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CLINICAL HEART FAILURE GUIDELINES: ARE THEY DIFFERENT FOR CANCER PATIENTS?

DIAGNOSIS AND TREATMENT

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INTRODUCTION

- **Myocardial dysfunction** and **heart failure** are considered to be the most concerning **cardiovascular complications** of cancer therapies

- **Cardiotoxicity** is defined as:

Symptomatic or asymptomatic LVEF reduction of more than 10% compared to baseline, with final LVEF < 53%

INTRODUCTION

Aetiologies of heart failure		
Ischaemic heart disease	Myocardial scar	
	Myocardial stunning/hibernation	
	Epicardial coronary artery disease	
	Abnormal coronary microcirculation	
	Endothelial dysfunction	
Toxic damage	Recreational substance abuse	Alcohol, cocaine, amphetamine, anabolic steroids.
	Heavy metals	Copper, iron, lead, cobalt.
	Medications	Cytostatic drugs (e.g. anthracyclines), immunomodulating drugs (e.g. interferons monoclonal antibodies such as trastuzumab, cetuximab), antidepressant drugs, antiarrhythmics, non-steroidal anti-Inflammatory drugs, anaesthetics.
	Radiation	
Immune-mediated and inflammatory damage	Related to infection	Bacteria, spirochaetes, fungi, protozoa, parasites (Chagas disease), rickettsiae, viruses (HIV/AIDS).
	Not related to infection	Lymphocytic/giant cell myocarditis, autoimmune diseases (e.g. Graves' disease, rheumatoid arthritis, connective tissue disorders, mainly systemic lupus erythematosus), hypersensitivity and eosinophilic myocarditis (Churg–Strauss).
Infiltration	Related to malignancy	Direct infiltrations and metastases.
	Not related to malignancy	Amyloidosis, sarcoidosis, haemochromatosis (iron), glycogen storage diseases (e.g. Pompe disease), lysosomal storage diseases (e.g. Fabry disease).
Metabolic derangements	Hormonal	Thyroid diseases, parathyroid diseases, acromegaly, GH deficiency, hypercortisolaemia, Conn's disease, Addison disease, diabetes, metabolic syndrome, phaeochromocytoma, pathologies related to pregnancy and peripartum.
	Nutritional	Deficiencies in thiamine, L-carnitine, selenium, iron, phosphates, calcium, complex malnutrition (e.g. malignancy, AIDS, anorexia nervosa), obesity.
Genetic abnormalities	Diverse forms	HCM, DCM, LV non-compaction, ARVC, restrictive cardiomyopathy (for details see respective expert documents), muscular dystrophies and laminopathies.

INTRODUCTION

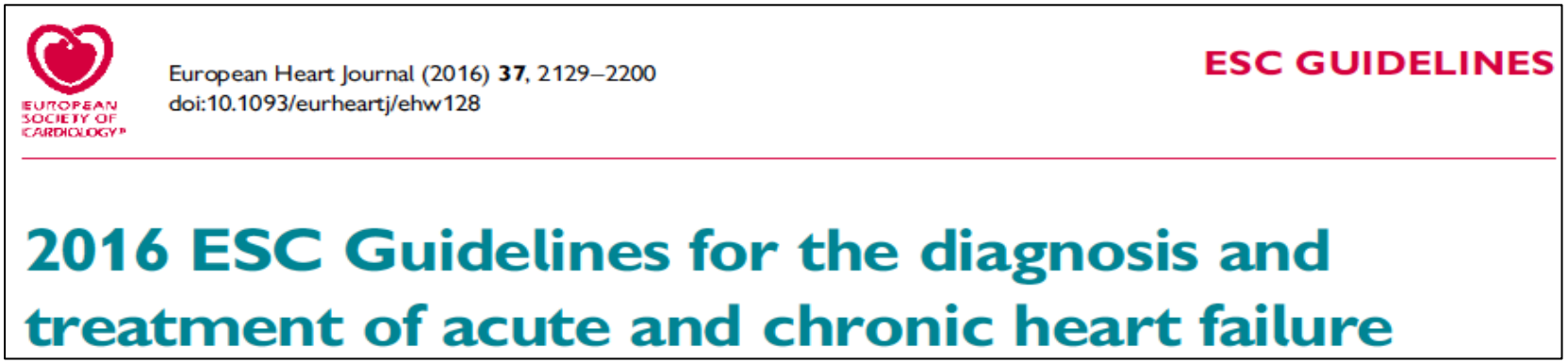
Anthracyclines are the major agent involved

Chemotherapy agents	Incidence (%)
Anthracyclines (dose dependent)	
Doxorubicin (Adriamycin) 400 mg/m² 550 mg/m² 700 mg/m²	 3–5 7–26 18–48
Idarubicin (>90 mg/m²)	5–18
Epirubicin (>900 mg/m²)	0.9–11.4
Mitoxanthone >120 mg/m²	2.6
Liposomal anthracyclines (>900 mg/m²)	2
Alkylating agents	
Cyclophosphamide	7–28
Ifosfamide <10 g/m² 12.5–16 g/m²	 0.5 17
Antimetabolites	
Clofarabine	27
Antimicrotubule agents	
Docetaxel	2.3–13
Paclitaxel	<1

Chemotherapy agents	Incidence (%)
Monoclonal antibodies	
Trastuzumab	1.7–20.1[28a]
Bevacizumab	1.6–4[14b]
Pertuzumab	0.7–1.2
Small molecule tyrosine kinase inhibitors	
Sunitinib	2.7–19
Pazopanib	7–11
Sorafenib	4–8
Dasatinib	2–4
Imatinib mesylate	0.2–2.7
Lapatinib	0.2–1.5
Nilotinib	1
Proteasome inhibitors	
Carfilzomib	11–25
Bortezomib	2–5
Miscellanous	
Everolimus	<1
Temsirolimus	<1

INTRODUCTION

Do we have different guidelines?



European Heart Journal (2016) **37**, 2129–2200
doi:10.1093/eurheartj/ehw128

ESC GUIDELINES

2016 ESC Guidelines for the diagnosis and treatment of acute and chronic heart failure

European Heart Journal (2016) **37**, 2768–2801
doi:10.1093/eurheartj/ehw211

ESC CPG POSITION PAPER

2016 ESC Position Paper on cancer treatments and cardiovascular toxicity developed under the auspices of the ESC Committee for Practice Guidelines

Rev Esp Cardiol. 2017;**70**(6):474–486

Special article

Cardio-Onco-Hematology in Clinical Practice. Position Paper and Recommendations

European Heart Journal – Cardiovascular Imaging (2014) **15**, 1063–1093
doi:10.1093/ehjci/jeu192

POSITION PAPER

Expert consensus for multimodality imaging evaluation of adult patients during and after cancer therapy: a report from the American Society of Echocardiography and the European Association of Cardiovascular Imaging

DIAGNOSIS

DIAGNOSIS

- **Early detection strategies**:

  -Identify **risk factors**

  -Referral for high-risk patients

Current myocardial disease	*Demographic and other CV risk factors*
• Heart failure (with either preserved or reduced ejection fraction) • Asymptomatic LV dysfunction (LVEF <50% or high natriuretic peptide[a]) • Evidence of CAD (previous myocardial infarction, angina, PCI or CABG, myocardial ischaemia) • Moderate and severe VHD with LVH or LV impairment • Hypertensive heart disease with LV hypertrophy • Hypertrophic cardiomyopathy • Dilated cardiomyopathy • Restrictive cardiomyopathy • Cardiac sarcoidosis with myocardial involvement • Significant cardiac arrhythmias (e.g. AF, ventricular tachyarrhythmias)	• Age (paediatric population <18 years; >50 years for trastuzumab; >65 years for anthracyclines) • Family history of premature CV disease (<50 years) • Arterial hypertension • Diabetes mellitus • Hypercholesterolaemia
Previous cardiotoxic cancer treatment	*Lifestyle risk factors*
• Prior anthracycline use • Prior radiotherapy to chest or mediastinum	• Smoking • High alcohol intake • Obesity • Sedentary habit

When to refer a patient with heart failure to Cardiology

- First episode of heart failure
- Refractory heart failure
- Multiple admissions
- Possible ischaemic aetiology
  - Previous MI, angina, chest pain
- Significant valvulopathy
- Sudden cardiac death
- Syncope
- Symptomatic arrhythmias
- LVEF <35% with refractory symptoms
- QRS > 120 ms
- **Need for cardiotoxic treatment(NSAIDs,Chemo…)**
- Any other criteria

DIAGNOSIS

- **Early detection strategies**:

  -Identify **risk factors**

  -Referral for high-risk patients

  -Screening strategies:

  *Cardiac imaging

  *Biomarkers

Current myocardial disease	*Demographic and other CV risk factors*
• Heart failure (with either preserved or reduced ejection fraction) • Asymptomatic LV dysfunction (LVEF <50% or high natriuretic peptide[a]) • Evidence of CAD (previous myocardial infarction, angina, PCI or CABG, myocardial ischaemia) • Moderate and severe VHD with LVH or LV impairment • Hypertensive heart disease with LV hypertrophy • Hypertrophic cardiomyopathy • Dilated cardiomyopathy • Restrictive cardiomyopathy • Cardiac sarcoidosis with myocardial involvement • Significant cardiac arrhythmias (e.g. AF, ventricular tachyarrhythmias)	• Age (paediatric population <18 years; >50 years for trastuzumab; >65 years for anthracyclines) • Family history of premature CV disease (<50 years) • Arterial hypertension • Diabetes mellitus • Hypercholesterolaemia
Previous cardiotoxic cancer treatment	*Lifestyle risk factors*
• Prior anthracycline use • Prior radiotherapy to chest or mediastinum	• Smoking • High alcohol intake • Obesity • Sedentary habit

DIAGNOSIS

Table 6 Proposed diagnostic tools for the detection of cardiotoxicity

Technique	Currently available diagnostic criteria	Advantages	Major limitations
Echocardiography: **- 3D-based LVEF** **- 2D Simpson's LVEF** **- GLS**	• LVEF: >10 percentage points decrease to a value below the LLN suggests cardiotoxicity. • GLS: >15% relative percentage reduction from baseline may suggest risk of cardiotoxicity.	• Wide availability. • Lack of radiation. • Assessment of haemodynamics and other cardiac structures.	• Inter-observer variability. • Image quality. • GLS: inter-vendor variability, technical requirements.
Nuclear cardiac imaging (MUGA)	• >10 percentage points decrease in LVEF with a value <50% identifies patients with cardiotoxicity.	• Reproducibility.	• Cumulative radiation exposure. • Limited structural and functional information on other cardiac structures.
Cardiac magnetic resonance	• Typically used if other techniques are non-diagnostic or to confirm the presence of LV dysfunction if LVEF is borderlines.	• Accuracy, reproducibility. • Detection of diffuse myocardial fibrosis using T1/T2 mapping and ECVF evaluation.	• Limited availability. • Patient's adaptation (claustrophobia, breath hold, long acquisition times).
Cardiac biomarkers: **- Troponin I** **- High-sensitivity Troponin I** **- BNP** **- NT-proBNP**	• A rise identifies patients receiving anthracyclines who may benefit from ACE-Is. • Routine role of BNP and NT-proBNP in surveillance of high-risk patient needs futher investigation.	• Accuracy, reproducibility. • Wide availability. • High-sensitivity.	• Insufficient evidence to establish the significance of subtle rises. • Variations with different assays. • Role for routine surveillance not clearly established.

DIAGNOSIS

Heart failure criteria

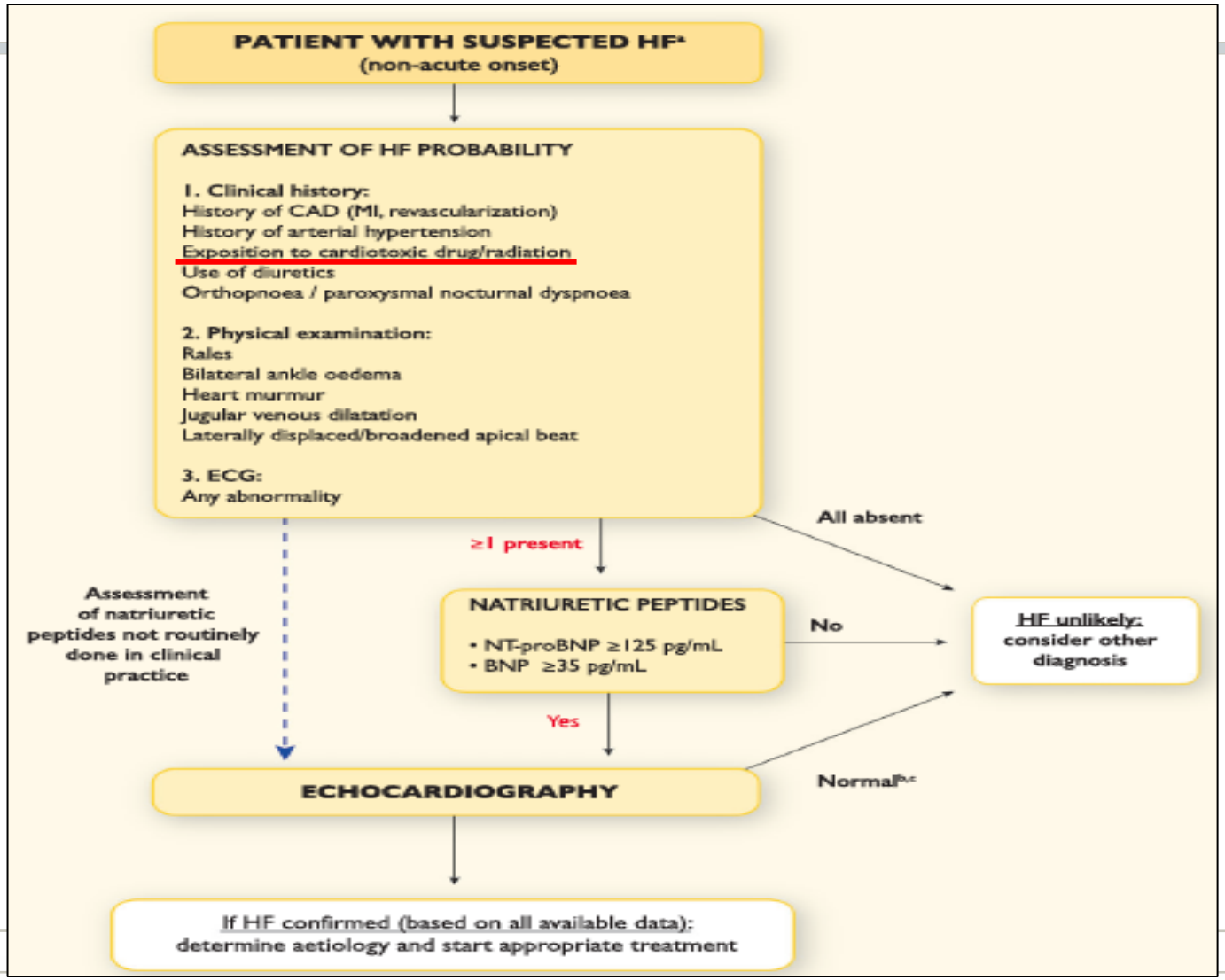
Type of HF		HFrEF	HFmrEF	HFpEF
CRITERIA	1	Symptoms ± Signs[a]	Symptoms ± Signs[a]	Symptoms ± Signs[a]
	2	LVEF <40%	LVEF 40–49%	LVEF ≥50%
	3	–	1. Elevated levels of natriuretic peptides[b]; 2. At least one additional criterion: a. relevant structural heart disease (LVH and/or LAE), b. diastolic dysfunction (for details see Section 4.3.2).	1. Elevated levels of natriuretic peptides[b]; 2. At least one additional criterion: a. relevant structural heart disease (LVH and/or LAE), b. diastolic dysfunction (for details see Section 4.3.2).

DIAGNOSIS

Table 4.1 Symptoms and signs typical of heart failure

Symptoms	Signs
Typical	**More specific**
Breathlessness Orthopnoea Paroxysmal nocturnal dyspnoea Reduced exercise tolerance Fatigue, tiredness, increased time to recover after exercise Ankle swelling	Elevated jugular venous pressure Hepatojugular reflux Third heart sound (gallop rhythm) Laterally displaced apical impulse
Less typical	**Less specific**
Nocturnal cough Wheezing Bloated feeling Loss of appetite Confusion (especially in the elderly) Depression Palpitations Dizziness Syncope Bendopnea[53]	Weight gain (>2 kg/week) Weight loss (in advanced HF) Tissue wasting (cachexia) Cardiac murmur Peripheral oedema (ankle, sacral, scrotal) Pulmonary crepitations Reduced air entry and dullness to percussion at lung bases (pleural effusion) Tachycardia Irregular pulse Tachypnoea Cheyne Stokes respiration Hepatomegaly Ascites Cold extremities Oliguria Narrow pulse pressure

DIAGNOSIS


PATIENT WITH SUSPECTED HF[a]
(non-acute onset)
ASSESSMENT OF HF PROBABILITY
1. Clinical history:
History of CAD (MI, revascularization)
History of arterial hypertension
Exposition to cardiotoxic drug/radiation
Use of diuretics
Orthopnoea / paroxysmal nocturnal dyspnoea
2. Physical examination:
Rales
Bilateral ankle oedema
Heart murmur
Jugular venous dilatation
Laterally displaced/broadened apical beat
3. ECG:
Any abnormality
All absent
≥1 present
Assessment of natriuretic peptides not routinely done in clinical practice
NATRIURETIC PEPTIDES
• NT-proBNP ≥125 pg/mL
• BNP ≥35 pg/mL
No
HF unlikely: consider other diagnosis
Yes
ECHOCARDIOGRAPHY
Normal[b,c]
If HF confirmed (based on all available data):
determine aetiology and start appropriate treatment

DIAGNOSIS

If **heart failure is diagnosed**

Rule out ischaemic heart disease → **CT**, **CMR**, **Angiography**

Invasive coronary angiography is recommended in patients with HF and angina pectoris recalcitrant to pharmacological therapy or symptomatic ventricular arrhythmias or aborted cardiac arrest (who are considered suitable for potential coronary revascularization) in order to establish the diagnosis of CAD and its severity.	I	C
Invasive coronary angiography should be considered in patients with HF and intermediate to high pre-test probability of CAD and the presence of ischaemia in non-invasive stress tests (who are considered suitable for potential coronary revascularization) in order to establish the diagnosis of CAD and its severity.	IIa	C
Cardiac CT may be considered in patients with HF and low to intermediate pre-test probability of CAD or those with equivocal non-invasive stress tests in order to rule out coronary artery stenosis.	IIb	C
CMR with LGE should be considered in patients with dilated cardiomyopathy in order to distinguish between ischaemic and non-ischaemic myocardial damage in case of equivocal clinical and other imaging data (taking account of cautions/contra-indications to CMR).	IIa	C

TREATMENT

TREATMENT

- **Conventional** heart failure **treatment algorithms** apply for heart failure secondary to cardiotoxicity

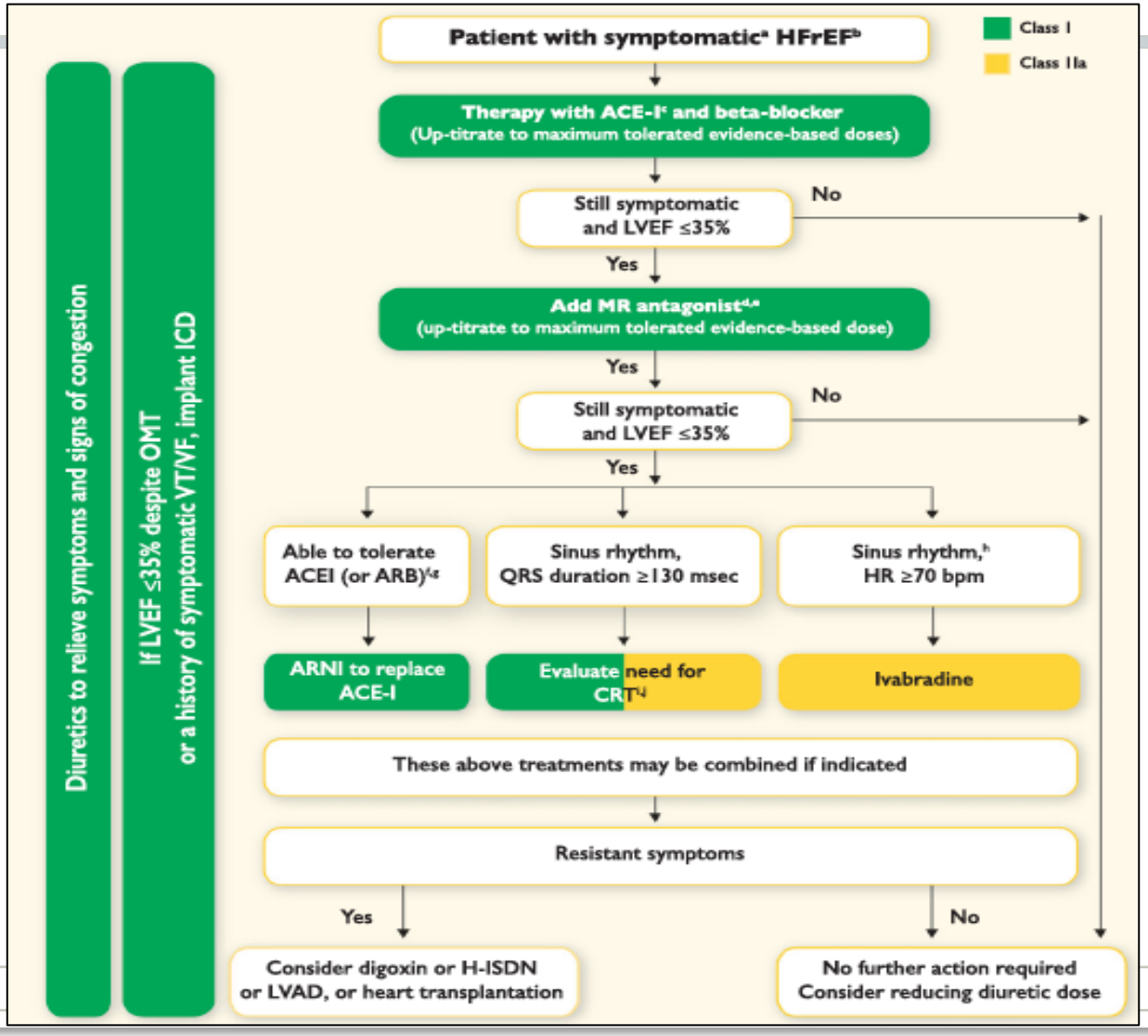
- **Heart failure therapies**:

  -Pharmacological treatment

  -Non-surgical devices

  -Advanced therapies

TREATMENT


Class I
Class IIa
Patient with symptomatic[a] HFrEF[b]
Therapy with ACE-I[c] and beta-blocker
(Up-titrate to maximum tolerated evidence-based doses)
Still symptomatic and LVEF ≤35%
No
Yes
Add MR antagonist[d,e]
(up-titrate to maximum tolerated evidence-based dose)
Yes
Still symptomatic and LVEF ≤35%
No
Yes
Able to tolerate ACEI (or ARB)[f,g]
Sinus rhythm, QRS duration ≥130 msec
Sinus rhythm,[h] HR ≥70 bpm
ARNI to replace ACE-I
Evaluate need for CRT[i,j]
Ivabradine
These above treatments may be combined if indicated
Resistant symptoms
Yes
No
Consider digoxin or H-ISDN or LVAD, or heart transplantation
No further action required
Consider reducing diuretic dose
Diuretics to relieve symptoms and signs of congestion
If LVEF ≤35% despite OMT or a history of symptomatic VT/VF, implant ICD

PHARMACOLOGICAL TREATMENT

Beta-blockers

-In symptomatic patients
-Mortality reduction
-Uptitrate to higher dose

Beta-blockers		
Bisoprolol	1.25 o.d.	10 o.d.
Carvedilol	3.125 b.i.d.	25 b.i.d.[d]
Metoprolol succinate (CR/XL)	12.5–25 o.d.	200 o.d.
Nebivolol[e]	1.25 o.d.	10 o.d.

ACEI/ARB

-In all patients
-Mortality reduction
-Uptitrate to higher dose

ACE-I		
Captopril[a]	6.25 t.i.d.	50 t.i.d.
Enalapril	2.5 b.i.d.	10–20 b.i.d.
Lisinopril[b]	2.5–5.0 o.d.	20–35 o.d.
Ramipril	2.5 o.d.	10 o.d.
Trandolapril[a]	0.5 o.d.	4 o.d.

ARBs		
Candesartan	4–8 o.d.	32 o.d.
Valsartan	40 b.i.d.	160 b.i.d.
Losartan[b,c]	50 o.d.	150 o.d.

MRA

-In symptomatic patients and LVEF ≤35%

MRAs		
Eplerenone	25 o.d.	50 o.d.
Spironolactone	25 o.d.	50 o.d.

ARNI

-In symptomatic patients and LVEF ≤35% despite OMT

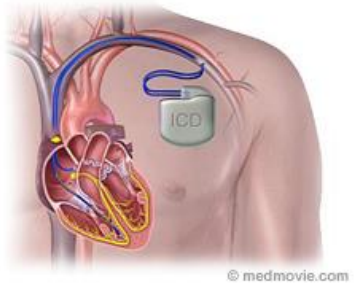
ARNI		
Sacubitril/valsartan	49/51 b.i.d.	97/103 b.i.d.

Ivabradine

-Sinus rhythm >70
-Symptomatic
-LVEF≤35%

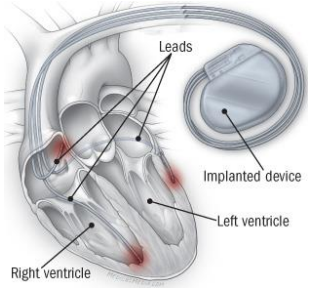
If-channel blocker		
Ivabradine	5 b.i.d.	7.5 b.i.d.

NON-SURGICAL DEVICES



- **ICD:**

  -LVEF ≤35% and NYHA class II-III despite OMT



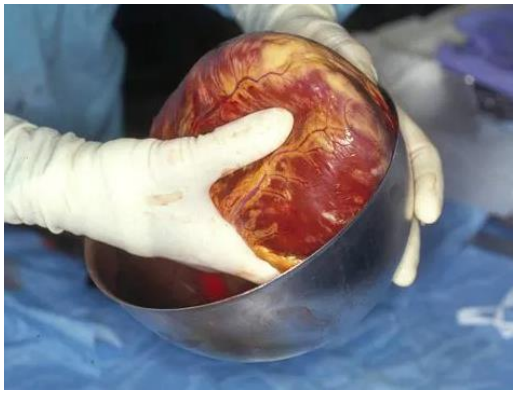
- **CRT:**

  -QRS >130ms, LVEF ≤35% and NYHA class II-III despite OMT

ADVANCED THERAPIES

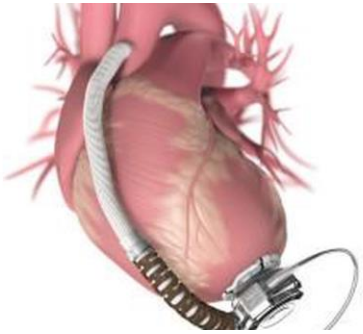
To be considered in symptomatic patients despite OMT

- Heart transplant:



Neoplasm in remission
MDT decision

- Left ventricular assist devices:



Life expectancy > 2 years
MDT decision

IS IT POSSIBLE TO PREVENT HEART FAILURE IN PATIENTS RECEIVING CARDIOTOXIC DRUGS?

CARDIOPROTECTIVE AGENTS

- **Cardioprotective agents** in **primary prevention**:

  -Beta-blockers

  -ACE inhibitors

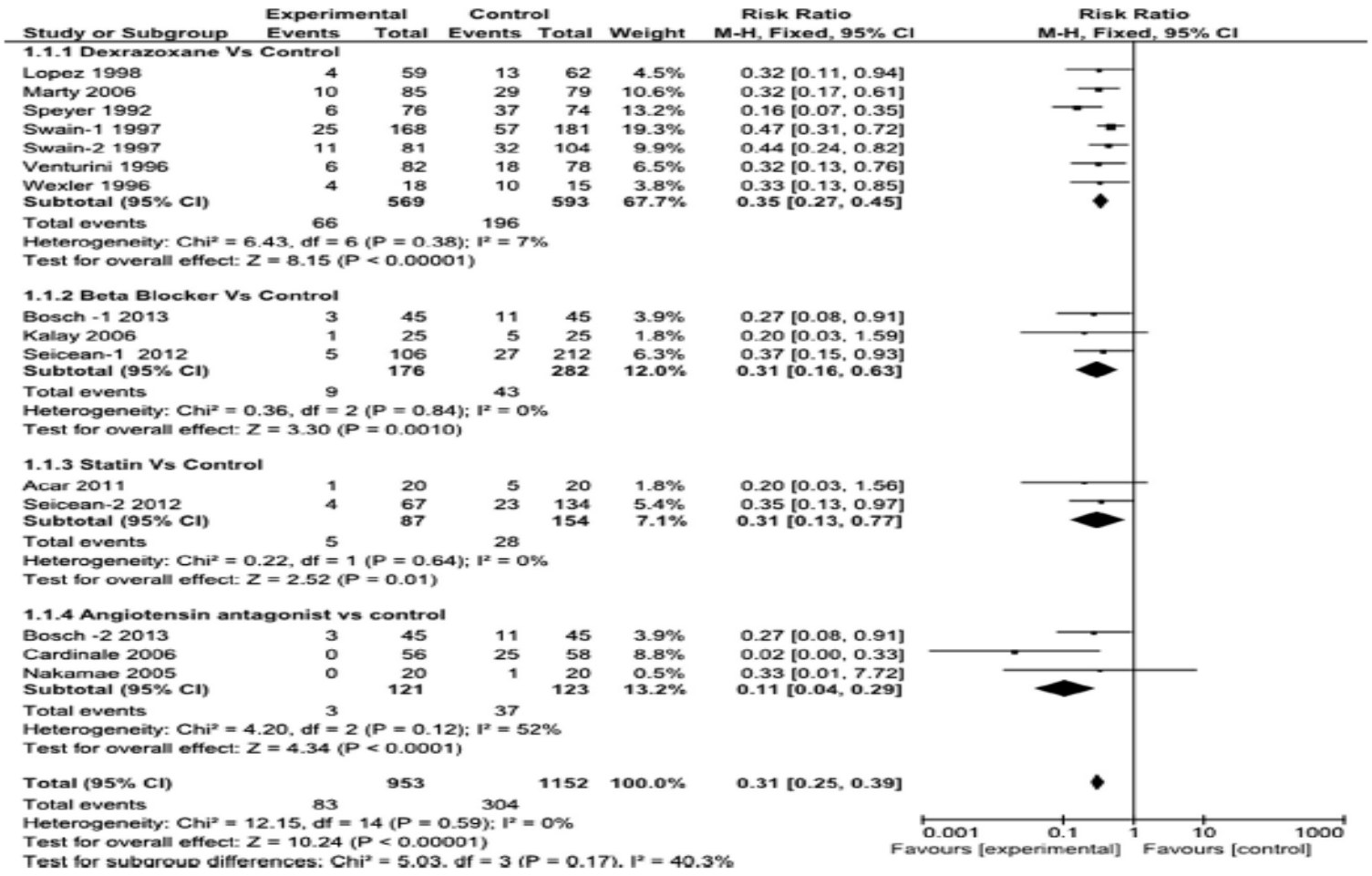
  -Combination therapy

  -Statins

  -Aldosterone inhibitors

Role of cardioprotective therapy for prevention of cardiotoxicity with chemotherapy: A systematic review and meta-analysis

Kashif Kalam, Thomas H. Marwick*



Kalam K, Marwick TH. Role of cardioprotective therapy for prevention of cardiotoxicity with chemotherapy: a systematic review and meta-analysis. Eur J Cancer. 2013 Sep;49(13):2900-9.

BETA-BLOCKERS

Original Article

Cardioprotective Effect of β-Adrenoceptor Blockade in Patients With Breast Cancer Undergoing Chemotherapy
Follow-Up Study of Heart Failure

Sinziana Seicean, MD, MPH, PhD; Andreea Seicean, PhD, MPH; Nima Alan, BS; Juan Carlos Plana, MD; G. Thomas Budd, MD; Thomas H. Marwick, MBBS, PhD, MPH

- **Methods:**

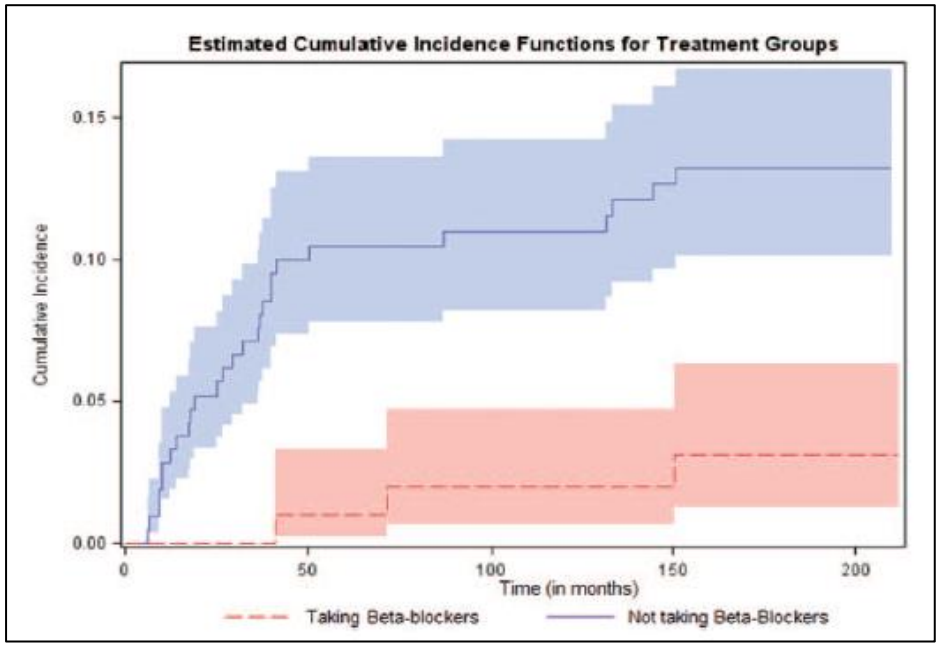
  -Breast cancer patients receiving anthracyclines and trastuzumab

- **Objective:**

  -HF admissions and death

- **Results:**

  - BB treatment was associated with lower risk of new HF events (HR 0.2; 95% CI, 0.1–0.5; p=0.003).



Seicean S et al. Cardioprotective effect of β-adrenoceptor blockade in patients with breast cancer undergoing chemotherapy: follow-up study of heart failure. Circ Heart Fail. 2013 May;6(3):420-6.

ACE INHIBITORS

Prevention of High-Dose Chemotherapy–Induced Cardiotoxicity in High-Risk Patients by Angiotensin-Converting Enzyme Inhibition

Daniela Cardinale, MD; Alessandro Colombo, MD; Maria T. Sandri, MD; Giuseppina Lamantia, MD; Nicola Colombo, MD; Maurizio Civelli, MD; Giovanni Martinelli, MD; Fabrizio Veglia, PhD; Cesare Fiorentini, MD; Carlo M. Cipolla, MD

- **Methods:**

  -114 patients (56 enalapril vs 58 no ACEi)

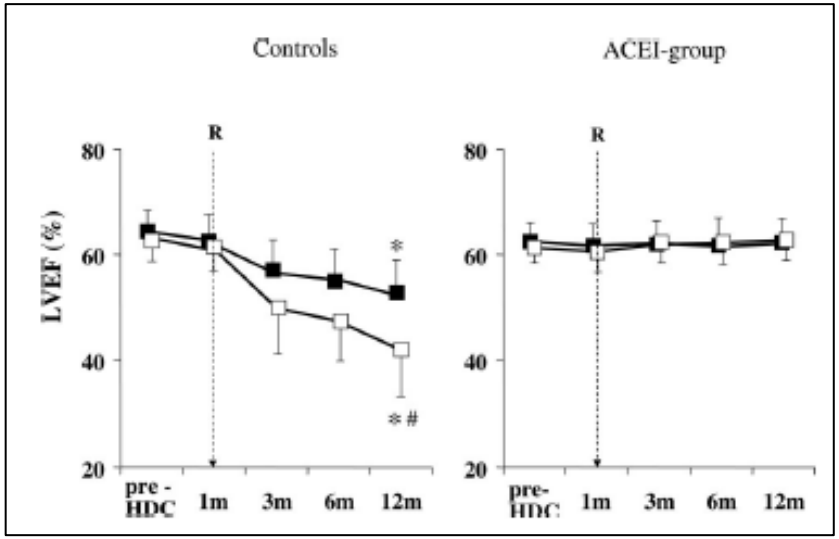
  -ACEi started after 1 month of treatment

- **Objective:**

  -The occurrence of cardiotoxicity

- **Results:**

  - Incidence of cardiotoxicity was significantly higher in control group (43% vs 0%; p=0.001).



Cardinale D et al. Prevention of high-dose chemotherapy-induced cardiotoxicity in high-risk patients by angiotensin-converting enzyme inhibition. Circulation. 2006 Dec 5;114(23):2474-81.

COMBINATION THERAPY

Enalapril and Carvedilol for Preventing Chemotherapy-Induced Left Ventricular Systolic Dysfunction in Patients With Malignant Hemopathies

The OVERCOME Trial (preventiOn of left Ventricular dysfunction with Enalapril and caRvedilol in patients submitted to intensive ChemOtherapy for the treatment of Malignant hEmopathies)

- **Methods**: Breast cancer patients receiving anthracyclines and trastuzumab
- **Objective:** Efficacy of enalapril and carvedilol to prevent cardiotoxicity
- **Results:** LVEF decrease was lower in the intervention group

Table 3 Differences in Change in LVEF Between the Intervention and Control Groups

	Enalapril + Carvedilol	Control	Intergroup Difference	p Value
Echocardiography				
LVEF (%)	n = 42	n = 37		
Baseline	61.67 ± 5.11	62.59 ± 5.38		
6 months	−0.17 (−2.24 to 1.90)	−3.28 (−5.49 to −1.07)	−3.11 (−6.10 to −0.11)	0.04
CMR				
LVEF (%)	n = 31	n = 27		
Baseline	56.00 ± 6.00	60.18 ± 7.16		
6 months	0.36 (−2.41 to 3.13)	−3.04 (−6.01 to −0.07)	−3.40 (−7.43 to 0.63)	0.09

Bosch X et al. Enalapril and carvedilol for preventing chemotherapy-induced left ventricular systolic dysfunction in patients with malignant hemopathies: the OVERCOME trial (preventiOn of left Ventricular dysfunction with Enalapril and caRvedilol in patients submitted to intensive ChemOtherapy for the treatment of Malignant hEmopathies). JACC 2013 Jun 11;61(23):2355-62.

COMBINATION THERAPY

Prevention of cardiac dysfunction during adjuvant breast cancer therapy (PRADA): a 2 × 2 factorial, randomized, placebo-controlled, double-blind clinical trial of candesartan and metoprolol

- **Methods**: Breast cancer patients receiving anthracyclines and trastuzumab
- **Objective:** Primary outcome was change in LVEF by CMR
- **Results:** The overall decline in LVEF was 2.6 (95% CI 1.5, 3.8) in the placebo group and 0.8 (95% CI 20.4, 1.9) in the candesartan group. No effect on metoprolol

Table 2 Primary and secondary endpoints, estimated values from linear mixed models (intention-to-treat analysis)

	n	Baseline	EOS	Change from baseline to EOS	Between-group difference in change from baseline to EOS	*P*-value
LVEF						
No candesartan	60	63.2 (62.0, 64.4)	60.6 (59.4, 61.8)	−2.6 (−3.8, −1.5)	1.9 (0.2, 3.5)[a]	0.026
Candesartan	60	62.1 (61.0, 63.3)	61.4 (60.2, 62.6)	−0.8 (−1.9, 0.4)		
No metoprolol	62	62.8 (61.6, 64.0)	61.0 (59.8, 62.2)	−1.8 (−3.0, −0.7)	0.2 (−1.4, 1.9)	0.772
Metoprolol	58	62.5 (61.3, 63.7)	61.0 (59.8, 62.2)	−1.6 (−2.8, −0.4)		
RVEF						
No candesartan	60	61.3 (60.0, 62.5)	58.9 (57.6, 60.1)	−2.4 (−3.7, −1.1)	0.8 (−1.0, 2.6)	0.370
Candesartan	60	60.2 (59.0, 61.4)	58.7 (57.4, 59.9)	−1.6 (−2.8, −0.3)		
No metoprolol	62	60.4 (59.2, 61.6)	58.0 (56.8, 59.3)	−2.4 (−3.7, −1.1)	0.8 (−1.0, 2.6)	0.377
Metoprolol	58	61.1 (59.8, 62.3)	59.5 (58.3, 60.8)	−1.6 (−2.9, −0.3)		
LV GLS						
No candesartan	48	−21.6 (−22.1, −21.1)	−21.0 (−21.5, −20.5)	0.6 (0.1, 1.1)	−0.7 (−1.4, 0.1)	0.076
Candesartan	45	−21.3 (−21.8, −20.7)	−21.3 (−21.9, −20.8)	−0.1 (−0.6, 0.5)		
No metoprolol	46	−21.4 (−21.9, −20.8)	−21.0 (−21.6, −20.5)	0.3 (−0.2, 0.8)	−0.1 (−0.8, 0.7)	0.824
Metoprolol	47	−21.5 (−22.0, −21.0)	−21.3 (−21.8, −20.7)	0.2 (−0.3, 0.7)		

Gulati G et al. Prevention of cardiac dysfunction during adjuvant breast cancer therapy (PRADA): a 2 × 2 factorial, randomized, placebo-controlled, double-blind clinical trial of candesartan and metoprolol. Eur Heart J. 2016 Jun 1;37(21):1671-80

CONCLUSIONS

- **Cardiotoxicity** is defined as **symptomatic or asymptomatic LVEF reduction of more than 10%** compared to baseline, with **final LVEF < 53%**
- **Conventional guidelines** on heart failure **apply** for cancer patients developing heart failure
- **High-risk patients** should be identified in order to be **closely monitored**
- **Cardiac imaging and biomarkers** are used for **early detection and diagnosis**

CONCLUSIONS

- **Conventional** heart failure **treatment algorithms should be used** for treating heart failure secondary to cardiotoxicity
- Several **heart failure drugs might prevent** myocardial dysfunction in these patients
- **Further studies are needed** in order to have more information on this topic

THANK YOU FOR YOUR ATTENTION