Vol. 18 No. 2 December 2022 p-ISSN: 0216-759X e-ISSN: 2614-X476X http://ejournal.umm.ac.id/index.php/sainmed

A Case of Heart Failure due to Triple Vessel Coronary Artery Disease: Role of General Practitioner

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Received: August 10th2022. Revised: September 6th2022. Published: Dec 30th2022

DOI: https://doi.org/10.22219/sm.Vol18.SMUMM2.17370

ABSTRACT

Heart failure with reduced ejection fraction (HFrEF) is a complex clinical syndrome characterized by a left ventricular ejection fraction ≤ of 40%. Primarily the etiology of heart failure is due to coronary artery disease (CAD). Patients with multivessel disease are considered for revascularization procedures with coronary artery bypass grafting (CABG). We report patient with HFrEF due to three-vessel disease (3VD) is not indicated for revascularization and only gets a conservative therapy to improve the patient's quality of life. A 52-year-old man visited the cardiologist to get control his condition. The patient experienced shortness of breath, heartburn, cold sweat, and fatigue. The physical and blood examination was normal, electrocardiography showed fragmented QRS complex, V1-V3 poor R waves progression, and V5-V6 T waves inverted. The echocardiography showed a result of ejection fraction is only 30%. He was treated with acetylsalicylic acid, bisoprolol, valsartan, furosemide, nitroglycerin, spironolactone, and isosorbide dinitrate. Treatment with CABG was not carried out. The ejection fraction was raised to 40% and he was clinically improved. HFrEF due to CAD3VD could be treated with fully conservative therapy and not indicated for revascularization treatment because of coronary anatomy and myocardial viability. A comprehensive approach should be evaluated by Heart Team.

Keywords: CABG, CAD, Conservative Therapy, General Practitioner, HfrEF.

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INTRODUCTION

Heart failure (HF) is a clinical syndrome characterized by typical symptoms (e.g. breathlessness, ankle swelling, and fatigue) that may be accompanied by signs (e.g. elevated jugular venous pressure, pulmonary crackles, and peripheral edema) caused by a structural and/or functional cardiac abnormality, resulting in reduced cardiac output and/ or elevated intracardiac pressures at rest or during stress (Ponikowski P, et al. 2016).

HF is a progressive health problem with high mortality and morbidity rates in both developed and developing countries, including Indonesia. In Indonesia, the age of heart failure patients is relatively younger than in Europe and America accompanied by a more severe clinical appearance. The prevalence of heart failure itself is increasing because patients who experience acute heart damage can progress to chronic heart failure. The World Health Organization (WHO) describes that the increasing number of heart failures in the world, including in Asia, is caused by the increasing number of smokers, the level of obesity, dyslipidemia, and diabetes. The incidence of heart failure also increases with age (Siswanto B.B., et al., 2020).

The estimated population incidence of HF in the developed countries ranges from 1% to 2% and at least one-half have HFrEF. HFrEF is commonly defined as a reduction in left ventricular ejection fraction (LVEF) to ≤40%, with CAD is the most common etiology for HFrEF and has increased with the growing incidence of associated mortality. This unfavorable prognosis is related to the progressive nature of ischemic left ventricle (LV) dysfunction and underlying comorbidities, such as chronic kidney disease, diabetes, and hypertension. (Hawranek M, et al., 2018, Wolff G, et al., 2017)

Contemporary pharmacology for most patients with HFrEF encompasses triple therapy, which includes the combination of (1) an ACEi (or ARB if ACEi-intolerant); (2) a b-blocker; and (3) an MRA. Working on various and complex pathways of the neurohormonal system, the combination of these agents has been shown to improve survival and quality of life in patients with HFrEF. Recently, trials and guidelines have demonstrated improved outcomes in people with HFrEF receiving sodium-glucose co-transporter 2 inhibitors (SGLT2i) as known as glifozin, and its added as four main pillars of heart failure. The presence of multivessel coronary artery disease (MVCAD) indicates an unfavorable prognosis and higher mortality than single vascular disease. In MVCAD, revascularization can be achieved either with percutaneous coronary intervention (PCI) or CABG. (De Innocentiis C, et. al., 2018)

This study is to report a patient with HFrEF due to three-vessel disease (3VD) is not indicated for revascularization treatment and only gets a conservative medication to improve the patient's quality of life. Also, to recognize the role of the general practitioner (GP) in the management of patients with heart failure.

CASE DESCRIPTION

A 52-year-old man "MN" and his wife visited our outpatient cardiac polyclinic with chronic heart failure. The patients experienced shortness of breath, heartburn, cold sweat, and fatigue. The shortness of breath as if feeling stabbed from the front of the chest and spreads to the back, the patient feels his complaints arise when the position changes and got stressed.

Blood pressure was 94/62 mmHg and heart rate was 88 bpm. Physical examination was normal. Blood examination results were also normal. He didn't have any underlying diseases such as diabetes, dyslipidemia, high uric acid, and kidney disease.

The result of electrocardiography was shown in figure 1. The echocardiography results were eccentric left ventricular hypertrophy (LVH), dilatation left atrium (LA), ejection fraction was 30%, regional wall motion abnormalities (RWMA) with decreased LV systolic function, grade II LV diastolic dysfunction (pseudonormal), mild mitral regurgitation (MR), and normal right ventricle (RV) systolic function.

He was treated with acetylsalicylic acid, bisoprolol, valsartan, furosemide, nitroglycerin, spironolactone, and isosorbide dinitrate. Treatment with PCI and CABG was not carried out because the cardiologist and Heart Team evaluate that the outcome would not be satisfactory due to the occlusion of plaque is too many and mostly placed on the proximal artery showed by the coronary angiography results in Figure 2. After two years of conservative medication, the ejection fraction was improved become 40%. Then he was clinically improved and felt better.

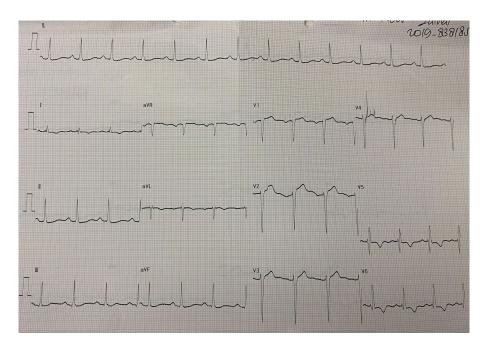


Figure 1. Electrocardiography showed sinus rhythm, regular, I fragmented QRS complex, V1-V3 poor R waves progression, V5 & V6 T waves inverted.

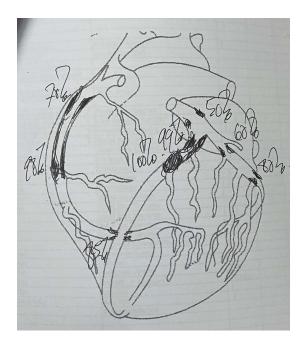


Figure 2. Result of coronary angiography, RCA 70% stenosis on proximal, 98% on mid, and 85% on distal. LM 50% stenosis on distal. LAD 99% stenosis on ostial and total occlusion on proximal. LCX 60% stenosis on proximal and 80% on distal.

DISCUSSION

The main terminology used to describe HF is based on the measurement of the LVEF. HF comprises a wide range of patients, from those with normal LVEF [typically considered as ≥50%; HF with preserved EF (HFpEF)] to those with reduced LVEF [typically considered as < 40%; HF with reduced EF (HFrEF)]. Patients with an LVEF in the range of 40 – 49% represent a 'grey area', which we now define as HFmrEF.¹ MVCAD is defined by the presence of ≥50 % diameter stenosis of two or more epicardial coronary arteries. The presence of MVCAD indicates a poorer prognosis and significantly higher mortality than the single-vessel disease (De Innocentiis C, et. al., 2018).

Diagnosis and treatment of heart failure aim to reduce morbidity and mortality of patients circumstances. Preventing the worsening of heart disease remains an important part of the management of heart disease. In addition, it is important to recognize and consider the treatment of accompanying cardiovascular and noncardiovascular comorbidities. Management of HFrEF is to relieve symptoms and signs, improve the quality of life, reduce fatigue and shortness of breath, reduce hospitalization requirements, and provide end-of-life care (Siswanto B.B., et al., 2020).

Optimal medical therapy with neuro-hormonal antagonists (ACEIs, MRAs, and betablockers) have been shown to improve survival in patients with HFrEF and are recommended for the treatment of every patient with HFrEF unless contraindicated or not tolerated. Recently, the trial has shown better results in people with HFrEF who received a sodium-glucose co-transporter 2 inhibitor (SGLT2i), dapagliflozin, and empagliflozin. The efficacy of SGLT2i in addition to standard therapies for people with HFrEF has been confirmed reducing nearly 25% risk of the primary endpoint of cardiovascular death or hospitalization for heart failure. However, it is currently unclear how these agents will be used in conjunction with established therapies. (Straw S, 2021)

ACE inhibitors are first-line therapy for heart failure patients. This class of drugs should be administered to patients with a left ventricular ejection fraction of \leq 40%. The mechanism of action of ACE-inhibitors is to inhibit the change from angiotensin I to angiotensin II which is mediated by ACE (Angiotensin Converting Enzyme). That way, the amount of angiotensin II will decrease followed by the amount of aldosterone. The reduction of these hormones will prevent myocardial fibrosis, myocyte apoptosis, cardiac hypertrophy, norepinephrine release, vasoconstriction, and fluid retention (Nurkhalis N, 2020).

ARB drugs work by blocking subtype 1 (AT1) angiotensin II receptors. Thus, the effects of angiotensin II will be inhibited. The impact of blocking AT1 receptors, namely vasodilation and inhibition of ventricular worsening. Because ARB drugs do not inhibit ACE, they do not affect bradykinin activity. Bradykinin is an inflammatory mediator that can cause coughing. Therefore, ARBs are usually given to patients who are intolerant of ACE inhibitors, especially coughing. Betablockers have been shown to reduce heart failure mortality by acting selectively to block β 1 receptors while carvedilol blocks β 1, β 2, and α 1 receptors. Spironolactone and eplerenone are aldosterone antagonists that work by blocking mineralocorticoid receptors. In the kidney, aldosterone antagonists inhibit sodium reabsorption and potassium excretion. So that aldosterone antagonists also have a diuretic effect. In the heart, aldosterone antagonists inhibit the formation of collagen and matrix deposits. Collagen and matrix deposits are one of the triggers for cardiac fibrosis and ventricular remodeling (Nurkhalis N., 2020).

When compared with medical therapy alone, coronary revascularization is a better action in improving survival in patients with HF of ischaemic origin and is recommended in clinical practice. However, the optimal revascularization strategy is not defined. The choice between CABG and PCI should be made by the cardiologist and team after a comprehensive evaluation of the patient's clinical status. The team should be considered and analyze the patient's coronary anatomy, expected completeness of revascularization myocardial viability, coexisting valvular disease, and comorbidities. There are currently no dedicated randomized clinical trials comparing PCI vs. medical therapy in patients with HFrEF. In addition, CABG vs. PCI randomized trials have excluded patients with severe HF (Neumann FI, et al., 2018).

Contraindication for CABG procedure is not considered appropriate in asymptomatic patients who are at low risk of MI or death. Patients who will experience little advantage from

coronary revascularization are also excluded. Although advanced age is not a contraindication, CABG should be carefully considered in the elderly, especially those older than 85 years. These patients are also more likely to experience perioperative complications after CABG. A multidisciplinary heart team approach that emphasizes shared decision-making in patients with multivessel CAD is essential to offer the patient the best chance of a successful revascularization strategy. A meta-analysis that examined data from Pubmed, Embase, and Cochrane Central stated that in stable coronary heart disease in the PCI and conservative treatment groups there was no statistically significant difference in survival rate (Setiawan AA, 2016).

The GPs play an essential role in the management of HF as the first clinical presentation usually takes place in the primary care setting, and as they are responsible for the daily follow-up of chronic HF patients. GPs has to deal with careful monitoring of early signs and adherence to medication, early intervention with appropriate medication, and active engagement with other specialists involved with the care team while at all times engaging with the family and caregivers to keep the patient following doctor's instructions to get a better quality of life (Piterman L, 2018, Verhestraeten C, et al., 2020).

CONCLUSION

HFrEF due to CAD3VD could be treated with optimal medical therapy to improve the quality of life and reduce hospitalization. Some patients are not indicated for revascularization procedure because of coronary anatomy and myocardial viability of the patient circumstances. A comprehensive and holistic approach should be evaluated by Heart Team. Multidisciplinary chronic care for HF between the cardiologist and the GPs needs to be considered.

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