HeFSSA Practitioners Program 2019
“Challenges in Heart Failure Management”

- Dyspnoea and leg swelling, when is it heart failure?
- Management of acute decompensated heart failure
- Heart failure during pregnancy
- Refractory oedema in heart failure patient
CASE STUDY:

Dyspnoea and leg swelling, when is it heart failure?

Left heart failure: dyspnea and orthopnea; no elevation of venous pressure

Acute, severe p[ edema due to ventricular systolic or diastolic
What is dyspnoea?

Dyspnoea = Abnormally uncomfortable awareness of breathing

“smothering feeling/tightness/tiredness in the chest”

“cannot get enough air”

Baseline level of exercise/physical activity and patient perceptions must be taken into account when evaluating patients with the symptoms of “dyspnoea”

“air does not go all the way down”

“choking sensation”

“fatigue/tiredness during exercise”
Figure 1. Efferent and Afferent Signals That Contribute to the Sensation of Dyspnea.

The sense of respiratory effort is believed to arise from a signal transmitted from the motor cortex to the sensory cortex coincidently with the outgoing motor command to the ventilatory muscles. The arrow from the brain stem to the sensory cortex indicates that the motor output of the brain stem may also contribute to the sense of effort. The sense of air hunger is believed to arise, in part, from increased respiratory activity within the brain stem, and the sensation of chest tightness probably results from stimulation of vagal-irritant receptors. Although afferent information from airway, lung, and chest-wall receptors most likely passes through the brain stem before reaching the sensory cortex, the dashed lines indicate uncertainty about whether some afferents bypass the brain stem and project directly to the sensory cortex.
Differential diagnosis

1. Obstructive airways disease e.g. asthma/COPD
2. Diffuse parenchymal lung disease e.g. interstitial lung disease
3. Pulmonary vascular occlusive disease e.g. thromboembolism
4. Diseases of the chest wall/weakness of respiratory muscle e.g. Guillain-Barre
5. Heart Disease
6. Anemia
7. [Anxiety]
### NYHA Class

<table>
<thead>
<tr>
<th>NYHA Class</th>
<th>Symptoms</th>
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<tbody>
<tr>
<td>I</td>
<td>No limitation of physical activity. Ordinary physical activity does not cause undue fatigue, palpitation, dyspnea (shortness of breath).</td>
</tr>
<tr>
<td>II</td>
<td>Slight limitation of physical activity. Comfortable at rest. Ordinary physical activity results in fatigue, palpitation, dyspnea (shortness of breath).</td>
</tr>
<tr>
<td>III</td>
<td>Marked limitation of physical activity. Comfortable at rest. Less than ordinary activity causes fatigue, palpitation, or dyspnea.</td>
</tr>
<tr>
<td>IV</td>
<td>Unable to carry on any physical activity with discomfort. Symptoms of heart failure at rest. If any physical activity is undertaken, discomfort increases.</td>
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Patterns of dyspnoea may be helpful

- **Sudden unexpected episodes of dyspnoea**
  Consider: PE, spontaneous pneumothorax

- **Orthopnoea [dyspnoea in supine position]**
  Consider: CCF but may occur in asthma/COPD and bilateral diaphragmatic paralysis [rare]

- **Trepopnoea [dyspnoea in the lateral decubitus position]**
  Consider: CCF

- **Platypnoea: dyspnoea in the upright position**
  Consider: positional changes in V/Q relationships usually account for this

- **Parosymal nocturnal dyspnoea [sudden episodes of dyspnoea at usually at night]**
  Consider: CCF, asthma [due to circadian rhythm of airway muscle tone], chronic bronchitis [hypersecretion of mucus]
“Cardiac” Dyspnoea usually diagnosed when underlying heart disease is diagnosed based on history and clinical examination supplemented by non-invasive testing.
Clinical signs suggestive of cardiac etiology

- Peripheral Oedema
- Previous History of Heart Disease
- LV enlargement
- Presence of S3 or S4
- Elevated JVP

**Peripheral Oedema**

**Previous History of Heart Disease**

**LV enlargement**

**Presence of S3 or S4**

**Elevated JVP**

a – atrial contraction
x – atrial relaxation
c – bulging of tricuspid valve with ventricular contraction
x’ – downward movement of tricuspid valve with ventricular contraction
v – passive atrial filling
y – atrial emptying with opening of the tricuspid valve
How useful are clinical signs?

Sensitivity and specificity of clinical symptoms and signs in HF

<table>
<thead>
<tr>
<th>Clinical Features</th>
<th>Sensitivity (%)</th>
<th>Specificity (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Breathlessness</td>
<td>66</td>
<td>52</td>
</tr>
<tr>
<td>Orthopnoea</td>
<td>21</td>
<td>81</td>
</tr>
<tr>
<td>PND</td>
<td>33</td>
<td>76</td>
</tr>
<tr>
<td>History of oedema</td>
<td>23</td>
<td>80</td>
</tr>
<tr>
<td>Tachycardia</td>
<td>7</td>
<td>99</td>
</tr>
<tr>
<td>Pulmonary crackles</td>
<td>13</td>
<td>91</td>
</tr>
<tr>
<td>Oedema on examination</td>
<td>10</td>
<td>93</td>
</tr>
<tr>
<td>3rd heart sound</td>
<td>31</td>
<td>95</td>
</tr>
<tr>
<td>Raised JVP</td>
<td>10</td>
<td>97</td>
</tr>
</tbody>
</table>

Sosin M et al, Mansion Publishing 2006
ECG – Arrhythmias – AF/Atrial Flutter
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CHEST X-RAYS:
Interstitial oedema, pulmonary vascular redistribution, accumulation of fluid in septal planes, pleural effusion, cardiomegaly
NT-proBNP <125 pg/mL has a negative predictive value of 0.94 – 0.98 (i.e. if the NT-proBNP is this low >95% of the time the patient does NOT have heart failure)

This cut-off is also valid to exclude HFpEF.

It is therefore useful to exclude heart failure as a cause of dyspnea.
Triggers of elevated BNP

Cardiac disease
• Heart failure with reduced ejection fraction
• Heart failure with preserved ejection fraction
• RV failure
• Valvular heart disease
• Myocarditis
• LVH
• Coronary artery disease
• Myocardial trauma
• Arrhythmias
• Pericardial disease

Obese patients may have relatively lower BNP levels compared to non-obese individuals

Pulmonary disease
• Acute PE
• Pulmonary hypertension
• OSA
• Infection
• COAD

Neurologic disorders
• Stroke

Critical illness
• Sepsis
• Burns
• Transfusion associated circulatory overload

Toxins
• Chemotherapy
• Snake bites

Other
• Renal insufficiency
• Anaemia
• Cirrhosis
• Hypertension
• Hyperaldosteronism
Lung function Tests

A. Normal

B. Emphysema

C. Unilateral main-stem bronchial obstruction

D. Fixed UAO

E. Variable extrathoracic UAO

F. Variable intrathoracic UAO

G. Restrictive parenchymal lung disease

H. Neuromuscular weakness
Myocardial disease - DCMO

Pericardial disease - effusion

Valvular heart disease – aortic stenosis
Leg swelling
Oedema

Oedema=clinically apparent increase in interstitial fluid volume – usually manifests with swelling of the extremities

Anasarca: gross, generalised oedema

Ascites: accumulation of excess fluid in the peritoneal cavity

Localised

Usually due to lymphatic or venous obstruction: thrombophlebitis, lymphangitis, lymphnode resection, tumour, DVT

Generalised

Heart failure
Kidney disease
Nephrotic syndrome
Cirrhosis
Malnutrition
Hypothyroidism
Pregnancy
Drugs
Low output heart failure, Pericardial tamponade
Constructive pericarditis

↓Oncotic pressure
and/or
↑capillary permeability

↓Cardiac output
Effective arterial volume

Activation of ventricular and arterial receptors

SNS stimulation

Nonosmotic vasopressin stimulation

↑Systemic and renal arterial vascular resistance

Renal H₂O retention

Renal Na⁺ retention

Restoration of effective arterial volume

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<table>
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<tr>
<th>Organ System</th>
<th>History</th>
<th>Physical Exam</th>
<th>Lab Findings</th>
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<tbody>
<tr>
<td>Cardiac</td>
<td>Dyspnoea/ortho-pnoea/PND</td>
<td>Elevated JVP S3 Small pulse pressure</td>
<td>Increased Urea:Crea ratio Elevated uric acid Na often lower LFTs may be abnormal due to hepatic congestion</td>
</tr>
<tr>
<td>Hepatic</td>
<td>Dyspnoea infrequent Ethanol abuse</td>
<td>Often associated with ascites JVP normal BP usually lower Other signs of chronic liver disease (jaundice, palmar erythema, Dupuytren, spider angiomata, gynaecomastia)</td>
<td>Reductions in serum albumin and cholesterol LFTs increased Raised INR Hypokalemia Macrocytosis</td>
</tr>
<tr>
<td>Renal</td>
<td>Dyspnoea less prominent Uraemic symptoms (LOA, restless legs, reduced concentration, altered taste)</td>
<td>Usually hypertensive Retinopathy (diabetic/hypertensive) Periorbital oedema may be prominent Pericardial friction rub</td>
<td>Albuminuria Hypoalbuminaemia Raised creatinine Hyperkalemia Hyperphosphatemia and hypocalcemia Normocytic anaemia</td>
</tr>
</tbody>
</table>
Drugs associated with oedema

NSAIDS

Antihypertensive agents
  Vasodilators
  • Minoxidil
  • Clonidine
  • Hydralazine
  • Methyldopa
  • CCBs
  • Alpha-adrenergic blockers

Steroids
Cyclosporine
Growth hormone
Certain immunotherapies
What catches us out?

- Anaemia
- Pregnancy
- Pulmonary emboli
- Kidney disease
- Anxiety
- Drugs

Patient does actually not have dyspnoea, symptoms wrongly interpreted.

Co-existing pathology – e.g. COAD and severe aortic stenosis.
1. Confirm the symptom – does this patient have dyspnoea? If so, how bad is it (NYHA classification)
2. What is the cause – recall the differential diagnosis
3. Distinguishing a cardiac cause from noncardiac causes requires integration of:
   • History and clinical signs
   • ECG
   • CXR
   • NT-proBNP
   • +/-Echo and lung function tests
THANK YOU