Does My Patient Have Transthyretin Amyloid Cardiomyopathy (ATTR-CM)? The Pharmacist Can Solve the Mystery

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Does My Patient Have Transthyretin Amyloid Cardiomyopathy (ATTR-CM)?
The Pharmacist Can Solve the Mystery!

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Learning Objectives

• Explain the pathophysiology and clinical presentation of patients with transthyretin amyloid cardiomyopathy (ATTR-CM)
• Develop a plan for the pharmacist to identify, diagnose, and treat patients who have ATTR-CM
• Interpret clinical trial data on available and emerging therapies for the treatment of ATTR-CM

“Unfolding” the myths that accompany the disease

Cardiac amyloidosis is an extremely rare disease
Diagnosis of amyloid cardiomyopathy is a death sentence for the patient
Limited treatment options are available for ATTR-CM

Diagnosing cardiac amyloidosis is complex, and the diagnostic algorithms are confusing
Pharmacists have limited roles in the diagnosis and treatment of this disease
A “rare” disease hidden in plain sight

<table>
<thead>
<tr>
<th>HFpEF</th>
<th>TAVR</th>
</tr>
</thead>
<tbody>
<tr>
<td>13% of patients with HFpEF</td>
<td>16% of patients undergoing TAVR for severe aortic stenosis</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>HCM</th>
<th>CTS</th>
</tr>
</thead>
<tbody>
<tr>
<td>5% of patients with presumptive hypertrophic cardiomyopathy</td>
<td>7% to 8% of patients with carpal tunnel syndrome</td>
</tr>
</tbody>
</table>

CTS = carpal tunnel syndrome; HCM = hypertrophic cardiomyopathy; HFpEF = heart failure with preserved ejection fraction; TAVR = transcatheter aortic valve replacement

Maurer MS et al. Circ Heart Fail. 2019 Sep;12(9):e006075

Background

Amyloidosis is a broad term for a condition characterized by protein misfolding

Multiple organ systems can be affected

More than 30 different precursor proteins can cause amyloidosis

Cardiac amyloidosis (CA) is predominantly caused by deposition of transthyretin (TTR) or immunoglobulin light chains (AL), accounting for >95% of cases

Does My Patient Have Transthyretin Amyloid Cardiomyopathy (ATTR-CM)?

The Pharmacists Can Solve the Mystery

**ATTR Amyloidogenesis**

- Tetrameric transthyretin
- Dissociation
- Monomers
- Rate-limiting step
- Misfolding
- Oligomers
- Amyloid fibrils

**AL vs. ATTR**

<table>
<thead>
<tr>
<th>AL Amyloidosis</th>
<th>ATTR Amyloidosis</th>
</tr>
</thead>
<tbody>
<tr>
<td>- Bone marrow “problem”</td>
<td></td>
</tr>
<tr>
<td>- Worst overall prognosis</td>
<td></td>
</tr>
<tr>
<td>- Treated with chemotherapy ± autologous stem cell transplant in select patients</td>
<td></td>
</tr>
<tr>
<td>- Liver “problem”</td>
<td></td>
</tr>
<tr>
<td>- More favorable prognosis</td>
<td></td>
</tr>
<tr>
<td>- Treatment aims to prevent TTR production, stabilize TTR protein, or enhance degradation and elimination of existing amyloid fibrils</td>
<td></td>
</tr>
</tbody>
</table>


Subtype Classification

**Nomenclature**

- **ATTR-CM**
  - A = Amyloidosis
  - TTR = Transthyretin
  - CM = Cardiomyopathy

Other terminology may be used:
- ATTRwt may also be referred to as acquired ATTR; previously known as senile cardiac amyloidosis
- ATTRv may also go by mutant ATTR (ATTRm), hereditary ATTR, or familial ATTR

Maurer MS et al. Circ Heart Fail. 2019 Sep;12(9):e006075

**ATTR - Pathophysiology**

- Transthyretin predominantly produced in liver
  - Responsible for transporting thyroxine and retinol binding protein

- Transthyretin amyloid cardiomyopathy occurs when normally **soluble** transthyretin dissociates, misfolds, and deposits as **insoluble** amyloid fibrils in the myocardium

- Etiology of amyloidogenesis in ATTRwt-CM is unclear

Maurer MS et al. Circ Heart Fail. 2019 Sep;12(9):e006075
ATTR - Pathophysiology

- In ATTRv, genetic mutations lead to a dysfunctional TTR protein
  - Autosomal dominant inheritance with variable penetrance
  - More than 120 mutations identified
    - V30M
      - Most common worldwide
      - Early onset with predominant polyneuropathy phenotype
      - Late onset with predominant cardiomyopathy phenotype
    - V122I
      - Identified in 3-4% of patients of African descent
      - Almost exclusively presents as cardiomyopathy
  - Phenotype varies depending on specific mutation involved

Patient Case

<table>
<thead>
<tr>
<th>History of Present Illness</th>
<th>Vital signs and Anthropometrics</th>
</tr>
</thead>
<tbody>
<tr>
<td>80-year-old African-American female with heart failure presents to clinic for routine follow up. Complains of dizziness after taking morning heart failure medications.</td>
<td>BP 100/60 mm Hg, HR 74 bpm, Wt: 85 kg; Ht: 65”</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Past Medical History</th>
<th>Cardiodynamics</th>
</tr>
</thead>
<tbody>
<tr>
<td>HFREF, HTN, DM, dyslipidemia, OA, history of CTS</td>
<td>TTE – EF 30-35%; Grade 2 diastolic dysfunction, GLS -8.1% with apical sparring of longitudinal strain</td>
</tr>
<tr>
<td></td>
<td>ECG – normal sinus rhythm with HR 72 bpm, age indeterminant inferior infarct</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Pertinent labs</th>
<th>Medications (all taken orally)</th>
</tr>
</thead>
<tbody>
<tr>
<td>NT-proBNP 5700 pg/mL</td>
<td>1. Carvedilol 25 mg twice daily</td>
</tr>
<tr>
<td>SCR 1.3 mg/dL</td>
<td>2. Sacubitril-valsartan 97 mg/103 mg twice daily</td>
</tr>
<tr>
<td>A1C 6.7%</td>
<td>3. Spironolactone 25 mg daily</td>
</tr>
<tr>
<td></td>
<td>4. Furosemide 20 mg daily</td>
</tr>
<tr>
<td></td>
<td>5. Hydralazine-isosorbide dinitrate 20 mg/37.5 mg twice daily</td>
</tr>
</tbody>
</table>

bpm = beats per minute; DM = diabetes mellitus; HTN = hypertension; HFREF = heart failure with reduced ejection fraction, EF = ejection fraction; GLS = global longitudinal strain; OA = osteoarthritis; SCR = serum creatinine; NT-proBNP = N-terminal-pro B-type natriuretic peptide
Patient Case

What clue in the patient’s history and presentation is suggestive of amyloidosis?

a) History of hypertension and diabetes
b) Apical sparing of longitudinal strain on echocardiogram
c) Elevation in SCr
d) Reduction in EF on echocardiogram

Signs and Symptoms

Cardiovascular
- Dyspnea
- Chest pain
- Fatigue
- Edema
- HF symptoms + conduction abnormalities

Extra-cardiac
- Sensory and autonomic neuropathy
- Gastrointestinal complaints
- Sexual dysfunction
- Orthostatic hypotension
- Proteinuria/nephrotic syndrome

Maurer MS et al. Circ Heart Fail. 2019 Sep;12(9):e006075
Clinical Clues and Red Flags

- H/o carpal tunnel syndrome
  - Often years prior to diagnosis
- H/o lumbar spinal stenosis
- H/o biceps tendon rupture
- H/o TKA/THA

- Autonomic and peripheral neuropathy
- Orthostatic hypotension
- Unexplained weight loss

- Heart failure
- Inability to tolerate HF therapies
- H/o HTN with BP that is now normalized
- Mild chronic elevation in troponin
- Low-flow, low-gradient AS

- Unexplained ventricular wall thickness and apical sparing of longitudinal strain on echocardiogram
- Discordance between left ventricular thickness and voltage on ECG
- CMR characteristics suggestive of amyloid cardiomyopathy

- Family history of the disorder (ATTRv)

TKA = total knee arthroplasty
THA = total hip arthroplasty
AS = aortic stenosis

Common with ATTRv

Common with ATTRwt

Maurer MS et al. Circ Heart Fail. 2019 Sep;12(9):e006075
Oerlemans MiFi et al. Neth Heart J. 2019; 27:525-36.

Patient Case

The cardiologist suspects cardiac amyloidosis and consults with the pharmacist to initiate the diagnostic workup. Which of the following tests do you recommend performing at this time?

a) Serum/urine electrophoresis
b) Serum free light chains and troponin T
c) Nuclear scintigraphy (technetium pyrophosphate [99mTc-PYP] scan)
d) Serum/urine immunofixation with serum free light chains
Diagnosis

- Appropriate **AND** early diagnosis is critical
  - Prognostic and therapeutic implications
- Abundant diagnostic options

Improper diagnosis and variable clinical presentation contribute to the perceived rarity of the disease with direct influence on prognosis and clinical outcome

Patient Case

The patient’s initial workup is negative for AL amyloidosis. Which of the following tests do you recommend to proceed with the diagnostic workup for cardiac amyloidosis?

a) Cardiac magnetic resonance (CMR) imaging  
b) Transesophageal echocardiogram (TEE)  
c) Nuclear scintigraphy (technetium pyrophosphate [99mTc-PYP] scan)  
d) Cardiac computed tomography (CT) scan
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### Diagnostic Arsenal

**Biomarkers:** Troponin T, BNP/NT-proBNP (Disease severity/prognostication)

**Electrocardiogram (ECG)**
- **Limited sensitivity**
  - *Features of CA:* low voltage or pseudo-infarct pattern; discordance between QRS voltage and LV wall thickness

**Transthoracic echocardiogram (TTE)**
- **Limited sensitivity**
  - *Features of CA:* wall thickness/mass; bi-atrial enlargement; apical sparing of longitudinal strain

**Nuclear Scintigraphy (99mTc-PYP scan)**
- Sensitive and specific for ATTR in absence of monoclonal antibodies
  - (Able to diagnose ATTR-CM without biopsy)
  - Needs to be combined with SPECT imaging
  - *Features of CA:* Grade 2 or 3 uptake diagnostic for ATTR-CM

**Cardiac magnetic resonance (CMR)**
- Useful for tissue characterization
  - Morphologic and functional assessment
  - Can differentiate between CA and other HCM
  - *Features of CA:* LV wall thickness/mass; bi-atrial enlargement; diffuse LGE with global ECV > 0.4; abnormal myocardial nulling kinetics

**Tissue Biopsy**
- Fat aspirate or clinically affected organ
- Always required for AL diagnosis
- Should be considered if clinical suspicion remains high or if above tests are indeterminate

### Amyloid Cardiomyopathy?

- **BNP** = B-type natriuretic peptide; **LGE** = late gadolinium enhancement; **ECV** = extracellular volume; **SPECT** = Single photon emission computed tomography; **99mTc-PYP** = 99m technetium pyrophosphate


### Diagnostic Algorithm

**Clinical suspicion**

**Screen for monoclonal proteins**
- SPIE/UPIE (electrophoresis + immunofixation)
- Serum free light chains

**Hematology referral**
- Present
- Absent

**Tissue or fat pad biopsy**
- Congo red and tissue typing

**Amyloidosis unlikely**
- Negative
- Positive

**AL, ATTR, or alternative diagnosis**
- Negative
- Positive

**Cardiac Scintigraphy (99mTc-PYP)**
- Positive
- Negative or indeterminate

**GENETIC TESTING**
- ATTRwt
- ATTRv

**ATTR-CM Unlikely**

- Consider endomyocardial biopsy if clinical suspicion remains high

*Signs/symptoms, red flags, ECG, TTE, CMR suggestive of amyloid cardiomyopathy

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Staging and Prognosis

- Different staging systems available using cardiovascular and/or renal parameters to assess prognosis in ATTR-CM

### Mayo Clinic Model for ATTRwt (NT-proBNP + Troponin T)

<table>
<thead>
<tr>
<th>Condition</th>
<th>Stage</th>
</tr>
</thead>
<tbody>
<tr>
<td>NT-proBNP ≤ 3000 pg/mL AND Troponin T ≤ 0.05 ng/mL</td>
<td>Stage I</td>
</tr>
<tr>
<td>NT-proBNP &gt; 3000 pg/mL OR Troponin T &gt; 0.05 ng/mL</td>
<td>Stage II</td>
</tr>
<tr>
<td>NT-proBNP &gt; 3000 pg/mL AND Troponin T &gt; 0.05 ng/mL</td>
<td>Stage III</td>
</tr>
</tbody>
</table>

### UK Model for ATTRwt and ATTRv (NT-proBNP + eGFR)

<table>
<thead>
<tr>
<th>Condition</th>
<th>Stage</th>
</tr>
</thead>
<tbody>
<tr>
<td>NT-proBNP ≤ 3000 pg/mL AND eGFR &gt; 45 mL/min/1.73 m²</td>
<td>Stage I</td>
</tr>
<tr>
<td>NT-proBNP &gt; 3000 pg/mL OR eGFR &lt; 45 mL/min/1.73 m²</td>
<td>Stage II</td>
</tr>
<tr>
<td>NT-proBNP &gt; 3000 pg/mL AND eGFR &lt; 45 mL/min/1.73 m²</td>
<td>Stage III</td>
</tr>
</tbody>
</table>

NYHA = New York Heart Association; eGFR = estimated glomerular filtration rate

Patient Case

The patient is diagnosed with ATTRv-CM (V122I mutation) and is referred to you for treatment. Which of the following is the most appropriate treatment for this patient’s ATTRv-CM?

a) Diflunisal
b) Tafamidis
c) Patisiran
d) Doxycycline with tauroursodeoxycholic acid (TUDCA)
Treatment – General

Avoid
- Digoxin
- Non-DHP CCB
- ACEI/ARB
- BB

Use
- Loop diuretic
- MRA
- Anticoagulants
- Amiodarone

- Supportive treatment limited to management of HF symptoms and arrhythmias.
- Goal is to maintain euvolemia and reduce ventricular filling pressures without causing hypotension
- Most HF therapies are generally not well tolerated, especially in advanced disease
  - Concomitant autonomic dysfunction
  - Rate dependent CO due to fixed SV

*Can be used in select cases with close monitoring targeting lower trough levels
*Consider in all patients with AF regardless of CHA2DS2-Vasc score

Non-DHP CCB = non-dihydropyridine calcium channel blocker; ACEI = angiotensin converting-enzyme inhibitor; ARB = angiotensin receptor blocker; BB = beta blocker; MRA = mineralocorticoid receptor antagonist; CO = cardiac output; SV = stroke volume; AF = atrial fibrillation


Treatment – TTR Amyloidosis

Prevent formation of TTR
- Small interfering RNA (siRNA)
- Antisense oligonucleotides (ASOs)

Stabilize TTR protein
- Tafamidis
- Diflunisal
- Epigallocatechin-3-gallate (EGCG)

Disrupt deposited amyloid fibrils
- Doxycycline + TUDCA/UDCA
- Monoclonal antibodies

Inhibit oligomer aggregation
- EGCG

Prevent
Stabilize
Disrupt

TUDCA = tauroursodeoxycholic acid; UDCA = ursodeoxycholic acid
**Gene Silencing Therapeutics**

**Goal:** Prevent translation of TTR mRNA to functional TTR protein

<table>
<thead>
<tr>
<th>Small Interfering RNA (siRNA)</th>
<th>Antisense Oligonucleotides (OSA)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Patisiran (OnPattro®)</strong></td>
<td><strong>Inotersen (Tegsedi®)</strong></td>
</tr>
<tr>
<td>• APOLLO trial</td>
<td>• NEURO-TTR trial</td>
</tr>
<tr>
<td>• Patisiran 0.3 mg/kg IV every 3 weeks vs. placebo in hereditary TTR amyloidosis with polyneuropathy</td>
<td>• Inotersen 300 mg subcutaneously once weekly vs. placebo in hereditary TTR amyloidosis with polyneuropathy</td>
</tr>
<tr>
<td>• In cardiac subpopulation (N = 126) patisiran decreased LV wall thickness, GLS, and NT-proBNP</td>
<td>• Evaluation of cardiac parameters showed no difference in LV wall thickness, GLS, or EF</td>
</tr>
<tr>
<td>• APOLLO-B trial evaluating patisiran in patients with ATTR-CM is ongoing</td>
<td>• Open-label extension trial of NEURO-TTR is ongoing</td>
</tr>
</tbody>
</table>

*Not currently FDA-approved for ATTR-CM*

**TTR Stabilizers**

**Goal:** Stabilize TTR tetramer to prevent dissociation into monomers

<table>
<thead>
<tr>
<th>Tafamidis meglumine (Vyndaqel®)</th>
<th>Tafamidis (Vyndamax®)*</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Selective small-molecule TTR stabilizer</td>
<td></td>
</tr>
<tr>
<td>• Binds to thyroxine-binding site on TTR, stabilizing the tetrameric structure and preventing dissociation into monomers</td>
<td></td>
</tr>
<tr>
<td>• Rate-limiting step in amyloidogenic process</td>
<td></td>
</tr>
<tr>
<td>• Extensively studied in both TTR polyneuropathy and cardiomyopathy</td>
<td></td>
</tr>
<tr>
<td>• Dosage for ATTR-CM is 80 mg orally daily (tafamidis meglumine) or 61 mg orally daily (tafamidis)</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Diflunisal (Off-label use only)</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Nonsteroidal anti-inflammatory drug (NSAID) with TTR stabilization properties</td>
</tr>
<tr>
<td>• Studied predominantly in patients with polyneuropathy</td>
</tr>
<tr>
<td>• Small non-randomized trials in patients with ATTR-CM using biomarkers and echocardiogram parameters as the principle endpoints</td>
</tr>
<tr>
<td>• Limited evidence shows diflunisal may prevent disease progression and improve outcomes</td>
</tr>
<tr>
<td>• Long-term risks of CV, GI, and renal toxicities may preclude use in many patients</td>
</tr>
<tr>
<td>• Dosage is 250 mg orally twice daily</td>
</tr>
</tbody>
</table>

*FDA-approved for both ATTRwt-CM and ATTRv-CM

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Does My Patient Have Transthyretin Amyloid Cardiomyopathy (ATTR-CM)?

The Pharmacists Can Solve the Mystery

**ATTR – ACT**

**Objective:** Evaluate the efficacy and safety of tafamidis in patients with transthyretin amyloid cardiomyopathy

**Multicenter, randomized, double-blind, parallel-design, placebo-controlled**

**Pertinent inclusion criteria:**
1. NYHA IV heart failure
2. Presence of AL amyloidosis
3. Implanted LVAD
4. eGFR < 25 mL/min/1.73 m²
5. Liver transaminase levels > 2 x ULN
6. Concurrent use of NSAIDs, tauroursodeoxycholate, doxycycline, CCBs, or digitalis

**Pertinent exclusion criteria:**
1. End-diastolic interventricular septal wall thickness > 12 mm
2. H/o heart failure with at least one HFH or clinical evidence of HF without hospitalization
3. NT-proBNP > 600 pg/mL
4. 6MWT distance > 100 m (328 ft)

**Key secondary outcomes: Change in 6MWT and change in KCCQ – OS score**

ATTR – ACT = The Transthyretin Amyloidosis Cardiomyopathy Clinical Trial; CM = cardiomyopathy; KCCQ – OS = Kansas City Cardiomyopathy Questionnaire – Overall Summary

HFH = hospitalization for heart failure; 6MWT = six minute walk test; LVAD = left-ventricular assist device


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**ATTR – ACT**

**Average patient was a 75-year-old White male with NYHA FC II HF. 76% had ATTRwt and 24% had ATTRv**

- **Tafamidis superior to placebo in primary efficacy analysis** (*P* < 0.001) with win ratio of 1.695
- **CV hospitalizations also lower in tafamidis group** (0.48 vs. 0.7 per year); RRR 0.68; (95% CI, 0.56-0.81); NNT = 4
- **Decreased rate of decline in 6MWT and KCCQ-OS with tafamidis compared with placebo with benefit observed at 6 months**
- **Safety profiles similar with tafamidis and placebo**

*Subgroup analyses favoring tafamidis except for patients with NYHA FC III HF at baseline


**All-cause mortality**

*Using the Finkelstein-Schoenfeld method*

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**Fibril Disruption**

**Goal:** Degrade and eliminate existing deposited amyloid fibrils

- **Doxycycline + TUDCA or UDCA**
  - Limited evidence with unclear benefit
  - Higher rate of ADRs (specifically with doxycycline) leading to treatment discontinuation

- **Monoclonal antibodies**
  - Targeted binding of amyloid deposits and remove amyloid fibrils from tissue
    - Anti-TTR antibodies
      - NI006 and PRX004
    - Anti-SAP antibodies
      - Dezamizumab
  - Early phase trials are ongoing

SAP = serum amyloid P component

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### Ongoing trials

- **ATTRibute-CM**
  - Acoramidis (AG-10)
  - Selective TTR stabilizer
- **APOLLO-B**
  - Patisiran
- **HELIOS-B**
  - Vutrisiran
  - Subcutaneous siRNA
- **CARDIO-TTRansform**
  - Eplontersen
  - Subcutaneous ASO

### Future directions

**CRISPR**

- **NTLA-2001**
- CRISPR/Cas9-based therapy
  - In vivo gene editing
  - Molecular “scissor” that slices DNA
  - Targeted knockout of TTR gene
  - One dose and done?
- Phase 1 clinical trials are ongoing

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### ATTR-CM Recommended Treatment

<table>
<thead>
<tr>
<th>ATTRwt-CM</th>
<th>ATTRv-CM</th>
<th>ATTRv-CM + Neuropathy</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tafamidis</td>
<td>Tafamidis</td>
<td>Tafamidis</td>
</tr>
<tr>
<td>Diflunisal+</td>
<td>Diflunisal+</td>
<td>Patisiran</td>
</tr>
</tbody>
</table>

*Off-label use only

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### Unanswered Questions

What is the “best” treatment (no head-to-head comparison, not a “**one size fits all**” approach)

Single vs. combination therapy?*

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*Tafamidis is the only FDA-approved treatment for ATTRwt-CM and ATTRv-CM

No data currently available supporting the use of combination therapy

Take Home Points

Cardiac amyloidosis is rare, although significantly underdiagnosed

Diagnosis of amyloid cardiomyopathy is NOT a death sentence for the patient

There ARE various treatment options available for ATTR-CM, and treatments are rapidly expanding

"Unfolding" the myths that accompany the disease

Diagnosing cardiac amyloidosis is straightforward, although one must "seek out" the disease

Pharmacists have EXTENSIVE limited roles in the diagnosis and treatment of this disease

How will you change your practice?
1. Screen patients for signs and symptoms of ATTR-CM
2. Collaborate with interprofessional teams to identify the potential for ATTR-CM in patients
3. Advocate for early, appropriate ATTR-CM diagnosis and treatment
4. Collaborate with the health care team to diagnose and treat patients who have ATTR-CM
5. Be aware of the pathophysiology and clinical presentation of patients with ATTR-CM

Take a moment to reflect on the changes you would make based on what you learned today.
Key Takeaways

• The workup for a patient with ATTR-CM almost always starts with a clinical suspicion
• Treatment options for ATTR-CM are rapidly expanding
• Time is KEY!
  – Time to suspicion, time to diagnosis, time to treatment