

A SZÍV IMMUNPATHOMECHANIZMUSÚ BETEGSÉGEI

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SE Reumatológiai és Immunológiai Klinika
Intenzív Terápiás osztály

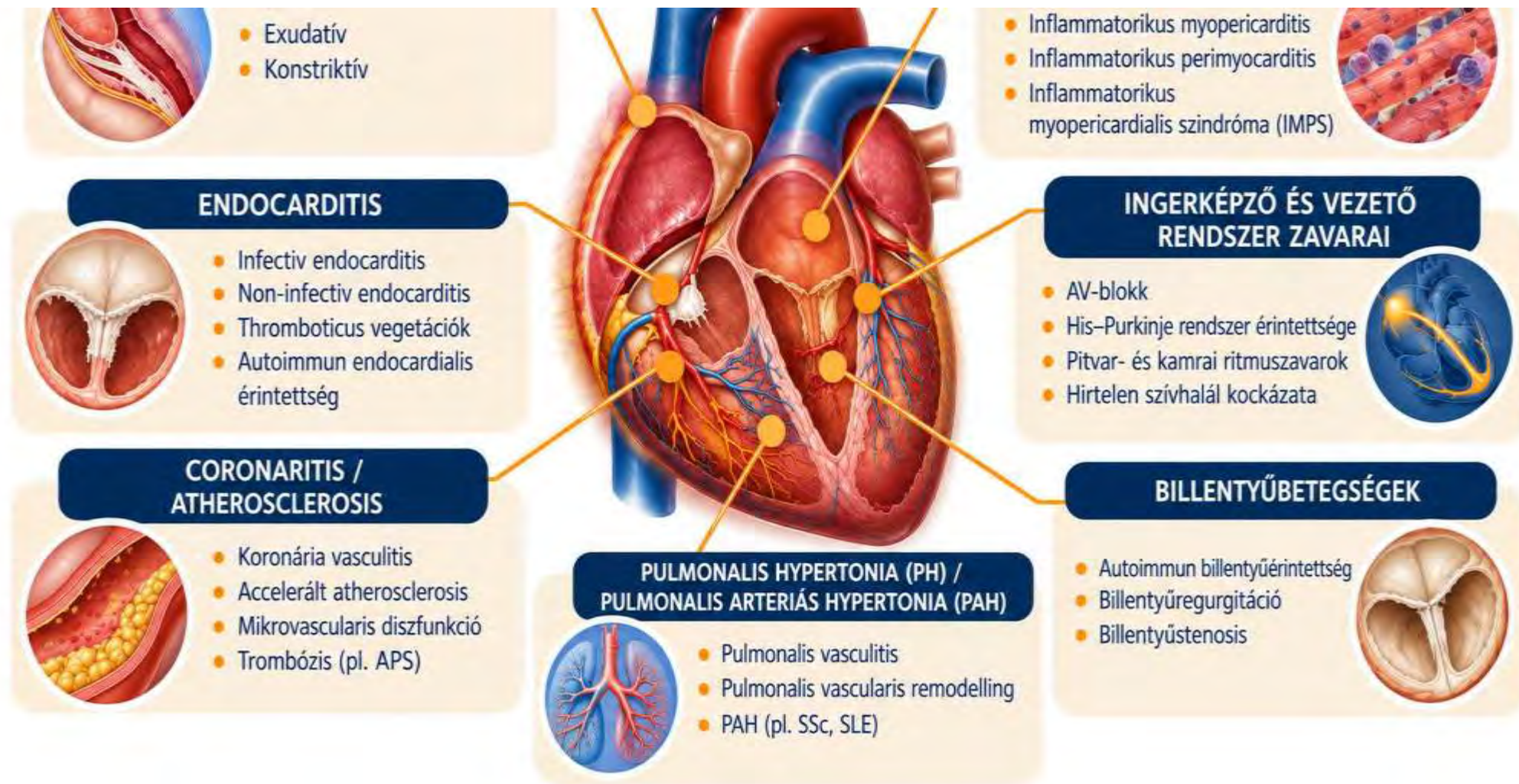
Klinikai Immunológia és allergológia szakvizsga előkészítő tanfolyam

2026.10.02.



SEMMELWEIS
EGYETEM 1769

A SZÍV IMMUNMECHANIZMUSÚ BETEGSÉGEI



A SZÍV IMMUNMECHANIZMUSÚ BETEGSÉGEI

Elsődlegesen a szív alkotórészei ellen irányul a gyulladásos folyamat (szervspecifikus autoimmun betegség)
iCMP, Reumás láz

- PRIMAER

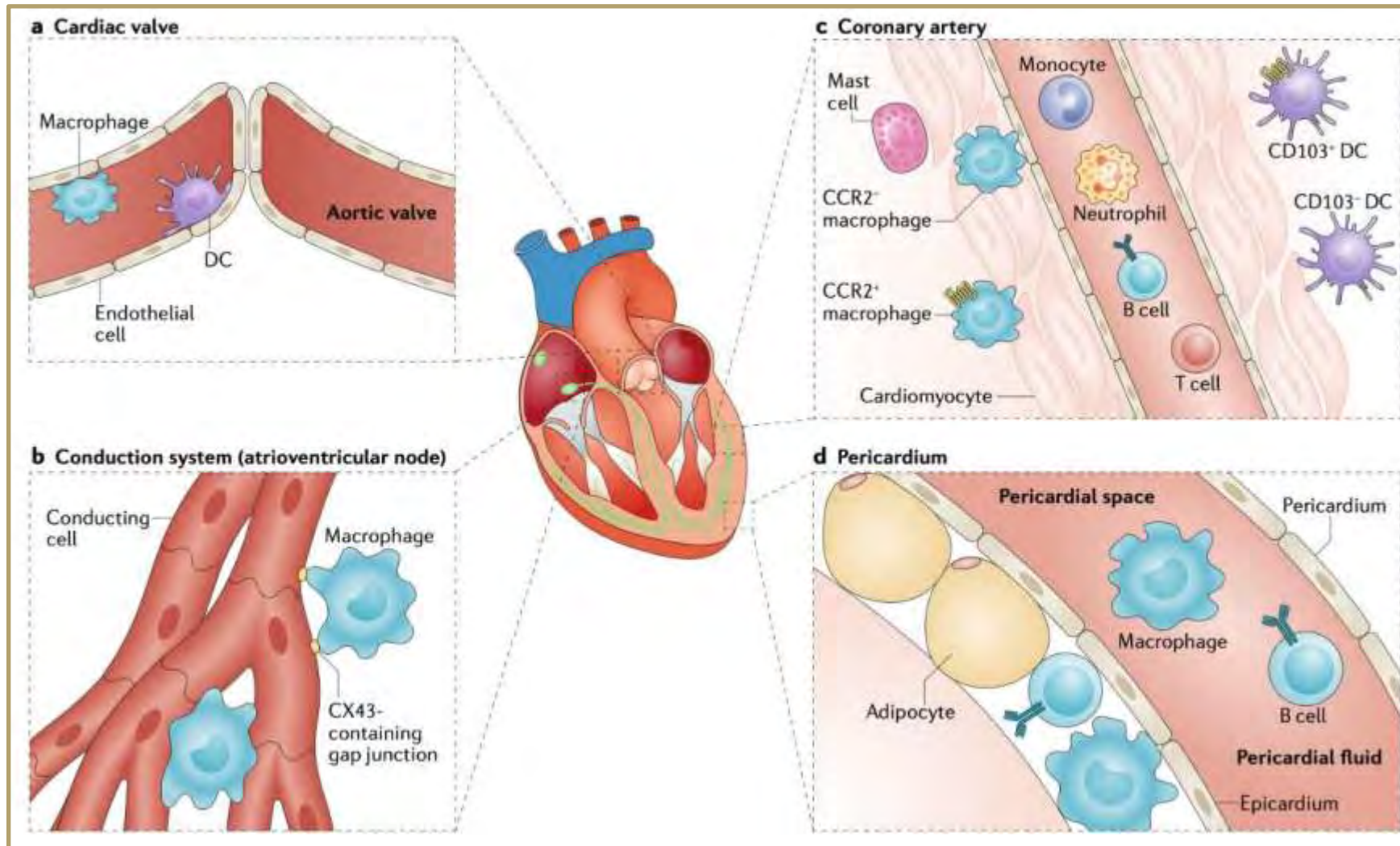
Egyéb autoimmun betegség részjelensége
PSS, RA, SLE, Sjögren sy, SPA, Vasculitis, PsA

- SECUNDAER



Betegség	Pericardium	Myocardium	Coronaria	Billentyű	Arrhythmia	PH
SLE	+++	++	++	+++	+	+
SSc	+	+++	++	+	+++	+++
Myositis	+	+++	+	-	++	+
EGPA	++	+++	+++	-	++	+
RA	++	+	+++	+	+	+
APS	-	+	+++	+++	-	++

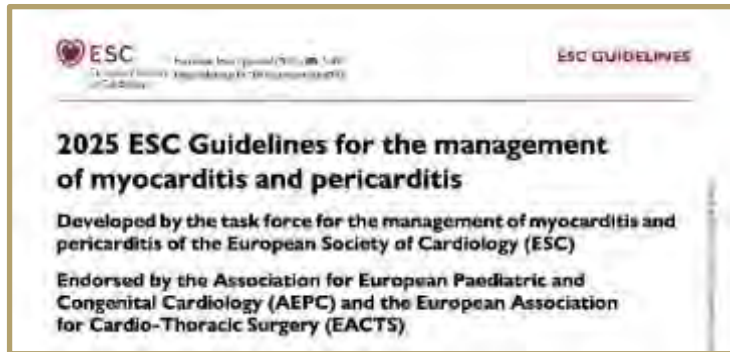
IMMUNHOMEOSZTÁZIS A SZÍVBEN



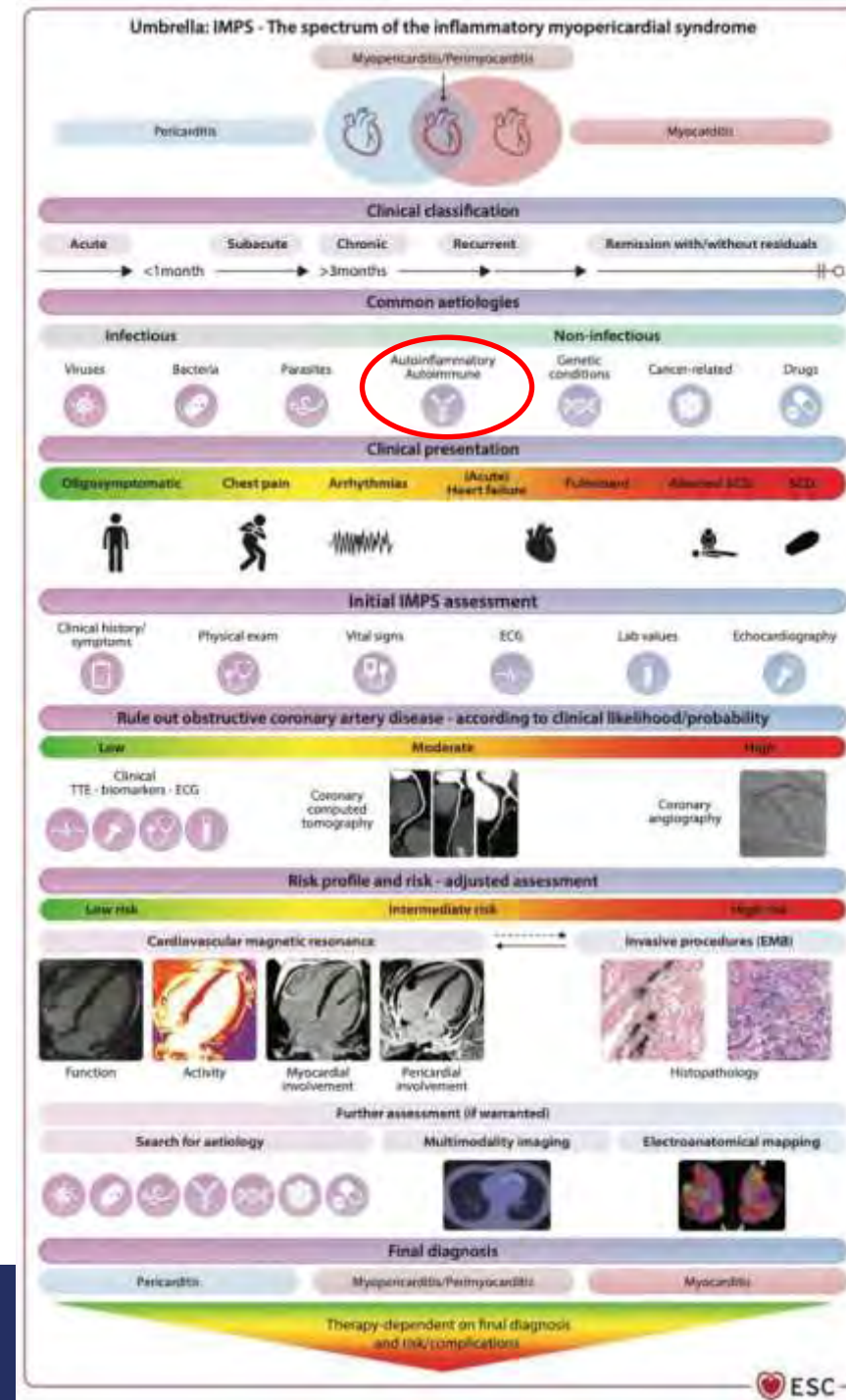
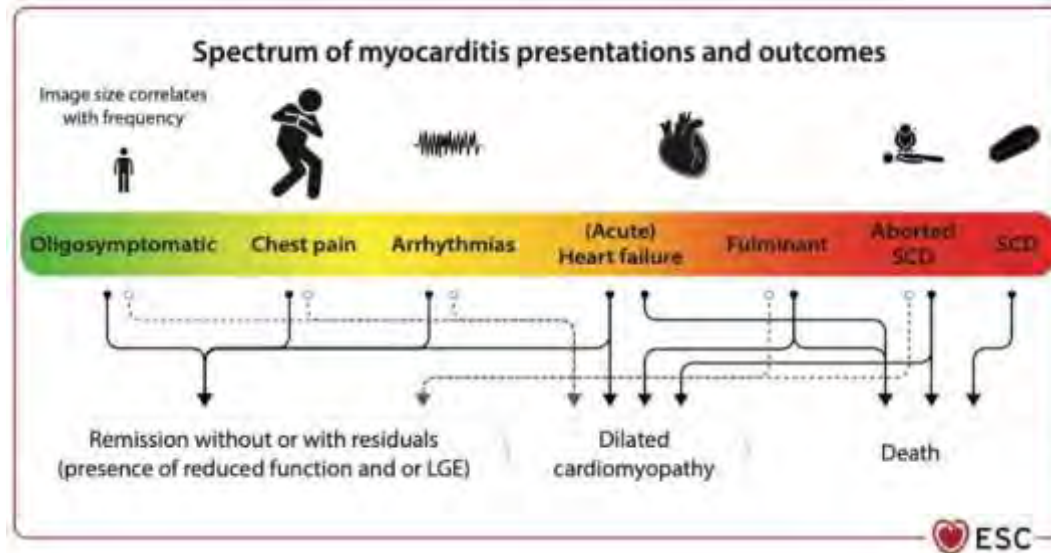
Cardioimmunology: the immune system in cardiac homeostasis and disease_Swirski FK, Nahrendorf M.Nat Rev Immunol. 2018 Dec;18(12):733-744. doi: 10.1038/s41577-018-0065-8.

**PERICARDITIS
MYOCARDITIS
CARDIOMYOPATHIÁK
IMMUNOLÓGIAI VONATKOZÁSAI**

IMPS - INFLAMMATÓRIKUS MYOPERICARDIALIS SZINDROMA



- Általában együtt fordul elő
perimyocarditis - myopericarditis
- Spektrumbetegség
- PARADIGMAVÁLTÁS



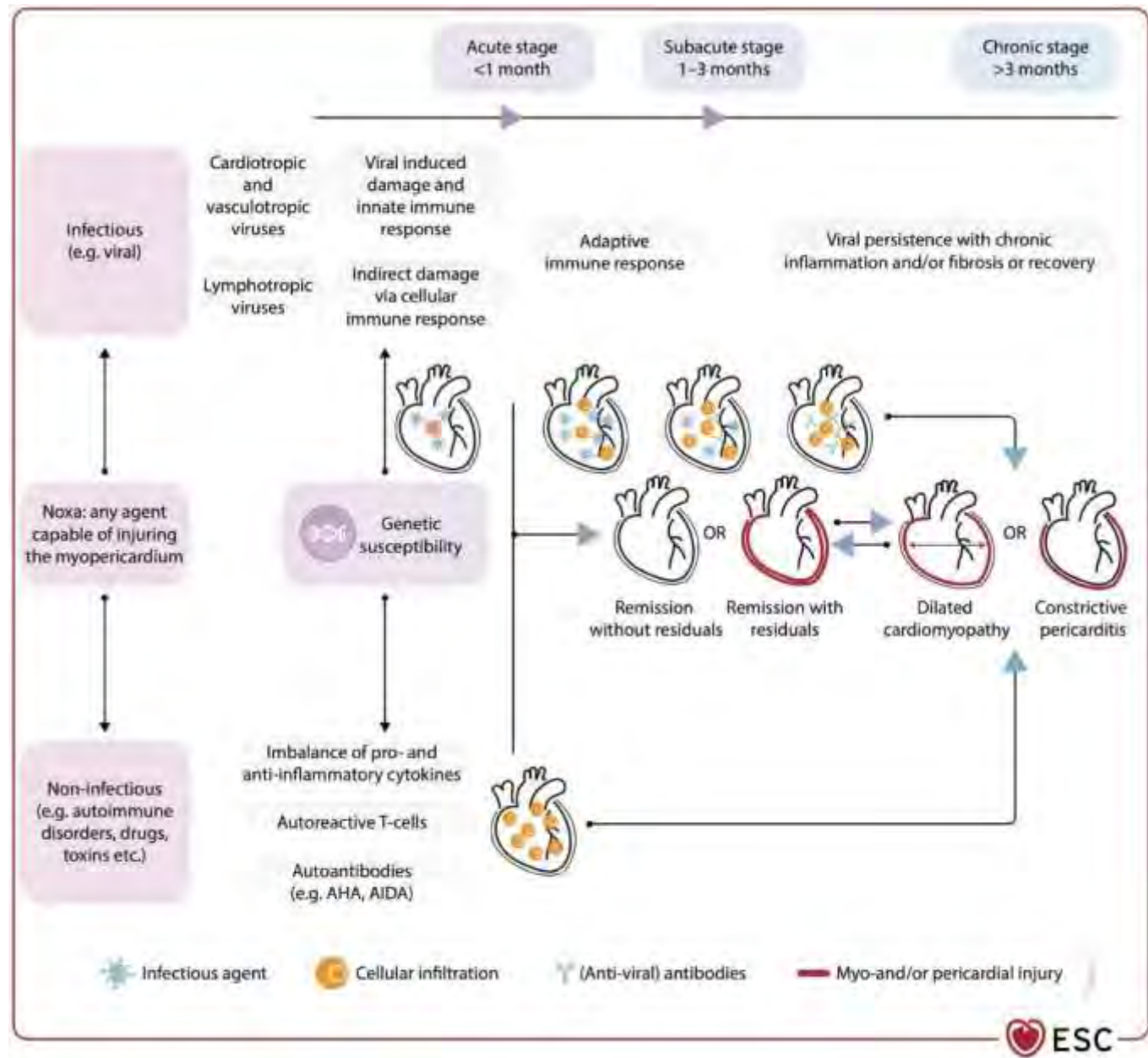
IMPS - terminológia - stádiumok

Terminology	Definition
IMPS	Umbrella term for inflammatory myocardial and pericardial syndromes
Myopericarditis	Predominant pericarditis ^a
Perimyocarditis	Predominant myocarditis ^b
Acute myocarditis	Duration of symptoms ≤ 4 weeks Fulminant if: Acute onset²⁸ and haemodynamically unstable patients requiring inotropes or mechanical circulatory support
Complicated myocarditis	AM and ≥ 1 of the following: ²⁸ - LVEF $< 50\%$ on echocardiogram - Sustained ventricular arrhythmias - Advanced heart block - Heart failure - Cardiogenic shock
Acute pericarditis	Duration of symptoms ≤ 4 weeks

[2025 ESC Guidelines for the management of myocarditis and pericarditis.](#)
Schulz-Menger J, et al. Eur Heart J. 2025. Aug 29;ehaf192. doi: 10.1093/eurheartj/ehaf192.

Terminology	Definition
Subacute/ongoing myocarditis	Duration of symptoms > 4 weeks to ≤ 3 months
Subacute/incessant pericarditis ^c	Duration of symptoms > 4 weeks to ≤ 3 months
Chronic myocarditis/pericarditis	Duration of symptoms > 3 months
Inflammatory cardiomyopathy	Chronic myocarditis in association with cardiac dysfunction and ventricular remodelling with clinical phenotype of hypokinetic, either dilated or non-dilated cardiomyopathy with/without arrhythmogenic substrate
Recurrent myocarditis/pericarditis	New symptoms or disease activity after clinical remission
Remission without residuals	Regression/absence of symptoms, normalization of ECG, biomarkers, imaging abnormalities (echocardiography and CMR)
Remission with residuals	Regression/absence of symptoms, persistence of abnormalities on ECG, biomarkers and/or imaging (functional and/or structural abnormalities in echocardiography or CMR)

IMPS - stádiumok



2025 ESC Guidelines for the management of myocarditis and pericarditis.
Schulz-Menger J, et al. Eur Heart J. 2025. Aug 29;ehaf192. doi: 10.1093/eurheartj/ehaf192.

PERICARDITIS

Autoimmun PERICARDITIS okai és kimenetele

SLE, RA, MCTD, SSc....

Systemic Autoimmune Disease



Auto-antibodies and immune-complexes

Autoinflammatory Disease



Genetic Mutation

Sarcoidosis, Still kór

Post-cardiac Injury Syndrome



Dressler sy

Post myocardial infarction,
Post cardiac surgery/intervention
Anti-heart antibodies

Pericardial Effusion

Small <10 mm
Moderate 10-20 mm
Large >20mm

Cardiac Tamponade (rare)

Pericardiocentesis
Pericardial window

Constrictive Pericarditis

Auto-inflammatory therapy
Pericardiectomy

Acute Pericarditis



Medical Management

Autoimmune Pericarditis: Multimodality Imaging

October 2022 - *Current Cardiology Reports* 24(5):1-13

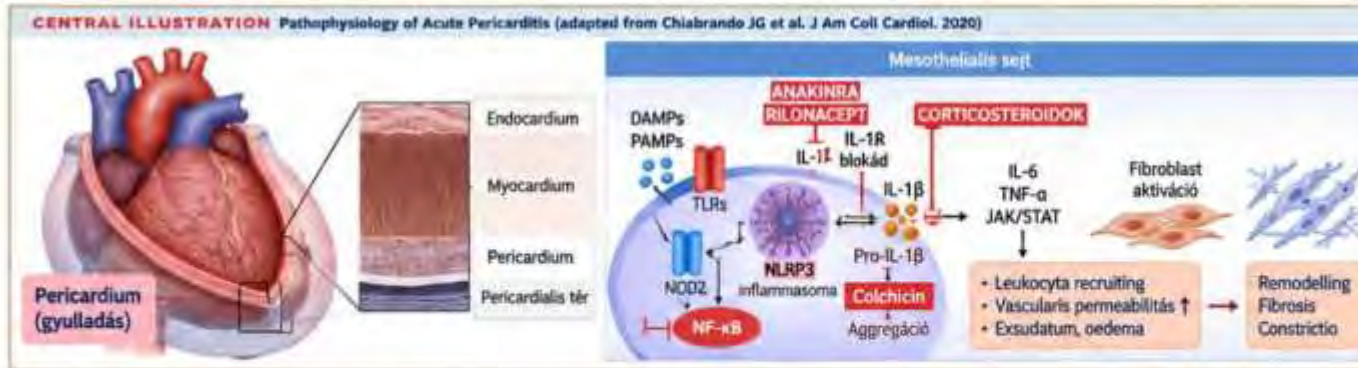
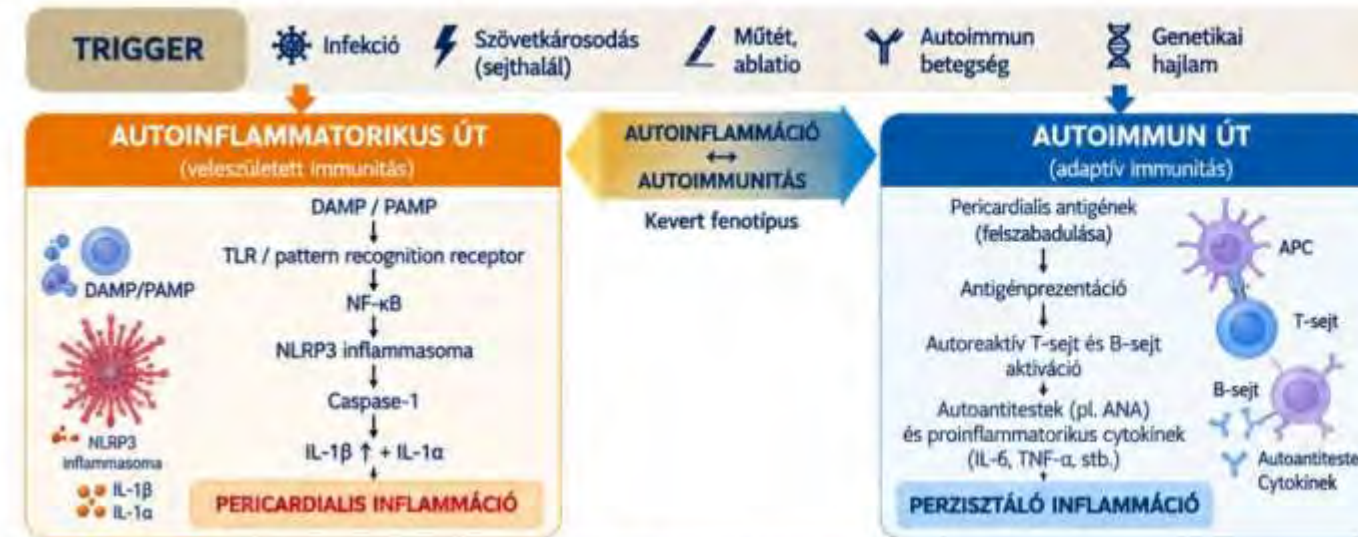
DOI:10.1007/s11886-022-01785-3



PERICARDITIS - PATHOGENEZIS

Chiabrando, J.G. et al. J Am Coll Cardiol. 2020;75(1):76-92.

2025 ESC Guidelines for the management of myocarditis and pericarditis.
Schulz-Menger J, et al. Eur Heart J. 2025. Aug 29;ehaf192. doi:
10.1093/eurheartj/ehaf192.



Diagnosztikus kritériumok (ESC 2025)

Pericarditis

Klinikai prezentáció (jellegzetes mellkasi fájdalom, pericardialis dörzsi zaj)	+ ≥1 további kritérium
Klinikai prezentáció (jellegzetes mellkasi fájdalom, pericardialis dörzsi zaj)	+ 1 további kritérium
Csak klinikai prezentáció (jellegzetes mellkasi fájdalom, pericardialis dörzsi zaj)	további kritérium nélkül (lehetséges eset)

További kritériumok

Pericardialis dörzsi zaj
PR depresszió, széleskörű ST-segmens elevatio
C-reaktív protein emelkedés
Új vagy fokozódó pericardialis folyadék
Pericardialis oedema és/vagy LGE (CMR-eltérés)

DAMP/PAMP → NLRP3 → IL-1 = a pericardialis gyulladás központi tengelye

Autoinflammatorikus dominancia ← Immune spectrum → Autoimmun dominancia

PERICARDITIS kezelés

Terápia	Adagolás	Időtartam ^a	Lépítés ^a
Aspirin ^b	750–1000 mg 3× naponta	1–2 hét	250 mg-mal csökkentés 1–2 hetente
Ibuprofen ^b	600–800 mg 3× naponta	1–2 hét	200 mg-mal csökkentés 1–2 hetente
Indomethacin	25–50 mg 3× naponta	1–2 hét	25 mg-mal csökkentés 1–2 hetente
Colchicin ^b	0,5 mg 1× naponta (<70 kg vagy súlyos vesekárosodás: 0,5 mg 2× naponta)	3–6 hónap	Nem szükséges
Prednison	0,2–0,5 mg/ttkg/nap	2–4 hét	Több hónap alatt

Kizárólag recidíva esetén adandó kezelés

Azathioprin	Kezdetben 1 mg/ttkg/nap, majd fokozatos emelés 2–3 mg/ttkg/nap	Több hónap	Több hónap
IVIG	400–500 mg/ttkg i.v. naponta 5 napon át	5 nap	Nem szükséges
Anakinra	1–2 mg/ttkg/nap, felnőttben max. 100 mg/nap	Legalább 6 hónap (>12 hónap)	Szükséges (legalább 3–6 hónap)
Rilonacept	320 mg 1× hetente, majd 160 mg hetente	>12 hónap	Ismeretlen

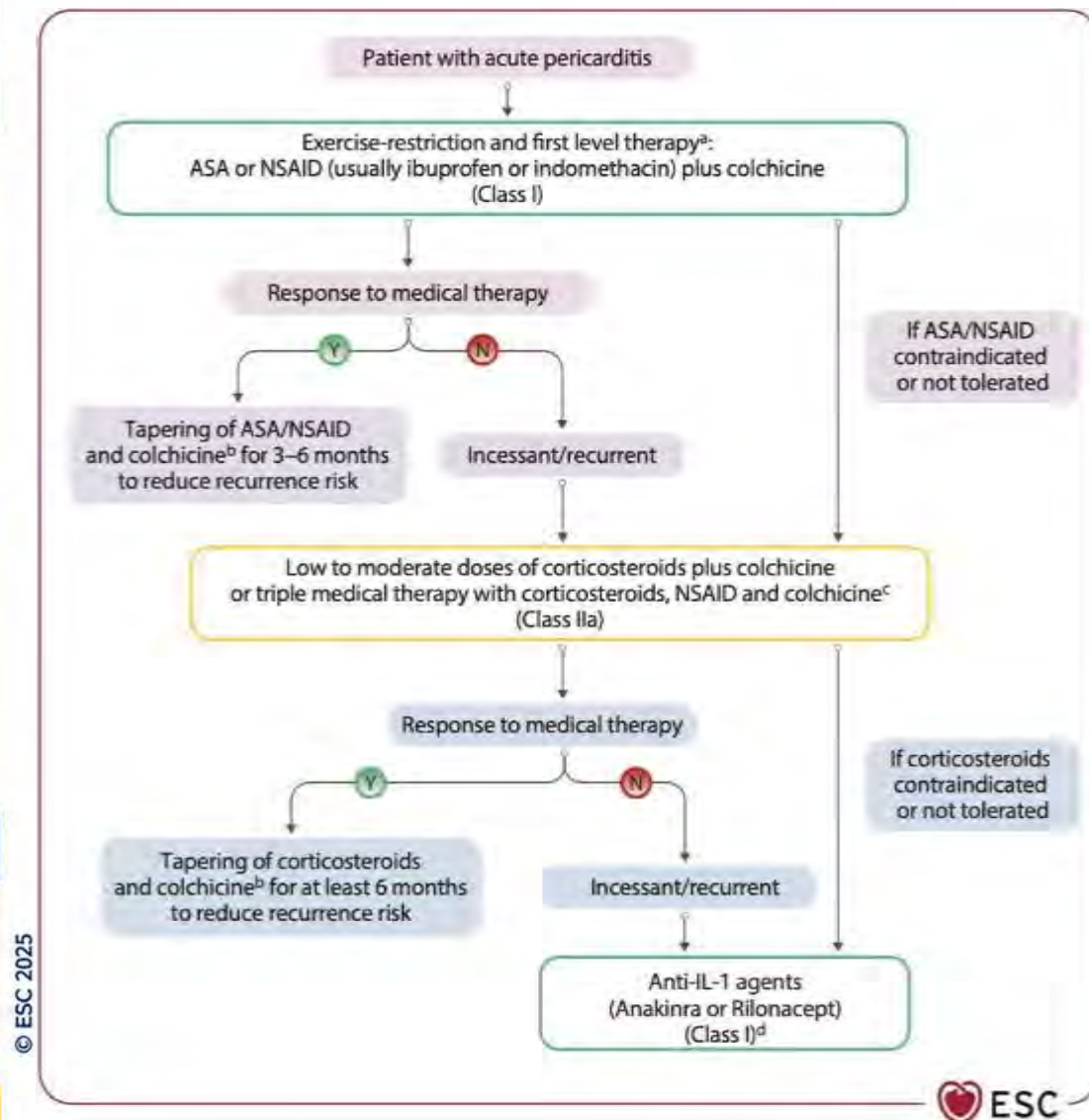
Prednison dózisa ^a	Kezdő dózis (0,20–0,50 mg/ttkg/nap)	Lépítés ^b
> 50 mg/nap	> 50 mg	10 mg/nap csökkentés 1–2 hetente
50–25 mg/nap	50–25 mg	5–10 mg/nap csökkentés 1–2 hetente
25–15 mg/nap	25–15 mg	2,5 mg/nap csökkentés 2–4 hetente
< 15 mg/nap	< 15 mg	1,25–2,5 mg/nap csökkentés 2–6 hetente



Autoimmun betegségek – lehet aktivitási jel

First line: szteroid

Scleroderma – szteroid ritkán, akkor igen !



2025 ESC Guidelines for the management of myocarditis and pericarditis. Schulz-Menger J, et al. Eur Heart J. 2025. Aug 29;ehaf192. doi: 10.1093/eurheartj/ehaf192.

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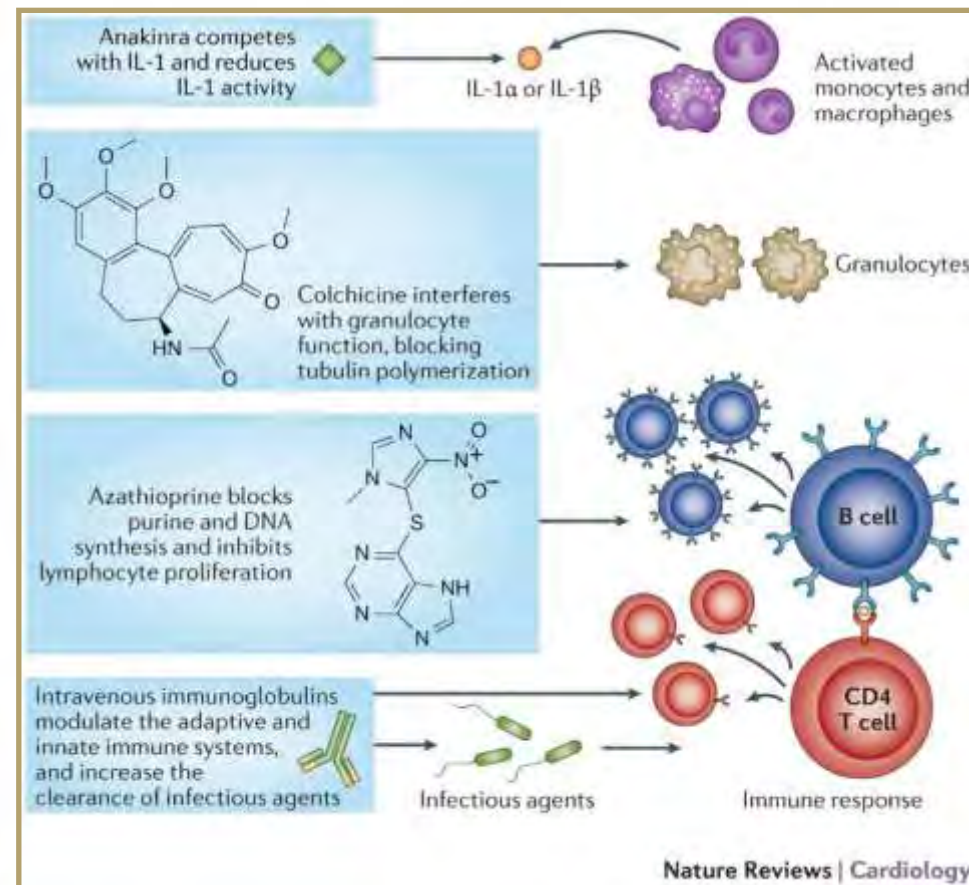
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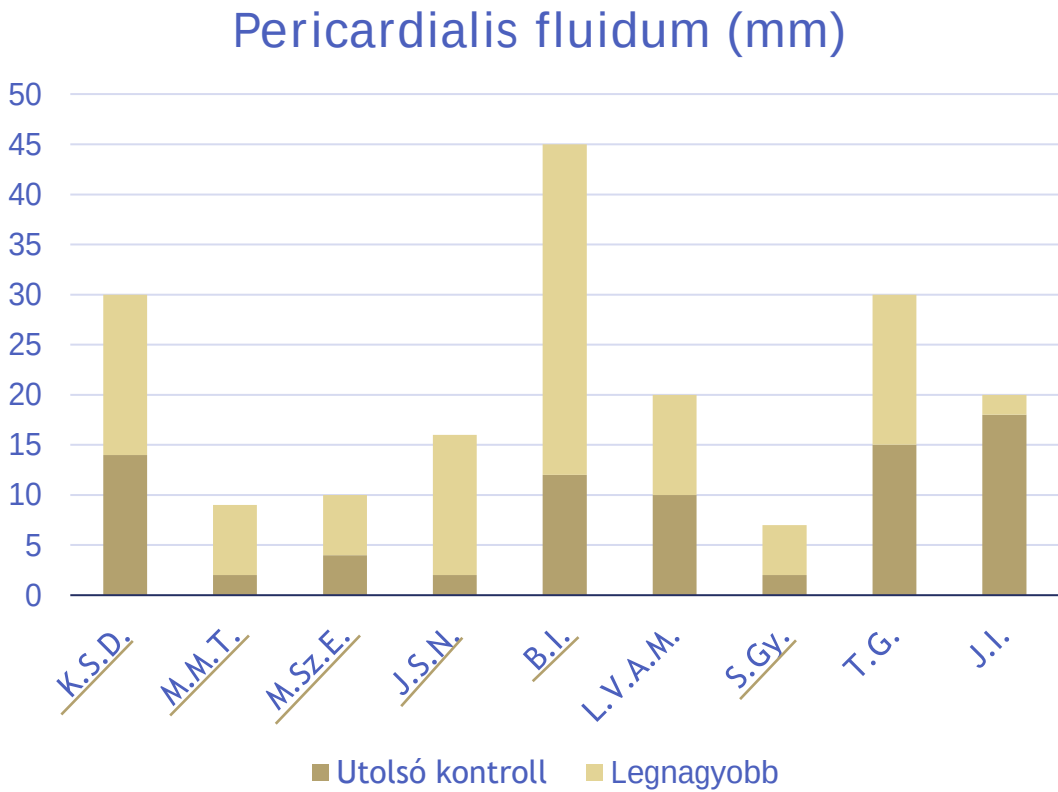
© ESC 2025

2025 ESC Guidelines for the management of myocarditis and pericarditis. Schulz-Menger J, et al. Eur Heart J. 2025. Aug 29;ehaf192. doi: 10.1093/eurheartj/ehaf192.

Név	AI betegség	Immunszerológia	CRP	We	PCT	Gyógyszeres terápia
K.S.D.	RP	negatív	6,1	34	<0,5	Kineret sc. inj + Colchicin, sz.e Ibuprofen
M.M.T.	RP	RF neg. ACCP 30	48	82	<0,5	Kineret sc. inj
M.Sz.E	RA	RF 112 ACCP > 1600 Nuklearis AT poz.	45,7	40	<0,5	Medrol + Kineret sc. inj.
J.S.N.	SLE+Sj	ANA poz 1:160 SSA 141, SSB 210 dsDNS 20, C1q 54	112	86	<0,5	Plaquenil + Imuran + Kineret sc. inj
B.I.	RA	RF 48 ACCP 620	76	55	<0,5	MTX + Medrol + Kineret sc. inj + Colchicin + Plaquenil
L.V.A.M	SSc+Sj	ANA 1:160 Centromer AT poz. SSA 119	8	48	<0,5	Nem kap terápiát Malignus folyamat kizárása
S.Gy.	RA	RF 195 ACCP >1600	111	69	<0,5	MTX + Kineret sc. inj
T.G.	NDC	ANA poz 1:80 C4 ↓	24	36	<0,5	Colchicin + Kineret sc
J.I	RP	Negatív QF poz.	12	20	<0,5	Medrol, látens TBC miatt Rifampicin

IL-1 gátlóval szerzett tapasztalatok

2024. szeptember és 2025. augusztus közötti időszak



PERICARDIOCENTEZIS



Egy betegnél pericardiocentesis, majd fenestráció történt
Saját felvétel, a beteg beleegyezésével



Injekció beadás helyén létrejött reakció
Saját felvétel, a beteg beleegyezésével

MYOCARDITIS

CARDIOMYOPATHIÁK IMMUNOLÓGIAI VONATKOZÁSAI

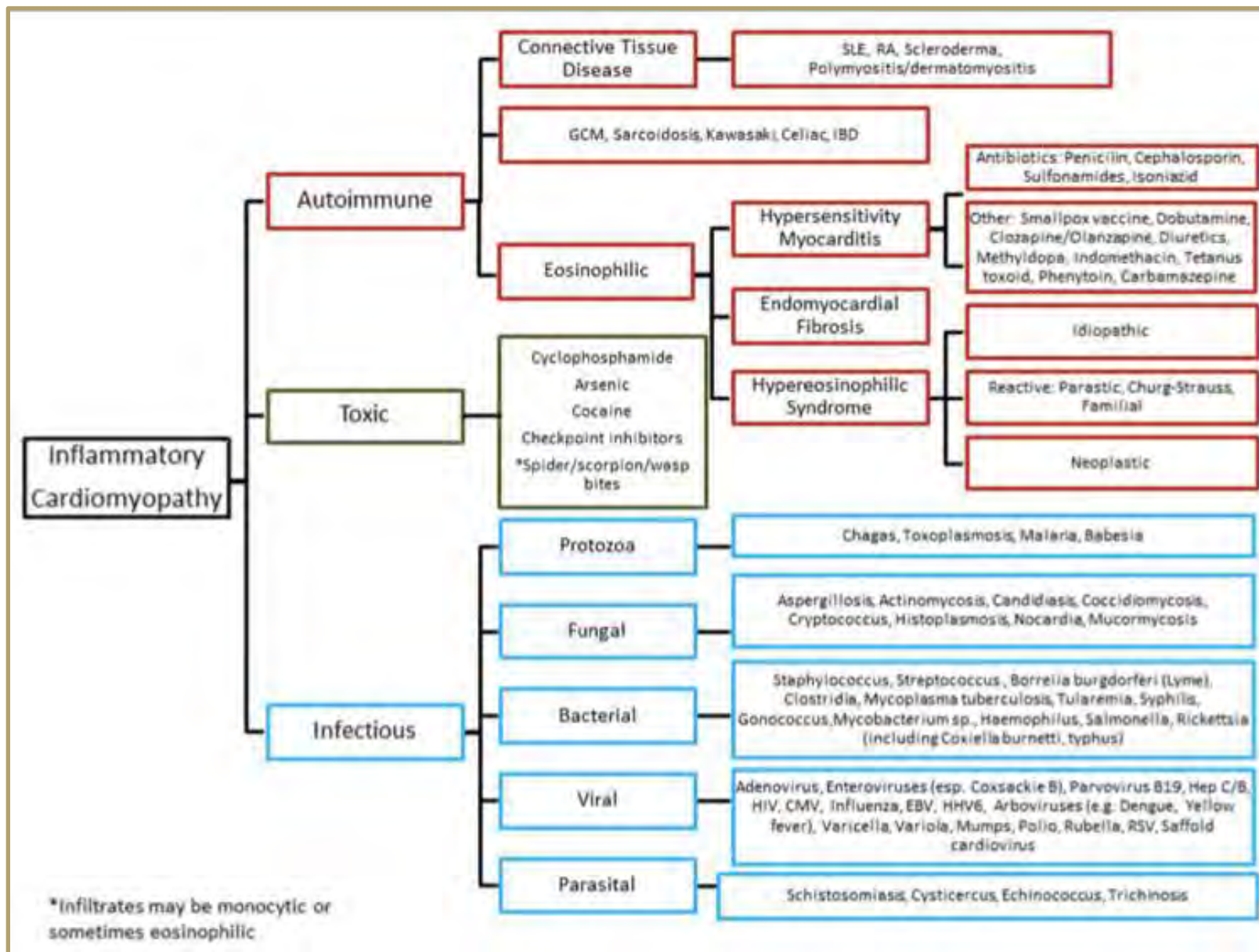
INFLAMMATÓRIKUS CARDIOMYPATHIA WHO 1995

Richardson, P., McKenna, W., Bristow, M., Maisch, B., Mautner, B., O'Connell, J., Olsen, E., Thiene, G., Goodwin, J., Györfi, L., et al. Report of the 1995 World Health Organization/International Society and Federation of Cardiology Task Force on the Definition and Classification of cardiomyopathies. *Circulation* 1996; 93, 841-842. [CrossRef] [PubMed]

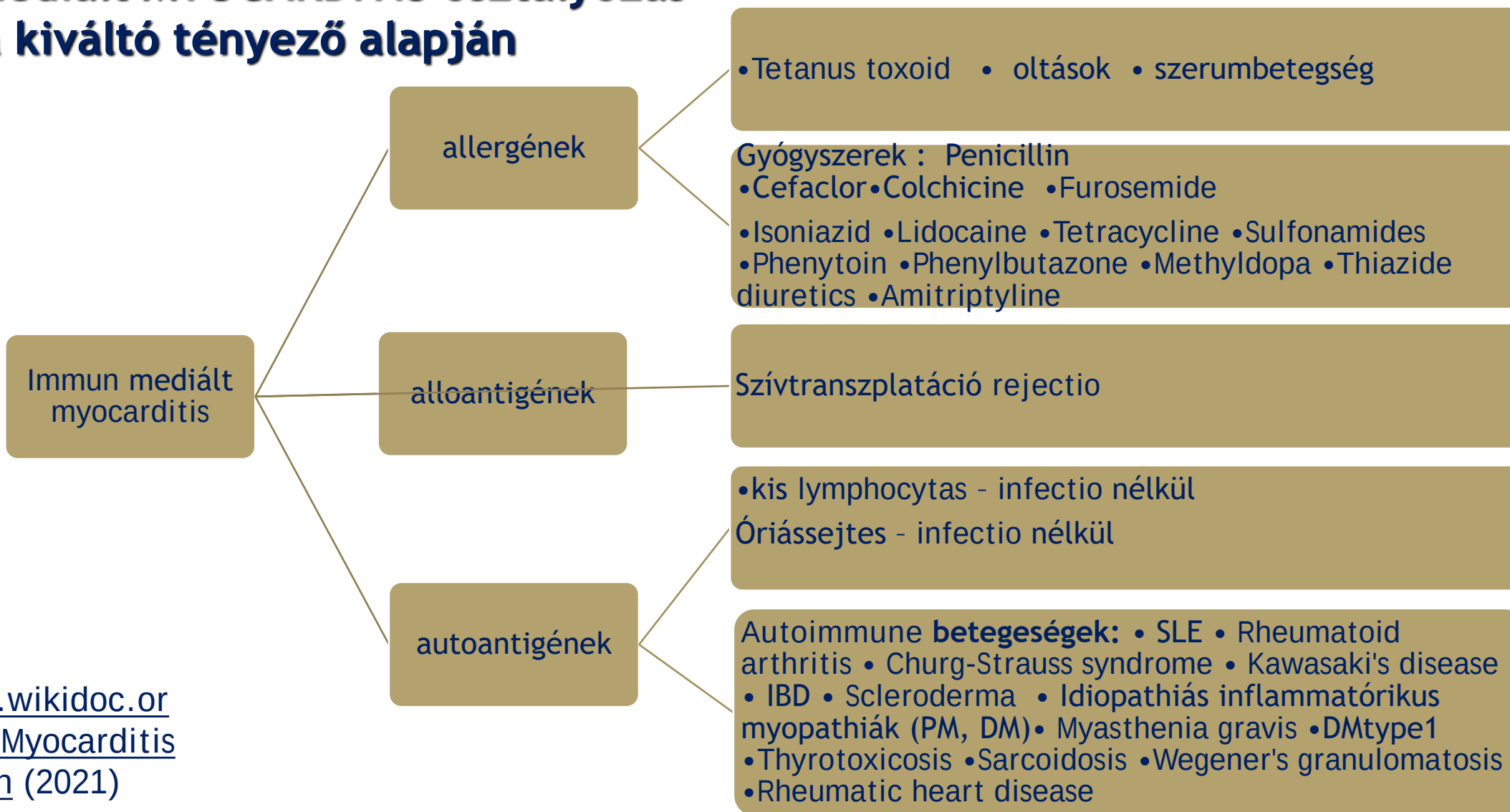
Trachtenberg BH, Hare JM: Inflammatory Cardiomyopathic syndromes. *Circ Res* 2017 Sep 15;121(7):803-818. doi: 10.1161/CIRCRESAHA.117.310221.

INFLAMMATÓRIKUS MYOPERICARDIALIS SZINDROMA ESC 2025

2025 ESC Guidelines for the management of myocarditis and pericarditis. Schulz-Menger J, et al. *Eur Heart J*. 2025. Aug 29;ehaf192. doi: 10.1093/eurheartj/ehaf192.



Immun mediált MYOCARDITIS osztályozás a kiváltó tényező alapján



https://www.wikidoc.org/index.php/Myocarditis_classification (2021)

MYOCARDITIS LEFOLYÁSA

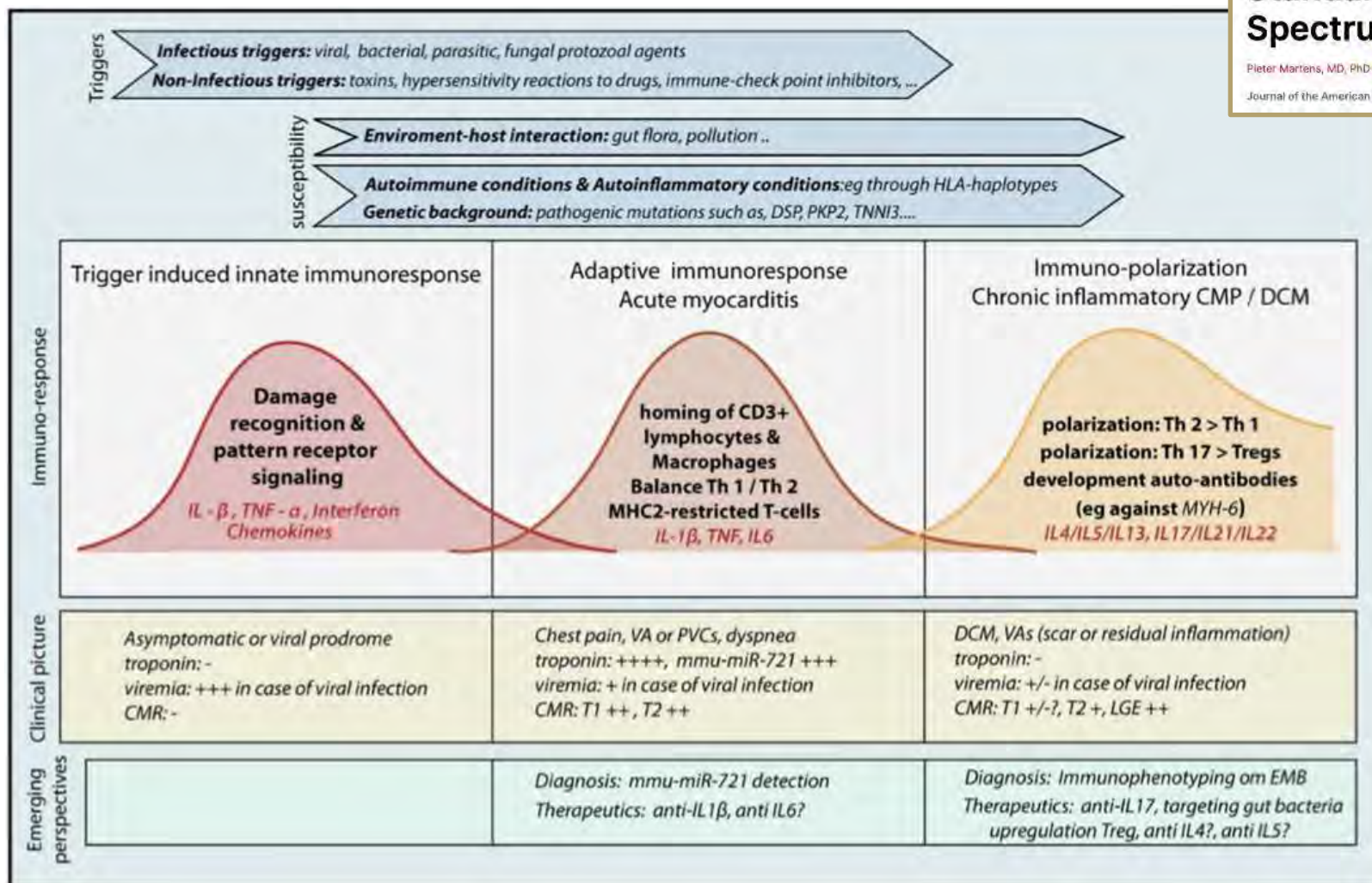
REVIEW ARTICLE

Originally Published 17 August 2023 | Check for updates

Diagnostic Approach for Suspected Acute Myocarditis: Considerations for Standardization and Broadening Clinical Spectrum

Pieter Martens, MD, PhD , Leslie T. Cooper, MD , and W. H. Wilson Tang, MD **AUTHOR INFO & AFFILIATIONS**

Journal of the American Heart Association • Volume 12, Number 17 • <https://doi.org/10.1161/JAHA.123.031454>

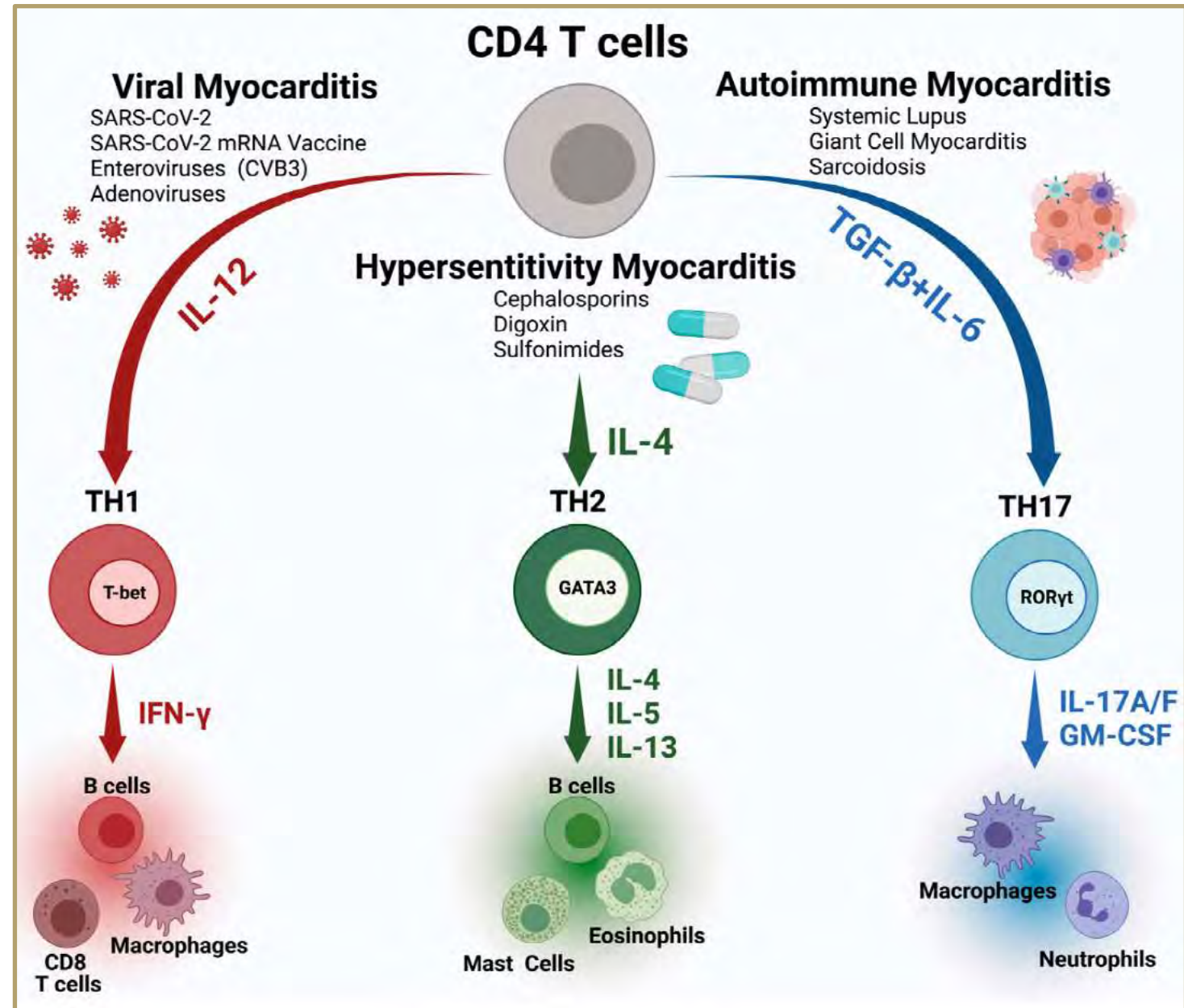


MYOCARDITIS - T HELPER SEJTEK

Meeting the Challenges of Myocarditis: New Opportunities for Prevention, Detection, and Intervention—A Report from the 2021 National Heart, Lung, and Blood Institute Workshop

Daniela Čiháková ^{1,2}, Yang Shi ³, Bishow Adhikari ³, W. Patricia Bandettini ³, Madeleine W. Cunningham ⁴, Narasimhan Danthi ³, Matthias G. Friedrich ⁵, Peter Liu ⁶, Lisa Schwartz Longacre ³, Douglas L. Mann ⁷, Filip K. Swirski ⁸, W. H. Wilson Tang ⁹, Guofei Zhou ¹⁰ and Leslie T. Cooper, Jr. ^{11,*} on behalf of the Workshop Speakers

J. Clin. Med. 2022, 11, 5721. <https://doi.org/10.3390/jcm11195721>



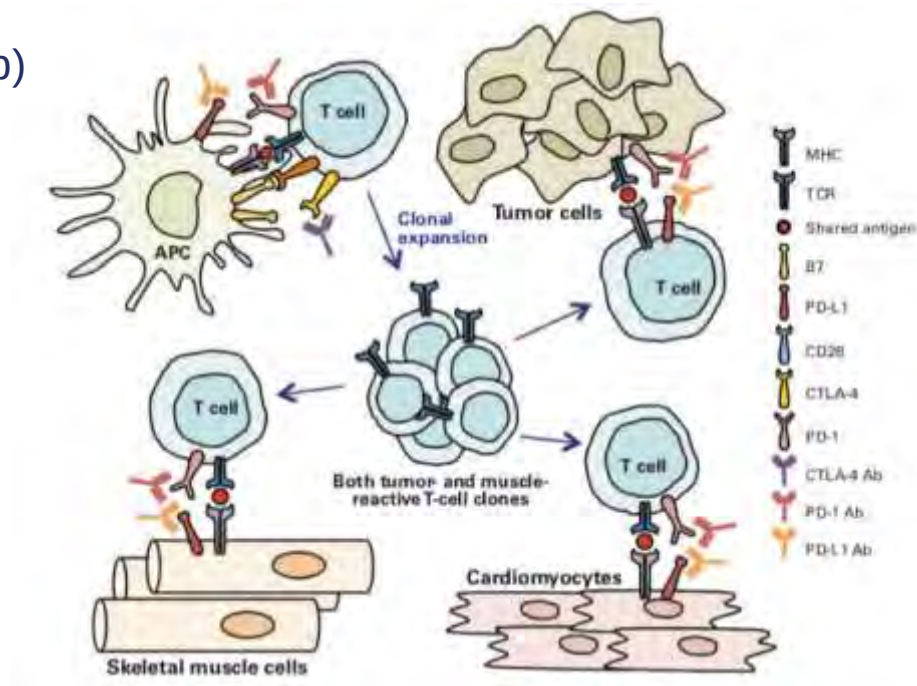
Immun CHECK POINT INHIBITOR related MYOCARDITIS

Fulmináns myocarditis

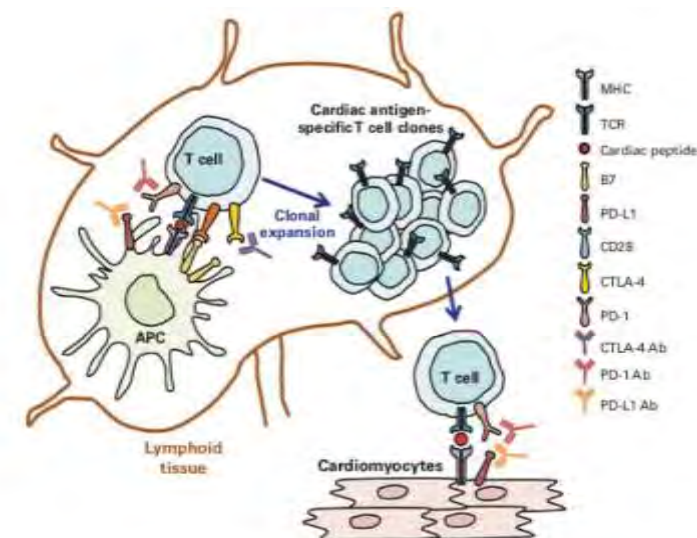
- kezelés 2-4 hetében (median 21 nap)
- Troponin, CK
- EKG, echocardiographia
- (nincs DCM, de akineticus)
- Dyspnoe, myalgia, dylopia, ptosis
- Mortalitas : 38%

Kombinációban különösen
(nivolumab + ipilimumab)
– mortalitás 60 %

Pemprolizumab - PD-1 eII AT
Nivolumab - PD-1 eII AT
Atezolimumab - PD-L1 eII AT
Ipilimumab - CTLA-4 eII AT
Relatlimab - LAG-3



A szívizomzaton és a simaizomzaton ugyanaz az ag presentálódik, mint a tumorsejteken



PD-1-PD-L1 és a CTLA-4-B7 kapcsolódás elmaradásával az autoreaktív T sejt klónok aktiválódhatnak



Myocarditis, Myositis, and Myasthenia Gravis Overlap Syndrome Associated with Immune Checkpoint Inhibitors: A Systematic Review

Dennis M. Tipton^{1,4,5}, Aikem Qadani^{2,6}, Fawzi E. Kishk^{2,6}, Trung D. Nguyen¹, Patrick Chaffin^{2,6}, Navee El Meuti¹, Aashir Srivastava^{2,6}, Elita Galis-Czarvaji¹, Claitor C. Reyes-Gibby^{2,6} and Nabeela K. Wadhwa¹

Diagnostics 2024, 14, 1794. <https://doi.org/10.3390/diagnostics14161794>

PERIPARTUM CARDIOMYOPATHIA

A terhesség utolsó hónapja
v szülés utáni első 5 hónap alatt



➤ Etiológiai teóriák:

- PROLAKTIN -**kóros** neurohormonalis **válasz**
- MYOCARDITIS (vírusinfeció) - biopsziás minta/MR
 - gyulladásos sejtek jelenléte, necrosis, fibroticus átalakulás
- Autoimmun folyamat - **KIMÉRIZMUS**
abnormalis immunválasz a foetalis antigénekre
- **TNF α , IL-6, CRP**
- Genetikai meghatározottság : családi előfordulás, esetleírások
- Selénhiány (egyéb táplálkozási hiányállapot)
- Abnormalis **HAEMODINAMIKAI VÁLASZ**
a terhesség alatti fiziológiás változásokra

Peripartum Cardiomyopathy (PPCM)	
Definition: • Non-ischemic cardiomyopathy with reduced LVEF (<45%) • Commonly presents in the first months postpartum or towards the end of pregnancy	Risk Factors: • African-American race, preeclampsia, hypertension, multigestational pregnancies, age >30 years
Symptoms: • Heart failure symptoms can be confused with common symptoms of normal pregnancy	
Management Options for PPCM	
 During Pregnancy: • Beta-blockers, loop diuretics, hydralazine/isosorbide dinitrate, digoxin, low-molecular-weight heparin • (No ACE/ARB/aldosterone receptor antagonists) • MCS for severe heart failure/cardiogenic shock • Consider early delivery if unstable	 Delivery: • Plan ahead with a Cardio-Obstetrics Team • If unstable, consider hemodynamic monitoring and optimization • Caution for fluid overload, especially after delivery
 After Pregnancy: • Heart failure management. Beta-blockers, enalapril, and spironolactone are compatible with breastfeeding. • Anticoagulation for LV thrombus; consider if severe LV dysfunction (LVEF <35%) • Consider a wearable cardioverter/defibrillator if severe LV dysfunction • Discuss Contraception	
Outcomes	
Worse prognosis with lower LVEF, dilated LV, African-American race, and delayed diagnosis.	
Long-term Outcomes	
• After recovery, optimal duration of medication treatment is unknown • In the case of stopping medications, wean gradually and observe closely • Continue surveillance after recovery	

MYOCARDITIS

Klinikai jellemzők, diagnoszis

Klinikum: nagyon széles skála
DCM esetén : szívelégtelenség tünetei
3:1 ffi dominancia

Vizsgálatok :

Általános labor - AST/ASO

Troponin (CK)

Immunserológia- szívizom ellenes AT

Infect serologia

Mellkas Rtg,

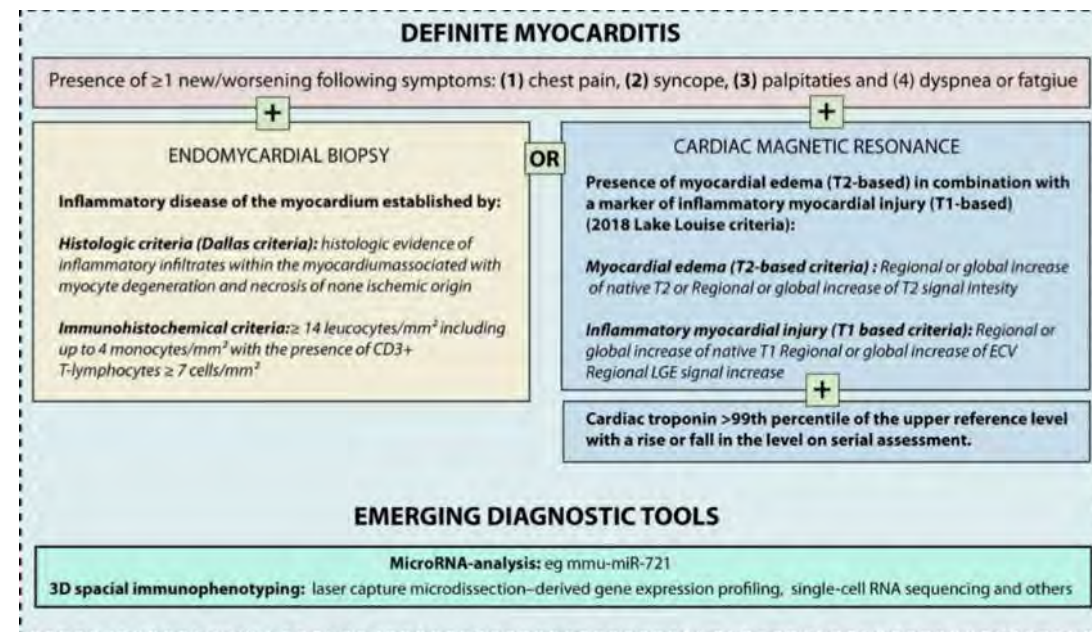
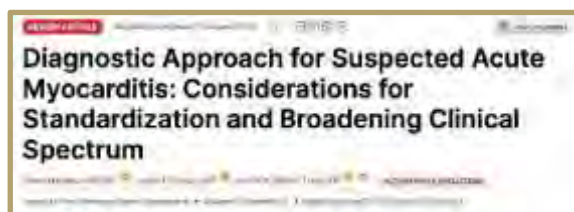
Echocardiographia

Cardiális MR

Izotópdiagnosztika

(Ga 67, In111-el jelölt antimyozinAT)

Endomyocardialis biopszia



TIER 2: CONFIRMATORY TIER	Fulminant myocarditis	Complicated myocarditis	Uncomplicated high Risk myocarditis	Uncomplicated low risk myocarditis
	In hospital management			Outpatient
	EMB			CMR
	EMB - indications: 1) Suspected acute myocarditis with cardiogenic shock (early EMB) or complicated myocarditis (potentially CMR/EVM guided) 2) Suspected chronic inflammatory CMP: with persistent reduced LVEF/VA or AV block and to respond to OMT after 3 months. 3) Myocarditis with eosinophilia or use of ICI (continuation ICI?) EMB - analysis: Histopathology, Quantitative real-time PCR* and immunohistochemistry** EMB - Optimization If stable consider EMB guided by imaging (CMR or FDG-PET) or guided using EVM left/right sided biopsy or biventricular			CMR- indications: 1) First line choice in patients with uncomplicated myocarditis 2) Can be used to guide EMB in complicated myocarditis 3) CMR should be performed in fulminant myocarditis after disease stabilization to evaluate extent/presece of inflammation CMR- prognostification: High risk CMR features: presence of LGE septal /midwal LGE, LV dysfunction (LVEF $<50\%$), myocardial fraction with T2 time >80 ms in acute phase CMR- optimization: timing: not too early (first days), not too late ($> 3-4$ weeks)

MYOCARDITIS

Klinikai jellemzők, diagnózis

Klinikum: r
DCM esetér
3:1 ffi dom

Vizsgálatok
Általános la
Troponin (C
Immunsero
Infect sero
Mellkas Rtg
Echocardio
Cardiális M
Izotópdiagr
(Ga 67, In1
Endomyoc

If diagnostic criteria for myocarditis and/or pericarditis are fulfilled^a

Myocarditis

Definite	Clinical presentation ^b and CMR- or EMB-proven
Possible	Clinical presentation ^b with at least 1 additional criterion CMR- or EMB-uncertain or not available
Unlikely/rejected	Only clinical presentation ^b without additional criteria

Additional criteria beyond clinical presentations^b

Myocarditis

Clinical ^b	Non-specific findings
ECG ^c	ST-T changes
Biomarkers	Troponin elevation
Imaging ^d	Abnormal strain, wall motion, reduced EF Myocardial oedema and/or LGE (CMR findings)



2025 ESC Guidelines for the management of myocarditis and pericarditis

Developed by the task force for the management of myocarditis and pericarditis of the European Society of Cardiology (ESC)

Endorsed by the Association for European Paediatric and Congenital Cardiology (AEPC) and the European Association for Cardio-Thoracic Surgery (EACTS)

ESC GUIDELINES



percentile of the upper reference level
level on serial assessment.

g, single-cell RNA sequencing and others

high
tis

Uncomplicated low
risk myocarditis

Outpatient

CMR

patients with uncomplicated myocarditis
de EMB in complicated myocarditis
formed in fulminant myocarditis after di-
evaluate extent/presece of inflammation

cation:

es: presence of LGE septal /midwal LGE, LV
>0%), myocardial fraction with T2 time >80

n:

imaging needs only (first days), not too late (> 3-4weeks)

CARDIALIS MR

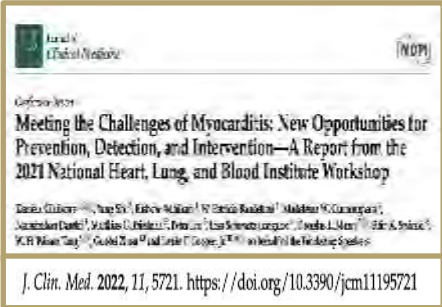
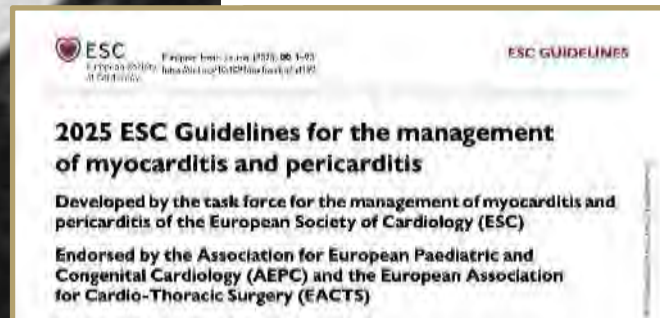
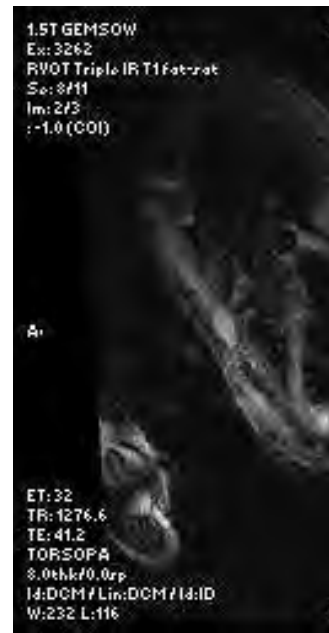


Table 1. An Overview of Imaging Modalities Used in the Evaluation of Patients Presenting with Myocarditis.

	Transthoracic Echocardiography	Positron Emission Tomography	Cardiovascular Magnetic Resonance
Ventricular volumes, myocardial mass, systolic function	+++	++	++++
Ventricular strain, myocardial mechanics	++++	++	+++
Inflammation	++	++++	++++
Fibrosis/Infiltration	++	+++	++++
Pericardium/Pericardial effusion	+++	++	++++
Alternate diagnoses of chest pain syndromes	++	+++	++++
Cost	Inexpensive	Most expensive	Increasingly affordable; cost-effective
Strengths	Portable, widely accessible in most medical institutions, rapid assessment of systolic function and wall motion abnormalities	Highly validated in inflammatory state; molecular and metabolic characterization	High spatial resolution, highly reproducible, volumetric coverage, multi-faceted tissue characterization
Limitations	Dependence on optimal acoustic window, nonspecific findings	Exposure to ionizing radiation with use of nuclear tracers, special preparation required	Specific Hardware/software requirements with fewer centers of excellence available; claustrophobia, use of gadolinium contrast

A higher number of “+” symbols indicates a higher strength or utility in the category.

CARDIALIS MR



Criterion	Methods	Example images and pathology		Parameters for reporting	
				For myocarditis	For pericardial involvement
T2-based criterion	T2-weighted imaging or T2 mapping	Myocardial oedema	Pericardial oedema	- Presence, extent, and location of oedema (T2-weighted) - Regional high T2 SI or global high T2 SI (T2-weighted) - Regional or global increase of myocardial T2 times	High signal intensity of the pericardium in T2-mapping or T2-weighted imaging
		Myocardial oedema/diffuse fibrosis	Pericardial oedema/diffuse fibrosis	- Description of focal increases - Regional or global increase of native myocardial T1 times - Regional or global increase ECV values	High signal intensity of the pericardium in T1-mapping
T1-based criterion	Native T1 mapping/post-contrast T1 mapping (ECV)/T1-weighted imaging				
	Late gadolinium enhancement	Focal myocardial fibrosis/scar	Pericardial inflammation/scar	- Presence, pattern, extent, and location of LGE (positive if areas with high SI in a non-ischaemic distribution pattern) - Thrombi (if present) - Total LGE/LV mass (%) (no routine)	High signal intensity of the pericardium in LGE images
Supportive criterion	Cine imaging	Functional and wall motion abnormalities	Haemodynamic compromise	- Regional wall-motion abnormalities - Cardiac function (e.g. LVEF, RVEF) and volume parameters	- Presence, composition, and extent of pericardial effusion - Haemodynamic relevance of pericardial effusion - Diameter of pericardial effusion
Updated Lake Louise Criteria (LLC) for myocarditis					
CMR-proven myocarditis= 2 out of 2 updated LLC main criteria fulfilled		T2-based criterion Myocardial oedema	Abnormal T2-mapping or T2-weighted imaging	Pericardial abnormalities	
CMR-uncertain myocarditis= only 1 out of 2 updated LLC main criteria fulfilled		Main criteria		Supportive criteria	
		T1-based criterion Non-ischaemic myocardial injury	Abnormal T1-mapping, ECV or LGE	Systolic LV-dysfunction	

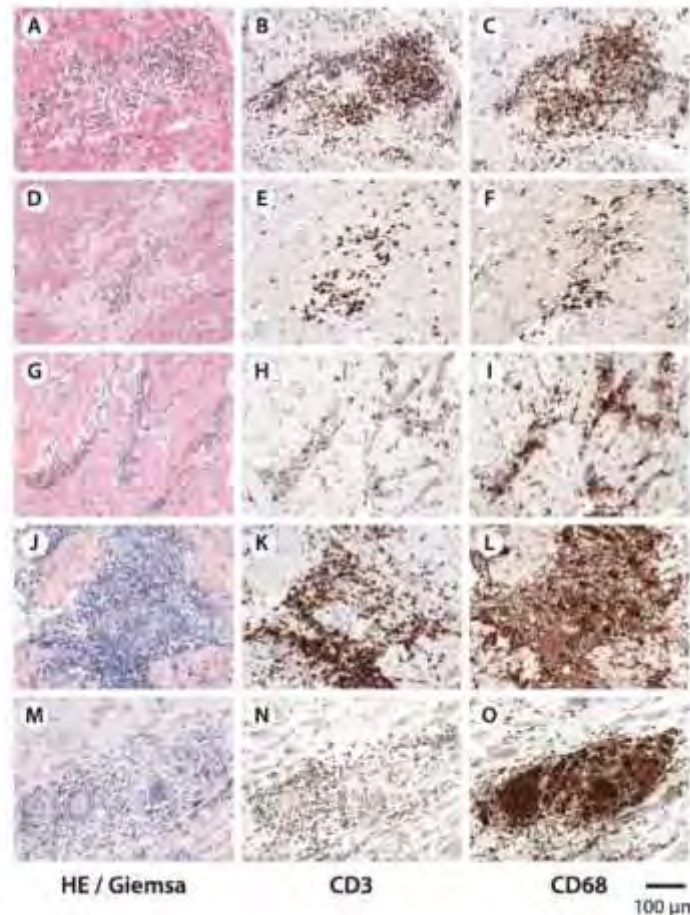
ENDOMYOCARDIALIS BIOPSZIA



Indikáció :

- High risk betegek
- Haemodinamikai instabilitás
- Intermediate risk, de nem reagál a th-ra
- Spec forma felmerül
- Vírus (kórokozó) genom kimutatás

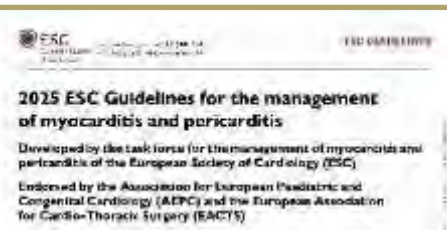
Myocarditis szövettani diagnosztikus kritériumok



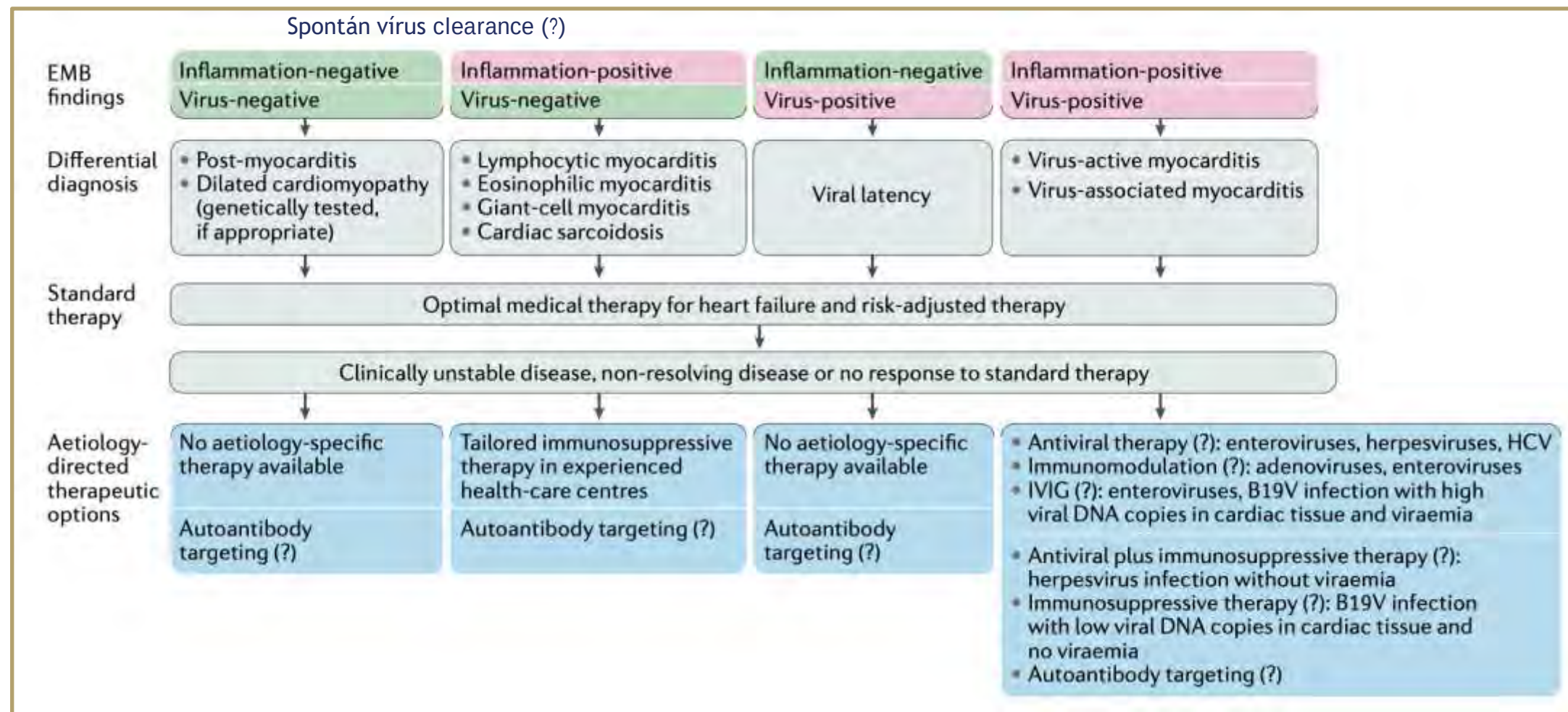
	Predominant inflammatory cells	Myocyte necrosis	Infections PCR positive (viruses, etc.)
Active lymphocytic myocarditis	CD3 ⁺ T lymphocytes >7/mm ² , CD68 ⁺ macrophages	yes	yes/no
Persistent lymphocytic myocarditis	CD3 ⁺ T lymphocytes >7/mm ² , CD68 ⁺ macrophages	yes	yes/no
Resolved lymphocytic myocarditis	–	no	yes/no
Eosinophilic myocarditis (acute stage)	Eosinophils, CD3 ⁺ T lymphocytes, CD68 ⁺ macrophages	yes	yes/no
Giant-cell myocarditis (acute stage)	Eosinophils, CD68 ⁺ giant cells, CD3 ⁺ T lymphocytes, CD68 ⁺ macrophages	yes	no
Sarcoidosis	CD68 ⁺ giant cells, granuloma, CD3 ⁺ T lymphocytes, CD68 ⁺ macrophages	yes/no	no

2025 ESC Guidelines for the management of myocarditis and pericarditis.
Schulz-Menger J, et al. Eur Heart J. 2025. Aug 29;ehaf192. doi: 10.1093/eurheartj/ehaf192.

MYOCARDITIS KEZELÉS



Carsten Tschöpe et al. Myocarditis and inflammatory cardiomyopathy: current evidence and future directions. Nat Rev Cardiol . 2021 Mar;18(3):169-193.
doi: 10.1038/s41569-020-00435-x. Epub 2020 Oct 12.



MYOCARDITIS KEZELÉS

Carsten Tschöpe et al. Myocarditis and inflammatory cardiomyopathy: current evidence and future directions. Nat Rev Cardiol . 2021 Mar;18(3):169-193. doi: 10.1038/s41569-020-00435-x.Epub 2020 Oct 12.

2025 ESC Guidelines for the management of myocarditis and pericarditis. Schulz-Menger J, et al. Eur Heart J. 2025. Aug 29;ehaf192. doi: 10.1093/eurheartj/ehaf192.

Szívtranszplantáció (HTX) - DCM, szívelégtelenség esetén

		1 vonal	2 vonal	3 vonal
Vírus - GYULLADÁS +	lymphocytas	Steroid : enyhe : 1 mg/ttkg /nap Súlyos : 4-14 mg/ttkg/nap, majd 1 mg/ttkg /nap	Steroid +AZA, Cy, MMF, MTX	IVIg , PEX
	Eosinophyl	Steroid : enyhe : 1 mg/ttkg /nap Súlyos : 4-14 mg/ttkg/nap, majd 1 mg/ttkg /nap	Steroid +AZA, Cy, MMF, MTX	IVIg , PEX
	Óriássejtes	Steroid : enyhe : 1 mg/ttkg /nap Súlyos : 4-14 mg/ttkg/nap, majd 1 mg/ttkg /nap + AZA, Cy, MMF,	ATG, CYC, RTX,	
	Sarcoidosis	Steroid : enyhe : 1 mg/ttkg /nap Súlyos : 4-14 mg/ttkg/nap, majd 1 mg/ttkg /nap	MTX, AZA, MMF, CYC	Infliximab/adali mumab, RTX
	Lyme	A:p.o AB - 21 nap - Doxyxcyclin, Amoxicillin, Cefuroxim - B: Iv-Cetriaxone	Iv Cefotaxim / penicillin G	
	ImmunCP inhibitor	Nagy dózisú steroid 500-1000 mg/nap 3 napig, majd leépítés Súlyos : 7-14 mg/ttkg/nap) - (24-48 óra)	MMF, ATG, abatacept, alemtuzumab	Infliximab/adali mumab, RTX
VÍRUS + Gyulladás -		Antiviralis, IVIg		
VÍRUS + GYULLADÁS +		BetaINF, Immunadszopcio+ IVIg pocapavir, pleconaril, telbivudine stb, HCQ,	IL-6 R antag., IL-1beta inhib	Anti-IL-17, IL-1 R antibody,

MYOCARDITIS KEZELÉS

Carsten Tschöpe et al. Myocarditis and inflammatory cardiomyopathy: current evidence and future directions. Nat Rev Cardiol . 2021 Mar;18(3):169-193. doi: 10.1038/s41569-020-00435-x.Epub 2020 Oct 12.

2025 ESC Guidelines for the management of myocarditis and pericarditis. Schulz-Menger J, et al. Eur Heart J. 2025. Aug 29;ehaf192. doi: 10.1093/eurheartj/ehaf192.

		1 vonal	2 vonal	3 vonal
Vírus - GYULLADÁS +	lymphocytas	Steroid : enyhe : 1 mg/ttkg /nap Súlyos : 4-14 mg/ttkg/nap, majd 1 mg/ttkg /nap	Steroid +AZA, Cy, MMF, MTX	IVIg , PEX
	Eosinophyl	Steroid : enyhe : 1 mg/ttkg /nap Súlyos : 4-14 mg/ttkg/nap, majd 1 mg/ttkg /nap	Steroid +AZA, Cy, MMF, MTX	IVIg , PEX
	Óriássejtes	DCM, szívelégtelenség, cardiogen shock esetén - Szívelégtelenség ajánlás szerinti kezelése - Keringéstámogatás, lélegeztetés stb - Mechanikus keringéstámogatás - ECMO, VAD Szívtranszplantáció (HTX)		
	Sarcoidosis			
	Lyme			
	ImmunCP inhibitor			
		ma		
		Súlyos : 7-14 mg/ttkg/nap) - (24-48 óra)		
VÍRUS + gyulladás		Antiviralis, IVIg		
VÍRUS + GYULLADÁS +		BetaINE, Immunadszopcio+ IVIg pocapavir, pleconaril, telbivudine stb, HCQ,	IL-6 Rantag., IL-1beta inhib	Anti-IL-17, IL-1 R antibody,

ENDOCARDITIS

ENDOCARDITIS / VALVULOPATHIA

➤ Infectiv

➤ Non-infectiv

-NBTE - non-bacterial-thrombotic
endocarditis

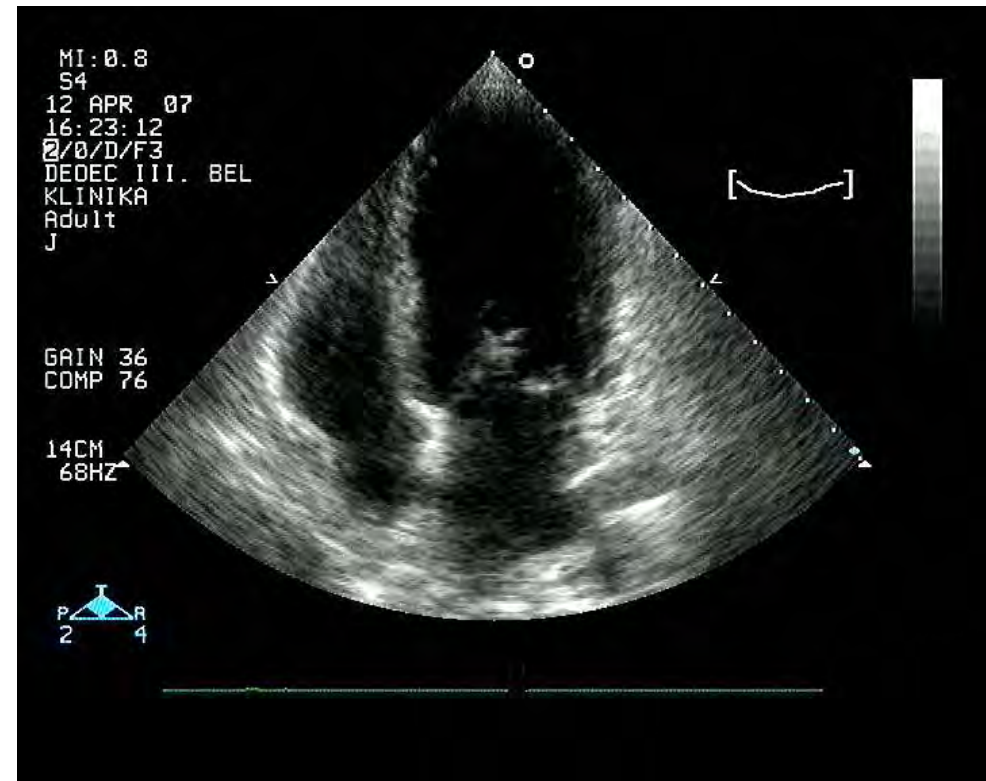
=Asepticus

=Libman-Sacks (SLE, APS)

=Verrucosus

=Marantic

➤ Loeffler féle (eosinophylia)



ENDOCARDITIS



NBTE
Libman-sacks

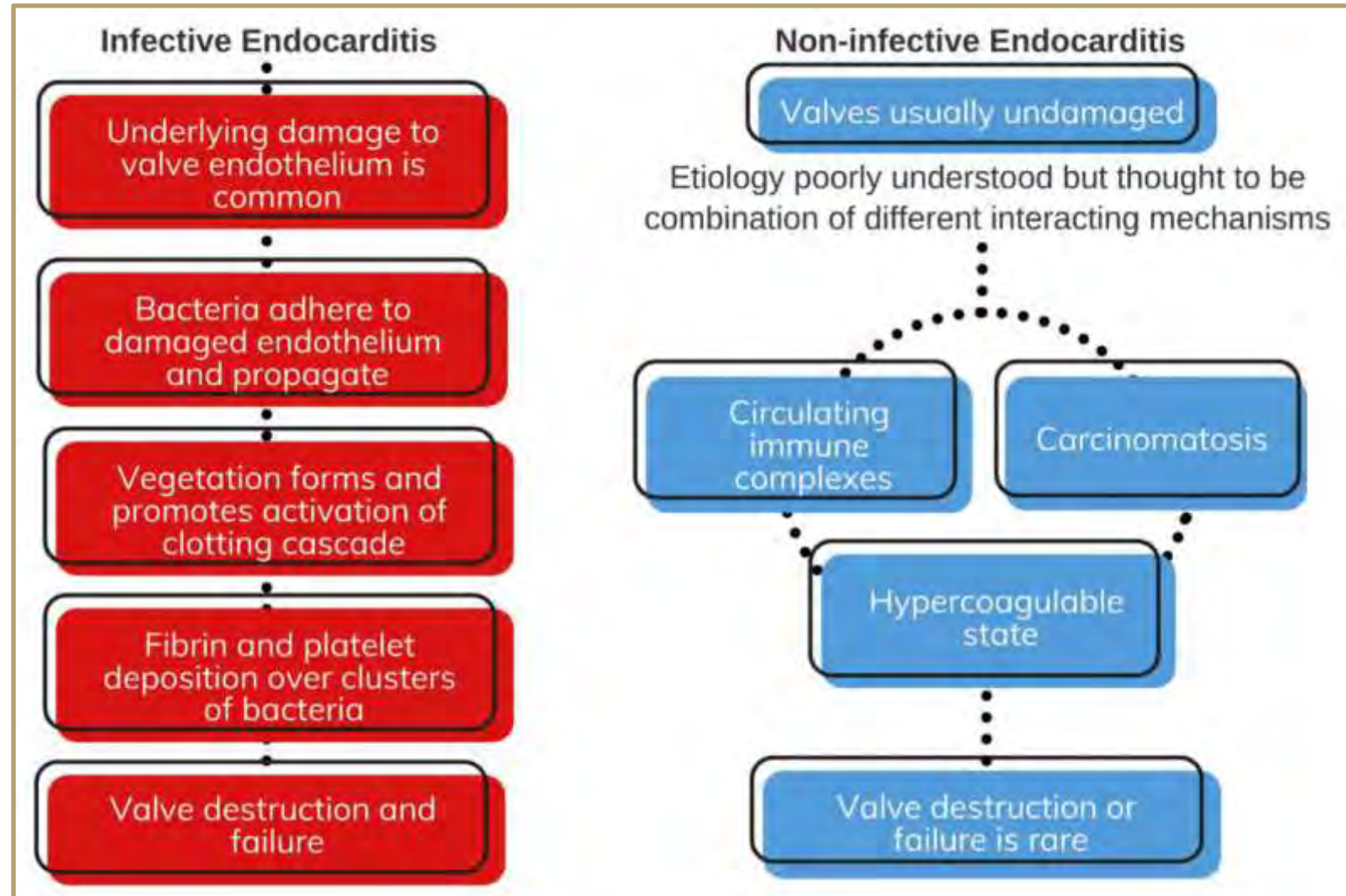
Harriet Hurrell , Ross Roberts-Thomson , Bernard D Prendergast : **Non-infective endocarditis**
Heart. 2020 Jul;106(13):1023-1029. doi: 10.1136/heartjnl-2019-315204.Epub 2020 May 6.

alapbetegség	NBTE előfordulása
Autoimmun betegségek	
• SLE	4-43 %
• APS	41 %
• RA	esetismertetések
• GPA (granulomatosis poliangitissel)	esetismertetések
• Behcet	esetismertetések
HIV	esetismertetések
Neoplasma	19 %
• adenocarcinoma	(neoplasma + NBTE esetek) 62 %-a
• Pancreas neoplasma	(neoplasma + NBTE esetek) 25-50 %-a
• Tüdő neoplasma	(neoplasma + NBTE esetek) 25-30 %-a
Hypercoagulabilitással járó állapotok	50 % fölött
• DIC	
• Sepsis	
• Égés	
• APS	
• Neoplasma	NBTE : non-bacterial-thromboticus endocarditis

ENDOCARDITIS

PATHOPHYSIOLOGIA

Harriet Hurrell , Ross Roberts-Thomson , Bernard D Prendergast : **Non-infective endocarditis**
Heart. 2020 Jul;106(13):1023-1029. doi:
10.1136/heartjnl-2019-315204.Epub 2020 May 6.

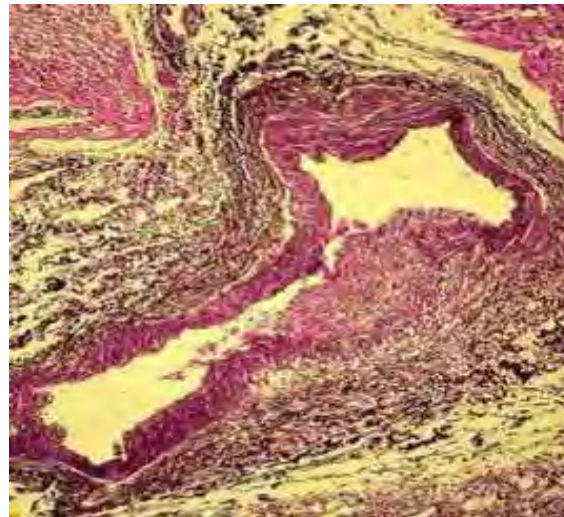


CORONARITIS

**ACCELERÁLT
ATHEROSCLEROSIS**

CORONARITIS

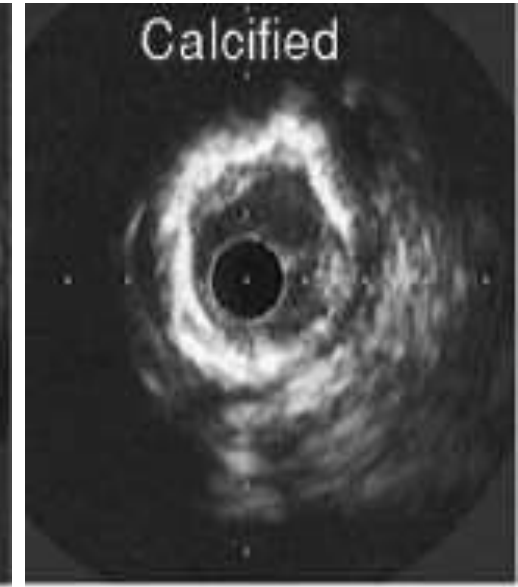
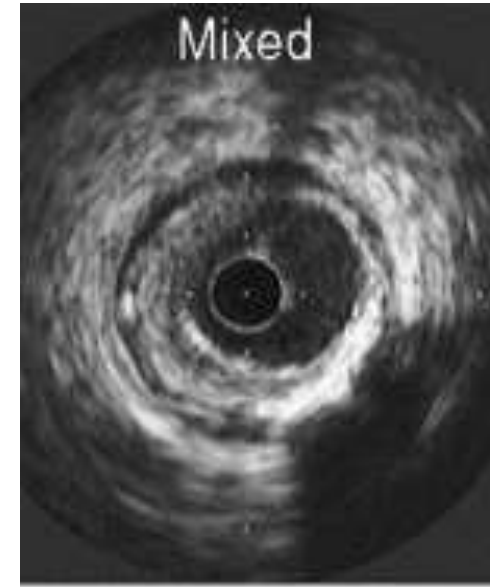
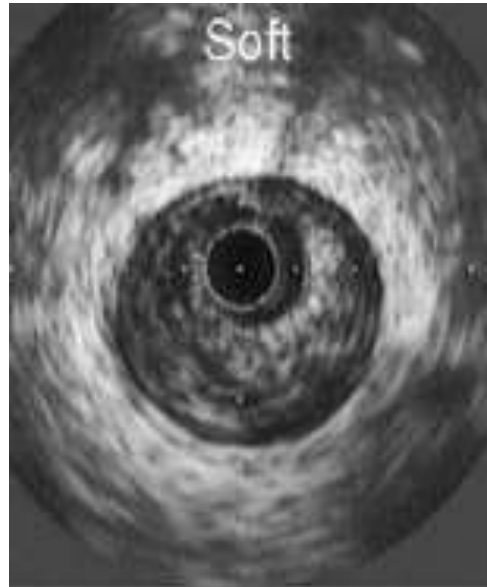
- VASCULITIS részjelensége
- Takayasu, Kawasaki, PAN, SLE, RA
- Klinikum:
- Acut coronaria syndroma képe
- Labor, EKG, echocardiographia, coronarographia



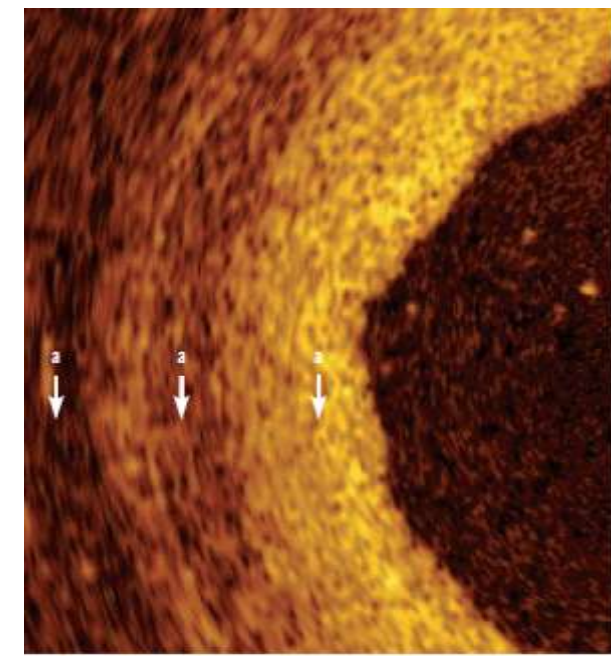
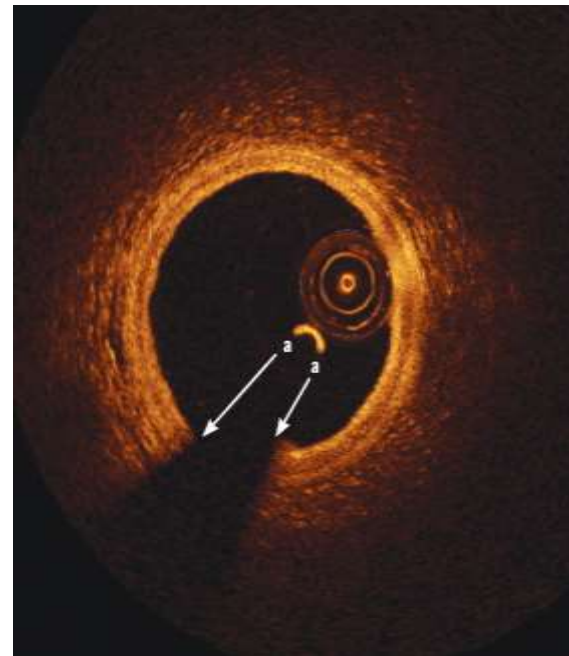
Arteriogramm- a.coronaria
aneurysma és occlusio

CORONARITIS

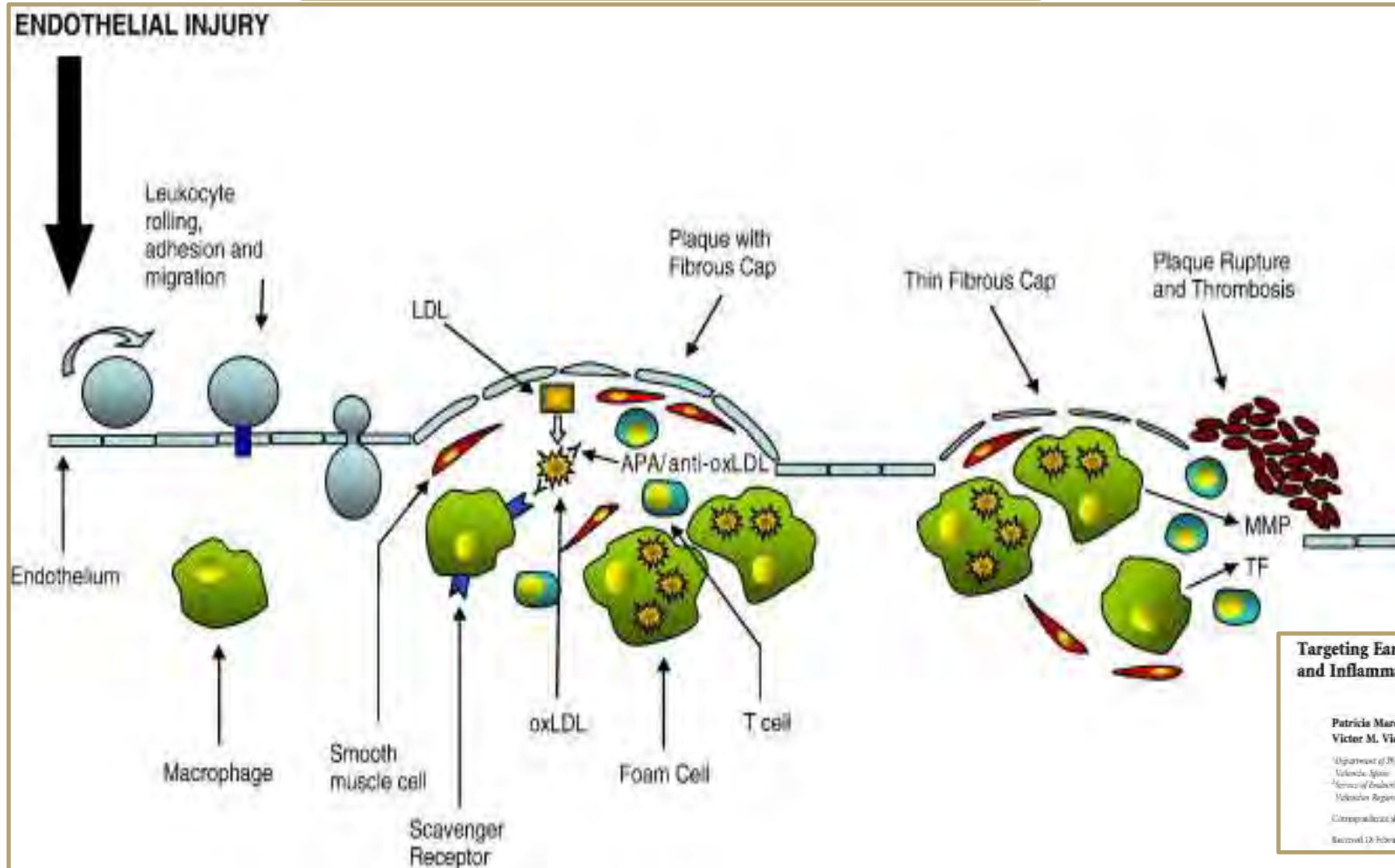
IVUS
Intravascularis ultrasonographia
Plaque morfológia



OCT
Optikai koherencia
tomographia
Normál coronaria anatómia



ATHEROSCLEROSIS



1871-lipid-elmélet

1986 krónikus gyulladás (nem spec)

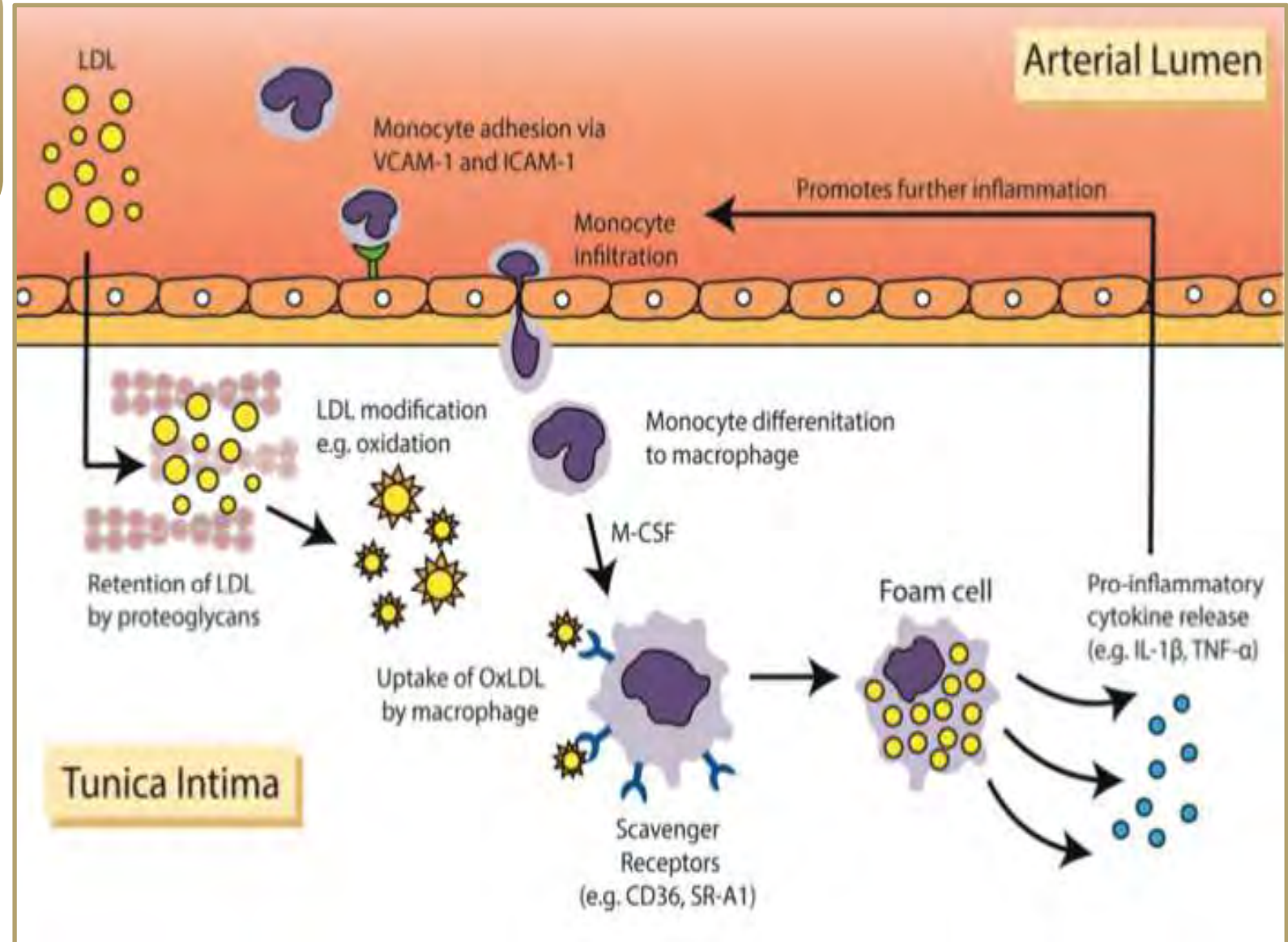
2012 erek falában
Tsejt-APC interakció
(Ag specifikus)

Elsődleges antigének :
oxLDL, HSP, béta 2GPI

ATHEROSCLEROSIS



MACROPHAGOK SZEREPE



ACS / PLAQUE RUPTÚRA KIALAKULÁSÁNAK MECHANIZMUSA

Instabil plaque

- vékony fibrin sapka + kollagén
- M1 macroph - MMPs termelésével a fibrinsapka kiszakadását elősegítik
- Innate immunrendszer sejtjei:
- Th1 - IFN- γ
- Th: CD28+null (IFN- γ , TNF- α termelés, apoptózis rezistens)

Plaque ruptúra

Plaque erosio

- nem kíséri kimutatható gyulladás
- lokális endothelsejt károsodás (oxidatív stressz, haemodinamikai zavar, hypoxia stb)
- TLR aktiváció - neutrofil NETózis -
- thrombusképződés

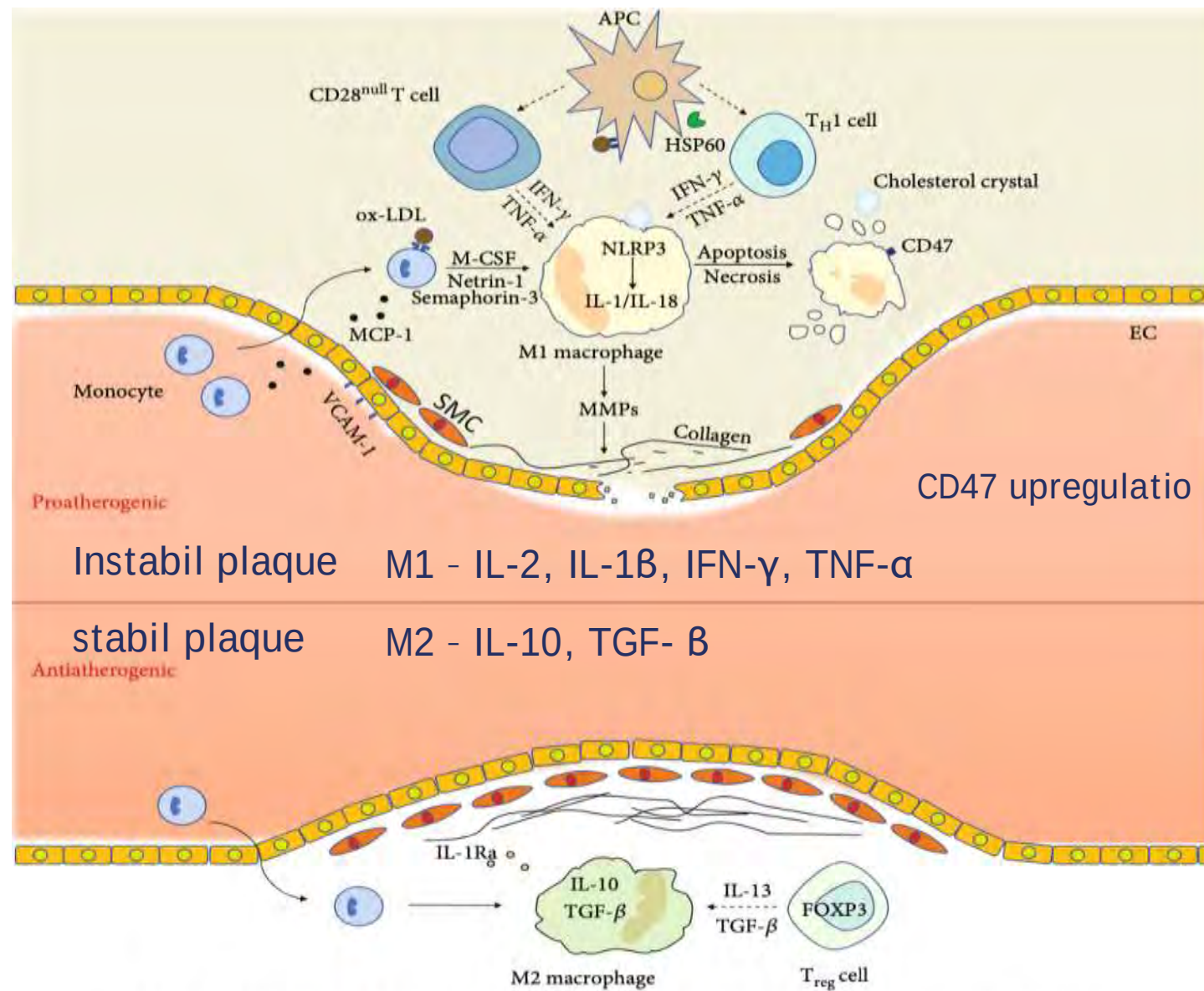


FIGURE 1: Immune and inflammation pathways in the pathogenesis of acute coronary syndrome.

ACCELERÁLT ATHEROSCLEROSIS GYULLADÁSOS RHEUMATOLÓGIAI KÓRKÉPEKBEN

- Chronicus autoimmun gyulladásal járó megbetegedésekben észlelhető (RA, SLE, AP sy, Vasculitis, Myositis, SP A)
- Szisztémás sclerosisban diffúz vasculopathia, a microvascularis eltérések dominálnak



- A tradicionális rizikófaktorokkal nem magyarázható fokozott cardiovascularis érintettség észlelhető
 - nem kontrollált krónikus gyulladás
- accelerált atherosclerosis

- Az autoimmun megbetegedésekhez társuló korai atherosclerosis nagyon gyakran tünetmentes

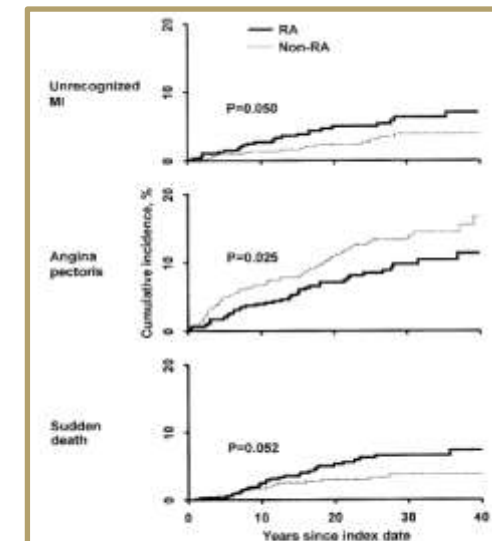


Figure 1. Cumulative incidence of unrecognized myocardial infarction (MI), angina pectoris, and sudden deaths in the rheumatoid arthritis (RA) and non-RA cohorts according to length of time since index date, adjusted for the competing risk of death.

ARTHRITIS & RHEUMATISM
Vol. 43, No. 3, February 2001, pp 465-471
DOI: 10.1002/1529-0928(200102)43:3<465::AID-ARTHR43>3.0.CO;2-1

Increased Unrecognized Coronary Heart Disease and Sudden Deaths in Rheumatoid Arthritis
A Population-Based Cohort Study

Hélène Maréchal-Konken, Cynthia N. Claessen, Paulo J. Nogueira, Karla V. Ballman, Véronique L. Roger, Steve J. Jacobson, and Sherine E. Gabriel

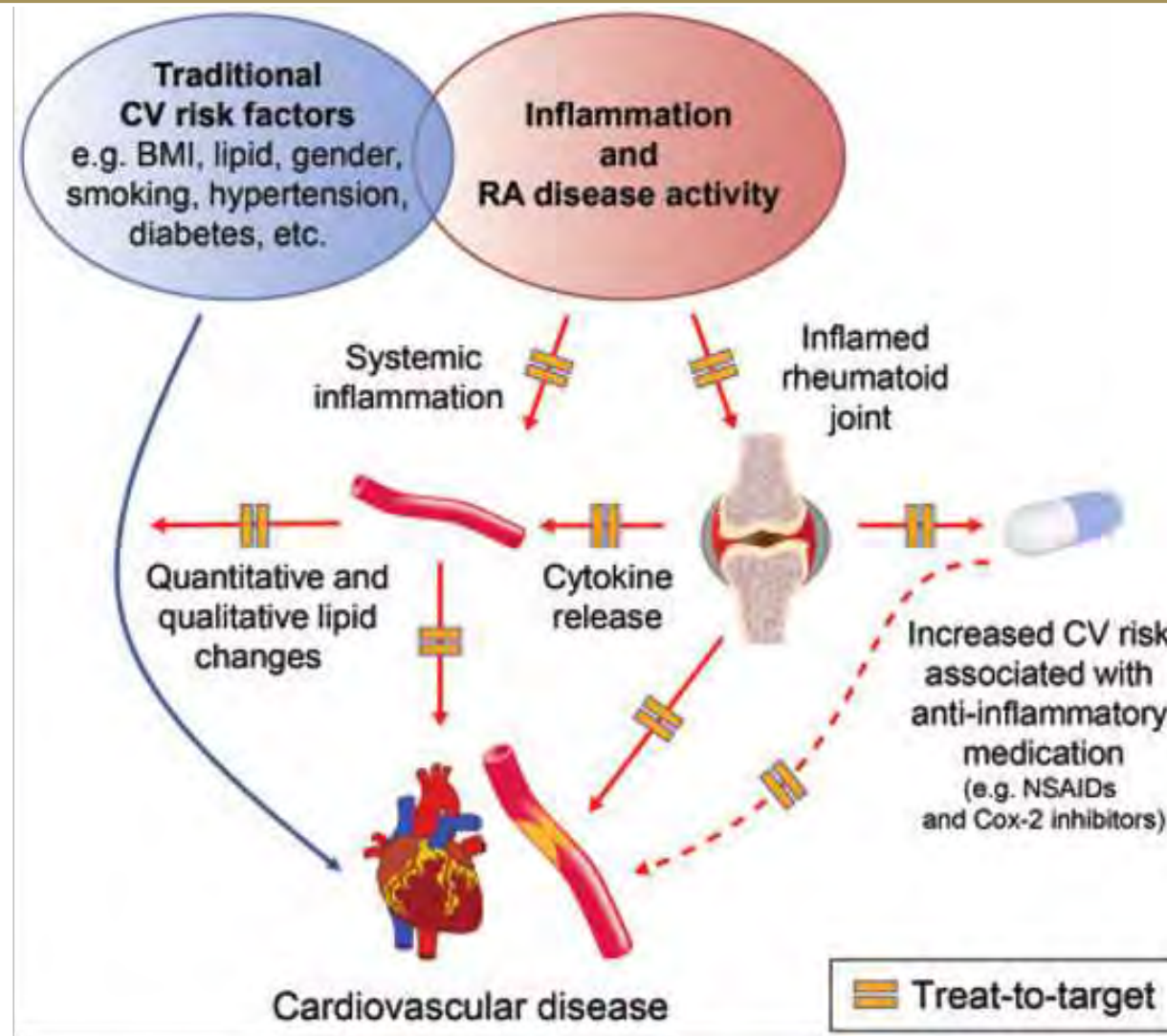
EMELKEDETT CARDIOVASCULARIS RIZIKÓ OKAI

TRADICIONALIS CV RIZIKÓ

általános CV
rizikófelmérés

DM, hypertonia,
doh, mobilitás

doi:10.1093/
rheumatology/k
eu224



ALAPBETEGSÉG FÜGGŐ RIZIKÓ

SPA: Aortitis, Aorta
billentyű elégtelenség,
AV block

SLE : Liebmann Sacks,
Loeffler endocarditis

SSC: myocardialis fibrosis

KEZELÉS DEPENDENS RIZIKÓ

NSAID
corticosteroidok
antiTNF α
JAK gátlók

ACCELERÁLT ATHEROSCLEROSIS THERAPIÁS MEGFONTOLÁSOK

Ann Rheum Dis. 2017 Jan;76(1):17-28. doi: 10.1136/annrheumdis-2016-209775. Epub 2016 Oct 3. EULAR recommendations for cardiovascular disease risk management in patients with rheumatoid arthritis and other forms of inflammatory joint disorders: 2015/2016 update. [Agca et.al.](#)

- **Szisztémás gyulladás tartós szuprimálása a cardiovascularis rizikót csökkenti**
(MTX, antiTNF alfa, MMF-SLE- Jak gátlók !)
- **A hagyományos rizikófaktorok tekintetében szigorúbb célérték betartása**
(EULAR 2015) – RA 1,5x
- **Statin kezelés – PLEIOTROPH HATÁSOK**
 - 30-50%-al csökkentik a morbiditást és mortalitást RA-ban (Packard)
 - Placebo controlált vizsgálat (Mc Garey):40 mg/nap atorvastatin:
LDL-C (45%), triglycerid (18%), betegség aktivitás (10%), CRP(50%), We(28%) csökkenés
- **Rendszeres szűrővizsgálatok**
- **Rendszeres CV szűrés**

KLASSZIKUS RIZIKÓMODELLEK

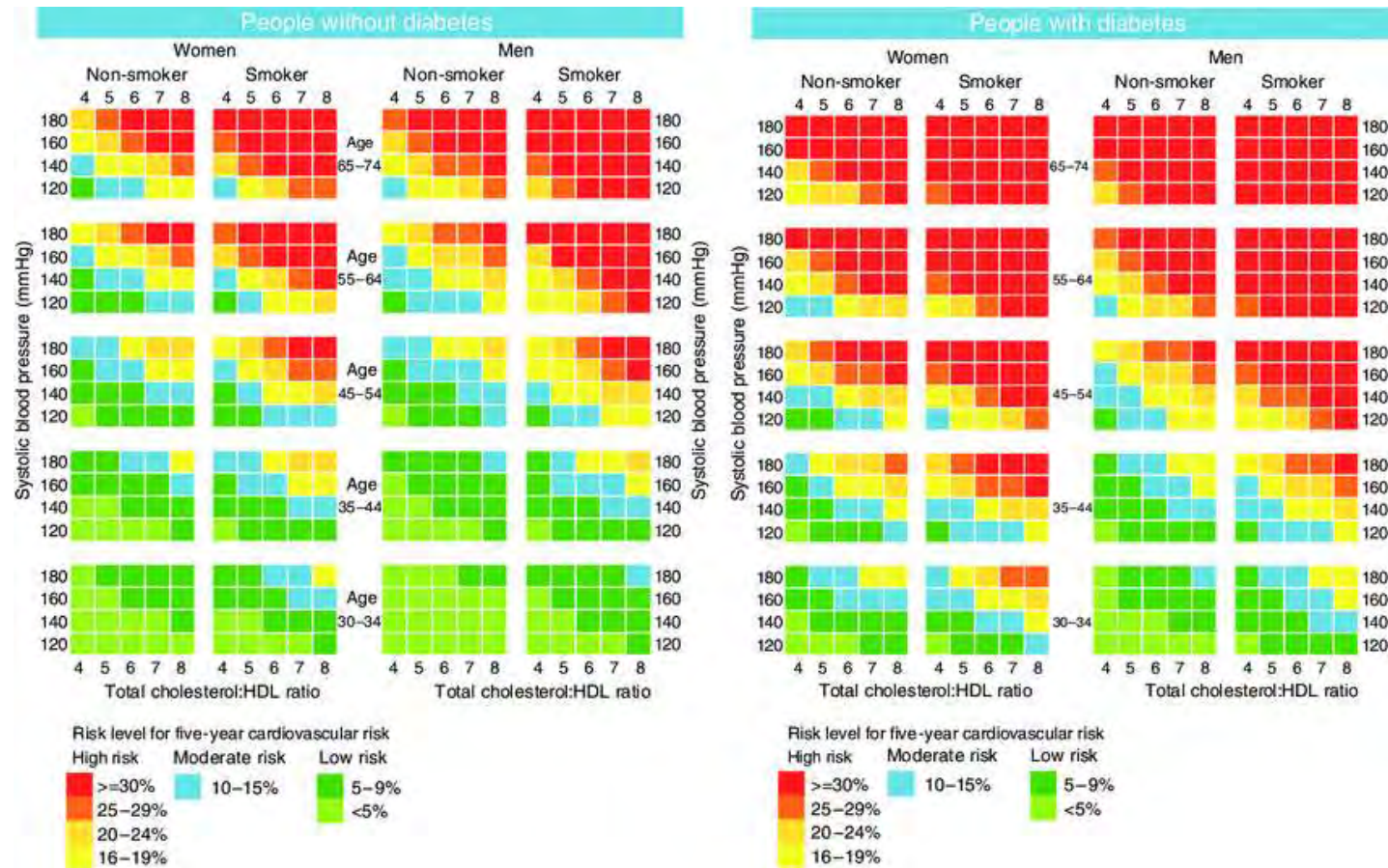
SCORE :

Framingham : www.mdcalc.com

ASCVD calc : www.tools.acc.org

széles körben használatosak

gyulladásos reumatológiai
kórképekben alulbecsülik a
tényleges rizikót
(nem számolnak a gyulladásos
aktivitással és immunmediált
tényezőkkel)



SCORE2 risk prediction algorithms: new models to estimate 10-year risk of cardiovascular disease in Europe. SCORE2 working group and ESC Cardiovascular risk collaboration. European Heart Journal (2021) 42, 2439–2454 .(doi: 10.1093/eurheartj/ehab310)

KARDIOVASCULARIS RIZIKÓ FELMÉRÉS - QRISK3

REUMA SPECIFIKUS MODELL

10 éves rizikófelmérés

Validálás 2017

Figyelembe veszi a krónikus
gyulladás okozta emelkedett
rizikót

www.qrisk.org

Development and validation of QRISK3 risk prediction algorithms to estimate future risk of cardiovascular disease: prospective cohort study

BMJ 2017; 357 doi: <https://doi.org/10.1136/bmj.j2099> (Published 23 May 2017) Cite this as: BMJ 2017;357:j2099

Welcome to the QRISK®3-2017 risk calculator

This calculator is only valid if you do not already have a diagnosis

Reset Copyright Algorithm

About you

Age (25-84):

Sex: ☒ Male ☐ Female

Ethnicity: ☒ White or not stated ☐ Other

UK postcode: leave blank if unknown

Postcode:

Clinical information

Smoking status: ☒ Heavy smoker (20 or over) ☐ Other

Diabetes status: ☒ None ☐ Yes

Angina or heart attack in a 1st degree relative <60? ☐

Chronic kidney disease (stage 3, 4, or 5)? ☐

Atrial fibrillation? ☐

On blood pressure treatment? ☐

Do you have migraines? ☒

Rheumatoid arthritis? ☐

Systemic lupus erythematosus (SLE)? ☐

Severe mental illness? ☐

On atypical antipsychotic medication? ☐

Are you on regular steroid tablets? ☒

A diagnosis of or treatment for erectile dysfunction? ☐

Leave blank if unknown

Total cholesterol: HDL cholesterol ratio:

Systolic blood pressure (mm Hg):

Standard deviation of at least two most recent systolic blood pressure readings (mm Hg):

Body mass index

Height (cm):

Weight (kg):

Calculate risk

Your results

Your risk of having a heart attack or stroke within the next 10 years is:

In other words, in a crowd of 100 people with the same risk factors as you, 8 are likely to have a heart attack or stroke within the next 10 years.

Risk of a heart attack or stroke

Your score has been calculated using estimated data, as some information was left blank.

Your body mass index was calculated as 31.22 kg/m².

CORONARIA CT-NATÍV

Ca-score



- A meszes plakk területének (mm^2) és plakkon belül mért legmagasabb denzitás érték (HU) alapján meghatározott koefficiensnek a szorzata
- Koefficiens 1-4: 1: 130-199 HU
2: 200-299 HU
3: 300-399 HU
4: ≥ 400 HU
- Meszes lézió detekciós küszöbe:

Példa:

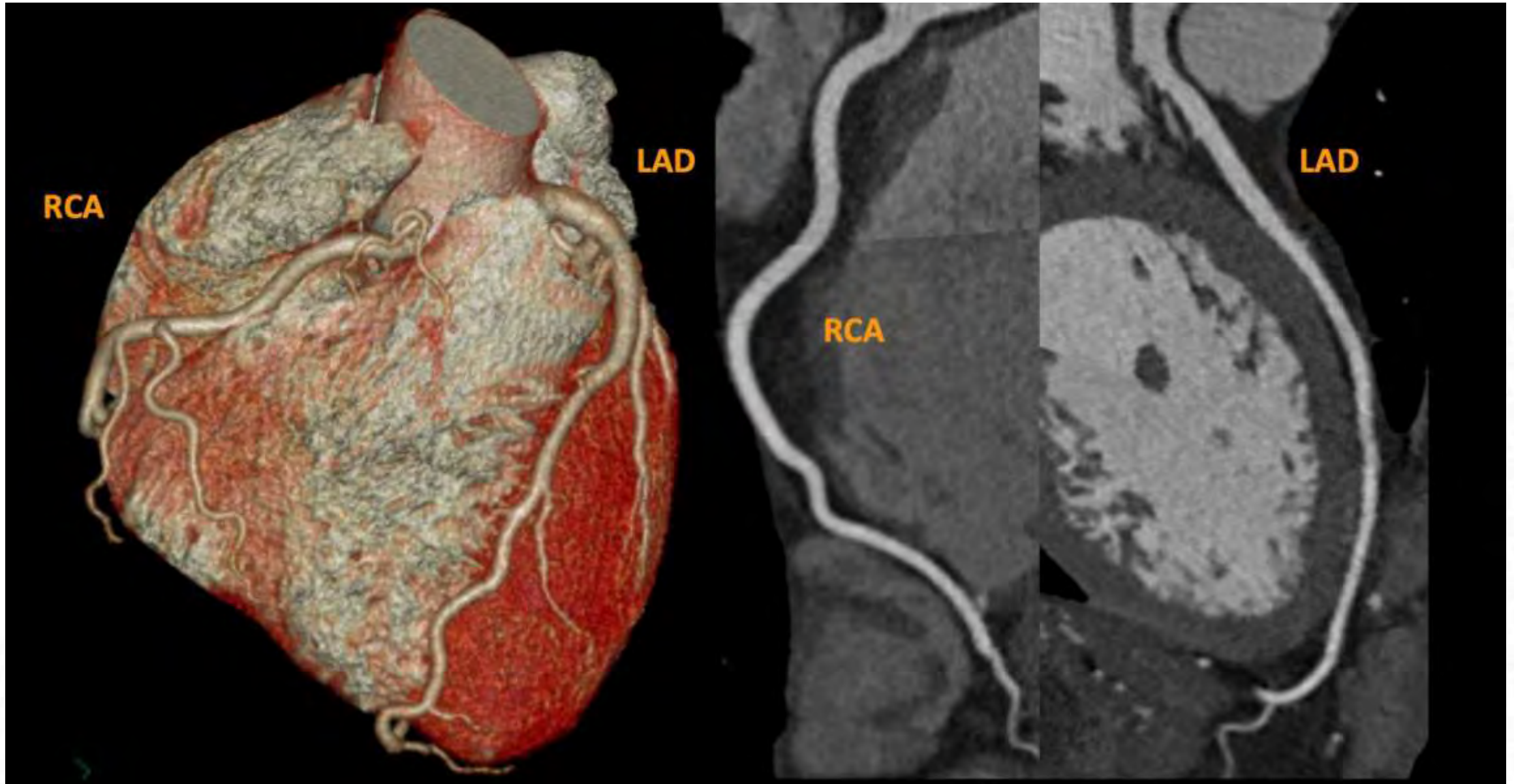
Egy meszes lézió maximális denzitása 400 HU, területe $8 \text{ mm}^2 \rightarrow$ A Ca-score: $4 \times 8 = 32$

SCORE	RIZIKÓ
0	Nincs
0-99	Alacsony
100-399	Közepes
400-999	Magas
≥ 1000	Extrém magas

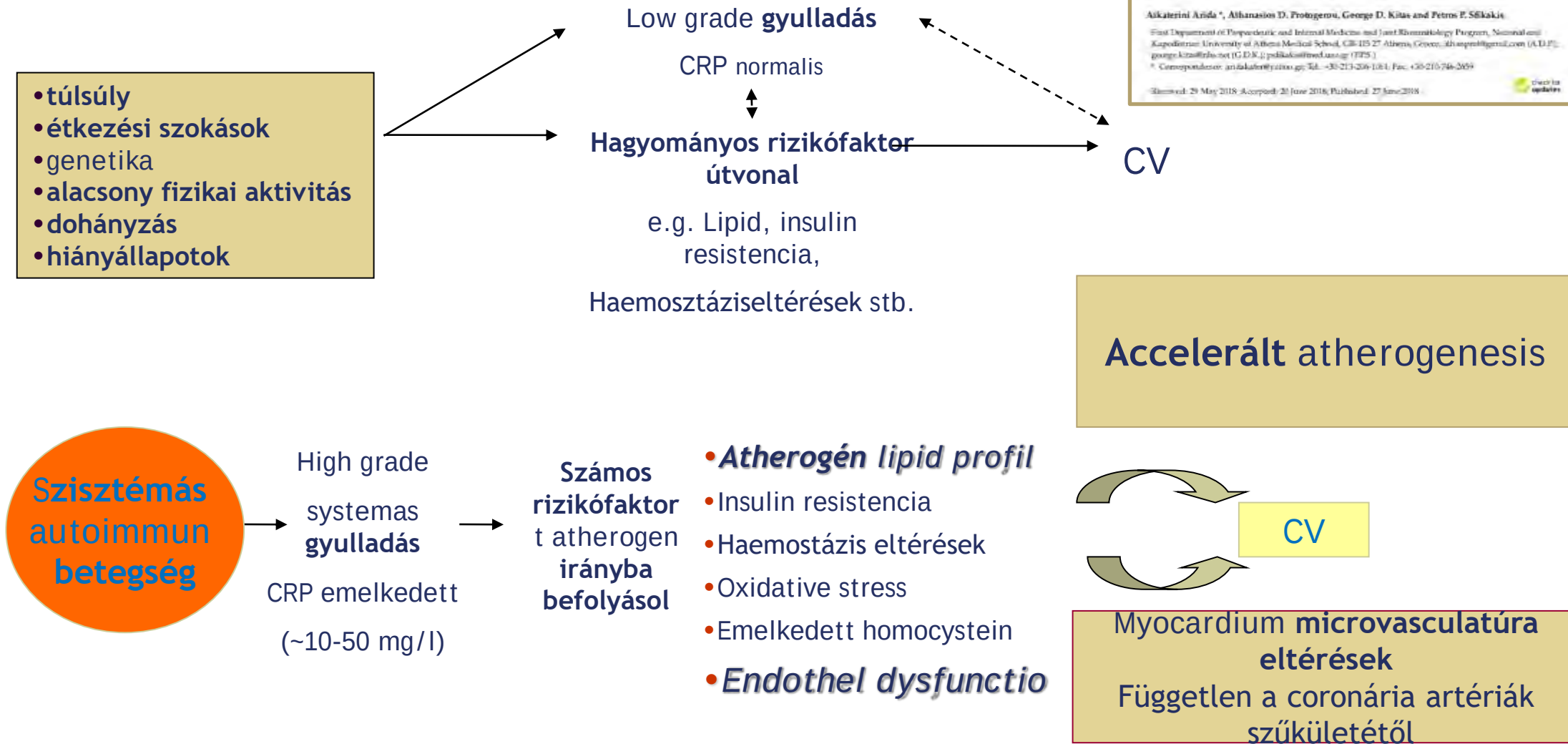
Detrano et al. NEJM 2008;358:1336-45
Budoff et al., J Am Coll Cardiol. 2007

DE !
Nem kalcifikált
plaque-ot
nem mutatja

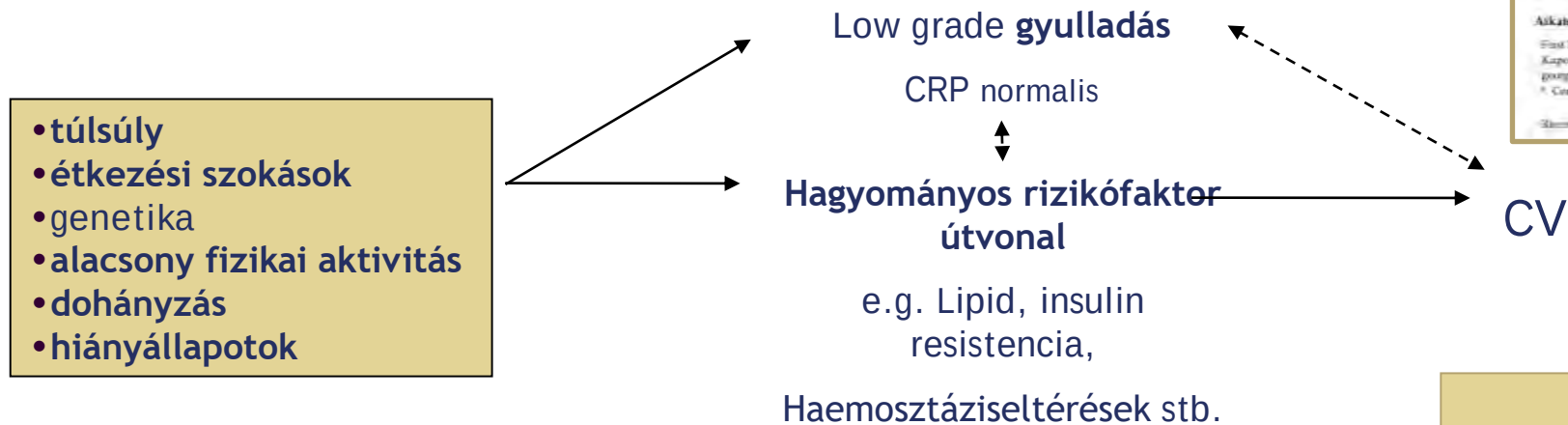
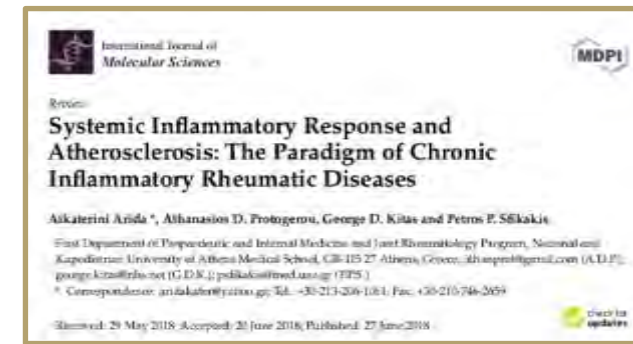
CORONARIA CT-ANGIOGRAPHIA



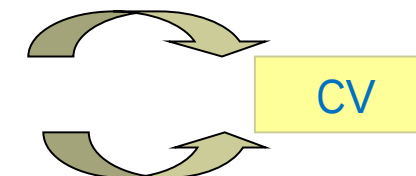
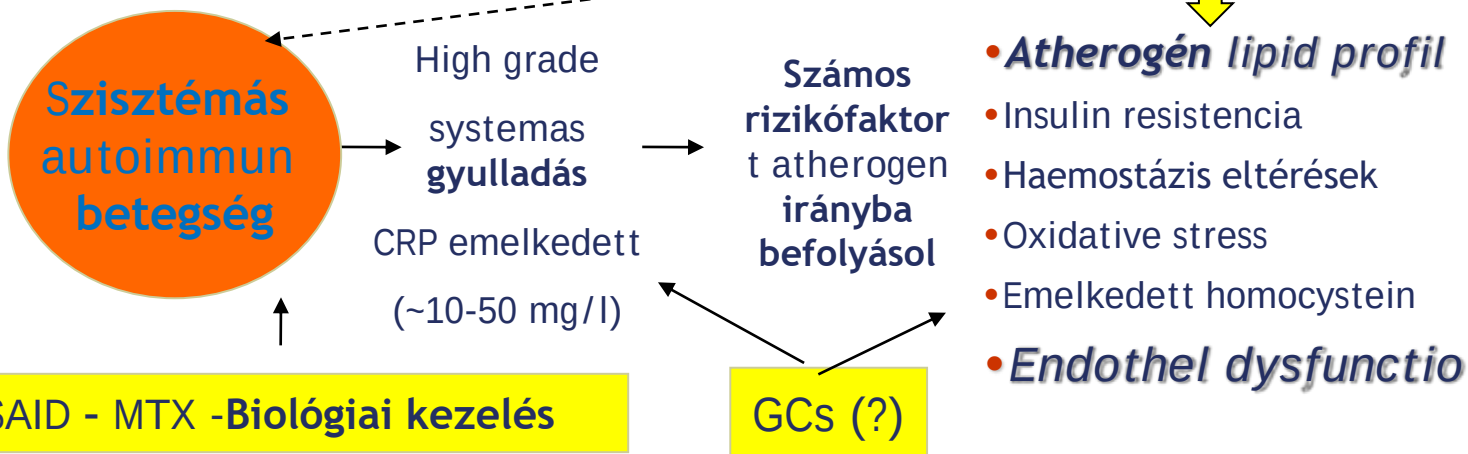
„LOW GRADE” - „HIGH GRADE” GYULLADÁS ÉS AZ ATHEROSCLEROSIS



„LOW GRADE” - „HIGH GRADE” GYULLADÁS ÉS AZ ATHEROSCLEROSIS



Accelerált atherogenesis



**Myocardium microvasculatúra
eltérések**
Független a coronária artériák
szűkületétől

Table 1. Major published clinical trials involving anti-inflammatory agents in atherosclerotic heart disease.

Trial Name	Study Design	Patient Number	Intervention	Primary Outcomes	Results	Benefit Observed
Broad-spectrum anti-inflammatory approach						
Methotrexate						
Cardiovascular Inflammation Reduction Trial (CIRT) [77]	Phase 3 multicentre, randomised, double-blind, placebo-controlled	4786	Oral low methotrexate (target dose of 15–20 mg weekly) vs. placebo	Non-fatal myocardial infarction, non-fatal stroke and cardiovascular death	HR 1.01; 95% CI 0.82–1.25; $p = 0.91$	
Colchicine						
Low-dose colchicine for secondary prevention of cardiovascular disease (LoDoCo) [78]	Phase 3 multicentre, randomised, double-blind, placebo-controlled	532	Colchicine 0.5 mg/day vs. placebo	MI, fatal or non-fatal out-of-hospital cardiac arrest, or non-cardioembolic ischaemic stroke	HR 0.33; 95% CI 0.18–0.59; $p < 0.001$	
Narrow-spectrum anti-inflammatory approach						
IL-1β						
Anti-inflammatory Therapy with Canakinumab for Atherosclerosis (CANTOS) [76]	Phase 3 multicentre, randomised, double-blind, placebo-controlled	10,061	Subcutaneous injection of canakinumab (50 mg, 150 mg or 300 mg) every 3 months vs. placebo	Non-fatal MI, non-fatal stroke and cardiovascular death	HR 0.85; 95% CI 0.74–0.98; $p = 0.021$ in the 150 mg-treated group	
Lipoprotein-associated phospholipase A2 (Lp-PLA₂)						
SOLID-TIMI 52 [79]	Phase 3 multicentre, randomised, double-blind, placebo-controlled	13,026	Daily oral darapladib 160 mg vs. placebo	Coronary heart disease death, non-fatal MI and urgent revascularisation for myocardial ischaemia	HR 1.00; 95% CI 0.91–1.09; $p = 0.93$	
P38 mitogen-activated protein kinase (MAPK)						
LATITUDE-TIMI 60 [80]	Phase 3 multicentre, randomised, double-blind, placebo-controlled	3503	Oral losmapimod 7.5 mg twice daily vs. placebo	Non-fatal MI, severe recurrent ischaemia requiring urgent coronary artery revascularisation and cardiovascular death	HR 1.16; 95% CI 0.91–1.47; $p = 0.24$	

Abbreviations: CI, confidence interval; HR, hazard ratio; IL, interleukin; mg, milligrams; MI, myocardial infarction. The green tick sign represents that the intervention was associated with a significant benefit whereas a red cross represents that there was no significant benefit observed.



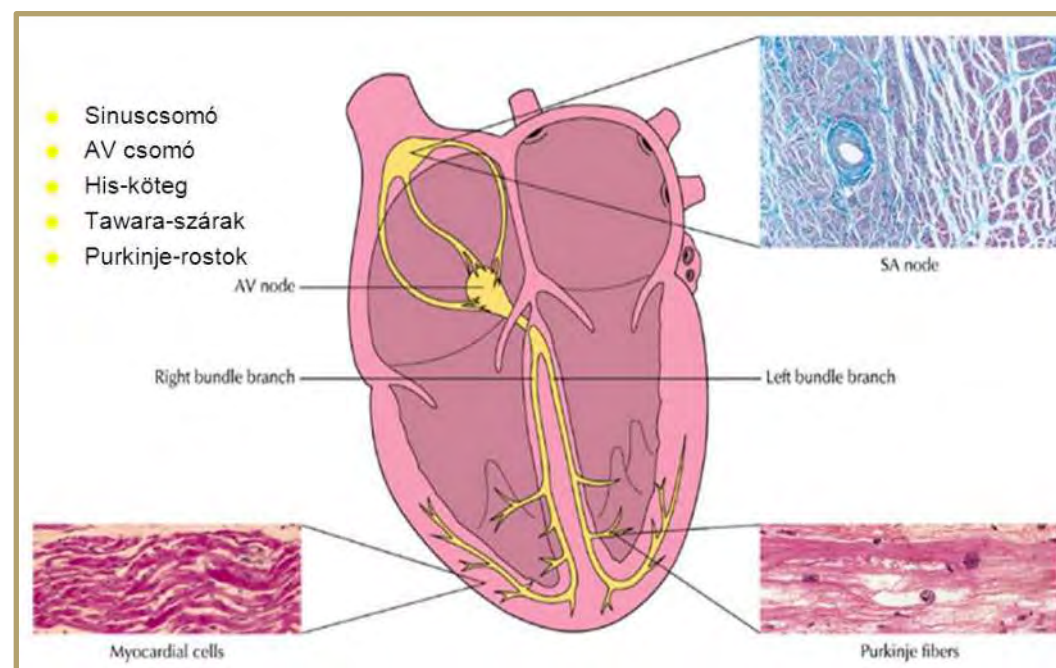
INGERKÉPZÉSI ÉS VEZETÉSI ZAVAROK IMMUNOLÓGIAI VONATKOZÁSAI

AZ INGERKÉPZŐ ÉS VEZETŐ RENDSZER AUTOIMMUN JELENSÉGEI

Sinus csomó ellenes AT jelenléte
AV csomó ellenes AT jelenléte
HIS köteg ellenes AT
Purkinje sejt ellenes AT

10x gyakoribb a SSS
3x gyakoribb az AV block
alacsony mindenkinben

Herz. 2000 May;25(3):181-8.
Cardiac rhythm and conduction disturbances: what is
the role of autoimmune mechanisms?
Ristic AD1, Maisch B.



CONGENITALIS SZÍVBLOCK

aSSA, aSSB anyák magzataiban alakulhat ki

IgG AB + FcRn + alb

Transplacentaris transzport : 16-22 hét - 10% IgG

SZŰRÉS - MAGZATI ECHOCARDIOGRAPHIA !!

Prevencao :

400 mg/nap hydroxichloroquin

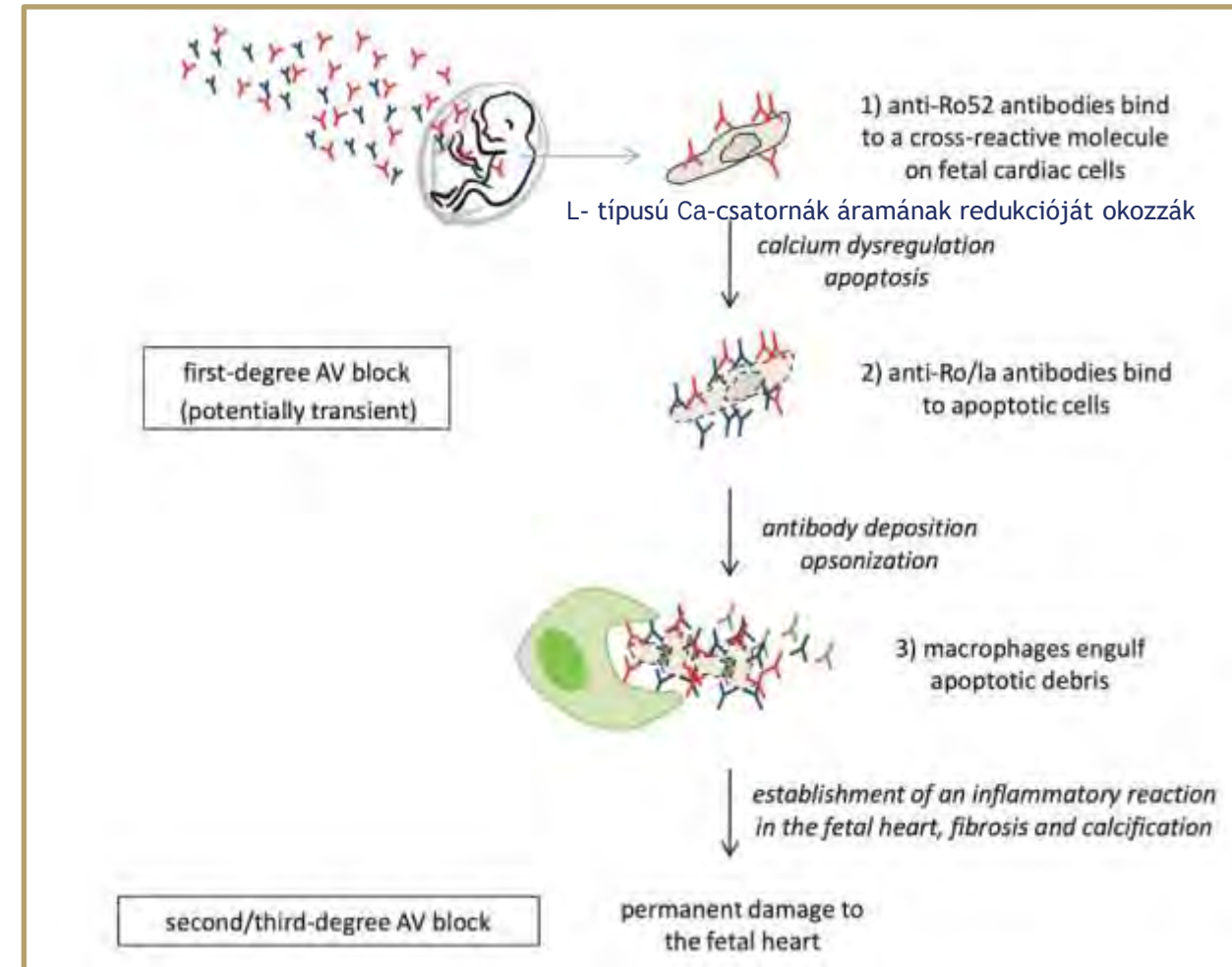
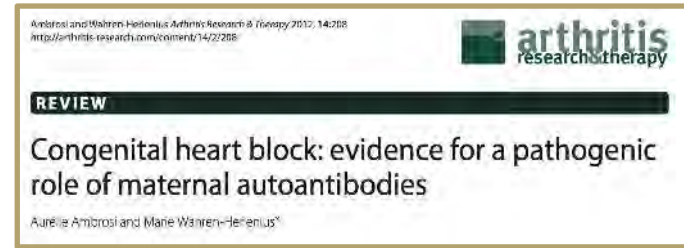
Szeroid-azatioprin- IVIg- autoimmun betegek

Kezelés :

400 mg/nap hydroxichloroquin

Steroid-azathioprin- IVIg (1 gr / ttkg / 2 hét)

PEX (2-3000 ml / alkalom)



AUTOANTITESTEK A RITMUSZAVAROK HÁTTÉRÉBEN

Sinus bradycardia- AV block :
aSSA -Ca csatorna,
NA csatorna ell at - Na csatorna

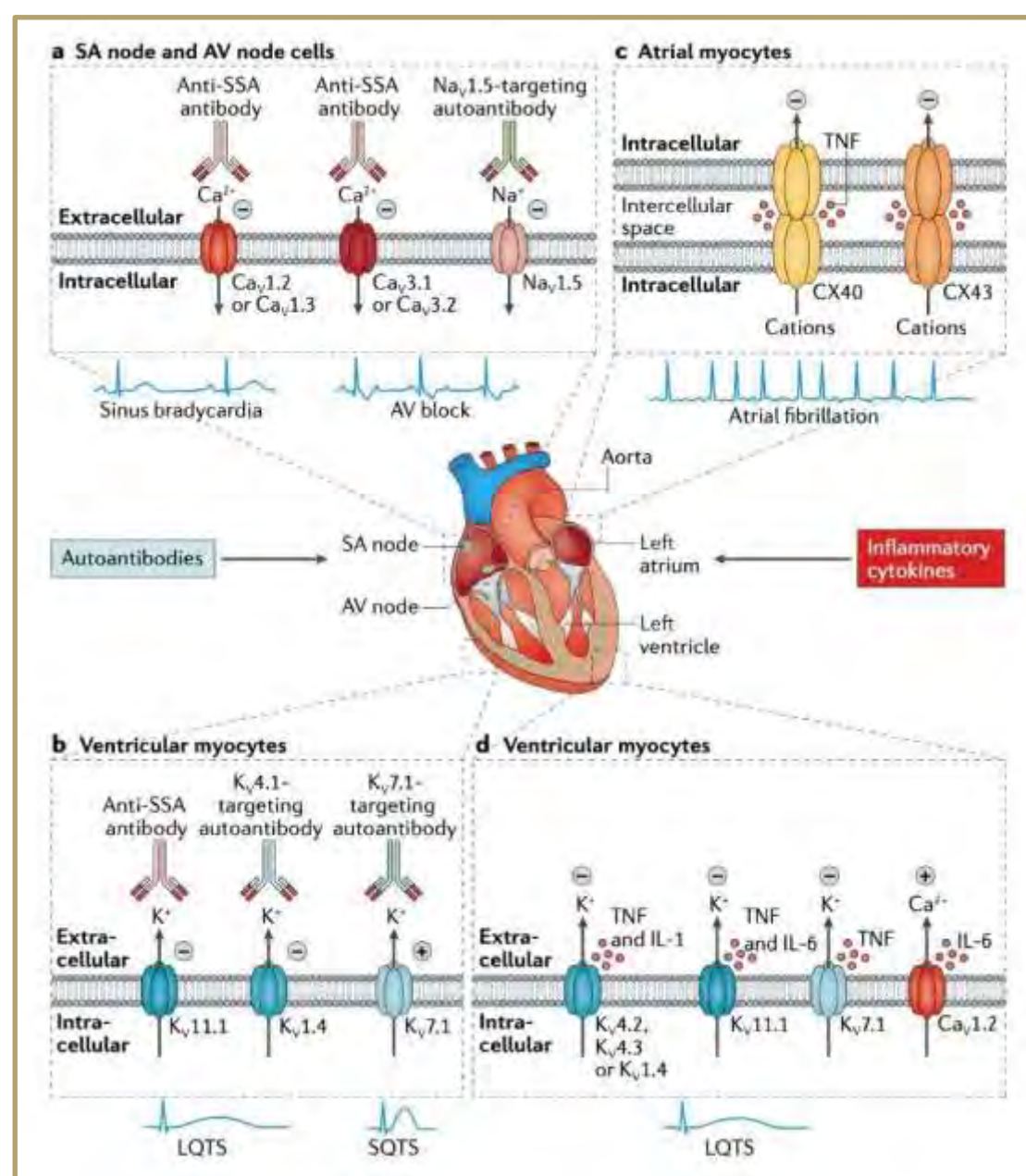
Pitvarfibrillatio :
TNF alfa - CX40, CX43-en keresztül a gap junction funkciót gátolja

longQT
aSSA - K csatorna
TNF alfa - K csatorna
IL-6- cs csatorna gátlás

Cardioimmunology of arrhythmias: the role of autoimmune and inflammatory cardiac channelopathies

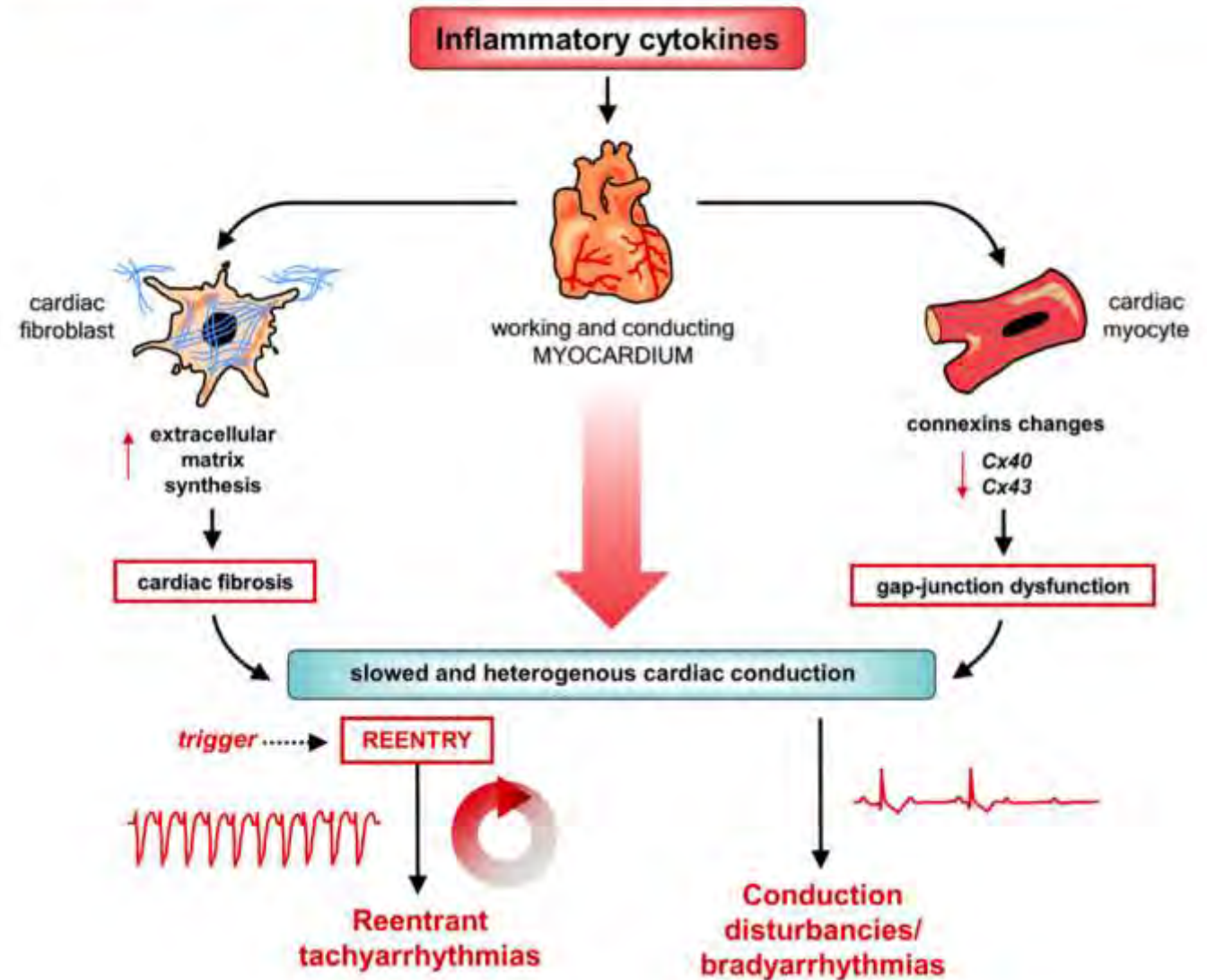
Pietro Enea Lazzerini , Franco Laghi-Pasini, Mohamed Boutjdir & Pier Leopoldo Capecchi

Nature Reviews Immunology 19, 63–64 (2019) | [Cite this article](#)



INFLAMMATORIKUS CYTOKINEK ARRYTHMOGEN HATÁSA

FIGURE 2 Arrhythmogenic Effects of Inflammatory Cytokines: Re-entrant Tachyarrhythmias and Conduction Disturbances/Bradyarrhythmias



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STATE-OF-THE-ART REVIEW

Fir(e)ing the Rhythm

Inflammatory Cytokines and Cardiac Arrhythmias

Pietro Enea Lazzarini, MD,^a Antonio Abbate, MD, PhD,^b Mohamed Boutjdir, PhD,^{c,d,*} Pier Leopoldo Capecchi, MD^{a,*}

INFLAMMATORIKUS CYTOKINEK HATÁSMECHANIZMUSA

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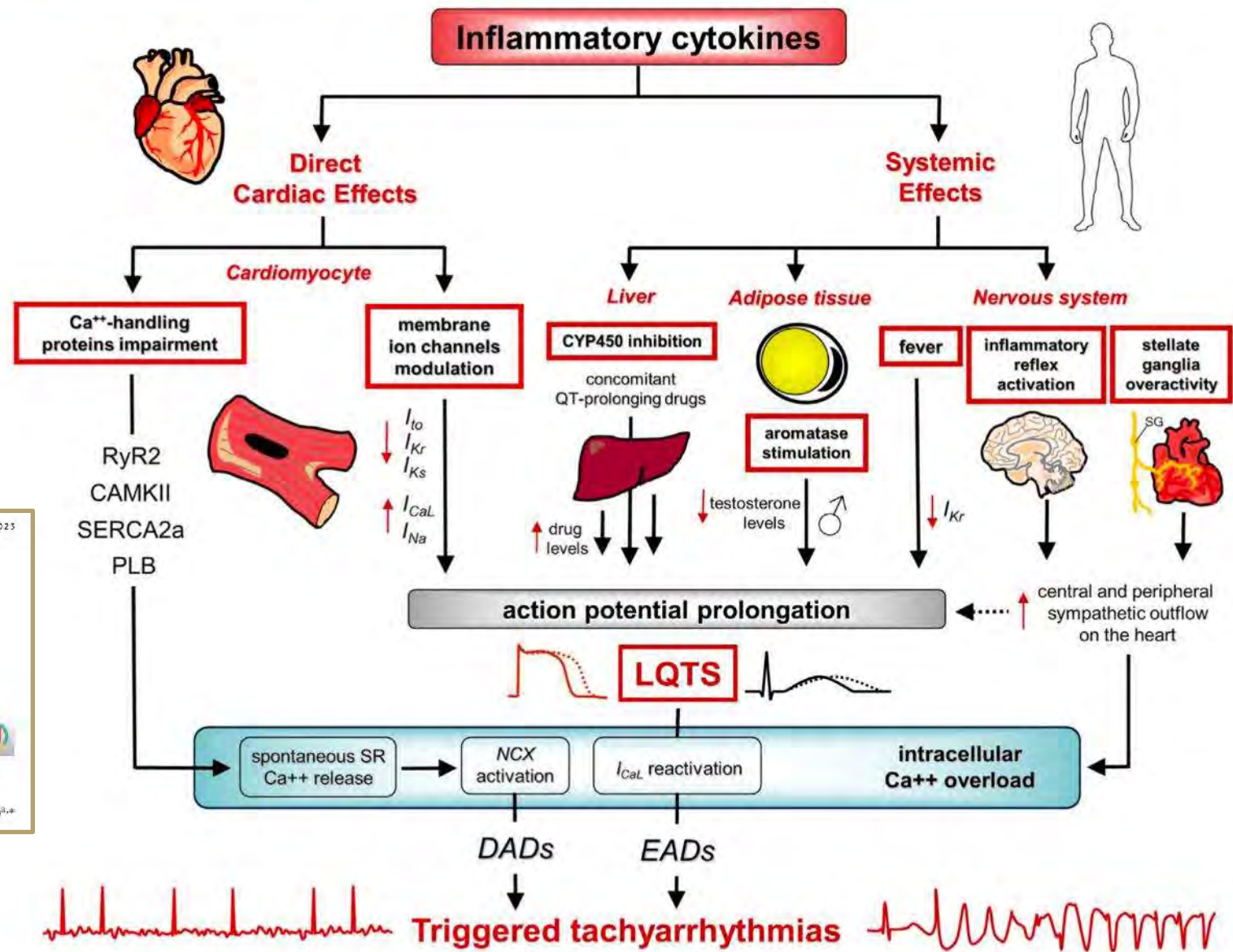
STATE-OF-THE-ART REVIEW

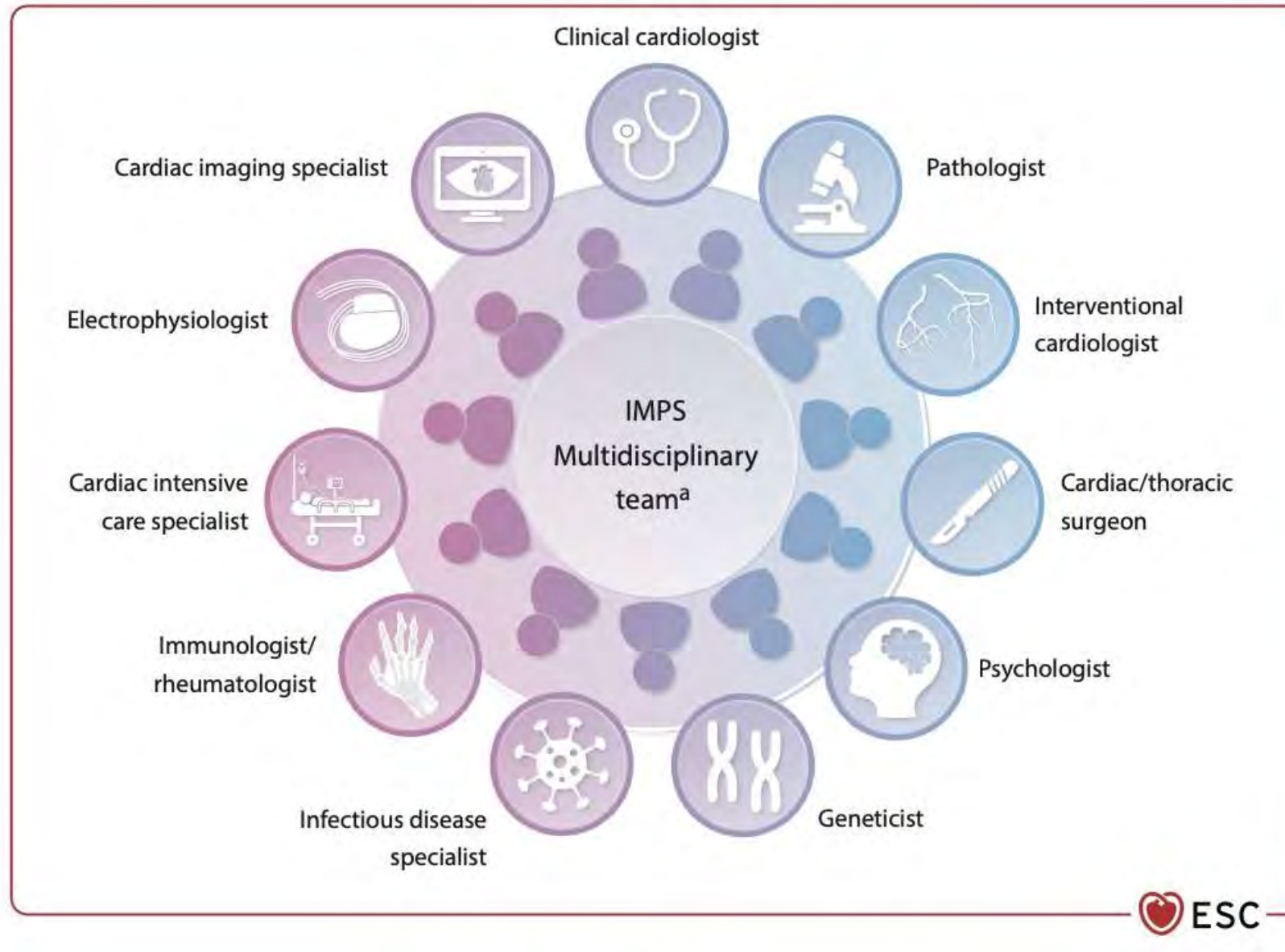
Fir(e)ing the Rhythm

Inflammatory Cytokines and Cardiac Arrhythmias

Pietro Enea Lazzerini, MD,^a Antonio Abbate, MD, PhD,^b Mohamed Boutjdir, PhD,^{c,d,*} Pier Leopoldo Capecchi, MD^{a,*}

FIGURE 1 Arrhythmogenic Effects of Inflammatory Cytokines: Triggered Tachyarrhythmias







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