Bacteremia and Endocarditis

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Bacteremia and Endocarditis

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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

| Frequency ratios†, in rank order, of the indicated taxa in isolates from |
|--------------------------|--------------------------|--------------------------|
|                          | endocarditis             | purulent disease          | bacteriaemia             |
|                          |                          |                          |                          |
| **S. mutans**            | 14:2:1                   | **S. milleri**           | 5:1:1                    | Group A                   |
| **S. bovis I**           | 5:9:1                    | Group B                  | 3:3:1                    | Group G                   |
| **Dx + mitior**          | 3:3:1                    | Miscellaneous streptococci| 2:4:1                    | **S. faecalis**           |
| **S. sanguis**           | 3:0:1                    | Group A                  | 1:5:1                    | **S. bovis II**           |
| **S. mitior**            | 1:8:1                    | Group G                  | 1:1:1                    | Group B                   |
| "Viridans"              | 1:4:1                    | **S. bovis II**          | 1:1:2                    | "Viridans"               |
| **S. faecalis**          | 1:1:2                    | **S. sanguis**           | 1:2:2                    | **S. mitior**             |
| Miscellaneous streptococci| 1:1:3                    | "Viridans"               | 1:3:0                    | **Dx + mitior**          |
| **S. bovis II**          | 1:1:7                    | **S. faecalis**          | 1:3:7                    | Miscellaneous streptococci|
| **S. milleri**           | 1:2:6                    | **S. bovis I**           | 1:3:8                    | **S. sanguis**            |
| Group C                  | 1:3:0                    | **S. mitior**            | 1:4:5                    | **S. milleri**            |
| **Group B**              | 1:7:4                    | **Dx + mitior**          | †                        | **S. bovis I**            |
| **Group A**              | 1:32:0                   | **S. mutans**            | ‡                        | **S. mutans**             |

† Ratios indicate frequency of occurrence in endocarditis, purulent disease, and bacteriaemia.

‡ Data not available.
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.**

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

**Activation of immune system → generalized immune response → Fever, Malaise.**

**Positive blood cultures**

**Infection of the thrombus typically produces a vegetation on the flow surface of a valve.**

**In subacute cases, valvular abnormality usually present beforehand.**

**In all cases, vegetation forms on affected valve.**

**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).**

**blood flow to organs perfused by the obstructed arteries.**

**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).**

**Infection destroys infected valve → Valve unable to fulfill normal functions.**

**Cardiac valve insufficiency, regurgitation.**

**Vegetation seen on ultrasound (echocardiogram).**

**Glomerulonephritis.**

**Mitrval Regurgitation, Aortic Stenosis, Aortic Insufficiency (Valve involvement: Mitrval > Aortic > Tricuspid).**

**Legend:** Pathophysiology, Mechanism, Sign/Symptom/Lab Finding, Complications, Published August 20, 2013 on www.thecalgaryguide.com
#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
Further Reading


