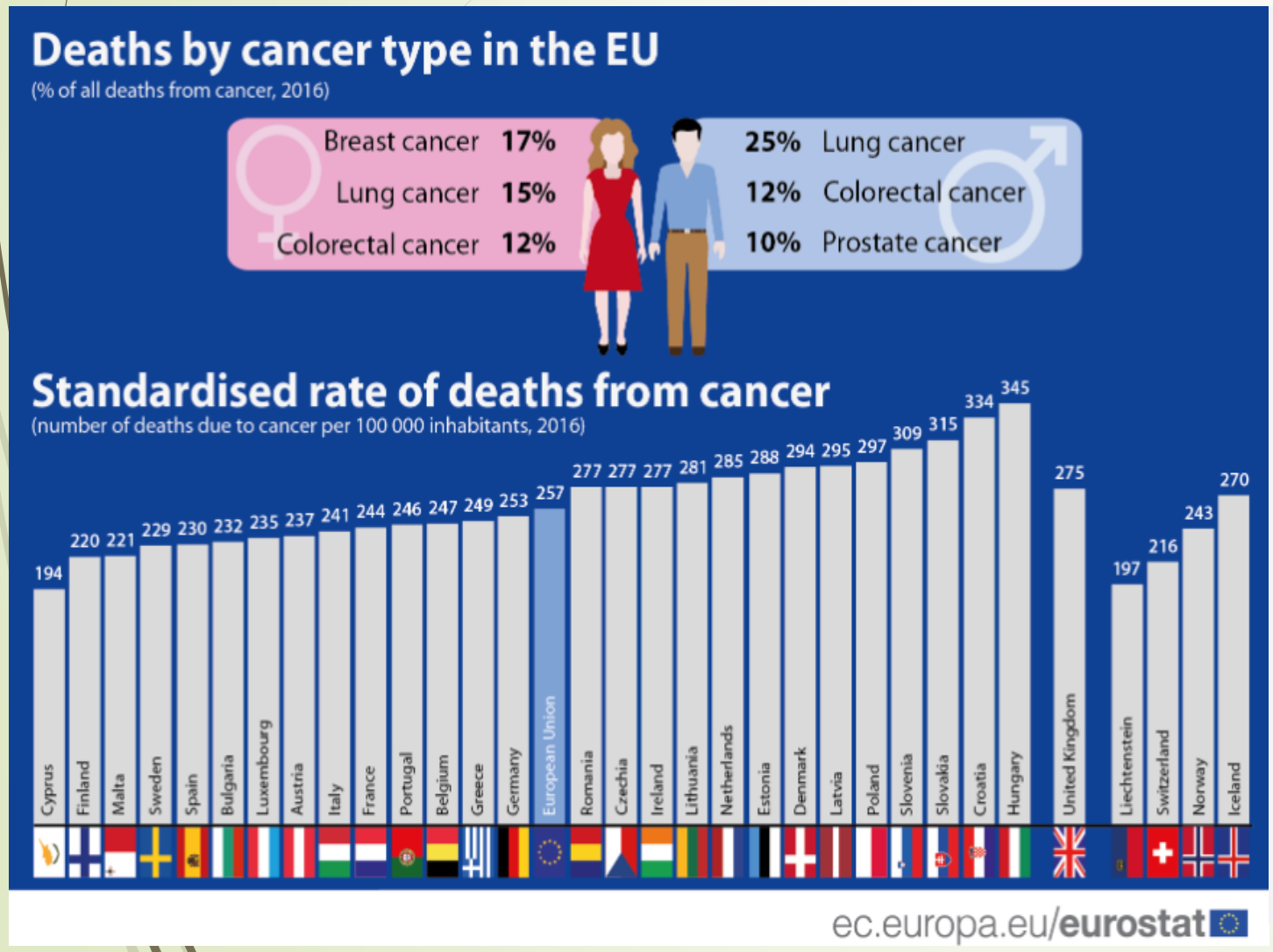
Onko-kardiológia

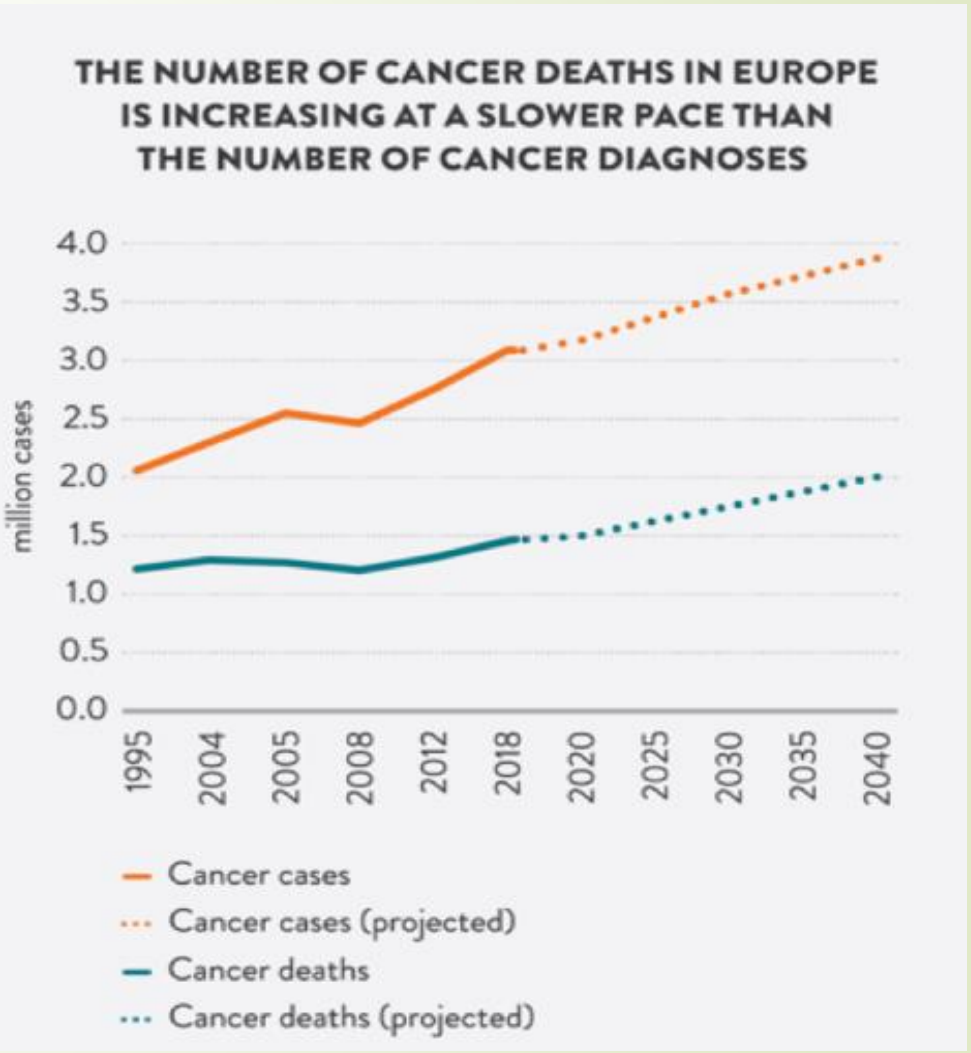
Dr. Gál Roland PhD
egyetemi adjunktus

PTE KK I. sz. Belgyógyászati Klinika, Kardiológiai Tanszék

Pécs, 2026.05.21.

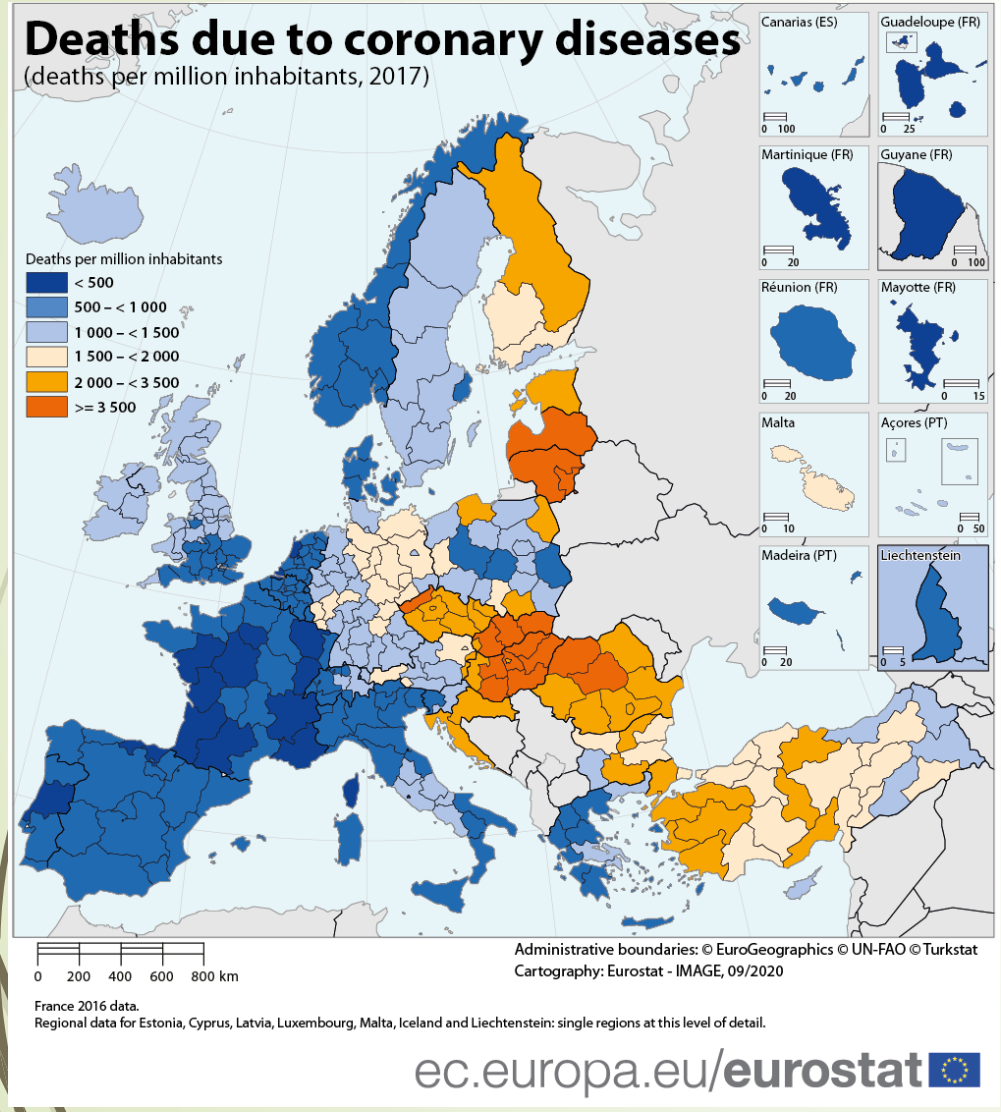
A daganatos betegségek előfordulása Európában

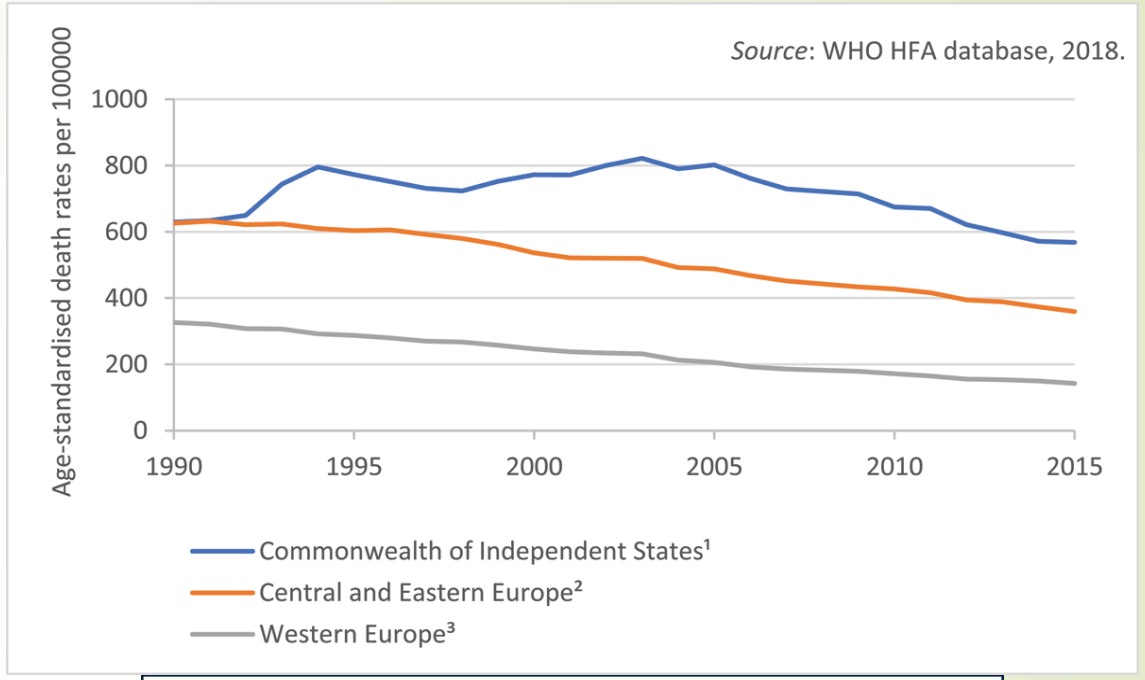




www.efpia.eu/news-events/the-efpia-view/blog-articles/cancer-care-in-europe-where-are-we-in-2020/

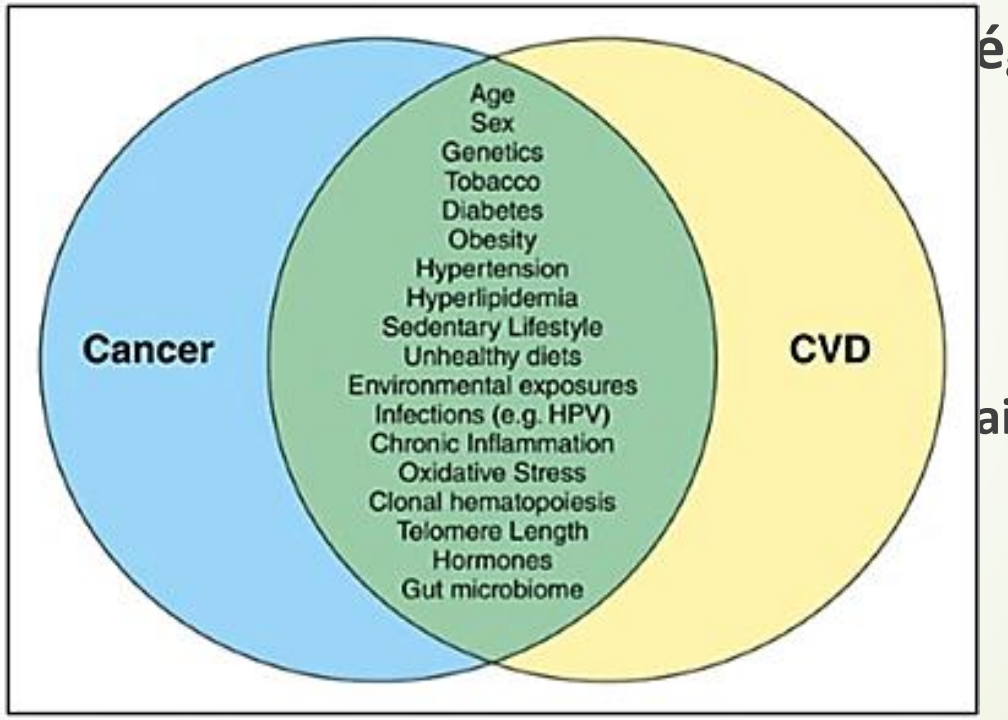
A kardiovaszkuláris betegségek halálozása Európában





Bevezetés

- Mind a daganatos, mind a kardiovaszkuláris (CV) betegségek előfordulása növekvő tendenciát mutat
- Évről-évre több[illegible] [illegible]égben is egyaránt
  - emelkedik a l[illegible]
  - javul a dagan[illegible]
  - genetikai prec[illegible]
  - környezeti str[illegible] [illegible]ai)



Forrás: https://www.summahealth.org/medicalservices/heart/conditions-and-services/heart-failure/cardio-oncology-program

Bevezetés

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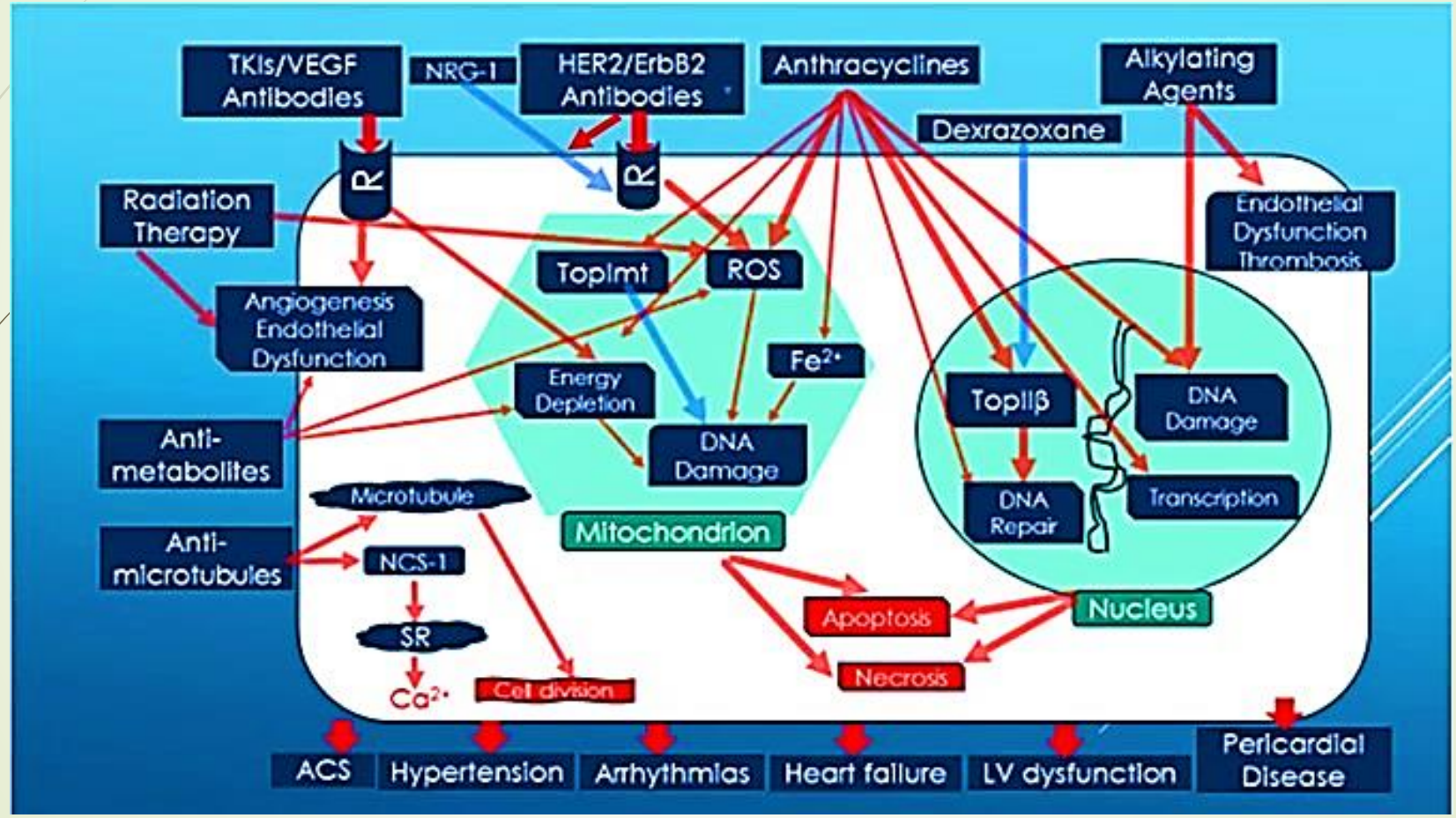
  - emelkedik a lakosság átlagéletkora
  - javul a daganatos betegek túlélése
  - genetikai prediszpozíció
  - környezeti stresszhatások fokozódása (emocionális és fizikai)
  - daganatellenes kezelések kardiotoxicitása

A daganatellenes kezelés fő kardiális hatásai



Han X. et. Al. NPJ Precis Oncol.2017;1(1):31.

Bevezetés

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- Évről-évre több daganatos beteg szenved CV betegségben is egyaránt
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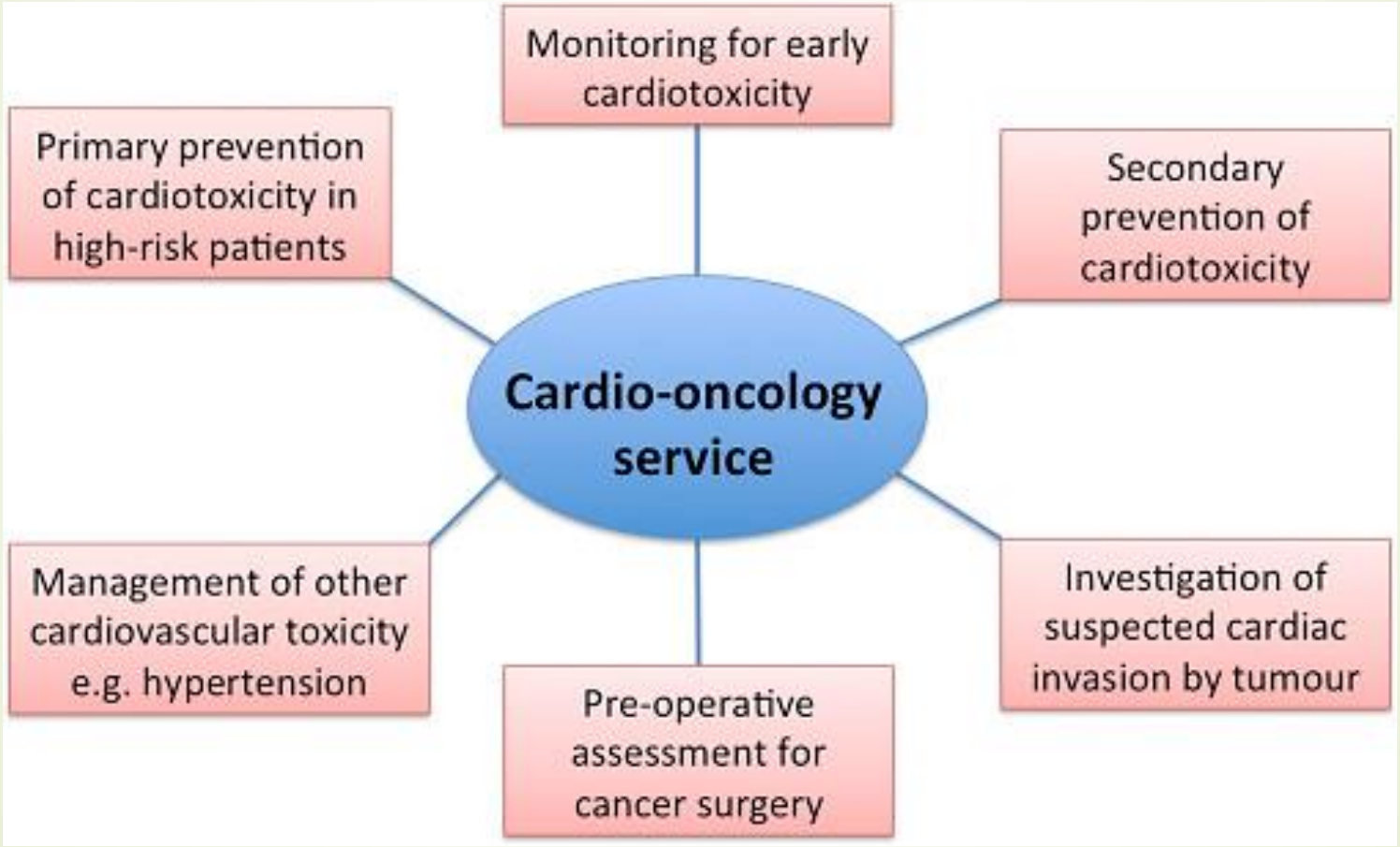
  - daganatellenes kezelések kardiotoxicitása
- A kardio-onkológia fő feladata a betegek multidiszciplináris megközelítése

Az onko-kardiológia fő feladatkörei



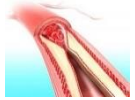
Cubon R.M. et al. Indian Heart J. 2016;68:S77-85.

Az onkológiai kezelések kardiovaszkuláris szövődményei

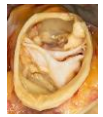
- ESC → Kardiotoxicitás = CTR-CVT (daganatterápia asszociált CV toxicitás)
- Mely kardiális szövődmények tartoznak ebbe a körbe?

1. Balkamra diszfunkció és szívelégtelenség

2. Koronáriabetegség



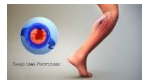
3. Billentyűbetegség



4. Aritmiák

5. Perifériás érbetegség és stroke

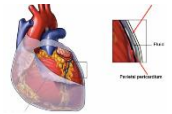
6. VTE



7. Szisztémás hipertónia

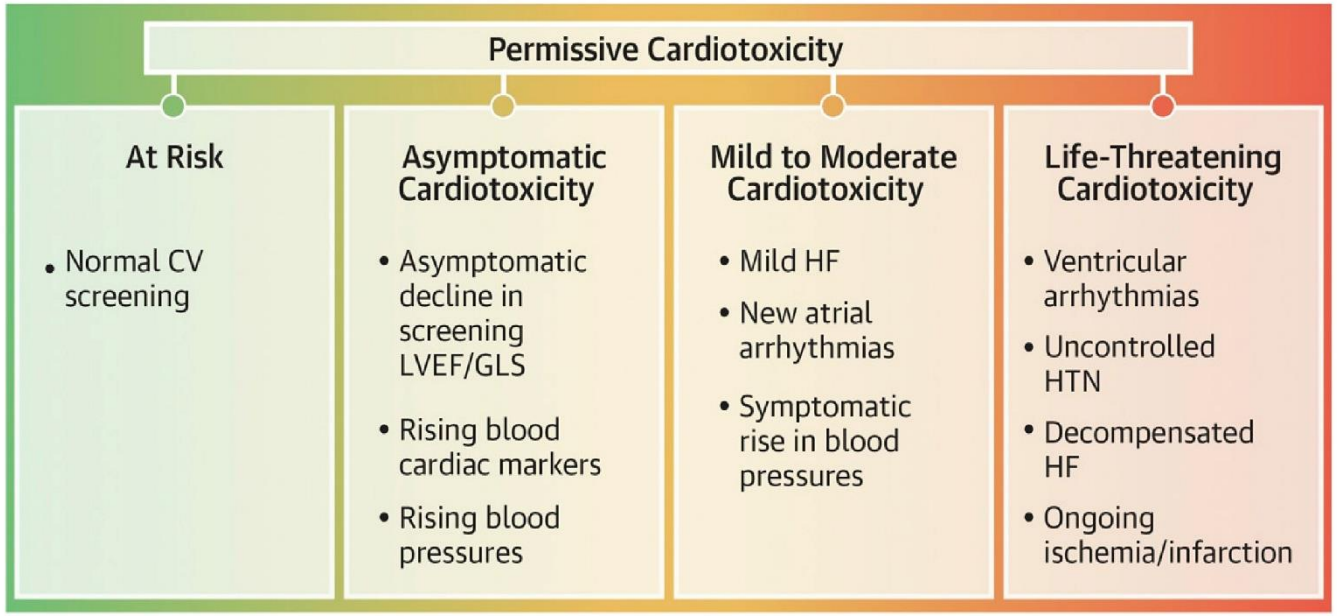
8. Pulmonális hipertónia

9. egyéb kardiovaszkuláris szövődmények

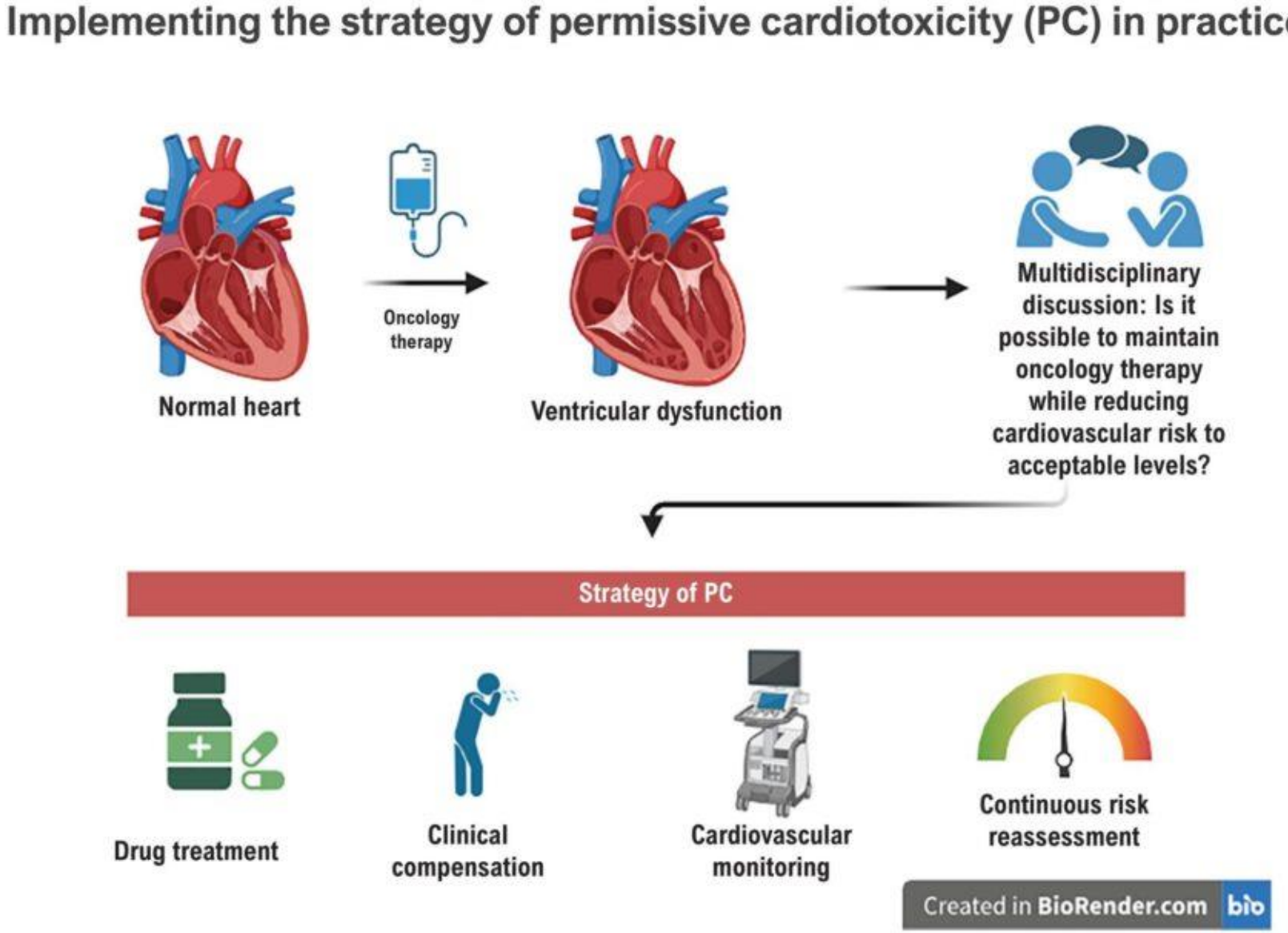


„Kardiotoxicitás”

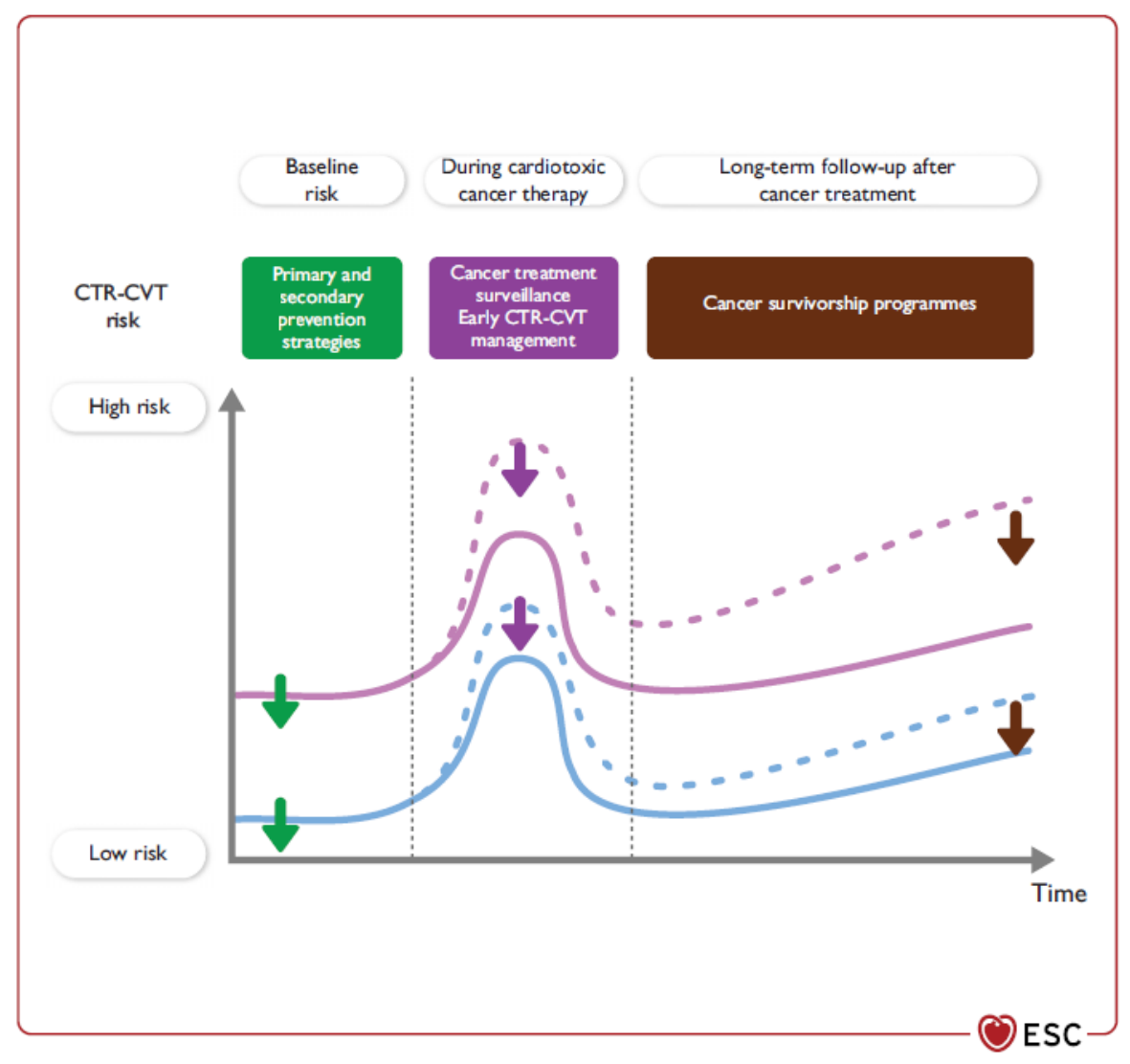
CENTRAL ILLUSTRATION: The Spectrum of Permissive Cardiotoxicity: From “At Risk” Through Life-Threatening Cardiotoxicity



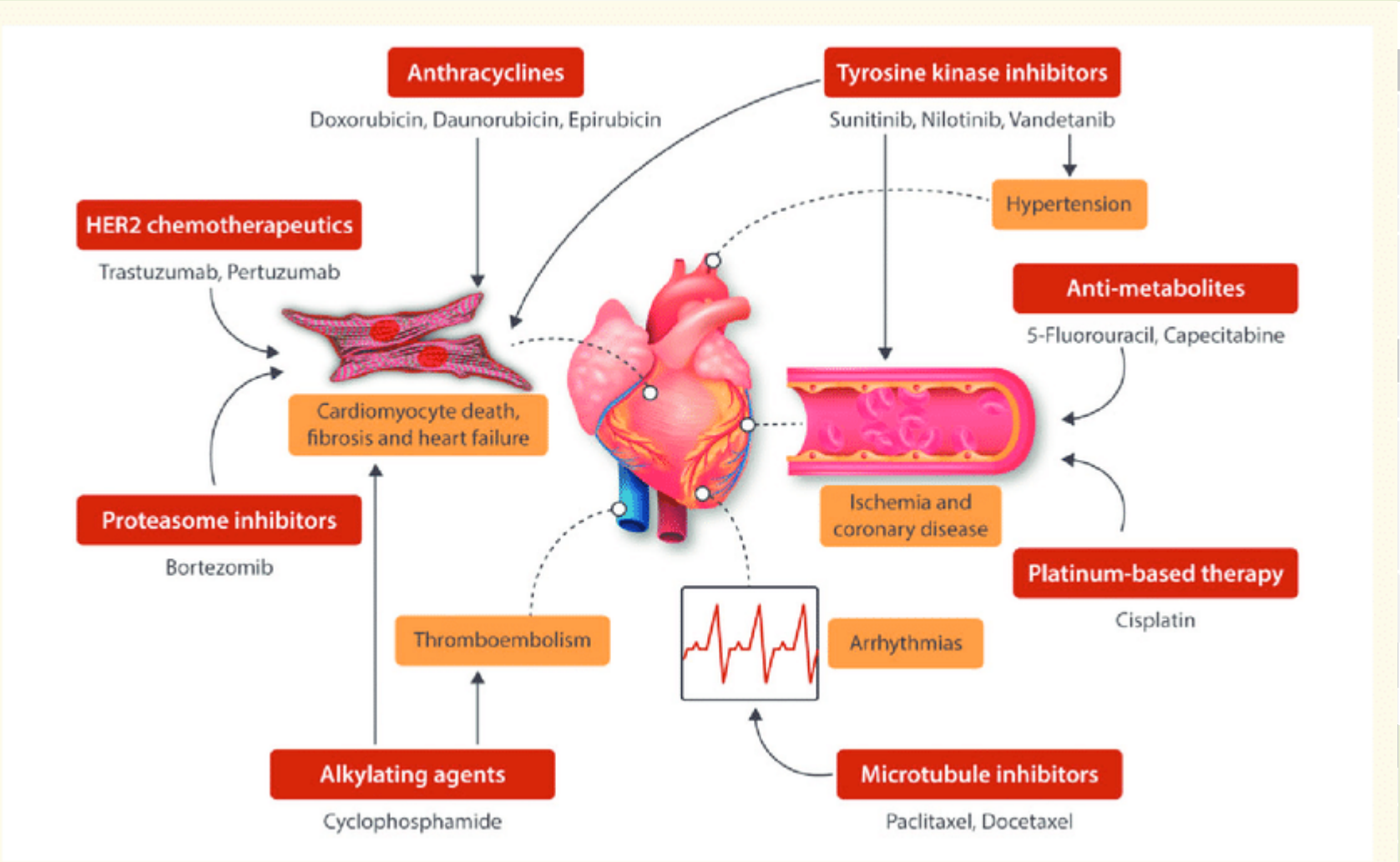
Porter C, et al. J Am Coll Cardiol CardioOnc. 2022;4(3):302-312.


Implementing the strategy of permissive cardiotoxicity (PC) in practice
Normal heart
Oncology therapy
Ventricular dysfunction
Multidisciplinary discussion: Is it possible to maintain oncology therapy while reducing cardiovascular risk to acceptable levels?
Strategy of PC
Drug treatment
Clinical compensation
Cardiovascular monitoring
Continuous risk reassessment
Created in BioRender.com

A kardiotoxicitás előfordulása a daganatellenes kezelés során



Kardiotoxicitás



	vasoreactivity		
	Coronary microvascular vasoreactivity		Microvascular angina Raynaud's phenomenon

Sayed N. et al. Cardiovasc Res. 2019 Apr 15; 115(5): 949–959.
Lyon AR et. Eur Heart J Cardiovasc Imaging. 2022 Sep 10;23(10):e333-e465.

ESC
European Society of Cardiology
European Heart Journal (2022) **00**, 1–133
https://doi.org/10.1093/eurheartj/ehac244

ESC GUIDELINES

2022 ESC Guidelines on cardio-oncology developed in collaboration with the European Hematology Association (EHA), the European Society for Therapeutic Radiology and Oncology (ESTRO) and the International Cardio-Oncology Society (IC-OS)

Developed by the task force on cardio-oncology of the European Society of Cardiology (ESC)

Az onko-kardiológia diagnosztikai lehetőségei- képalkotók

Right ventricle-right atrium[a]

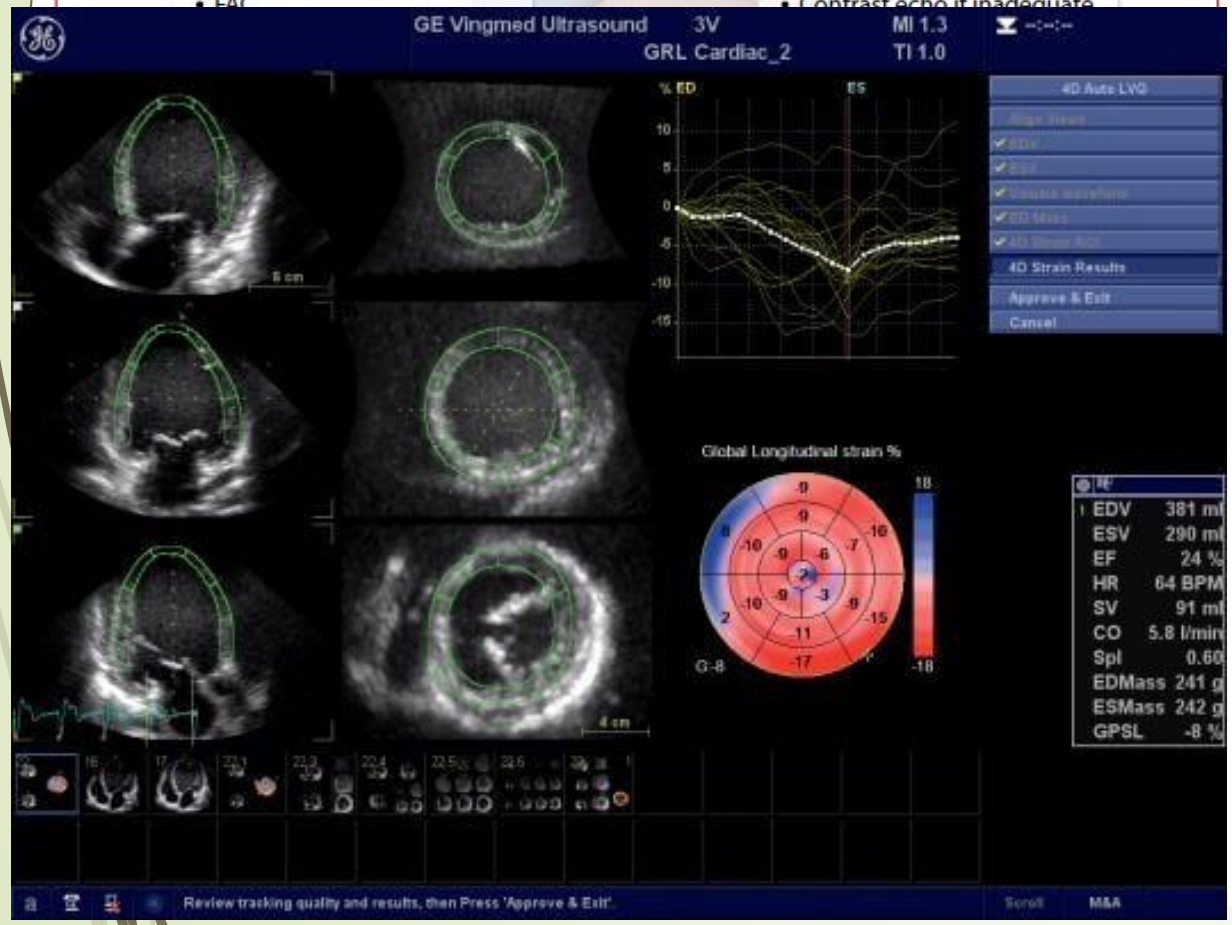
TTE

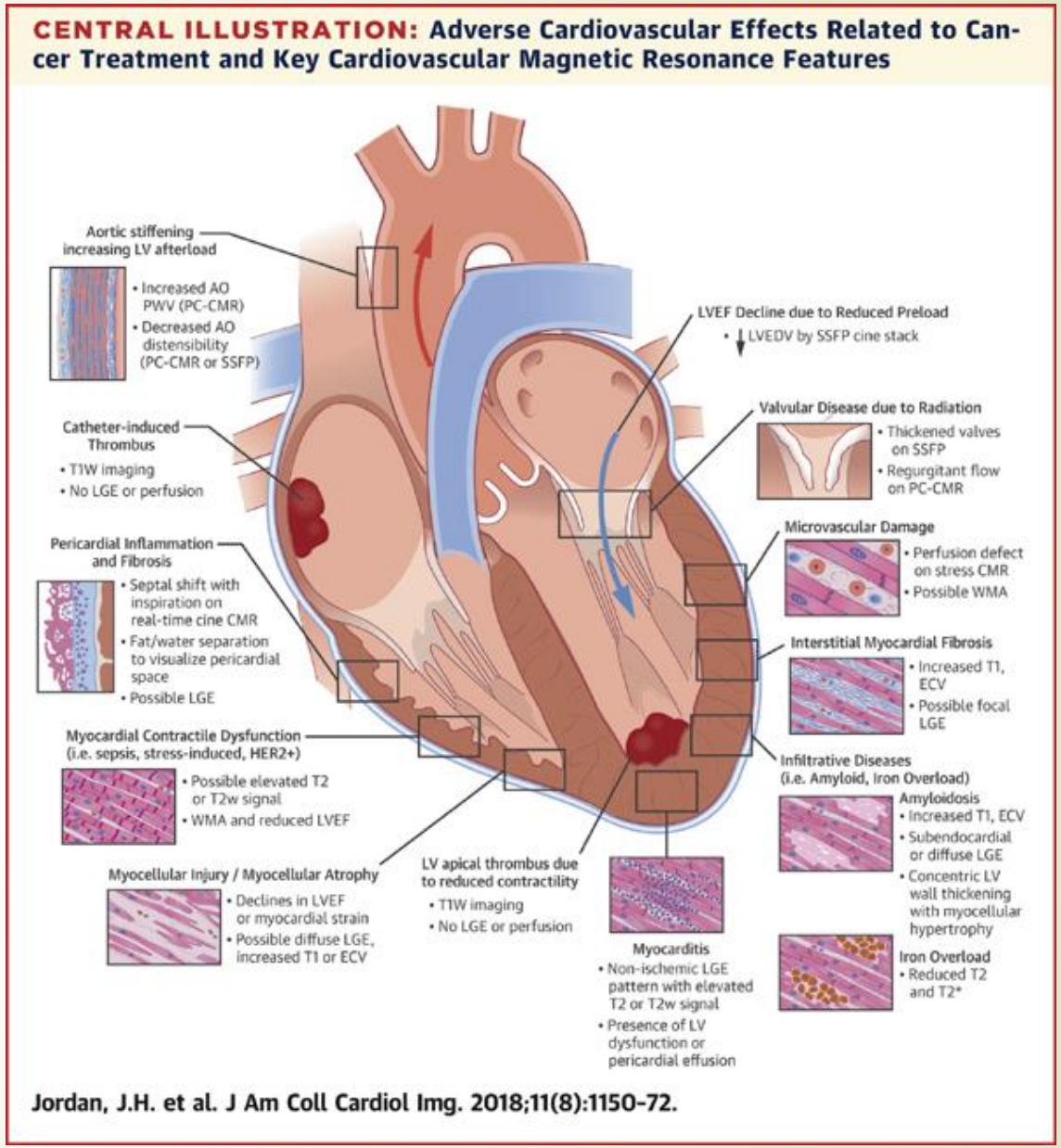
- RV dimensions
- S´
- TAPSE
- FAC

Left ventricle-left atrium[a]

TTE

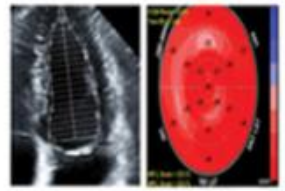
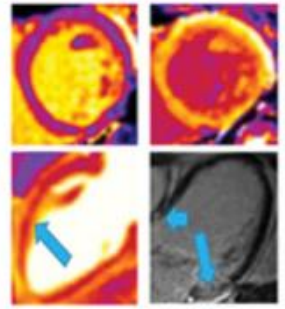
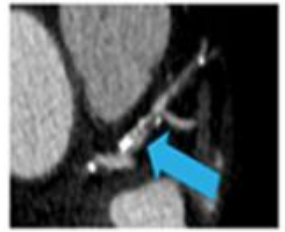
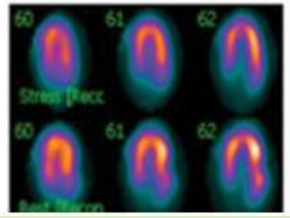
- LVV and LV mass
- 3D-LVEF (2D-LVEF if 3D not available)
- Contrast echo if inadequate





Jordan, J.H. et al. J Am Coll Cardiol Img. 2018;11(8):1150-72.

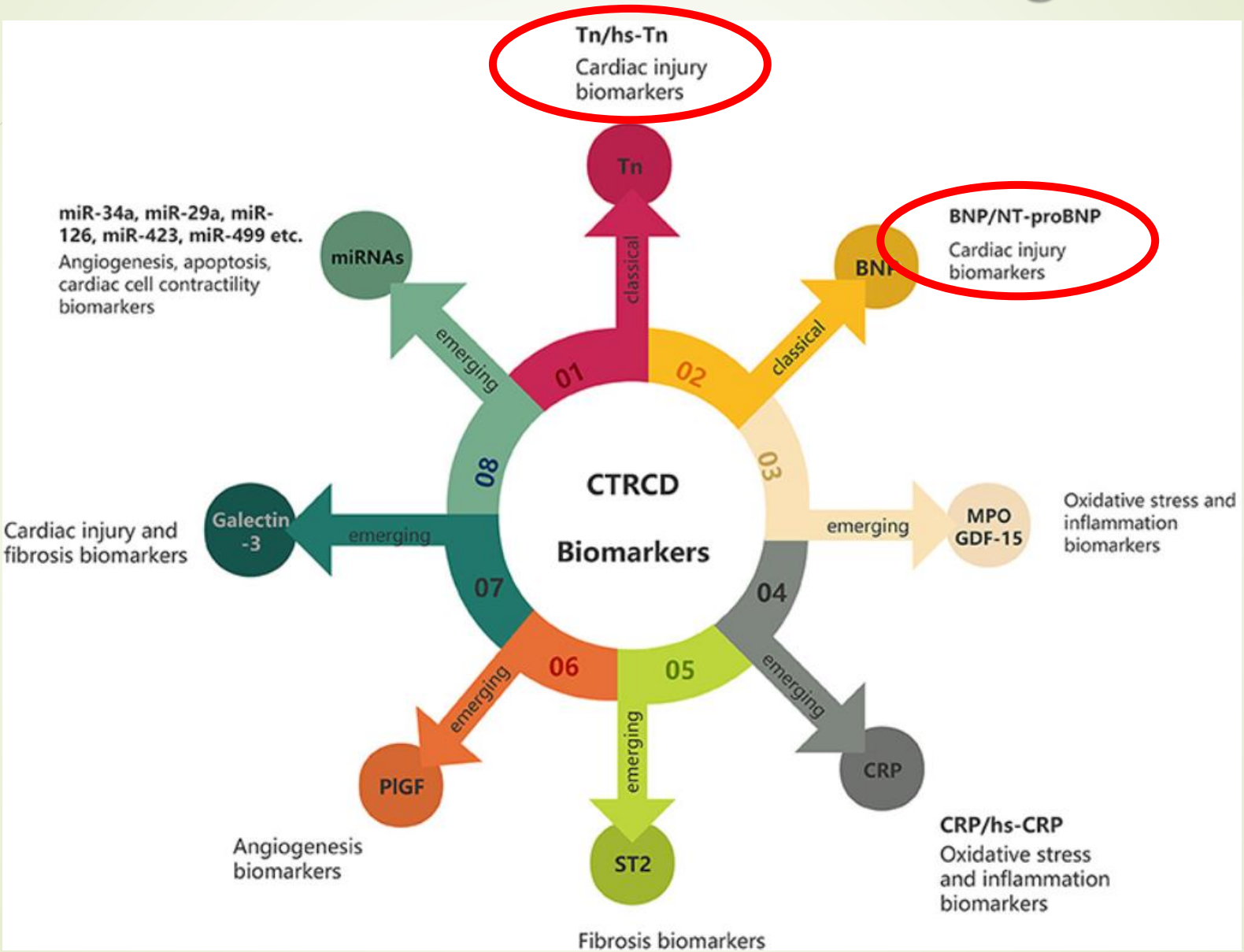
Lyon AR et. Eur Heart J Cardiovasc Imaging. 2022 Sep 10;23(10):e333-e465.

Before Treatment	During Treatment	After Treatment	Modality
Echocardiography			
LV Function Assessment Cardiac mass Valvular disease	LV Function Assessment Consider as needed for cardiac mass, or Other significant cardiovascular states (eg, valve disease)	LV Function Assessment Consider as needed for cardiac mass, or Other significant cardiovascular states (eg, valve disease)	
Cardiac Magnetic Resonance (CMR)			
Unexplained heart failure Recent myopericarditis Accurate LVEF or valve assessment before cancer treatment Cardiac mass assessment Cardiac amyloid	Unexplained heart failure Recent myopericarditis Accurate LVEF or valve assessment before cancer treatment Cardiac mass assessment Consider as needed other cardiovascular states (eg, amyloid)	Unexplained heart failure Recent myopericarditis Accurate LVEF or valve assessment before cancer treatment Cardiac mass assessment Consider as needed other cardiovascular states (eg, amyloid)	
Cardiac Computed Tomography (CCT)			
Symptomatic CAD evaluation prior to pro-ischemic or prothrombotic treatment Guidance of primary prevention therapy with intermediate ASCVD risk Structural planning before TAVR or TMVR	Use as needed for general symptoms (eg, suspected CAD)	Use as needed for general symptoms (eg, suspected CAD)	
Nuclear or Positron Emission Tomography (PET)			
Symptomatic CAD evaluation prior to pro-ischemic or pro-thrombotic treatment Cardiac Amyloid Metabolic activity of cardiac mass	Use as needed for general symptoms (eg, suspected CAD) Consider as needed for cardiac mass, or other cardiovascular states (eg, amyloid)	Use as needed for general symptoms (eg, suspected CAD) Consider as needed for cardiac mass, or other cardiovascular states (eg, amyloid)	

Addison D et al. Circulation. 2023 Oct 17;148(16):1271-1286.

A biomarkerek helye a onko-kardiológiában

A biomarkerek az onko-kardiológiában

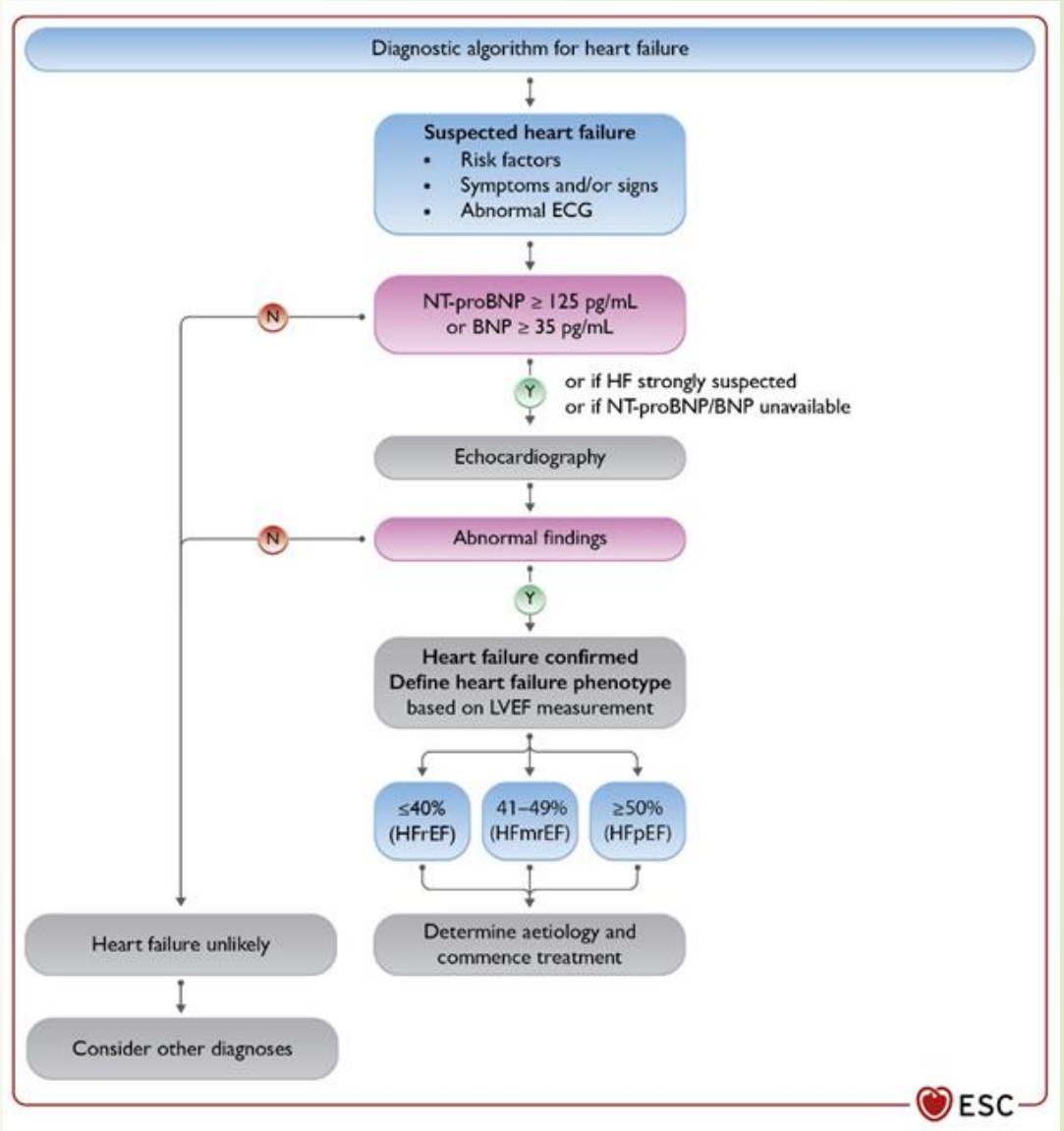


CTRCD: kemoth. asszociált kardiális diszfunkció

Xiao H. et al. Front Cardiovasc Med. 2021 Nov 10;8:753313.

Nátriuretikus peptidek

- B-típusú NP (BNP) és N-terminális proBNP (NT-proBNP)használatos
- magas szenzitivitása és specificitása erősíti a szívelégtelenség diagnózis pontosságát (diff. dg., rule-out)
  - CAVE: obezitás (50% alacsonyabb cut-off), életkor
- Prognosztikus értéke van a szívelégtelenségben (mortalitás és HHF), emellett szívinfarktus, szívbillentyű-betegség, pitvarfibrilláció és tüdőembólia esetén



McDonagh TA et al. Eur Heart J. 2021;42(36):3599-726

Kardiális Troponin (cTnT/I)

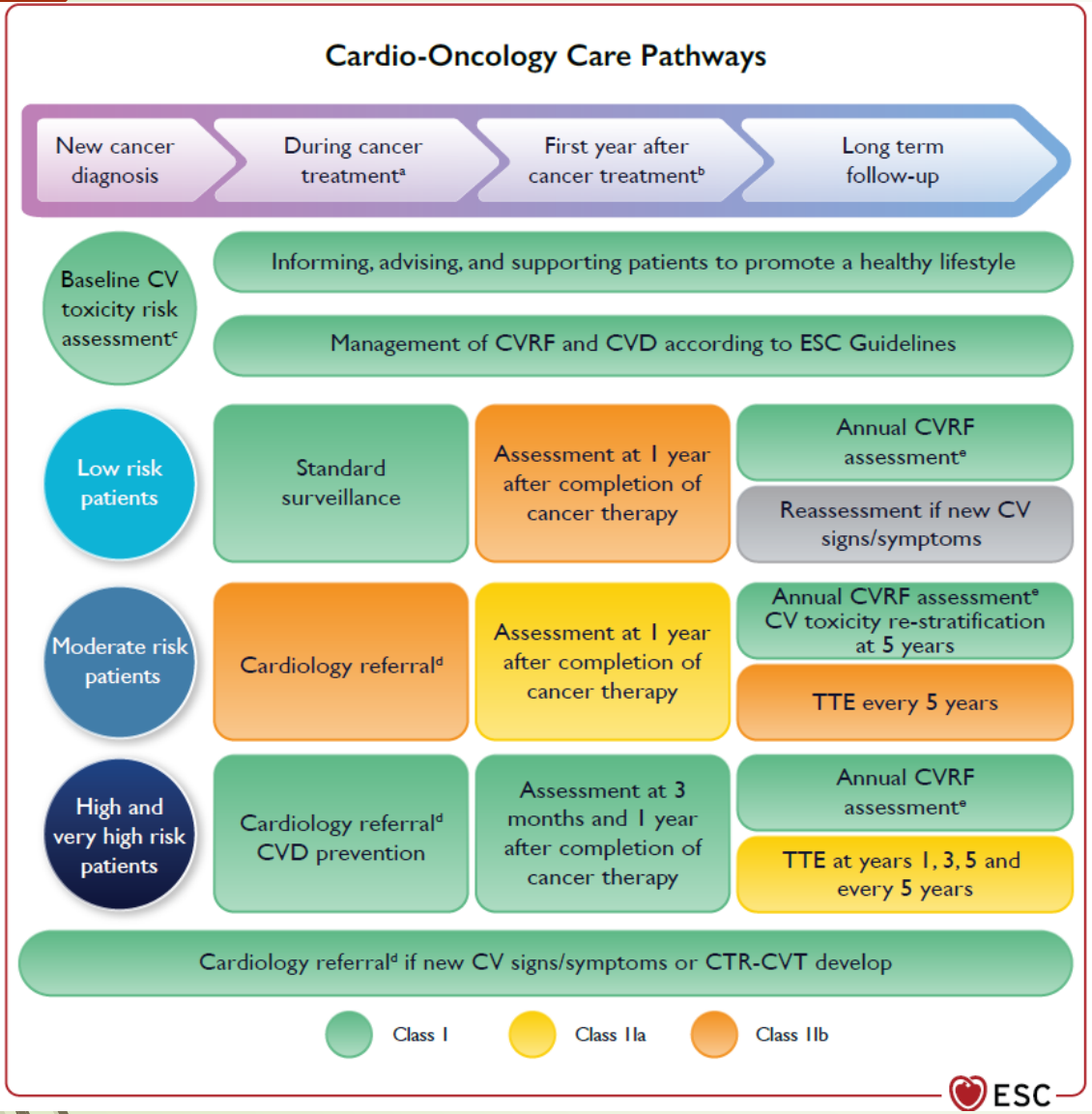
- Kardiális troponin T (cTnT) és kardiális troponin I (cTnI) használatosak
- Szervspecifikus, de nem betegségspecifikus markerek
- Változása, dinamikája alapján el lehet különíteni az akut és kr. miokardiális sérüléseket
- Összefüggés (közel lineáris) van a kardiomiociták sérülésének mértéke (TnT/I szint) valamint a HF kialakulása, HHF, mortalitás között
- A magas szenzitivitású analitikai módszerek → sokkal alacsonyabb detektálhatósági küszöböt eredményeztek (CAVE: gyártók közötti különbségek)

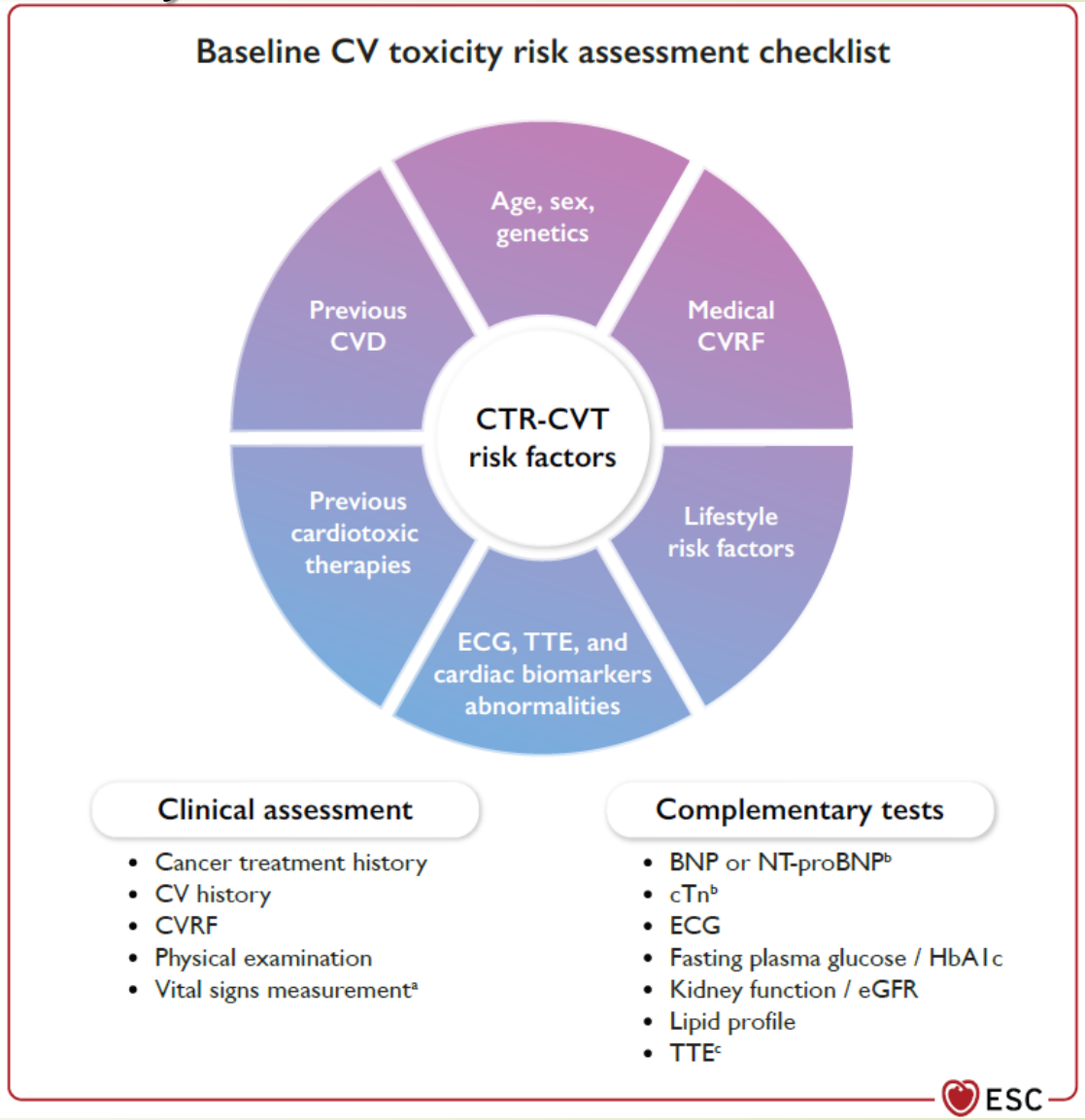
Table 2 Main causes of troponin release in cancer patients

Acute coronary syndromes – atherosclerotic plaque rupture, vasospasm
Anthracycline chemotherapy
Acute pulmonary embolus
Immune checkpoint inhibitor-mediated myocarditis
Atrial tachycardias including fast atrial fibrillation
Ventricular tachycardias
Acute pericarditis
Takotsubo syndrome
Supply–demand mismatch • Anaemia • Hypotension • Hypertensive crises • Sepsis • Acute rises in intracranial pressure
Direct myocardial infiltration (primary or metastatic cancer)

Pudil R. et al. Eur J Heart Fail. 2020;22(11):1966-1983.

Az onko-kardiológia betegek gondozása, rizikó stratifikációja





Lyon AR et. Eur Heart J Cardiovasc Imaging. 2022 Sep 10;23(10):e333-e465.

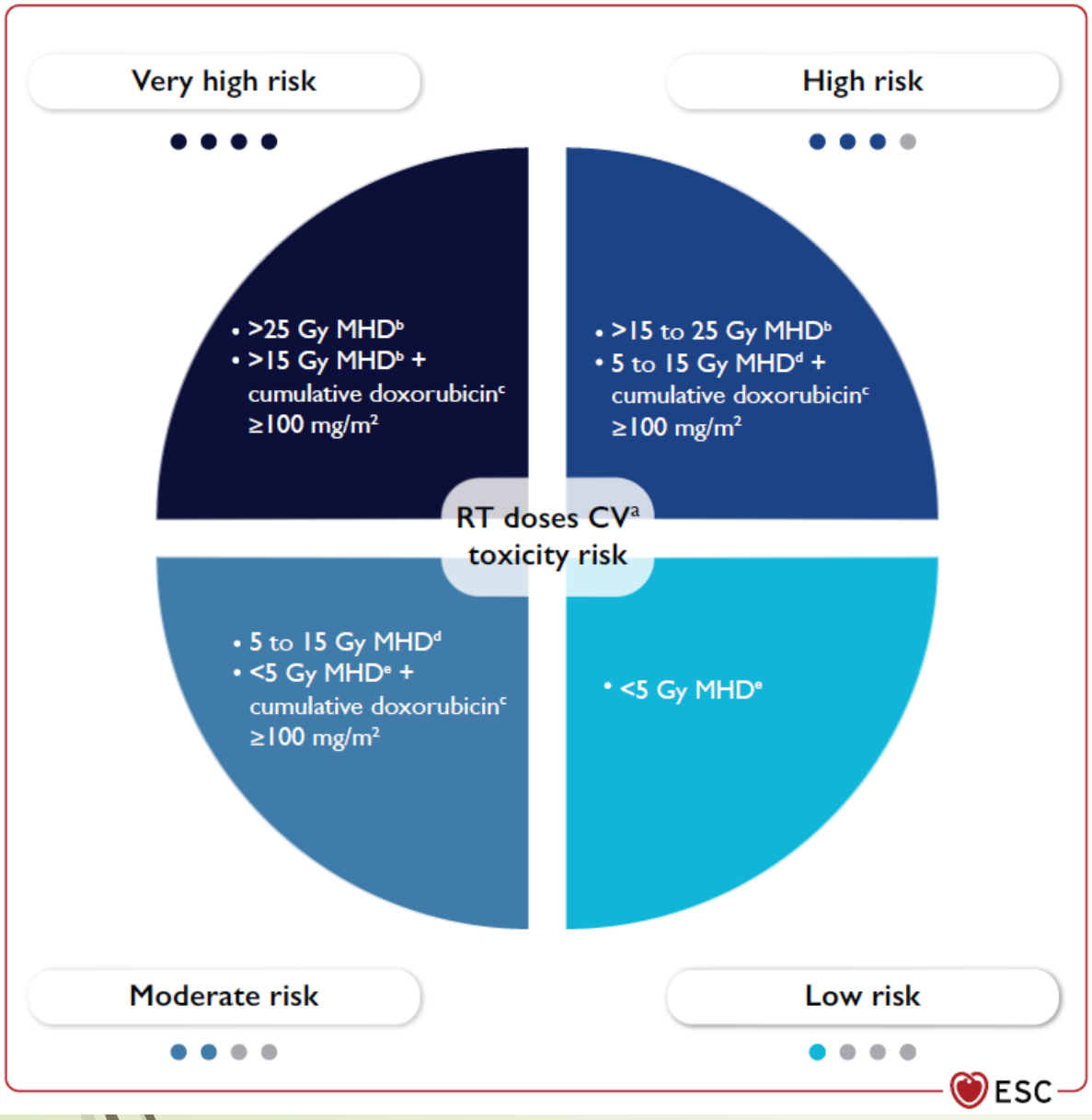
Table 4 Heart Failure Association–International Cardio-Oncology Society baseline cardiovascular toxicity risk stratification

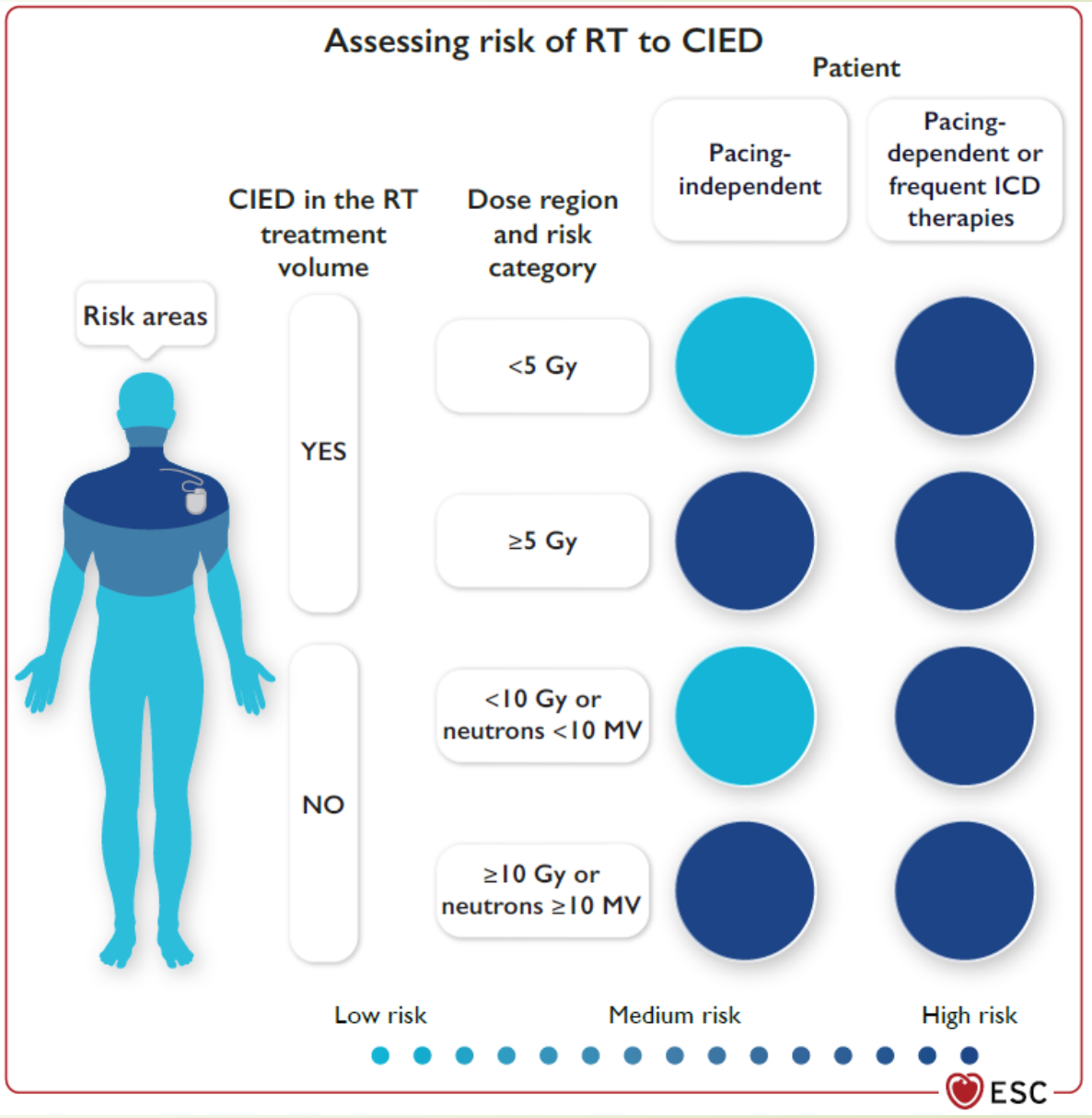
Baseline CV toxicity risk factors	Anthracycline chemotherapy	HER2-targeted therapies	VEGF inhibitors	BCR-ABL inhibitors	Multiple myeloma therapies	RAF and MEK inhibitors
Current cancer treatment						
Dexamethasone > 160 mg/month	–	–	–	–	M1	–
Includes anthracycline before HER2-targeted therapy	–	M1[g]	–	–	–	–
Previous exposure to						
Anthracycline	H	M2[h]	H	–	H	H
Trastuzumab	–	VH	–	–	–	–
RT to left chest or mediastinum	H	M2	M1	–	M1	M2
Lifestyle risk factors						
Current smoker or significant smoking history	M1	M1	M1	H	M1	M1
Obesity (BMI > 30 kg/m^2) (women)	M1	M1	M1	M1	M1	M1
Prior PI CV toxicity	–	–	–	–	VH	–
Prior IMiD CV toxicity	–	–	–	–	H	–
Cardiac imaging						
LVEF < 50%	H	H	H	H	H	H
LVEF 50–54%	M2	M2	M2	–	M2	M2
LV hypertrophy	–	–	–	–	M1	–
Cardiac amyloidosis	–	–	–	–	VH	–
Cardiac biomarkers						
Elevated baseline cTn[b]	M1	M2	M1	–	M2	M2
Elevated baseline NP[b]	M1	M2	M1	–	H	M2

Risk level: Low risk = no risk factors OR one moderate1 risk factor; **moderate risk (M)** = moderate risk factors with a total of 2–4 points (Moderate 1 [M1] = 1 point; Moderate [M2] = 2 points); **high risk (H)** = moderate risk factors with a total of ≥5 points OR any high-risk factor; **very-high risk (VH)** = any very-high risk factor.

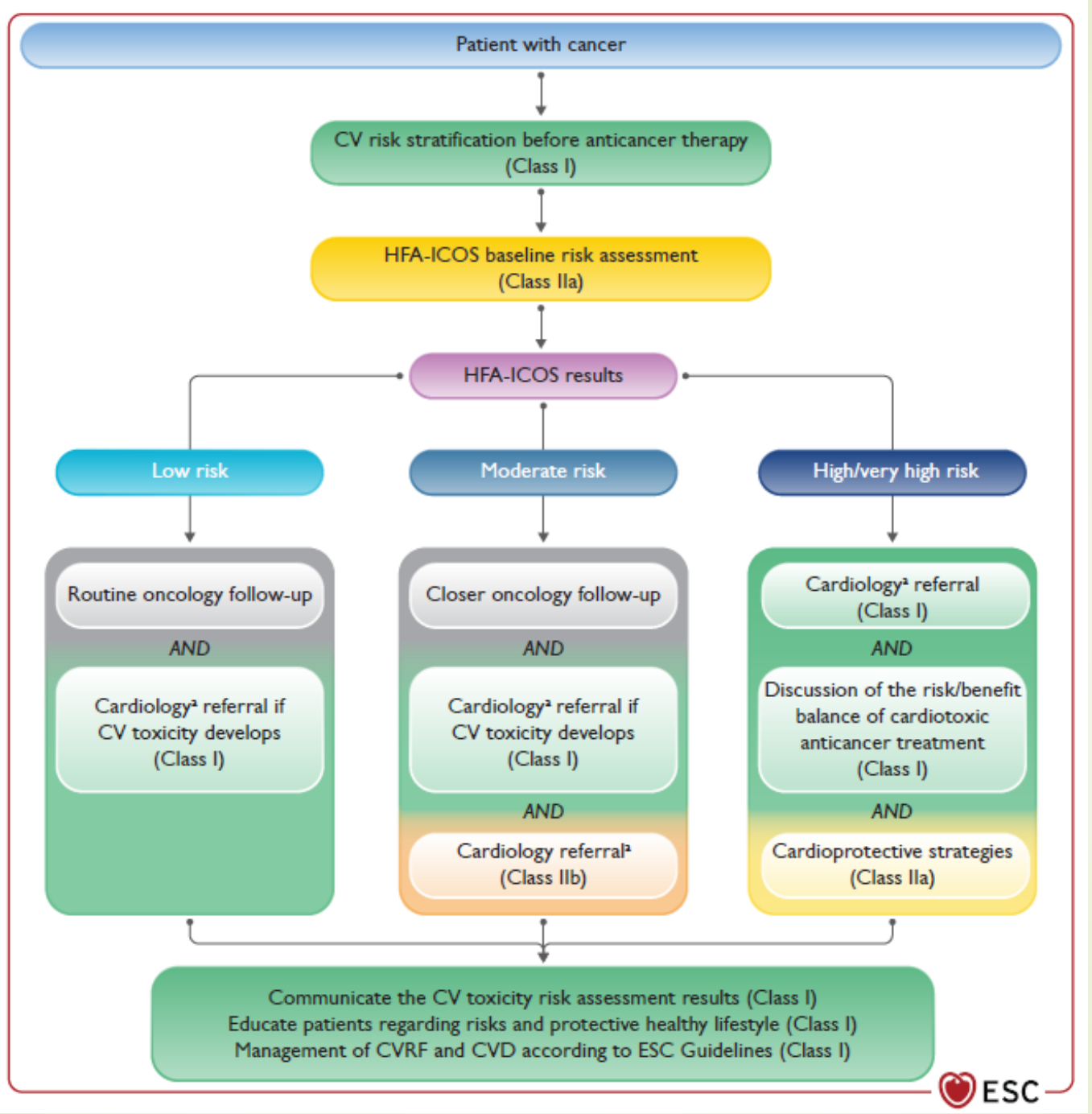
Lyon AR et. Eur Heart J Cardiovasc Imaging. 2022 Sep 10;23(10):e333-e465.

Radioterápia okozta kardiotoxicitás





Lyon AR et. Eur Heart J Cardiovasc Imaging. 2022 Sep 10;23(10):e333-e465.



Lyon A.R. et al. Eur Heart J. 2022 Nov 1;43(41):4229-4361.

Biomarkerek daganatellenes kezelés esetén

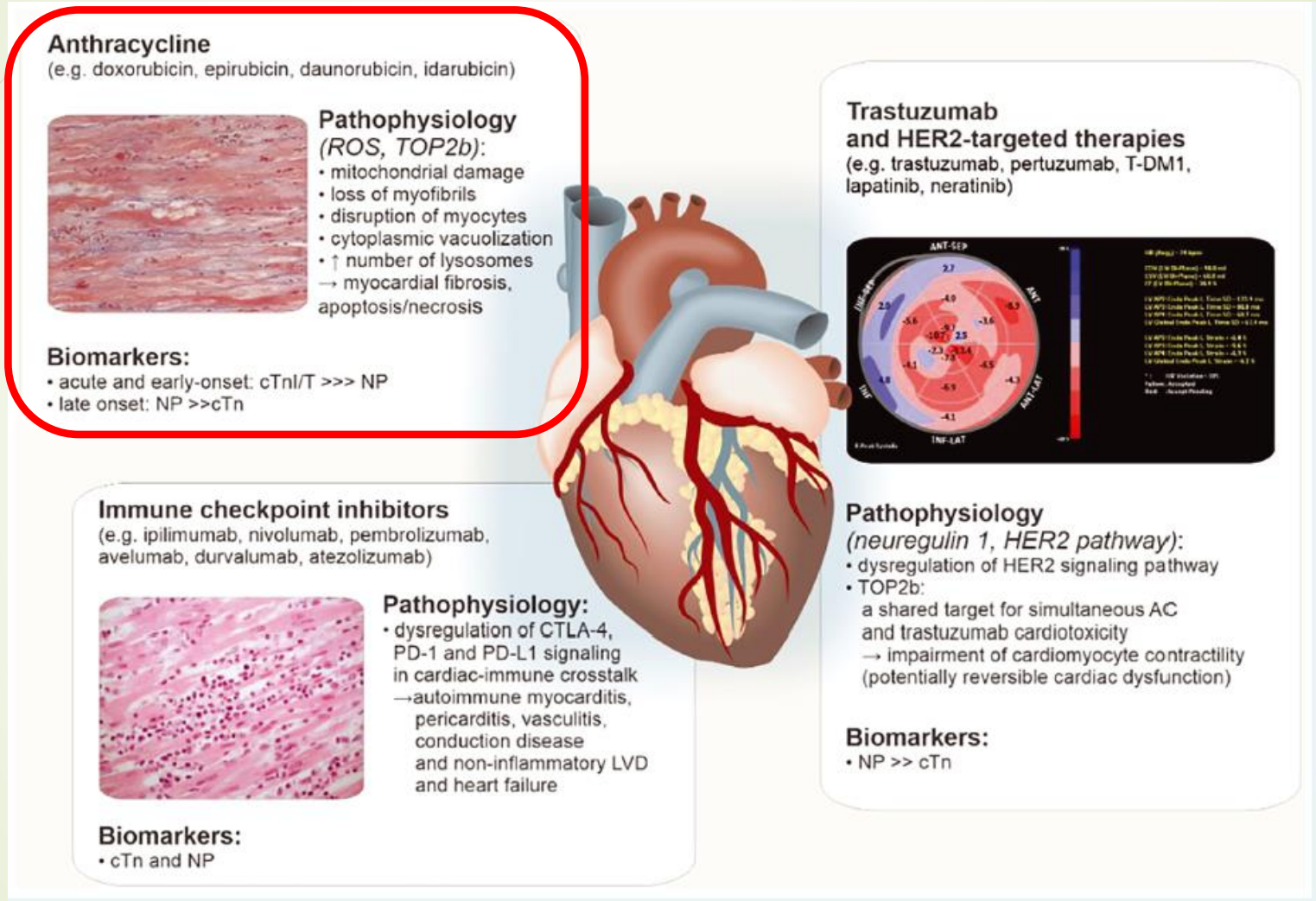


Figure 2 Biomarkers and pathophysiology in cardiotoxic cancer therapies. AC, anthracycline chemotherapy; CTLA-4, cytotoxic T-lymphocyte antigen-4; cTn, cardiac troponin; cTnI/T, cardiac troponin I/T; LVD, left ventricular dysfunction; NP, natriuretic peptide; PD-1, programmed cell death 1; PD-L1, programmed cell death ligand 1; ROS, reactive oxygen species; T-DM1, trastuzumab emtansine; TOP2b, topoisomerase II beta.

Pudil R. et al. Eur J Heart Fail. 2020;22(11):1966-1983.

Antraciklinek

- Előfordulás 5-6 %, de dózisfüggően meredeken nő
- Rossz prognózis, magas mortalitás (~50%/1 év)
- Akut forma: 3-5 nappal a szer adását követően perimyocarditis képe
- Krónikus forma (kumulatív dózis): 30 nap <, de akár évek múlva is: oxidatív stressz, krónikus gyulladás, fibrózis, DCM képe
- Számos kis betegszámú (néhány 100 beteg) vizsgálat alapján a hs-TnT/I akut forma esetén szenzitíven jelzi a miokardium toxicitást, viszont nem specifikus (más ok is okozhat pozitivitást), ilyenkor javasolt BNP/NT-proBNP-vel kiegészíteni
- Több kis betegszámú vizsgálat, metaanalízis alapján → NT-proBNP szignifikáns korrelációt mutat az BK diszfunkció (EF, GLS, diasztolés funkció) tekintetében AC kezelés kapcsán → krónikus forma

Pudil R. et al. Eur J Heart Fail. 2020;22(11):1966-1983.

Szívelégtelenség, tünetmentes balkamra diszfunkció: Antraciklinek

Toxicitás kialakulása dózis-dependens

- de: igen nagy a betegek közötti különbségek
- magasabb rizikó: korábbi szívbetegség, egyéb citotoxikus kezelés, magasabb dózis, életkor – idősek, gyerekek (fejlődő szív!!!)

Doxorubicin (Adriamycin): 400 mg/m^2: 3–5%
550 mg/m^2: 7–26%
700 mg/m^2: 18–48%
Idarubicin: >90 mg/m^2: 5–18%
Epirubicin: >900 mg/m^2: 0.9–11.4%
Mitoxantron: >120 mg/m^2: 2.6%
Liposzómális antraciklinek: >900 mg/m^2: 2%

Table 5 Anthracycline equivalence dose considering doxorubicin in rapid infusion as a reference[94]

Drug	Relative cardiotoxicity	Incidence of HF rises to >5% when cumulative dose exceeds (mg/m^2)
Doxorubicin rapid infusion	1	400
Epirubicin	0.7	900
Daunorubicin	~0.75	800
Idarubicin	0.53	150

Cardiac serum biomarkers		
Baseline measurement of NP and cTn is recommended in high- and very high-risk patients prior to anthracycline chemotherapy.[55,65,211]	I	B
Baseline measurement of NP and cTn should be considered in low- and moderate-risk patients prior to anthracycline chemotherapy.[211]	IIa	C
cTn and NP monitoring before every cycle during anthracycline chemotherapy and 3 and 12 months after therapy completion is recommended in high- and very high-risk patients.[55,175,211]	I	B
cTn and NP monitoring every two cycles during anthracycline chemotherapy and within 3 months after therapy completion should be considered in moderate-risk patients and in low-risk patients receiving a cumulative dose of ≥250 mg/m^2 of doxorubicin or equivalent.[55,59,212,213]	IIa	C
cTn and NP monitoring every two cycles during anthracycline chemotherapy and within 3 months after therapy completion may be considered in low-risk patients.[55,59,212,213]	IIb	C

Biomarkerek daganatellenes kezelés esetén

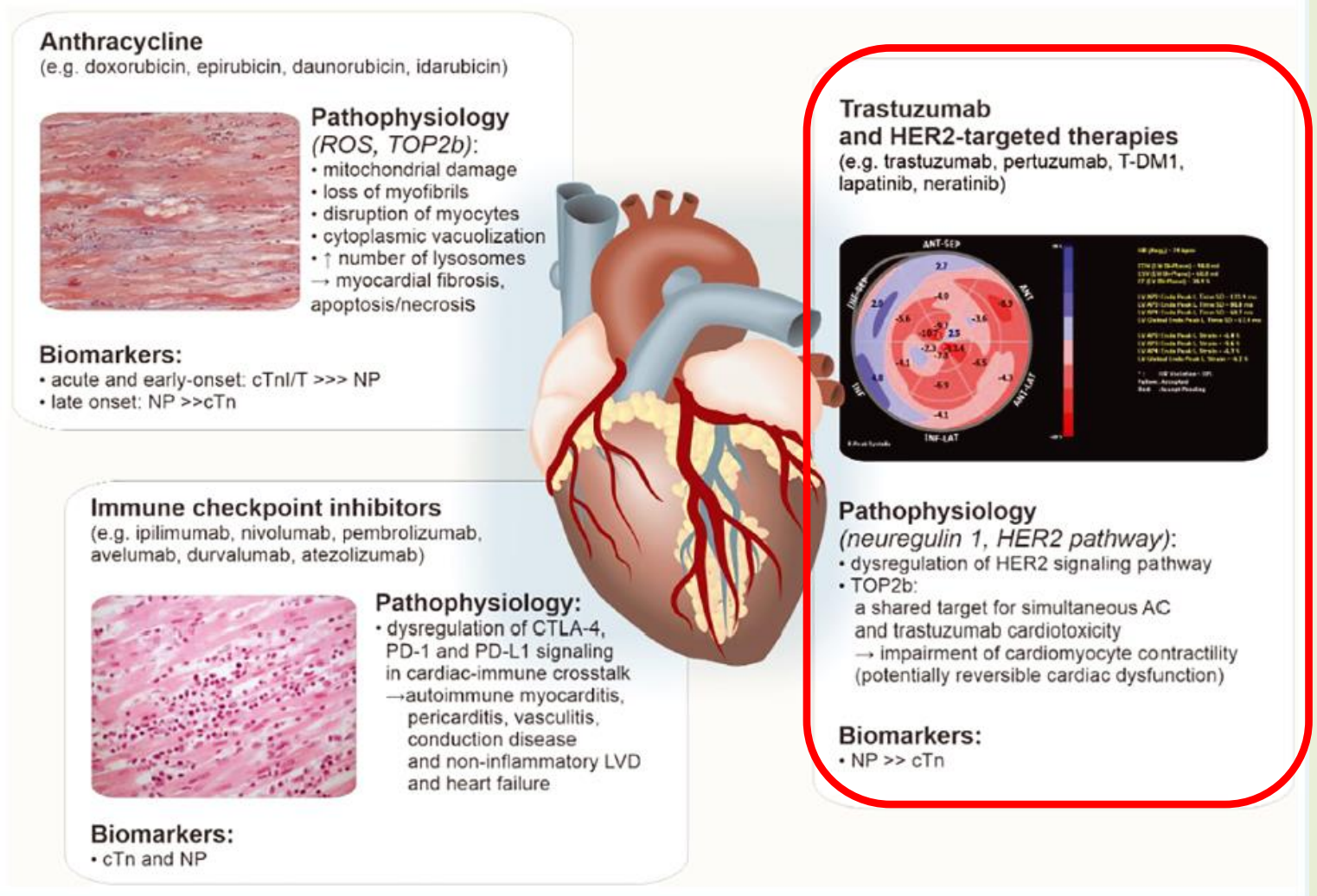


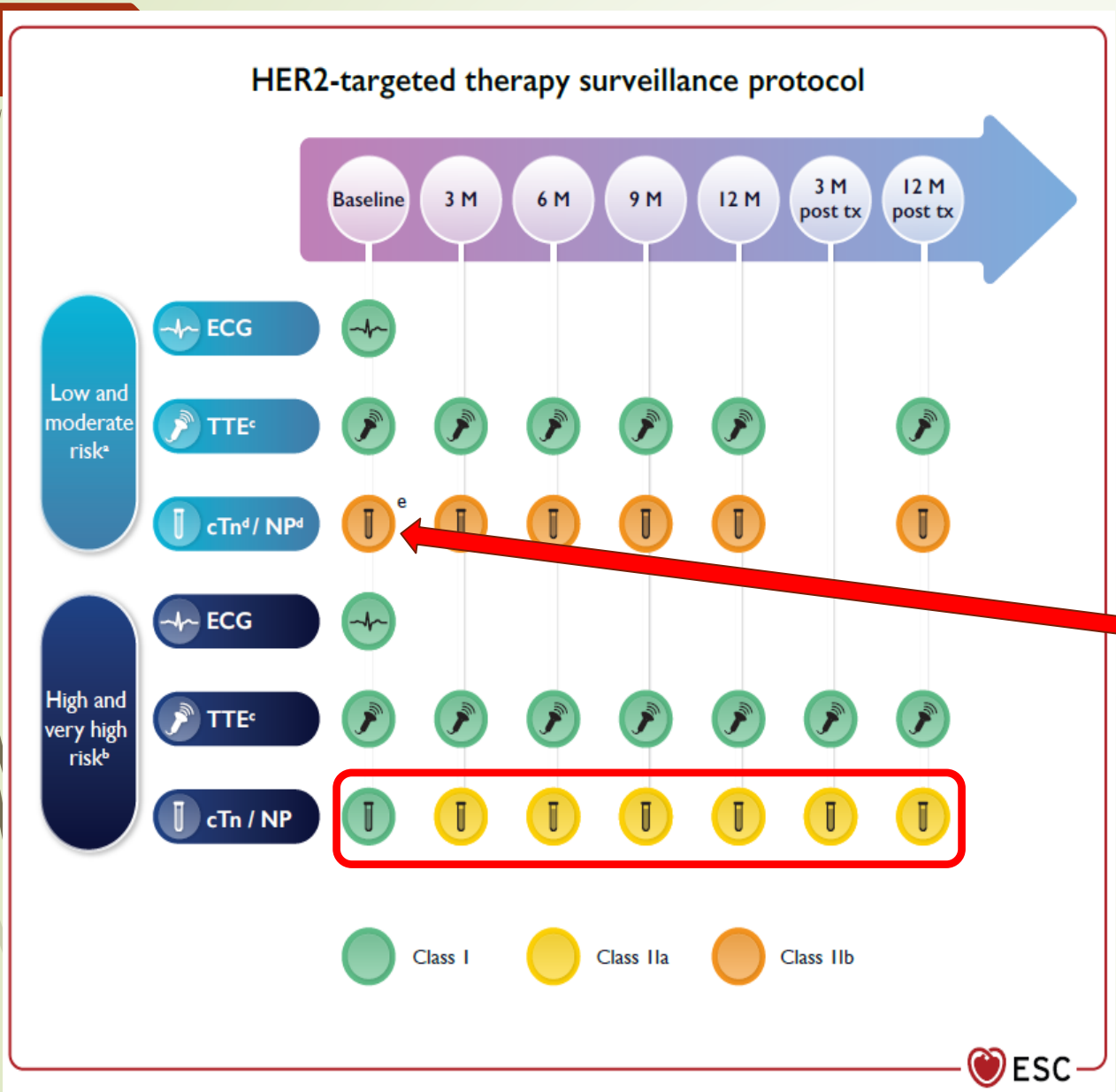
Figure 2 Biomarkers and pathophysiology in cardiotoxic cancer therapies. AC, anthracycline chemotherapy; CTLA-4, cytotoxic T-lymphocyte antigen-4; cTn, cardiac troponin; cTnI/T, cardiac troponin I/T; LVD, left ventricular dysfunction; NP, natriuretic peptide; PD-1, programmed cell death 1; PD-L1, programmed cell death ligand 1; ROS, reactive oxygen species; T-DM1, trastuzumab emtansine; TOP2b, topoisomerase II beta.

Pudil R. et al. Eur J Heart Fail. 2020;22(11):1966-1983.

Anti-HER2 terápia

- Létfontosságú támadáspont a HER2 pozitív (human epidermális növekedési faktor receptor) emlőtumor esetén (neoadjuváns, adjuváns)
- Akár 15–20%-ban okozhat bal kamra diszfunkciót (LVD)
- Gyakran kombinációban alkalmazzák (5-FU, platinaszármazékok, antraciklin)→ nagyobb rizikó
- Mechanizmus: neuroregulin-1 jelátviteli út gátlása → protektív pro-survival utak sérülnek → miokardium vulnerabilitás fokozódik (pl. ROS)
- korai és késői forma egyaránt jellemző → DCM képe (potenciálisan reverzibilis)
- NT-proBNP szenzítívebben jelzi a trastuzumab okozta LVD a cTroponin-nál (HERA trial, 533 emlőtumoros nő)
- AC kezelés után tervezett anti-HER2 terápia előtt mért induló cTroponin rendkívül szenzitív kardiotoxicitás (LV diszfunkció) irányába (Michel et al., meta-analízis)

Pudil R. et al. Eur J Heart Fail. 2020;22(11):1966-1983.
Michel L, et al. Eur J Heart Fail 2020;22:350–361.



Cardiac biomarkers		
Baseline NP and cTn measurement are recommended in high- and very high-risk patients prior to anti-HER2-targeted therapies.[227,228]	I	C
NP and cTn monitoring every 2–3 cycles during therapy and 3 and 12 months after the end of therapy should be considered in high- and very high-risk HER2+ EBC patients.[d,55]	IIa	C
Baseline cTn measurement should be considered in low- and moderate-risk patients post-anthracycline chemotherapy but prior to starting anti-HER2-targeted therapies.[55,62]	IIa	A
NP and cTn monitoring at baseline, every 3 months, and 12 months after therapy may be considered in low- and moderate-risk HER2+ EBC patients.[d,55]	IIb	C

Biomarkerek daganatellenes kezelés esetén

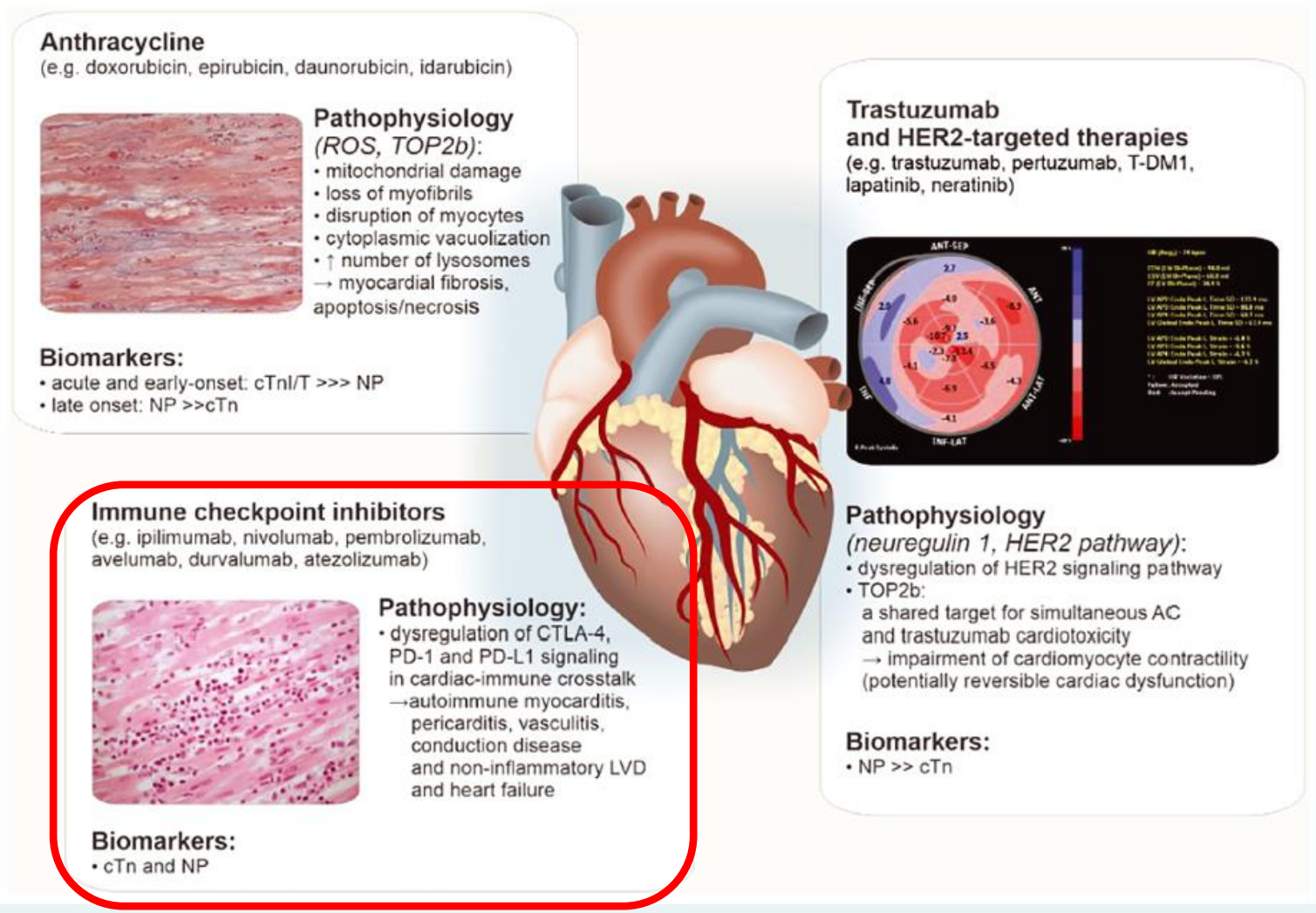


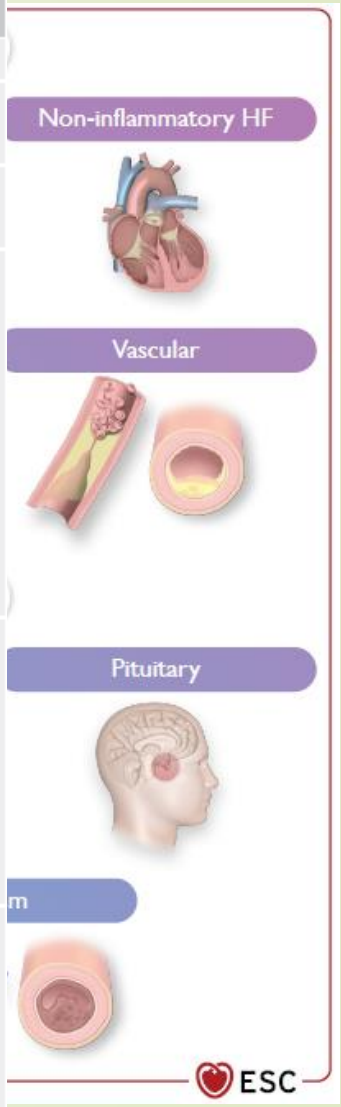
Figure 2 Biomarkers and pathophysiology in cardiotoxic cancer therapies. AC, anthracycline chemotherapy; CTLA-4, cytotoxic T-lymphocyte antigen-4; cTn, cardiac troponin; cTnI/T, cardiac troponin I/T; LVD, left ventricular dysfunction; NP, natriuretic peptide; PD-1, programmed cell death 1; PD-L1, programmed cell death ligand 1; ROS, reactive oxygen species; T-DM1, trastuzumab emtansine; TOP2b, topoisomerase II beta.

Pudil R. et al. Eur J Heart Fail. 2020;22(11):1966-1983.

ICI terápia

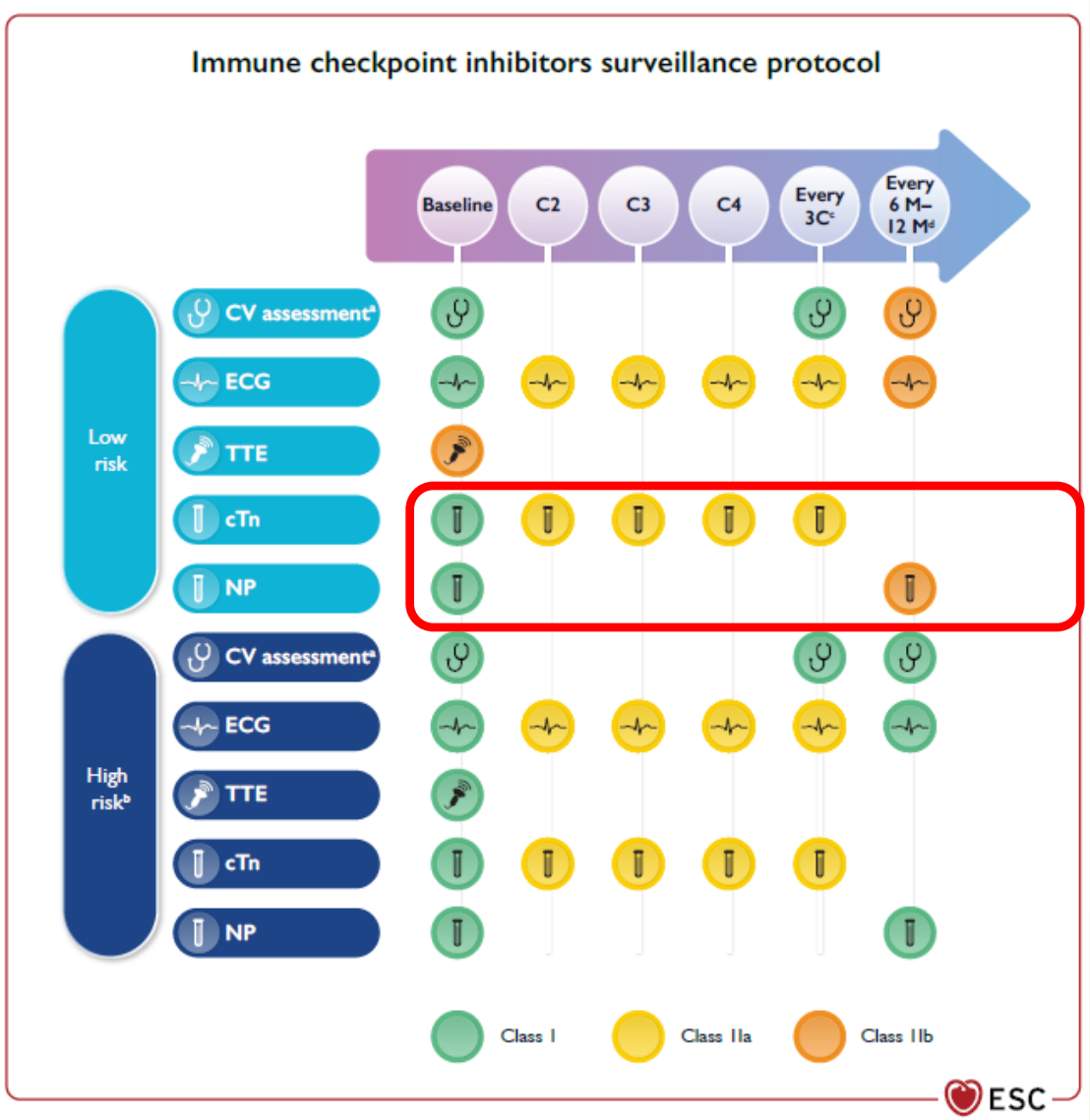
- ICI
- Im
- Elő
- Dg
- A b

ICI myocarditis (either pathohistological diagnosis or clinical diagnosis)	
Pathohistological diagnosis (EMB)	Multifocal inflammatory cell infiltrates with overt cardiomyocyte loss by light microscopy
Clinical diagnosis[d]	**cTn elevation** (new or significant change from baseline)[e] **with 1 major criterion or 2 minor criteria,** after exclusion of ACS and acute infectious myocarditis based on clinical suspicion[f]
	Major criterion: • CMR diagnostic for acute myocarditis (modified Lake Louise criteria)[g]
	Minor criteria: • Clinical syndrome (including any one of the following: fatigue, myalgias, chest pain, diplopia, ptosis, shortness of breath, orthopnoea, lower-extremity oedema, palpitations, light-headedness/dizziness, syncope, muscle weakness, cardiogenic shock) • Ventricular arrhythmia (including cardiac arrest) and/or new conduction system disease • Decline in LV systolic function, with or without regional wall motion abnormalities in a non-Takotsubo pattern • Other immune-related adverse events, particularly myositis, myopathy, myasthenia gravis • Suggestive CMR[h]
Severity of myocarditis	• **Fulminant:** Haemodynamic instability, HF requiring non-invasive or invasive ventilation, complete or high-grade heart block, and/or significant ventricular arrhythmia • **Non-fulminant:** including symptomatic but haemodynamically and electrically stable patients and incidental cases diagnosed at the same time as other immuno-related adverse events. Patients may have reduced LVEF but no features of severe disease • **Steroid refractory**: non-resolving or worsening myocarditis (clinical worsening or persistent troponin elevation after exclusion of other aetiologies) despite high-dose methylprednisolone
Recovery from myocarditis	• **Complete recovery**: Patients with complete resolution of acute symptoms, normalization of biomarkers, and recovery of LVEF after discontinuation of immunosuppression. CMR may still show LGE or elevated T1 due to fibrosis, but any suggestion of acute oedema should be absent • **Recovering**: Ongoing improvement in patient clinical symptoms, signs, biomarkers, and imaging parameters, but not yet normalized, while on tapering doses of immunosuppression



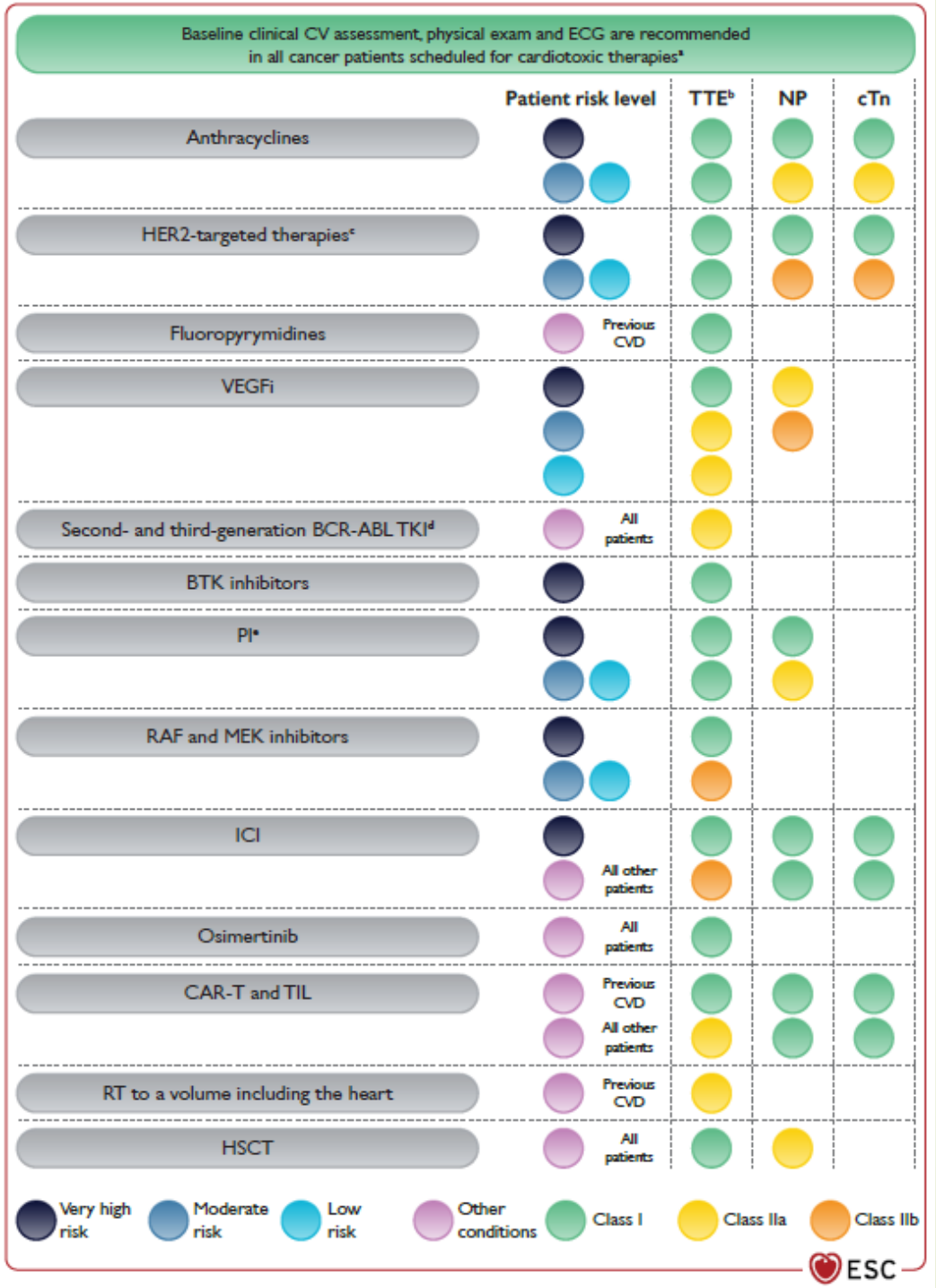
Pudil R. et al. Eur J Heart Fail. 2020;22(11):1966-1983.

ICI terápia



Recommendations	Class[a]	Level[b]
ECG, NP, and cTn measurements are recommended in all patients before starting ICI therapy.[333]	I	B
Serial ECG and cTn measurements should be considered before ICI doses 2, 3, and 4, and if normal, reduce to every three doses until completion of therapy to detect subclinical ICI-related CV toxicity.[333]	IIa	B

Lyon A.R. et al. Eur Heart J. 2022 Nov 1;43(41):4229-4361.



Lyon A.R. et al. Eur Heart J. 2022 Nov 1;43(41):4229-4361.

Kezelési lehetőségek

Kezelési lehetőségek

Lifestyle modifications

- Smoking cessation

- Aerobic exercise

- Weight loss

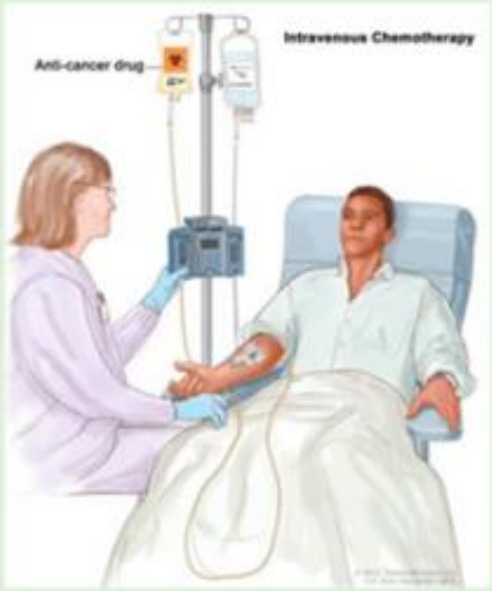
- Healthy diet

Pharmacological therapy

- Beta blockers
- Renin-angiotensin inhibitors
  - ACE inhibitors
  - ARBs
- Mineralocorticoid receptor antagonists
  -Aldosterone antagonists
- Sacubitril/valsartan
- Statins
- Dexrazoxane

Chemotherapy modifications

- Reduction of dose
- Slow infusion
- Special formulations



Courek C. J. Cardiovasc. Dev. Dis. 2022, 9(8), 259

2022-es Kardio-onkológiai guideline ajánlásainak evidenciaszintjei

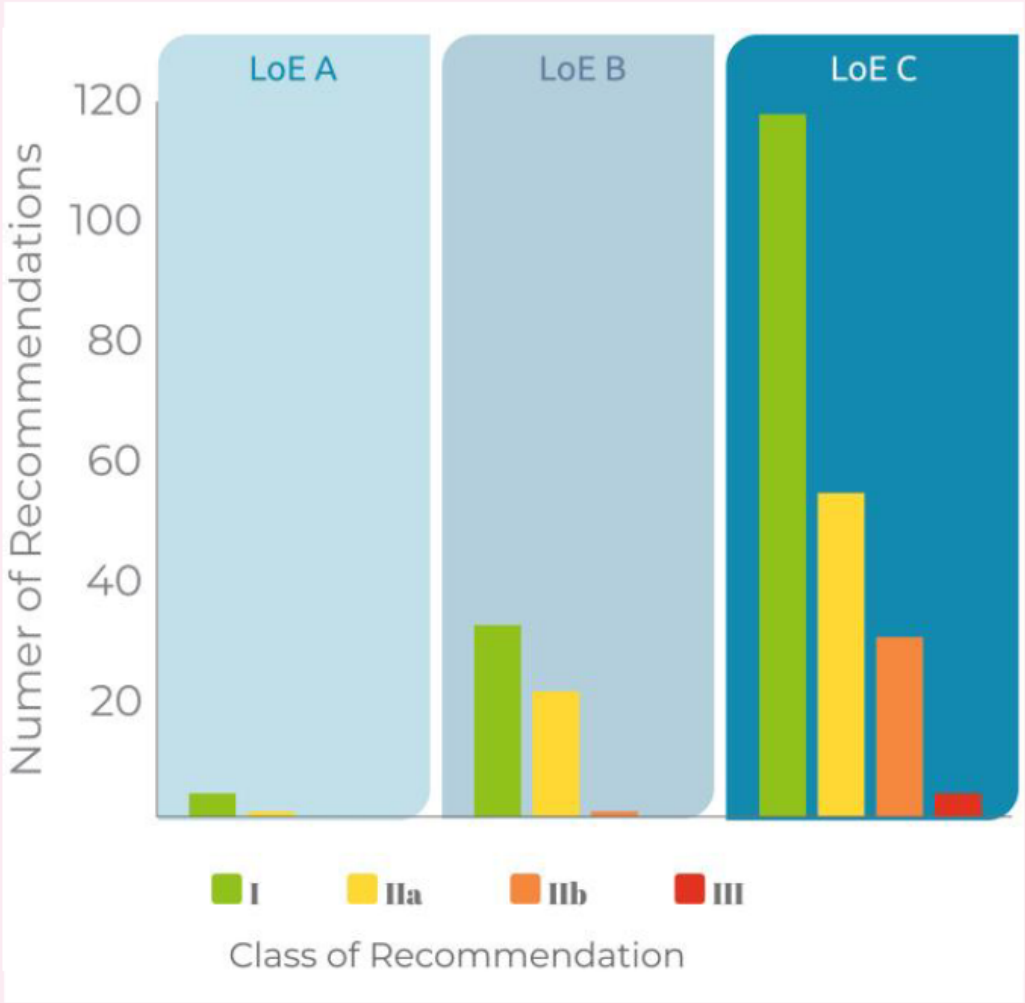
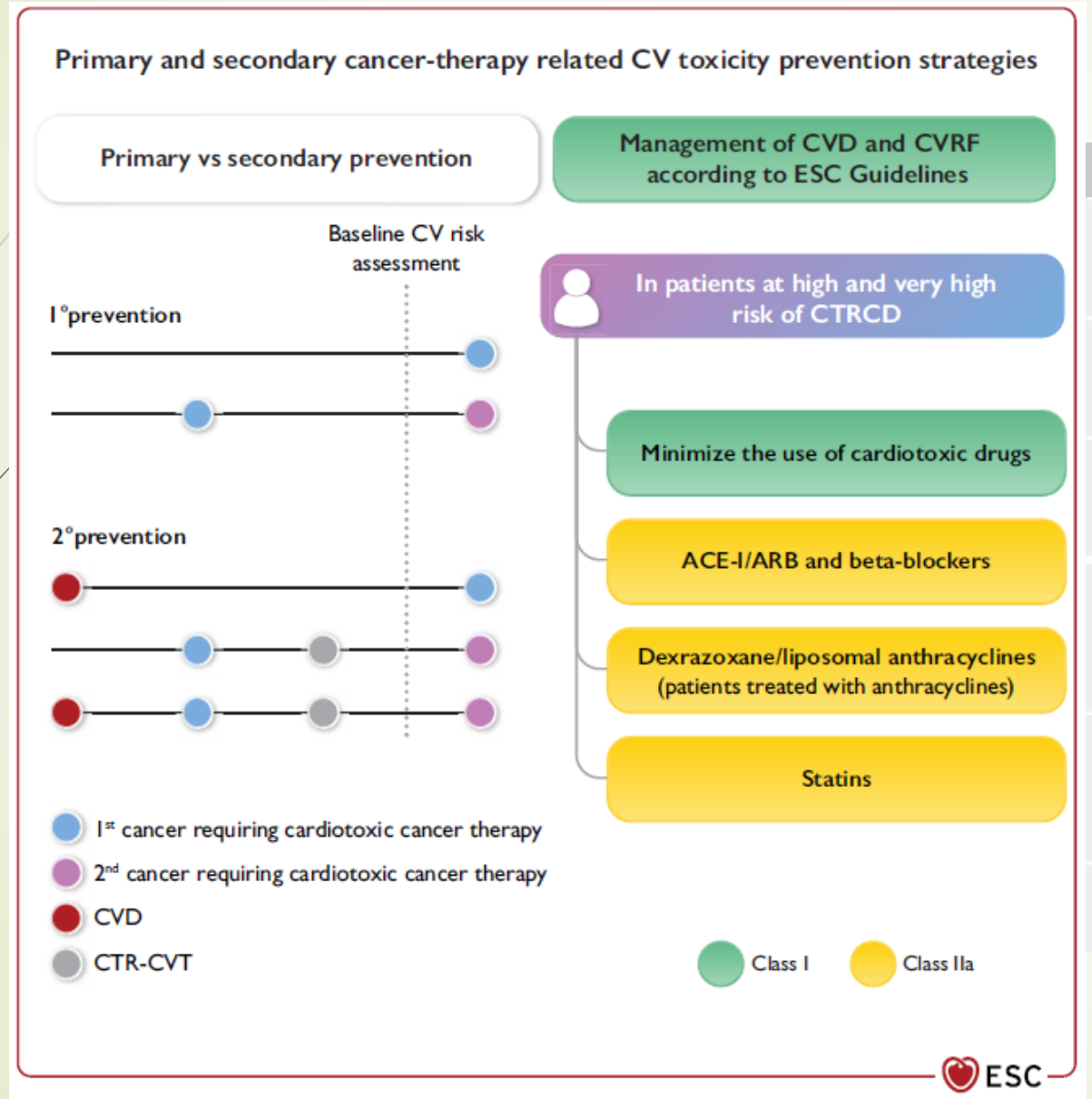


Figure 1 Summary of all guideline recommendations and corresponding level of evidence.

Eur Heart J - Cardiovasc Imaging 24, e45–e46, 2023.

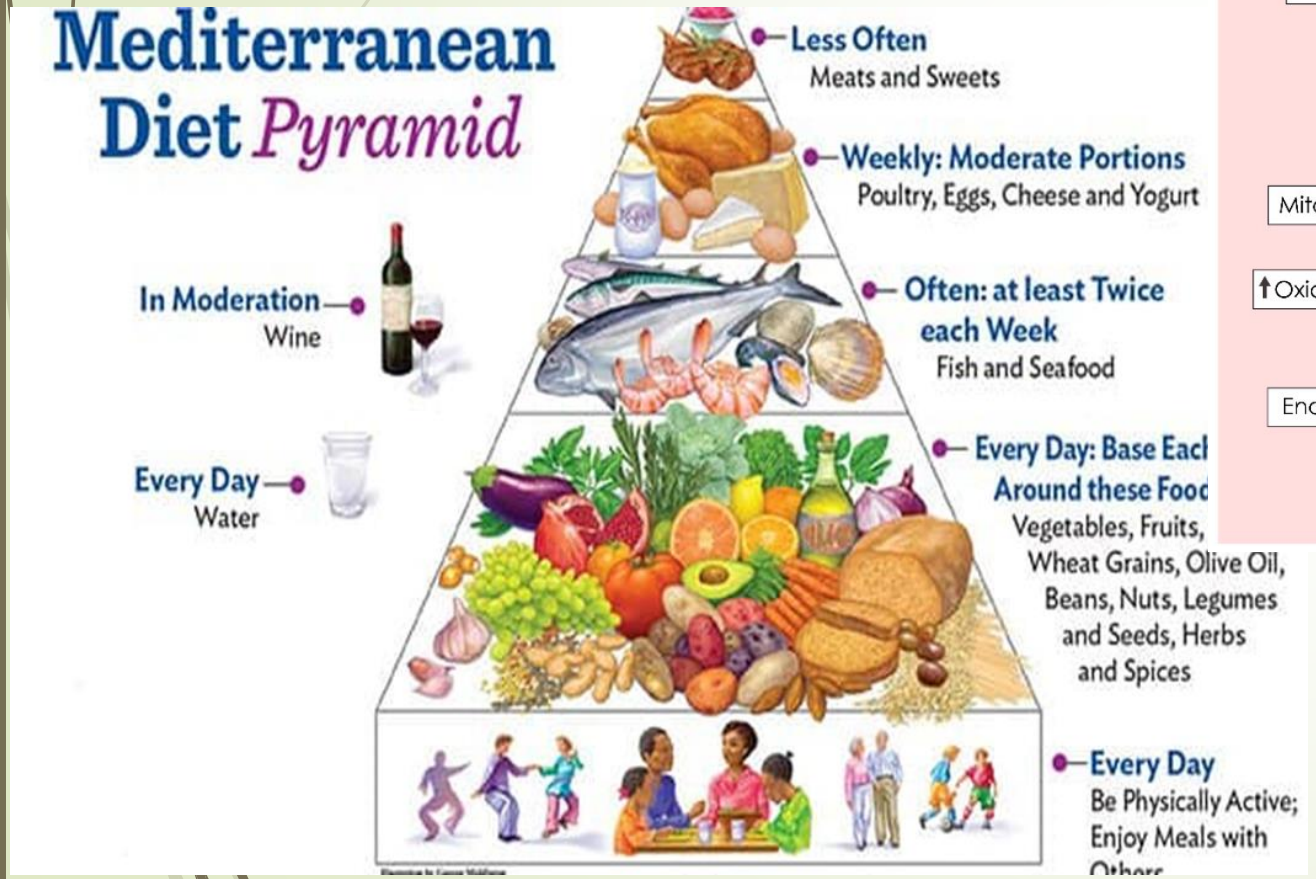
Prevenció, terápiás lehetőségek

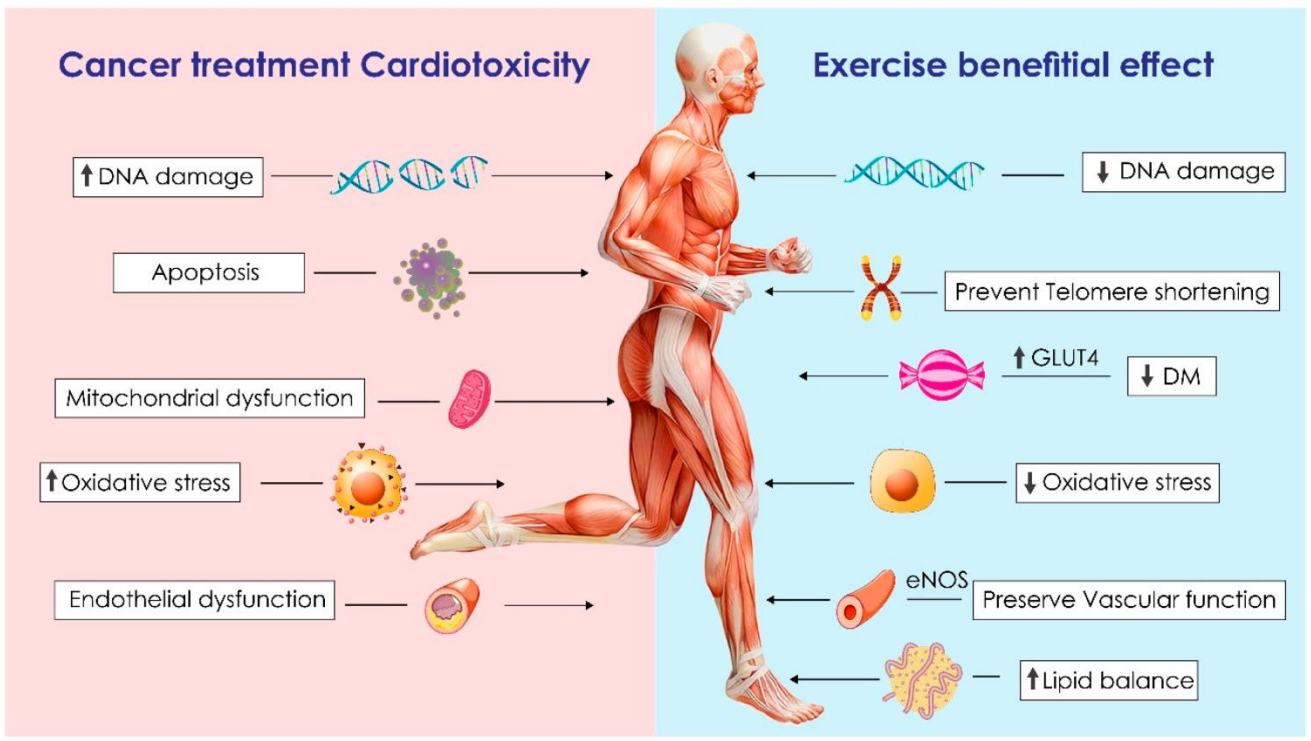


Recommendations	Class[a]	Level[b]
Management of CVRF according to the 2021 ESC Guidelines on CVD prevention in clinical practice is recommended before,[c] during, and after cancer therapy.[19]	I	C
Dexrazoxane should be considered in adult patients with cancer at high and very high CV toxicity risk when anthracycline chemotherapy is indicated.[d,158]	IIa	B
Liposomal anthracyclines should be considered in adult patients with cancer at high and very high CV toxicity risk when anthracycline chemotherapy is indicated.[e,164,165,168]	IIa	B
ACE-I or ARB and beta-blockers recommended for HF[f] should be considered for primary prevention in high- and very high-risk patients receiving anthracyclines and/or anti-HER2 therapies.[145,150,155–157,159,160,175]	IIa	B
ACE-I or ARB and beta-blockers recommended for HF[f] should be considered for primary prevention in high- and very high-risk patients receiving targeted cancer therapies that may cause HF.[g]	IIa	C
Statins should be considered for primary prevention in adult patients with cancer at high and very high CV toxicity risk.[h,149,176–185]	IIa	B

Lyon A.R. et al. Eur Heart J. 2022 Nov 1;43(41):4229-4361.

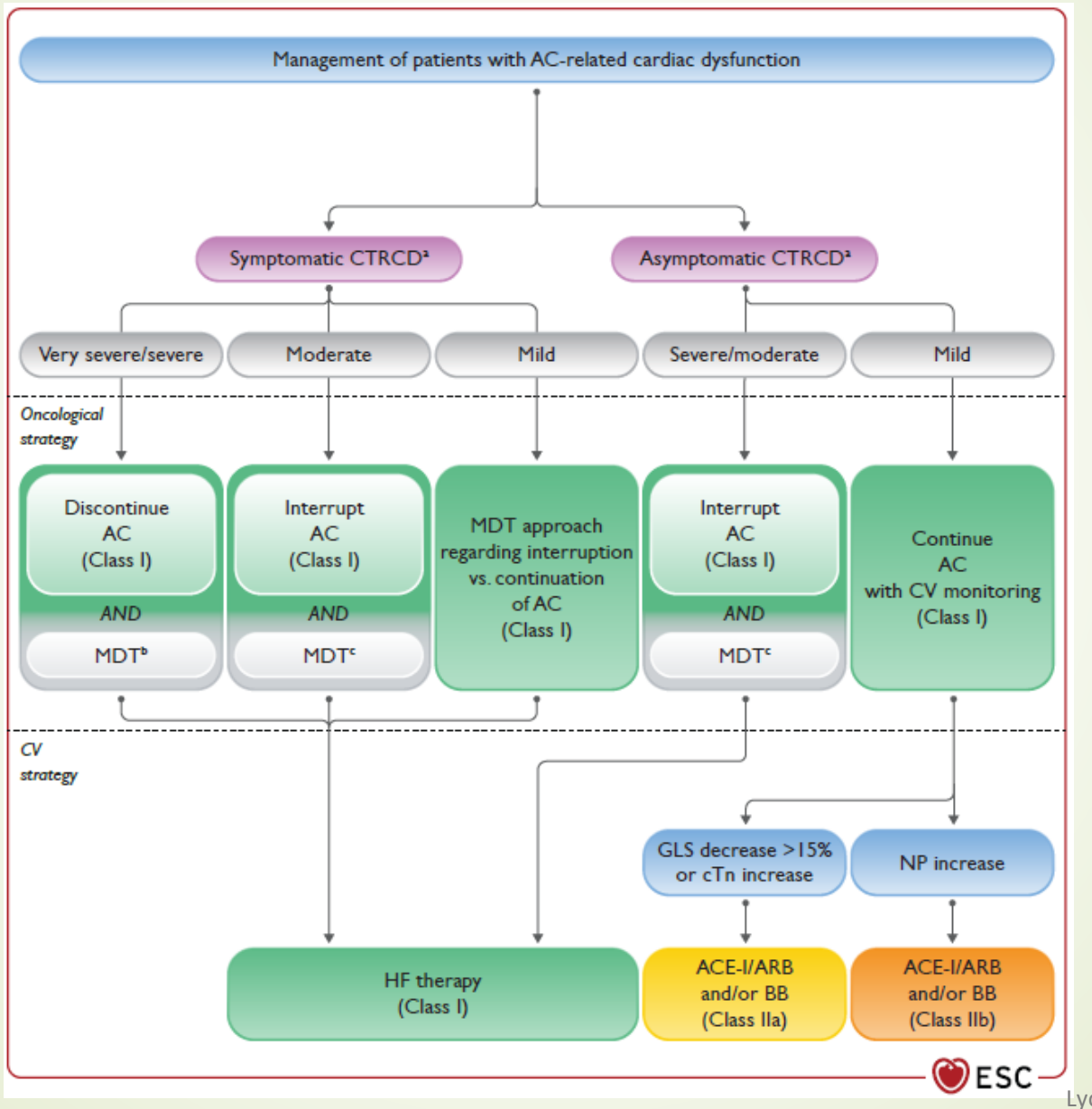
Életmódi javaslatok onkológiai betegeknek (is)





Life 12(7), 1006, 2022.
Eur Heart J 37, 2768–2801, 2016.

Antraciklin kardiotoxicitás ellátási protokollja



Lyon A.R. et al. Eur Heart J. 2022 Nov 1;43(41):4229-4361.

A jelen és a jövő kardioprotektív gyógyszerei

Table 1 Summary of currently available and emerging cardioprotective therapies

Therapy	Proposed mechanism(s)	Level of evidence	Limitations
Cu			
Em			
		models[37,38]	unknown efficacy in patients
CYP1 inhibitors	Under investigation	Preclinical studies—*in vivo* animal models[39]	Unknown efficacy in patients

TABLE 2 Recent Randomized Controlled Trials on Cardioprotection During Trastuzumab Therapy

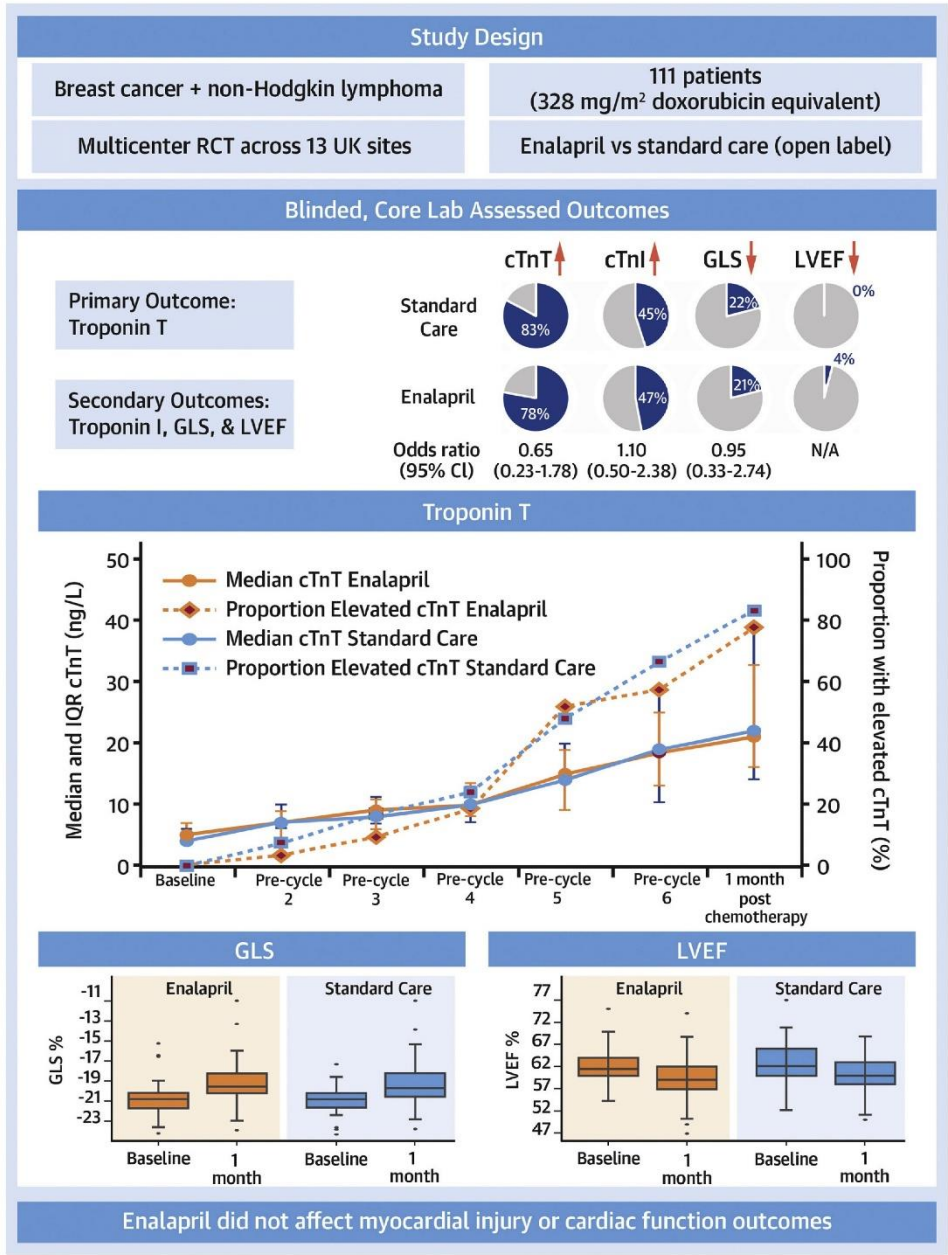
Trial	Trial Design	Trial Intervention	Imaging Method	N	Result of Primary Endpoint	Result of Key Secondary Endpoints
Pharmacologic intervention						
MANTICORE 101-Breast[44]	Randomized Placebo-controlled Double-blind Few were treated with anthracyclines	Bisoprolol/ perindopril/placebo	CMR	99	No between-group difference in LVEDVi	• Bisoprolol attenuated the decline in LVEF • Perindopril attenuated the decline in LVEF
Boekhout et al[45]	Randomized Multicenter Placebo-controlled Double-blind All were treated with anthracycline in advance	Candesartan/placebo	MUGA	210	No between-group difference in incidence of cardiotoxicity, defined as decline in LVEF of ≥15% or ≤15% to an absolute value < 45%	No between-group differences in changes in LVEF, troponin T, or NT-proBNP
Guglin et al[46]	Randomized Multicenter Placebo-controlled 189 were treated with anthracyclines	Lisinopril/carvedilol/ placebo	Echocardiography MUGA	468	No between-group difference in incidence of cardiotoxicity, defined as a reduction in LVEF of ≥10% or a decrease of ≥5% to a value <50%	• Reduction in the incidence of cardiotoxicity if patients treated with sequential anthracyclines in both lisinopril and carvedilol arms • No between-group difference if no anthracycline exposure

LVEDVi = left ventricular end-diastolic indexed volume; MANTICORE 101-Breast = Multidisciplinary Approach to Novel Therapies in Cardio-Oncology Research; MUGA = multigated acquisition; NT-proBNP = N-terminal pro-B-type natriuretic peptide; other abbreviations as in Table 1.

JACC CardioOnc 4, 19–37, 2022.
Cardiovasc Res 115, 915–921, 2019.

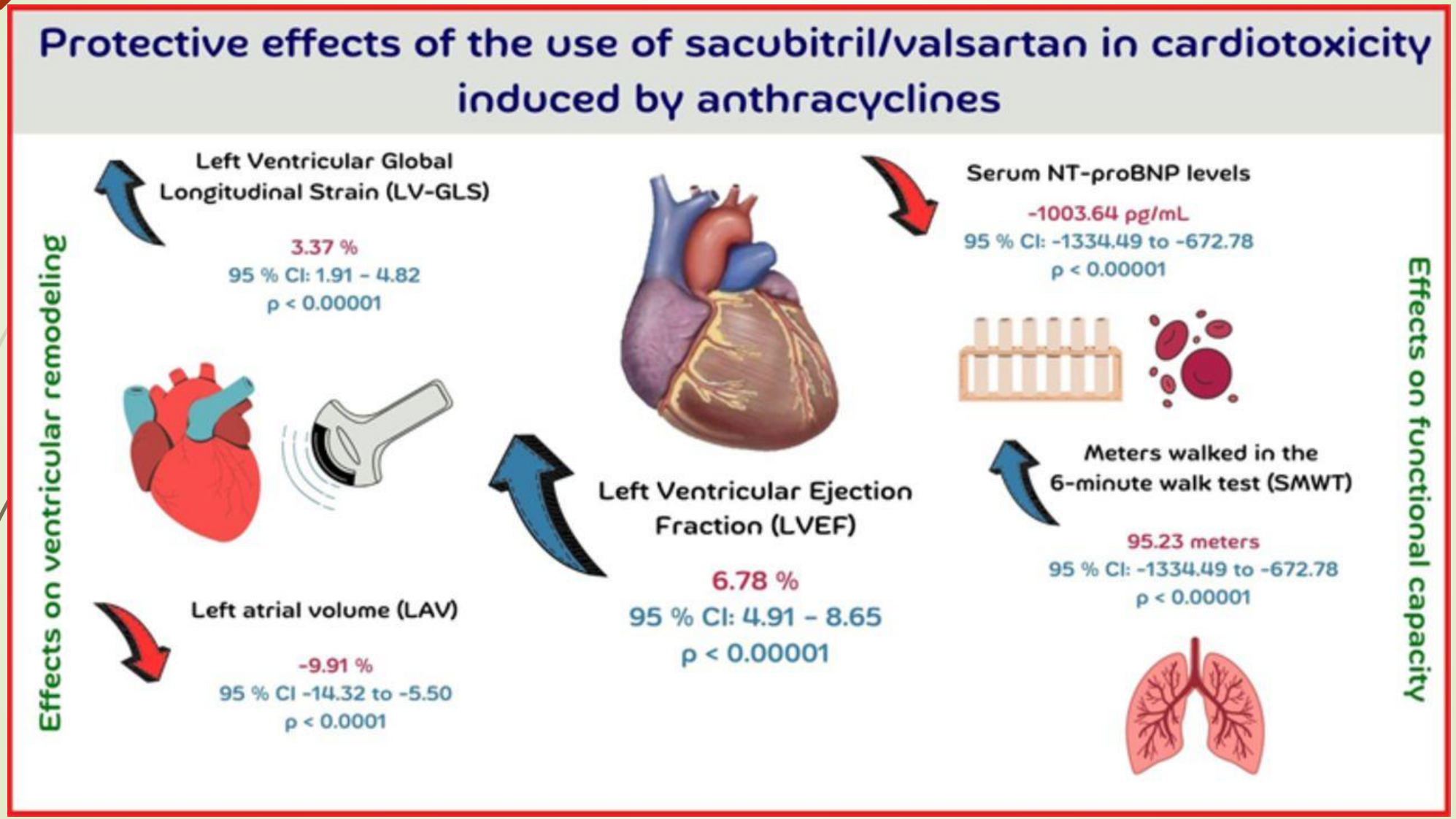
PROACT Clinical Trial

CENTRAL ILLUSTRATION: Does Enalapril Prevent Cardiotoxicity When Given Before and During High-Dose Anthracycline Chemotherapy?



Austin D, et al. JACC CardioOncol. 2024;10.1016/j.jaccao.2024.07.010

ARNI
–meta-analízis



Trejo F.I.L. BMJ. doi: https://doi.org/10.1101/2023.08.14.23294099

ARNI hatása daganatos betegek szívelégtelenségére- szekunder prevenciós vizsgálat

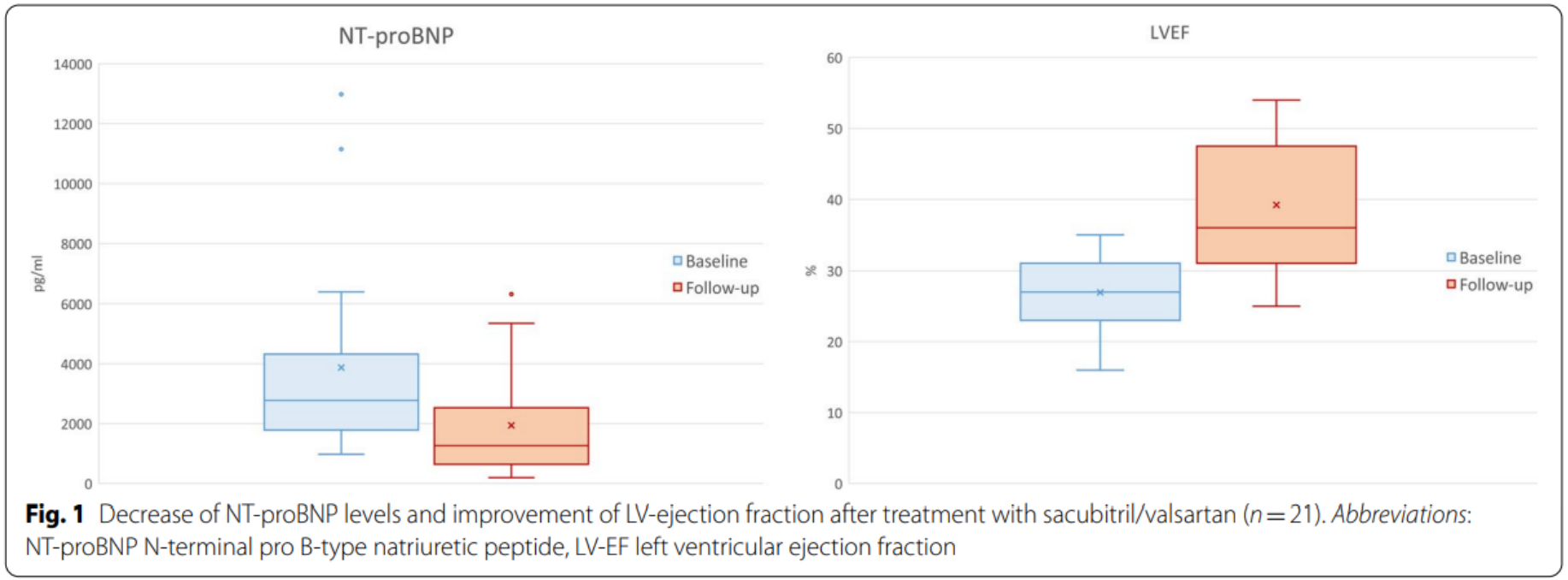
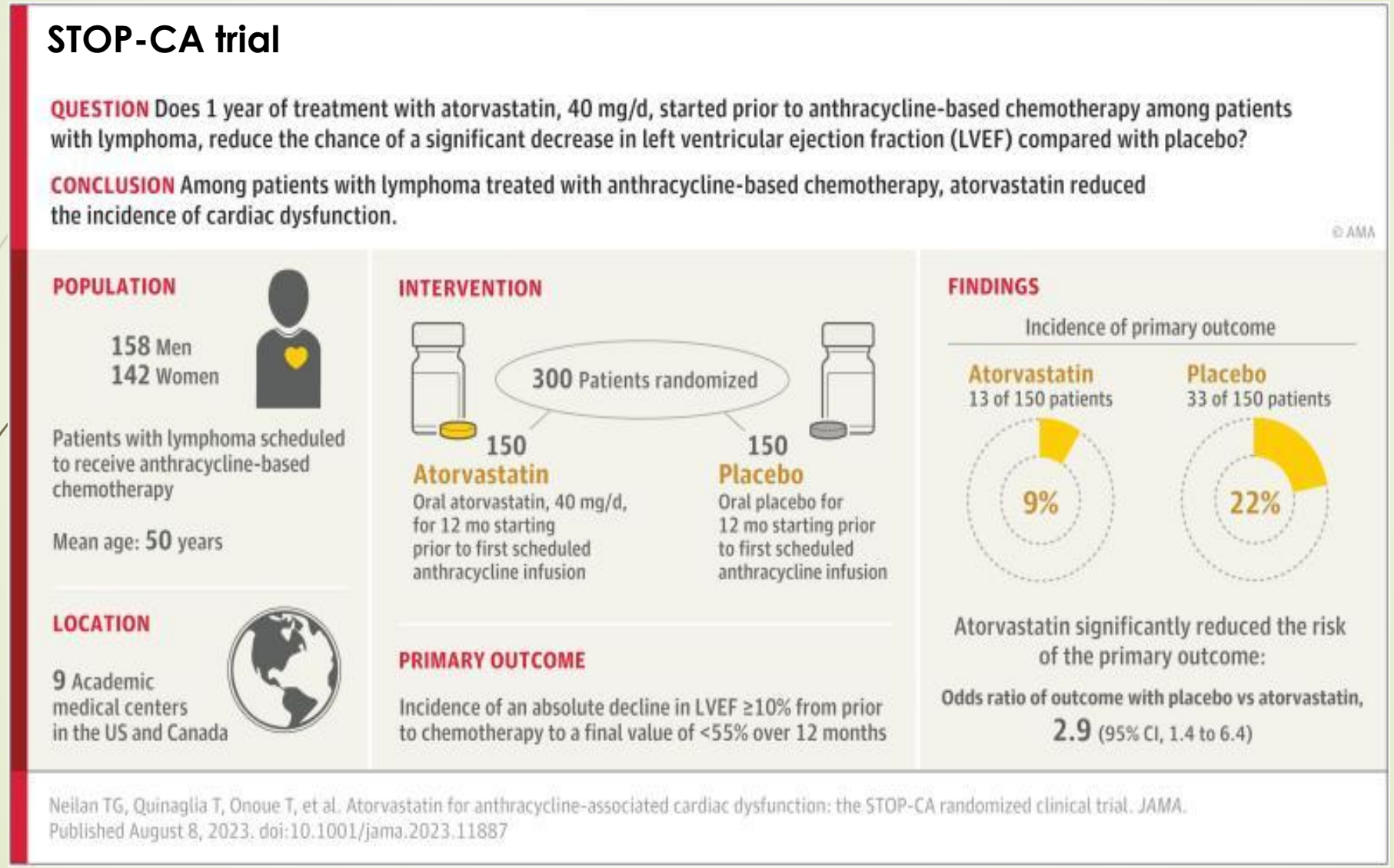


Fig. 1 Decrease of NT-proBNP levels and improvement of LV-ejection fraction after treatment with sacubitril/valsartan ($n = 21$). *Abbreviations*: NT-proBNP N-terminal pro B-type natriuretic peptide, LV-EF left ventricular ejection fraction

N=21
Stage C szívelégtelenség
Korábban daganatos betegség miatti kezelés – kemotherápia (53%) sugárkezelés (43%)
Medián utánkövetés 12 hónap

Cardio oncology. 2021 Oct 13;7(1):35.

Sztatinok



Neilan TG, Quinaglia T, Onoue T, et al. Atorvastatin for anthracycline-associated cardiac dysfunction: the STOP-CA randomized clinical trial. *JAMA*. Published August 8, 2023. doi:10.1001/jama.2023.11887

SGLT2-gátlók

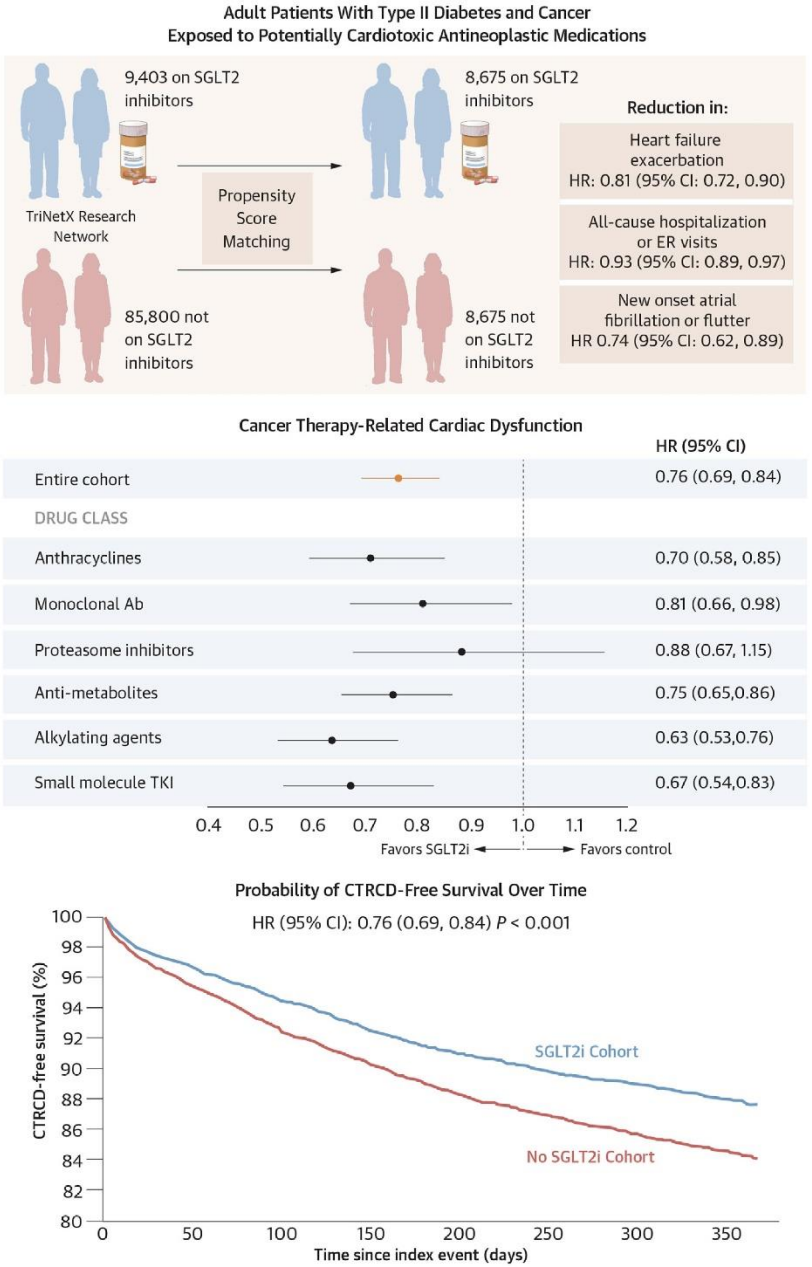
TABLE 1 Clinical Studies Supporting the Use of SGLT2 Inhibitors in Cardio-Oncology

First Author (Year)	Study Type	Population	Study Groups	Treatments	Key Findings
Gongora et al (2022)[16]	Observational retrospective cohort study	Patients with DM and cancer treated with anthracyclines, aged >18 y, and patients with prior HF	Cases (n = 32): patients with DM and cancer on SGLT2 inhibitors during anthracycline treatment Control (n = 96): patients with DM cancer on anthracycline treatment without SGLT2 inhibitors	CANA (34% [n = 11]), DAPA (16% [n = 5]), EMPA (50% [n = 16])	Patients on SGLT2 inhibitors had ↓ cardiac events after anthracycline therapy, including ↓ HF admissions and a ↓ rate of cardiac dysfunction No new cases of anthracycline-induced cardiac dysfunction were observed in patients taking SGLT2 inhibitors
Abdel-Qadir et al (2023)[18]	Observational population-based cohort study using medical records data sets	Patients aged ≥65 y with treated diabetes, no prior HF, receiving anthracycline-based chemotherapy for cancer	SGLT2 inhibitor–treated patients (n = 99) SGLT2 inhibitor–unexposed patients (n = 834)	CANA, DAPA, EMPA	SGLT2 inhibitor exposure ↓ risk of HF hospitalization, but no significant difference in incident HF diagnosis SGLT2 inhibitor use was associated with a statistically nonsignificant ↓ rate of mortality
Avula et al (2024)[38]	Retrospective cohort analysis of deidentified, aggregated patient data	Patients aged ≥18 y with histories of T2DM, cancer, and exposure to potentially cardiotoxic antineoplastic therapies, with subsequent diagnoses of cardiomyopathy or HF	Patients on SGLT2 inhibitors (n = 640) SGLT2 inhibitor–unexposed patients (n = 640), after propensity score matching	CANA, DAPA, EMPA	Patients on SGLT2 inhibitors had ↓ risk of acute HF exacerbation and all-cause mortality Less frequent all-cause hospitalizations or emergency department visits, atrial fibrillation/flutter, acute kidney injury, and need for renal replacement therapy in patients on SGLT2 inhibitors

CANA — canagliflozin; DAPA — dapagliflozin; DM — diabetes mellitus; EMPA — empagliflozin; HF — heart failure; SGLT2 — sodium-glucose cotransporter-2; T2DM — type 2 diabetes mellitus; ↑ — increase; ↓ — decrease.

Dabour MS, et al. J Am Coll Cardiol CardioOnc. 2024;6(2):159-182.

CENTRAL ILLUSTRATION: Sodium Glucose Co-Transporter 2 Inhibitors in the Prevention of Cancer Therapy-Related Cardiac Dysfunction



Bhatti AW, et al. JACC CardioOncol. 2024;10.1016/j.jaccao.2024.08.001

Folyamatban lévő kardio-onkológiai vizsgálatok

Trial	Trial Number	Cancer	Cancer Therapy	Trial Intervention	Masking/ Design	N	Outcome Measures
Pharmacologic intervention: neurohormonal blockade							
PRADA II (Prevention of Cardiac Dysfunction During Breast Cancer Therapy)	NCT03760588	Breast cancer	Anthracyclines with/ without trastuzumab/ pertuzumab	Sacubitril-valsartan/ placebo	Blinded	214	Change in LVEF assessed by CMR from baseline to 18 mo
Carvedilol in Preventing Cardiac Toxicity in Patients With Metastatic HER-2-Positive Breast Cancer	NCT03418961	Metastatic HER2-positive breast cancer	HER2-targeted therapy without concurrent anthracyclines	Carvedilol/no study intervention/ observation in patients with increased risk for cardiotoxicity	Single-blinded (outcomes assessor)	817	Time to the first identification of cardiac dysfunction assessed by echocardiography
PROACT (Can We Prevent Chemotherapy-Related Heart Damage in Patients With Breast Cancer and Lymphoma?)	NCT03265574	Breast cancer/ lymphoma	Epirubicin	Enalapril/usual care	Single-blinded (outcomes assessor)	170	Cardiac troponin T release during anthracycline treatment (1 mo after last dose of anthracycline)
Effect of Angiotensin Converting Enzyme and Sacubitril Valsartan in Patients After Bone Marrow Transplantation	NCT04092309	Hematological malignancies	Hematopoietic cell transplantation	ACE inhibitor/ sacubitril-valsartan/ control	Open	90	LVEF by 3D echocardiography/ GLS/PWV/ glycocalyx thickness
CardioTox (Effects of Carvedilol on Cardiotoxicity in Cancer Patients Submitted to Anthracycline Therapy)	NCT04939883	Cancer patients submitted to anthracycline therapy	Anthracyclines	Carvedilol/placebo	Blinded	1,018	Decline in ejection fraction within 12 mo of starting treatment (>10% to values <50%)/ cardiac events
Carvedilol in Preventing Heart Failure in Childhood Cancer Survivors	NCT02717507	Childhood cancer survivors	Anthracyclines	2-y course of low-dose carvedilol/ placebo	Blinded	182	LV posterior wall thickness, LV systolic and diastolic function, and afterload; natriuretic peptides, troponins, and galectin-3
Pharmacological interventions: statins							
PREVENT (Preventing Anthracycline Cardiovascular Toxicity With Statins)	NCT01988571	Breast cancer/ lymphoma	Anthracyclines	Atorvastatin/placebo	Blinded	279	Change in LVEF by CMR from baseline to 24 mo
STOP-CA (Statins to Prevent the Cardiotoxicity From Anthracyclines)	NCT02943590	Lymphoma	Anthracyclines	Atorvastatin/placebo	Blinded	300	Change in LVEF from baseline to 12 mo assessed by CMR
SPARE-HF (Statins for the Primary Prevention of Heart Failure in Patients Receiving Anthracycline Pilot Study)	NCT03186404	Cancer patients with high CVD risk	Anthracyclines	Atorvastatin/placebo	Blinded	112	Change in LVEF assessed by CMR from baseline to within 4 wk of anthracycline completion
Pharmacological interventions: other							
IPAC (Ivabradine to Prevent Anthracycline-Induced Cardiotoxicity)	NCT03650205	Cancer diagnosis	Anthracyclines	Ivabradine/placebo	Blinded	160	Reduction in GLS of ≥10% from baseline to 12 mo
IPAC (Ivabradine to Prevent Anthracycline-Induced Cardiotoxicity)	NCT04030546	Cancer diagnosis	Anthracyclines	Ivabradine/usual care	Single-blinded (outcomes assessor)	128	Change in GLS at 1, 3, and 6 mo of ≥3%
TRIMETA	EudraCT: 2016-002270-12	HER2-positive breast cancer	Anthracyclines, taxanes, and trastuzumab	Trimetazidine/control	Open	242	Absolute and relative frequency of cardiotoxicity (24 mo) assessed by echocardiography/ CREC criteria
Effect of Trimetazidine on Radiotherapy-Induced Heart Damage	NCT04939857	Lung cancer	Stereotactic radiotherapy	Trimetazidine/control	Single-blinded (outcomes assessor)	80	GLS by echocardiography from baseline to 12 mo
Protective Effects of the Nutritional Supplement Sulforaphane on Doxorubicin-Associated Cardiac Dysfunction	NCT03934905	Breast cancer	Doxorubicin	Sulforaphane/ placebo	Blinded	70	Change in cardiac function by 2D echocardiography from baseline to 12 mo

TABLE 3 Continued

Trial	Trial Number	Cancer	Cancer Therapy	Trial Intervention	Masking/ Design	N	Primary Outcome Measures
Exercise							
ATOPE (Attenuating Cancer Treatment-Related Toxicity in Oncology Patients With a Tailored Physical Exercise Program)	NCT03787966	Breast cancer	Surgery, chemotherapy, and radiotherapy	Therapeutic exercise before vs after medical treatment	Single-blinded (outcomes assessor)	110	Change in LVEF by echocardiography from baseline to 12 mo
CAPRICE (Cancer Adverse Effects Prevention With Care & Exercise)	NCT03850171	Breast cancer/ lymphoma	Anthracyclines	Exercise training/ usual care	Single-blinded (outcomes assessor)	120	Changes in GLS from baseline to 13 wk
ONCORE (Exercise-Based Cardiac Rehabilitation for the Prevention of Breast Cancer Chemotherapy-Induced Cardiotoxicity)	NCT03964142	Breast cancer	Anthracyclines and/or anti-HER2 antibodies	Cardiac rehabilitation program/usual care	Open	122	Change in LVEF and GLS by transthoracic echocardiography during and every year after study completion up to a maximum of 5 y
EXACT2 (Exercise to Prevent Anthracycline-Based Cardio-Toxicity Study 2.0)	NCT03748550	Breast cancer	Anthracyclines	Aerobic exercise/ standard care	Single-blinded (outcomes assessor)	100	Change in LVEF from baseline, postintervention (week 13) and 6 mo
Choice of therapy							
RadComp (Pragmatic Randomized Trial of Proton vs Photon Therapy for Patients With Non-Metastatic Breast Cancer: A Radiotherapy Comparative Effectiveness)	NCT02603341	Breast cancer	Radiotherapy	Proton or photon	Open	1,278	Major cardiovascular events at 10 y
RadComp ancillary	NCT04361240	Patients with breast cancer enrolling in the RadComp trial	Radiotherapy	Proton or photon	Open	155	Change in LVEF and RV FAC assessed by echocardiography and NT-proBNP, PIGF, and GDF-15 from baseline to 14 mo
The DBCG Proton Trial: Photon Versus Proton Radiation Therapy for Early Breast Cancer	NCT04291378	Early breast cancer	Radiotherapy	Proton or photon	Open	1,502	Radiation-associated ischemic and valvular heart disease (10 y)
Remote ischemic preconditioning							
ERIC-ONC (Effect of Remote Ischemic Conditioning in Oncology Patients)	NCT02471885	Cancer diagnosis	Anthracyclines	Remote ischemic preconditioning/ placebo (sham)	Blinded	128	High-sensitivity troponin T AUC before and after each chemotherapy cycle and at 1-, 3-, 6-, and 12-mo follow-up

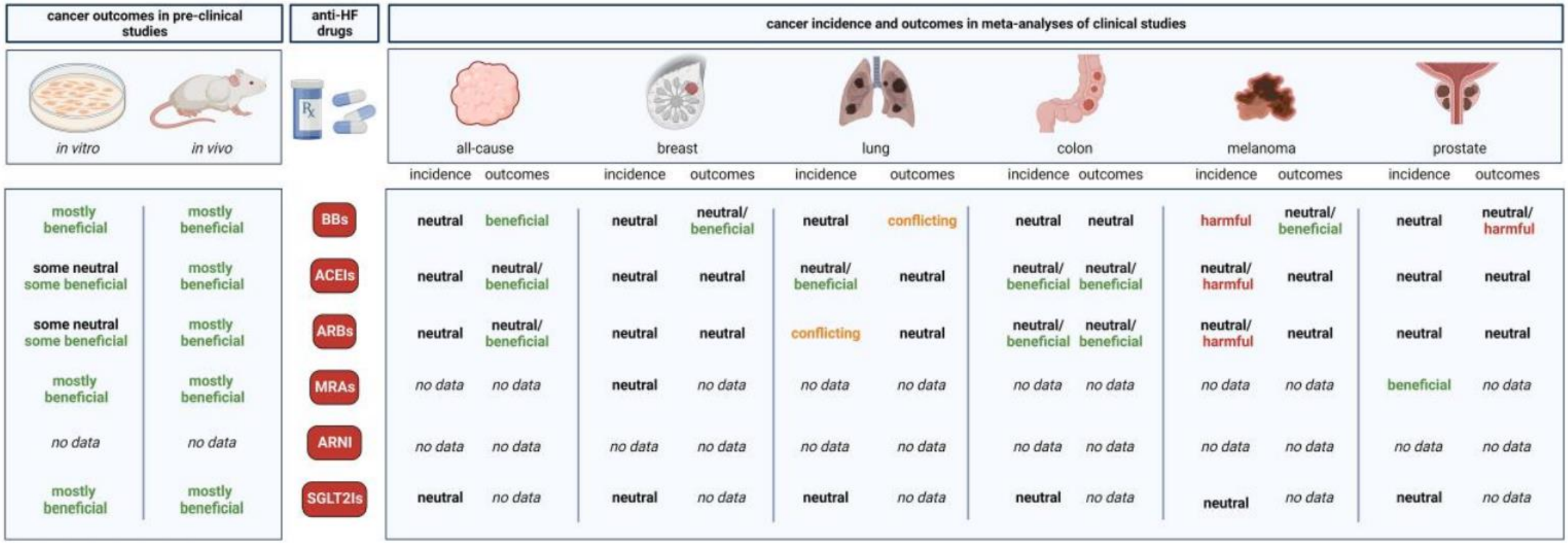


Figure 5 A graphical summary for both the pre-clinical and clinical evidence on the effect of different heart failure pharmacotherapies on cancer. The terms were defined as follows: beneficial: decreases cancer incidence, or improves any patient outcome; neutral: no effect on cancer incidence, or no effect on any patient outcome; harmful: increased incidence or worsening of any patient outcome; conflicting: there are studies showing either benefit or harm on cancer incidence or outcomes. Figure created with BioRender.com.

Vaszkuláris betegségek

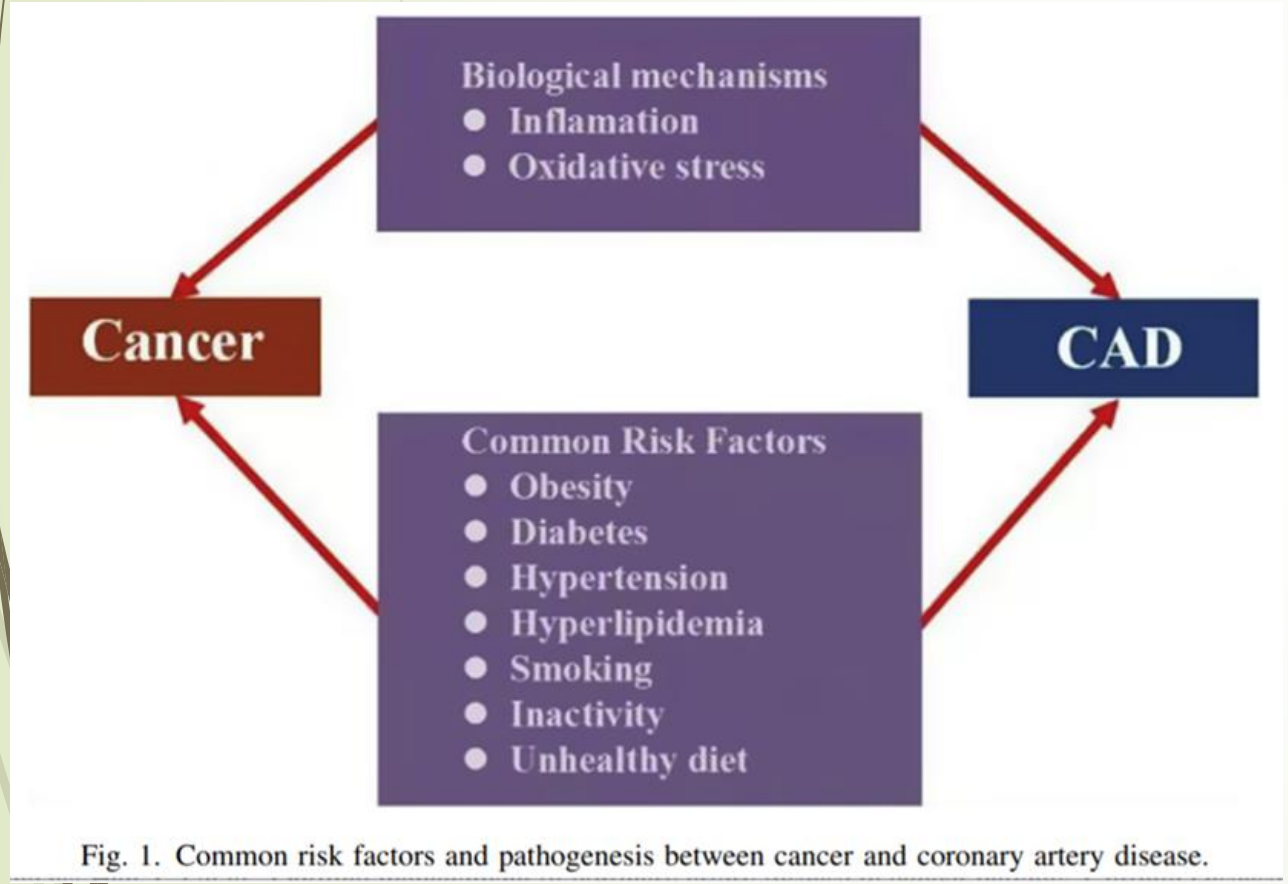
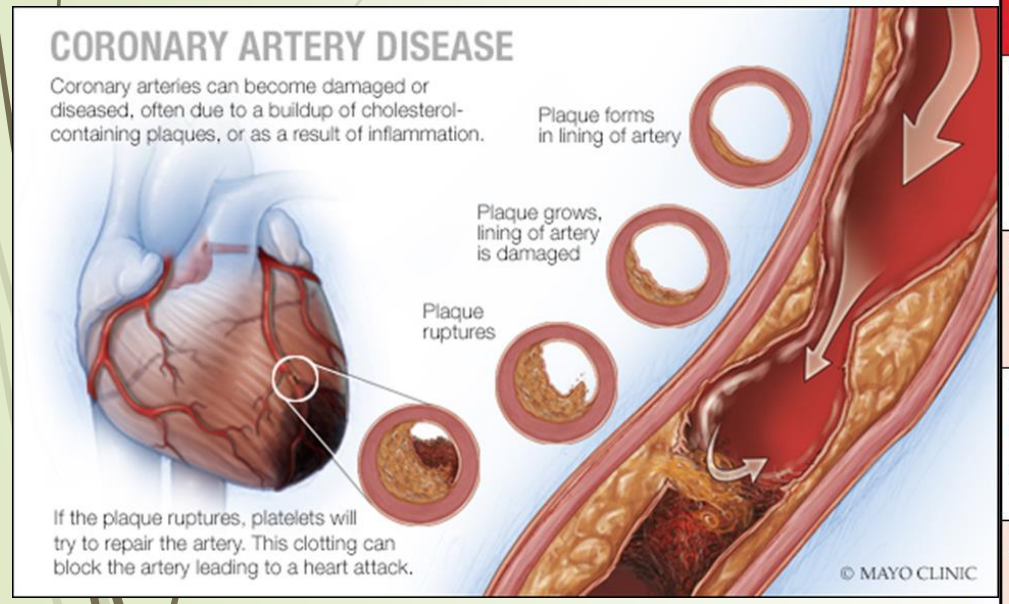


Fig. 1. Common risk factors and pathogenesis between cancer and coronary artery disease.

– **Onkotherápiás kezelések érkárosító hatásai:**
  - endothel sérülés
  - direkt vazospasztikus hatás
  - felgyorsult atherosclerosis
    – lipid metabolizmus változása
    – ROS
  - akut artériás thrombosis

Onkotherápiás kezelés hatásai a koszorúér betegség kialakulására



Pathophysiological mechanisms of coronary artery disease in cancer treatment

Agent	Pathophysiological mechanism	Risk of coronary artery disease and acute coronary syndrome
Fluoropyrimidines (5-FU, capecitabine, gemcitabine)	• Endothelial injury • Vasospasm	• Up to 18% manifest myocardial ischaemia • Up to 7–10%: silent myocardial ischaemia
Platinum compounds (cisplatin)	• Procoagulant status • Arterial thrombosis	• 20-year absolute risk of up to 8% after testicular cancer • 2% risk of arterial thrombosis
VEGF inhibitors (bevacizumab, sorafenib, sunitinib)	• Procoagulant status • Arterial thrombosis • Endothelial injury	• Risk of arterial thrombosis: bevacizumab 3.8%, sorafenib 1.7%, sunitinib 1.4%
Radiotherapy	• Endothelial injury • Plaque rupture • Thrombosis	• 2–7-fold increased relative risk of myocardial infarction • Cumulative 30-year coronary events incidence of 10% in Hogdkin lymphoma survivors • Risk proportional to irradiation dose

European Heart Journal doi:10.1093/eurheartj/ehw211 - EHJ 2016;37:2768-2801

Aritmiák

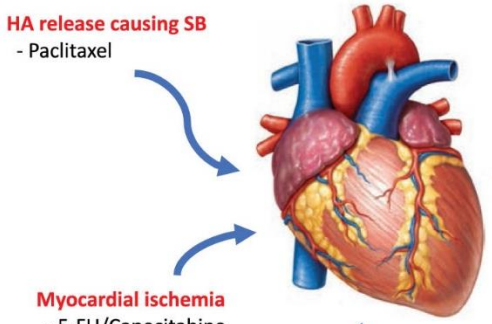
Cardiac arrhythmias	
QT prolongation	Prolonged: QTcF > 500 ms[j]
Bradycardia	For general cardiology definitions, see Supplementary data, *Table S1*
Supraventricular tachycardia	
Ventricular arrhythmias	
AF	

Non-chemotherapeutic factors

1. Prior underlying substrate for arrhythmias
2. Post cancer surgery arrhythmias
3. Radiation induced pericarditis/atherosclerosis
4. Adjuvant medications – e.g. Antiemetics

Direct cardiac involvement

1. Primary cancer
2. Metastasis to heart
3. Cardiac amyloidosis



Electrolyte disturbances

1. Vomiting
2. Diarrhea from colitis
3. Drug induced imbalance (amsacrine, cetuximab, cisplatin and necitumumab)

Effects on cardiac myocytes

1. hERG blockade - ATO, PKI
2. Abnormal calcium homeostasis – Taxanes, ATO, AC
3. Mitochondrial injury - Sunitinib, AC
4. Cardiac apoptosis – Sorafenib, antimetabolites, AC
5. Inhibition of PI3K - PKI

Lyon A.R. et al. Eur Heart J. 2022 Nov 1;43(41):4229-4361.; Circulation: Arrhythmia and Electrophysiology. 2017;10:e005443

Kemoterápia okozta ritmuszavar

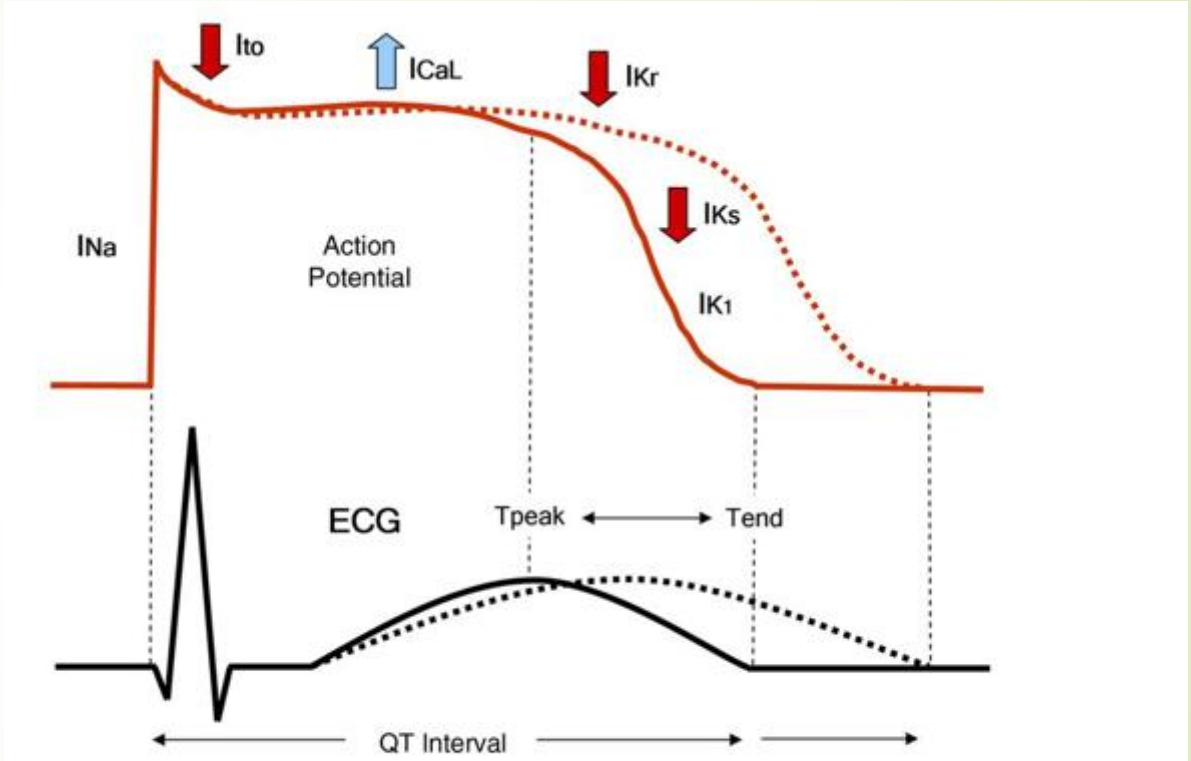
Ritmuszavar típusa	Kiváltó gyógyszer
Bradycardia	Arzén-trioxid, bortezomib, capecitabin, ciszplatin, ciklofoszfamid, doxorubicin, epirubicin, 5-FU, ifosfamid, IL-2, metotrexát, mitoxantron, paclitaxel, rituximab, talidomid.
Sinus tachycardia	Antracyclinek, carmustin.
Atrioventrikuláris blokk	Antracyclinek, arzén-trioxid, bortezomib, ciklofoszfamid, 5-FU, mitoxantron, rituximab, taxánok, thalidomid.
Vezetési zavarok	Antracyclinek, ciszplatin, 5-FU, imatinib, taxánok.
Pitvarfibrilláció	Alkiláló szerek (ciszplatin, ciklofoszfamid, ifoszfamid, melfalán), antracyclinek, antimetabolitok (capecitabin, 5-FU, gemcitabin), IL-2, interferonok, rituximab, romidepszin, kis molekulájú TKI-k (ponatinib, sorafenib, sunitinib, ibrutinib), topoizomeráz-II gátlók (amsacrin, etopozid), taxánok, vinka alkaloidok.
Szupravent-rikuláris tachycardiák	Alkiláló szerek (ciszplatin, ciklofoszfamid, ifoszfamid, melfalán), amsacrin, antracyclinek, antimetabolitok (capecitabin, 5-FU, metotrexát), bortezomib, doxorubicin, IL-2, interferonok, paclitaxel, ponatinib, romidepszin.
Kamrai tachycardia, kamrafibrilláció	Alkiláló szerek (ciszplatin, ciklofoszfamid, ifoszfamid), amsacrin, antimetabolitok (capecitabin, 5-FU, gemcitabin), arzén-trioxid, doxorubicin, interferonok, IL-2, metotrexát, paclitaxel, proteaszóma-gátlók (bortezomib, carfilzomib), rituximab, romidepszin.
Hirtelen szívhalál	Antracyclinek (nyagyon ritkán), arzén-trioxid (torsade de pointes következtében), 5-FU (valószínűleg iszkémia és koronáriaspazmus miatt), interferonok, nilotinib, romidepszin.

Előfordulás 16-36%

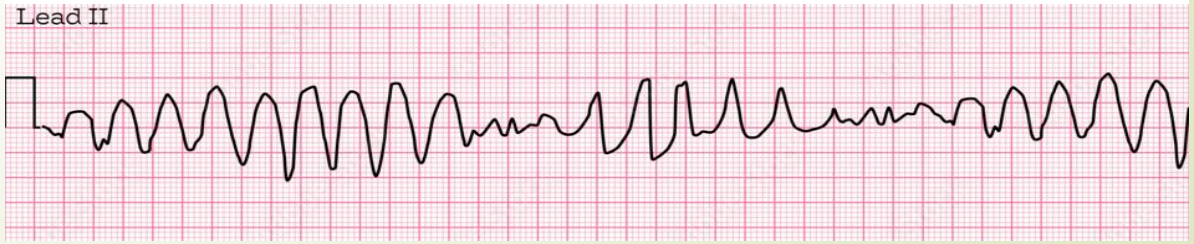
2016 ESC Position Paper on cancer treatments and cardiovascular toxicity developed under theauspices of the ESC Committee for Practice Guidelines, The Task Force for cancer treatments and cardiovascular toxicity of the European Society of Cardiology (ESC)

QT megnyúlás

- **QT idő:** a kamrai depolarizáció és repolarizáció ideje (gyakorlatilag a kamrai systole ideje az izovolumetrikus kontrakciótól a relaxációig)
- **A QT idő változik a szívfrekvencia függvényében**, magasabb frekvenciánál rövidül, az alacsonyabbnál hosszabbodik.
- normál QT távolság >350 és <450 ms felnőtt férfiakban, >360 and <460 ms felnőtt nők esetén
- **QT időt befolyásoló tényezők**:
  - Hypokalaemia
  - Hypomagnesaemia
  - Hypocalcaemia
  - Hypothermia
  - Miokardiális iszkémia
  - Emelkedett intrakraniális nyomás
  - Kongenitális hosszú QT szindróma
  - Gyógyszerek



Pietro Enea Lazzerini et al. Lupus Sci Med 2016;3:e000189



QTcF megnyúlás - onkológiában

Table 8 Risk factors for drug-induced QT prolongation and torsade de pointes

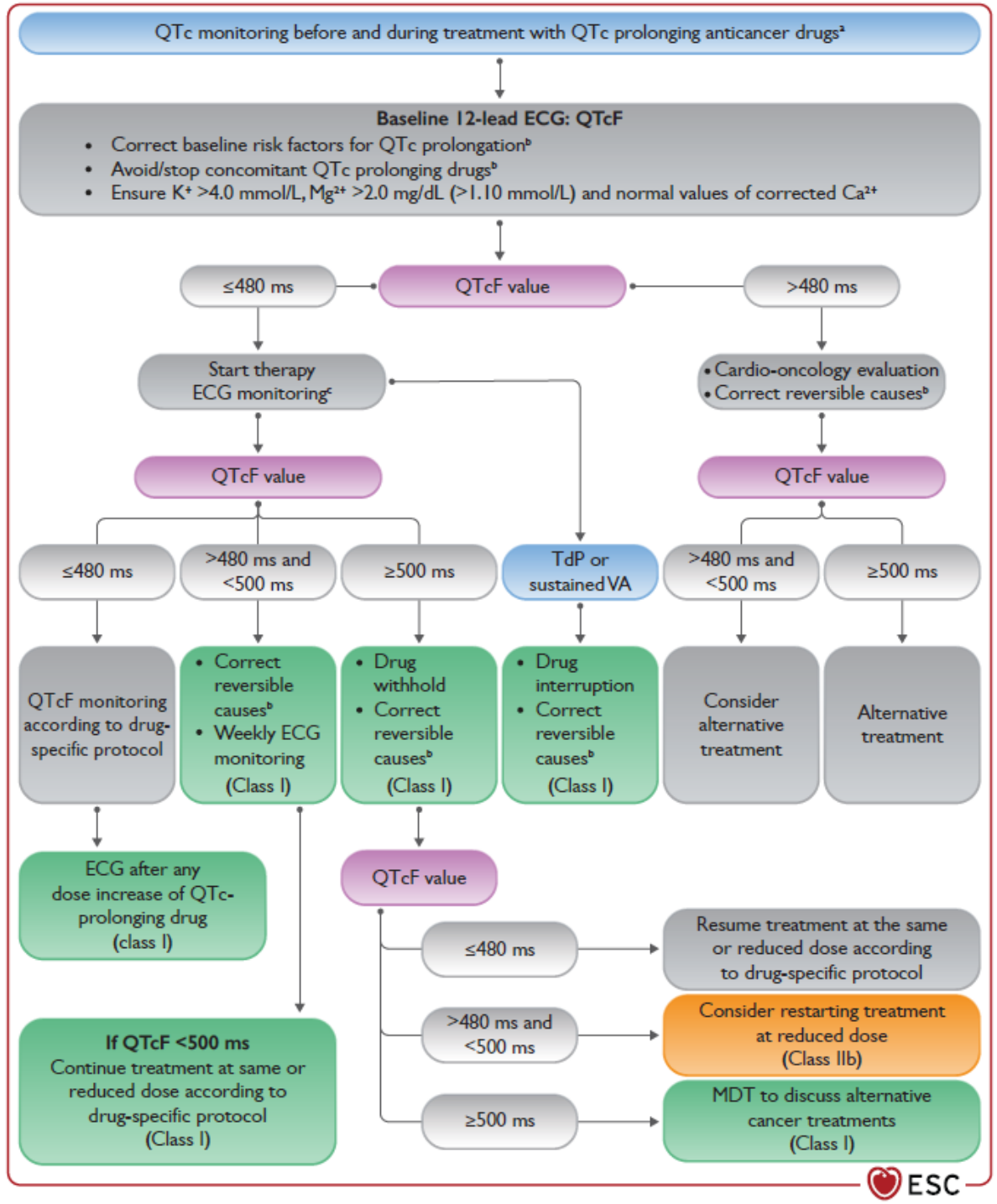
Correctable	Non-correctable
QT-prolonging drugs[a] • Antiarrhythmics • Antibiotics • Antidepressants • Antifungals • Antiemetics • Antihistamines • Antipsychotics • Loop diuretics • Opioids (methadone) Bradyarrhythmia Electrolyte imbalance/ abnormalities • Hypokalaemia (≤3.5 mEq/L) • Hypomagnesaemia (≤1.6 mEq/L) • Hypocalcaemia (≤8.5 mEq/L) Inadequate dose adjustment of renal or hepatic cleared QT-prolonging drugs	Acute myocardial ischaemia Age > 65 years Baseline QTc interval prolongation[b] Family history of sudden death (congenital LQTS or genetic polymorphism) Female sex Impaired renal function (for renally excreted drugs) Liver disease (for hepatically excreted drugs) Personal history of syncope or drug-induced TdP Pre-existing CVD (CAD, HF, LV hypertrophy)

Table 9 Classification of corrected QT interval prolongation induced by cancer drug therapy

Classification	Drugs
High risk: QTcF prolongation ≥ 10 ms and risk of TdP	• Aclarubicin • Arsenic trioxide • Glasdegib • Nilotinib • Oxaliplatin • Pazopanib • Ribociclib • Sunitinib • Toremifene • Vandetanib

Lyon A.R. et al. Eur Heart J. 2022 Nov 1;43(41):4229-4361.

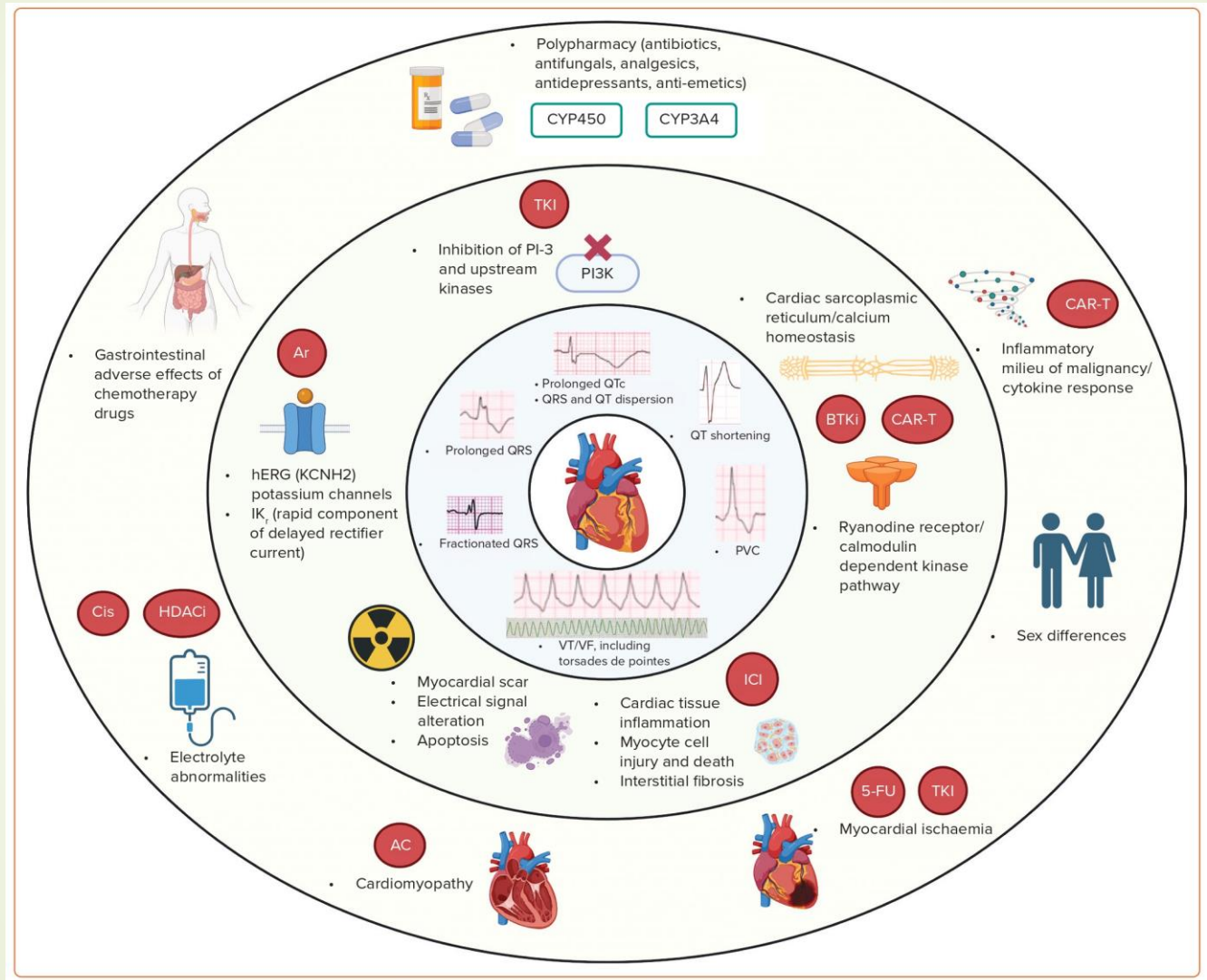
QT megnyúlás kezelése daganatellenes terápia mellett



- **Felismerés – EKG**
- **Egyéb befolyásoló tényezők ismerete (pl. elektrolitok), korrekciója**
- **Gyógyszerfelfüggesztés – indokolt esetben (>500 ms)**
- **EKG követés – újraindítás mérlegelése**
- **Fontos!!! – az egyes szerek egyéni tulajdonságainak mérlegelése-alkalmazási előirat!**
- **Multidiszciplináris team (pl. onkológus, kardiológus) döntés**

Lyon A.R. et al. Eur Heart J. 2022 Nov 1;43(41):4229-4361.

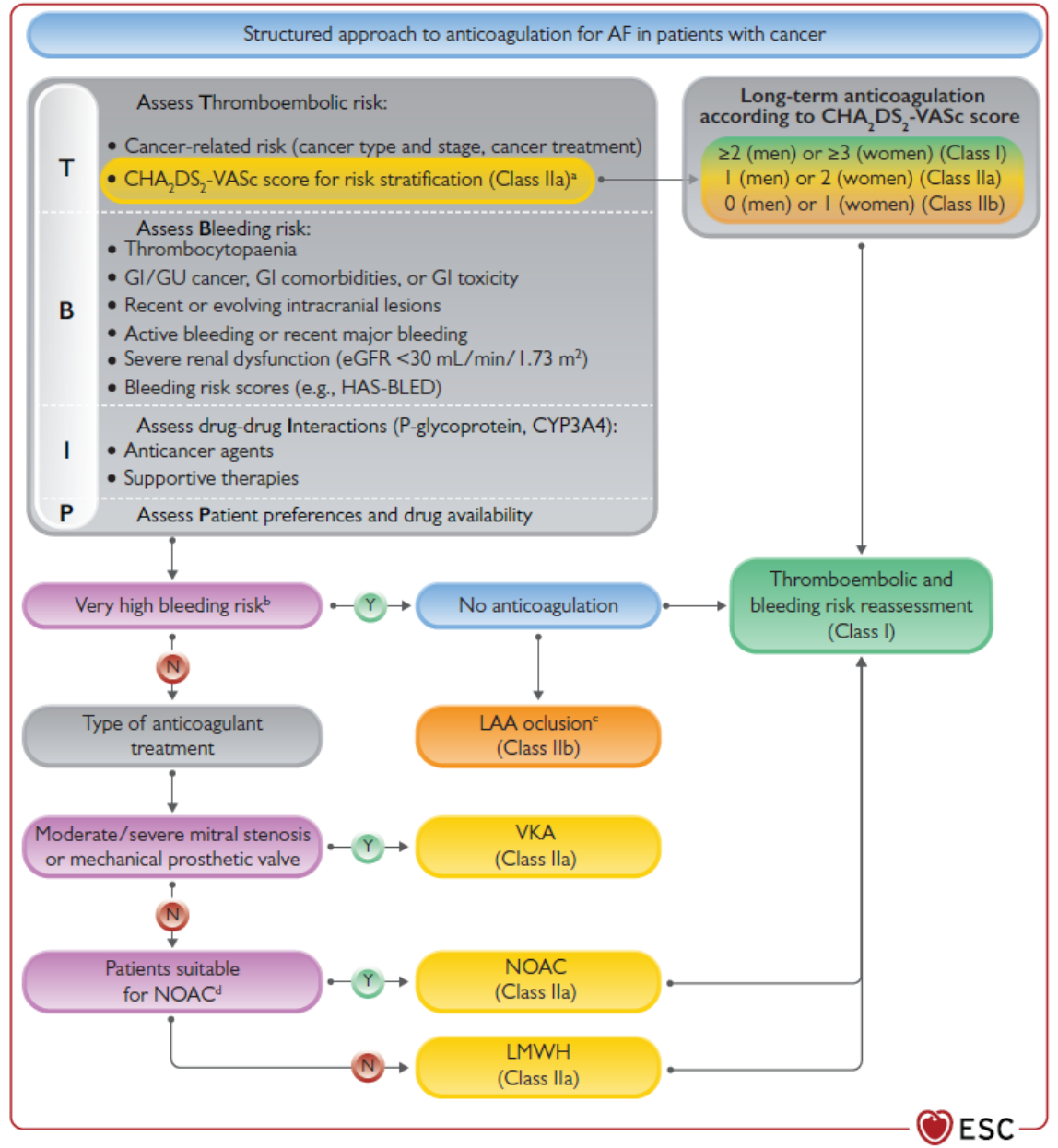
Malignus kamrai ritmuszavarok egyéb okai



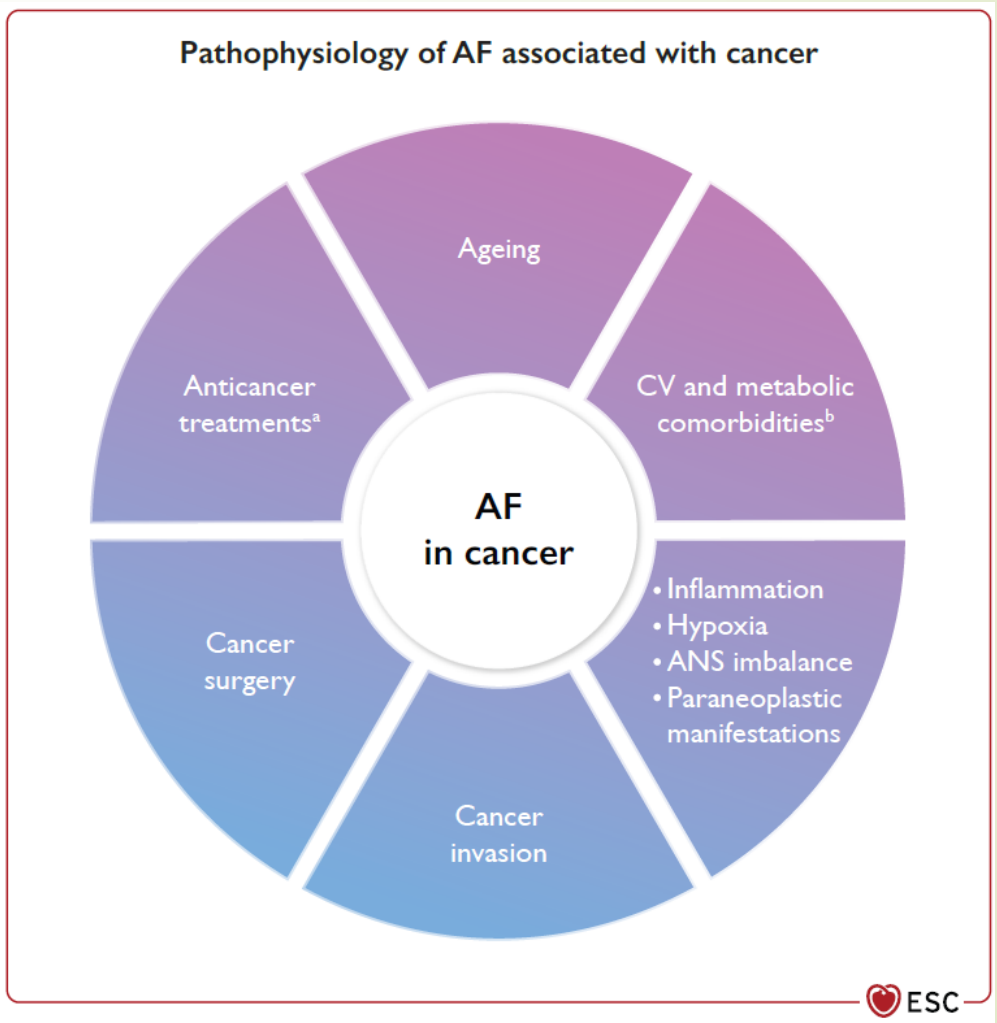
5-FU = 5-fluorouracil; AC = anthracyclines; Ar = arsenic trioxide; BTKi = Bruton tyrosine kinase inhibitor; CAR-T = chimeric antigen receptor T cell; Cis = cisplatin; CYP3A4 = cytochrome P450 3A4; CYP450 = cytochrome P450; HDACi = histone deacetylase inhibitor; hERG; human ether-a-go-go-related gene; ICI = immune checkpoint inhibitor; PI3K = phosphotidyl inositol 3–kinase; PVC = premature ventricular contractions; TKI = tyrosine kinase inhibitor; VT = ventricular tachycardia. Source: Created with BioRender.com. Reproduced with permission from BioRender agreement number BK24Y7V8FC.

Agarwal MA at e. Arrhythmia & Electrophysiology Review 2023;12:e16.

Pitvarfibrilláció

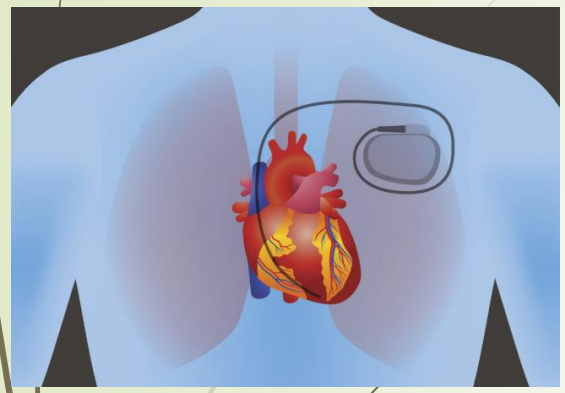


~ 2-5% az előfordulása, multifaktoriális

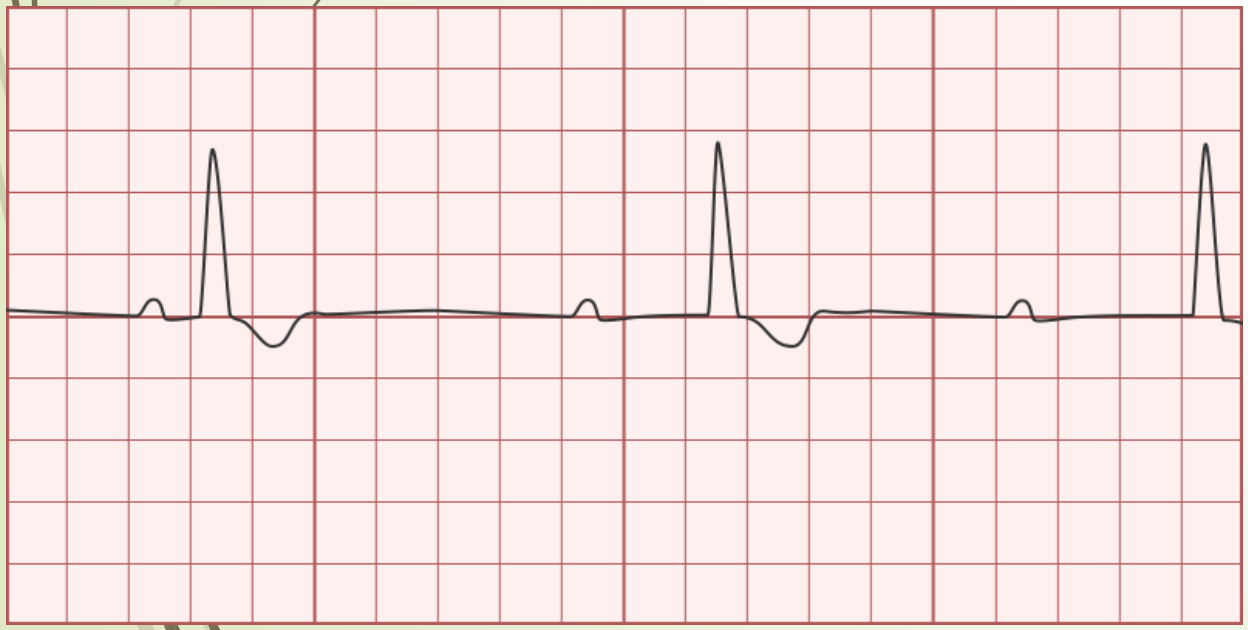


Lyon A.R. et al. Eur Heart J. 2022 Nov 1;43(41):4229-4361.

Sick sinus szindróma és vezetési zavarok



- AV csomó infiltrációja (lymphoma, amyloidosis)
- Vagus paraganglioma, katecholamint szekretáló tumorok, n. vagust involváló nyaki terime
- Cisplatin, irinotecan, **paclitaxel (29%)**, mitoxantrone, ritkán doxorubicin, octreotid**, thalidomide (26-53%)**,
- methotrexate, 5-fluorouracil, arzen-trioxid
- Mellkasi radioterápiát követően - SSS, AV blokk



European Heart Journal (2013) 34, 2281–2329.

Vénás thrombemboliák

- A 2. leggyakoribb halálok daganatos betegeknél
- A daganat 5x VTE rizikót jelent
- Az összes VTE eset 30%-a daganat-asszociált
- A nem provokált VTE lehet sok esetben a tumoros betegség első megjelenési formája, 5%-ban daganat igazolódik 12 hónapon belül

Recommendation Table 34 — Recommendations for the management of venous thromboembolism in patients receiving anticancer treatment

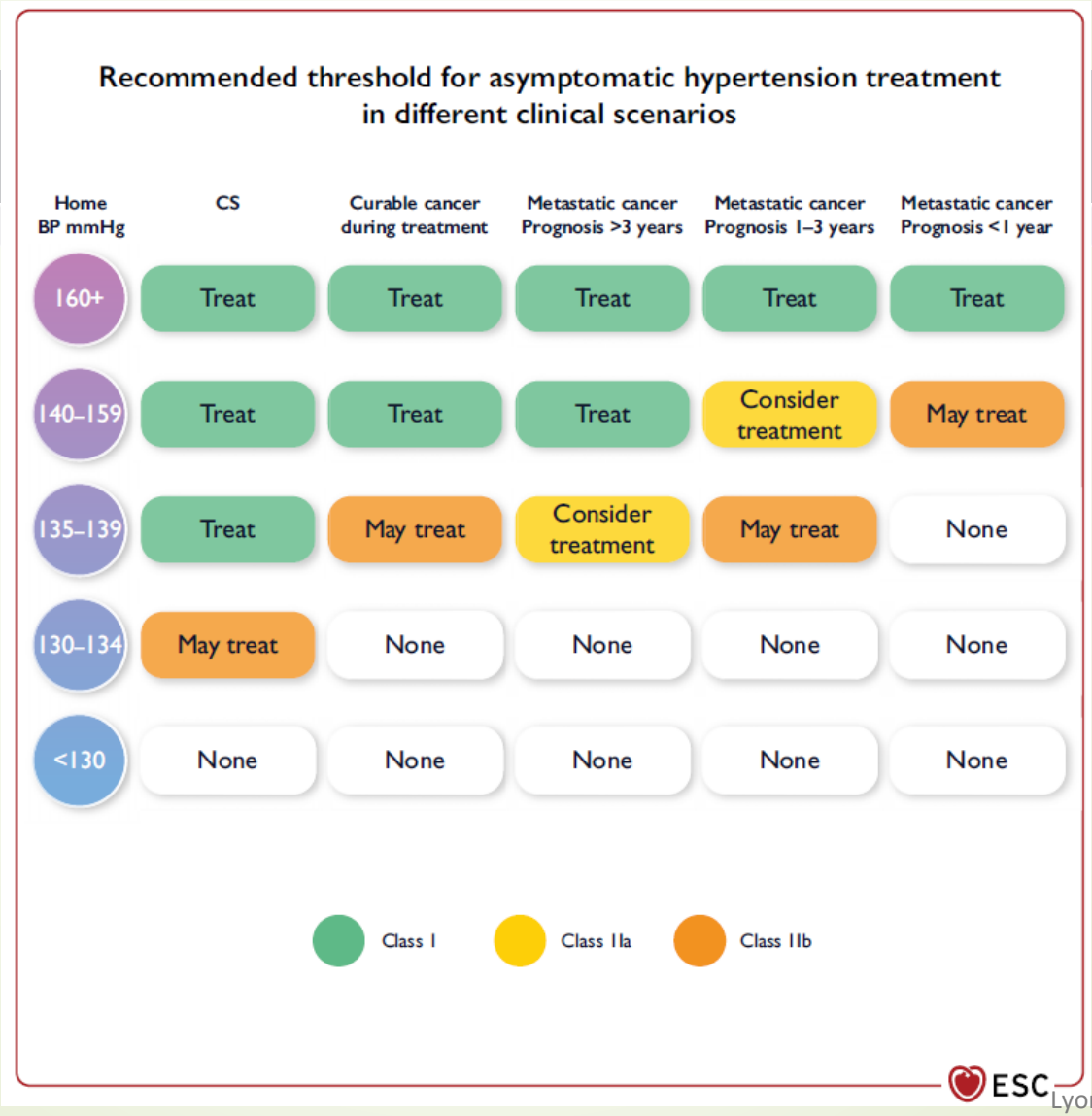
Recommendations	Class[a]	Level[b]
Apixaban, edoxaban, or rivaroxaban[c] are recommended for the treatment of symptomatic or incidental VTE in patients with cancer *without* contraindications.[d,578–581,584,585]	I	A
LMWH are recommended for the treatment of symptomatic or incidental VTE in patients with cancer with platelet count >50 000/μL.[298,299,578–581,584,585]	I	A
In patients with cancer with platelet counts of 25 000–50 000/μL, anticoagulation with half-dose LMWH may be considered after a multidisciplinary discussion.[591]	IIb	C
Prolongation of anticoagulation therapy beyond 6 months should be considered in selected patients with active cancer[e] including metastatic disease.[589,590]	IIa	A
Catheter-associated VTE		
Duration of anticoagulation in patients with cancer with a catheter-associated VTE is recommended for a minimum of 3 months and continuing longer if the catheter remains *in situ*.	I	C

ESC

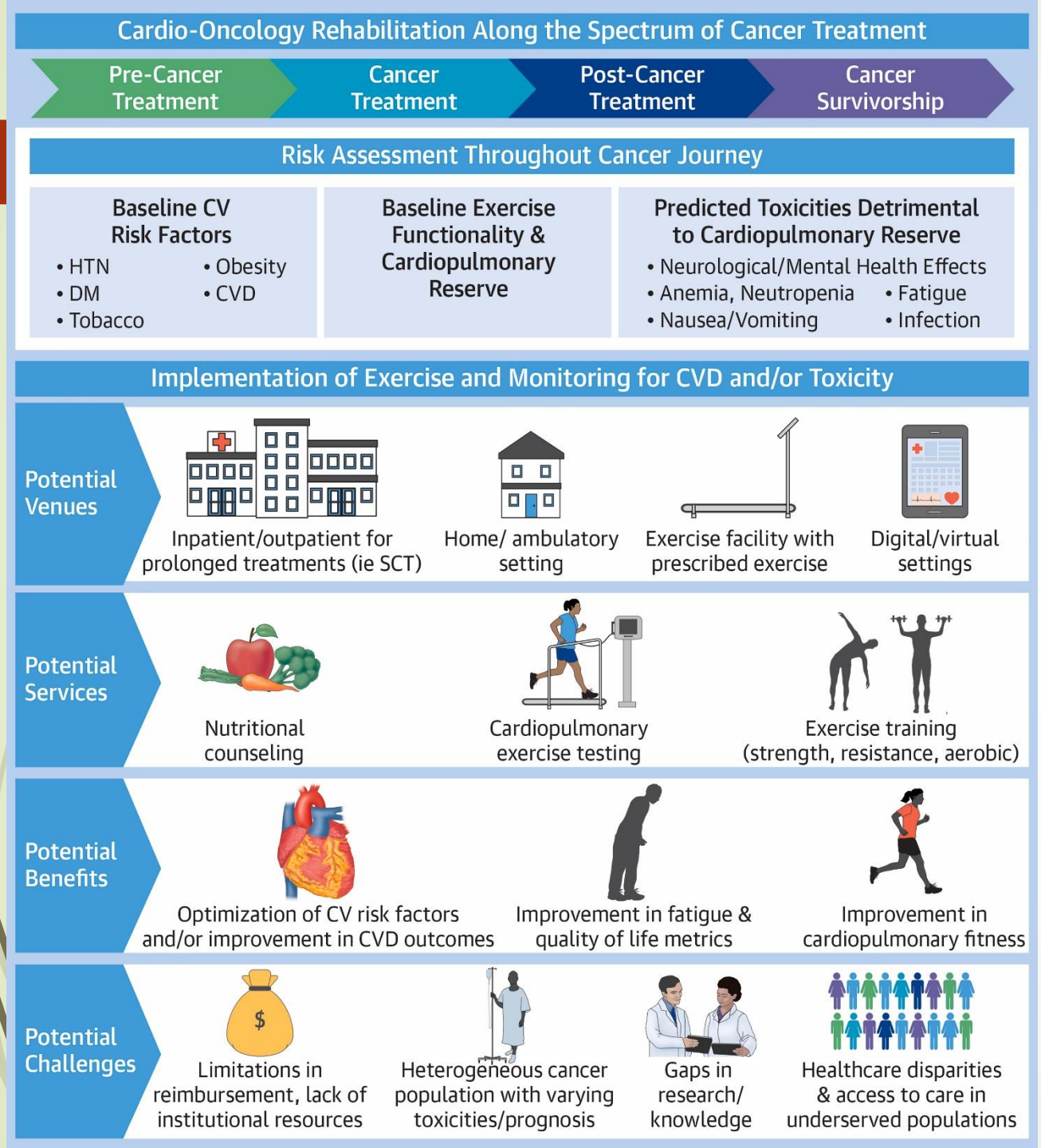
Lyon A.R. et al. Eur Heart J. 2022 Nov 1;43(41):4229-4361.

Hypertonia

Baseline CV toxicity risk factors	RAF and MEK inhibitors
Hypertension[c]	M2



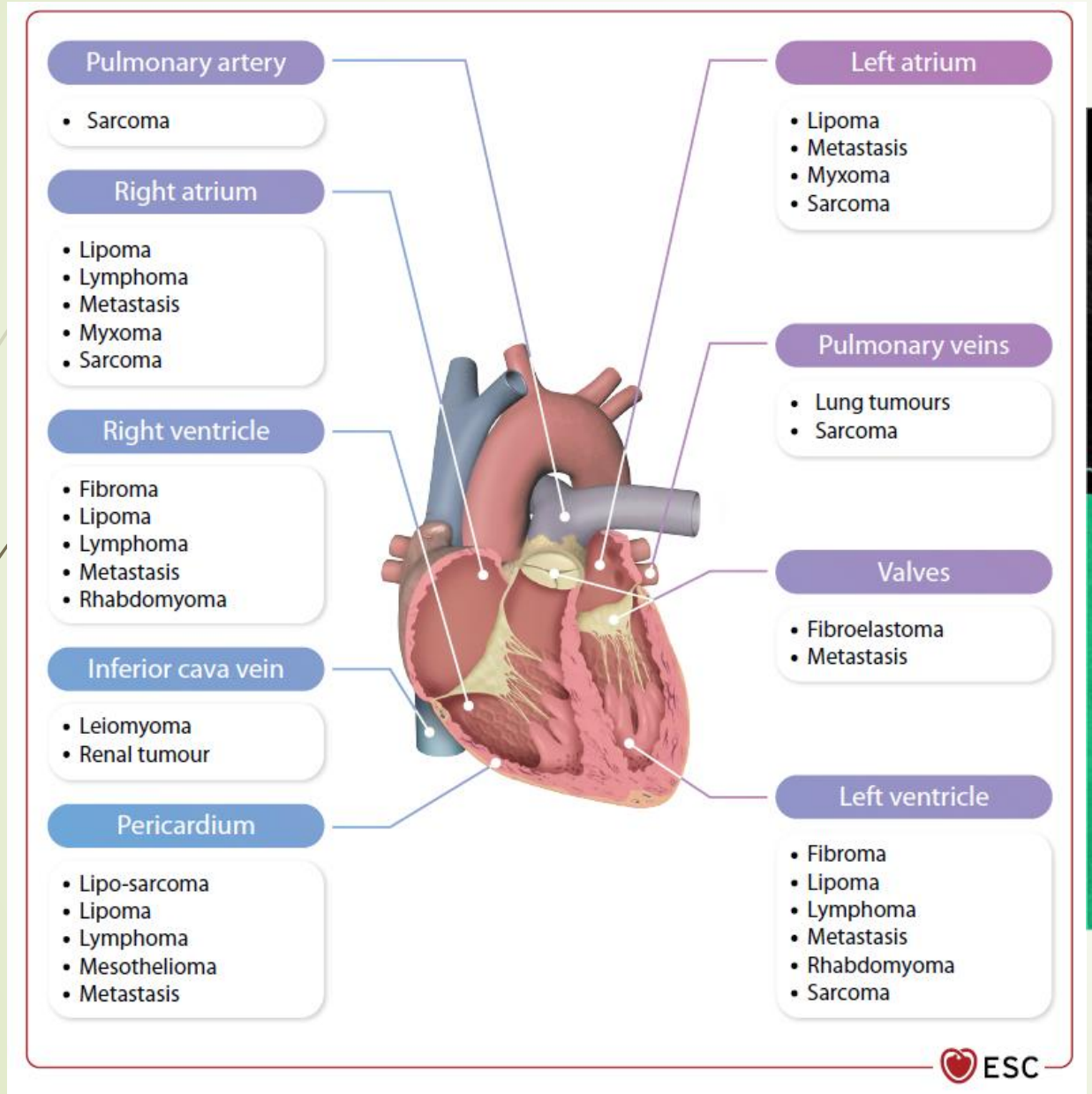
Lyon A.R. et al. Eur Heart J. 2022 Nov 1;43(41):4229-4361.

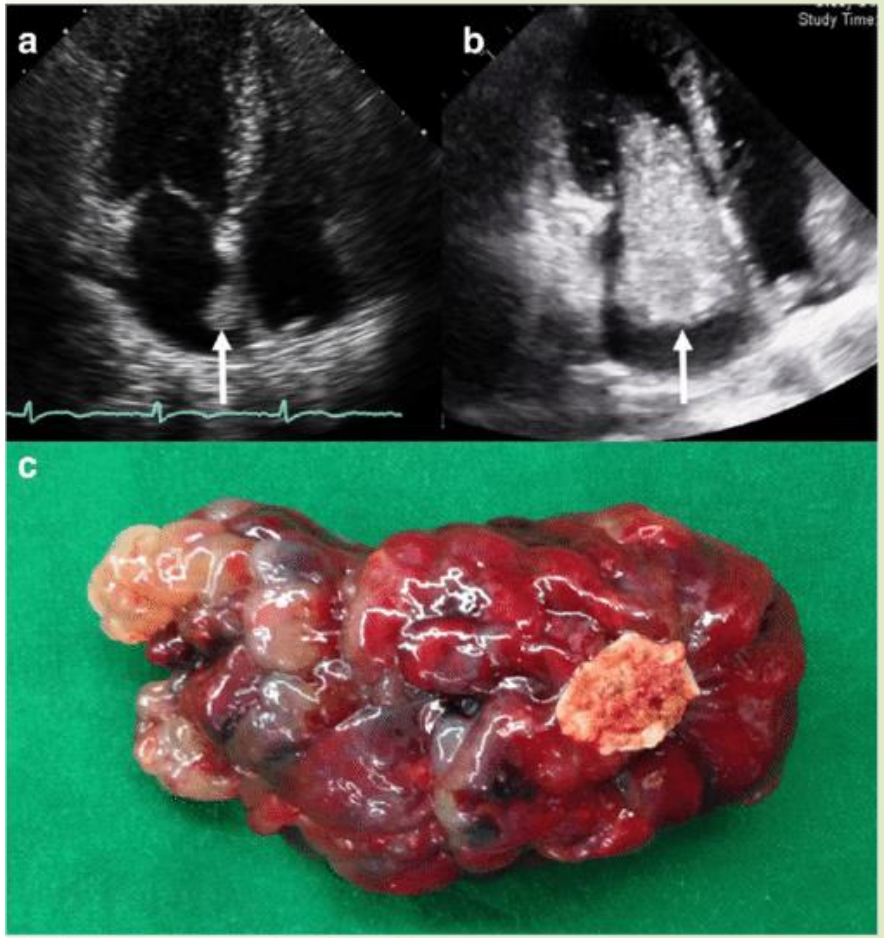


Rehabilitáció az onko-kardiológiában

Wilson RL et al . JACC CardioOncol. 2023 Oct 17;5(5):553-569.

Szívtumorok





Lyon A.R. et al. Eur Heart J. 2022 Nov 1;43(41):4229-4361.

Összefoglalás

- Az onko-kardiológia célja elsősorban primer és szekunder prevenció daganatellenes kezelés kapcsán
- kardiotoxicitás előrejelzése, felismerése, követése és a terápiás hatás lemérése → onkológiai kezelés biztonságossága javul, kimenetel (CV és onkológiai) javul
- Nagy, randomizált klinikai vizsgálat nem áll rendelkezésre, leginkább szakértői véleményre és kis betegszámú vizsgálatokra támaszkodunk mind a diagnosztika, mind a kezelés tekinetében
- Fontos a rizikóstratifikáció és a CV rizikó felmérése minden tumoros betegnél
- A diagnosztikus modalitásokat (EKG, biomarker, képalkotók, klinikum) együttesen kell értékelni
- Kardiotoxicitásnak más a dinamikája, megjelenése az egyes kezeléseknél
- Egyéb állapotokról sem szabad megfeledkezni (CCS, PAD, HT, VTE, aritmiák)
- Fontos a multidiszciplináris megközelítés (onkológus+kardiológus), team döntések meghozatala, risk-benefit értékelése

Köszönöm megtisztelő figyelmüket!