ACUTE DECOMPENSATED HEART FAILURE (ADHF)

Sheba Medical Center, ICU Department

By Kai Chi Chan

Penultimate year Medical Student

St George's University of London at the University of Nicosia

Some definitions

- Heart Failure: A state in which the heart is unable to pump enough blood to adequately perfuse tissues and organs
- Decompensation: Where a condition has progressed to a stage at which it overcomes the body's compensatory mechanisms.
- ADHF: Sudden or gradual onset of signs and symptoms (i.e. dyspnoea, fatigue, and oedema)of Heart failure requiring medical attention.

Epidemiology of ADHF

- > 390,000 patients were diagnosed with heart failure between 2010 – 2011 in the UK
  - 1-2% of the adult population in developed world
  - 10% of those aged 70+
- 30,000 patients were admitted for acute decompensated heart failure in the same year
- It is the leading cause of hospitalisation of patients aged 65 years and older
- Equal male:female ratio
  - In younger populations = men more affected due to earlier onset of ischemic heart disease
- Accounts for up to 4% of all emergency admissions
- 83% will be re-admitted at least once during the following 12 months
- 1/3 of patients die during their first admission
- 40% mortality within the following year.
- Prevelance is increasing!
  - better survival rates after coronary events predisposes more people to the possibility of ADHF
  - More efficacious secondary prevention

Aetiology of ADHF

- Usually occurs (80%) as an exacerbation in patients with a previous history of heart failure / those with myocardial dysfunction (systolic, diastolic or both)
  - But can present as new onset (20%) in those with no previous history of heart disease

- Some precipitating factors include:
  - Acute coronary syndromes
  - Atrial fibrillation
  - Anaemia
  - Infection
  - Renal dysfunction
  - Hyperthyroidism
  - Fluid overload

Secondary Causes of Heart failure

- Coronary artery disease
  - i.e. MI
- Hypertension
  - Increased vascular resistance →LV hypertrophy
- Cardiomyopathies
- Drugs
  - Betablockers, Calcium antagonists, cocaine
- Endocrine
  - Hyper/hypothyroidism, diabetes, Cushings,
- Infiltrative
  - Sarcoidosis, haemochromatosis, amyloidosis

Classification

- Those who present with heart failure for the 1st time (De Novo)
- Those who have pre-existing heart failure that worsens or becomes exacerbated.
- Can be further "classified" according to symptoms (see later)

Pathophysiology: The neurohormonal hypothesis

- Over-activation of the neurohormonal system
- Eventually overcomes the counter-regulatory measures put in place by the body
- An event that causes decreased perfusion of the kidney's, for example:
  - Myocardial infarction
  - Vasoconstrictive changes in hypertension
- → Activation of RAAS + sympathetic nervous system

RAAS

- Renin-angiotensin-aldosterone system
- Promotion of Na^+ and water resorption by the kidney's + release of proinflammatory cytokines
- Peripheral vasoconstriction via Angiotensin II
- Angiotensin II + Aldosterone
  - directly increase myocardial hypertrophy and fibrosis.
  - Reduce Nitric Oxide (NO) release by endothelial cells
    - NO is a potent vasodilator
- Decrease in vascular compliance as well as the initiation / progression of LV dysfunction

Sympathetic adrenergic system

- Release of noradrenaline:
  - Vasoconstriction
  - Direct myocardial toxicity
  - Possibly arrhythmias

Counter-regulatory measures

- Natriuretic peptides
  - Atrial natriuretic peptide (Myocardium)
  - B-type natriuretic peptide (Ventricular Myocardium)
  - C-type peptide (Vascular endothelium)
- Promotion of Natriuresis and diuresis
- Inhibition of RAAS + sympathetic vascular tone

Pathophysiology cont.

- Change in CO or change in the body's requirements → neurohormonal imbalance
- Retention of fluid and vasoconstriction via RAAS and sympathetic nervous system
  - Increased preload and afterload
  - LV dysfunction
  - Starling's law of the heart
  - Therefore decreased CO
- Decreased CO = decreased renal perfusion
  - → increased activation of RAAS and SNS
- See the problem??

Signs and Symptoms

- Symptoms:
  - Breathlessness
  - Fatigue
  - Ankle swelling
- Signs:
  - Tachycardia
  - Tachypnoea
  - Raised JVP
  - Peripheral oedema
  - Pleural effusion

Diagnosis

- Clinical Evaluation
- History
- Vital Signs
- ECG
- Chest X-Ray
- Echocardiography
- Bloods:
  - FBC
  - U+E's
  - B-type Naturetic peptide
  - Pro-BNP

B-type Natriuetic peptide

- Released mainly from the ventricles
- Increased levels in heart failure
  - <100 pg/ml = high negative predictive value
    - Cut-off in AF = 200pg/ml
  - > 400pg/ml = high positive predictive value
  - Persistant elevation = possible chronic HF
- Should always be used in conjunction with other evidence!

Example of a "typical" patient

- 68 year old male, obese, diabetic, smoker
- Previous MI 2 years ago → Chronic HF
- Decided he didn't want to take his medications anymore and also randomly decided it would be a good idea to drink a bucket of sea water as he didn't care about his fluid/salt intake anymore.
- Already compromised heart from MI, that was previously compensating, will start to decompensate due to neurohormonal imbalance = progression to ADHF

Which ADHF patients should be admitted?

Presenting With ADHF	
Recommendation	**Clinical Circumstances**
(a) Hospitalization Recommended	Evidence of severe ADHF, including: Hypotension Worsening renal function Altered mentation Dyspnea at rest Typically reflected by resting tachypnea Less commonly reflected by oxygen saturation $<90\%$ Hemodynamically significant arrhythmia Including new onset of rapid atrial fibrillation

(b) Hospitalization Should Be Considered	Worsened congestion Even without dyspnea Signs and symptoms of pulmonary or systemic congestion Even in the absence of weight gain Major electrolyte disturbance Associated comorbid conditions Pneumonia Pulmonary embolus Diabetic ketoacidosis Symptoms suggestive of transient ischemic accident or stroke Repeated ICD firings Previously undiagnosed HF with signs and symptoms of systemic or pulmonary congestion

Treatment: Diuretics

- Patient with Fluid Overload:
  - Loop Diuretics (IV)
  - i.e. Furosemide 20mg IV → increasing dose to desired effect
  - Avoid rapid reduction of intravascular volume:
    - Hypotension
    - Gout
    - Exacerbate or induce renal dysfunction
    - Electrolyte imbalance → arrhythmias / muscle cramps
      - Monitor potassium levels and correct as needed

In the case of diuretic resistance:

- Consider the following options:
  - Fluid restriction (< 2 L/day)
    - Depends whether severe or moderate hyponatremia is present.
    - Severe (<125mEq/L) = more restriction
  - Na^+ restriction (2g/day)
  - Increase dosage of diuretics
  - Add a second loop diuretic (usually oral)
    - i.e. Metolazone / spironolactone
    - Or IV – chlorothiazide
  - Continuous infusion of a loop diuretic
  - Ultrafiltration

Treatment: Vasodilators

- Diuretics + Vasodilators can be used in conjunction to accelerate recovery from pulmonary congestion
- Should take regular BP as they may cause severe hypotension

IV Nitroglycerine

- Venous dilator → reduces filling pressure
  - Decreases preload
- Arterial dilator (in higher doses) → reduces afterload, increases stroke volume and cardiac output
- Advantage: treating concomitant MI
- Disadvantage: possible Tachyphylaxis
  - Sudden decrease In response to a drug after it has been administered

IV Nitroprusside

- Useful in severe hypertension / valvular dysfunction
- Venodilation > Aretiodilation
- Decreased pre-load, afterload and peripheral resistance
- MOA:
  - Breaks down in circulation → NO
  - NO activates Guanylate cyclase = increase cGMP
  - cGMP activates PKG → phosphatase activation
  - Phosphatases inactivate myosin light chains in smooth muscle surrounding blood vessel = vasodilation

IV Nesiritide

- Recombinant B-type Natruietic peptide (normally produced by ventricles in response to wall stress)
- Counter-regulates RAAS
- Activation of cGMP → Causes Vasodilation

Treatment: Inotropes

- To improve cardiac output in those with advanced Heart Failure:
  - Reduced LV ejection fraction
  - LV dilation
  - Hypotension
  - Low output syndrome:
    - Decreased peripheral perfusion
    - End organ dysfunction
- Short term use only: Long-term use is associated with increased mortality

Dobutamine

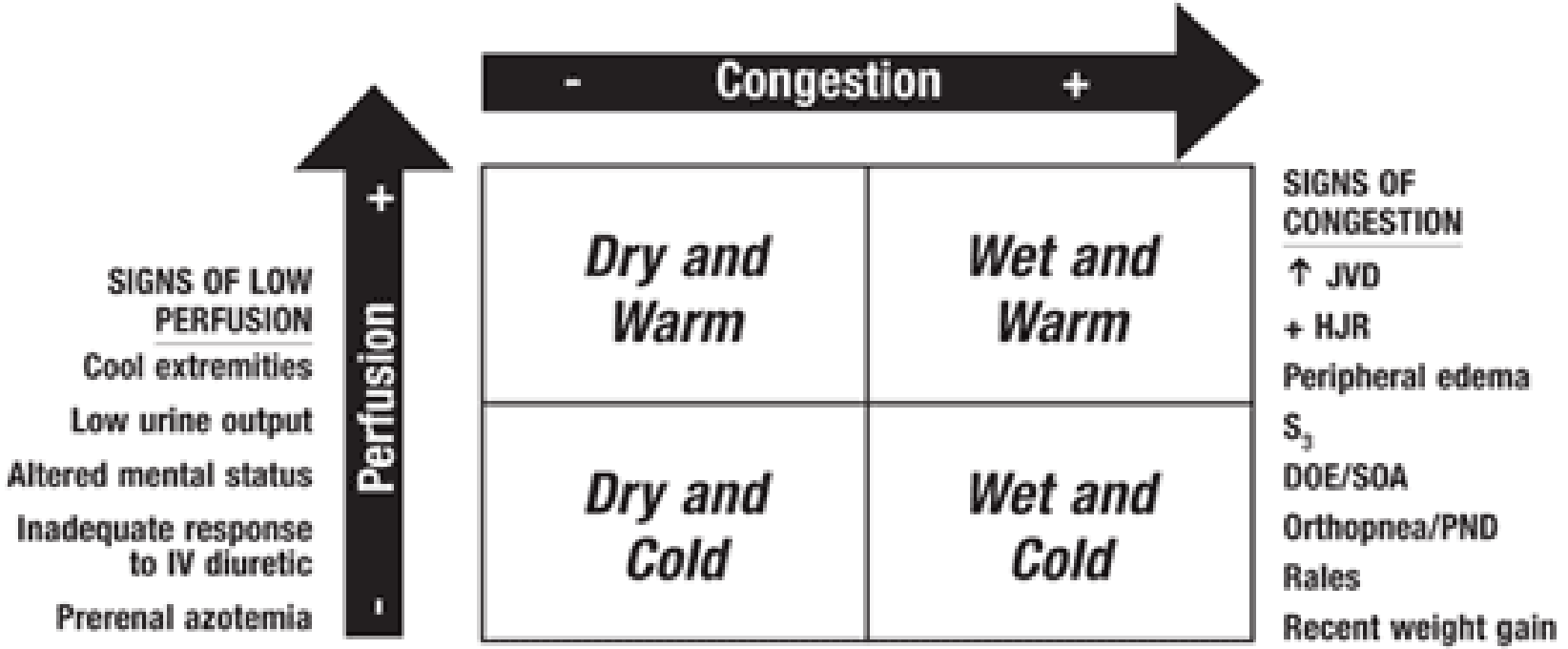
- Beta-1 Agonist
  - Increases adenylyl cyclase activity
  - Increase cAMP (degraded by Phosphodiseterases)
  - Increased PKA
  - PKA increases Ca^{2+} influx into cardiac myocytes
  - Result = Increased contractility
- Side effects:
  - Tachycardia, hypertension, arrhythmias

Milrinone

- Phosphodiesterase-3 inhibitor
  - Inhibits the breakdown of cAMP
  - Therefore:
    - increased PKA
    - Increased Ca^{2+} channel opening
    - Increased Ca^{2+} influx into cardiac myocytes
    - Increased contractility
  - Also affects K^{+} channels, allowing them to repolarise the cell more quickly
    - Therefore also has a positive chronotropic effect

Treatment choice depends on the clinical state of the patient

Figure 1. Hemodynamic/Clinical State in Acute Heart Failure



↑: increased; +: positive; -: negative; DOE: dyspnea on exertion; HJR: hepatojugular reflux; JVD: jugular venous distention; PND: paroxysmal nocturnal dyspnea; S_3: ventricular filling murmur; SOA: shortness of air.

Source: References 10, 11.

Treatment: Respiration

- Hypoxia
  - O_2 therapy is only indicated in the presence of hypoxia
- Severely dyspnoeic patients due to pulmonary oedema
  - Can consider non-invasive positive pressure ventilation

Venous thromboembolic prophylaxis

- Anticoagulation
  - Low dose unfractionated heparin
  - Low molecular weight heparin
  - For those who are not already anticoagulated and have no contraindications to anticoagulation
- Where anticoagulation is contraindicated:
  - Consider Intermittent pneumatic compression device
  - Or graded compression stockings.

Monitoring

- At least once per day:
  - Weight
  - Fluid input/output (Fluid balance chart)
    - Catheters are only recommended if there is suspicion of bladder outlet obstruction that is contributing to renal dysfunction
  - Reassessment for signs and symptoms of ADHF
  - Serum Electrolytes
  - Renal function
- More than once per day:
  - Vital signs

Discharge Criteria

- Return to near normal fluid volume
- No IV vasodilators or inotropes needed for the past 24 hours
- Continuation on only oral medication is well tolerated
- Patient and family have been educated with regards to medication, dietary and fluid concordance
- Resolution of exacerbating factors
- Referral for follow up has been arranged

References

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3. Lindenfeld J, et al. HFSA Guidelines. Journal of Cardiac Failure. 2010. 16(6): 475-539