

CMR IN HEART FEALURE

FESC. DR. JAMIL BABAYEV

TO DECLARE

- **I AM CMR SPECIALIST**



**IN DER MEDIZIN GEHT ES NICHT NUR UM DAS, WAS WIR
WISSEN, SONDERN AUCH UM DAS, WAS WIR ERKENNEN**

**MEDICINE IS NOT ONLY ABOUT WHAT WE KNOW, BUT ALSO
ABOUT WHAT WE RECOGNIZE**

(ANONYM)



CASE (12.2023)

- YOUNG PATIENT (2004), M
- STUDENT
- DYSPNEA DURING WALKING, NIGHT CUFF, PRETIBIAL OEDEMA, FATIGUE, EXHAUSTION
- ANAMNESTIC: SYPTOMS WORSENERD LAST 3 MONTH, HE HAS SKALIOSIS (RECIEVES FISIOTHERAPY). FIRST VISIT TO DOCTOR AT 12.2023. NO SIGNS OF COPD OR ANOTHER LUNG DISEASES.
- ALCOHOL-, DRUGS-,SMOKING-, FAMILY HISTORY OF HD-

OBJECTIVES

- HEIGHT-176 SM
- WEIGHT-67 KG
- NORMOSTENIC BODY CONFIGURATION
- AUSCULTATION: MUFFLED HEART SOUNDS, DOBLE SIDES WET RALES ON BASE LUNG LOBULES
- GIS: WITHOUT ANY SPECIAL FEATURES

LABORATORY AND INSTRUMENTAL EXAM

- ECG: NSR. SINUS TACHYCARDIA. QRS-NORMAL.
- ECHO: ENLARGED LV, MODERATE LOW EF(GLOBAL), WALLSICKNES-NORMAL, MILD AI AND MI.
- NT PRO BNP: 2379 PG/ML
- CREATININ: 0,8 MG/DL
- CRP: 1,7 MG/DL
- ALT AND AST: NORMAL
- HEMOGRAMM: NORMAL

Diagnostic algorithm for heart failure

Suspected heart failure

- Risk factors
- Symptoms and/or signs
- Abnormal ECG

NT-proBNP \geq 125 pg/mL
or BNP \geq 35 pg/mL

Y

or if HF strongly suspected
or if NT-proBNP/BNP unavailable

Echocardiography

Abnormal findings

Y

N

N

DIAGNOSIS

- DILATED CARDIOMOPATY ←
- HIPERTROPHIC CARDIOMOPATY
- POSTPARTUM CARDIOMOPATY
- VALVE DISEASE
- ISHEMIC CARDIOMOPATY

Management of patients with HFrEF

- ACE-I/ARNI^a
- Beta-blocker
- MRA
- Dapagliflozin/Empagliflozin
- Loop diuretic for fluid retention (Class I)

LVEF $\leq 35\%$ and
QRS < 130 ms and
where appropriate

ICD

Non-ischaemic
(Class IIa) Ischaemic
(Class I)

LVEF $> 35\%$ or device
therapy not indicated
or inappropriate

SR and
LVEF $\leq 35\%$ and
QRS ≥ 130 ms

CRT-D^{b/-P}

QRS 130–149 ms QRS ≥ 150 ms
(Class IIa) (Class I)

If symptoms persist, consider therapies
with Class II recommendations

TREATMENT

- SACUBETRIL/VALSARTAN 24/26 MG 1-0-1

(1 MONTH LATER MAXIMAL DOSAGE OF 97/103 MG WAS REACHED)

- SPIRONOLACTONE 50 MG 0-1-0

- BISOPROLOL 2,5 MG 0-0-1

- EPAGLIFLOZIN 10 MG 1-0-0

- TORSEMID 10 MG 1-0-0-0 (STOPPED AFTER 1 MO)

"That's all Folks!"

**LOONEY
TUNES**

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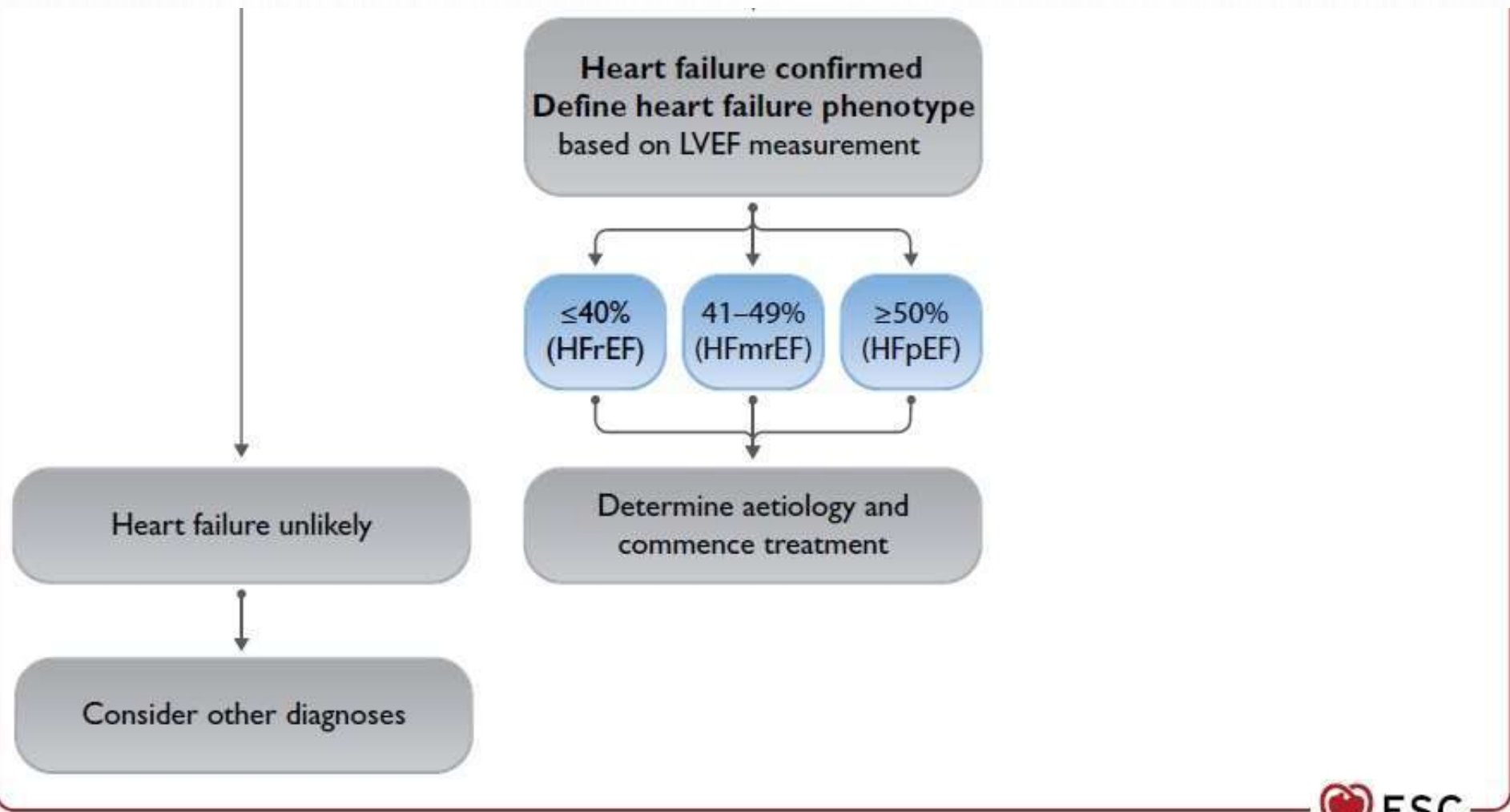


Table 5 Causes of heart failure, common modes of presentation and specific investigations

Cause	Examples of presentations	Specific investigations
CAD	Myocardial infarction Angina or “angina-equivalent” Arrhythmias	Invasive coronary angiography CT coronary angiography Imaging stress tests (echo, nuclear, CMR)
Hypertension	Heart failure with preserved systolic function Malignant hypertension/acute pulmonary oedema	24 h ambulatory BP Plasma metanephrines, renal artery imaging Renin and aldosterone
Valve disease	Primary valve disease e.g., aortic stenosis Secondary valve disease, e.g. functional regurgitation Congenital valve disease	Echo – transoesophageal/stress
Arrhythmias	Atrial tachyarrhythmias Ventricular arrhythmias	Ambulatory ECG recording Electrophysiology study, if indicated
CMPs	All Dilated Hypertrophic Restrictive ARVC Peripartum Takotsubo syndrome Toxins: alcohol, cocaine, iron, copper	CMR, genetic testing Right and left heart catheterization CMR, angiography Trace elements, toxicology, LFTs, GGT

Recommendations for specialized diagnostic tests for selected patients with chronic heart failure to detect reversible/treatable causes of heart failure

Recommendations	Class ^a	Level ^b
CMR		
CMR is recommended for the assessment of myocardial structure and function in those with poor echocardiogram acoustic windows.	I	C
CMR is recommended for the characterization of myocardial tissue in suspected infiltrative disease, Fabry disease, inflammatory disease (myocarditis), LV non-compaction, amyloid, sarcoidosis, iron overload/haemochromatosis.	I	C
CMR with LGE should be considered in DCM to distinguish between ischaemic and non-ischaemic myocardial damage.	IIa	C

Non-invasive testing

CTCA should be considered in patients with a low to intermediate pre-test probability of CAD or those with equivocal non-invasive stress tests in order to rule out coronary artery stenosis.

IIa

C

Non-invasive stress imaging (CMR, stress echocardiography, SPECT, PET) may be considered for the assessment of myocardial ischaemia and viability in patients with CAD who are considered suitable for coronary revascularization.^{90–93}

IIb

B

Exercise testing may be considered to detect reversible myocardial ischaemia and investigate the cause of dyspnoea.^{94–96}

IIb

C

2023 ESC Guidelines for Management of Cardiomyopathies: Key Points

Aug 30, 2023 | [Debabrata Mukherjee, MD, FACC](#)

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Authors: Arbelo E, Protonotarios A, Gimeno JR, et al., on behalf of the ESC Scientific Document Group.

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MULTIMODALITY IMAGING TO CHARACTERIZE THE CARDIAC PHENOTYPE (MORPHOLOGY AND FUNCTION)—INCLUDING TISSUE CHARACTERIZATION FOR NONISCHEMIC MYOCARDIAL SCAR DETECTION IS NECESSARY, IN COMBINATION WITH A DETAILED PERSONAL AND FAMILY HISTORY, CLINICAL EXAMINATION, ELECTROCARDIOGRAPHY, AND LABORATORY INVESTIGATIONS. HOWEVER, IMAGING RESULTS SHOULD ALWAYS BE INTERPRETED IN THE OVERALL CLINICAL CONTEXT, INCLUDING GENETIC TESTING RESULTS, RATHER THAN IN ISOLATION.

TISSUE CHARACTERIZATION BY CARDIAC MAGNETIC RESONANCE (CMR) IS OF VALUE IN DIAGNOSIS, MONITORING OF DISEASE PROGRESSION, AND RISK STRATIFICATION IN EACH OF THE MAIN CARDIOMYOPATHY PHENOTYPES.

Ischemic

Transmural infarct



Non-transmural (subendocardial) infarct



Non-ischemic

Midwall



HCM
RV overload



DCM



Myocarditis
Anderson-fabry
Chagas
Sarcoidosis

Subendocardial



Sarcoidosis

Epicardial



Sarcoidosis
Myocarditis
Anderson-fabry
Chagas

Global endocardial



Amyloidosis
Scleroderma
Post-cardiac
transplant

Transmural

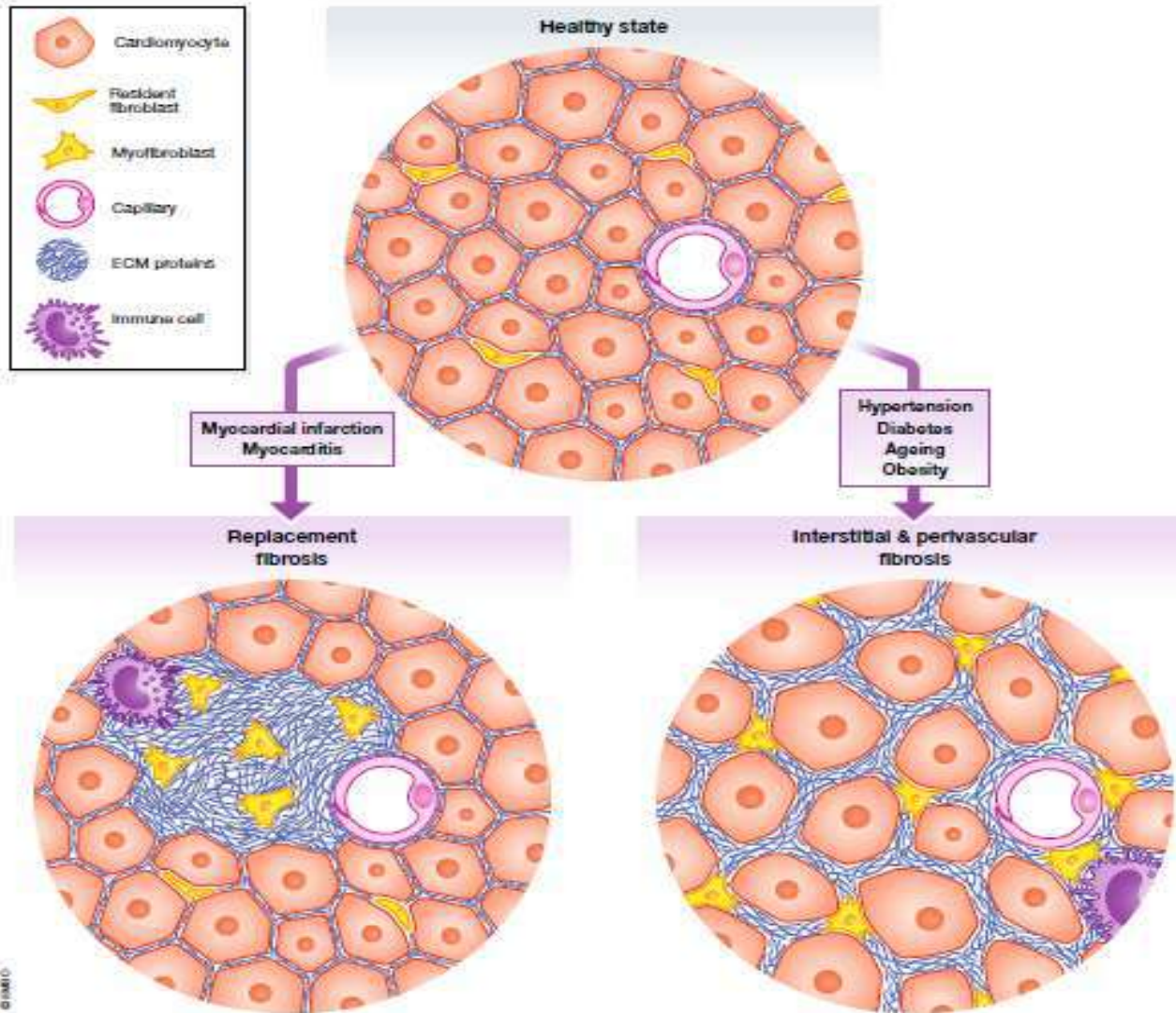


Sarcoidosis

RV involvement



Sarcoidosis



- HISTOLOGICAL DIFFERENCES BETWEEN REPARATIVE FIBROSIS AND INTERSTITIAL/PERIVASCULAR FIBROSIS

BIOPSY VS CMR

TABLE 1 Overview of Studies Validating Various T1-Mapping Methods Against Histological Specimens

First Author (Ref. #)	T1-Mapping Method	Patient Population	r and p Values
Flett et al. (8)	EQ-CMR ECV	18 AS and 8 HCM patients	$r^2 = 0.796$, $p < 0.001$
Fontana et al. (33)	ShMOLLI EQ ECV	18 AS patients	$r^2 = 0.685$, $p < 0.001$
	Multiple breath-hold ECV		$r^2 = 0.589$, $p < 0.001$
White et al. (37)	ShMOLLI single-bolus ECV	18 AS patients	$r^2 = 0.69$, $p < 0.001$
	ShMOLLI EQ ECV		$r^2 = 0.71$, $p < 0.001$
Bull et al. (32)	ShMOLLI native T1	19 AS patients	$r = 0.655$, $p = 0.002$
Miller et al. (36)	DynEq-CMR MOLLI ECV	6 Explanted hearts	$r^2 = 0.893$, $p = 0.004$
Iles et al. (34)	Multiple breath-hold post contrast	9 Patients with heart failure after orthotopic heart transplantation	$r = -0.70$, $p = 0.003$
Mascherbauer et al. (13)	Multiple breath-hold post contrast	9 Patients with heart failure and preserved ejection fraction	$r = 0.977$, $p < 0.001$
Iles et al. (35)	Multiple breath-hold post contrast	4 Explanted hearts; 8 patients with myectomy for HCM	$r = -0.78$, $p = 0.003$
Aus dem Siepen et al. (31)	MOLLI ECV	24 Patients with DCM	$r = 0.85$, $p < 0.01$

AS = aortic stenosis; CMR = cardiac magnetic resonance; DCM = dilated cardiomyopathy; DynEq = dynamic equilibrium; ECV = extracellular volume; EQ = equilibrium; HCM = hypertrophic cardiomyopathy; (Sh)MOLLI = (shortened) modified Look-Locker inversion recovery sequence.



JACC: CARD
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PUBLISHED

T1 M
From

Andreas A
Franz Duc
Andreas G

CMR

- **LV SIZE MILD-MODERATE ENLARGED**
- **LV FUNCTION SEVERE REDUCE (LVEF-18%)**
- **LA, RA SIZE-NORMAL**
- **RV SIZE AND FUNCTION ARE NORMAL**
- **NO SIGN OF LATE GADOLINIUM ENHANCEMENT IN LV, RV, LA AND RA**
- **NORMAL T1 AND T2 WEIGHTED SEQUENCES**

Im: 1/25

Se: 25

A

01.01.2004 M

Hospital

1

KARDIAK^RUTIN

cine_t2d12_retro_4ch

RR: 73b +/- 24; 7 heartbeats

R

L

FS: 3

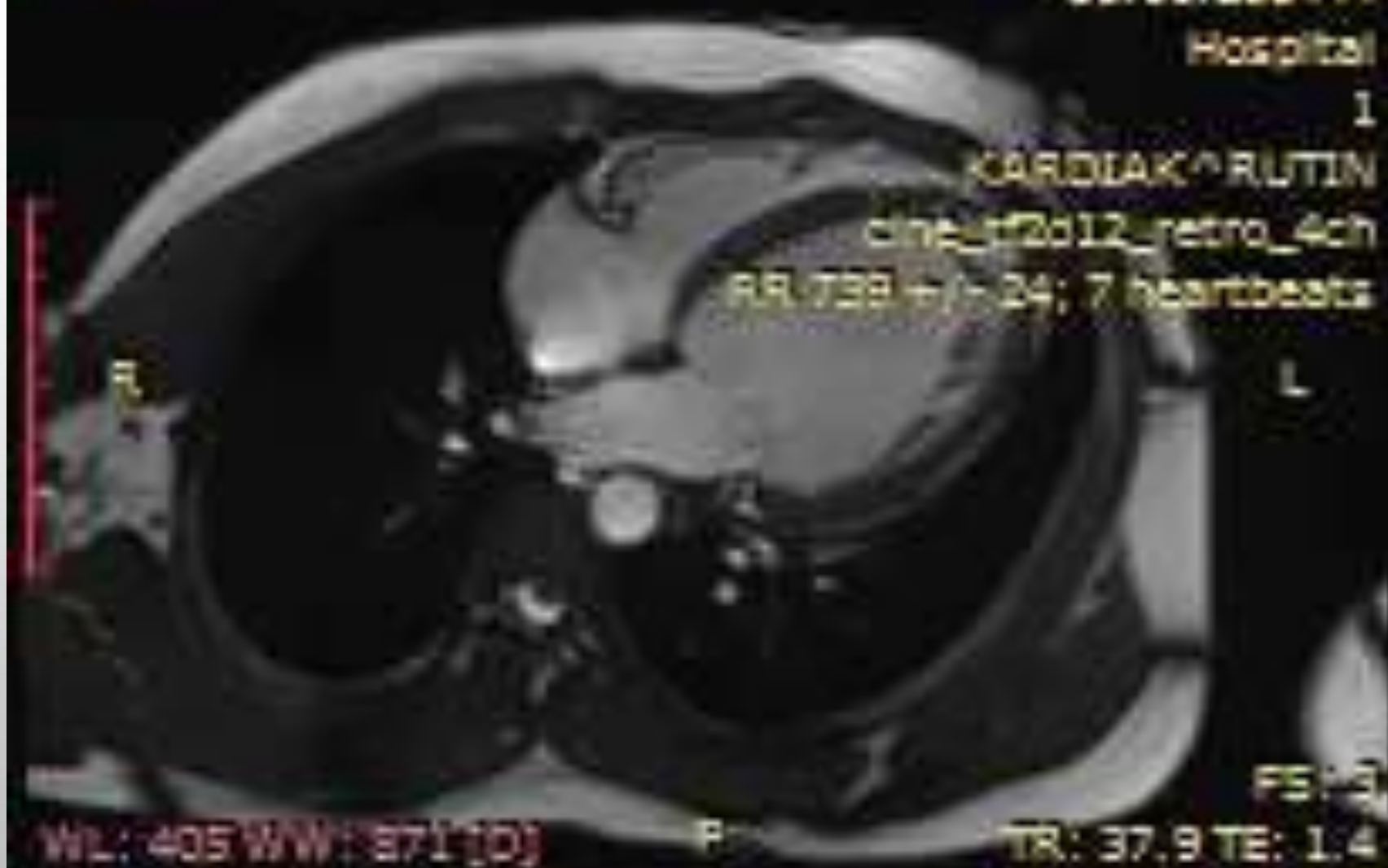
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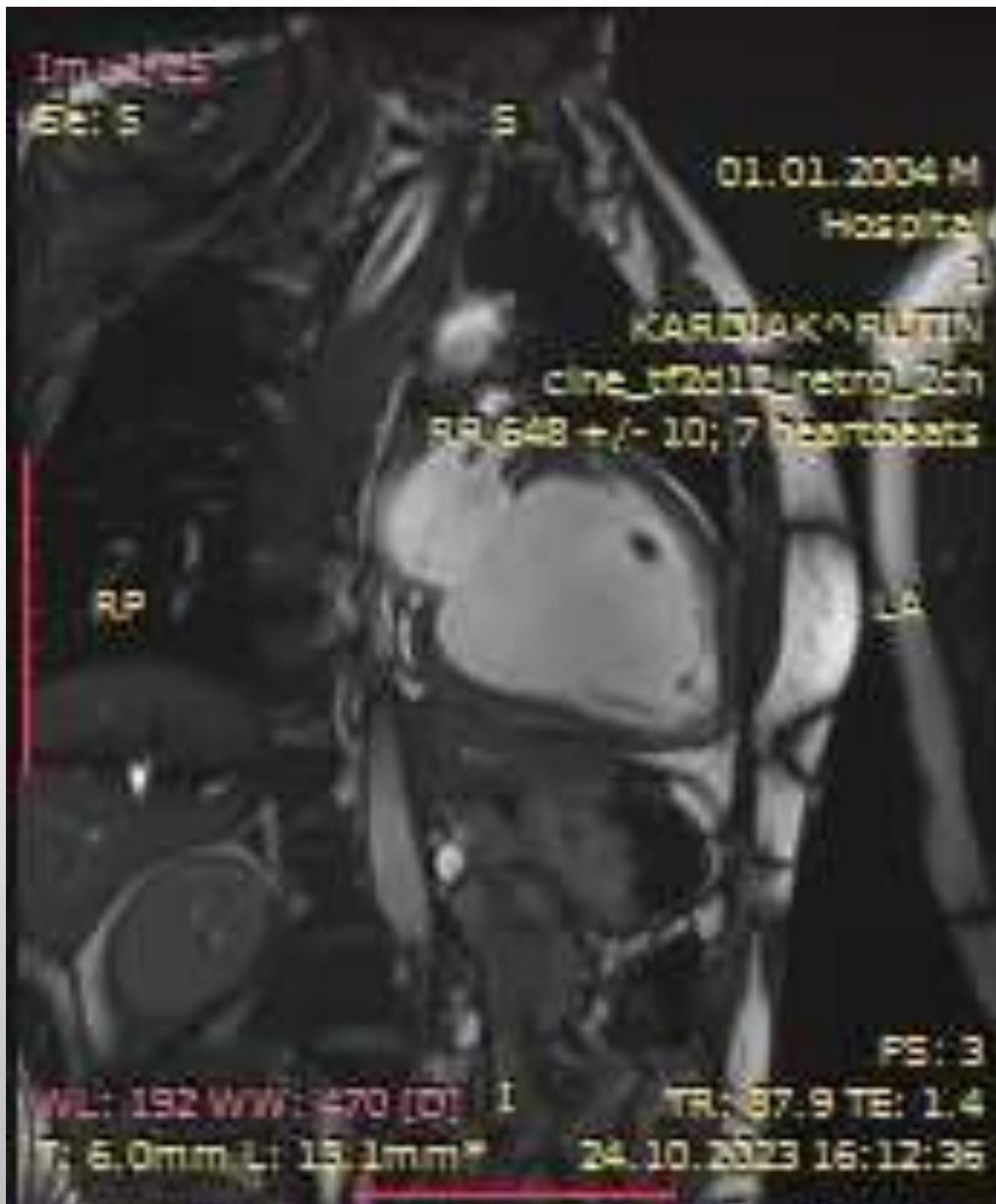
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TR: 37.9 TE: 1.4

T: 6.0mm L: 46.0mm*

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Images

SE: S

S

01.01.2004 M

Hospital

1

KARDIAK-RTIN

cline_H2d12_retro_2ch

RR: 648 +/- 10; 7 heartbeats

RP

LA

PS: 3

WL: 192 WW: 470 (D) 1

TR: 87.9 TE: 1.4

T: 5.0mm L: 15.1mm*

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Im: 1/25

Se: 24

A

01.01.2004 M

Hospital

1

KARDIAK-RUTIN

cine_t12d12_retro_3ch

RR: 715 +/- 45; 7 heartbeats

RS

LI

FS: 3

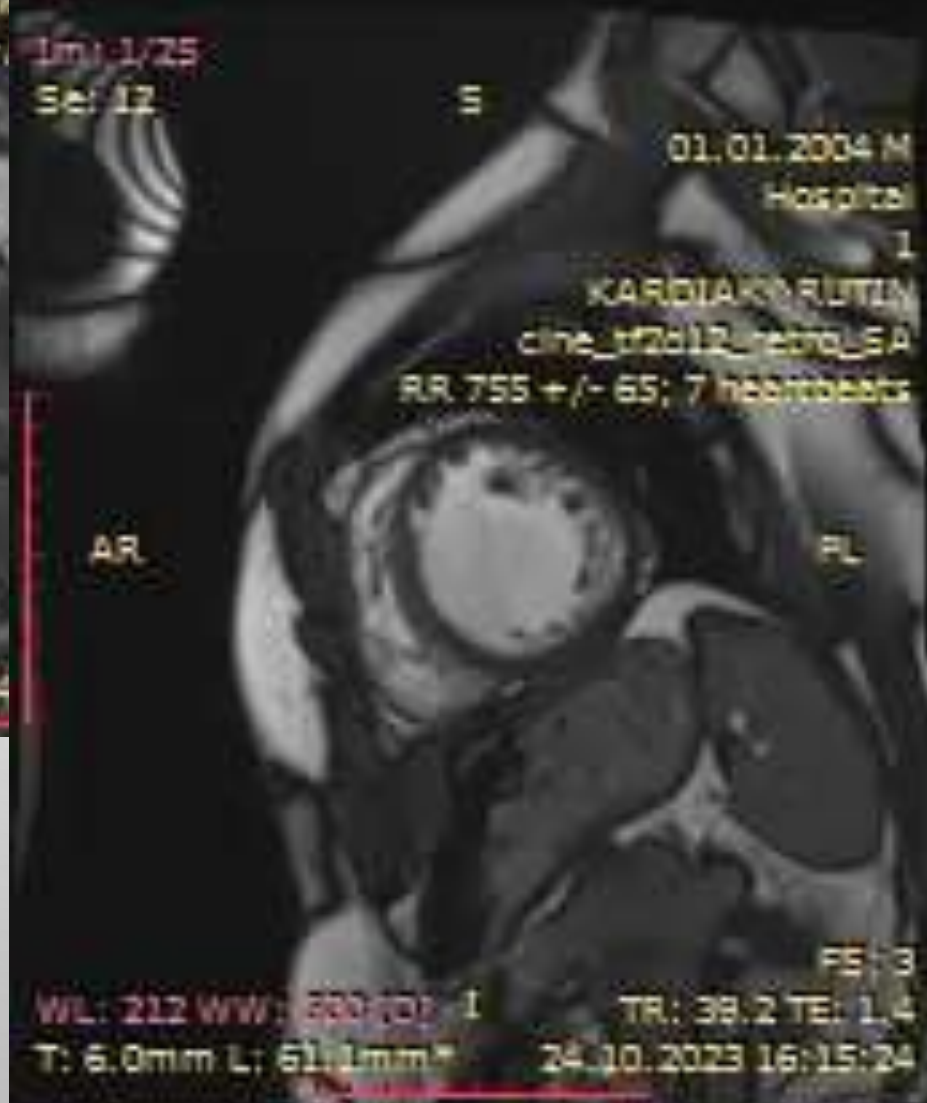
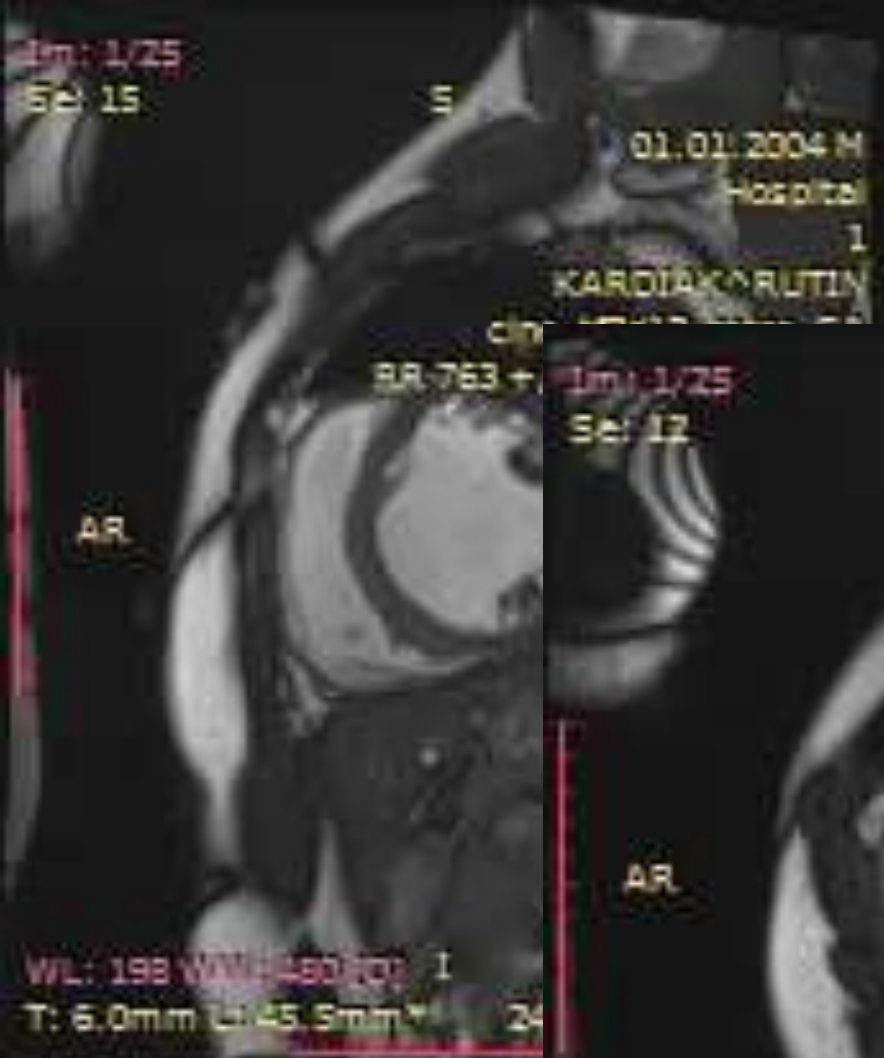
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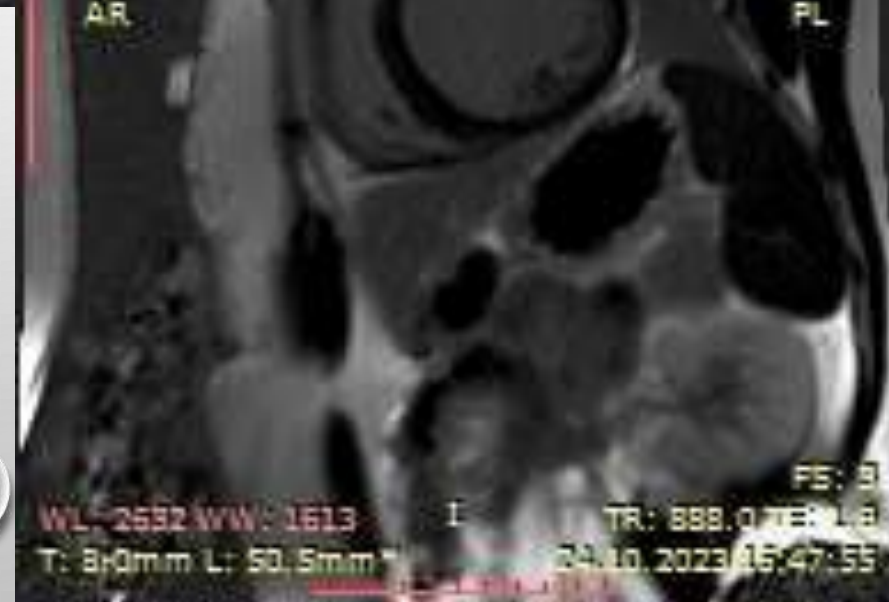
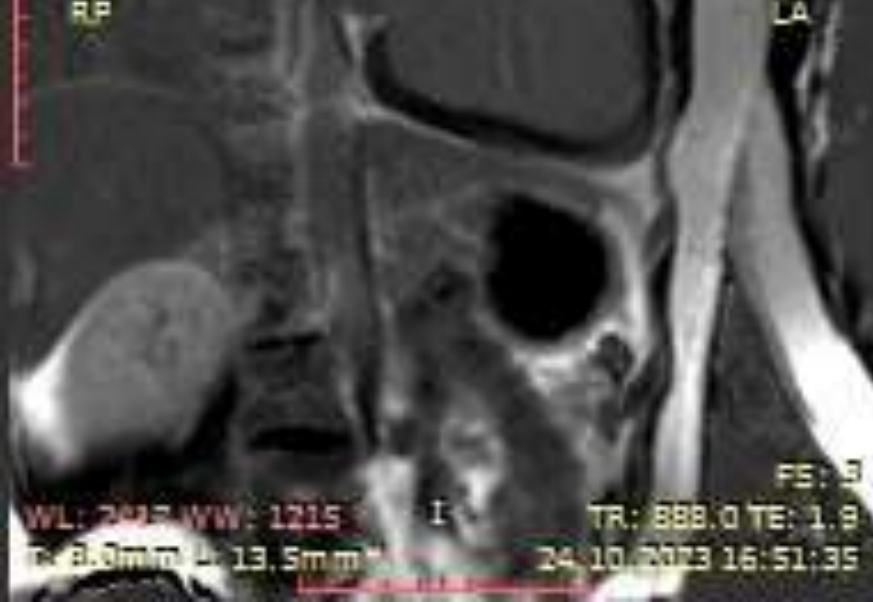
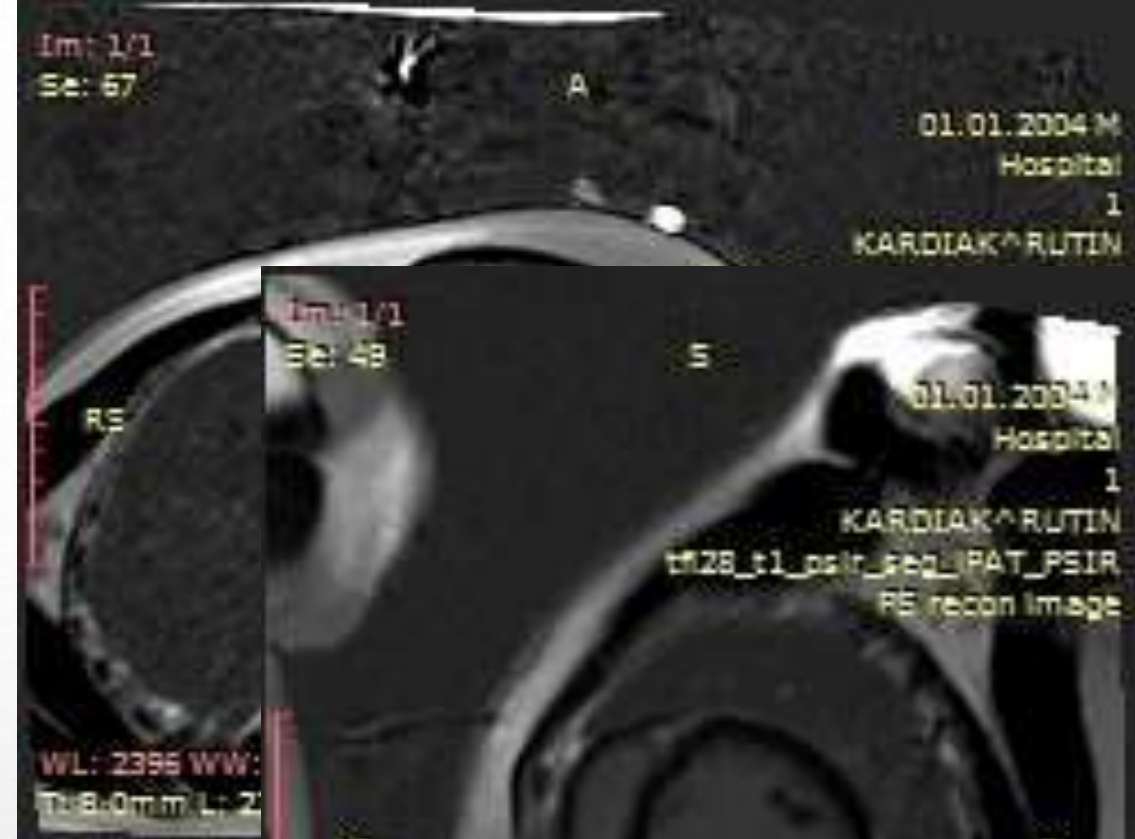
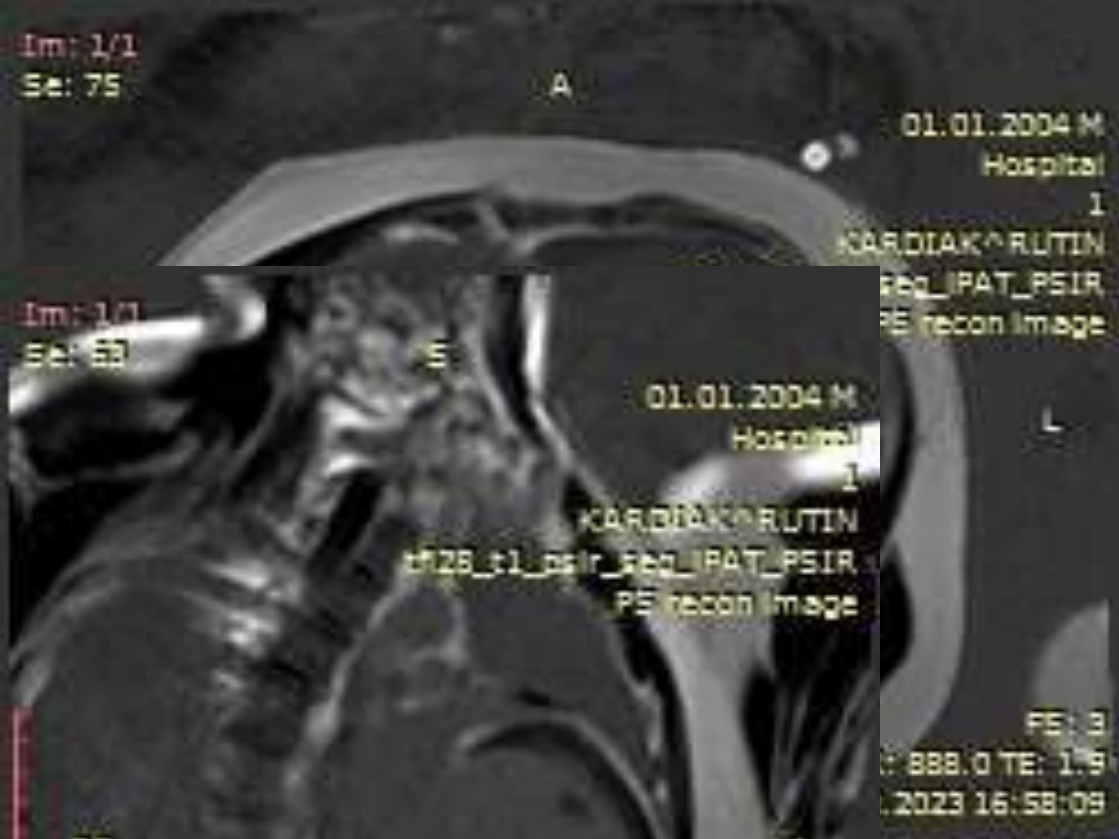
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TR: 37.9 TE: 1.4

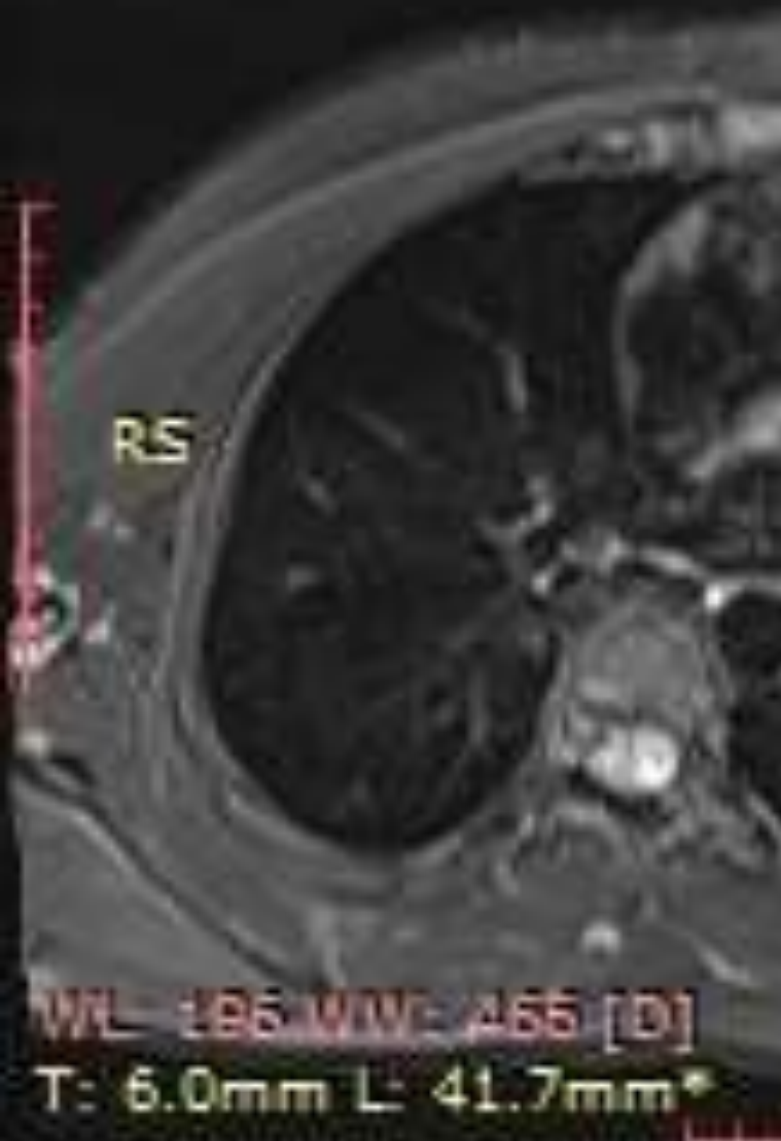
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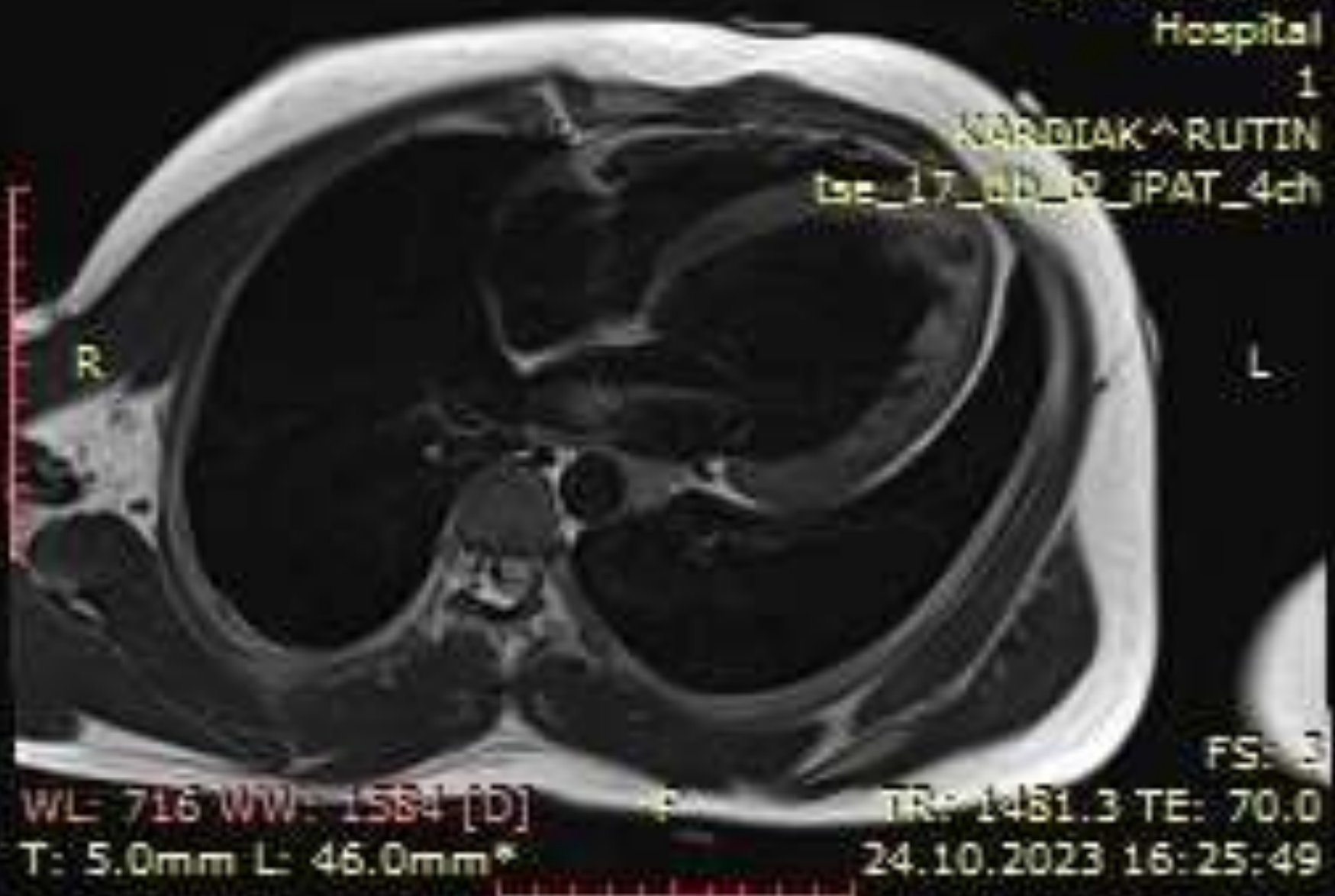




Im: 1/1
Se: 26



Im: 1/1
Se: 31



MSCT

(RCA) Sağ Koronar Arter

1. Proksimal: Normal
2. Orta: Normal
3. Distal: Normal
4. PDA-PLA: Normal

(LMA) Sol Ana Koronar Arter

5. LMA: Normal

(LAD) Sol Ön Enen Koronar Arter

1. Proksimal: Normal
2. Orta: Normal
3. Distal: Normal
4. D1: Normal
5. D2: Normal

(LCX) Sirkumfleks Koronar Arter

6. Proksimal: Normal
7. OM1: Normal
8. Orta: Normal
9. OM2: Normal

Kalsium Skoru

Kalsiyum skorlamasında **Agatston skoru 0.0% Percentile**

Koronar Anomaliya

Yox



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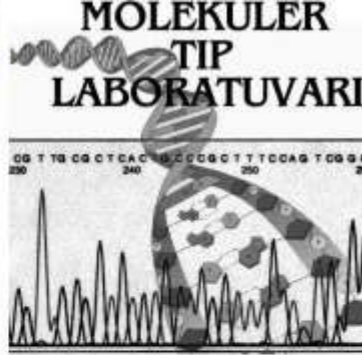
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EGE ÜNİVERSİTESİ TIP FAKÜLTESİ ÇOCUK SAĞLIĞI VE
HASTALIKLARI ANABİLİM DALI

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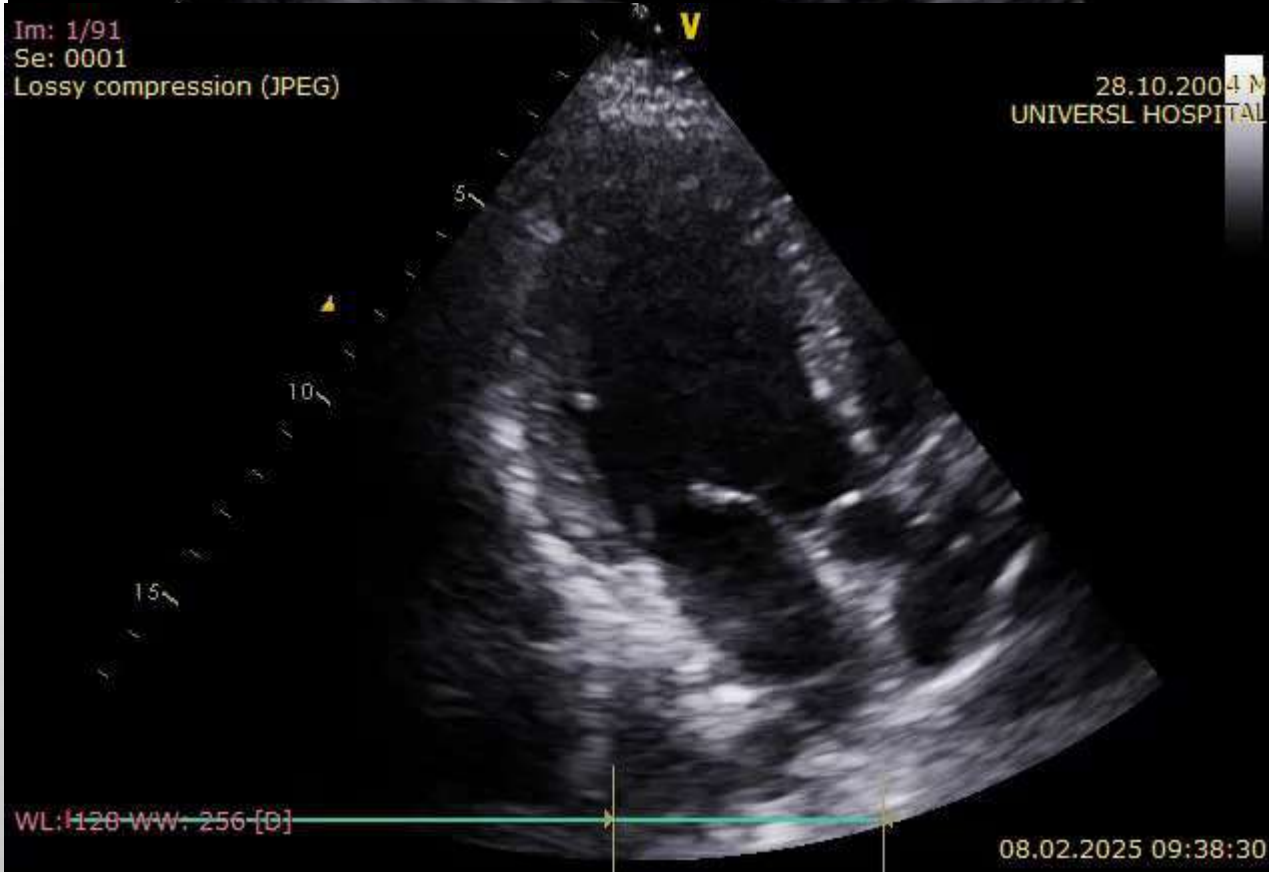
Genetik Analiz Raporu

SONUÇ

- SCN5A** (NM_000335.5) geninde heterozigot p.T1303M (c.3908C>T) varyantı saptanmıştır.
- ACADVL** (NM_000018.4) geninde heterozigot p.K278del (c.833_835del) varyantı saptanmıştır.
- PCSK9** (NM_174936.4) geninde heterozigot p.G394S (c.1180G>A) varyantı saptanmıştır.

-**SCN5A** genindeki heterozigot mutasyonlar otozomal dominant olarak kalıtılan "Long QT Sendromu Tip 3 (OMIM No:603830)" ve "Kardiyomiyopati, Dilate, 1E (OMIM No:601154)" tablosuna neden olmaktadır. Bu gendeki deęişiklikler ACMG kriterlerine göre bildirilmesi gerekli deęişiklikler arasındadır. Olguda heterozigot olarak saptanan p.T1303M (c.3908C>T) varyantı daha önce HGMD veritabanında tanımlanmıştır (CM992663) ve "Long QT Sendromu" ile ilişkilendirilmiştir. Franklin programı saptanan deęişikliği ACMG kriterlerine göre "Olası Patojenik" olarak sınıflandırmaktadır. Bu deęişiklik açısından kardiyolojik deęerlendirme ve aile taraması önerilir.

-HETEROZYGOUS MUTATIONS IN THE SCN5A GENE CAN CAUSE THE AUTOSOMAL DOMINANT INHERITED "LONG QT SYNDROME TYPE 3 (OMIM NO:603830)" AND "CARDIOMYOPATHY, DILATED, 1E (OMIM NO:601154)" TABLE. ...THE P.T1303M (C.3908C>T) VARIANT DETECTED HETEROZYGOUS IN THE CASE WAS PREVIOUSLY IDENTIFIED IN THE HGMD DATABASE (CM992663) AND WAS ASSOCIATED WITH "LONG QT SYNDROME". THE FRANKLIN PROGRAM CLASSIFIES THE DETECTED CHANGE AS "POSSIBLY PATHOGENIC" ACCORDING TO THE ACMG CRITERIA. CARDIOLOGICAL EVALUATION AND FAMILY SCREENING ARE RECOMMENDED FOR THIS CHANGE.



08.02.2025

- **NT PRO BNP-NORMAL**
- **ECG-NSR**
- **ECHO-NORMAL LV SIZE. MILDE REDUSE LFEF-45%(SIMPSON BIPLAN). NORMAL SIZE ANF FUNCTION OF RV. NORMAL SIZE OF LA AND RA. MILDE AI AND MI.**
- **BEGINING_GYNECOMASTY-CHANGE SPIRONALAKTON TO FINERENON**

TREATMENT

- SACUBETRIL/VALSARTAN 97/103 MG 1-0-1
- FINERENON 20 MG 0-1-0
- BISOPROLOL 2,5 MG 0-0-1
- EPAGLIFLOZIN 10 MG 1-0-0

THANK YOU FOR YOUR ATTENTION!!!

