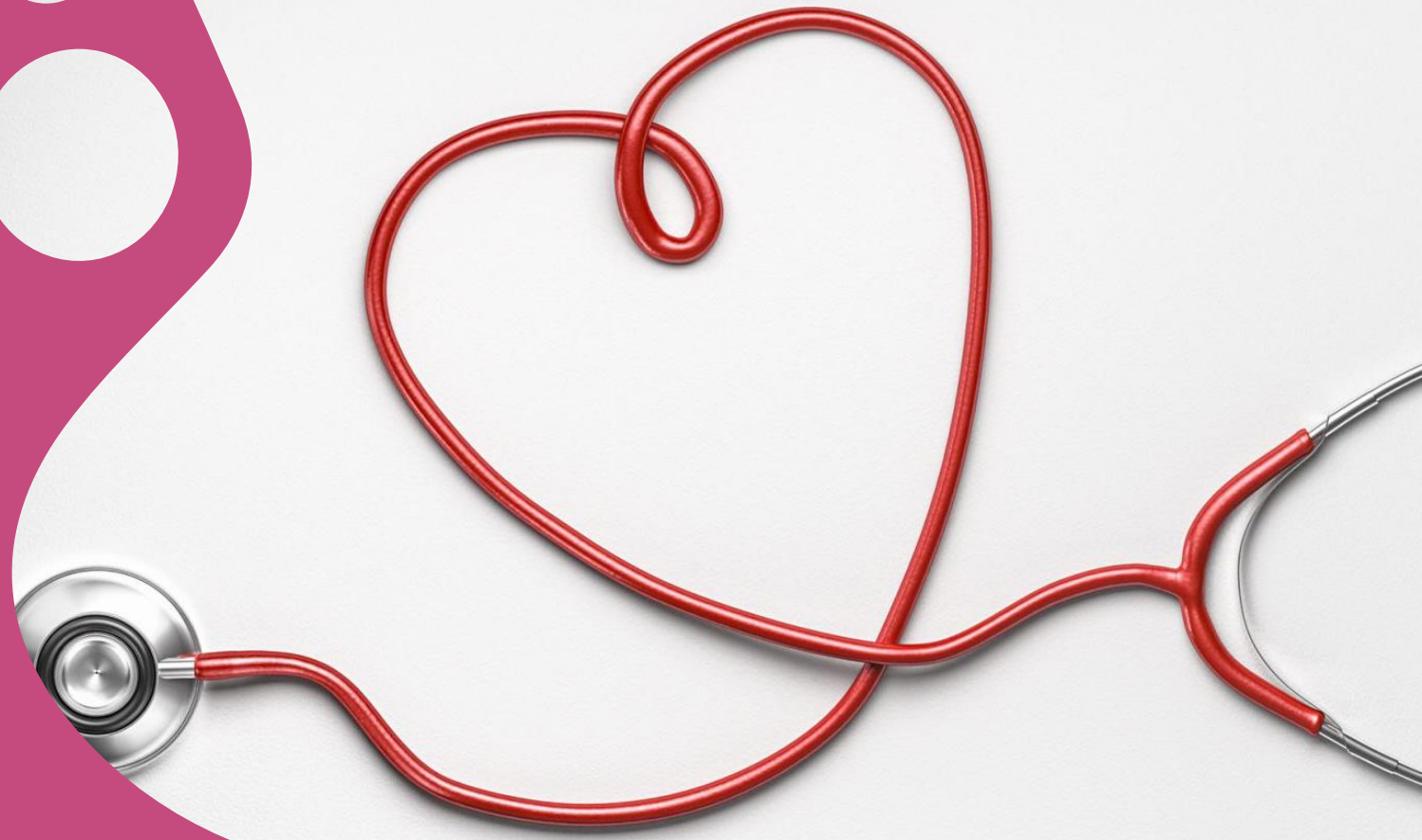
Don’t Break My Heart: Cardiology and Cancer

Shantanu Patil, MD

Assistant Professor of Medicine,

Creighton University School of Medicine, Omaha.

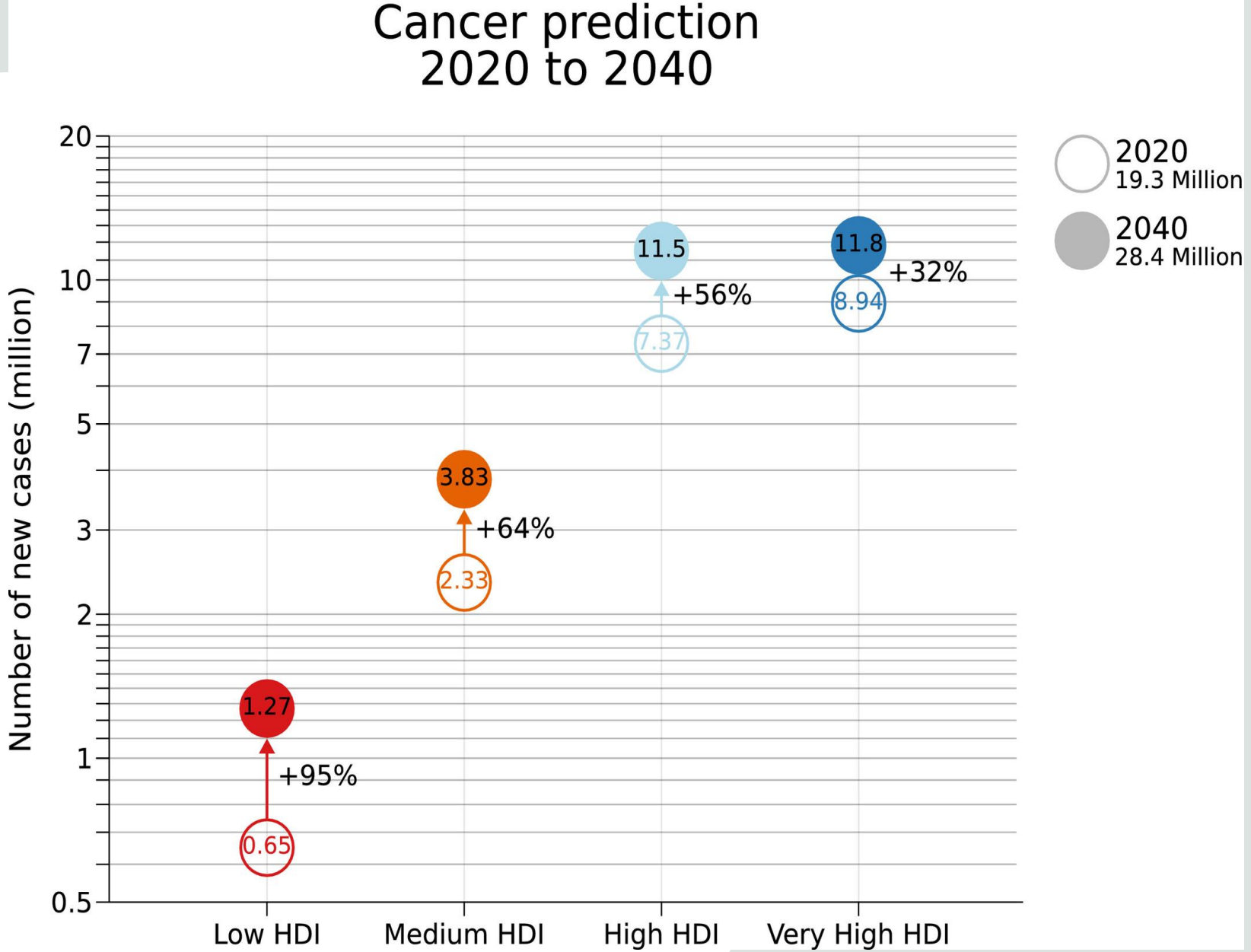


Conflict of Interest

None

Objectives

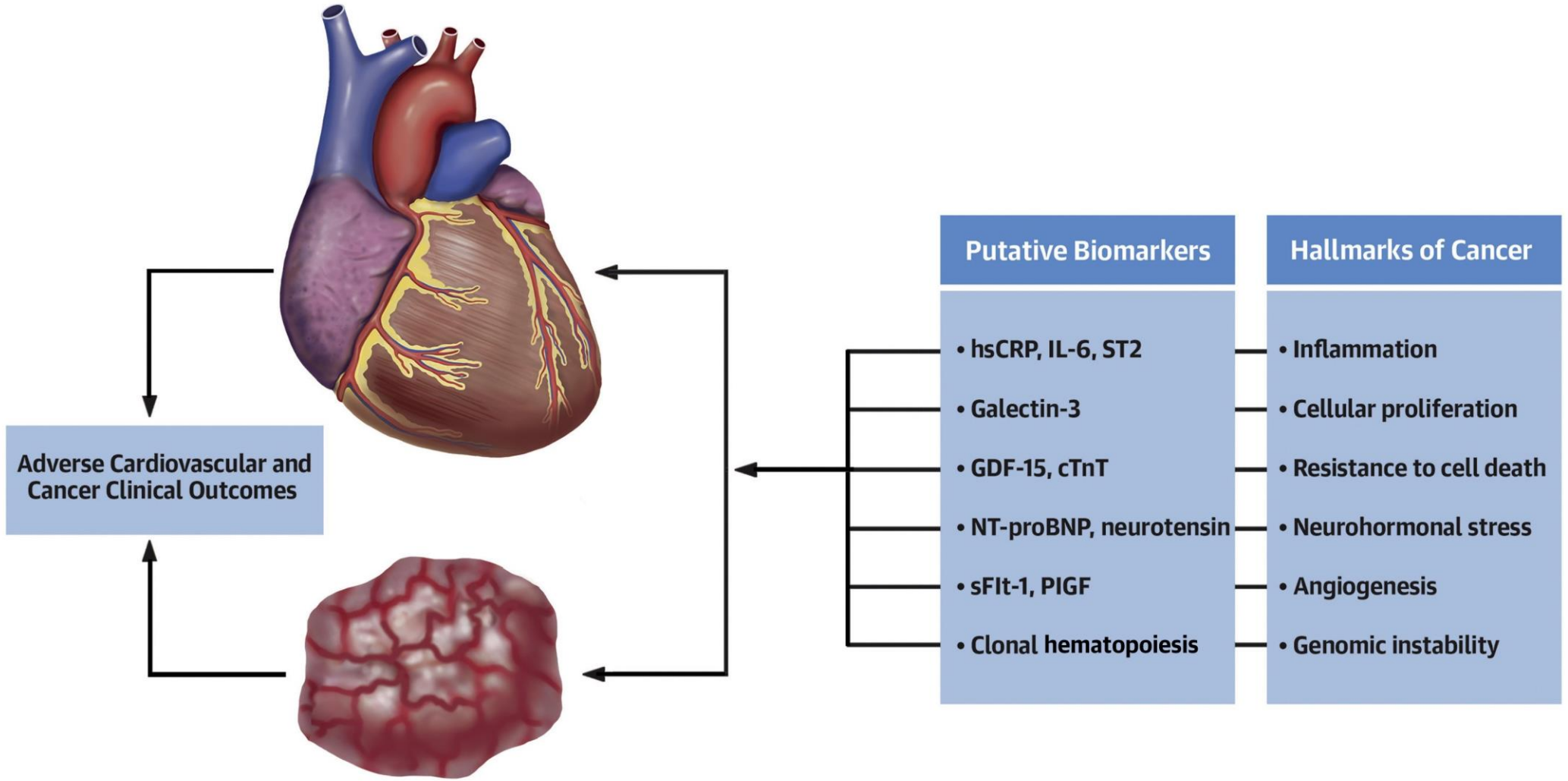
1. Discuss the common types of cardiotoxicities
2. Outline the significance of cardiac screening in oncological patients to recognize and mitigate cardiomyopathy risks
3. Describe effective management strategies for cardiotoxicities induced by chemotherapeutic agents in cancer patients



Author: Freddie Bray, Ahmedin Jemal, Isabelle Soerjomataram, et al

CA A Cancer J Clinicians, Volume: 71, Issue: 3, Pages: 209-249, First published: 04 February 2021, DOI: (10.3322/caac.21660)

CENTRAL ILLUSTRATION: Shared Pathophysiological Mechanisms Between Cardiovascular Disease and Cancer



Narayan, V. et al. J Am Coll Cardiol. 2020;75(21):2726–37.

Types of Cardiotoxiticies

1. Cancer therapeutic related Cardiac Dysfunction (CTRCD)

Symptomatic CTRCD (HF)[a,b]	Very severe	HF requiring inotropic support, mechanical circulatory support, or consideration of transplantation
	Severe	HF hospitalization
	Moderate	Need for outpatient intensification of diuretic and HF therapy
	Mild	Mild HF symptoms, no intensification of therapy required
Asymptomatic CTRCD	Severe	New LVEF reduction to <40%
	Moderate	New LVEF reduction by ≥10 percentage points to an LVEF of 40–49% *OR* New LVEF reduction by <10 percentage points to an LVEF of 40–49% AND either new relative decline in GLS by >15% from baseline OR new rise in cardiac biomarkers[c]
	Mild	LVEF ≥ ***50%*** AND new relative decline in GLS by >15% from baseline AND/OR new rise in cardiac biomarkers[c]

2.Immune check point inhibitors related myocarditis

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

3. Vascular toxicity

1. Coronary artery disease
2. Peripheral arterial disease
3. Deep venous thrombosis/ Pulmonary embolism
4. Vasospasm

4. Arterial Hypertension

1. Asymptomatic hypertension
2. Hypertensive emergency

5 Arrythmias

1. Supraventricular Tachycardia
2. Atrial fibrillation
3. QTc Prolongation

1. CRTCD – anthracyclines

Doxorubicin, daunorubicin, idarubicin, epirubicin, mitoxantrone

Doxorubicin targets the enzyme Top2 (topoisomerase 2) and binds to DNA to form the Top2-doxorubicin-DNA cleavage complex, which triggers cell death

Dose dependent

Preexisting cardiovascular condition dependent

Incidence 2-10 percent (depending on preexisting cardiovascular condition)

Median time interval is 3-6 months after chemotherapy,80 percent recover, 10 percent total and 70 percent with partial recovery. (which is why initiating cardioprotective medications before cardiotoxicity develops is utmost important)

Prevention

Screen high risk individuals:

1. Any patient receiving high-dose anthracycline (eg, cumulative doxorubicin ≥250 mg/m2, epirubicin ≥600 mg/m2).

2 Patients receiving lower-dose anthracycline (eg, cumulative doxorubicin <250 mg/m2, epirubicin <600 mg/m2) who have at least one of the following factors:

a)Concurrent low-dose radiotherapy (<30 Gy) where the heart is in the treatment field

b)Two or more CVD risk factors including smoking, hypertension, diabetes, dyslipidemia, or obesity.

c)Older age (≥60 years) at initial cancer treatment.

d) history of myocardial infarction or cardiomyopathy

e)Sequential treatment with a HER2-targeted agent (eg, trastuzumab)

3. High-dose radiotherapy (≥30 Gy) where the heart is in the treatment field.

Prevention

1) Alternative to anthracyclines

If LV EF < 40% before or during treatment

2) Infusional (over 48 hrs) rather than bolus regimens

3) Liposome-encapsulated of doxorubicin over unencapsulated

4) Dexrazoxane (iron chelator) lowers radical oxygen, approved in pts with > 500 mg/m2

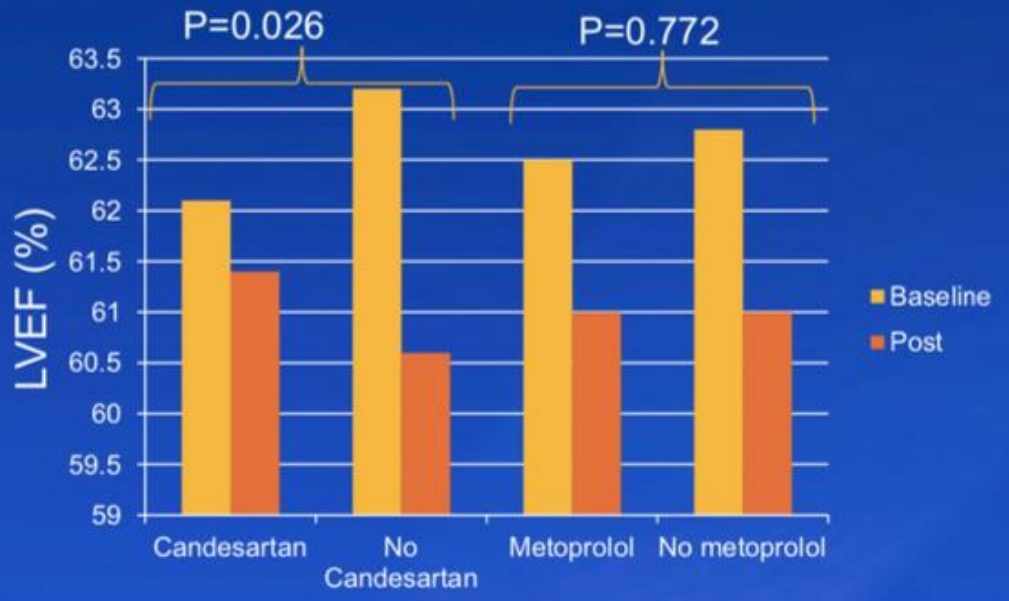
5) Routine cardiac screening

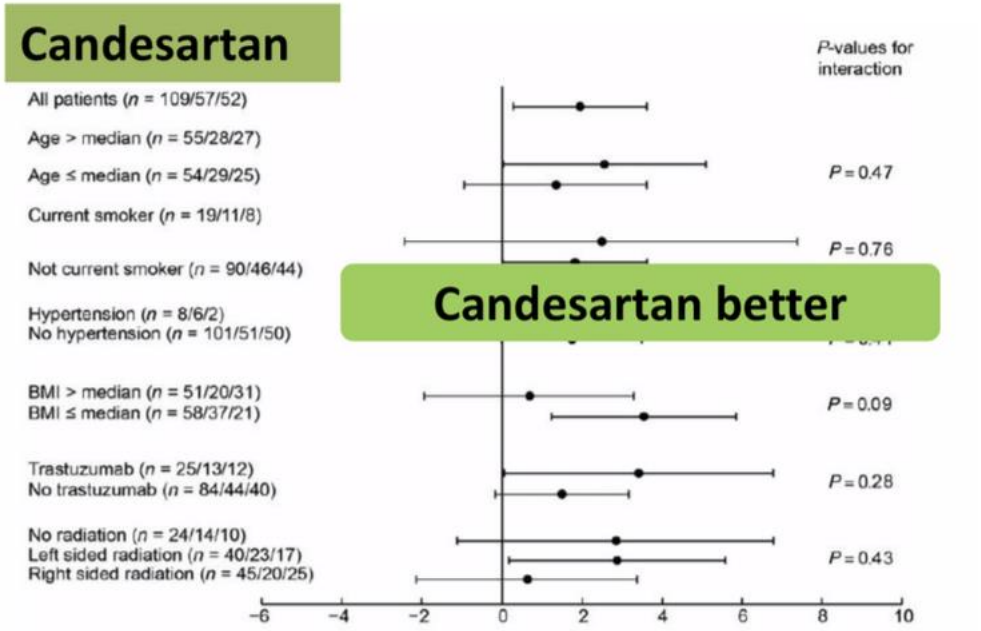
Cardioprotective medications ?

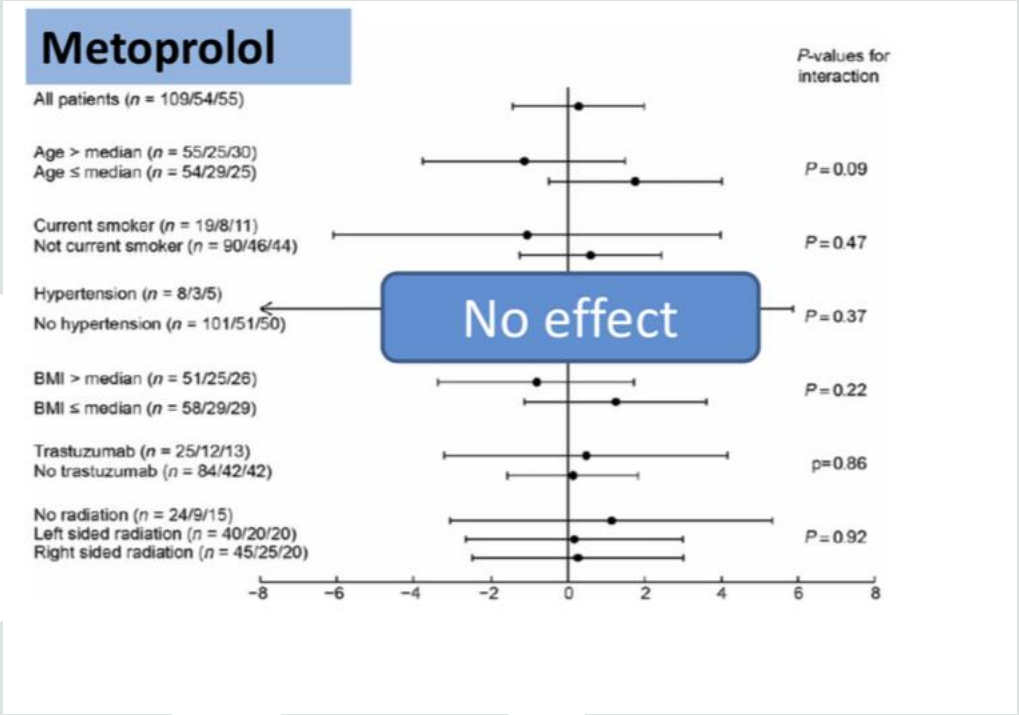
PRADA Trial: Prevention of Cardiac Dysfunction During Adjuvant Breast Cancer Therapy

ARB and Beta Blocker: the PRADA trial

- 130 women receiving fluorouracil, epirubicin, and cyclophosphamide (FEC) for breast cancer randomized to
  - Candesartan and placebo
  - Metoprolol succinate and placebo
  - Candesartan and metoprolol
  - Placebo and placebo
- Primary endpoint:
  - change in LVEF by CMR
- Results:
  - No CHF
  - LVEF dropped by 2.6% in placebo and 0.8% candesartan group (P=0.026)




Candesartan
P-values for interaction
All patients (n = 109/57/52)
Age > median (n = 55/28/27)
Age ≤ median (n = 54/29/25)
P = 0.47
Current smoker (n = 19/11/8)
P = 0.76
Not current smoker (n = 90/46/44)
Hypertension (n = 8/6/2)
No hypertension (n = 101/51/50)
Candesartan better
BMI > median (n = 51/20/31)
BMI ≤ median (n = 58/37/21)
P = 0.09
Trastuzumab (n = 25/13/12)
No trastuzumab (n = 84/44/40)
P = 0.28
No radiation (n = 24/14/10)
Left sided radiation (n = 40/23/17)
Right sided radiation (n = 45/20/25)
P = 0.43
−6 −4 −2 0 2 4 6 8 10


Metoprolol
P-values for interaction
All patients (n = 109/54/55)
Age > median (n = 55/25/30)
Age ≤ median (n = 54/29/25)
P = 0.09
Current smoker (n = 19/8/11)
Not current smoker (n = 90/46/44)
P = 0.47
Hypertension (n = 8/3/5)
No hypertension (n = 101/51/50)
No effect
P = 0.37
BMI > median (n = 51/25/26)
BMI ≤ median (n = 58/29/29)
P = 0.22
Trastuzumab (n = 25/12/13)
No trastuzumab (n = 84/42/42)
p=0.86
No radiation (n = 24/9/15)
Left sided radiation (n = 40/20/20)
Right sided radiation (n = 45/25/20)
P = 0.92
−8 −6 −4 −2 0 2 4 6 8

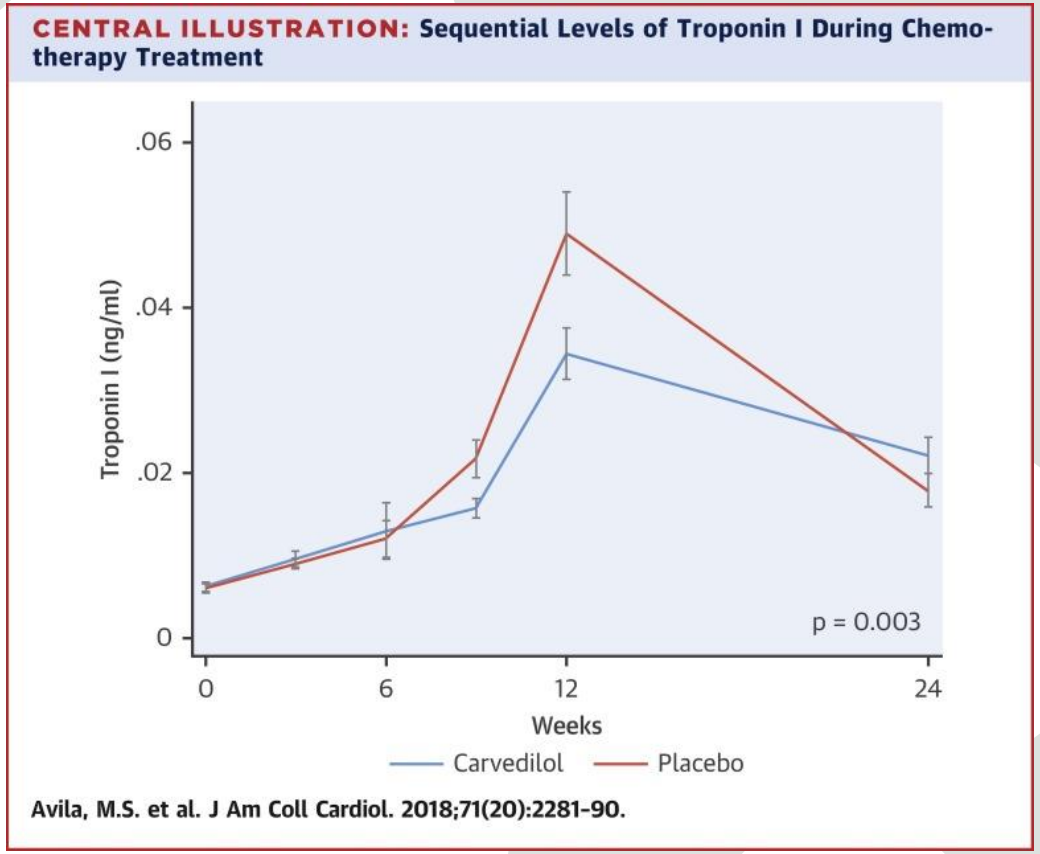
CECCY Trial (Carvedilol for Prevention of Chemotherapy-Related Cardiotoxicity)

CECCY Trial

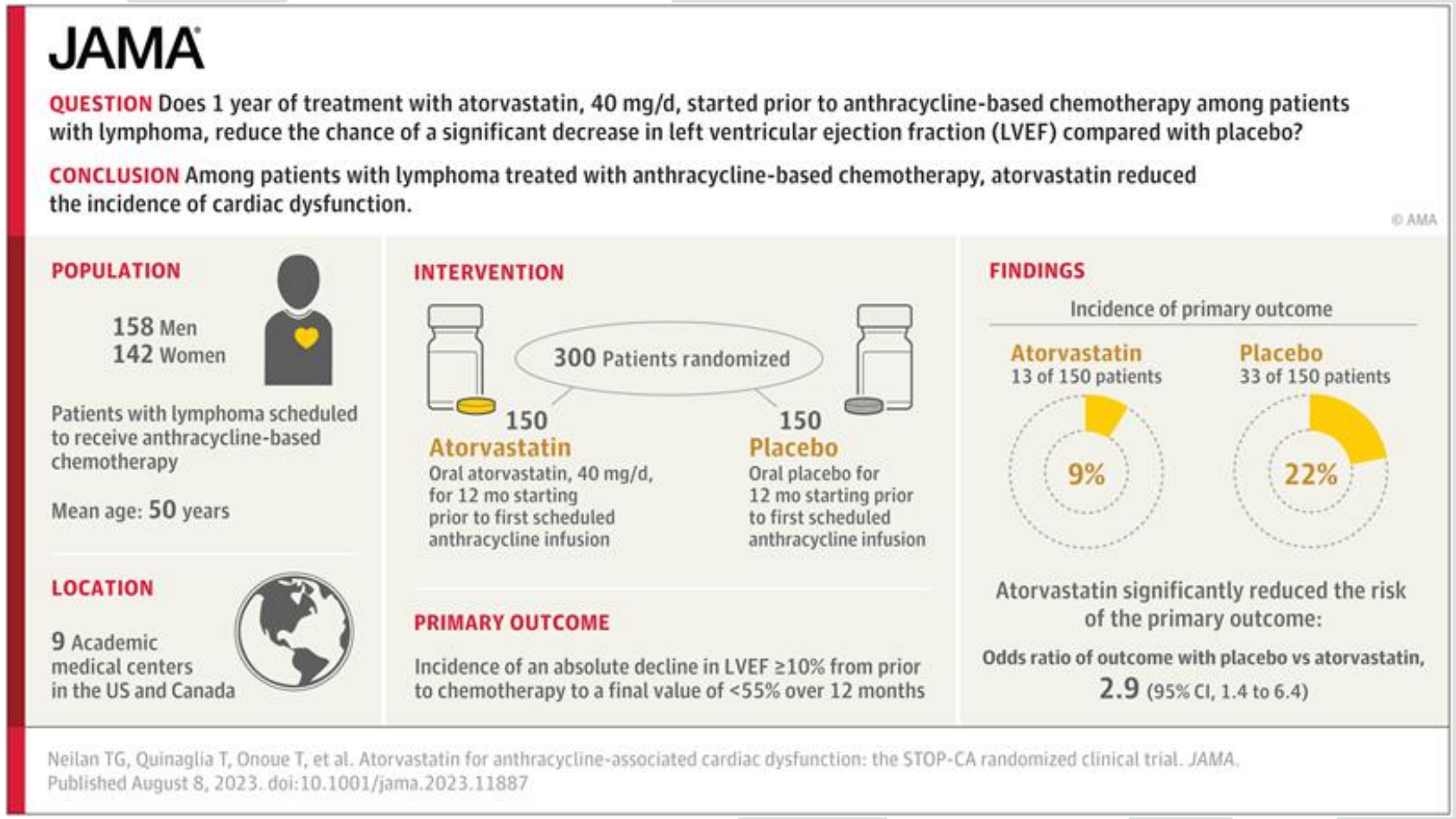
- 196 women receiving anthracycline chemotherapy for breast cancer
  - Cumulative dose 240 mg/m^2
- Randomized to carvedilol versus placebo
- 6 mo follow-up
- No significant difference in primary endpoint of a 10% decrease in EF
  - 14.5% vs 13.5%; p = 1.0
- No significant difference in the change in mean EF
  - 0.9% vs 1.3%; p = 0.84

Avila, *et al*. Carvedilol for prevention of chemotherapy related cardiotoxicity: The CECCY Trial. *J Am Coll Card* 2018;71:2281-90.

CECCY Trial (Carvedilol for Prevention of Chemotherapy-Related Cardiotoxicity)



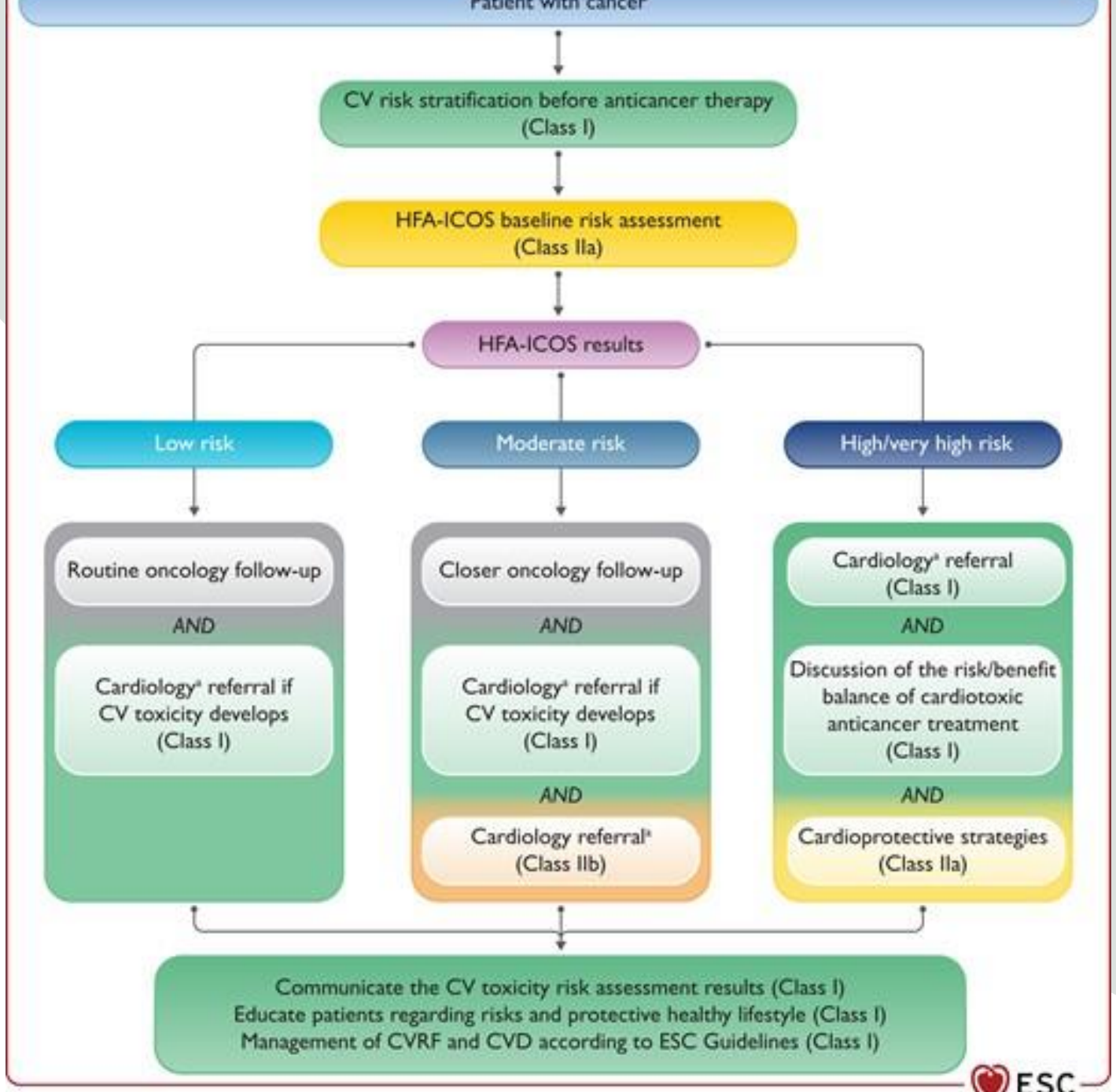
Statin ?


JAMA
QUESTION Does 1 year of treatment with atorvastatin, 40 mg/d, started prior to anthracycline-based chemotherapy among patients with lymphoma, reduce the chance of a significant decrease in left ventricular ejection fraction (LVEF) compared with placebo?
CONCLUSION Among patients with lymphoma treated with anthracycline-based chemotherapy, atorvastatin reduced the incidence of cardiac dysfunction.
© AMA
POPULATION
158 Men
142 Women
Patients with lymphoma scheduled to receive anthracycline-based chemotherapy
Mean age: 50 years
LOCATION
9 Academic medical centers in the US and Canada
INTERVENTION
300 Patients randomized
150
Atorvastatin
Oral atorvastatin, 40 mg/d, for 12 mo starting prior to first scheduled anthracycline infusion
150
Placebo
Oral placebo for 12 mo starting prior to first scheduled anthracycline infusion
PRIMARY OUTCOME
Incidence of an absolute decline in LVEF ≥10% from prior to chemotherapy to a final value of <55% over 12 months
FINDINGS
Incidence of primary outcome
Atorvastatin
13 of 150 patients
9%
Placebo
33 of 150 patients
22%
Atorvastatin significantly reduced the risk of the primary outcome:
Odds ratio of outcome with placebo vs atorvastatin,
2.9 (95% CI, 1.4 to 6.4)
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

Primary prevention

Broadly administered cardioprotective approach may not be required in most patients with early breast cancer without preexisting cardiovascular disease.

Instead, if there is an indication to treat , such as very high-risk patients (preexisting cardiovascular disease) use carvedilol and angiotensin receptor blocker


Patient with cancer
CV risk stratification before anticancer therapy (Class I)
HFA-ICOS baseline risk assessment (Class IIa)
HFA-ICOS results
Low risk
Moderate risk
High/very high risk
Routine oncology follow-up
AND
Cardiology[a] referral if CV toxicity develops (Class I)
Closer oncology follow-up
AND
Cardiology[a] referral if CV toxicity develops (Class I)
AND
Cardiology referral[a] (Class IIb)
Cardiology[a] referral (Class I)
AND
Discussion of the risk/benefit balance of cardiotoxic anticancer treatment (Class I)
AND
Cardioprotective strategies (Class IIa)
Communicate the CV toxicity risk assessment results (Class I)
Educate patients regarding risks and protective healthy lifestyle (Class I)
Management of CVRF and CVD according to ESC Guidelines (Class I)
ESC

Screening for cardiotoxicity

Cardiac MRI – accurate (patient and labor intensive, cost)

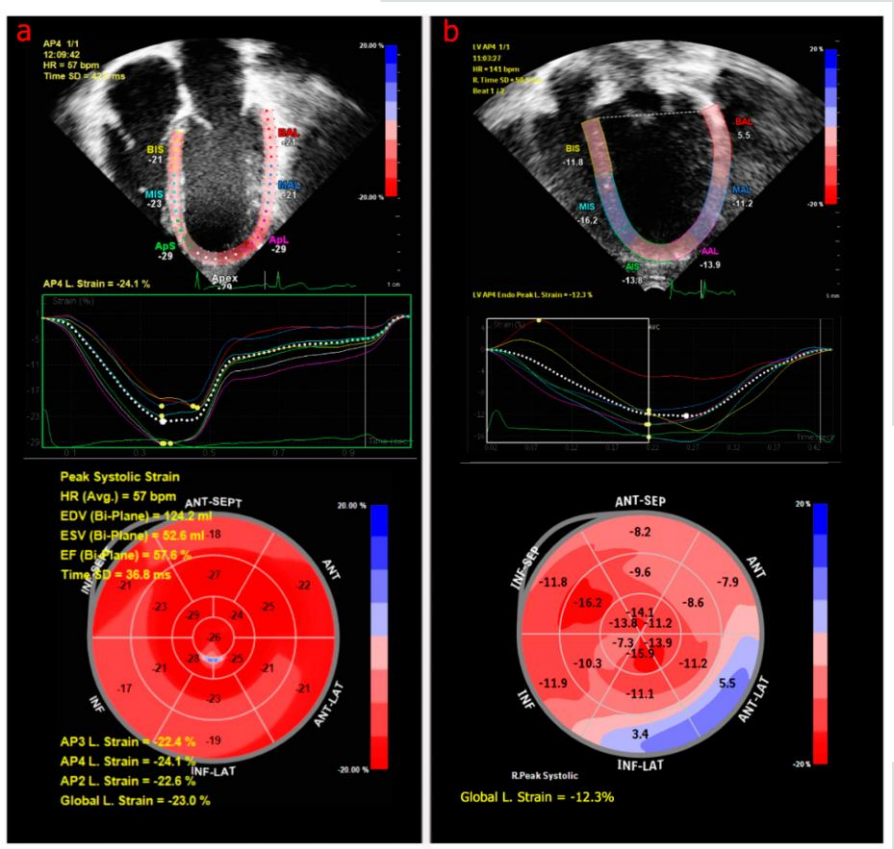
Echocardiogram

Radionuclide scan

How to screen

General	Class[a]	Level[b]
Echocardiography is recommended as the first-line modality for the assessment of cardiac function in patients with cancer.[4,12,54,94]	I	C
3D echocardiography is recommended as the preferred echocardiographic modality to measure LVEF.[77–79,89]	I	B
GLS is recommended in all patients with cancer having echocardiography, if available.[75,80,81,89,90,92,93,102,103]	I	C
CMR should be considered for the assessment of cardiac function when echocardiography is unavailable or non-diagnostic.[83,104,105]	IIa	C
MUGA may be considered when TTE is not diagnostic and CMR is not available.[106–108]	IIb	C

Echo with Strain Imaging



SUCCOUR TRIAL

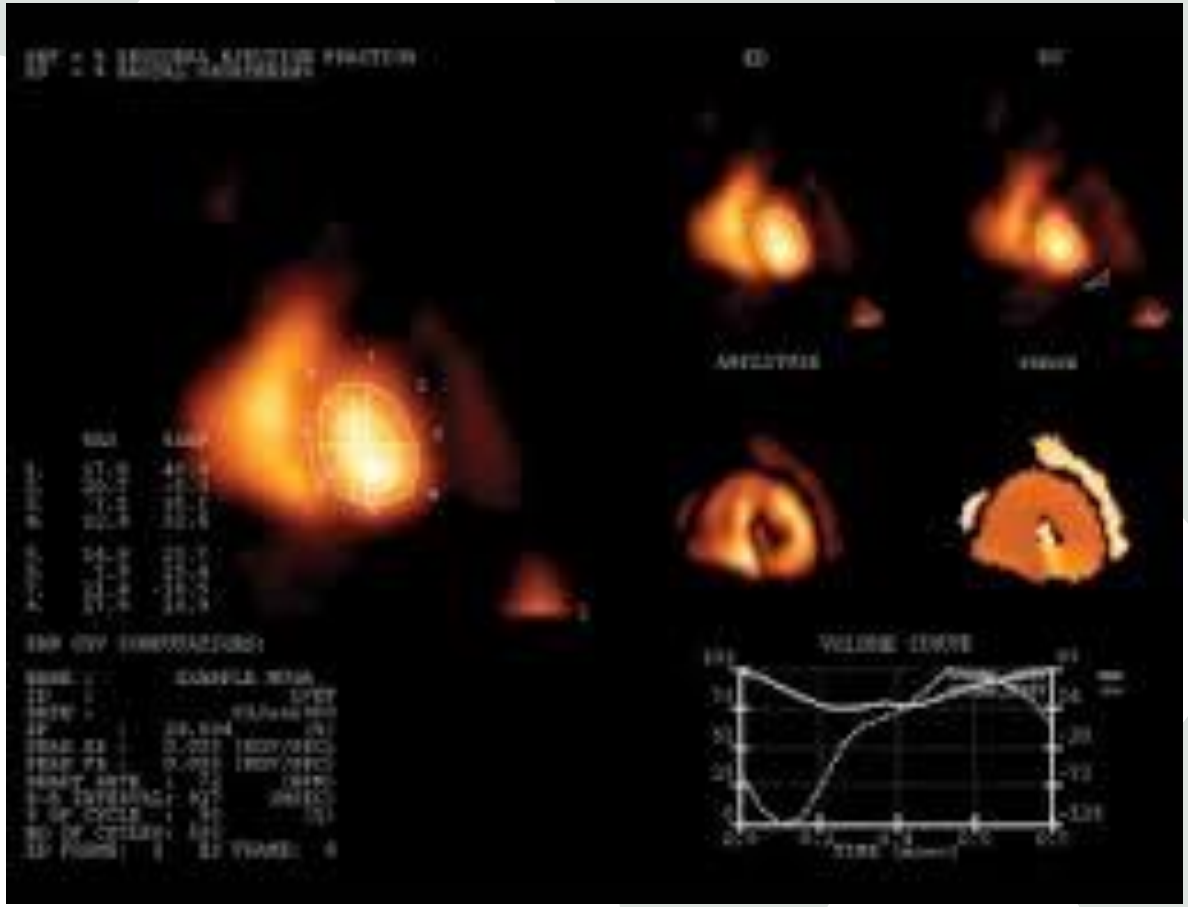
JACC: Cardiovascular Imaging
Volume 16, Issue 3, March 2023, Pages 269-278

Special Issue: Evidence-Based Imaging

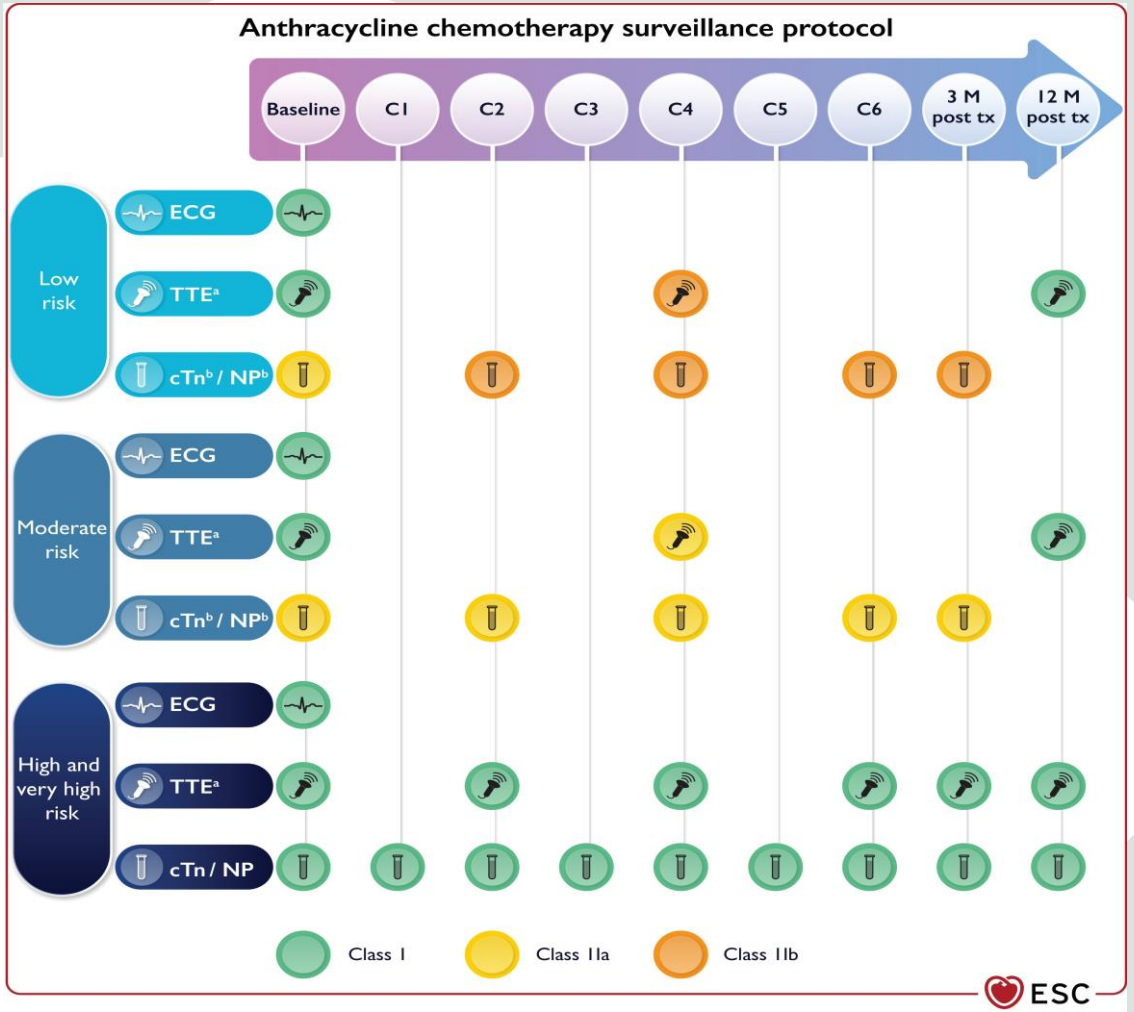
Randomized-Controlled Trial

Cardioprotection Using Strain-Guided Management of Potentially Cardiotoxic Cancer Therapy: 3-Year Results of the SUCCOUR Trial

MUGA scan



How often to screen



Examples- case 1

JM-75 yr old w lymphoma in 1990 , treated with bleomycin, vincristine, Cytoxan, novantrone, prednisolone.

2022, found to have positive lymph nodes on survelliance

Cardiac risk factors (paroxysmal atrial fibrillation s/p ablation, age > 60)

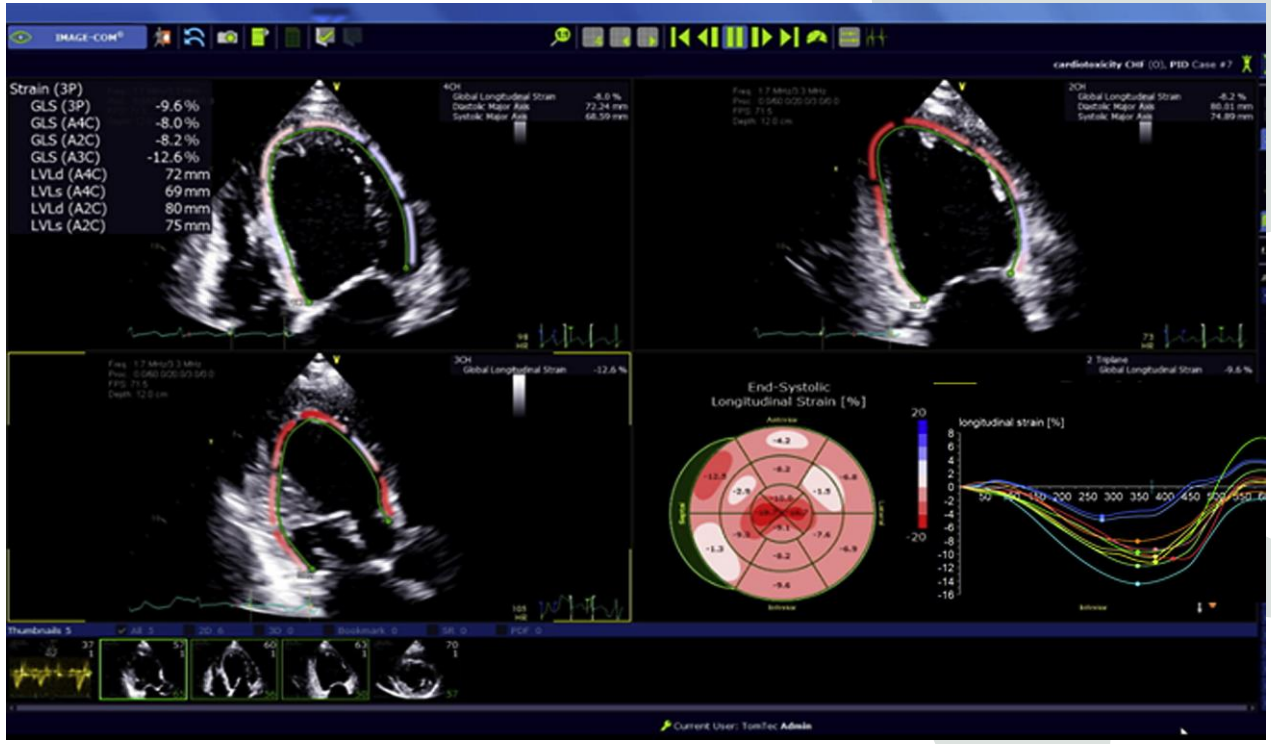
Received 6 cycles of R-pola – CHP(Rituximab, Polatuzumab, **Doxirubicin**, Prednisone)

Echo before chemotherapy

EF- 60%, GLS strain -18%, EKG – normal sinus rhythm

Noticed shortness of breath, leg swelling

On follow up EF -30-35%. GLS strain -8%



Started on Entresto, Toprol Xl, Jardiance, Lasix

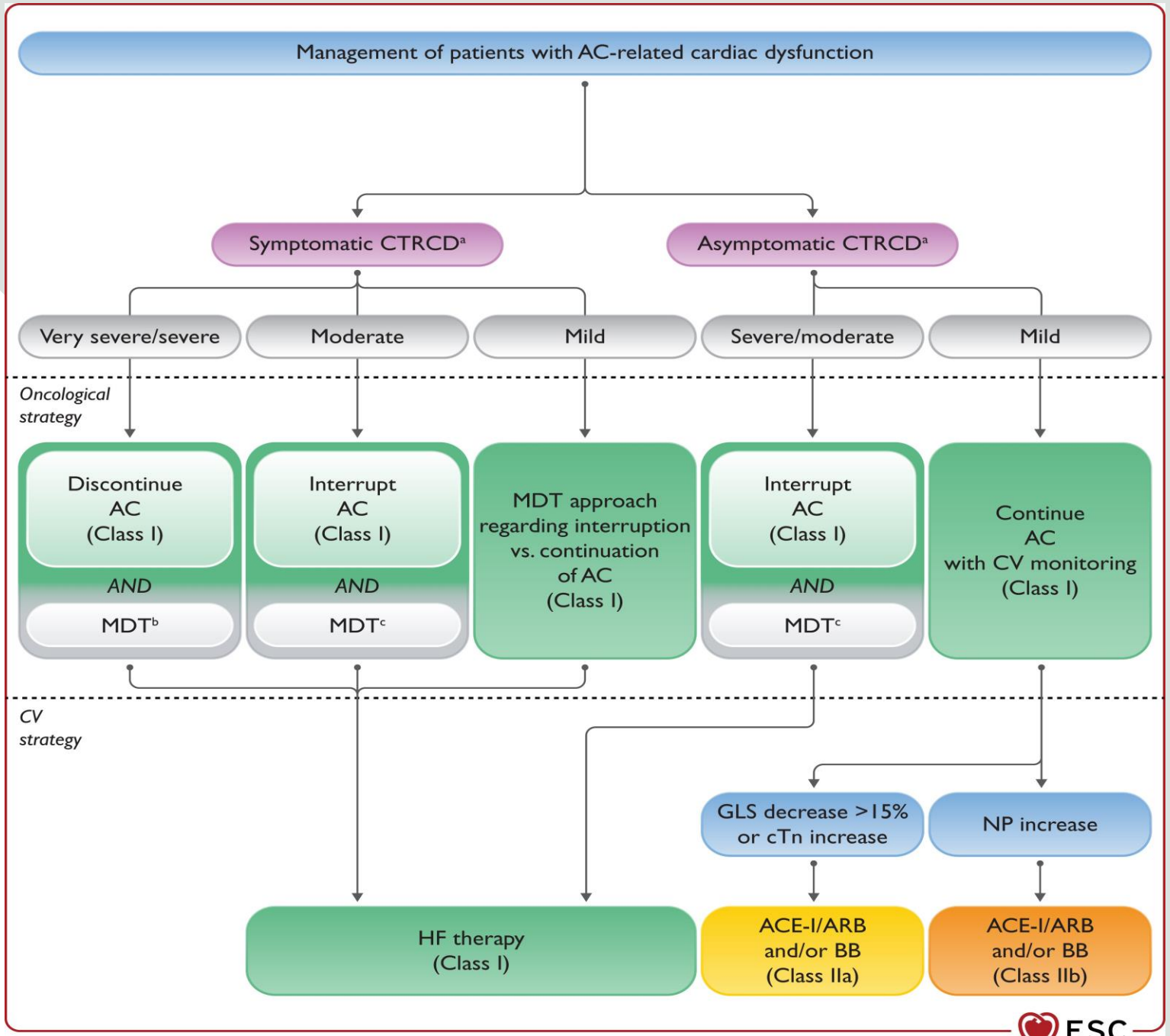
Cardiac cath – normal coronaries

Doxorubicin related cardiac dysfunction- symptomatic moderate HF

Follow up : Echo in 3 months

EF improved to 45-50% , NYHA class 1

Cancer Survelliance : resolution of lymphadenopathy on PET/CT


Management of patients with AC-related cardiac dysfunction
Symptomatic CTRCD[a]
Asymptomatic CTRCD[a]
Very severe/severe
Moderate
Mild
Severe/moderate
Mild
Oncological strategy
Discontinue AC (Class I)
AND
MDT[b]
Interrupt AC (Class I)
AND
MDT[c]
MDT approach regarding interruption vs. continuation of AC (Class I)
Interrupt AC (Class I)
AND
MDT[c]
Continue AC with CV monitoring (Class I)
CV strategy
GLS decrease >15% or cTn increase
NP increase
HF therapy (Class I)
ACE-I/ARB and/or BB (Class IIa)
ACE-I/ARB and/or BB (Class IIb)
ESC

2. CTRCD-HERCEPTIN

20% of breast cancer tumors human epidermal growth factor receptor 2 (HER2), which is associated with a worse prognosis

Monoclonal antibodies antagonistic to HER2 such as trastuzumab (Herceptin) have led to a paradigm shift in treatment of breast cancer

Incidence of HF was as high as 27% in initial trials when used concomitantly with Adriamycin but much lower when used sequentially (only1.6%)

New HER2-targeted agents ado-trastuzumab emtansine (T-DM1), fam-trastuzumab,pertuzumab may be less cardiotoxic than trastuzumab

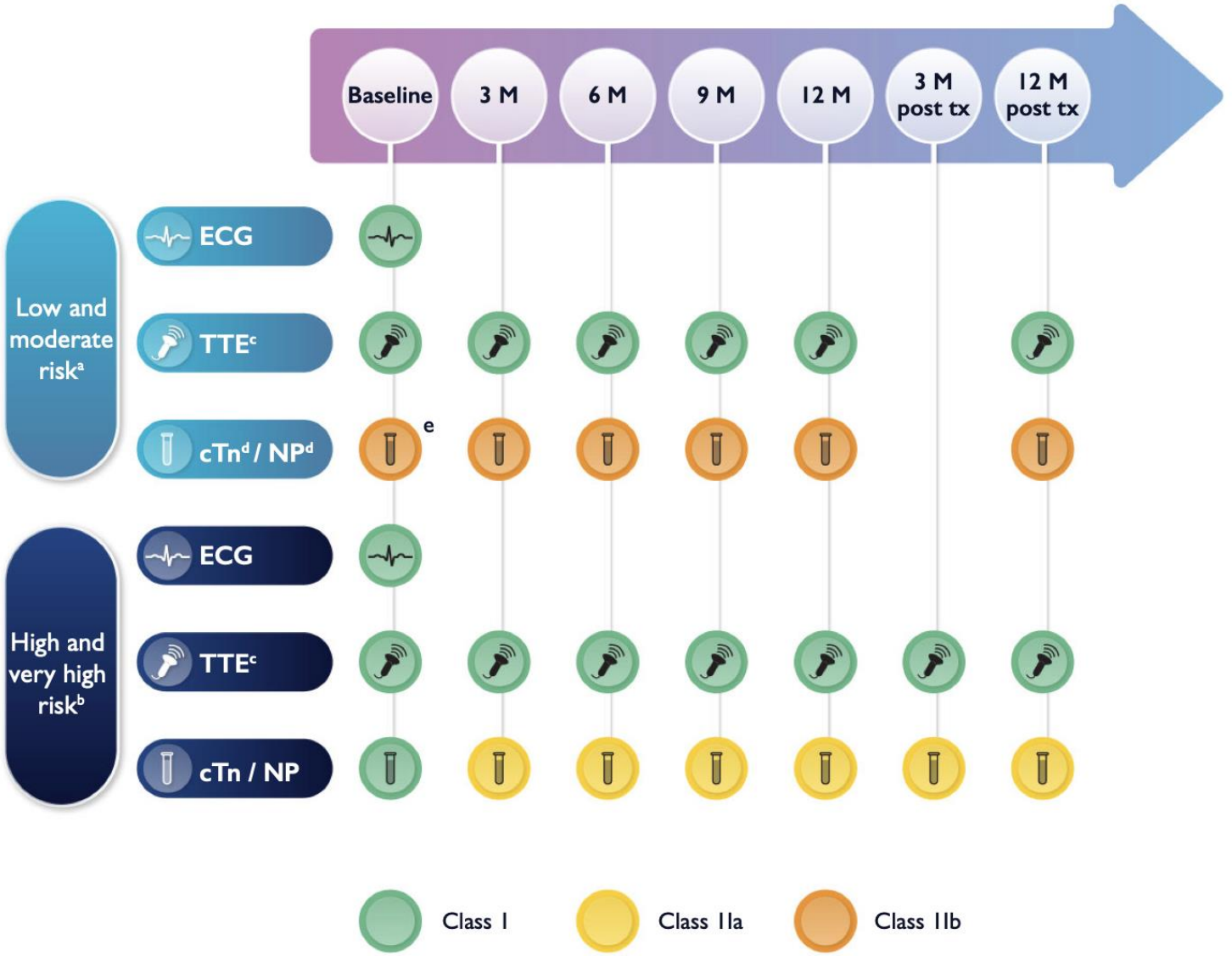
Not related to cumulative dose

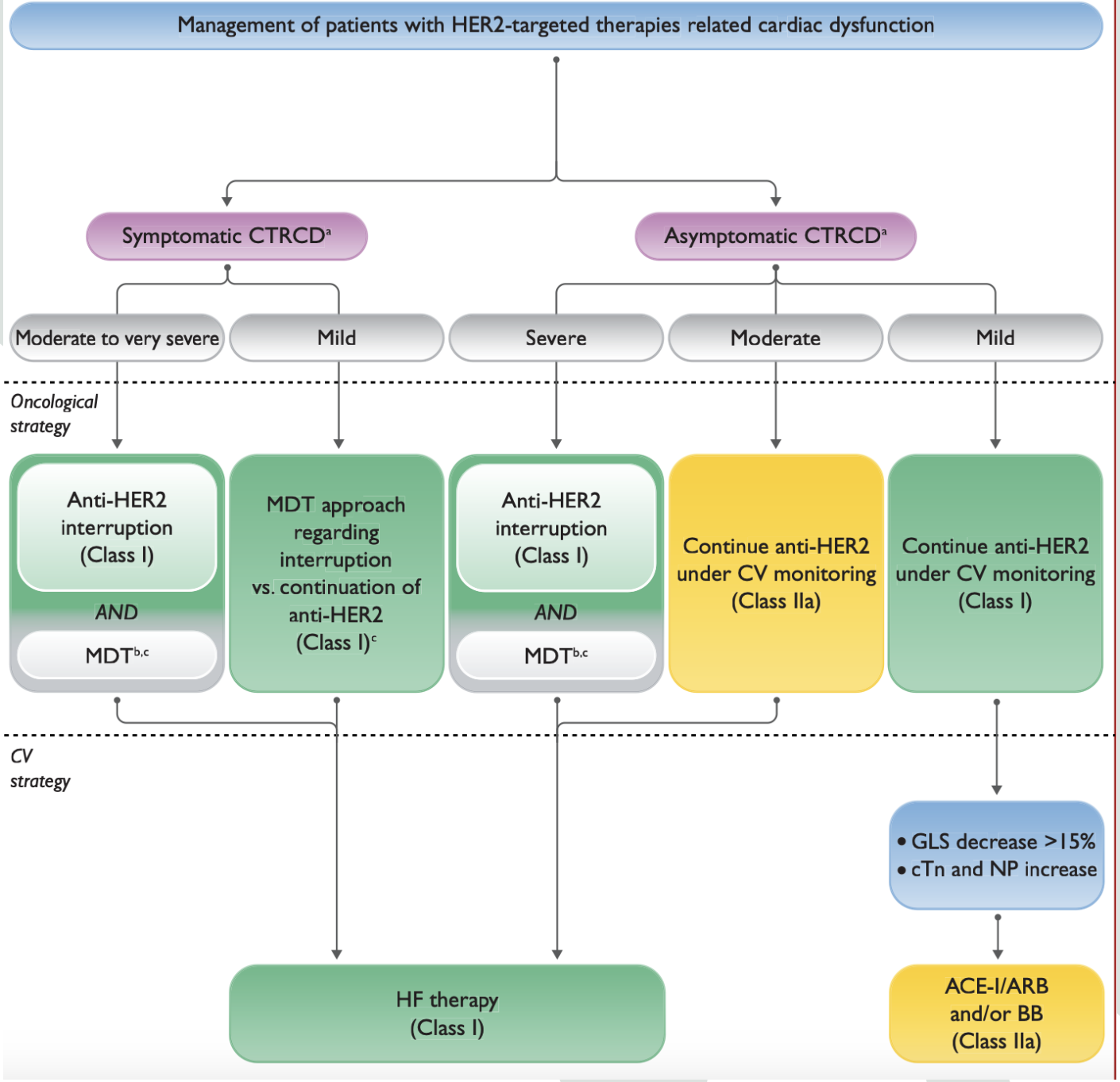
Occurs during therapy and rarely post therapy

Cessation of therapy (combined with beta-blockade and ACE inhibition) lead to recovery of EF

Rarely, herceptin cardiotoxicity can sometimes be irreversible

HER2-targeted therapy surveillance protocol




Management of patients with HER2-targeted therapies related cardiac dysfunction
Symptomatic CTRCD[a]
Asymptomatic CTRCD[a]
Moderate to very severe
Mild
Severe
Moderate
Mild
Oncological strategy
Anti-HER2 interruption (Class I)
AND
MDT[b,c]
MDT approach regarding interruption vs. continuation of anti-HER2 (Class I)[c]
Anti-HER2 interruption (Class I)
AND
MDT[b,c]
Continue anti-HER2 under CV monitoring (Class IIa)
Continue anti-HER2 under CV monitoring (Class I)
CV strategy
• GLS decrease >15%
• cTn and NP increase
HF therapy (Class I)
ACE-I/ARB and/or BB (Class IIa)

Example 2

76 yr old F , hx of breast cancer in 2006, lumpectomy and radiation.

On follow up found to have multifocal breast cancer, started treatment with ddAC(dense dose doxorubicin and cyclophosphamide) followed by THP(taxotere, trastuzumab, pertuzumab)

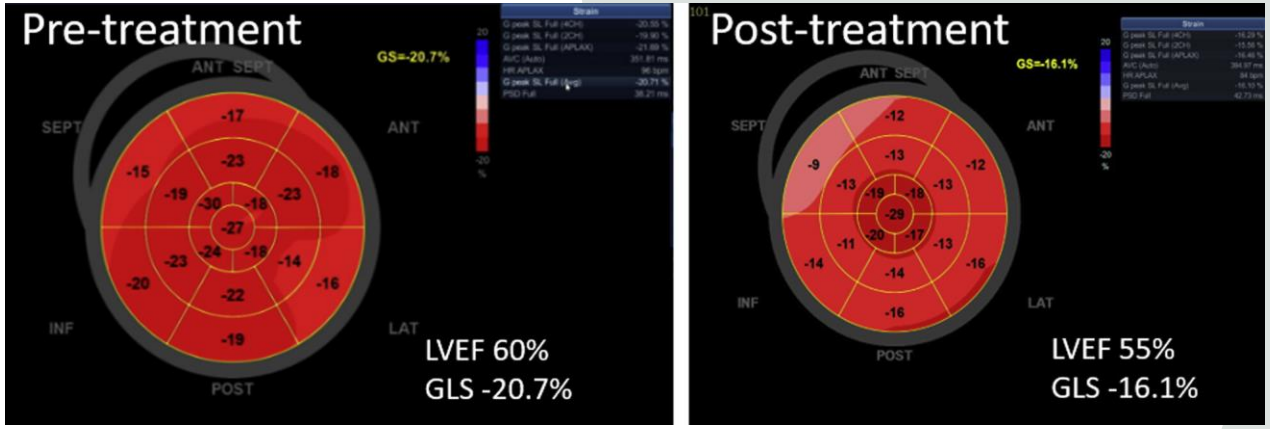
Cardiac risk factors : age >60, HTN

Routine screening Echo every 3 months.

Baseline Echo: EF 60%. GLS -20.7%

6 months into Herceptin based therapy

On routine followup , no symptoms, exam normal



Management

Discussion with Oncologist

Started on BB and ARB

Repeat echo planned in 6 weeks

Improvement in EF 60%, GLS -18.1% (still slightly low)

Repeat echo in 12 weeks, EF 60%, GLS -18.3%

Plan to continue Herceptin and continue serial Echocardiograms for now

Vascular toxicity
e.g. Fluoropyrimidines

5-fluorouracil (5-FU) and capecitabine

Coronary vasospasm and endothelial dysfunction

Angina (2-20%)

Common with longer infusions of 5-FU

Pre-exisiting CAD

Screening

Recommendations	Class[a]	Level[b]
Baseline CV risk assessment and evaluation including BP measurement, ECG, lipid profile, HbA1c measurement, and SCORE2/ SCORE2-OP[c] or equivalent is recommended[19] before starting fluoropyrimidines.	I	C
A baseline echocardiogram is recommended in patients with a history of symptomatic CVD before starting fluoropyrimidines.	I	C
Screening for CAD[d] may be considered in patients at high and very high risk of CAD[c] before fluoropyrimidines.	IIb	C

Management

Stop Infusion

EKG, cardiac enzymes, Angiography

Rechallenge ?

Vasodilators, Nitrates

Bolus regimen

In Hospital challenge in severe cases

Antidote- Uridine triacetate (not found easily)

Example

60 yr old male with hx of HLD diagnosed with colon ca referred for pre 5-FU screening.

Very active, gifted a new puppy (roughly 4 Mets)

Echo EF 55%, GLS -16%

EKG normal sinus rhythm

Started 5 FU , sent home on infusion

Day 2 , developed chest pain → came to ER

Positive troponins, EKG new t wave inv, Bedside echo apical hypokinesis EF 45%

Infusion stopped, slg nitro given with resolution of chest pain, taken to cath lab

Normal Coronaries

5 FU related -Suspected Coronary Vasospam

Rechallenge ?

D/w Oncologist

CCB and Nitrates initiated

Bolus regimen given ; first dose administered with direct observation

No recurrence of pain

Immune check point inhibitors

The immune checkpoints are proteins expressed in the T cells that inhibit their activation when they contact a body cell.

By blocking these checkpoints from binding with their partner proteins, ICI inhibit the ‘off’ signal, activating T cells and promoting killing of cancer cells.

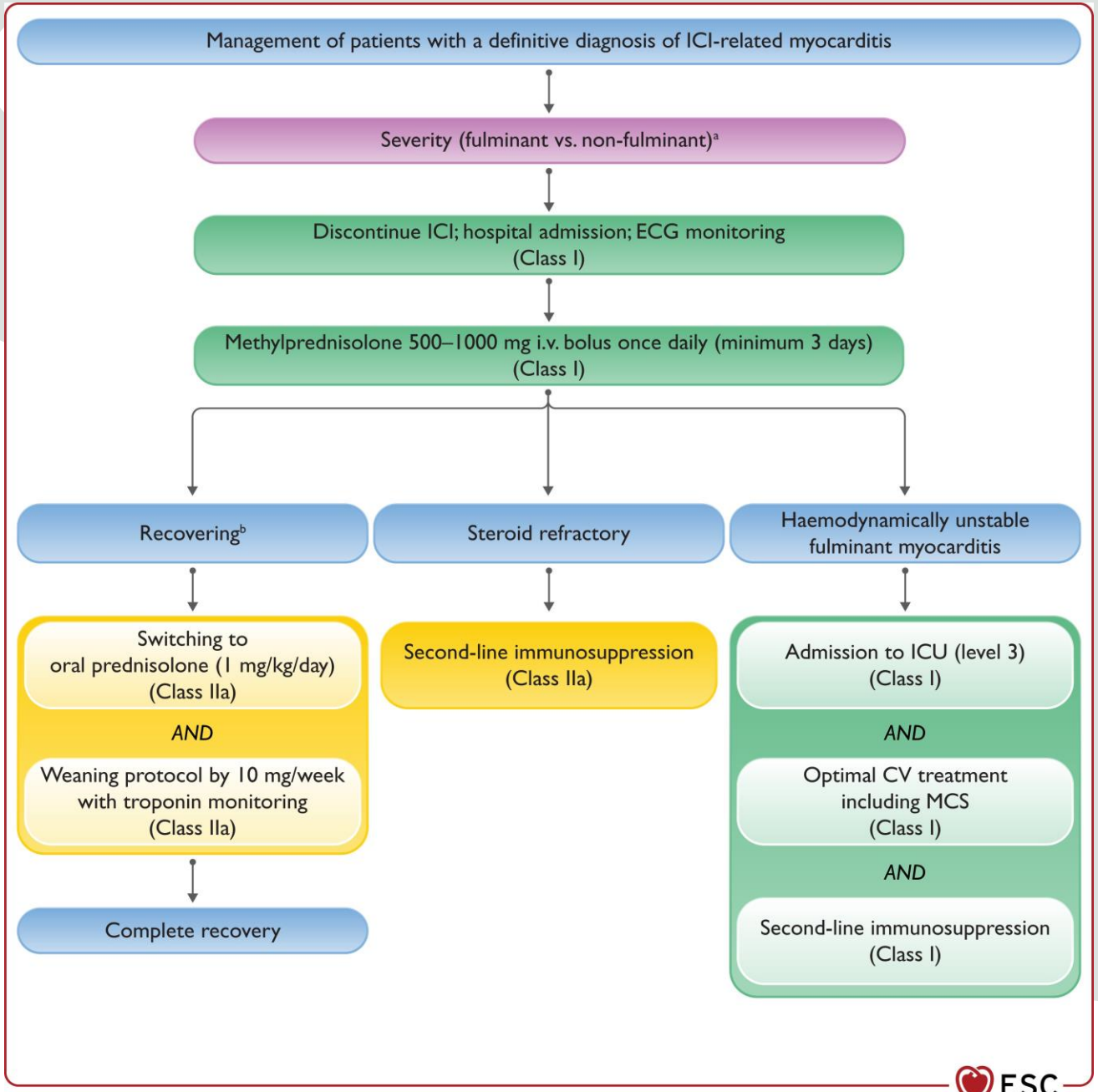
Overactivation of T cells against non-cancerous tissues, can lead immune-related adverse events such as myocarditis

True incidence low (<1%) , Median onset 30 days (can occur upto 6 months as well)

Risk -dual ICI therapy (e.g. ipilimumab and nivolumab), combination ICI therapy with other cardiotoxic therapies, and patients with ICI-related non-CV events

Recommendations	Class[a]	Level[b]
ECG, NP, and cTn measurements are recommended in all patients before starting ICI therapy.[333]	I	B
Baseline echocardiography is recommended in high-risk patients[c] before starting ICI therapy.[333]	I	B
Baseline echocardiography may be considered in all patients before starting ICI therapy.	IIb	C
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
CV assessment[d] is recommended every 6–12 months in high-risk patients[c] who require long-term (>12 months) ICI treatment.[321–323,335,336]	I	C
CV assessment[d] may be considered every 6–12 months in all patients who require long-term (>12 months) ICI treatment.	IIb	C

Grading	Management
G1: Abnormal cardiac biomarker testing without symptoms and with no ECG abnormalities.	▪ All grades warrant work-up and intervention, given the potential for cardiac compromise. ▪ Hold ICPi for G1 elevated troponin[¶] and recheck troponin 6 hours later. May consider resuming once normalized or if believed not to be related to ICPi. ▪ Hold ICPi and discontinue for ≥G2: • For patients with grade ≥2, early (ie, within 24 hours) initiation of high-dose corticosteroids (prednisone 1 to 2 mg/kg/day, oral or IV equivalent depending on symptoms) should be considered as it is likely to be beneficial without adverse effects. • Admit patient for cardiology consultation. ▪ Management of cardiac symptoms according to ACC/AHA guidelines and with guidance from cardiology. ▪ Immediate transfer to a coronary care unit should be considered for patients with elevated troponin or conduction abnormalities. ▪ For new conduction delay, consider a pacemaker. ▪ In patients without an immediate response to high-dose corticosteroids, consider early institution of cardiac transplant rejection doses of corticosteroids (methylprednisolone 1 g every day) and the addition of either mycophenolate, infliximab, or antithymocyte globulin. Consider abatacept (costimulatory molecule blockade) or alemtuzumab (CD52 blockade) as additional immunosuppression in life-threatening cases.
G2: Abnormal cardiac biomarker testing with mild symptoms or new ECG abnormalities without conduction delay.	
G3: Abnormal cardiac biomarker testing with either moderate symptoms or new conduction delay.	
G4: Moderate to severe decompensation, IV medication or intervention required, life-threatening conditions.	


Management of patients with a definitive diagnosis of ICI-related myocarditis
Severity (fulminant vs. non-fulminant)[a]
Discontinue ICI; hospital admission; ECG monitoring (Class I)
Methylprednisolone 500–1000 mg i.v. bolus once daily (minimum 3 days) (Class I)
Recovering[b]
Steroid refractory
Haemodynamically unstable fulminant myocarditis
Switching to oral prednisolone (1 mg/kg/day) (Class IIa)
AND
Weaning protocol by 10 mg/week with troponin monitoring (Class IIa)
Second-line immunosuppression (Class IIa)
Admission to ICU (level 3) (Class I)
AND
Optimal CV treatment including MCS (Class I)
AND
Second-line immunosuppression (Class I)
Complete recovery

Example

65 yr old diagnosed with metastatic non small cell lung cancer PD L1 level > 50% recently started on Keytruda (pembroluzimab) .

Cardiac hx: smoking hx, left SFA stent

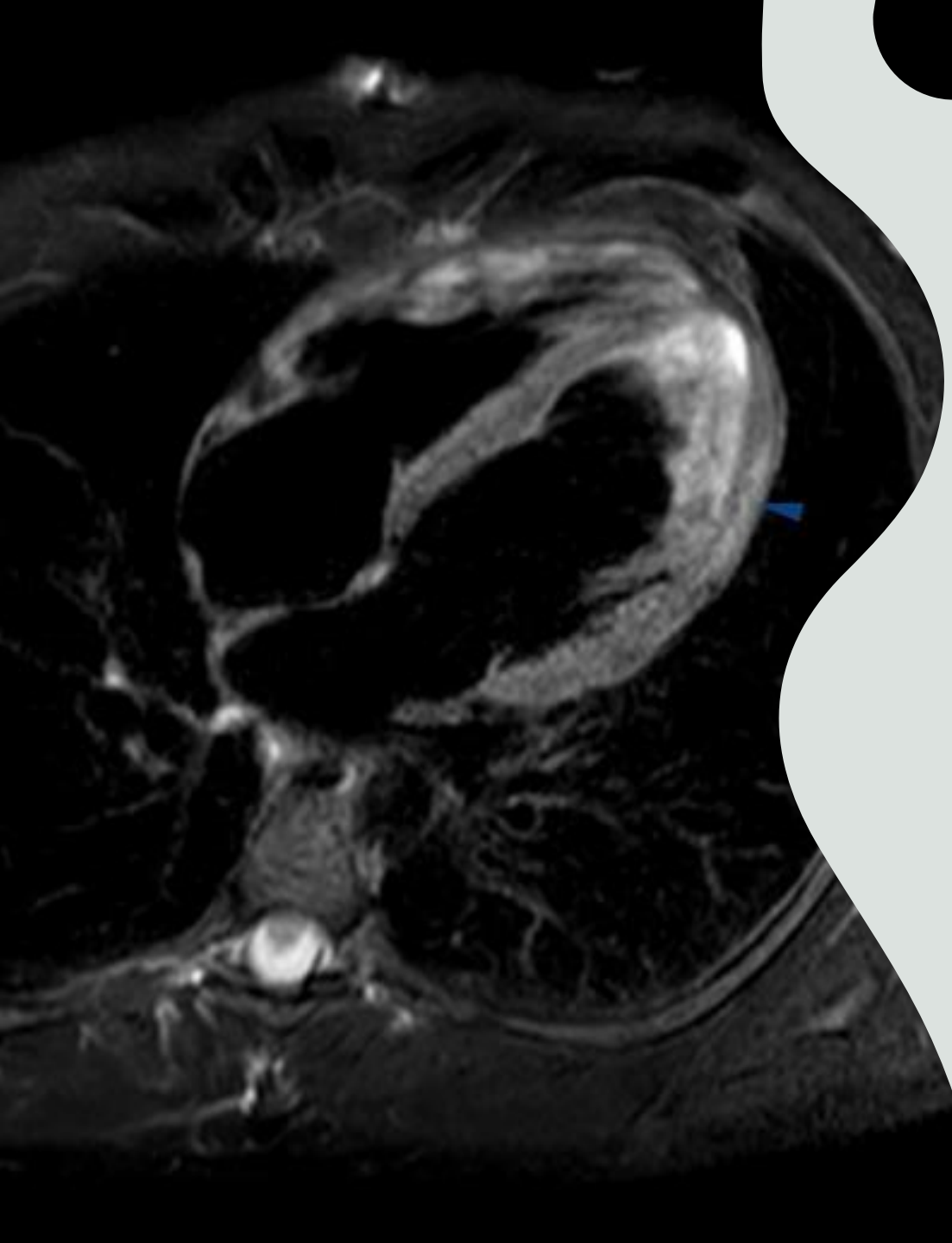
Called Oncologist with chest pain, office visit with EKG changes, instructed to go to ER

Trop 2462→ 2514→2458

Echo with EF 40-45%, Global Hypokinesis

Taken to the Cath Lab

Mild Coronary Artery Disease, non obstructive



Started on 1 gm IV methylprednisone x 3 days

MRI showed Myocardial Wall Edema and Epicardial LGE meets Lake Louise Criteria for myocarditis

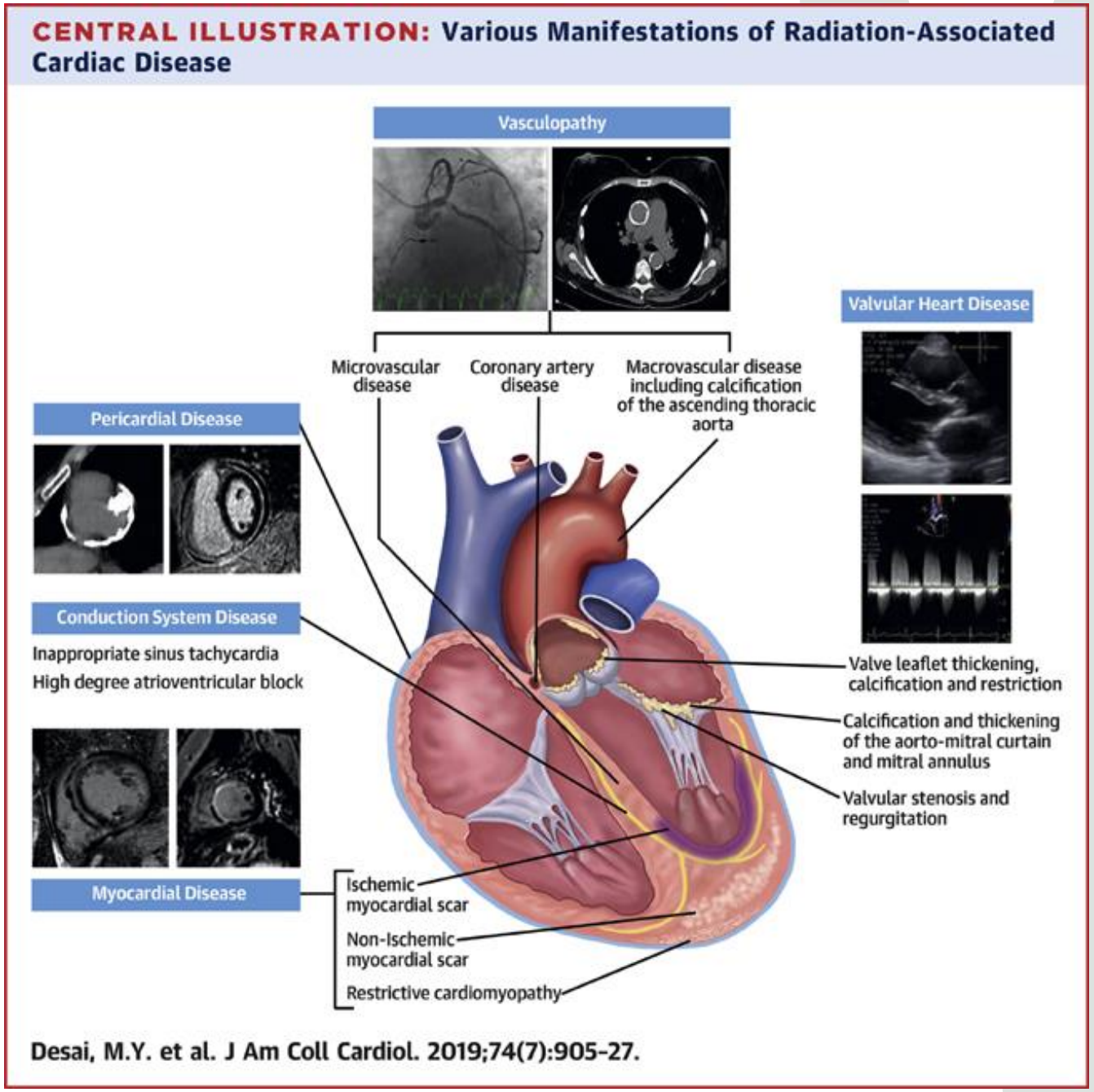
Troponins decreased to 1200

Chest Pain improved

Clinically stable

Discharged on Oral Prednisone “ slow” taper

60mg , decrease by 10 mg every 1-2 weeks.



Radiation therapy

Radiation therapy

Mantle radiation and extended field mantle radiation were the standard of care for several decades for Hodgkin's lymphoma or breast cancer, with radiation exposures significantly higher than 30 Gy

Partial breast irradiation have been shown to result in significantly lower radiation doses to the left anterior descending artery (LAD) (mean 2.13 Gy ± 0.11 vs. 1.02 ± 0.17), with right-sided partial breast radiation resulting in minimal exposure.

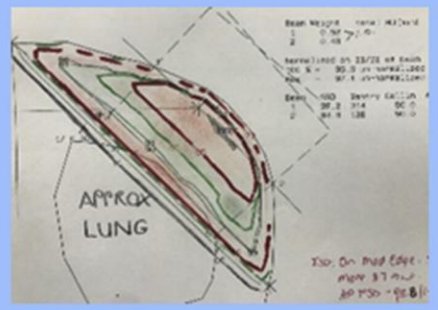
The development of newer cardiac-shielding techniques, such as the multi-leaf collimator modification technique, deep inspiration breath holding and intensity-modulated radiation therapy minimize total cardiac radiation dose.

Breast Cancer

Hand/Digitized Planning

- Based on patient contours
- Patient in free breathing
- Dosimetry using 2D tangential fields
- Heart volume unable to be defined

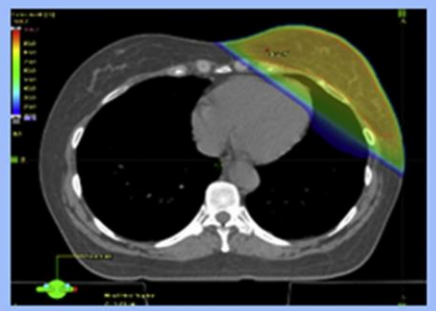
Increased Mean Heart Dose - Unable to Quantify



CT Planning

- Supine position common
- Patient in free breathing
- Dosimetry using 3D conformal radiotherapy (3DCRT)

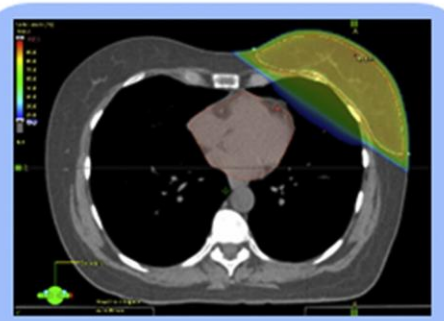
Mean Heart Dose > 1-3Gy



Advanced CT Planning

- Supine v Prone
- Deep inspiration breath hold
- Dosimetry using IMRT/VMAT
- Partial breast or omission of RT if clinically appropriate

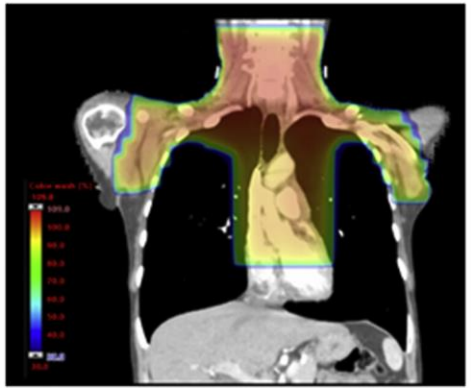
Mean Heart Dose Often < 1 Gy



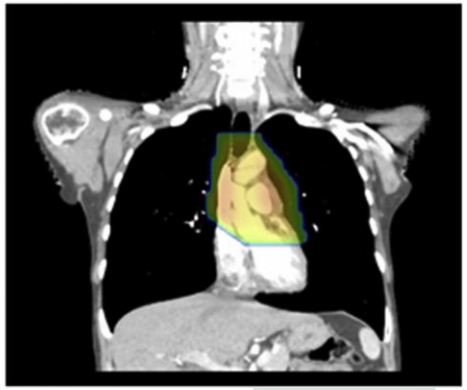
Pre-1990s 2000s 2010s 2020s

Lymphoma

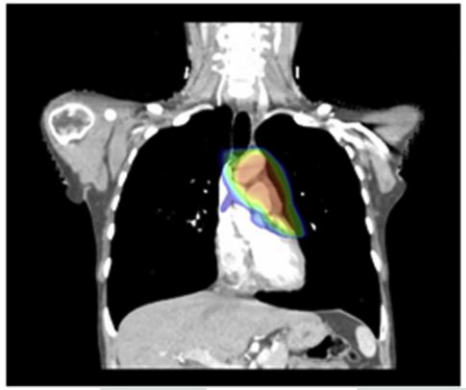
A



B

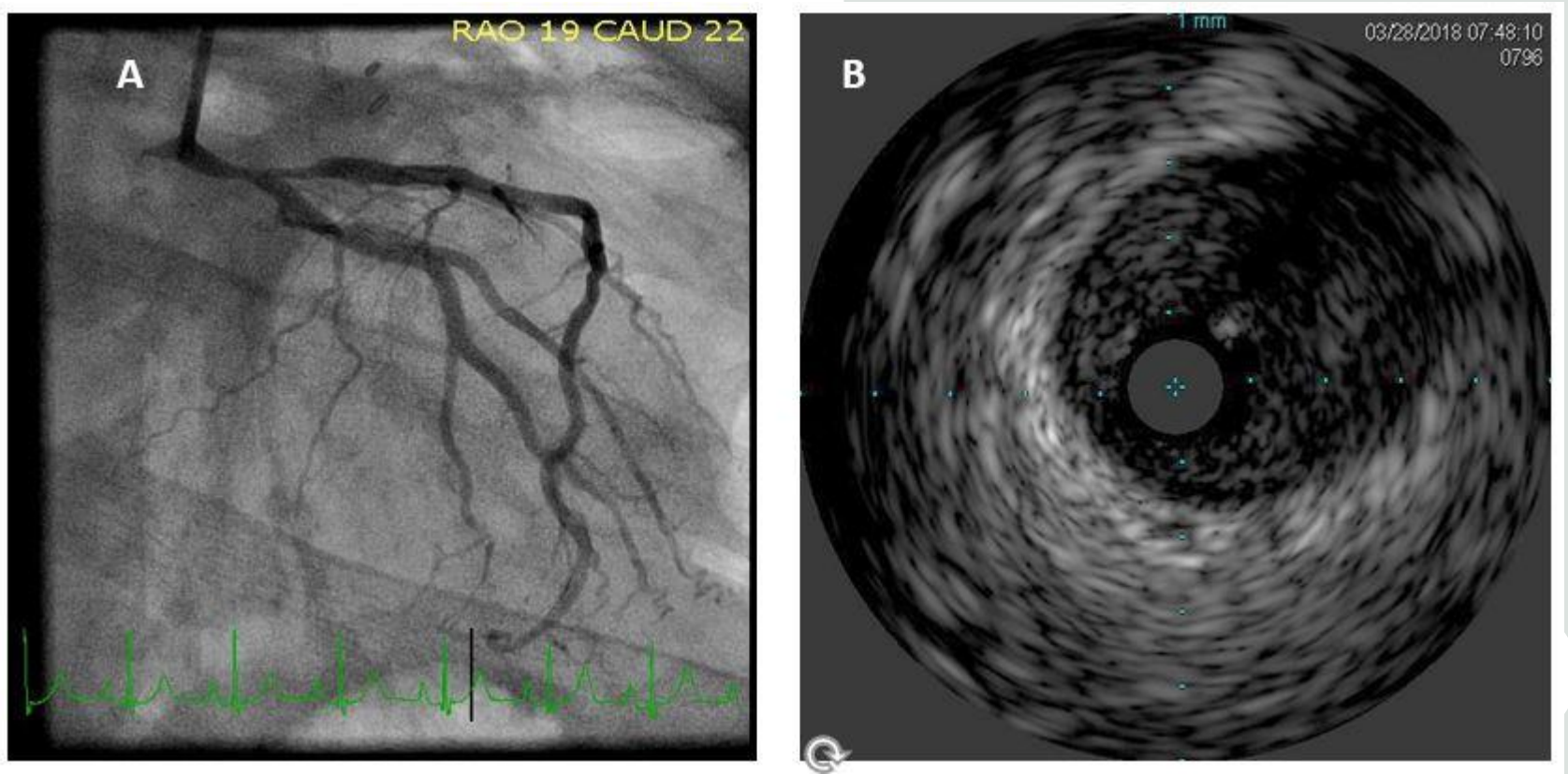


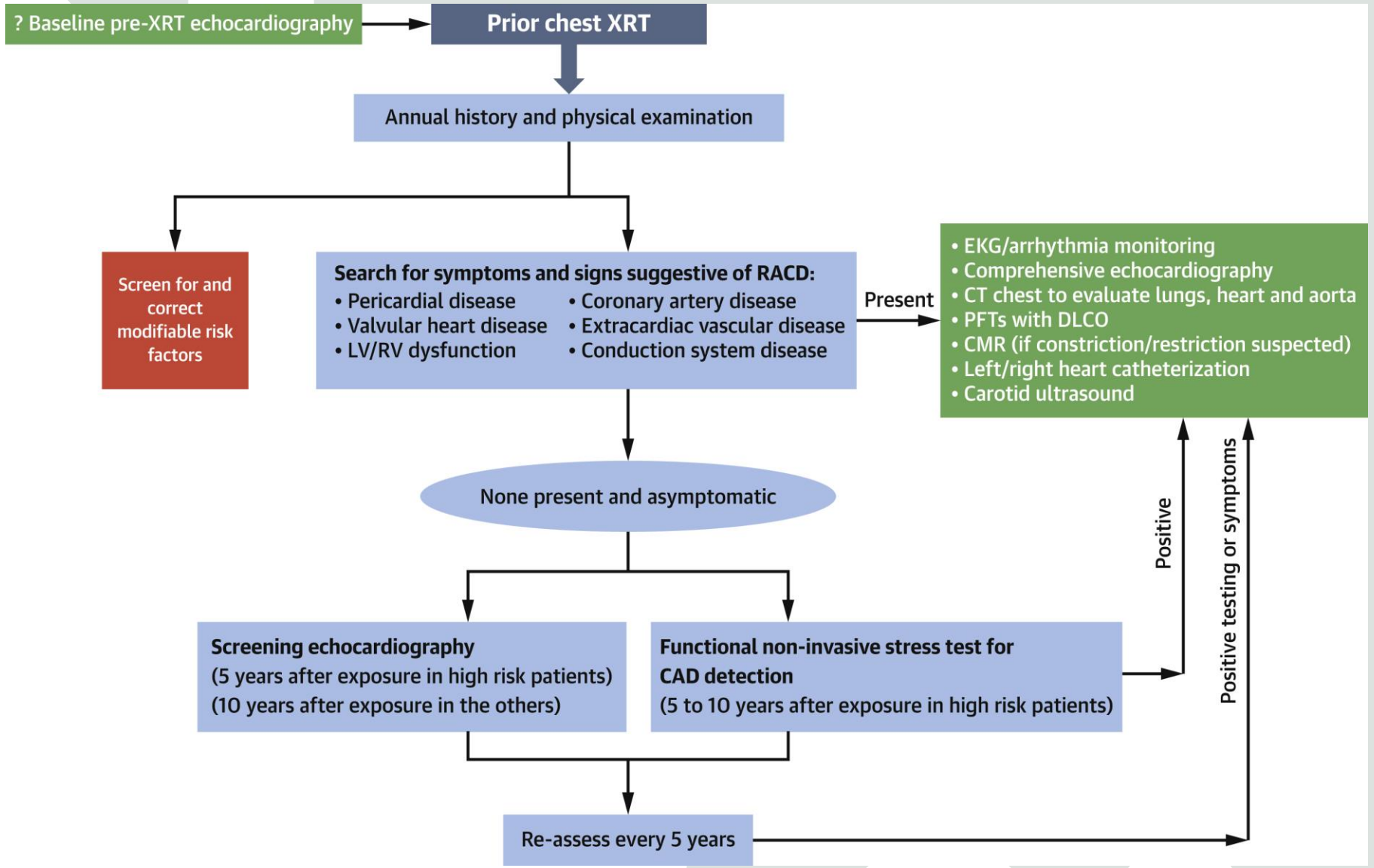
C

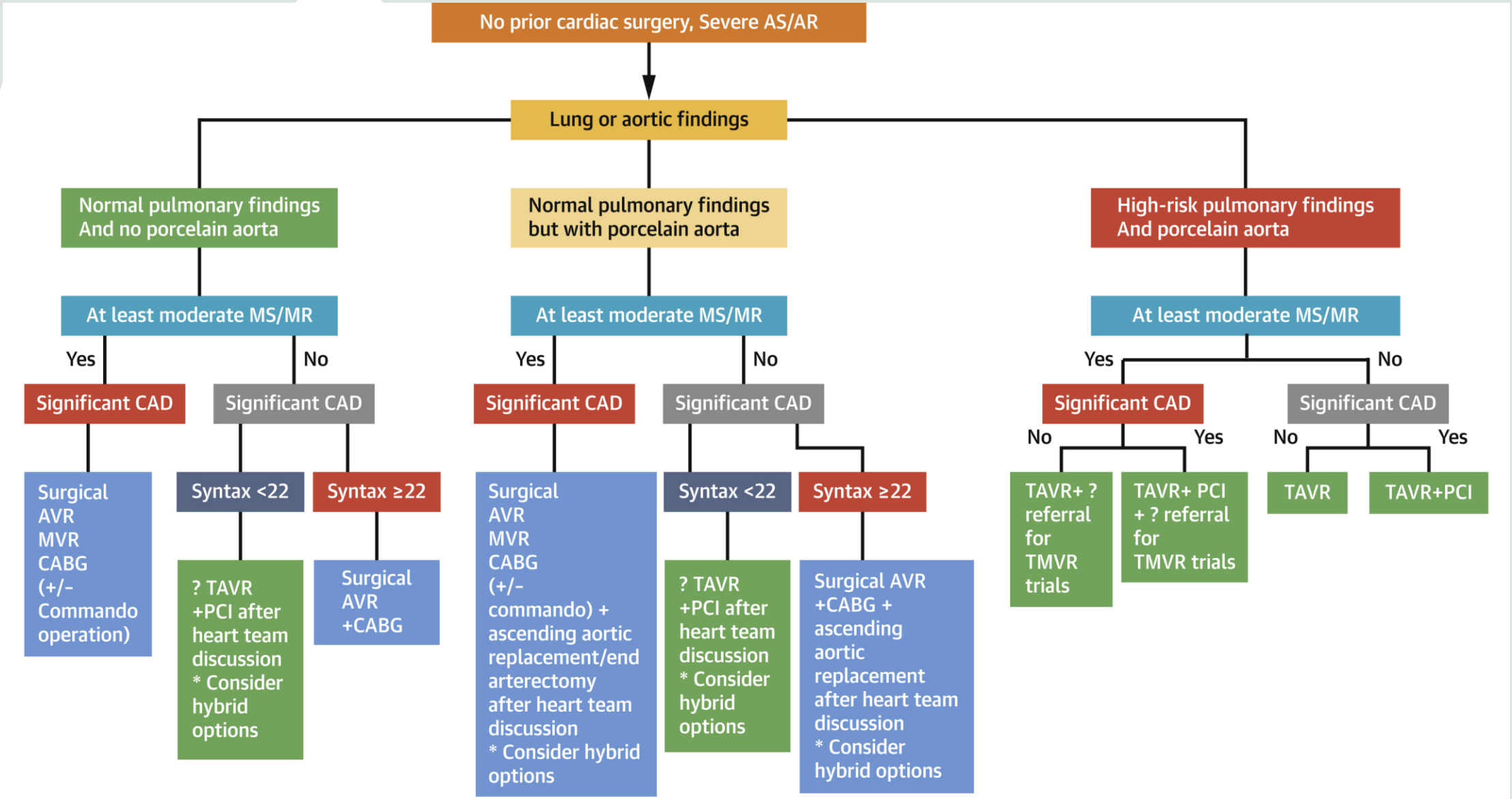


Example

74 yr old , hx of lymphoma 30 years ago treated with chemo and radiation comes with exertional chest pain. Echo normal.




? Baseline pre-XRT echocardiography
Prior chest XRT
Annual history and physical examination
Screen for and correct modifiable risk factors
Search for symptoms and signs suggestive of RACD:
• Pericardial disease
• Valvular heart disease
• LV/RV dysfunction
• Coronary artery disease
• Extracardiac vascular disease
• Conduction system disease
Present
• EKG/arrhythmia monitoring
• Comprehensive echocardiography
• CT chest to evaluate lungs, heart and aorta
• PFTs with DLCO
• CMR (if constriction/restriction suspected)
• Left/right heart catheterization
• Carotid ultrasound
None present and asymptomatic
Screening echocardiography
(5 years after exposure in high risk patients)
(10 years after exposure in the others)
Functional non-invasive stress test for CAD detection
(5 to 10 years after exposure in high risk patients)
Positive
Positive testing or symptoms
Re-assess every 5 years



All evaluation and management should be performed at an experienced center with a heart team of cardiologists and cardiac surgeons experienced in management of RACD. Many treatment decisions might have to be individualized, especially in the setting of cardiac reoperation.

Thank you