Bacteremia and Endocarditis

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Bacteremia and Endocarditis

Steven Hatch, MD
USAID PEER/Liberia ID Lecture Series
26 November 2020
Goals

- Consider major pathogens that cause bacteremia and endocarditis in both adults and children
- Discuss pathogenesis
- Review clinical manifestations
- Discuss basic treatment strategies
- Highlight useful sources of information
A 34 year-old woman with ongoing fevers

- Generalized malaise & fevers for ~6 weeks
- Also notes some shortness of breath, DOE
- Denies wt loss but clothes more loose
- She has no major PMHx; had two uncomplicated childbirths, children now 12 and 16, healthy
- No recent trauma or prodrome, though does note minor toothache past 2-3 months
- Lives in Duazon with husband & children; has chickens & a dog
A 34 year-old woman with ongoing fevers con’t

- Vitals: 38.6°C, HR 106, BP 110/60, RR 22, O2 Sats 94%
- Exam: neck supple; one L upper molar with cavity
- Cardiac/Pulm: IV/VI systolic “whoosh” murmur at apex; lungs clear
- Abd: splenomegaly
- Painless papules on palms
A 34 year-old woman with ongoing fevers con’t

- CBC: WBC 14.6 (90% neut), Hct 30.4; Plt 288; Cr. 1.1
- Blood cultures are ordered
- 24 hours later the lab notifies you that a bacteria is growing in both aerobic and anaerobic bottles
#1: which of the following bacteria is the most likely to have grown?
#2: which was the most likely cause of the positive blood cultures?

- A. consequences of childbirth
- B. the toothache
- C. the macules on the palms
- D. the chicken
- E. the dog
- F. her husband
#3: What would be an appropriate antibiotic? (May be more than one, choose all effective abx)

- A. IV penicillin
- B. IV ceftriaxone
- C. IV amoxicillin
- D. Fluconazole
- E. RIF-INH-PZA-EMB-B6
- F. IV gentamicin
Your body is under attack every hour of every day

- Bacteria *always* trying to multiply in, on, around us
- We are commensal clouds of bacteria
- Typical adult has ~10 trillion cells
- There are at least as many bacteria, maybe much more
- Generally these bacteria benefit our lives
- But not so good when they are in bloodstream
Risks for bacteremia and endocarditis

- Anything that violates the skin and/or mucous membranes:
  - Trauma/cuts & jobs that cause them (e.g. butchering, construction)
  - Injection drug use (drugs of any kind—heroin or insulin)
  - Dental caries, inflamed gums
  - Animal exposures or bites

- Immune suppression:
  - Diabetes, Type I or Type II, worse with higher A1Cs
  - HIV

- Cardiac valvular abnormalities:
  - Rheumatic heart disease (esp in children)
  - Mitral valve prolapse
How did she develop this disease?

- Infection is a *thrombogenic* process
- Vegetations are mainly aggregations of fibrin & platelet remnants
- Bacteria get into your blood every day, but *constant* seeding of blood with bacteria increases risk of deposition on valves
Not all bacteria are equally likely to cause endocarditis

<table>
<thead>
<tr>
<th>Frequency ratios†, in rank order, of the indicated taxa in isolates from</th>
<th>endocarditis</th>
<th>purulent disease</th>
<th>bacteriæmia</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td><strong>S. mutans</strong></td>
<td>14·2:1</td>
<td><strong>S. milleri</strong></td>
</tr>
<tr>
<td></td>
<td><strong>S. bovis I</strong></td>
<td>5·9:1</td>
<td><strong>Group B</strong></td>
</tr>
<tr>
<td></td>
<td><strong>Dx + mitior</strong></td>
<td>3·3:1</td>
<td>Miscellaneous streptococci</td>
</tr>
<tr>
<td></td>
<td><strong>S. sanguis</strong></td>
<td>3·0:1</td>
<td><strong>Group A</strong></td>
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<tr>
<td></td>
<td><strong>S. mitior</strong></td>
<td>1·8:1</td>
<td><strong>Group G</strong></td>
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<td></td>
<td><strong>“Viridans”</strong></td>
<td>1·4:1</td>
<td><strong>S. bovis II</strong></td>
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<tr>
<td></td>
<td><strong>S. faecalis</strong></td>
<td>1·1:2</td>
<td><strong>S. sanguis</strong></td>
</tr>
<tr>
<td></td>
<td>Miscellaneous streptococci</td>
<td>1·1:3</td>
<td><strong>“Viridans”</strong></td>
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<td><strong>S. bovis II</strong></td>
<td>1·1:7</td>
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<td></td>
<td><strong>S. milleri</strong></td>
<td>1·2:6</td>
<td><strong>S. bovis I</strong></td>
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<td><strong>Group G</strong></td>
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<td><strong>S. mitior</strong></td>
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<td><strong>Group B</strong></td>
<td>1·7:4</td>
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<tr>
<td></td>
<td><strong>Group A</strong></td>
<td>1·32:0</td>
<td><strong>S. mutans</strong></td>
</tr>
</tbody>
</table>

† The ratio indicates the frequency of occurrence of the specified taxa in isolates from the indicated conditions.
Infective Endocarditis: Pathogenesis, complications, and clinical findings

In acute endocarditis, valve trauma or the invading bacterium damages the valve endothelium.

In subacute endocarditis, non laminar flow (due to pre-existing valvular stenosis or regurgitation) damages valve endothelium, a sterile thrombus forms.

Poor dental hygiene / recent dental procedure

Intravenous drug use (mostly cause R sided endocarditis)

Invasive procedure / indwelling device

Bacteria [see Microbiology Note] enter the bloodstream (Bacteremia)

Positive blood cultures

Activation of immune system → generalized immune response

Fever, Malaise

Thrombus forms on the surface of a cardiac valve

Bacteria adhere to thrombi on the cardiac valve endothelium

In subacute cases, valvular abnormality usually present beforehand

Mitrail Regurgitation, Aortic Stenosis, Aortic Insufficiency / Valve Involvement: Mitrail > Aortic > Tricuspid

In all cases, vegetation forms on affected valve

Vegetation seen on ultrasound (echocardiogram)

Formation of immune complexes (complexes of antibody bound to antigen) secondary to infection

Immune complexes deposit in kidney → damage to glomeruli

Immune complexes cause vasculitis in retinal vessels

Immune complexes deposit subcutaneously

Roth’s Spots (retinal hemorrhages with pale centers due to coagulated fibrin)

Osler Nodes (tender, raised, red lesions found on the hands & feet)

Smaller emboli block smaller vessels on hands / feet → microinfections

Splinter hemorrhages (small red streaks under nails)

Janeway Lesions (non-tender, red macules / nodules on palms / soles – only a few millimeters wide)

Valve unable to fulfill normal functions

Cardiac valve insufficiency, regurgitation

Parts of vegetation embolize systemically, obstructing arteries

Blood flow to organs perfused by the obstructed arteries

Organ infarction

Infection destroys infected valve

Blood flow to organs perfused by the obstructed arteries

Microbiology Note:

bacteria causing endocarditis varies depending on the type of valve:

- Native Valve: Viridans streptococci (in young patients), Enterococcus (in older patients), Staph aureus (for both)
- Prosthetic Valve: Staph aureus or Coagulase-negative Staph
#4: what would be the main sequelae of her infection if left untreated? (check all that apply)

- A. Immunologic phenomena
- B. Renal failure
- C. Hepatotoxicity from bacterial toxins
- D. CVA
- E. Splenic rupture
Once vegetations are established, then what happens?

- Sustained bacteremia, sepsis & septic shock
- Emboli: often cause infarcts of downstream tissue
  - Left: CVA; L main coronary artery; renal & splenic infarcts, toes/fingers (Janeway lesions, Roth’s spots); vertebra
  - Right: pulmonary infarcts
- Emboli part 2: metastatic abscesses (mainly w/ *Staph*)
- Valve destruction
  - Left: aortic insufficiency, flash pulmonary edema, congestive hepatopathy
  - Right: less physiologically consequential due to low-pressure system
- Immune phenomena: Osler nodes
Roth’s spots
Mimics of Endocarditis

- Noninfectious endocarditis:
  - Libmann-Sacks endocarditis (Lupus)
  - Marantic endocarditis (cancer)
- Acute rheumatic fever
- Thrombi (hypercoagulability)
- Myxoma
#5. Treatment length should be:

- A. 3 days
- B. 7 days
- C. 14 days
- D. 28 days
- E. 42 days
- F. It depends


