

CARDIO-ONCOLOGY

Relevant Guidelines

ESC 2022 Guidelines on cardio-oncology

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). *Eur Heart J*. 2022.

Relevant Scientific Statements

AHA 2022 Statement on cardio-oncology

Cardio-Oncology Drug Interactions: A Scientific Statement From the American Heart Association. *Circulation*. 2022:CIR0000000000001056

CANCER THERAPY-RELATED CARDIOVASCULAR TOXICITY

General Principles

Several anticancer therapies (including chemotherapeutic agents and radiotherapy) may affect the heart through direct cardiotoxic effects or, indirectly, through other mechanisms, such as ischaemia, systemic or pulmonary hypertension, arrhythmias or valve disease.¹⁻⁵ A genetic background may also be involved.⁶ Beyond the development of asymptomatic or symptomatic LV dysfunction, the spectrum of CV complications of modern cancer therapy may include acute coronary syndromes, thrombo-embolic events, hypertension, QTc prolongation and arrhythmias, myocarditis, pericarditis, and metabolic disorders.⁵

Chemotherapy-induced ventricular arrhythmias, including sustained ventricular tachycardia and fibrillation, are relatively rare. The incidence is higher in patients receiving chemotherapy for advanced metastatic disease but can also be attributable to the cancer itself.^{4, 7} Figures 1-3 and Table 1 provide definitions and Tables 2 and 3 risk stratification parameters. Specific recommendations for patients with known comorbidities are also provided in relevant Chapters.

Cardiovascular toxicity risk stratification before anticancer therapy

Recommendations are provided in Figures 4 and 5, and Tables 4-6

References

1. 2021 ESC Guidelines for the diagnosis and treatment of acute and chronic heart failure. *Eur Heart J*. 2021.
2. Herrmann J, et al. Defining cardiovascular toxicities of cancer therapies: an International Cardio-Oncology Society (IC-OS) consensus statement. *Eur Heart J*. 2022;43:280-299.
3. Beavers CJ, et al. Cardio-Oncology Drug Interactions: A Scientific Statement From the American Heart Association. *Circulation*. 2022:CIR0000000000001056.
4. Lyon AR, et al. 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). *Eur Heart J*. 2022.
5. Fabiani I, et al. Use of new and emerging cancer drugs: what the cardiologist needs to know. *Eur Heart J*. 2024;45:1971-1987.
6. Garcia-Pavia P, et al. Genetic Variants Associated With Cancer Therapy-Induced Cardiomyopathy. *Circulation*. 2019;140:31-41.
7. Fradley MG, et al. Recognition, Prevention, and Management of Arrhythmias and Autonomic Disorders in Cardio-Oncology: A Scientific Statement From the American Heart Association. *Circulation*. 2021;144:e41-e55.

Table 1. ESC 2022 GL on cardio-oncology. Cancer therapy-related cardiovascular toxicity definitions

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	
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		
Vascular toxicity (for general cardiology definitions, see Supplementary data, Table S1)			
Asymptomatic vascular toxicity	CAD	Symptomatic vascular toxicity	Stroke
	PAD		Transient ischaemic attack
	Carotid artery disease		MI
	Venous thrombosis		ACS
	Arterial thrombosis		CCS
	Peripheral vasoreactivity		PAD

	Coronary epicardial vasoreactivity		Vasospastic angina
	Coronary microvascular vasoreactivity		Microvascular angina
			Raynaud's phenomenon
Arterial hypertension			
Treatment threshold for hypertension before, during, and after therapy	In patients with high CV risk ^d : ≥130 mmHg systolic and/or ≥80 mmHg diastolic		
	Otherwise: ≥140 mmHg systolic and/or ≥90 mmHg diastolic		
Cancer therapy holding threshold	≥180 mmHg systolic and/or ≥110 mmHg diastolic		
Hypertensive emergency	(Very high) BP elevation associated with acute hypertension-mediated organ damage (heart, retina, brain, kidneys, and large arteries), requiring immediate BP reduction to limit extension or promote regression of target organ damage		
Cardiac arrhythmias			
QT prolongation	Prolonged: QTcF > 500 ms ^j		
Bradycardia	For general cardiology definitions, see Supplementary data, Table S1		
Supraventricular tachycardia			
Ventricular arrhythmias			
AF			

ACS, acute coronary syndromes; AF, atrial fibrillation; BNP, B-type natriuretic peptide; BP, blood pressure; CAD, coronary artery disease; CCS, chronic coronary syndromes; CMR, cardiac magnetic resonance; cTn, cardiac troponin; CTRCD, cancer therapy-related cardiac dysfunction; CV, cardiovascular; ECV, extracellular volume fraction; EMB, endomyocardial biopsy; ESC, European Society of Cardiology; GLS, global longitudinal strain; HF, heart failure; HFmrEF, HF with mildly reduced ejection fraction; HFpEF, heart failure with preserved ejection fraction; HFrEF, heart failure with reduced ejection fraction; ICI, immune checkpoint inhibitors; LGE, late gadolinium enhancement; LV, left ventricular; LVEF, left ventricular ejection fraction; MI, myocardial infarction; NT-proBNP, N-terminal pro-B-type natriuretic peptide; PAD, peripheral artery disease; QTcF, corrected QT interval using Fridericia correction; SCORE2, Systematic Coronary Risk Estimation 2; SCORE2-OP, Systematic Coronary Risk Estimation 2—Older Persons.

a With LVEF and supportive diagnostic biomarkers based on the 2021 ESC Guidelines for the diagnosis and treatment of acute and chronic HF.

b Symptomatic CTRCD represents HF, which is a clinical syndrome consisting of cardinal symptoms (e.g. breathlessness, ankle swelling, and fatigue) that may be accompanied by signs (e.g. elevated jugular venous pressure, pulmonary crackles, and peripheral oedema) and has traditionally been divided into distinct phenotypes based on the measurement of LVEF: $\leq 40\%$ =HFrEF; $41\text{--}49\%$ =HFmrEF; $\geq 50\%$ =HFpEF.

c cTnI/cTnT.99th percentile, BNP ≥ 35 pg/mL, NT-proBNP ≥ 125 pg/mL or new significant rise from baseline beyond the biological and analytical variation of the assay used.

d Clinical diagnoses should be confirmed with magnetic resonance imaging or EMB if possible and without causing treatment delays. Treatment with immunosuppression should be promptly initiated while awaiting further confirmatory testing in symptomatic patients.

e Both troponin I and troponin T can be used; however, clinical observations suggest that troponin T may be falsely elevated in patients with concomitant myositis and without myocarditis.

f According to local protocols.

g Diagnostic CMR: Based on updated Lake Louise criteria: T2-based criterion+T1-based criterion+supportive criteria (T2-based criteria: regional or global increase of native T2, or T2 signal intensity; T1-based criteria: regional or global increase of native T1, or regional or global increase in the ECV, or presence of LGE; supportive criteria: pericarditis and/or regional or global LV systolic dysfunction).

h Suggestive CMR: meeting some but not all of the modified Lake Louise criteria. The presence of T2- or T1-based criteria may support a diagnosis of acute myocardial inflammation in the appropriate clinical scenario.

i SCORE2 (<70 years), SCORE2-OP (≥ 70 years) or equivalent. CV risk stratification: <50 years: low risk, 2.5%, moderate risk 2.5% to 7.5%, high risk $\geq 7.5\%$; 50–69 years: low risk, 5%; moderate risk 5% to 10%; high risk $\geq 10\%$; ≥ 70 years: low risk, 7.5%, moderate risk 7.5% to 15%, high risk $\geq 15\%$.

j QTcF 480–500 ms: correct reversible causes, minimize other QT prolonging medications, close QTcF monitoring. Fridericia correction is recommended ($QTcF = QT/3\sqrt{RR}$).

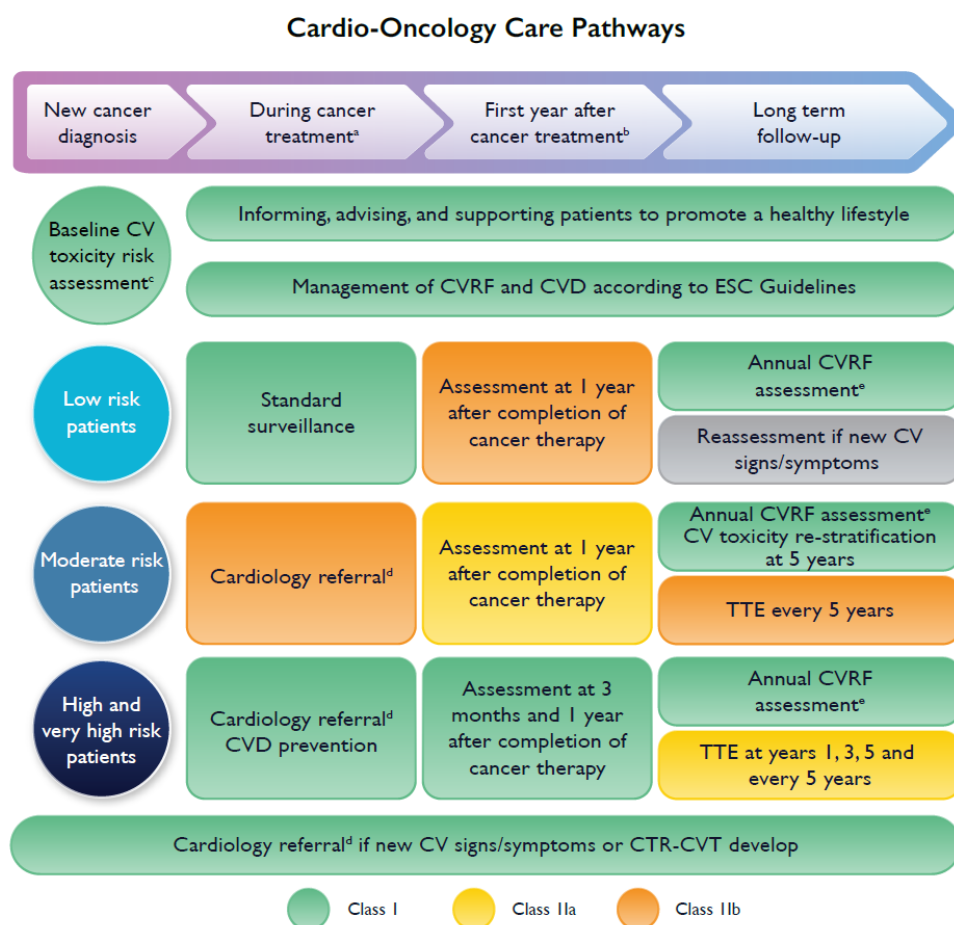


Figure 1. ESC 2022 GL on cardio-oncology. Cardio-oncology care pathways.

BP, blood pressure; CS, cancer survivors; CTR-CVT, cancer therapy-related cardiovascular toxicity; CV, cardiovascular; CVD, cardiovascular disease; CVRF, cardiovascular risk factors; ECG, electrocardiogram; ESC, European Society of Cardiology; HbA1c, glycated haemoglobin; HFA, Heart Failure Association; ICOS, International Cardio-Oncology Society; NP, natriuretic peptides; RT, radiotherapy; TTE, transthoracic echocardiography.

a CV surveillance according to baseline CV toxicity risk, type of cancer, cancer stage, and cancer therapy.

b CTR-CVT risk assessment is recommended during the first year after cardiotoxic cancer treatment to establish a long-term follow-up care plan.

c The use of HFA-ICOS risk assessment tools should be considered to assess CTR-CVT risk in patients with cancer scheduled to receive cardiotoxic anticancer therapy. Clinical assessment and ECG are recommended at baseline in all patients with cancer and echocardiography, cardiac biomarkers, or other cardiac imaging tests in selected patients according to baseline CV toxicity risk and cancer treatment type.

d Cardio-oncology referral is recommended when available, alternatively patients should be referred to a specialized cardiologist with expertise in managing CVD in patients with cancer.

e Annual CV risk assessment (including clinical review, BP, lipid profile, HbA1c, ECG, and NP) and CVRF management is recommended in CS who were treated with a potentially cardiotoxic cancer drug or RT to a volume exposing the heart.

Baseline CV toxicity risk assessment checklist

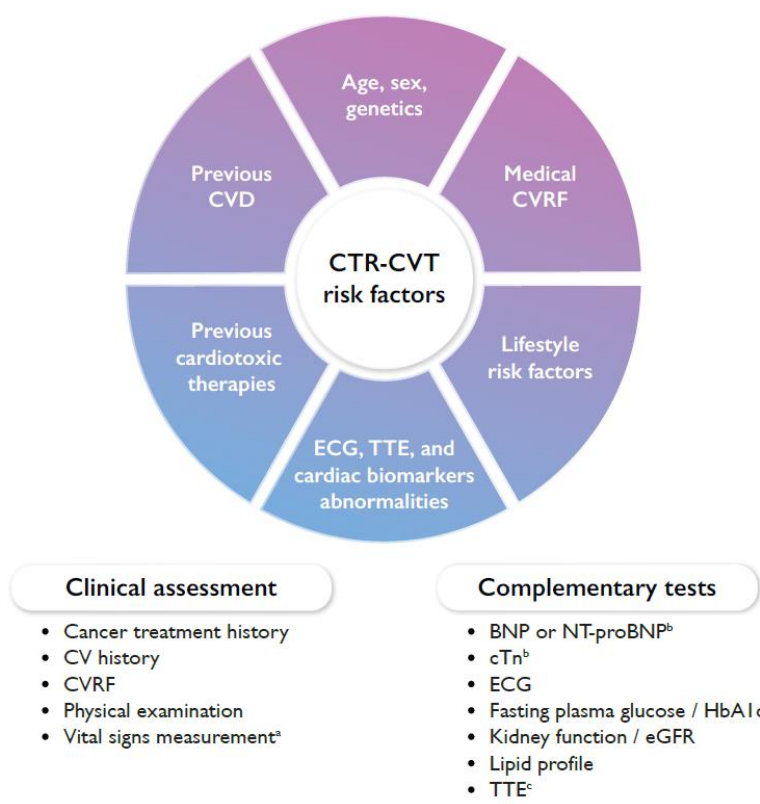


Figure 2. ESC 2022 GL on cardio-oncology. Baseline cardiovascular toxicity risk assessment checklist.

BNP, B-type natriuretic peptide; cTn, cardiac troponin; CTR-CVT, cancer therapy-related cardiovascular toxicity; CV, cardiovascular; CVD, CV disease; CVRF, cardiovascular risk factors; ECG, electrocardiogram; eGFR, estimated glomerular filtration rate; HbA1c, glycated haemoglobin; NT-proBNP, N-terminal pro-BNP; TTE, transthoracic echocardiography.

a Including blood pressure, heart rate, height, weight, and body mass index.

b Cardiac biomarkers (troponin and NP) should be measured in patients at risk of CTRCD where available and results should be interpreted according to the patient clinical status, type of cancer treatment, and kidney function.

c Consider other CV complementary tests in selected patients: cardiac magnetic resonance, coronary computed tomography angiography, CPET (in selected patients for pre-operative [lung, colon, and rectal cancers] risk stratification).

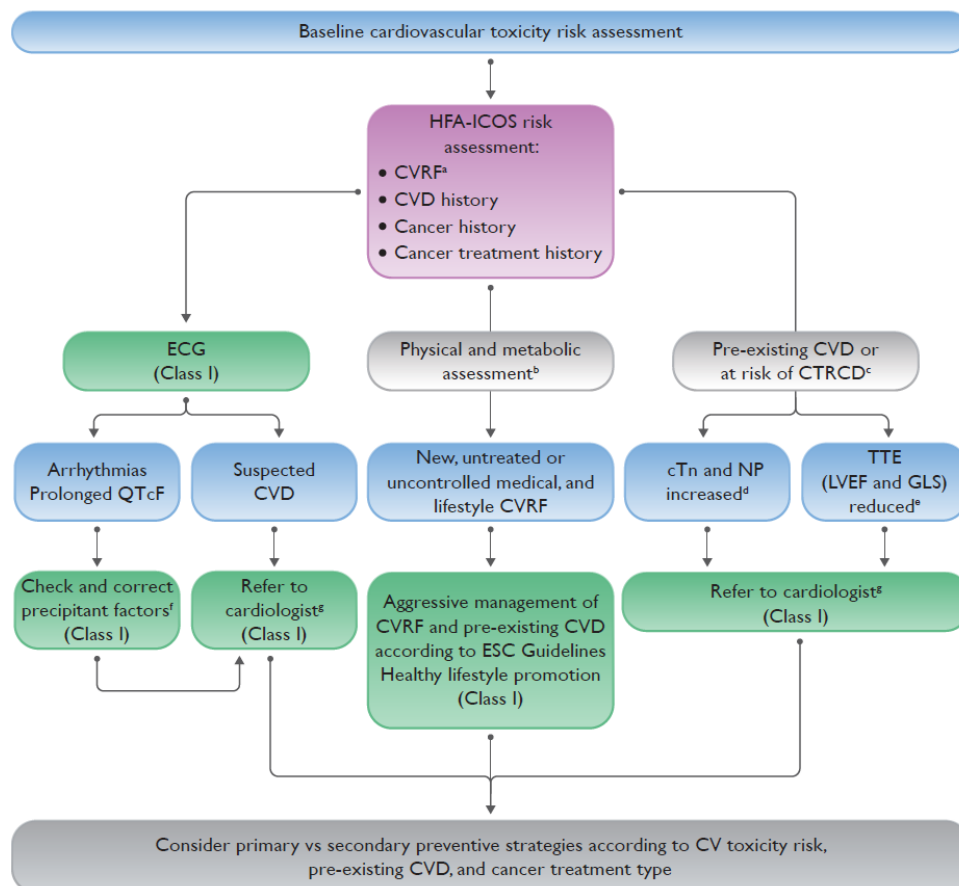


Figure 3. ESC 2022 GL on cardio-oncology. Baseline cardiovascular toxicity risk assessment before anticancer therapy.

BNP, B-type natriuretic peptide; cTn, cardiac troponin; CTRCD, cancer therapy-related cardiac dysfunction; CV, cardiovascular; CVD, CV disease; CVRF, CV risk factors; ECG, electrocardiogram; ESC, European Society of Cardiology; GLS, global longitudinal strain; HFA, Heart Failure Association; ICOS, International Cardio-Oncology Society; LVEF, left ventricular ejection fraction; NP, natriuretic peptides (including BNP and NT-proBNP); NT-proBNP, N-terminal pro-BNP peptide; QTc, corrected QT interval; QTcF, corrected QT interval using Fridericia correction; TTE, transthoracic echocardiography.

^a When assessing CVRF, include information

about unhealthy lifestyle including sedentary behaviour, smoking, and alcohol intake.

^b See Figure 2

^c According to cancer treatment and HFA-ICOS risk assessment.

^d cTnI/T.99th percentile, BNP $\geq$ 35 pg/mL, NT-proBNP $\geq$ 125 pg/mL.

^e Patients with baseline LVEF $\geq$ 50% or in the low normal range (LVEF 50–54%) should be referred to a specialized cardiologist or cardio-oncologist. When TTE is used, ideally three-dimensional-LVEF and GLS should be measured. If GLS assessment is not available, other markers of longitudinal function (e.g. annular Doppler velocity) should be considered.

Cardiac magnetic resonance should be considered if echocardiography is of non-diagnostic quality.

^f Anaemia, infections, electrolyte abnormalities, metabolic problems, other QTc-prolonging drugs.

^g Cardio-oncology referral is recommended when available; alternatively, patients should be referred to a specialized cardiologist with expertise in managing CVD in patients with cancer.

Table 2. ESC 2022 GL on cardio-oncology. 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
Previous CVD						
HF/cardiomyopathy/CTRCD	VH	VH	VH	H	VH	VH
Severe VHD	H	H	–	–	–	H
MI or PCI or CABG	H	H	VH	–	–	H
Stable angina	H	H	VH	–	–	H
Arterial vascular disease	–	–	VH	VH	VH	–
Abnormal ankle-brachial pressure index	–	–	–	H	–	–
PH	–	–	–	H	–	–
Arterial thrombosis with TKI	–	–	–	VH	–	–
Venous thrombosis (DVT/PE)	–	–	H	M2	VH	–
Arrhythmia ^a	–	M2	M2	M2	M2	M1
QTc $\geq$ 480 ms	–	–	H	H	–	–
450 $\leq$ QTc < 480 ms (men); 460 $\leq$ QTc < 480 ms (women)	–	–	M2	M2	–	–
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
Age and CVRF						
Age $\geq$ 80 years	H	H	–	–	–	M1
Age 65–79 years	M2	M2	–	–	–	M1
Age $\geq$ 75 years	–	–	H	H	H	M1
Age 65–74 years	–	–	M1	M2	M1	M1
Age $\geq$ 60 years	–	–	–	M1	–	–
CVD 10-year risk score > 20%	–	–	–	H	–	–
Hypertension ^c	M1	M1	H	M2	M1	M2
Chronic kidney disease ^d	M1	M1	M1	M1	M1	M1
Proteinuria	–	–	M1	–	–	–
DM ^e	M1	M1	M1	M1	M1	M1
Hyperlipidaemia ^f	–	–	M1	M1	M1	–
Family history of thrombophilia	–	–	–	M1	M1	–

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
Non-anthracycline chemotherapy	M1	–	–	–	–	–
Lifestyle risk factors						
Current smoker or significant smoking history	M1	M1	M1	H	M1	M1
Obesity (BMI > 30 kg/m ²)	M1	M1	M1	M1	M1	M1

AF, atrial fibrillation; BCR-ABL, breakpoint cluster region–Abelson oncogene locus; BMI, body mass index; BNP, B-type natriuretic peptide; BP, blood pressure; CABG, coronary artery bypass graft; cTn, cardiac troponin; CTRCD, cancer therapy-related cardiac dysfunction; CV, cardiovascular; CVD, cardiovascular disease; CVRF, cardiovascular risk factors; DM, diabetes mellitus; DVT, deep vein thrombosis; eGFR, estimated glomerular filtration rate; H, high risk; HbA1c, glycated haemoglobin; HER2, human epidermal receptor 2; HF, heart failure; IMiD, immunomodulatory drugs; LV, left ventricular; LVEF, left ventricular ejection fraction; M, moderate risk; MEK, mitogen-activated extracellular signal-regulated kinase; MI, myocardial infarction; MM, multiple myeloma; NP, natriuretic peptides (including BNP and NT-proBNP); NT-proBNP, N-terminal pro-B-type natriuretic peptide; PCI, percutaneous coronary intervention; PE, pulmonary embolism; PH, pulmonary hypertension; PI, proteasome inhibitors; QTc, corrected QT interval; RAF, rapidly accelerated fibrosarcoma; RT, radiotherapy; TKI, tyrosine kinase inhibitors; ULN, upper limit of normal; VEGFi, vascular endothelial growth factor inhibitors; VH, very high risk; VHD, valvular heart disease. Risk level: Low risk=no risk factors OR one moderate risk factor; moderate risk (M)=moderate risk factors with a total of 2–4 points (Moderate 1 [M1]=1 point; Moderate 2 [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.

a AF, atrial flutter, ventricular tachycardia, or ventricular fibrillation.

b Elevated above the ULN of the local laboratory reference range.

c Systolic BP.140 mmHg or diastolic BP.90 mmHg, or on treatment.

d eGFR,60 mL/min/1.73 m².

e HbA1c.7.0% or .53 mmol/mol, or on treatment.

f Non-high density lipoprotein cholesterol .3.8 mmol/L (.145 mg/dL) or on treatment.

g High risk if anthracycline chemotherapy and trastuzumab delivered concurrently.

h Previous malignancy (not current treatment protocol).

Table 3. ESC 2022 GL on cardio-oncology. Recommendations for a general approach to cardiovascular toxicity risk categorization

CV toxicity risk stratification ^a before starting potentially cardiotoxic anticancer therapy in all patients with cancer.	I B
Communicate the results of the CV toxicity risk assessment to the patient and other appropriate healthcare professionals	I C
Use of HFA-ICOS risk assessment to stratify CV toxicity risk in patients with cancer scheduled to receive cardiotoxic anticancer therapy.	IIa C
Patients categorized to be at low CV toxicity risk should proceed to anticancer therapy without delay.	I C
Cardiology referral ^b of patients categorized at moderate CV toxicity risk, cardiology referral ^d may be considered. ^c	IIb C
Cardiology referral ^b of high-risk and very high-risk patients before anticancer therapy. ^d	I C
Discussion of the risk/benefit balance of cardiotoxic anticancer treatment in high- and very high-risk patients in a multidisciplinary approach prior to starting treatment is recommended.	I C
Cardiology referral ^e of patients with cancer and pre-existing CVD or abnormal findings at baseline CV toxicity risk assessment who require potentially cardiotoxic anticancer therapy.	I C

CV, cardiovascular; CVD, CV disease; ECG, electrocardiogram; GLS, global longitudinal strain; HbA1c, glycated haemoglobin; HFA, Heart Failure Association; ICOS, International Cardio-Oncology Society; LVEF, left ventricular ejection fraction; TTE, transthoracic echocardiography; ULN, upper limit of normal; VHD, valvular heart disease.

a Including clinical history and physical examination, ECG, general blood test, HbA1c, lipid profile, and cardiac serum biomarkers and/or TTE (according to cancer drug type and CV toxicity risk).

b Cardio-oncology referral is recommended when available; alternatively, the patients should be referred to a specialized cardiologist with expertise in managing CVD in patients with cancer.

c Without delaying cancer treatments.

d Unless there is an oncology emergency requiring immediate cancer treatment.

e Moderate-to-severe pre-existing CVDs or new abnormal findings (baseline cardiac serum biomarkers. ULN, LVEF ≤ 50%, GLS under normal local values, previously undiagnosed moderate-to-severe myocardial, pericardial, or VHDs, abnormal baseline ECG).

Table 4. ESC 2022 GL on cardio-oncology. Recommendations for electrocardiogram baseline assessment

ECG in all patients starting cancer therapy as part of their baseline CV risk assessment. I C

In patients with an abnormal baseline ECG,^a referral to a cardiologist^b I C

AF, atrial fibrillation; CV, cardiovascular; ECG, electrocardiogram; LV, left ventricular; QTc, corrected QT interval; QTcF, corrected QT interval using Fridericia correction.

a Advanced conduction disease (left bundle branch block, right bundle branch block, second degree heart block, severe first degree heart block with a PR interval >300 ms); Q waves in two or more contiguous leads; LV hypertrophy; AF/atrial flutter if previously undiagnosed; QTc prolongation using Fridericia correction formula ($QTcF = QT/3\sqrt{RR}$) >450 ms for men and >460 ms for women or other ECG abnormality raising concern.

b Cardio-oncology referral is recommended when available; alternatively, the patients should be referred to a specialized cardiologist with expertise in managing CVD in patients with cancer.

c Recommendation for cardiac biomarker assessment prior to potentially cardiotoxic therapies

Baseline measurement of NP^a and/or cTn^b in all patients with cancer at risk of CTRCD if these biomarkers are going to be measured during treatment to detect CTRCD.^c I C

cTn, cardiac troponin; CTRCD, cancer therapy-related cardiac dysfunction; NP, natriuretic peptides.

a NPs including B-type natriuretic peptide or N-terminal pro-B-type natriuretic peptide.

b cTn includes any of troponin I, troponin T, or hs-cTnT.

c For specific recommendations for baseline cardiac biomarkers in patients with cancer at low, moderate, high, and very high risk of cancer therapy-related cardiovascular toxicity see text.

Table 5. ESC 2022 GL on cardio-oncology. Recommendation for cardiac biomarker assessment prior to potentially cardiotoxic therapies

Baseline measurement of NP^a and/or cTn^b in all patients with cancer at risk of CTRCD if these biomarkers are going to be measured during treatment to detect CTRCD.^c I C

cTn, cardiac troponin; CTRCD, cancer therapy-related cardiac dysfunction; NP, natriuretic peptides.

a NPs including B-type natriuretic peptide or N-terminal pro-B-type natriuretic peptide.

b cTn includes any of troponin I, troponin T, or hs-cTnT.

c Specific recommendations for baseline cardiac biomarkers in patients with cancer at low, moderate, high, and very high risk of cancer therapy-related cardiovascular toxicity are included below.

Table 6. ESC 2022 GL on cardio-oncology. Recommendations for cardiac imaging modalities in patients with cancer

General

Echocardiography as the first-line modality for the assessment of cardiac function	I C
3D echocardiography as the preferred echocardiographic modality to measure LVEF	I B
GLS for all patients with cancer having echocardiography, if available	I C
CMR for the assessment of cardiac function when echocardiography is unavailable or non-diagnostic	IIa C
MUGA when TTE is not diagnostic and CMR is not available	IIb C

Baseline cardiac imaging prior to potentially cardiotoxic therapies

Baseline comprehensive TTE in all patients with cancer at high risk and very high risk of CV toxicity before starting anticancer therapy.^a

3D, three-dimensional; CMR, cardiac magnetic resonance; CTR-CVT, cancer therapy-related CV toxicity; CV, cardiovascular; GLS, global longitudinal strain; LVEF, left ventricular ejection fraction; MUGA, multigated acquisition nuclear imaging; TTE, transthoracic echocardiography.

^a Except asymptomatic patients referred to breakpoint cluster region-Abelson oncogene locus therapy (BCR-ABL) where baseline TTE should be considered

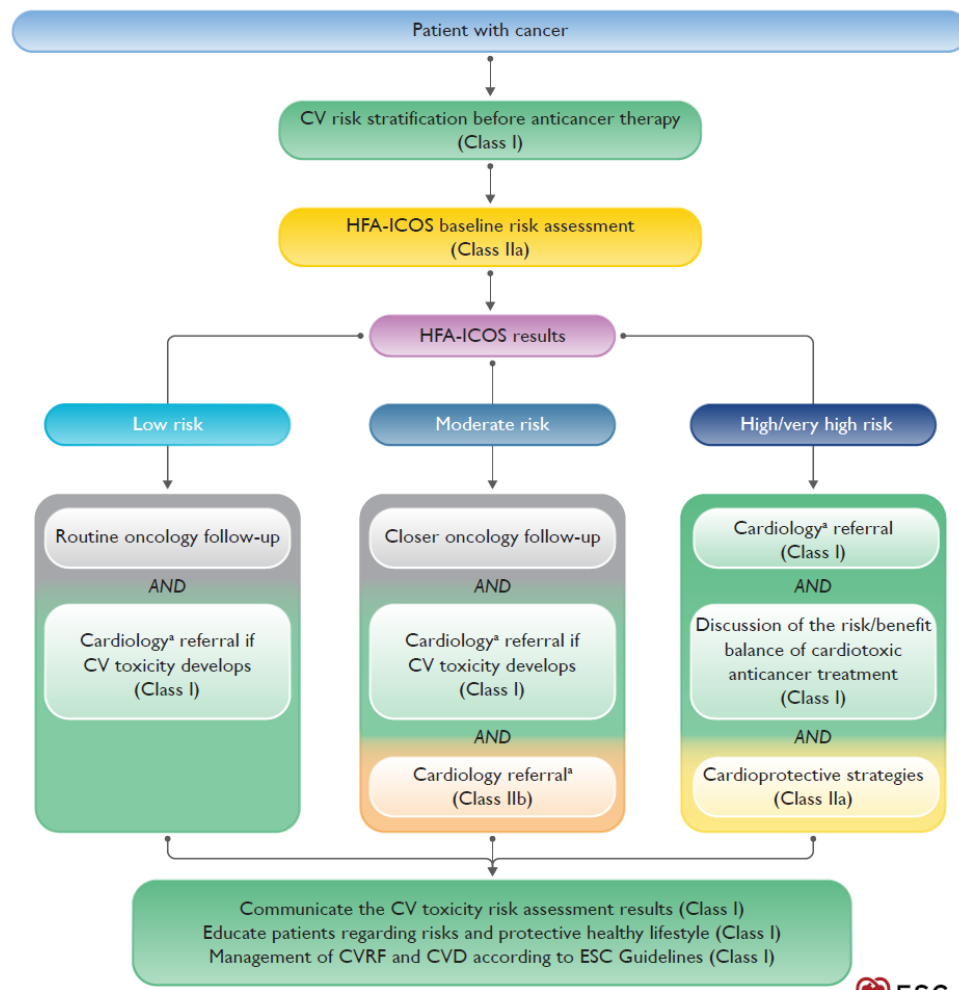


Figure 4. ESC 2022 GL on cardio-oncology. General cardio-oncology approach after Heart Failure Association–International Cardio-Oncology Society cardiovascular toxicity risk assessment.

CV, cardiovascular; CVD, CV disease; CVRF, CV risk factors; ESC, European Society of Cardiology; HFA, Heart Failure Association; ICOS, International Cardio-Oncology Society.

^a Cardio-oncology referral is recommended when available; alternatively, patients should be referred to a specialized cardiologist with expertise in managing CVD in patients with cancer.

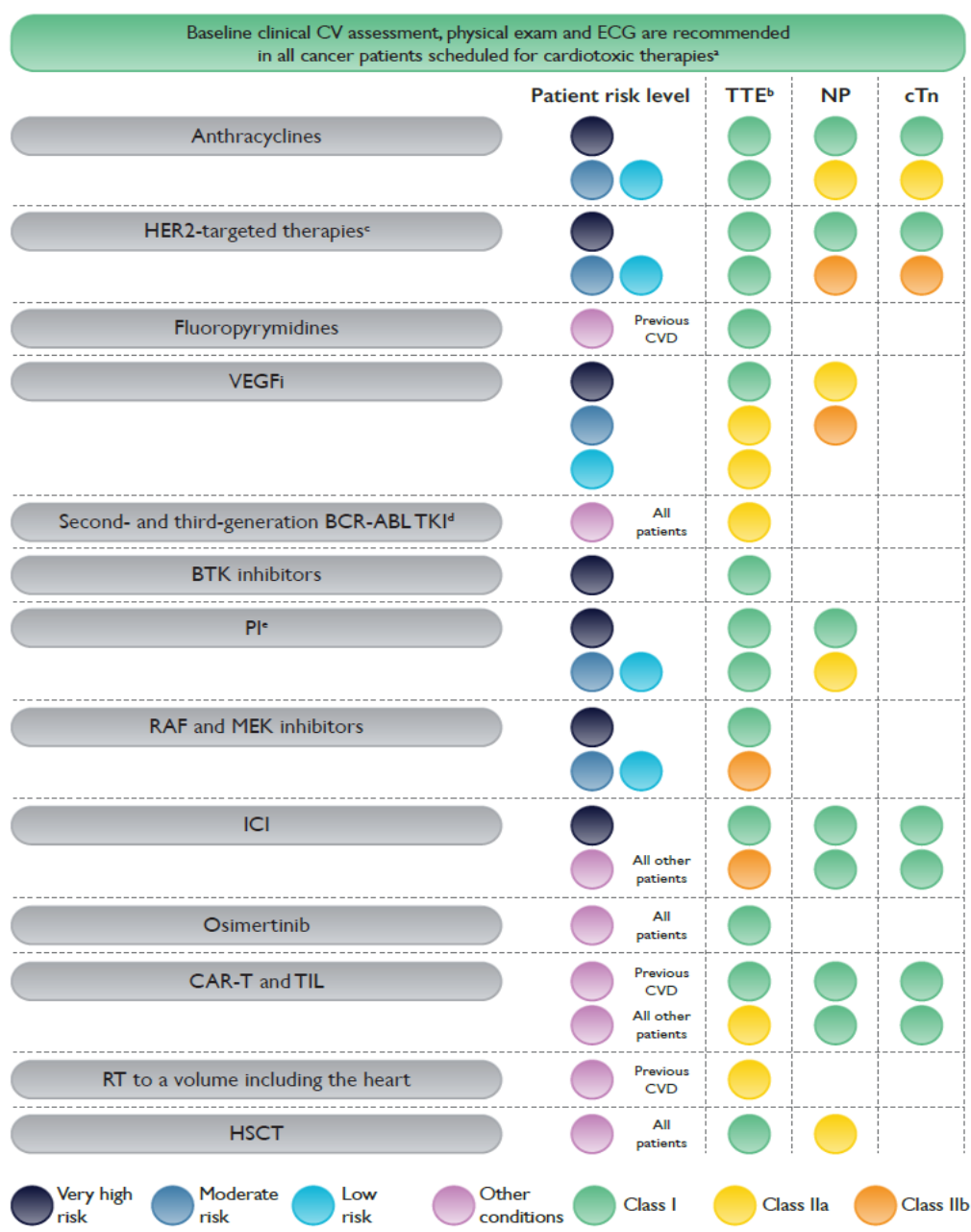


Figure 5. ESC 2022 GL on cardio-oncology. Baseline screening recommendations for patients with cancer treated with potentially cardiotoxic drugs.

3D, three-dimensional; ADT, androgen deprivation therapy; AL-CA, amyloid light-chain cardiac amyloidosis; BC, breast cancer; BCR-ABL, breakpoint cluster region-Abelson oncogene locus; BNP, B-type natriuretic peptide; BTK, Bruton tyrosine kinase; CAR-T, chimeric antigen receptor T cell; CDK, cyclin-dependent kinase; CMR, cardiac magnetic resonance; cTn, cardiac troponin; CV, cardiovascular; CVD, cardiovascular disease; ECG, electrocardiogram; GLS, global longitudinal strain; HER2, human epidermal receptor 2; HSCT, haematopoietic stem cell transplantation; ICI, immune checkpoint inhibitors; LVEF, left ventricular ejection fraction; MEK, mitogen-activated extracellular signal-regulated kinase; NP, natriuretic peptides (including BNP and NT-proBNP); NT-proBNP, N-terminal pro-B-type natriuretic peptide; PI, proteasome inhibitors; RAF, rapidly accelerated fibrosarcoma; RT, radiotherapy; TIL, tumour-infiltrating lymphocytes; TKI, tyrosine kinase inhibitors; TTE, transthoracic echocardiography; VEGFi, vascular endothelial growth factor inhibitors.

- a Including patients scheduled to receive ADT for prostate cancer, CDK 4/6 inhibitors, endocrine hormone therapy for BC and anaplastic lymphoma kinase inhibitors.
- b TTE is recommended as the first-line modality for the assessment of cardiac function. 3D echocardiography is recommended to measure LVEF. GLS is recommended in all patients with cancer having echocardiography, if available. CMR should be considered when echocardiography is unavailable or not diagnostic.
- c Baseline cTn measurement should be considered (Class IIa, Level A) in low- and moderate-risk patients post-anthracycline chemotherapy but prior to starting HER2-targeted therapies. Baseline NP and cTn measurement may be considered (Class IIb, Level C) in low- and moderate-risk patients.
- d Baseline echocardiography is recommended in patients scheduled to receive dasatinib (Class I, Level C).
- e NP and cTn measurements are recommended at baseline in patients with AL-CA (Class I, Level B).