricular hypertrophy; LVOTO = left ventricular outflow tract obstruction; NSVT = non-sustained ventricular

HKMP-İzlem

Risk factor
Non-sustained ventricular tachycardia
Syncope
Severe left ventricular hypertrophy
QT dispersion
Myocardial bridging
Voltage criteria for left ventricular hypertrophy on ECG
Heart rate variability
Young age at diagnosis
Inducible ventricular tachycardia by electrophysiological study

Recommendations on prevention of sudden cardiac death

Recommendations	Class[a]	Level[b]	Ref.[c]
Avoidance of competitive sports[d] is recommended in patients with HCM	I	C	395
ICD implantation is recommended in patients who have survived a cardiac arrest due to VT or VF, or who have spontaneous sustained VT causing syncope or haemodynamic compromise, and have a life expectancy of >1 year.	I	B	327,367, 391–393
HCM Risk-SCD is recommended as a method of estimating risk of sudden death at 5 years in patients aged ≥16 years without a history of resuscitated VT/VF or spontaneous sustained VT causing syncope or haemodynamic compromise.	I	B	73
It is recommended that the 5-year risk of SCD be assessed at first evaluation and re-evaluated at 1–2 year intervals or whenever there is a change in clinical status.	I	B	73
ICD implantation should be considered in patients with an estimated 5-year risk of sudden death of ≥6% and a life expectancy of >1 year, following detailed clinical assessment that takes into account the lifelong risk of complications and the impact of an ICD on lifestyle, socio-economic status and psychological health.	IIa	B	73,327, 393,396

Recommendations on prevention of sudden cardiac death

Recommendations	Class[a]	Level[b]	Ref.[c]
ICD implantation may be considered in individual patients with an estimated 5-year risk of SCD of between ≥4% and <6% and a life expectancy of >1 year, following detailed clinical assessment that takes into account the lifelong risk of complications and the impact of an ICD on lifestyle, socio-economic status and psychological health.	IIb	B	73,327, 393,396
ICD implantation may be considered in individual patients with an estimated 5-year risk of SCD of <4% only when they have clinical features that are of proven prognostic importance, and when an assessment of the lifelong risk of complications and the impact of an ICD on lifestyle, socio-economic status and psychological health suggests a net benefit from ICD therapy.	IIb	B	73,327, 393,396
ICD implantation is not recommended in patients with an estimated 5-year risk of SCD of <4% and no other clinical features that are of proven prognostic importance.	III	B	73,327, 393,396

ICD = implantable cardioverter defibrillator; VF = ventricular fibrillation; VT = ventricular tachycardia

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Hypertrophic Cardiomyopathy in Children, Adolescents, and Young Adults Associated With Low Cardiovascular Mortality With Contemporary Management Strategies

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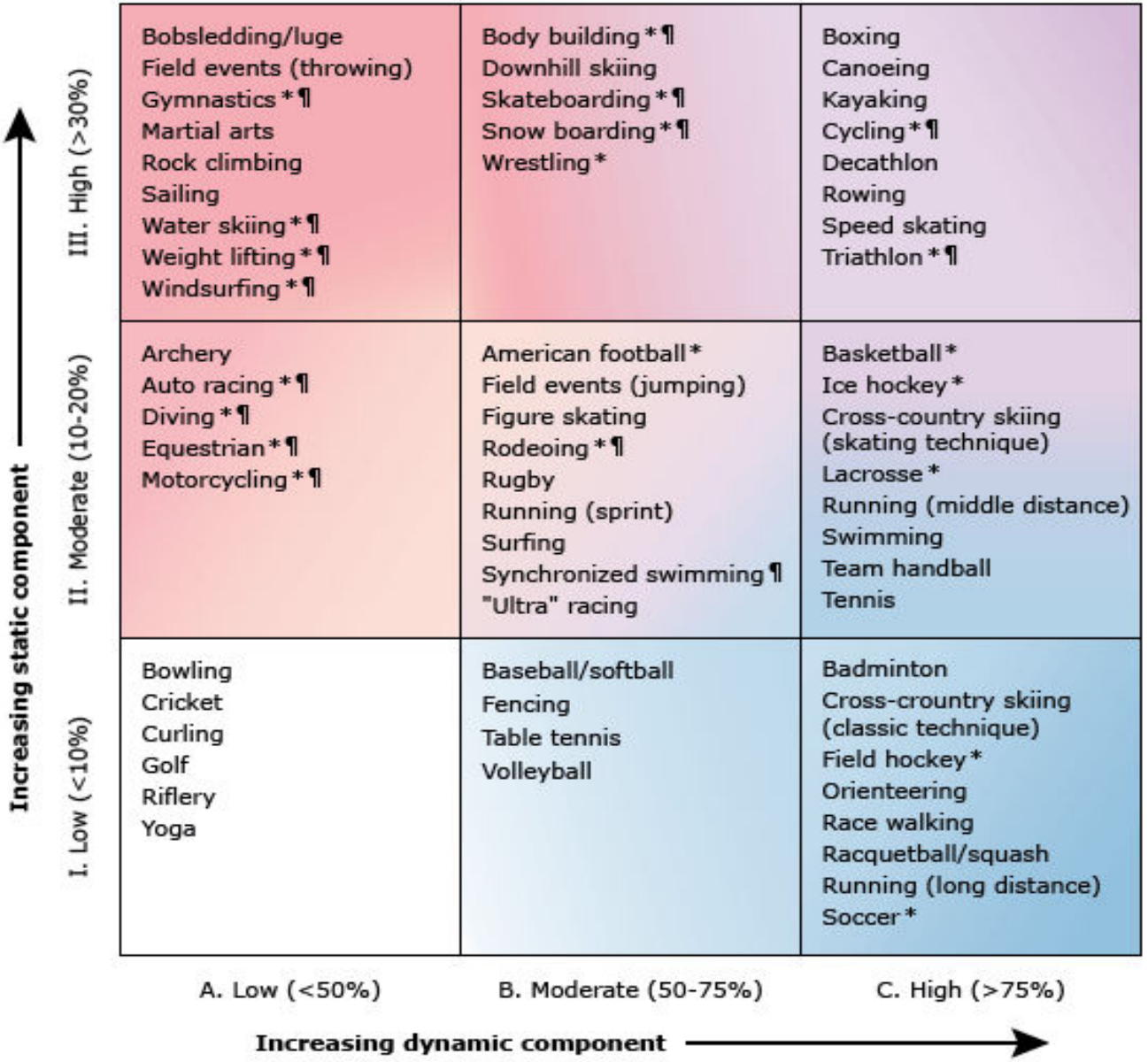
Background—Youthful age has been considered the time of greatest risk for patients with hypertrophic cardiomyopathy (HCM), largely because of the possibility of sudden death. The last 2 decades have witnessed more reliable identification of at-risk patients and utilization of implantable cardioverter-defibrillators for prevention of sudden death, and other contemporary treatment options. Whether such management advances have significantly altered the considerable mortality rate for young HCM patients remains unresolved.

Methods and Results—We studied long-term outcome in 474 consecutive HCM patients between 7 and 29 years of age presenting at 2 referral institutions. Over 7.1±5.1 years of follow-up (6.0 [3.0, 10.0]), 452 patients (95%) survived, with 95% experiencing no or mild symptoms. HCM-related death occurred in 18 patients (3%; 0.54%/y): arrhythmic sudden death (n=12), progressive heart failure and heart transplant complications (n=5), or postoperatively (n=1). In contrast, aborted life-threatening events occurred in 63 other high-risk patients (13%) with implantable cardioverter-defibrillator interventions for ventricular tachyarrhythmias (n=31), resuscitated out-of-hospital cardiac arrest (n=20), or heart transplant for advanced heart failure (n=12), 1.8%/y, 3-fold higher than HCM mortality. Five- and 10-year survival (considering only HCM deaths) was high (97% and 94%, respectively), virtually identical to that reported in middle-aged adult HCM patients (98% and 94%, P=0.23).

Conclusions—In a large hospital-based cohort of young HCM patients, representing an age group considered at greatest risk, low mortality rates can be achieved with the application of contemporary cardiovascular treatment strategies, largely because of reliable identification of high-risk patients who benefited from implantable cardioverter-defibrillators for sudden death prevention, thereby creating the opportunity for extended longevity and good quality of life. (*Circulation*. 2016;133:62-73. DOI: 10.1161/CIRCULATIONAHA.115.017633.)

Classification of sports based on peak static and dynamic components during competition



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