

Chronic Heart Failure: Impact of the Current Guidelines

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ABSTRACT

Despite the persistent high mortality, many adults are living with chronic heart failure. Recent updates to the clinical guidelines for managing heart failure provide substantive recommendations on how to treat patients with heart failure with preserved ejection fraction or heart failure with reduced ejection fraction. Key changes in these guidelines include 2 new medications, use of biomarkers, a focus on specific comorbidities, and prevention strategies. This report provides recommendations from the updated 2017 guideline for the management of heart failure for nurse practitioners in caring for patients with chronic heart failure.

Keywords: angiotensin receptor-neprilysin inhibitor, biomarkers, chronic heart failure, heart failure comorbidities, ivabradine

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INTRODUCTION

As a chronic illness, heart failure (HF) challenges clinicians to stay informed of relevant changes in clinical practice. As the incidence and prevalence of HF continues to rise, it is imperative that health care providers are prepared to address issues associated with this chronic illness and the acute exacerbations that occur throughout the trajectory of this disease.

According to the American Heart Association (AHA), the number of adult patients who are chronically ill with HF will increase by 46% by 2030.^{1,2} Although the incidence is stable, there is an increase in the number of patients with HF with preserved ejection fraction (HFpEF) compared with HF with reduced ejection fraction (HFrEF), and this shift is likely due to the aging population as well as the implementation of therapies for patients with HFrEF.²

In an effort to improve morbidity and mortality, primary care clinicians must become familiar with these updated HF guidelines, which include new treatments.³ In addition, to new treatments, there are recommendations that focus on clinicians collaborating with the patient to identify individualized goals of care, provide patient education, and enhance care coordination.^{3,4} This report provides recommendations

from the updated 2017 guideline for the management of HF for nurse practitioners in caring for patients with chronic HF.³

REVIEW OF HF PATHOPHYSIOLOGY AND DEFINITIONS

HF is a clinical syndrome that results in cardiac dysfunction associated with myocardial loss, left ventricular hypertrophy, or a combination of the two.⁴ This cardiac dysfunction manifests as an inability for the heart to fill or empty, or both.² HF stimulates multiple circulatory and neurohormonal pathways, such as cytokine activation, which in turn result in cardiac remodeling, often leading to ventricular dilation and hemodynamic compromise.⁵ The goal of HF treatment is to mitigate this remodeling and disrupt the neurohormonal activation in an effort to halt disease progression.

The 2013, American College of Cardiology Foundation/AHA guidelines offered new definitions for HF that provided clarity to discriminate between systolic (HFrEF) and diastolic dysfunction (HFpEF) using EF (Supplementary Table 1, available online at <http://www.npjjournal.org>).⁵ New definitions have emerged to better define the populations. HF with midrange EF (HFmrEF) are those patients with an EF of 41% to 49%, and HF with recovered EF refers to those who initially had a reduced EF

and over a period of time have recovered left ventricular function.⁶

Many patients who are currently classified as HFpEF should be reclassified as HF-recovered EF.⁶ In 1 study, patients with HF-recovered EF demonstrated persistent activation of neurohormonal pathways and oxidative stress despite recovery of EF.⁶ Basuray et al⁷ attempted to characterize the phenotype and prognosis of patients with HF-recovered EF compared with HFrEF and HFpEF. The sample of 1,821 patients included 122 patients with HFpEF, 1,523 with HFrEF, and 176 with HF-recovered EF.⁷ Results indicated that there were different phenotypes among the 3 groups and that those in the HF-recovered EF group had differences in symptom severity as well as demographics and comorbid conditions.⁷ These new definitions allow the NP to consider fine tuning a diagnosis and provide guideline-directed medical therapy (GDMT).⁵ As with any new terminology, it will take time for this nomenclature to be adopted into clinical practice as well as future updates to billing codes.

PHARMACOLOGY

Current practice for HFrEF consists of the initiation of an angiotensin-converting enzyme inhibitor (ACEi) or angiotensin receptor blocker (ARB) and β -blocker therapy titrated to a target dose. Once patients are on GDMT and their EF remains $\leq 35\%$ they should be referred for implantation of a cardioverter/defibrillator for prevention of sudden cardiac death.⁵ In addition, if there is ventricular dyssynchrony, as determined by a QRS complex greater than 120 milliseconds, then cardiac resynchronization therapy is indicated.⁵ Current practice for HFpEF focuses on reducing congestion and managing comorbidities.

The 2016 European Society of Cardiology guidelines and 2017 updated HF guideline recommendations included the use of mineralocorticoid receptor antagonists (MRAs), such as spironolactone and eplerenone, and the addition of 2 new medications to the armamentarium for treatment of HFrEF^{8,9} (Supplementary Table 2, available online at <http://www.npjjournal.org>). Aldosterone is a mineralocorticoid that has deleterious effects on the heart, such as hypervolemia, left ventricular

remodeling, endothelial dysfunction, and myocardial fibrosis, all of which contribute to worsening HF.⁸ MRAs inhibit aldosterone, and when added to other neurohormonal agents, these deleterious effects can be mitigated.⁸ In the 2013 HF Guidelines, the RALES (Randomized Aldactone Evaluation Study Investigators) and EPHEUS (Eplerenone Post-Acute Myocardial Infarction Heart Failure Efficacy and Survival Study) trials both supported the use of MRAs in symptomatic patients with HFrEF in reducing the risk of sudden cardiac death and thereby improving mortality.⁵

In a more recent study, the Treatment of Preserved Cardiac Function Heart Failure with an Aldosterone Antagonist (TOPCAT) trial explored the use of spironolactone to reduce HF hospitalization or cardiovascular death in patients with HFpEF.¹⁰ There was a small reduction in HF hospitalizations, although the composite end points did not achieve statistical significance. As a result, there is a new recommendation in the 2017 guidelines to consider the addition of an aldosterone receptor antagonist in patients with an EF $\geq 45\%$, HF hospitalization within 1 year, or elevated brain (B-type) natriuretic peptide (BNP) level, potassium < 5.0 mEq/L, creatinine < 2.5 mg/dL, or glomerular filtration rate > 30 mL/min in an effort to reduce hospitalization.³ The initial dose of spironolactone is 12.5 to 25 mg daily and eplerenone is 25 mg daily. Checking the potassium level within 1 week of initiation is recommended.

A new target to prevent stimulation of the renin-angiotensin-aldosterone system is neprilysin. Combined with an ARB, inhibiting neprilysin has demonstrated beneficial effect on mortality in patients with HFrEF. A major result of the PARADIGM-HF (Efficacy and Safety of LCZ696 Compared to Enalapril on Morbidity and Mortality of Patients With Chronic Heart Failure) study was the 20% reduction in the combined primary end point of HF hospitalization or death and a 16% reduction in all-cause mortality comparing the combination therapy to enalapril alone.¹¹

Combining an ARB and neprilysin inhibitor created a new class of medications: angiotensin receptor-neprilysin inhibitor (ARNi). For symptomatic patients with HFrEF, New York Heart

Association (NYHA) Functional Classification II and III symptoms, stable blood pressure (BP), and no contraindication to sacubitril or an ARB, there is a Level I recommendation to transition from an ACEi or ARB to ARNi.^{3,4}

One key point to keep in mind when transitioning from an ACEi to an ARNi is that a 36-hour washout period is required before initiating an ARNi.⁵ This washout period is required to reduce the potential for angioedema. ARBs do not inhibit kinase, so there is less incidence of angioedema.⁵ Because the combination of sacubitril and valsartan may increase creatinine, careful monitoring of kidney function is recommended after initiating therapy.⁵

When initiating sacubitril/valsartan if the patient is on a low-dose ACEi or ARB or not currently on either of these medications, the patient should be prescribed the lowest dosage, which is 24/26 mg twice a day. If the patient is already taking > 10 mg enalapril or an equivalent dose of another ACEi, then it is reasonable to start with 49/51 mg of sacubitril/valsartan twice a day. Titration to higher doses is recommended every 2 to 4 weeks and should be guided by patient symptoms, BP, and kidney function, with a maximum dose of 97/103 mg twice a day.⁵

Elevation in resting heart rate is known to confer a poor prognosis in patients with HF. In the Systolic Heart failure Treatment With the If Inhibitor Ivabradine Trial (SHIFT), 6558 patients were treated with ivabradine, a new medication that targets the I(f) channel to inhibit the sinus node. The results of this clinical trial indicate that with the addition of ivabradine to GDMT, there was an 18% relative risk reduction in hospitalizations for HF or cardiovascular death in patients.^{12,13}

Ivabradine is a class IIa recommendation for use in patients with HFrEF and EF ≤ 35% who remain symptomatic and are on GDMT with dosing as tolerated who are in sinus rhythm with a heart rate > 70 beats/min.³ Starting dose of ivabradine is 5 mg twice a day up to a maximum dose of 7.5 mg twice a day for a heart rate > 60 beats/min. If a patient develops symptoms of dizziness or fatigue with a heart rate of < 50 beats/min, then the dose should be reduced by 2.5 mg twice a day, and if already on this low dose and symptomatic, then the medication

should be stopped.¹⁴ As a class IIa recommendation, it is reasonable and may be effective as an additional medication to the HF regimen in patients who meet the above criteria.

Pharmacologic treatment of HF can be overwhelming because there are so many evidence-based medications that are recommended in the clinical guidelines.⁵ A visual representation of a sequential process for how and when to initiate these new pharmacologic treatments amidst the ongoing medical therapy is in [Supplementary Table 2](#) (available online at <http://www.npjjournal.org>).

BIOMARKERS

Since the introduction of BNP as a biomarker for use in management of heart failure, there have been recommendations for its use in diagnosis and potential use for guiding treatment. BNP and N-terminal pro-BNP (NT-proBNP) are secreted by the cardiac ventricle and are the primary biomarkers used in the management of HF.¹⁵ Over the years a variety of clinical trials have been designed to determine how to use BNP to guide diuretic use.¹⁵ One of these clinical trials, GUIDE IT (Guiding Evidence Based Therapy Using Biomarker Intensified Treatment in Heart Failure), was designed to determine whether using NT-proBNP to titrate GDMT improved clinical outcomes in patients with HFrEF compared with usual care.¹⁶ The study was halted early due to futility, and the final outcome was that NT-proBNP-guided therapy was not more effective than usual care in high-risk patients with HFrEF.¹⁶

A variety of factors can affect BNP values resulting in elevated or reduced results. Including HF, the following conditions may cause an increase in BNP and NT-proBNP levels: hypertension, left ventricular hypertrophy, chronic kidney disease, valvular heart disease, ischemic heart disease, atrial fibrillation, pulmonary embolism, pulmonary hypertension, and advanced age.¹⁷

BNP and NT-proBNP levels may be low in patients who are obese, and therefore, additional clinical assessment data and diagnostic testing, such as a transthoracic echocardiogram, should be considered in the workup.¹⁷ In addition, patients who are currently being treated with medications that interfere with the renin-angiotensin-aldosterone

system, such as ARNi, will have reduced levels of natriuretic peptides and require the same diagnostic considerations.¹⁷

Current recommendations suggest specific circumstances in which the use of natriuretic peptide measurement can guide clinical decision making from a prognostic perspective.² There is evidence to support the use of biomarkers in discriminating between a cardiovascular and pulmonary cause of symptoms, such as dyspnea and shortness of breath, and can be done in a primary care setting. Measuring natriuretic peptide biomarkers in the acute care setting provides an opportunity to look for downward trends in levels as a reflection of reduced congestion. The current guidelines recommend obtaining an NT-proBNP level in hospitalized patients just before discharge as a method for risk stratification.³

HEART FAILURE WITH PRESERVED EJECTION FRACTION

Hypertension

The focus for HFpEF treatment remains on effectively managing comorbid conditions, such as hypertension, coronary artery disease (CAD), and arrhythmias.^{3,4} Diuretics are the primary treatment used for symptomatic relief of volume excess and symptoms of congestion. As previously mentioned, the TOPCAT trial provided a recommendation for the addition of an aldosterone receptor antagonist, such as spironolactone or eplerenone, in patients with an EF \geq 45% who met specific potassium and creatinine criteria as a method to reduce hospitalization.^{3,10}

The guidelines for managing hypertension were updated in 2017, and the new target for adequate BP control is \leq 130/80 mm Hg.¹⁸ In addition to recommendations for lifestyle changes regarding diet and exercise, antihypertensive therapy with multiple medications is necessary for optimal BP management.¹⁹ There are 9 medication classes available to treat hypertension, and most patients require 2 to 3 medications, 1 of them a diuretic. The overlap in HF and antihypertensive medications provides an opportunity to use 1 medication to treat both conditions.

Symptomatic With Angina and CAD

Patients with HF should have any symptoms of myocardial ischemia evaluated. To improve symptoms of both HF and CAD, coronary revascularization is advised.⁵ Tobacco use can also precipitate angina, and addressing smoking cessation should be a priority in this patient. Worsening angina may be a precipitating factor in symptom exacerbation in a patient with HFpEF, and treatment with long-acting nitrates may reduce both angina and afterload.

Arrhythmias

Atrial fibrillation can be a precipitant for worsening HF owing to rapid ventricular response or loss of atrial kick in an already compromised heart. Patients with HFpEF frequently develop atrial fibrillation and may not be aware of the change in heart rhythm. When a patient presents with an HF exacerbation, obtaining an electrocardiogram to assess for rhythm changes is a reasonable diagnostic approach to provide insight to the potential cause of worsening symptoms.

COMORBIDITIES

There are specific comorbid conditions that influence HF symptoms and may precede exacerbations. Each of these conditions is addressed in the updated HF guidelines in an effort to highlight their significance and provide recommendations on how to manage these comorbidities in chronic HF.

Anemia

Anemia is often seen in patients with HF and may be related to renal disease, a coagulopathy, or iron deficiency. Many patients with HF have iron-deficient anemia, and for those with NYHA class II and III symptoms, intravenous infusion of iron replacement has been shown to improve functional status and quality of life.⁹ The European Society of Cardiology has recommended iron infusions for patients with HFrEF and iron-deficiency anemia, serum ferritin $<$ 100 μ g/L or ferritin between 100 and 299 μ g/L and transferrin saturation $<$ 20%.⁹ According to the European Society of Cardiology guidelines, intravenous ferric carboxymaltose in patients with HFrEF has shown improved quality of

life, HF symptoms, and exercise capacity and a reduction in hospitalizations.⁹ Ongoing studies are needed to determine the long-term safety of iron therapy in patients with HFpEF/HFmrEF.

Hypertension

As previously mentioned, the 2017 AHA update of the guidelines for management of hypertension identified a new target of 130/80 mm Hg.¹⁸

Aggressively titrating antihypertensive medications to achieve this target is beneficial in both HFrEF and HFpEF. Barriers to achieving goal BP include polypharmacy, finances, and adverse effects of multiple medications. These issues are often more prevalent in older adults and may be worsened by cognitive and visual impairment, social isolation, and functional limitations.

Sleep Apnea

Since the last HF guidelines were published, there have been updates to the best approach to manage sleep disorders in patients with HF. Differentiating between obstructive and central sleep apnea in patients with NYHA class II to IV HF is clinically significant.³ Continuous positive airway pressure in patients with obstructive sleep apnea does improve sleep quality; however, recent randomized clinical trials did not demonstrate a cardiovascular benefit except in patients with concomitant atrial fibrillation.³ Data from the SERVE-HF (Treatment of Sleep-disordered Breathing with Predominant Central Sleep Apnea by Adaptive Servo Ventilation in Patients with Heart Failure) trial showed that adaptive servo ventilation was harmful in patients with central sleep apnea and therefore not recommended.^{3,20}

SKILLED NURSING FACILITIES

The prevalence of chronic HF in skilled nursing facilities (SNF) is high, estimated between 20% and 37.4% and is often listed as a secondary diagnosis.²⁰ Patients with HF discharged from a hospital to a SNF are most often older, female, white, have a higher EF, longer hospital stays, and multiple comorbidities.²⁰ These patients have a higher mortality rate and have a higher rate of rehospitalization compared with patients discharged to home.²¹ Therefore, early

recognition of HF symptoms and the physical examination changes that may accompany them are key to prompt treatment in a SNF.²¹

If there has not been a previous diagnosis of HF, then it is imperative to recognize the frequent causes of HF in older adults. Hypertension, CAD, valvular heart disease, pericardial disease, cardiomyopathy, and age-related diastolic dysfunction are the most common.²¹ Patients receiving treatment in SNFs fall into 1 of 3 categories; long-term care, rehabilitation, and uncertain prognosis.²¹ Chronic HF treatment in these groups is based on goals of care, which may range from aggressive treatment to palliation.

Fluid overload is one of the most common signs of HF and manifests as lower extremity edema, abdominal edema, elevated neck veins, and 1 of the most sensitive signs is worsening orthopnea. Although most evidence-based clinical guidelines are not based on clinical trials that included older adults, treatment of chronic HF should be based on whether or not you are treating HFrEF or HFpEF, NYHA class symptoms, and comorbid conditions. Two key points to consider when evaluating a patient in a SNF: cognitive impairment can limit accurate and timely reporting of symptoms, and lack of overt HF symptoms at rest may mask symptoms of decompensated HF.

PREVENTION OF HF

There is a new focus on HF prevention in the updated guidelines. Reducing cardiovascular risk and early recognition of signs and symptoms of the onset of HF is imperative to reduce the development and prevalence of chronic HF. Determining the risk of developing HF is predicated on the presence of several common comorbid conditions. Individuals with cardiovascular diagnoses, such as hypertension, CAD, diabetes mellitus, or metabolic syndrome, need to have these conditions aggressively treated in an effort to reduce the risk of developing HF.

Another group of individuals who should be informed about the potential to develop HF are those who may have had exposure to cardiotoxins, recreational drugs, such as cocaine or alcohol, and those with a family history or cardiomyopathy. Educating patients who have received chemotherapy about the signs and symptoms of HF will provide an

opportunity to intervene earlier in the course of the disease. For individuals who use cocaine or consume excessive amounts of alcohol, discussing the potential cardiovascular consequences of these behaviors is another strategy to address prevention.

PARTNERING WITH PATIENTS

In addition to self-management strategies, such as daily weight monitoring, dietary sodium and fluid restrictions, and early symptom recognition, there are tools to engage patients beyond patient-reported monitoring. There are a variety of methods in which you can collaborate with patients beyond face-to-face visits and remote telemonitoring using scales and blood pressure devices. Since the implementation of electronic health records, there are options to contact the health care provider directly through secure messaging. In addition, text options and mobile phone applications are available to partner with patients to manage chronic HF.²² Mobile phones are an integral component of daily life for most individuals, thereby providing a platform for telemonitoring that is scalable and sustainable.

IMPLICATIONS FOR PRIMARY CARE PRACTICE

The increase in prevalence of chronic HF requires us to be diligent as we strategize to prevent, diagnose, and treat this condition. Distinguishing between the various types of HF, specifically those individuals who have HF-recovered EF and may currently be classified as HFpEF, has implications for clinical practice because these individuals have significant symptoms, exacerbations, and hospitalizations, and further investigation is required on the cellular level.⁷ Despite ongoing clinical trials, pharmacologic therapy for patients with HFpEF is lacking behind evidence-based therapies for HFrEF. Tissue engineering to replace a failing heart and targeted pharmacology using pharmacogenomics both hold promise for future treatments.²³

Management of a patient with acute HF exacerbations places a significant burden of time and resources on a primary care practice. In an effort to improve symptoms, reduce congestion, avoid acute exacerbations, and keep patients out of the hospital, escalation of diuretic dosing, including addition of metolazone, to provide an additive effect to loop

diuretic is one strategy that can be implemented in a primary care practice. In addition, there are times when the administration of intravenous loop diuretics in the home or ambulatory clinic setting may be appropriate as long as the patient is hemodynamically stable and has prompt follow-up by telephone and in person.

CONCLUSIONS

Managing chronic HF requires a multifaceted approach and vigilance as the course of the disease waxes and wanes. As advanced practice providers, there are opportunities to adjust medical therapy to improve symptoms, reduce mortality, and reduce hospitalizations. All patients with HF require diligence in managing cardiac medications and maximizing dosing strategies in an effort to address this chronic illness. Advanced practice providers practicing in primary care settings are well situated to address issues of HF prevention, manage comorbidities, and initiate GDMT. By engaging patients through shared decision-making strategies, there are opportunities to gradually reduce the effect of chronic HF and improve quality of life.

SUPPLEMENTARY DATA

Supplementary Tables associated with this article can be found in the online version at <https://doi.org/10.1016/j.nurpra.2018.10.016>. **JNP**

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1555-4155/18/\$ see front matter
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<https://doi.org/10.1016/j.nurpra.2018.10.016>

Supplemental Table 1. Definition of Heart Failure

Classification	Ejection Fraction	Description
I. Heart failure with reduced ejection fraction (HFrEF)	< 40%	Also referred to as systolic HF. Randomized clinical trials have mainly enrolled patients with HFrEF and it is only in these patients that efficacious therapies have been demonstrated to date.
II. Heart Failure with preserved ejection fraction	>50%	Also referred to as diastolic HF. Several different criteria have been used to further define HFpEF. The diagnosis of HFpEF is challenging because it is largely one of excluding other potential noncardiac causes of symptoms suggestive of HF. To date, efficacious therapies have not been identified.
a. HFpEF Borderline *HFmrEF	41% to 49%	These patients fall into a borderline or intermediate group. Their characteristics, treatment patterns, and outcomes appear similar to those of patients with HFpEF.
b. HFpEF **HF-recovered EF	>40%	It has been recognized that a subset of patients with HFpEF previously had HFrEF. These patients with improvement or recovery in EF may be clinically distinct from those with persistently preserved or reduced EF. Ongoing research is in progress to better characterize these patients.(Unkovic & Basuray, 2018)

* (Ponikowski, 2016) European Society of Cardiology 2016 refer to this as HFmrEF Heart failure with mid-range ejection fraction.

** HF-Recovered EF.

Source: Yancy, CW et al, 2013 ACCF/AHA Heart Failure Guidelines Executive Summary

Supplemental Table 2. Medication Update

Generic (Trade)	Indication	Mechanism of Action (MOA)	Most Common Adverse Effects	Starting dose range	Key points in Monitoring
Ivabradine (Corlanor [®]) COR IIa LOE B-R	Symptomatic HFrEF with NYHA Class II or III on GDMT* with beta blocker at maximum tolerated dose in sinus rhythm with HR 70 or greater	Inhibits <i>f</i> channel to inhibit the sinus node	Atrial fibrillation, dizziness, bradycardia, hypertension, temporary visual changes (flashes of light)	5 mg bid	Not recommended in pregnancy. Unknown if medication passes into breast milk.
Sacubitril/Valsartan (Entresto [®]) COR I LOE B-R	Chronic symptomatic HFrEF with NYHA Class II or III	Valsartan (V) inhibits angiotensin II by binding to angiotensin II receptors Sacubitril (S) Inhibits neprilysin and prevents breakdown of endogenous vasoactive peptides, such as natriuretic peptides.	V-hypotension, dizziness, headache, hyperkalemia, impaired kidney function, fatigue and diarrhea. S-angioedema, hypotension, hyperkalemia, impaired kidney function.	24/26 mg po bid—49/51 mg po bid Titrate up to 97/ 103 mg po bid	36 hr washout period if transitioning from an ACE inhibitor No washout required if already on ARB. Do not start if patient has symptomatic hypotension or is in a decompensated state. Estimated GFR should be >30mL/Min/1.73m ²
Spironolactone (Aldactone [®]) Eplerenone (Inspra [®]) COR I in HFrEF COR IIb in HFpEF LOE B-R	HFrEF and NYHA class II–IV, with CrCl >30 mL/min & K ⁺ <5.0 mEq/L or In patients with EF≥ 45% with hospitalization within one year or elevated BNP level, potassium <5.0 mEq/L, creatinine <2.5 mg/dl and GFR>30mL/min	Inhibits aldosterone resulting in retention of K ⁺ and excretion of Na ⁺ /Cl ⁻ /Water	Hyperkalemia Gynecomastia (10% incidence only with Spironolactone)	12.5-25 mg po daily	Monitor serum potassium 1 week and one month after initiating. Avoid foods and medications high in potassium. Avoid salt substitutes containing potassium

COR-Class of Recommendation

1- Strong, IIa-Moderate, IIb-Weak, III-No benefit, III-Harm

LOE-Level of Evidence:

A- More than 1 high quality randomized clinical trial (RCT)

B-R Moderate quality evidence from 1 or more RCT

B-NR Moderate quality evidence from 1 or more non-randomized study

C-LD Randomized or nonrandomized observational or registry with limited data

C-EO Expert opinion

* GDMT= Guideline Directed Management and Therapy