Clinical Practice Guidelines

Malaysia CPG for Heart Failure

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Cardiovascular disease is an important cause of morbidity and mortality in Malaysia. Heart Failure (HF), the end stage of most diseases of the heart, is a common medical problem encountered in general practice and is an important cause of hospital admissions. With aging of the population the prevalence of HF is expected to increase.

The 1st Clinical Practice Guidelines (CPG) in HF was published in 2000 with the 2nd edition in 2007. This current document is an update of the last edition.

Objectives:
The objectives of this CPG are to assist the health care provider in:

- the prevention of HF
- the diagnosis and treatment of HF
- improving survival of patients with HF
- reducing the morbidity associated with the condition and improving the quality of life of these patients

Process:
Evidence was obtained by systematic review of current medical literature on HF using the usual search engines – PubMed and Ovid. International guidelines on HF were also studied. The draft was then submitted to the Technical Advisory Committee for Clinical Practice Guidelines, Ministry of Health Malaysia and key health personnel for review and feedback.

The level of recommendation and the grading of evidence used in this CPG was adapted from the American Heart Association and the European Society of Cardiology.

Clinical Questions Addressed:
- How do you make a diagnosis of HF.
- Who are individuals at high risk of developing HF and how do you prevent them from developing HF?
- How do you treat acute and chronic HF effectively using current knowledge and available resources?
- How do you prevent recurrent admissions for acute decompensated HF.
- How do you treat the following special groups?
  - the asymptomatic individual with reduced left ventricular (LV) function.
  - the individual with HF due to preserved LV function.
  - the pregnant patient with HF.
  - infants and children with HF.
  - the individual with refractory and terminal HF.

Target Group:
This CPG is directed at all healthcare providers treating patients with HF – general practitioners, general and family physicians, both adult and pediatric cardiologists and obstetricians.

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SUMMARY

- Heart Failure (HF) is a clinical diagnosis. To satisfy the definition of HF, symptoms, signs and/or objective evidence of cardiac dysfunction must be present. (Fig. 1)

- HF may be the result of any disorder of the endocardium, myocardium, pericardium or great vessels although commonly, it is due to myocardial dysfunction. It may occur in the presence of reduced left ventricular (LV) function, the left ventricular ejection fraction (LVEF) <40% (HFrEF) or with normal LV function, the LVEF > 50% (HF with with preserved LV function -HFpEF). If the LVEF is 41-49% it is called HFpEF, borderline.

- It may be classified as Acute HF or chronic HF depending on the acuteness of the clinical presentation.

- HF is not a complete diagnosis. It is important to identify the underlying disease and the precipitating cause(s), if present. Common causes are coronary artery disease and hypertension. Patients with Chronic HF may occasionally develop acute decompensation. Important causes that can lead to this Acute HF include acute myocardial infarction/myocardial ischemia, arrhythmias (e.g. atrial fibrillation) and uncontrolled Blood Pressure. (Fig. 2)

- Prevention and early intervention wherever appropriate, should be the primary objective of management. (Fig. 3)

- Management of HFrEF (both Acute HF and Chronic HF) and grades of recommendations are as outlined in Flow Charts 1 & 2 and Tables 1 & 2.

- Management of HFpEF remains empiric since trial data are limited.

- Non pharmacological measures includes counseling the patient and family about the disease, diet and fluid intake, regular exercise and appropriate lifestyle changes such as smoking cessation and abstinence from alcohol.

- HF in pregnancy and in children are best managed in tertiary centres.

- Performance measures should be instituted to assess quality of care.

LEVELS OF EVIDENCE AND GRADES OF RECOMMENDATIONS

<table>
<thead>
<tr>
<th>GRADES OF RECOMMENDATION</th>
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<tbody>
<tr>
<td>I</td>
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<tr>
<td>II</td>
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<tr>
<td>II-a</td>
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<tr>
<td>II-b</td>
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<tr>
<td>III</td>
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LEVELS OF EVIDENCE

<table>
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<tr>
<td>A</td>
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<tr>
<td>B</td>
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<td>C</td>
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Adapted from the American College of Cardiology Foundation / American Heart Association and the European Society of Cardiology (Available at: http://assets.cardiosource.com/Methodology_Manual_for_ACC_AHA_Writing_Committees and at http://www.escardio.org/guidelines-surveys/escguidelines/about/Pages/rules-writing.aspx).
Figure 1: Algorithm for the diagnosis of Heart Failure or LV dysfunction

Suspected Heart Failure because of symptoms/signs → ECG, Chest Radiograph, Natriuretic Peptides (where available)

- Tests abnormal
  - Echocardiography
    - Tests abnormal
      - Determine:
        - Underlying cause
        - Severity
        - Precipitating factors
        - Type of LV dysfunction (systolic +/- diastolic)
        - Treat accordingly
    - Additional diagnostic tests where appropriate (eg: Coronary Angiography, Nuclear Imaging & CMR)
  - Tests normal
    - Heart Failure or LV dysfunction unlikely. Consider other diagnosis such as:
      - coronary artery disease (angina equivalent),
      - pulmonary disease,
      - obesity
Fig 2: Factors Contributing to Decompensation in a Patient with Stable HF

**Patient factors**
- Non compliance to medications
- Dietary indiscretion especially salt and fluid intake
- Inappropriate medications e.g. NSAIDS and COX-2 inhibitors
- Alcohol consumption

**Cardiac Causes**
- Superimposed myocardial ischaemia or infarction (often asymptomatic)
- Uncontrolled hypertension
- Arrhythmias
- Pulmonary embolism
- Secondary mitral or tricuspid regurgitation

**Systemic Conditions**
- Superimposed infections
- Anemia
- Thyroid disease
- Electrolyte disturbances
- Worsening renal disease

Fig 3: Prevention of Heart failure

Identifying Individuals at High Risk of developing HF/CAD. These include those with:
- multiple risk factors for developing CAD or who already have evidence of atherosclerotic disease in other vascular beds (e.g. cerebral, peripheral vascular disease)
- hypertension
- diabetes
- the metabolic syndrome
- severe hyperlipidemia
- a family history of cardiomyopathy
- thyroid disorders
- renal disease
- cardiotoxins – excessive alcohol consumption, chemotherapeutic agents
- sleep-disordered breathing especially obstructive sleep apnoea

Individuals with cardiac disease but who do not as yet have evidence of myocardial dysfunction and who should be treated according to guidelines. These include patients with:
- acute coronary syndrome.
- Coronary artery disease
- Hypertension and left ventricular hypertrophy
- Valve disease
- Arrythmias
- Congenital cardiac lesions

Individuals with myocardial dysfunction but who do not as yet have signs and symptoms of HF. (Asymptomatic Left Ventricular Dysfunction)
- Treat underlying cause
- Prevent progression to HF
Flowchart I: Management of Acute HF

NOTE:
* It is important to look for tissue hypoperfusion - cool peripheries, sweating, low volume pulse, decreasing urine output
** Flow Chart II

From onset, evaluate to identify correctable/reversible lesions
Special situations: Myocardial ischaemia / infarction: Treat accordingly Hypertension: Control BP quickly Valvular heart disease: Corrective surgery/balloon valvuloplasty
Flowchart II: Optimizing Drug Therapy in Chronic HF

SIGN & SYMPTOMS OF Volume Overload

- ACE-I (or ARB if ACE-I intolerant)
- β-blockers

Yes

- ACE-I (or ARB if ACE-I intolerant)
- Diuretics

No

Clinical Improvement

- Add:
  - MRA
  - Consider β-blocker
  - If no pulmonary congestion

Yes

Clinical Improvement

- Add:
  - Digoxin
  - And/or ivabradine (if sinus rhythm & HR > 70bpm)

No

Clinical Improvement

- See flowchart I (pg12)
  - Loop diuretics + thiazides
  - Short term parenteral positive inotrops
  - Consider if suitable:
    - CRT
    - IABP
    - VAD
    - Cardiac transplant

Yes

- Continue with:
  - Diuretics: low maintenance dose
  - ACE-I/ARB: titrate to max tolerated dose
  - + β-blocker

No

Clinical Improvement

- Continue with:
  - Diuretics
  - ACE-I / ARB
  - MRA
  - (if not already on)
### Table I: Grading of Recommendations in the Management of Acute HF

<table>
<thead>
<tr>
<th>Intervention</th>
<th>Grades of Recommendation</th>
<th>Levels of Evidence</th>
<th>Comments</th>
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</thead>
<tbody>
<tr>
<td><strong>INITIAL MANAGEMENT CONSISTS OF:</strong></td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>Oxygen</td>
<td>I</td>
<td>C</td>
<td>Maintain the oxygen saturation above 95%</td>
</tr>
<tr>
<td>Diuretics</td>
<td>I</td>
<td>B</td>
<td>Indicated for fluid retention</td>
</tr>
<tr>
<td>Nitrates</td>
<td>I</td>
<td>B</td>
<td>Contraindicated if SBP&lt; 100mmHg. Use with caution in valvular stenosis.</td>
</tr>
<tr>
<td>Morphine</td>
<td>IIb</td>
<td>B</td>
<td>Indicated in pts who are dyspneic and restless</td>
</tr>
<tr>
<td><strong>NOT RESPONSIVE TO INITIAL TREATMENT AND SBP&lt;100mmHg</strong></td>
<td></td>
<td></td>
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<tr>
<td>Diuretics</td>
<td>IIA</td>
<td>B</td>
<td>continuous infusion; combination with nitrates, dopamine, dobutamine or thiazide</td>
</tr>
<tr>
<td>Dopamine (&lt;2-3μg/kg/min)</td>
<td>IIA</td>
<td>B</td>
<td>To improve renal perfusion and promote diuresis</td>
</tr>
<tr>
<td>Dobutamine</td>
<td>IIA</td>
<td>B</td>
<td>Indicated for peripheral hypoperfusion +/- pulmonary congestion</td>
</tr>
<tr>
<td><strong>NOT RESPONSIVE TO INITIAL TREATMENT AND SBP&gt;100mmHg</strong></td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>Noradrenaline</td>
<td>IIA</td>
<td>B</td>
<td>Indicated to increase the BP</td>
</tr>
<tr>
<td>Dopamine (&gt;5μg/kg/min)</td>
<td>IIB</td>
<td>B</td>
<td>Indicated to increase the BP</td>
</tr>
<tr>
<td>IABP</td>
<td>I</td>
<td>B</td>
<td>Indicated as a bridge till myocardial recovery or heart transplant</td>
</tr>
<tr>
<td>Ventricular Assist Device (VAD)</td>
<td>IIA</td>
<td>B</td>
<td>Indicated as a bridge till myocardial recovery or heart transplant</td>
</tr>
</tbody>
</table>

### Table II: Grading of Recommendations in the Management of Chronic HF

<table>
<thead>
<tr>
<th>Intervention</th>
<th>Grades of Recommendation</th>
<th>Levels of Evidence</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>INDICATED FOR FLUID RETENTION IN NYHA II – IV</strong></td>
<td></td>
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<tr>
<td>Diuretics</td>
<td>I</td>
<td>B</td>
<td>Not shown to improve survival.</td>
</tr>
<tr>
<td><strong>INDICATED IN ALL PATIENTS</strong></td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>ACE-I</td>
<td>I</td>
<td>A</td>
<td>Improves survival and delays progression in all classes of HF</td>
</tr>
<tr>
<td>ARB</td>
<td>I</td>
<td>A</td>
<td>In ACE-I intolerant patients</td>
</tr>
<tr>
<td>β -blockers</td>
<td>I</td>
<td>A</td>
<td>Improves survival and delays progression in all classes of HF</td>
</tr>
<tr>
<td><strong>IN ADDITION TO THE ABOVE , THE FOLLOWING ARE INDICATED IN SELECTED PATIENTS</strong></td>
<td></td>
<td></td>
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<tr>
<td>ARB</td>
<td>I</td>
<td>B</td>
<td>In pts post MI and LVEF&lt;40%, Valsartan shown to be comparable to captopril</td>
</tr>
<tr>
<td>Mineralocorticoid receptor antagonists</td>
<td>I</td>
<td>B</td>
<td>Improves survival and reduces hospitalizations in moderate to severe HF and in post MI pts with mild HF</td>
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<tr>
<td>(Spironolactone, Eplerenone)</td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>Digoxin</td>
<td>I</td>
<td>B</td>
<td>In pts with HF and AF</td>
</tr>
<tr>
<td>Ivabradine</td>
<td>IIa</td>
<td>B</td>
<td>No effect on survival. Reduces hospitalizations when added to optimal medical therapy</td>
</tr>
<tr>
<td>ICD (implantable cardioverter defibrillator)</td>
<td>IIa</td>
<td>B</td>
<td>Reduces hospitalizations when added to optimal medical in sinus rhythm &gt; 70bpm</td>
</tr>
<tr>
<td>CRT (cardiac resynchronization therapy)</td>
<td>I</td>
<td>A</td>
<td>Improves survival in pts with resuscitated cardiac arrest, VF or sustained VT</td>
</tr>
<tr>
<td></td>
<td>I</td>
<td>A</td>
<td>Improves survival in pts &gt; 40 days post MI, LVEF &lt; 30%, on optimal medical treatment, and in NYHA II or III</td>
</tr>
<tr>
<td></td>
<td>IIa</td>
<td>B</td>
<td>Improves survival in pts (no prior MI), LVEF ≤ 35%, on optimal medical treatment, and in NYHA II or III</td>
</tr>
</tbody>
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