

MANAGEMENT OF CHRONIC HEART FAILURE IN PRIMARY CARE: WHAT EVIDENCE DO WE HAVE FOR HEART FAILURE WITH PRESERVED SYSTOLIC FUNCTION?

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ABSTRACT

Chronic Heart Failure (CHF) is a debilitating illness commonly encountered in primary care. Its prevalence in developing countries is rising as a result of an ageing population, and an escalating epidemic of hypertension, type 2 diabetes and coronary heart disease. CHF can be specifically diagnosed as Heart Failure with Reduced Systolic Function (HF-RSF) or Heart Failure with Preserved Systolic Function (HF-PSF). This paper illustrates a common presentation of HF-PSF in primary care; and critically appraises the evidence in support of its diagnosis, prognosis and management. Regardless of the specific diagnosis, long term management of CHF is intricate as it involves a complex interplay between medical, psychosocial, and behavioural factors. Hence, there is a pressing need for a multidisciplinary team management of CHF in primary care, and this usually takes place within the broader context of an integrated chronic disease management programme. Primary care physicians are ideally suited to lead multidisciplinary teams to ensure better co-ordination, continuity and quality of care is delivered for patients with chronic conditions across time and settings. Given the rising epidemic of cardiovascular risk factors in the Malaysian population, preventive strategies at the primary care level are likely to offer the greatest promise for reducing the growing burden of CHF.

Keywords: Chronic heart failure, preserved systolic function, chronic disease management, primary care, Malaysia.

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CASE SCENARIO

Madam YW, a 70-year-old woman with a long history of hypertension, type 2 diabetes mellitus and osteoarthritis; presented to her primary care physician complaining of increasing shortness of breath while walking over the last six months. She noted swelling in her feet and ankles. Her breathlessness worsened over the last three days, where she has been unable to lie flat in bed at night and has been sleeping on three pillows.

This lady lives with her 76-year-old husband who has recently been diagnosed with colon cancer. Her daughter, who lives

nearby, noted that her mother has been deeply affected by the news. She complained of feeling tired all the time, and has been unable to cope with looking after him and doing her usual house chores. She is a non-smoker and does not drink alcohol.

Her current medications are as follows:

- o Hydrochlorothiazide 50 mg *od*.
- o Metformin 1 g *bd*.
- o Gliclazide 160 mg *bd*.
- o Diclofenac 50 mg *tds. prn*.

She admitted that she has not been taking her medications regularly and has frequently defaulted on her follow up appointments.

On examination, this lady has a body mass index (BMI) of 31 kg/m² and waist circumference of 98 cm. Her pulse rate was regular at 95 beats per minute, blood pressure was 170/90 mmHg and her jugular veins were noticeably distended. Her apex beat was displaced to the sixth left intercostals space and auscultation of the heart revealed normal heart sounds. There were bibasal crepitations heard on auscultation of her

lungs, and lower limbs examination revealed bilateral pitting oedema of feet and ankles.

Chest X-ray (CXR) and Electrocardiogram (ECG) were performed. Results are shown in Figure 1 and 2 respectively.

Madam YW was suspected to have an acute heart failure and was then admitted to the nearest hospital for stabilisation and confirmation of diagnosis. One week later, she was discharged from the hospital to her primary care physician with the following information:

Trans-thoracic echocardiography report:

Concentric LV hypertrophy with normal left ventricular ejection fraction (LVEF) of 55%.

Bi-atrial dilatation, impaired LV filling and elevated filling pressure.

Cardiac stress perfusion study (thallium):

No evidence of reversible ischaemia. LVEF of 55% is confirmed.

Blood results:

HbA_{1c}: 7.5%

Renal profile (RP): normal

Liver function test (LFT): normal

Full blood count (FBC): normal

Fasting serum lipid (FSL): TC 6.1, LDL-C 3.0, TG 2.1, HDL-C 1.19 (mmol/litre)

Urinalysis:

Urine protein negative, urine microalbumin negative.

Figure 1: Madam YW's Chest X-ray showing bilateral pulmonary congestion and cardiomegaly



Figure 2: Madam YW's 12-lead ECG showing large voltages, left axis deviation, ST-T strain pattern in lateral chest leads (without digoxin) and interventricular conduction delay typical of left ventricular hypertrophy (LVH)



Medication on discharge:

- o Frusemide 40 mg *bd*.
- o Hydrochlorothiazide 50 mg *od*.
- o Bisoprolol 2.5 mg *od*.
- o Perindopril 4 mg *od*.
- o Metformin 1 g *bd*.
- o Gliclazide 160 mg *bd*.
- o Simvastatin 40 mg *nocte*.
- o Aspirin 75 mg *od*.
- o Slow K 600 mg *od*.
- o Paracetamol 1 g *qds*.

Clinical Questions

1. How do we make the diagnosis?
2. How common and how significant is this condition?
3. How do we manage this lady?

REVIEW OF EVIDENCE

Diagnosing Heart Failure

Heart Failure (HF) is a common chronic condition encountered in primary care. The prevalence of HF in developing countries is gradually rising as a result of an ageing population, and an escalating epidemic of coronary heart disease, hypertension, type 2 diabetes and obesity.¹ In Malaysia, HF accounts for 6%-10% of all medical admissions.^{2,3} The economic burden of HF is substantial, in which the cost of hospitalisations represents the greatest proportion of total expenditure.⁴

This case illustrates a common primary care presentation of an Acute Heart Failure (AHF), which is defined as rapid onset of symptoms and signs due to acute deterioration of cardiac function.⁵ Patients with AHF may present *de novo* or as an acute decompensation of chronic heart failure (CHF).⁵ In the case of Madam YW - an elderly, obese woman with long history of hypertension and type 2 diabetes, it is more likely that she has an acute decompensation of an undiagnosed CHF which may have developed gradually. There are several common factors which can precipitate an acute cardiac

decompensation, and in this scenario it may have been contributed to her uncontrolled hypertension, excessive salt and water intake, and adverse effects of non-steroidal anti-inflammatory drug (NSAID). Exercise capacity should be assessed to determine functional class in CHF patients (Table 1)⁵, and this lady is most likely to be in the New York Heart Association (NYHA) Class III (marked limitation of physical activities such as walking and doing her house chores).

HF however, may present insidiously in the elderly, obese and in women, making it difficult to interpret.⁵ It is absolutely vital that primary care physicians recognize these characteristics early and have a high index of suspicion especially in patients with cardiovascular co-morbidities and risk factors.

Patients suspected to have AHF require hospital admission for i) stabilisation of haemodynamics ii) management of symptoms aimed at reducing pulmonary venous pressure and congestion iii) identification and management of the underlying cause or precipitating factors and iv) confirmation of diagnosis by echocardiography.⁵⁻⁷ CXR and ECG should be performed in primary care where available;⁵ and when the patient is relatively stable. Natriuretic peptides, which may be useful as a 'rule-out' test in doubtful cases,⁵ is not commonly available in Malaysian primary care setting.

This lady's echocardiogram confirmed that she has a normal systolic function with pre-dominantly diastolic dysfunction. Diastolic dysfunction can also occur in asymptomatic patients; therefore, it is important to distinguish this terminology with diastolic heart failure (DHF).^{8,9} Controversies surrounding the optimal diagnostic criteria for DHF is a subject of ongoing debate in the literature.^{10,11,12} The recently updated consensus statement by the European Society of Cardiology suggests that the diagnosis of DHF is made when three criterias are fulfilled simultaneously; i) presence of sign or symptoms of congestive HF ii) objective evidence of normal or mildly abnormal left ventricular systolic function (LVEF >50%) and iii) objective evidence of left ventricular diastolic dysfunction (abnormal relaxation, filling, diastolic distensibility or stiffness).¹² It is well known that the gold standard assessment for left ventricular diastolic dysfunction is by cardiac

Table 1: The New York Heart Association (NYHA) functional classification

NYHA grading	Functional description	Untreated 1 year mortality
Class I	No limitation. Ordinary physical activity does not cause fatigue, dyspnoea or palpitation.	5-10%
Class II	Slight limitation of physical activity. Such patients are comfortable at rest. Ordinary physical activity results in fatigue, dyspnoea, palpitation or angina.	10-15%
Class III	Marked limitation of physical activity. Less than ordinary physical activity leads to symptoms.	15-20%
Class IV	Inability to carry on any physical activity without discomfort. Symptoms of CHF present at rest.	20-50%

catheterization,¹² but this invasive procedure is not practical or widely available especially in developing countries. The Malaysian Clinical Practice Guidelines (CPG) for Management of HF recommends trans-thoracic echocardiography as a mean of diagnosis for diastolic dysfunction.⁵ It is also important to rule out cardiac ischaemia as a potential underlying cause for HF,^{5,6} therefore cardiac stress perfusion study was performed in this case.

DHF has also been described as Heart Failure with Preserved left ventricular Systolic Function (HF-PSF) in the literature.^{5,6,13,14} There is an ongoing debate regarding the most appropriate terminology for this condition.¹⁵

How common and how significant is HF-PSF?

Current epidemiological evidence from various parts of the world have established that HF-PSF is just as common as HF with reduced left ventricular Systolic Function (HF-RSF), representing 50% of all patients with heart failure.^{14,16-22} Compared to the latter, individuals presenting with HF-PSF are more likely to be female, older, have hypertension and atrial fibrillation; and less likely to have coronary heart disease.^{14,16,17,18,20,22} The prevalence of HF-PSF was found to have increased over time, and was significantly higher among community patients.¹⁸ A survey in everyday practice has also shown that elderly, female patients with HF were more likely to be managed in primary care as compared to secondary care.²³

The clinical presentation of patients with HF-PSF is similar to those with HF-RSF.¹⁴ In pathophysiological terms, the symptoms and signs of HF-PSF result from elevated left ventricular filling pressure due to diminished relaxation and reduced distensibility during diastole, while its contractility during systole (ejection fraction) is preserved.⁸

A growing body of evidence now shows that HF-PSF carries a comparably poor prognosis to HF-RSF.¹⁸⁻²² The recently published data from OPTIMIZE-HF registry comparing 21,149 patients with HF-PSF and 20,118 patients with HF-RSF shows that both groups had similar hospital readmission rates (29.2% vs. 29.9%; $p=0.591$) and similar mortality risks (9.5% vs. 9.8%; $p=0.459$).²⁰ Another study from Japan shows similar outcome where the combined all cause mortality or rehospitalisation rates did not differ between the two groups (adjusted HR 1.082 and 95%CI 0.858-1.365; $p=0.507$).²² Five year survival rates were also not significantly different in patients with HF-PSF compared to HF-RSF (43% vs. 46%; $p=0.95$).²¹

How do we manage this lady?

Regardless of whether the diagnosis is HF-PSF or HF-RSF, long term management of patients with CHF in primary care is intricate as they often present with multiple co-morbidities

which require persistent lifestyle modification and multiple drug therapies. As illustrated in the case of Madam YW, not only that she has osteoarthritis and metabolic syndrome (central obesity, dyslipidaemia, hypertension and type 2 diabetes);²⁴ she is also frail and not coping with her activities of daily living. The fact that her husband is terminally ill adds to her psychological stress and she may be at risk of developing depression. Prevalence of depression has been reported to be as high as 39% in patients with CHF, and it is related to a higher risk of functional decline.²⁵ Therefore, all individuals presenting with CHF should be routinely screened for depression.⁶

Management goals and lifestyle interventions

In principal, management goals for HF-PSF are similar to those with HF-RSF. The goals are to i) relieve symptoms ii) manage underlying causes and risk factors iii) improve physical activity tolerance and quality of life iv) reduce hospital readmissions and v) improve survival.^{5,6} Lifestyle and behavioural modification are pivotal to the successful management of CHF. Therefore, patients and their families should be educated and counselled regarding the diagnosis, potential complications, prognosis and management goals; and be empowered with skills to self care.^{5,6}

Management of congestive symptoms should be directed at achieving euvolaemia with diuretics, fluid restriction of <1.5 L/day and sodium restriction <2 gm/day (translated to no added salt to food or cooking).^{5,6} It is important to note however, that over-diuresis with diuretic therapy can cause intravascular contraction and deterioration of renal function. Therefore, patients with CHF should be educated and empowered with skills to manage and monitor their volaemic state e.g. by daily weighing and flexible titration of diuretic regimen.⁶ They need to seek medical help if there is a change of more than 2 kg in weight over two to three days.^{5,6}

Regular physical activity has also been shown to be highly beneficial in patients with CHF.^{5,6} It should be tailored to the individual's capacity, and this may include light exercise such as walking for 20-30 minutes daily, five to seven days per week.^{5,6} Participation in a supervised group exercise programme offers the opportunity of additional socialisation, support and education.⁶

Pharmacological management

While the evidence to support pharmacological treatment of HF-RSF is now well established, the enigma surrounding therapeutic options for HF-PSF is continuing. The survival rates of patients with HF-PSF remain unaltered over time as opposed to those with HF-RSF, despite frequent use of similar pharmacological agents.¹⁸ The CHARM-Preserved trial, which randomised 3,023 patients with HF-PSF to either candesartan

or placebo, failed to demonstrate significant effect on cardiovascular mortality, but there were significantly fewer hospital readmissions in the candesartan group.²⁶ The PEP-CHP study showed no difference in mortality and HF hospitalisations when perindopril was compared to placebo in elderly individuals (>70 years) with HF-PSF.²⁷ The I-PRESERVE trial also showed no improvement in mortality and hospitalisation rates when irbesartan was compared to placebo.²⁸ A recent review which summarised all the major outcome trials involving patients with HF-PSF to date concluded that these trials had excessively recruited HF patients with eccentric LV remodelling and ischaemic heart disease compared to patients with concentric LV remodelling and hypertension.²⁹ More clinical trials which adhere to diagnostic guidelines for HF-PSF are needed to form an evidence-based therapeutic decision. Pending completion of these trials, treatment for HF-PSF remains largely empirical with many guidelines recommending the use of similar pharmacological agents as used for HF-RSF.^{5,6}

In the case of Madam YW, it is important to note that her β -blocker and ACE-Inhibitor (ACE-I) require an up-titration to optimal dose following discharge. An international survey showed that primary care physicians prescribed ACE-I at 50% of the target doses suggested by guidelines and were generally reluctant to use β -blockers in CHF patients.³⁰ Using a guideline-based computerised decision support system was shown to have an impact on primary care physicians' confidence in the diagnosis and management of CHF.³¹ It is also important to note that her NSAID has been discontinued, as it can cause fluid retention, renal dysfunction and vasoconstriction.⁵

Management of risk factors

Since the evidence-base to support pharmacological management of HF-PSF is not yet established, identifying the underlying cause and aggressive management of risk factors has become the primary focus.^{5,6} Hypertension plays a central role in the pathophysiology of HF-PSF.³² In the case of Madam YW, it is clear that her long history of uncontrolled hypertension has induced compensatory hypertrophy of her left ventricular wall (LVH), which in turn contributed to the HF-PSF. Severe uncontrolled hypertension is also a common precipitator for acute cardiac decompensation.³² Treating hypertension to target level has been shown to reduce the incidence of CHF by as much as 50%.³³ Therefore, it is absolutely vital that her blood pressure, lipid level, glycaemic control and weight are managed according to guidelines' recommendations.^{34,35} Antihypertensive agents such as ACE-I or ARB which has been shown to be beneficial in promoting LVH regression should be used.^{5,6} ACE-I is also the first line agent of choice recommended for treating hypertensive patients with type 2 diabetes without proteinuria.^{34,35}

Addressing medication adherence

Madam YW also admitted to have poor adherence to her medications and follow up appointments prior to her hospital admissions. With the additional morbidity of having CHF, this lady is now on polypharmacy of ten different medications. Only 50% of patients with chronic diseases have been reported to adhere to their medications.³⁶ Non-adherence significantly contributes to increasing morbidity and mortality, as well as wastage of health care resources.³⁷ A Cochrane systematic review shows that interventions which were effective in improving medication adherence for long term care were complex and multifaceted; including combinations of more convenient care, information, reminders, self-monitoring, reinforcement, counselling, family therapy, psychological therapy, crisis intervention, manual telephone follow up, and supportive care.³⁸

Bridging the gap in long term heart failure management in the community

In summary, this case illustrates the complex interplay between medical, psychosocial, and behavioural factors in the management of patients with CHF. This underscores the need for a comprehensive health care delivery in long term management of these patients in the community.

There is now substantial evidence that multidisciplinary strategies in CHF management reduce HF hospitalisations, improve quality of life and save costs.³⁹⁻⁴² A systematic review suggests that patient education to enhance self care, follow up monitoring by specially trained staff, and access to specialised HF clinics are the most efficacious approaches.³⁹ Strategies which employed telephone contact and advised patients to attend their primary care physicians in the event of deterioration were shown to reduce hospitalisations.³⁹ Home visits by pharmacist are pertinent in optimising medication adherence,⁴³ and the inclusion of a pharmacist in the multidisciplinary team has been shown to reduce HF hospitalisations.⁴⁴ Multidisciplinary care teams, centred on primary care, are highly effective in improving coordination of care, disease control and health outcomes.⁴⁵ Therefore, long term care of CHF patients in the community warrants a multidisciplinary team management involving primary care physician, nurse, pharmacist, physiotherapist and psychologist; and it must be well supported by the secondary care services.^{7, 46}

In primary care, multidisciplinary management of CHF usually takes place within the broader context of an integrated chronic disease management. Numerous models of care have been developed and the most notable is the Chronic Care Model (CCM).⁴⁷ Primary care practice redesigned in accordance with the CCM has been shown to improve the quality of care and outcomes for patients with various chronic conditions.^{48,49}

However, chronic disease management in the Malaysian primary care settings, be it in the public or private sector; is still largely being done in a sporadic, unplanned and uncoordinated manner.⁵⁰ There is an urgent need to address this issue, and the CCM provides a useful framework for the restructuring of healthcare delivery across all levels of service provision.⁵¹ The recently announced proposal by the Malaysian government to establish a national health financing scheme which will integrate all public and private primary care clinics under a common network of care, offers the promise of better coordination and quality of care for chronic conditions.⁵²

In response to this, Malaysia has now produced more than 400 highly trained specialist primary care physicians, who are in an ideal position to champion patient-centred chronic disease care and take a leadership role in system redesign from a grassroots' perspective. These numbers, however, are far from adequate. Imperative measures are currently being undertaken by the public universities and the Academy of Family Physicians Malaysia (AFPM) to produce more primary care/family medicine specialists in order to meet the country's demand. Availability of highly trained allied health personnel is also critical to support the deployment of multidisciplinary teams in primary care. There must be a constant endeavour

to increase the numbers of highly skilled nurses, pharmacists, dieticians, physiotherapists, psychologists and other allied health personnel to deal with the challenge of managing chronic conditions. Primary care physicians must ultimately become the agent of change and lead multidisciplinary primary care teams to ensure that better coordination, continuity and quality of care is delivered for patients with chronic disease across time and settings.

A case for prevention

The incidence of CHF is likely to escalate in the Malaysian population as hypertension, type 2 diabetes and coronary heart disease remain the most common risk factors. The prevalence rates for hypertension and diabetes in Malaysian adults >30 years are reaching epidemic proportion at 42.6% and 14.9% respectively.⁵³ The overall control rates for both conditions however, have remained alarmingly poor over the past 20 years,⁵³ despite the existence of succinct clinical practice guidelines and improved availability of pharmacological agents in primary care. If this trend continues, Malaysia would not be able to cope with the impending burden of treating complications of these conditions; which is already causing strain to its health system. Preventive strategies at the primary

Table 2: Similarities and differences between HF-PSF and HF-RSF

Elements	HF-PSF	HF-RSF
Epidemiology ^{14, 16-18, 20-21}	<ul style="list-style-type: none"> o 50% of HF o More likely to be female and/or elderly o More likely to have hypertension and/or atrial fibrillation 	<ul style="list-style-type: none"> o 50% of HF o More likely to be male o More likely to have coronary heart disease
Prognosis ¹⁸⁻²²	Similar rehospitalisation, all-cause mortality and five years survival rates	
Symptoms and signs ⁵	Breathlessness, ankle swelling, fatigue, reduced exercise capacity, peripheral oedema, elevated jugular venous pressure, hepatomegaly	
Diagnosis ⁵⁻⁷	Can be diagnosed with trans-thoracic echocardiogram	
Management goals ⁵⁻⁷	i) relieve symptoms ii) manage underlying causes and risk factors iii) improve physical activity tolerance and quality of life iv) reduce hospital readmissions v) improve survival	
Lifestyle interventions ⁵⁻⁶	Fluid restriction <1.5 L/day, sodium restriction <2 gm/day and light exercise (e.g. walking for 20-30 minutes daily, 5-7 days/week)	
Pharmacological management ⁵	<ul style="list-style-type: none"> o Diuretics – relieve congestive symptoms o Empirical use of ACE I, ARB and β-blockers pending completion of more clinical trials 	<ul style="list-style-type: none"> o Diuretics – relieve congestive symptoms o ACE I and β-blockers – improve symptoms and survival, and delay progression o ARB – for patients intolerant of ACE I o Aldosterone antagonists – improve survival and reduce hospitalisation o Digoxin – reduce hospitalisation when added to optimal medical therapy, no effect on survival

care level, directed towards an early and aggressive management of cardiovascular risk factors, effective patient education and lifestyle change, improved prescribing habits; and efficient delivery of multidisciplinary care, are likely to offer the greatest promise for reducing the burden of HF morbidity and mortality.

Summary and recommendations

This case illustrates a common presentation of HF-PSF in primary care. While its epidemiology, pathophysiology and prognosis are well established, the enigma surrounding its therapeutic options is continuing. Table 2 summarises the

Table 3: Management recommendations for Madam YW

Case summary Madam YW, 70-year-old lady was recently diagnosed to have HF-PSF. Her co-morbidities include metabolic syndrome (central obesity, hypertension, type 2 diabetes mellitus, dyslipidaemia), osteoarthritis, frailty and risk of depression. She has an increased HbA_{1c} of 7.5%, TC of 6.1, LDL-C of 3.0 and TG of 2.1. Her RP, LFT and FBC were normal. This lady was discharged to her primary care physician for the up-titration of her medications and long term care. She was given a follow up appointment to be seen at a cardiology clinic in one month.

Role of primary care physician and allied health team

- o Educate Madam YW and her family regarding the diagnosis, potential complications, prognosis and self management goals.
- o Motivate her to change her lifestyle – reduce fluid intake to <1.5 L/day, reduce salt and fat intake, increase fibre intake, perform regular light exercise and reduce weight.
- o Empower her with skills to self care – chart daily weight and seek medical help if there is a change of >2 kg in her weight over 2-3 days.
- o Assess her fluid status at every follow-up – weight change, JVP, lung crackles, lying and standing BP, peripheral oedema.
- o Adjust her diuretic therapy (frusemide) according to her fluid status and kidney function (usual daily dose 20-80 mg).⁵⁻⁷
- o Continue combination with thiazide diuretic (hydrochlorothiazide, usual daily dose 25-50 mg) as these drugs work synergistically to improve diuresis.
- o Titrate her ACE I (perindopril) to target dose (8 mg *od.*) at 2-4 weekly intervals.⁵⁻⁷
- o Titrate her β -blocker (bisoprolol) to target dose (10 mg *od.*) at 2-4 weekly intervals.⁵⁻⁷
- o Treat blood pressure to target (<130/80 mmHg).³⁴
- o Monitor side effects (e.g. cough due to ACE I), heart rate and rhythm (bradycardia due to β -blockers) and blood pressure (SBP <100 mmHg requires specialist referral).⁵
- o Monitor RP 1-2 weeks after initiation and 1-2 weeks after final dose titration (in particular potassium, urea and creatinine).⁵⁻⁷
- o Monitor her glycaemic control (target HbA_{1c} <7% - acceptable in elderly) and consider insulin if glycaemic control remains poor.³⁵
- o Titrate her HMG-CoA reductase inhibitor (simvastatin – maximum dose 80 mg *od.*) at 4 weekly intervals until her FSL achieve the target levels (TC<4.5, LDL-C<2.6, TG<1.7).³⁵
- o Once targets are achieved and her condition is stable - monitor RP, HbA_{1c}, FSL and LFT at least yearly.⁵⁻⁷
- o Assess adherence to medications, functional capacity, mental state, cognitive functions and nutritional status regularly.⁷
- o Arrange follow up visits of 2-4 weekly at the initial stage and 1-3 monthly thereafter.⁷
- o Arrange multidisciplinary home visits where necessary.

Problem solving⁵⁻⁷

- o Increasing congestion – up-titrate dose of diuretics (20 mg/kg of body weight every 3 days) and/or halve dose of β -blockers. Monitor renal function closely.
- o Marked fatigue (and/or bradycardia) - halve dose of β -blockers.
- o Low heart rate <50 beats/minute – stop or halve dose of β -blockers and arrange ECG.
- o Still hypertensive – consider changing to carvedilol (more powerful vasodilator) and/or add other antihypertensive agents (short acting nifedipine and diltiazem are contraindicated as they depress myocardial contractility).
- o Asymptomatic low BP – no change in therapy.
- o Low BP causing symptoms (e.g. dizziness) – discontinue other vasodilators. SBP <100 mmHg requires specialist referral.
- o Increase K⁺ >5.9 mmol/L and/or creatinine >200 μ mol/L or >50% above baseline – halve dose of ACE-I and seek specialist advice.
- o Serious deterioration – seek specialist advice/referral.

Note: β -blockers should not be stopped suddenly – seek specialist advice before discontinuation.

similarities and the differences between HF-PSF and HF-RSF, while Table 3 summarises the management recommendations for Madam YW.

CONCLUSION

In conclusion, long term care of CHF patients involves managing complex interplays between medical, psychosocial, and behavioural factors. This underscores the need for a multidisciplinary primary care team management which usually takes place within the broader context of an integrated chronic disease management programme. Primary care physicians must become the change agent and take a leadership role in system redesign from a grassroots' perspective. They are ideally positioned to lead multidisciplinary primary care teams to ensure better coordination, continuity and quality of care for patients with chronic conditions. Given the rising epidemic of cardiovascular risk factors in the Malaysian population, preventive strategies at the primary care level are likely to offer the greatest promise for reducing the growing burden of CHF.

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