5 Important Things to Know About Heart Failure

Kia Afshar, MD
Disclosures

• I have no conflicts of interest to disclose

• I will not be discussing any off label medications and/or devices
Objectives

• 1) Understand certain relationships between heart failure and renal dysfunction
• 2) To recognize the two broad categories of heart failure presentations
• 3) Realize that ejection fraction is not the end-all-be-all of heart failure management
• 4) Understand the importance of reaching goal doses of heart failure treatments
• 5) Recognize the signs that your patient needs an advanced heart failure specialist
What is Heart Failure?

• 1. A clinical “syndrome” characterized by...
• 2. Inability to provide sufficient cardiac output to meet tissue demands...
• 3. At normal ventricular filling pressures...

Packer M, Cohn JN, eds. Consensus recommendations for the management of heart failure. Am J Cardiol 1999;83(2A):1-38A)
Chronic Systolic Dysfunction

- Defined as ejection fraction (EF) < 50%

- Decreased stroke volume and effective arterial pressure results in compensatory neurohormonal activation

- Neurohormonal activation is initially important to maintain BP and CO but eventually results in deleterious myocardial changes and remodeling which increases morbidity and mortality.
How to think about Systolic HF:

• 1. Hemodynamic
• 2. Neurohumoral
• 3. Remodeling
Cardiomyopathies

Most common
Ischemic (most common in Western world)
idiopathic/nonischemic dilated cardiomyopathy (NIDCM)
hypertensive (long standing)
valvular (severe AR, severe AS, severe MR)

(relatively common)
Familial
hemochromatosis
amyloidosis
sarcoidosis
viral myocarditis
Giant cell myocarditis
drugs - chemotherapy, alcohol, cocaine
peripartum
anomalous coronary arteries
tachycardia induced cardiomyopathy (more common than you think)
stress cardiomyopathy (takotsubo's)
radiation treatment (causes coronary disease, pericardial, valvular, and muscular disease)
HIV cardiomyopathy
septic shock

• (relatively rare)
• high-output heart failure
• constriction
• restriction
• rheumatic myocarditis
• hypereosinophilic (rare)
• endocardial fibroelastosis (rare)
• pheochromocytoma (rare)
• PVC induced (rare)
• LBBB induced
• chronic RV pacing (rare)
• LV noncompaction (rare)
• glycogen storage disease (very rare)
• Fabry's (very rare)
• Friedreich's ataxia (very rare)
• duschenne (very rare)
• carcinoid (rare)
Heart Failure Paradigm (HFrEF vs HFpEF)
Heart Failure Paradigm (HFrEF vs HFpEF)

- Low EF
  - Ischemic
  - Non-ischemic

- Normal EF
Chronic Systolic Dysfunction

Goals

1) Identify underlying cause

2) Improved quality of life (e.g. improve symptoms, decrease hospitalizations, increase functional status)

3) Increase length of life (i.e. decrease mortality)
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2) Improved quality of life (e.g. improve symptoms, decrease hospitalizations, increase functional status)

3) Increase length of life (i.e. decrease mortality)
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idiopathic/viral
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Valvular
Familial
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Giant cell myocarditis
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Normal EF

high-output heart failure
constriction
restriction
rheumatic myocarditis
Hypereosinophilic
endocardial fibroelastosis
Pheochromocytoma
PVC induced
LBBB induced
chronic RV pacing
LV noncompaction
glycogen storage disease
Fabry's
Friedreich's ataxia
Duschenne
Carcinoid
Chaga's

Ischemic
Non-ischemic
Low EF

Normal EF

Ischemic

idiopathic/viral

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Valvular

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Non-ischemic

ischemic

Valvular

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glycogen storage disease

Fabry’s

Friedreich's ataxia

Duschenne

Carcinoid

Chaga’s
Important Thing # 1

• The best way to manage heart failure is to know the **TRUE** underlying cause. Treat every case as if you are Sherlock Holmes.

• Idiopathic and viral should be diagnoses of exclusion once you exhausted your brain’s ability to figure out a cause.
Chronic Systolic Dysfunction

Goals

1) Identify underlying cause

2) Improved quality of life (e.g. improve symptoms, decrease hospitalizations, increase functional status)

3) Increase length of life (i.e. decrease mortality)
1 Year Mortality (EF < 35%, NYHA III-IV)
Chronic Systolic Dysfunction

Management

1 Year Mortality (EF < 35%, NYHA III-IV)

- Diuretics, digoxin
- ACE inhibitors
- ACE + BB
- ACE + BB + spironolactone
- ACE + BB + spironolactone + ICD
- ACE + BB + spironolactone + ICD + CRT
Chronic Systolic Dysfunction

Management

1 Year Mortality (EF < 35%, NYHA III-IV)

- Diuretics, digoxin
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Year:
- 1988
- 1994
- 1999
- 2006
- 2008
- 2012
ACEI and Renal Function

Benefit Even If Creatinine Rises

(+) WRF/Enalapril

(−) WRF/Enalapril

(−) WRF/Placebo

(+) WRF/Placebo

Testani JM, et al. CircHF 2011
Chronic Systolic Dysfunction

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**Chronic Systolic Dysfunction**

**Management**
Chronic Systolic Dysfunction

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- ICD
- CRT
Chronic Systolic Dysfunction

Admissions/deaths in Canada in the months after the RALES trial was published.
Chronic Systolic Dysfunction

Management

1 Year Mortality (EF < 35%, NYHA III-IV)

- 1988: Diuretics, digoxin
- 1994: ACE inhibitors
- 1999: ACE + BB
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- 2008: ICD
- 2012: CRT
Chronic Systolic Dysfunction

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- 2012: ACE + BB + spironolactone + ICD + CRT
1 Year Mortality (EF < 35%, NYHA III-IV)

- Diuretics, digoxin (52%)
- ACE inhibitors
- ACE + BB
- ACE + BB + spironolactone
- ACE + BB + spironolactone + ICD
- ACE + BB + spironolactone + ICD + CRT (15%)

Chronic Systolic Dysfunction

Management
Important Thing # 2

• Heart Failure medications (at goal doses used in clinic trials) increase quality and quantity of life.

• Don’t be afraid of slight increase in creatinine when using ACE inhibitors, likely beneficial!

• Aldosterone antagonists need diligent K+ monitoring, if you cant, don’t use them.
HF consult for fear to diurese due to ARF
40 year old man with 2 months of DOE, abdominal distension, and weight gain. Came to the ED due to difficulty breathing.

Exam showed obese gentleman in NAD. JVD to angle of jaw sitting upright, 1+ bilateral edema to thighs. Warm extremities. Regular, no S3 or S4 appreciated.

Echocardiogram showed no significant valvular lesions, mild RV dysfunction, LVIDD of 6.8cm, LVEF of 25% with global hypokinesis.
HF consult for fear to diurese due to ARF

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No hemodynamics data available

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**24 HOUR TOTALS (06:01 - 06:00)**

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HF consult for fear to diurese due to ARF

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<th>Cl mmol/L</th>
<th>CO₂ mmol/L</th>
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<th>Glucose mg/dL</th>
<th>BUN mg/dL</th>
<th>Creatinine mg/dL</th>
<th>Ca mmol/L</th>
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HF consult on 1/20/15 for increasing Cr with diuresis

59 year old man with 2 months of DOE, abdominal distension, and 40-50 lb weight gain.

PHYSICAL EXAMINATION
General: Comfortable. Recumbant with CPAP on.
Vital signs: BP 117/55, HR 83, temp 35.5 c, sat 95% on RA
Lungs: CTA anteriorly.
Cardiovascular: Very distant heart tones. Regular. No murmur/S3.
Abdomen: Obese.
Extremities: Wrapped LE. Upper extremities with pulses palpable.
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### Basic Metabolic Panel

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<th>Test Status</th>
<th>Na</th>
<th>K</th>
<th>Cl</th>
<th>CO2</th>
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<th>Glucose</th>
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A rising BUN / Cr does not always mean that you need to slow down/stop your diuresis.

• If there is significant venous congestion, patients can get renal venous congestion which reduces their net renal perfusion pressure and raises the creatinine.
  — In these cases – with aggressive diuresis – the creatinine often will come down.

• **THE KEY: ACCURATE ASSESSMENT OF FLUID STATUS**

• If you can’t tell by the JVP and/or the patient is not responding predictably—then you may have to put in a Swan-Ganz catheter to assess hemodynamics.
Heart Failure and Worsening Renal Function
Heart Failure and Worsening Renal Function

NORMAL PATIENT:

Mean Arterial Pressure: 70mmHg
Heart Failure and Worsening Renal Function

NORMAL PATIENT:

Mean Arterial Pressure: 70mmHg
RAP/Renal Vein Pressure: 5mmHg
Heart Failure and Worsening Renal Function

NORMAL PATIENT:

Mean Arterial Pressure: 70mmHg
RAP/Renal Vein Pressure: 5mmHg
Renal Perfusion Pressure: 70 – 5 = 65mmHg
DECOMPENSATED HEART FAILURE PATIENT:

Mean Arterial Pressure: 65mmHg
HEART FAILURE AND WORSENING RENAL FUNCTION

DECOMPENSATED HEART FAILURE PATIENT:

Mean Arterial Pressure: 65 mmHg
RAP/Renal Vein Pressure: 20 mmHg
DECOMPENSATED HEART FAILURE PATIENT:

Mean Arterial Pressure: 65mmHg
RAP/Renal Vein Pressure: 20mmHg

Renal Perfusion Pressure: $65 - 20 = 45$mmHg
Heart Failure and Worsening Renal Function

DECOMPENSATED HEART FAILURE PATIENT:

Mean Arterial Pressure: 65mmHg
RAP/Renal Vein Pressure: 20mmHg
Renal Perfusion Pressure: 65–20 = 45mmHg

NORMAL PATIENT:

Mean Arterial Pressure: 70mmHg
RAP/Renal Vein Pressure: 5mmHg
Renal Perfusion Pressure: 70 – 5 = 65mmHg
Heart Failure and Worsening Renal Function

Figure 1. Prevalence of Worsening Renal Function During Hospitalization According to Categories of Admission CVP, CI, SBP, and PCWP

CI = cardiac index; Cr = serum creatinine; CVP = central venous pressure; PCWP = pulmonary capillary wedge pressure; SBP = systolic blood pressure.
Heart Failure and Worsening Renal Function

![Graph showing GFR (ml/min) for different CVP and CI conditions.]

**Figure 3**  Relative Contributions of CVP and CI to GFR at Time of PAC Removal

Error bars represent 95% confidence intervals. Cutoff values for CI = 2.4 l/min/m² and CVP = 8 mm Hg. GFR = glomerular filtration rate; PAC = pulmonary artery catheter; other abbreviations as in Figure 1.
Important Thing # 3

• A rising BUN/creatinine does not mean the patient is dry and/or that you cannot diurese a hypervolemic patient

• If there is significant venous congestion, patients can get renal venous congestion which reduces their net renal perfusion pressure and raises the creatinine.
  – In these cases – with aggressive diuresis – the creatinine often will come down.

• THE KEY: ACCURATE ASSESSMENT OF FLUID STATUS
Acute cardiogenic pulmonary edema (aka – “flash pulmonary edema”)
• **Causes:**
  
  – Acute hypertension
  – Acute mitral/aortic valve regurgitation
  – Acute myocardial ischemia
  – Acute left sided valvular obstruction (thrombosed prosthetic mitral valve, myxoma, HOCM physiology)
  – Acute and aggressive volume loading
  – Acute ventricular septal rupture
Acute cardiogenic pulmonary edema (aka – “flash pulmonary edema”)

• **Causes:**
  - **Acute** hypertension
  - **Acute** mitral/aortic valve regurgitation
  - **Acute** myocardial ischemia
  - **Acute** left sided valvular obstruction (thrombosed prosthetic mitral valve, myxoma, HOCM physiology)
  - **Acute** and aggressive volume loading
  - **Acute** ventricular septal rupture
Two Major Presentations of ADHF:
Two Major Presentations of ADHF:

1. “Vascular” Failure:
   Acute dyspnea, pulmonary edema, often due to acute hemodynamic ∆
Acute cardiogenic pulmonary edema (aka – “flash pulmonary edema”)
Two Major Presentations of ADHF:

2. "Cardiac" Failure:
   Gradual edematous decompensation
Chronic heart failure patients usually don’t have crackles.

- Most decompensated HF patients don’t have crackles
  - Crackles may be absent in up to 80% of patients.
    - These patients have increased lymphatic drainage and chronic perivascular compensation
  - They generally don’t have hypoxemia.
  - With diuresis – their shortness of breath and energy does however improve substantially.

- Up to 50% of patients may not have edema.

- GI symptoms: Abdominal discomfort, fullness or n/v
  - It is not uncommon that we will be consulted for a pre-op eval for gall bladder surgery and discover the real problem is liver congestion from decompensated HF
• Acute cardiogenic pulmonary edema is an ACUTE pressure overload problem as opposed to CHRONIC decompensated heart failure which is a volume issue
• If you see “flash” pulmonary edema, aim for BP control first rather than reaching for diuretics.
• If BP is normal, investigate for other causes of acute hemodynamic changes
The Course of Heart Failure
Kaplan-Meier Survival by Era
(Transplants: January 1982 - June 2010)

Survival (%)

0 10 20 30 40 50 60 70 80 90 100

0 1 2 3 4 5 6 7 8 9 10 11 12 13 14 15

1982-1992 vs. 1993-2002: p < 0.0001
1982-1992 vs. 2003-6/2010: p < 0.0001
1993-2002 vs. 2003-6/2010: p < 0.0001

Half-Life:
- 1982-1992: 8.5 years
- 1993-2002: 10.9 years

ISHLT 2012
Heart Transplants Reported per Year

NOTE: This figure includes only the heart transplants that are reported to the ISHLT Transplant Registry. As such, the presented data may not mirror the changes in the number of heart transplants performed worldwide.

ISHLT 2012
Improvements in Survival

- HEARTWARE
- TRANSPLANT
- HM II NATL
- HM II - UAHP
- POST-REMATCH
- LVAD REMATCH
- OMM REMATCH

Survival rates at 6, 12, 18, and 24 months post-implant for different categories.
Our Program’s Outcomes (DT)

![Graph showing program outcomes over time and across different studies.](image-url)
Consider an evaluation when **three** of the following indications are present:

- Class III – IV heart failure symptoms
- Inability to walk < 1 block without dyspnea
- Sodium < 136 mEq/L
- BUN > 40 mg or Cr > 1.8 mg/dL
- ACE/ ARB/ BB intolerance
- Diuretic dose > 1.5 mg/kg/d
- 1 HF admit in the past 6 months
- No clinical improvement with CRT
Importance of Timing – “Goldilocks Effect”

• Heart transplantation is the gold standard treatment for advanced heart failure in eligible candidates.

• LVADs are becoming necessary to keep patients in good enough health in order to get heart transplantation safely.

• LVADs can be used as “destination therapy” to improve quality/quantity of life.

• Early referral is safer than late referral.
Evaluation Criteria

Consider an evaluation when **three** of the following indications are present:

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- 1 HF admit in the past 6 months
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Heart Failure Team:

Outpatient Appointments: 801-507-4000
MD-to-MD Line: 801-507-HFMD (4363)