Telltale Signs of Sarcoidosis:
Evaluation and Management of the Great Imitator

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Disclosures

- No relevant financial relationships with a commercial interest

- As with virtually any sarcoidosis talk, I will most definitely be talking about off-label medications
Take home points

- Assessment of extrapulmonary organ involvement should be performed in all patients
  - Especially cardiac, neuro, hypercalcemia
- Biomarkers can be helpful in assessing disease activity assessment, but should never be used as a primary treatment endpoint
- Adhere to Well’s Law!
- Second line therapy = steroid sparing therapy
- Vitamin D supplementation is safe, as long as sarc-associated vitamin D dysregulation is excluded
Epidemiology

- All races, ethnicities
- F>M
- Prevalence: not sure, but
  - 185,000 patients with sarcoidosis seek medical care annually (Baughman 2016)
- Race and Age

<table>
<thead>
<tr>
<th>Study</th>
<th>Race (%)</th>
<th>Age of onset</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Black</td>
<td>White</td>
</tr>
<tr>
<td>ACCESS</td>
<td>44</td>
<td>53</td>
</tr>
<tr>
<td>MUSC</td>
<td>65</td>
<td>35</td>
</tr>
<tr>
<td>Ohio St</td>
<td>40</td>
<td>57</td>
</tr>
</tbody>
</table>
Etiology

- Lock and Key

- Candidate antigens
  - Atypical Mycobacteria
  - Propionobacterium
  - Mold/organ dust

Genetic predisposition

Antigen

Not me
Pathophysiology

Unknown Antigen

Th1 > Th2
Inflammatory Response

GRANULOMA

Iannuzzi, 2007
Dropping the Ball

- Up to 60% of patients will have spontaneous resolution
- BUT 1/3 will have chronic, fibrotic disease
- Why do some patients progress while others spontaneously resolve?
- Can we predict which direction patients will go?

Iannuzzi, 2007
Sarcoidosis Autoantigen Leading to Fibrosis?

- **Serum Amyloid A**
  - Results in increased intensity of Th1 response to antigens (Chen 2010) and increased IFN$\gamma$ and TNF$\alpha$ expression
  - Initial SAA levels higher in patients who required prolonged treatment

- **Is chronic, fibrotic sarcoidosis a result of “autoimmunity”?**

<table>
<thead>
<tr>
<th>Antibody</th>
<th>n</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>ANA</td>
<td>12 (10-1:100, 2-1:320)</td>
<td>28.5</td>
</tr>
<tr>
<td>RF</td>
<td>7</td>
<td>16.6</td>
</tr>
</tbody>
</table>
What about genetics?

- 2.5-fold increase in risk for siblings and parents of sarcoidosis patients
- Certain HLA-DRB1 alleles associated with risk
- ANXA11 and BTNL2 associated with granuloma formation
- Others…

Diagnosis and Evaluation

- Review of the “Rules”
  - Do all patients need a biopsy?
- Disease Assessment
  - Biomarkers
  - Phenotyping
Making the diagnosis…

- ATS statement (1999): The diagnosis of sarcoidosis needs…
  1. a compatible clinical picture
  2. histologic demonstration of noncaseating granulomas (most of the time)
  3. exclusion of other diseases capable of producing a similar histologic or clinical picture
Building a compatible clinical picture
(Assessing organ involvement and excluding alternative diagnoses)

- **History and Physical exam**
  - occupational/environmental exposures, symptoms
  - Chest x-ray/CT chest
  - Pulmonary function tests
  - Labs: CBC, CMP (serum calcium, BUN/creatinine, liver enzymes)
  - ECG
  - Routine ophthalmologic exam (even in the absence of eye complaints)
  - TB skin test/Quantiferon
  - Urinalysis?
A Word about Scadding Stages

- Provides generally good prognostication in regards to what will happen to chest radiograph

- In population studies correlates with FVC, weak correlation to level of dyspnea

- Not predictive of need for treatment at follow-up (Baughman 2005)

- Fair inter-observer agreement (kappa = 0.43) (Baughman 2009)
Biopsy

- **Lofgren’s Syndrome**
  - Hilar adenopathy, symmetric polyarthritis, erythema nodosum
- **Asymptomatic bilateral hilar adenopathy**
  - 99.95% of patients in the US will have stage I sarcoidosis (versus TB or lymphoma) (Reich 1998)
Disease Assessment/Evaluation

- Includes phenotyping and measures of disease activity

- Goal is to improve our ability to
  - Prognosticate
    - How likely is this patient to experience morbidity/mortality?
  - Determine potential benefit of treatment
    - Is the patient symptomatic because there is active disease?
Phenotyping Theory in Sarcoidosis

<table>
<thead>
<tr>
<th>Individual</th>
<th>Disease</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td># organs involved</td>
</tr>
<tr>
<td>Gender</td>
<td>“critical” organ involvement</td>
</tr>
<tr>
<td>Race</td>
<td>Acuity of onset</td>
</tr>
</tbody>
</table>

Phenotypic Traits

1. Symptoms
   - Symptomatic
   - Asymptomatic
2. Disease Status
   - Morbidity
   - Involved
   - Resolved

Patient 1
- Lung
- Eye
- Skin

Patient 2
- Lung
- Eye
- Skin

Patient 3
- Lung
- Eye
- Skin
Phenotyping Panacaea

1. Symptoms
   - Symptomatic
   - Asymptomatic

2. Disease Status
   - Morbidity
   - Involved
   - Resolved

Patient X

Presentation
- Follow-up

Treat

Don’t Treat
Phenotyping organ involvement

- Characteristic of disease
  - Which organs involved
  - Number of organs involved

- Good association with prognosis/response to treatment
Expand your knowledge of cereal grains
...and organ assessment

**WASOG OAT (2014)**

<table>
<thead>
<tr>
<th>Highly Probable</th>
<th>At Least Probable</th>
<th>Possible</th>
<th>No Consensus</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Lung</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CXR: bilateral hilar adenopathy (19-2-0)</td>
<td>CXR: diffuse infiltrates (4-13-3)</td>
<td>CXR: localized infiltrate (1-2-18)</td>
<td>PFT: restriction (2-6-12)</td>
</tr>
<tr>
<td>Chest CT: perilymphatic nodules (18-2-1)</td>
<td>CXR: upper lobe fibrosis (9-10-2)</td>
<td>PFT: obstruction (1-2-17)</td>
<td>PFT: isolated reduction in diffusing capacity (2-6-12)</td>
</tr>
<tr>
<td>Chest CT: symmetrical hilar/mediastinal adenopathy (21-0-0)</td>
<td>Chest CT: peribronchial thickening (10-8-3)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>PET/Gallium-67: mediastinal/hilar enhancement (17-4-0)</td>
<td>BAL: lymphocytic alveolitis (6-14-1)</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>BAL: elevated CD4/CD8 ratio (11-9-1)</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Nervous System</strong></td>
<td>Clinical syndrome consistent with granulomatous inflammation of the meninges, brain, ventricular (CSF) system, cranial nerves, pituitary gland, spinal cord, cerebral vasculature or nerve roots plus An abnormal MRI characteristic of neurosarcoidosis, defined as ex-</td>
<td>Isolated facial palsy, negative MRI (6-8-5)</td>
<td>Seizures, negative MRI (0-3-15)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Clinical syndrome consistent with granulomatous inflammation of the meninges, brain, ventricular (CSF) system, cranial nerves, pituitary gland, spinal cord, cerebral vasculature, nerve roots but</td>
<td>Cognitive decline, negative MRI (0-1-17)</td>
</tr>
</tbody>
</table>

“At least probable” = sufficient for clinical diagnosis (assuming biopsy proven sarcoidosis in another organ)
“Using” organ involvement in clinical practice

- Assess disease extent
  - Number of organs involved: >3 (HR 2.45 for progression, p=0.015)
    - Inoue 2015(Japan) 150 steroid naïve pts followed >2 yrs
    - 21% progression

<table>
<thead>
<tr>
<th></th>
<th>ACCESS (%)</th>
<th>MUSC (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pulmonary</td>
<td>95.0</td>
<td>89.1</td>
</tr>
<tr>
<td>Skin</td>
<td>15.9</td>
<td>26.0</td>
</tr>
<tr>
<td>Liver</td>
<td>11.5</td>
<td>20.0</td>
</tr>
<tr>
<td>Eye</td>
<td>11.8</td>
<td>23.0</td>
</tr>
<tr>
<td>ENT</td>
<td>3.0</td>
<td>9.6</td>
</tr>
<tr>
<td>Neurologic</td>
<td>4.6</td>
<td>9.1</td>
</tr>
<tr>
<td>Extrathoracic lymph node</td>
<td>15.2</td>
<td>12.3</td>
</tr>
<tr>
<td>Cardiac</td>
<td>2.3</td>
<td>4.5</td>
</tr>
<tr>
<td>Spleen</td>
<td>6.7</td>
<td>7.5</td>
</tr>
<tr>
<td>Renal</td>
<td>0.7</td>
<td>0.9</td>
</tr>
<tr>
<td>Parotid/SG</td>
<td>3.9</td>
<td>2.7</td>
</tr>
</tbody>
</table>
Screening for “dangerous” organ involvement

- **Cardiac**
  - Palpitations/arrhythmia, heart failure, abnormal ECG or echo
    - cardiac MRI or cardiac PET/CT

- **Neurologic**
  - “significant” neurologic symptoms (severe/intractable HA, seizures, focal weakness, paresthesias, ataxia, vision loss)
    - MRI (must be with & without contrast)

- **Hypercalcemia/Vit D dysregulation**
  - Prevalence:
    - Hypercalcemia =~10%; hypercalciuria = ~35%
    - h/o hematuria, nephrolithiasis, renal insufficiency (with no other RF)
    - Can result in CKD → dialysis, most commonly from hypercalcemia

HRS screening guidelines, 2014
MED SCHOOL FLASHBACK
Calcium Regulation

**NORMAL**
- PTH

**SARCOIDOSIS**
- IFN-γ, TNF, IL-1/2

Kidney
- 1α hydroxylase

**25 hydroxy Vit D**
- ↓

**1,25 dihydroxy Vit D**
- ↑

**PAM/monocytes**
- +

**Ca^2+**
- ↑

### Table: Calcium Regulation

<table>
<thead>
<tr>
<th>Disorder</th>
<th>25 Vit D</th>
<th>1,25 Vit D</th>
<th>PTH</th>
</tr>
</thead>
<tbody>
<tr>
<td>Vit D Deficiency</td>
<td>↓</td>
<td>↓/nl</td>
<td>↑</td>
</tr>
<tr>
<td>Hyperparathyroid</td>
<td>↓</td>
<td>↑</td>
<td>↑↑</td>
</tr>
<tr>
<td>Sarcoidosis</td>
<td>↓</td>
<td>↑</td>
<td>↓/nl</td>
</tr>
</tbody>
</table>
Hypercalcemia and Vit D labs

<table>
<thead>
<tr>
<th></th>
<th>Screening</th>
<th>Evaluation of hypercalcemia</th>
</tr>
</thead>
<tbody>
<tr>
<td>Urinalysis (ATS statement)</td>
<td>✓</td>
<td></td>
</tr>
<tr>
<td>Serum calcium</td>
<td>✓</td>
<td></td>
</tr>
<tr>
<td>Serum creatinine</td>
<td>✓</td>
<td>✓</td>
</tr>
<tr>
<td>Hydroxy and dihydroxy Vit D levels</td>
<td>✓?</td>
<td>✓</td>
</tr>
<tr>
<td>PTH</td>
<td>✓?</td>
<td>✓</td>
</tr>
<tr>
<td>24hr urine calcium (abnormal: &gt;300mg/24h)</td>
<td>✓ (if h/o stones)</td>
<td>✓</td>
</tr>
</tbody>
</table>

- **What about Vit D supplementation?**
  - 25 hydroxyl D level of 10-20ng/mL → significantly higher BM
  - Inc risk for hypercalcemia in those on Ca/VitD (42% v 18%) in one study, but only 23% had Vit D levels measured pre-supplementation

Saidenberg 2014
Factor Analysis (Rodrigues 2011)
- 137 patients followed for ≥6 months in Brazil (median 35 mos)

Four dominant phenotypes

<table>
<thead>
<tr>
<th>Relevant residual pulmonary fibrosis (&gt;1/3 lung)</th>
<th>Relapse</th>
<th>Residual airflow limitation</th>
<th>Acute disease</th>
</tr>
</thead>
<tbody>
<tr>
<td>Older</td>
<td>BDI≤6 (49%)</td>
<td>FEV1/FVC 69</td>
<td>Younger</td>
</tr>
<tr>
<td>Longer Dz duration</td>
<td>≥2 organs involved</td>
<td>Res. Fibrosis 61%</td>
<td>BDI≤6 (27%)</td>
</tr>
<tr>
<td>44% stage II, III</td>
<td>Calcium metab.</td>
<td>White (85%)</td>
<td>Stage II (44%)</td>
</tr>
<tr>
<td>Lower FVC at dx</td>
<td>Cardiac (27.3%)</td>
<td></td>
<td>Weight loss</td>
</tr>
<tr>
<td>FEV1/FVC 75</td>
<td>Neurosarc (12.5%)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Res. Obstruction 54%</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
Sarcoidosis Associated Pulmonary Hypertension

- Always consider, especially in patients with:
  - Exertional dyspnea “out of proportion” to PFTs
  - Exertional dyspnea with isolated severe reduction in DLCO with no alternative cause

- SAPH most commonly seen in stage IV disease, but can occur in stage I
- Not all patients with SAPH have “classic” signs of right heart failure
Biomarkers for Assessment of Disease Activity

- ACE?
- Soluble IL-2 receptor levels
- Chitotriosidase
- [Serum Amyloid A, CRP/ESR, lysozyme (high sens, low spec)]

Other “biomarkers”
  - PET
  - Micro RNA (potentially)
    - 8 signature miRNA’s found to have PPV of 88% for diagnosis and associated with outcomes (Ascoli 2017)
ACE levels: Einstein’s Insanity?

- Angiotensin Converting Enzyme (ACE)
  - Controls 10%, TB 9%, Silicosis 48%, Gaucher’s 100%, MM, psoriasis, lymphoma, histo

- Sarcoidosis:

<table>
<thead>
<tr>
<th>Cohort</th>
<th>N</th>
<th>Sens</th>
<th>Spec</th>
<th>Location</th>
<th>Study</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pulm</td>
<td>251</td>
<td>41.4</td>
<td>89.9</td>
<td>US</td>
<td>Ungprasert ‘16</td>
</tr>
<tr>
<td>Pulm</td>
<td>128</td>
<td>58</td>
<td>83.8</td>
<td>S. Africa</td>
<td>Ainslie ‘85</td>
</tr>
<tr>
<td>Pulm</td>
<td>51</td>
<td>86</td>
<td>61</td>
<td>Pakistan</td>
<td>Kahn ‘98</td>
</tr>
<tr>
<td>Pulm</td>
<td>73</td>
<td>68</td>
<td>75</td>
<td>Neth</td>
<td>Rothkrantz ‘03</td>
</tr>
<tr>
<td>Ocular</td>
<td>261</td>
<td>22</td>
<td>99.5</td>
<td>Germany</td>
<td>Gundlach ‘16</td>
</tr>
<tr>
<td>Ocular</td>
<td>12</td>
<td>84</td>
<td>94</td>
<td></td>
<td>Baarsma ‘87</td>
</tr>
</tbody>
</table>
So not a great diagnostic test, but…

- If significantly elevated, can help support the diagnosis
  - >100: PPV 83%  vs  >52: PPV 65%

- Can help predict tx response
  - ACE>90 predictive of >10% improvement in PFTs on methotrexate (Vorselaars ‘14)

- Can be used to follow treatment response (but NEVER use as the only treatment endpoint)

- Side note:
  - Genetic polymorphisms can affect levels
  - Be sure your patient is not on an ACE-inhibitor
Soluble IL2R & Chitotriosidase

- Released by lymphocytes and alveolar macrophages

<table>
<thead>
<tr>
<th></th>
<th>cutoff</th>
<th>Sens</th>
<th>Spec</th>
</tr>
</thead>
<tbody>
<tr>
<td>sIL-2R</td>
<td>710</td>
<td>79</td>
<td></td>
</tr>
<tr>
<td></td>
<td>1200</td>
<td>64</td>
<td>88</td>
</tr>
<tr>
<td>Chito</td>
<td>48.8</td>
<td>88.6</td>
<td>92.8</td>
</tr>
</tbody>
</table>

- Clinical use
  - sIL-2R and Chito are both higher in active disease and respond to treatment
  - sIL-2R
    - Predictive of EPS
    - Predictive of PET positivity (cutoff value >3154 pg·mL$^{-1}$)
- But, both are send out labs

Grutters 2003, Rothkrantz 2003, Bargagli 2013
PET/CT for phenotyping disease activity

- PET positive patients
  - More likely to respond to infliximab (Keijsers 2008)
  - More likely to have future decline in DLCO if treatment is withheld (Keijsers 2011)
  - Can identify occult organ involvement

- Stage IV disease
  - 14/15 PET positive (Mostard 2011)

- Currently, insurance will only pay for as a means of excluding cardiac sarcoidosis
Treatment

- When to Treat
  - Well’s Law
- Prednisone
- Second therapy
- Third Line and Beyond
When to Treat?

- Considerations
  - What’s the usual course of disease?
    - The majority of patients will have resolution of disease by 2 years without treatment
    - 10-30% of patients will go on to develop chronic/relapsing disease
  - Treatment can increase likelihood of disease chronicity
    - Treat stage 1 → increased likelihood of chronic disease
      (Neville 1983 and Pietinalho 1999)

**Only a minority of sarcoidosis patients require treatment**
Well’s Law

- There are only 2 indications to treat sarcoidosis
  - 1. Quality of Life
    - Skin: cosmetic concerns/disfiguring lesions
    - Pulmonary: dyspnea, cough
  - 2. Situations of Danger
    - Hypercalcemia
    - Cardiac/Neuro/Ophtho/Renal
    - Progressive pulmonary disease in the absence of symptoms
Treatment Algorithm

Symptoms/danger?

Yes

Rule out other causes

No

Scadding Stage 0-I

Observation Alone

Scadding Stage II-IV

Extrapulmonary sarcoidosis?

Consider steroids

First Line

Predictive alone

Taper to <10mg/d ?

Second Line

Methotrexate Azathioprine Leflunomide

Failure/Toxicity

Third Line

Infliximab Adalimumab

Other treatment options

Adapted from Korsten et al and Baughman et al
Prednisone

- Initial dose 20-40mg daily
  - Taper dose by ~50% every 6-8 weeks (as tolerated) to lowest effective dose (which should be continued for 6-9 months)

Adapted from Judson 2008
Treatment Algorithm

Symptoms/danger?

Yes

Rule out other causes

No

Scadding Stage 0-I

Scadding Stage II-IV

Observation Alone

Extrapulmonary sarcoidosis?

Consider steroids

First Line

Prednisone alone

Taper to <10mg/d ?

Second Line

Methotrexate
Azathioprine
Leflunomide

Failure/Toxicity

Third Line

Infliximab
Adalimumab

Other treatment options

Adapted from Korsten et al and Baughman et al
## Second-line Agents

<table>
<thead>
<tr>
<th>Medication</th>
<th>Administration</th>
<th>Monitoring</th>
<th>Notes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Methotrexate</td>
<td>Oral, weekly (injectable form available)</td>
<td>CBC, LFT</td>
<td>- Nausea/low WBC/liver function/oral ulcers (folate) - 2/3 will respond</td>
</tr>
<tr>
<td>(10 to 25mg)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Azathioprine</td>
<td>Oral, daily</td>
<td>CBC, LFT</td>
<td>- Nausea/rash/fever - Response rate similar to MTX - Higher infection risk</td>
</tr>
<tr>
<td>(50 to 200mg)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Leflunomide</td>
<td>Oral, daily</td>
<td>CBC, LFT</td>
<td>- Less side effects than Methotrexate</td>
</tr>
<tr>
<td>(10 to 20mg)</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

### Treatment goal: Steroid sparing agents
- Can take months to be effective
- Contraindicated in pregnancy
Treatment Algorithm

Symptoms/danger?

- Yes
  - Rule out other causes

- No
  - Scadding Stage 0-I
  - Observation Alone
  - Scadding Stage II-IV
  - Extrapulmonary sarcoidosis?
  - Consider steroids

First Line

- Prednisone alone
  - Taper to <10mg/d ?

Second Line

- Methotrexate
  - Azathioprine
  - Leflunomide
  - Failure/Toxicity

Third Line

- Infliximab
  - Adalimumab
  - Other treatment options

Adapted from Korsten et al and Baughman et al
TNF-alpha inhibitors

<table>
<thead>
<tr>
<th>Medication</th>
<th>Dosing</th>
<th>Administration</th>
<th>Monitoring</th>
<th>Notes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Infliximab (Remicade®)</td>
<td>3-5mg/kg</td>
<td>IV infusion Start at 0, 2 and 6 weeks, then continue every 4-8 weeks</td>
<td>CBC</td>
<td>Risk of allergic reaction</td>
</tr>
<tr>
<td>Adalimumab (Humira®)</td>
<td>40mg</td>
<td>Subcutaneous injection every 1-2 weeks</td>
<td>CBC</td>
<td>Less potent Longer time to reach maximum effect</td>
</tr>
</tbody>
</table>

- Insurance companies require negative TB test and Hepatitis B antigen
Infliximab Efficacy

- **Jamilloux 2017 (152 patients)**
  - 64% clinical response rate (neuro/cardiac/skin/lung/SURT)
  - No difference in use as sole agent or when added to other immunosuppressant
  - 52% experience adverse events (23% requiring tx interruption, during which 42% had flares within 14 mos)

- **Aubart 2017 (18 neurosarcoidosis patients)**
  - 89% clinical response (33% complete, 56% partial, 11 stabilized)
  - 20 month f/u: 5 had flare, 4/4 who stopped had relapse

- Can be used (& improve outcomes) in cardiac sarcoidosis patients with reduced EF
Other Therapy

- Repository Corticotropin Injection (Acthar gel®)
  - Effective for steroid dose-reduction and improving DLCO and quality of life. (Baughman 2016 and 2017)
    - Observational study using 80 units every 2 weeks reported 37% of patients required cessation of RCI, with 61% having to stop treatment due to toxicity
    - Subsequent single blinded study comparing 80 units to 40 units found no difference in efficacy between the 2 doses (significantly fewer adverse events on 40 units)
  - RCI is currently considered 4th line therapy, being limited by a relative lack of data and high cost
Other Therapy

- **Mycophenolate**
  - Only 2 observational studies in pulmonary sarcoidosis (47 total patients)
  - Suggestion of benefit (steroid sparing and improvement in FVC)

- **Cyclosporine**
  - Ineffective in pulmonary sarcoidosis and has a high side effect profile (Wyser 1997)

- **Pentoxifylline**
  - Oral medication with TNF inhibitory activity
  - Has been shown in 2 small observational studies to be steroid sparing and potentially result in improved DLCO as monotherapy (Zabel 1997, Park 2009)
Currently enrolling clinical trials:
  › CLEAR for progressive pulmonary sarcoidosis
    › Contact Robyn Do (843-792-1221) for patient screening

For new patient appointments:
  › Katie Shoptaw (Sarcoidosis Nurse Coordinator)
    › 843-792-0373

Visit the MUSC Sarcoidosis page for educational resources for patients
My email: jamesw@musc.edu
My cell: 864-993-4375
The Susan Pearlstine Sarcoidosis Center of Excellence at the Medical University of South Carolina

Charleston, SC

jamesw@musc.edu