Kitchen Sink: Syndromes Not Covered Elsewhere

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Disclosures of Financial Relationships with Relevant Commercial Interests

- None

Session Plan

- Case-based discussions of topics not extensively covered in other sessions
- Highlight points likely to be assessed on ID Boards (rather than comprehensive overview)

Question #1

- A 51-year-old male with past medical history significant for insulin dependent diabetes presents with a six-month history of progressive arthralgias, abdominal pain, diarrhea, weight loss, and low-grade fevers.

- Work up thus far:
  - Negative blood cultures x 2
  - Negative Rheumatoid factor
  - Normal metabolic panels
  - Mild normocytic anemia

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Speaker: Stacey Rose, MD

Question #1

Which of the following tests will most likely yield the diagnosis?

A. Anti-streptolysin O Antibody
B. Anti-nuclear Antibody
C. Stool ova and parasite
D. Duodenal biopsy

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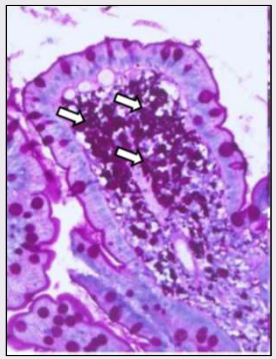
D. **Duodenal biopsy***

Diagnosis: Whipple's disease

Whipple's Disease

- Caused by *Tropheryma whipplei* (gram variable bacterium, difficult to cultivate)
- More common in middle-aged white males
- Diagnosis often delayed due to indolent clinical presentation
- Most commonly diagnosed via duodenal biopsy, stained with PAS
- PCR increasingly used



Periodic acid-Schiff-diastase (PAS-D)-stained duodenal biopsy specimens with PAS-D-positive granules in the foamy macrophages (arrows).

Dolmans RAV, Boel CHE, Lacle MM, Kusters JG. 2017. Clinical manifestations, treatment, and diagnosis of Tropheryma whipplei infections. Clin Microbiol Rev 30:529 –555.

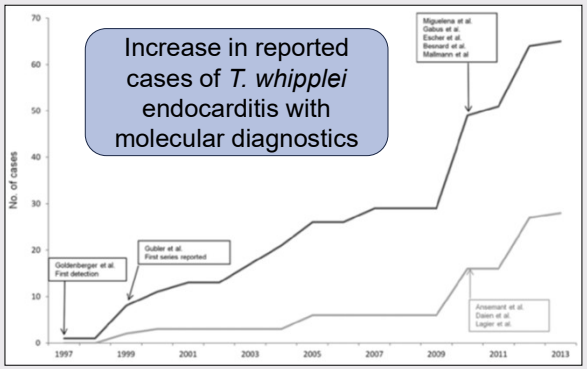
Whipple's: Clinical Presentations

TABLE 1 Clinical manifestations of *Tropheryma whipplei* infection[a]

Classic Whipple's disease (% incidence)	Chronic localized infections[b]	Acute infections[b]
Weight loss (79–99)	Endocarditis	Gastroenteritis
Gastroenteritis (63–85)	Encephalitis	Pneumonia
Abdominal pain (23–60)		Bacteremia
Arthritis (20–83)		
Neurological symptoms (6–63)		

Dolmans RAV, Boel CHE, Lacle MM, Kusters JG. 2017. Clinical manifestations, treatment, and diagnosis of Tropheryma whipplei infections. Clin Microbiol Rev 30:529 –555.

Whipple's Endocarditis - Increasingly Recognized



- Consider in patients with arthralgias plus **"culture negative" endocarditis**
- *T. whipplei* PCR from blood added to Duke's criteria (2023) for diagnosis of endocarditis

Fenollar F, Célard M, Lagier JC, Lepidi H, Fournier PE, Raoult D. Tropheryma whipplei endocarditis. *Emerg Infect Dis*. 2013
Fowler VG, et al. The 2023 Duke-International Society for Cardiovascular Infectious Diseases Criteria for Infective Endocarditis: Updating the Modified Duke Criteria. *Clin Infect Dis*. 2023.

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Whipple's: Treatment

No Gold Standard

Symptoms improve, but relapse is common without prolonged treatment / suppression

Options:

- Ceftriaxone or meropenem plus prolonged trimethoprim-sulfamethoxazole (~1 year)

OR

- Doxycycline plus hydroxychloroquine (12-18 mos)

Clinical manifestations, treatment, and diagnosis of Tropheryma whipplei infections. Clin Microbiol Rev 2017.
Whipple's disease and Tropheryma whipplei infections: from bench to bedside. Lancet Infect Dis. 2022
Principles and Practice of Infectious Diseases, 9th ed

Whipple's Disease
Take Home Points

- Cause: *Tropheryma Whipplei*
- Epidemiology: middle-aged, white males
- Clinical presentation: classic – *arthralgia, diarrhea, weight loss*
- Localized infection e.g., *endocarditis* (increasingly recognized)
- Diagnosis with *duodenal biopsy* (*PAS* stain; foamy macrophages)
  or *PCR* of infected tissue or blood
- Prolonged treatment needed to prevent relapse

Question #2

- A 20-year-old female schoolteacher presents with a 1-week history of fever and pain / swelling in knees, elbows and wrists. She notes that the pain moves from joint to joint.
- She reports being ill ~3 weeks prior with sore throat and headache which resolved without specific treatment.
- She has no rash or lymphadenopathy.
- She denies travel. She is sexually active with one male partner, using barrier protection (condoms).
- Labs are notable for elevated ESR and CRP and + ASO and Anti-DNase B titers; pregnancy and HIV tests (4th generation Ag/Ab) are negative.

Question #2

Which of the following is the best explanation for her symptoms?

A. Acute HIV infection
B. Mononucleosis due to Epstein Barr Virus
C. Acute rheumatic fever
D. Lemierre's syndrome

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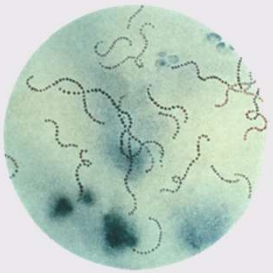

Streptococcus pyogenes (group A *Streptococcus*) on Gram stain. Source: Public Health Image Library, CDC

Explanation

- ✗ **Acute HIV** – joint symptoms not characteristic; HIV 4th gen testing should detect early infection
- ✗ **Mononucleosis due to EBV** – joint pains not characteristic; no mention of lymphadenopathy
- ✓ **Acute Rheumatic fever** – multisystem disease following group A strep; meets Jones criteria
- ✗ **Lemierre's** – septic thrombophlebitis of internal jugular vein following pharyngitis, typically caused by *Fusobacterium necrophorum*; joint pains not characteristic, no neck swelling

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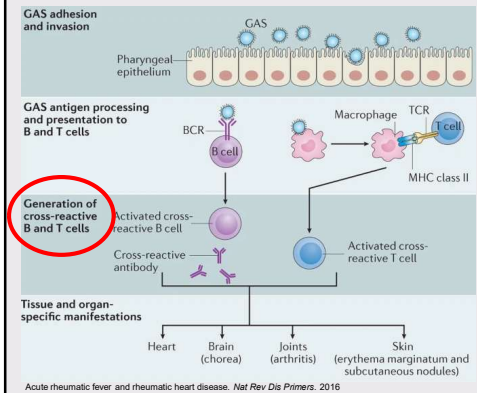
Acute Rheumatic Fever



Acute rheumatic fever and rheumatic heart disease. *Nat Rev Dis Primers*. 2016

- Rare in US (0.5 per 100K per year), but common worldwide (0.5 million per year)
- Affects ***children / young adults***
- Recurrence common
- *Pathogenesis*: immune response following *Streptococcus pyogenes* pharyngitis
- Leads to systemic manifestations (**arthritis**, **carditis**, **chorea**, **skin**)

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Revised Jones Criteria

For patients with evidence of prior GAS infection*,
Acute Rheumatic fever =
2 MAJOR
OR
1 MAJOR plus 2 MINOR

Major	Minor
Arthritis (usually migratory polyarthritis)	Arthralgia
Carditis (clinical or subclinical)	Fever
Chorea	Elevated ESR or CRP
Erythema marginatum	Prolonged PR interval (unless carditis is a major criterion)
Subcutaneous nodules	

*e.g., rapid strep test; culture; anti-streptolysin-O titer (ASO) or anti-DNase B (ADB)

Revision of the Jones Criteria for the diagnosis of acute rheumatic fever in the era of Doppler echocardiography: a scientific statement from the American Heart Association. *Circulation*. 2015

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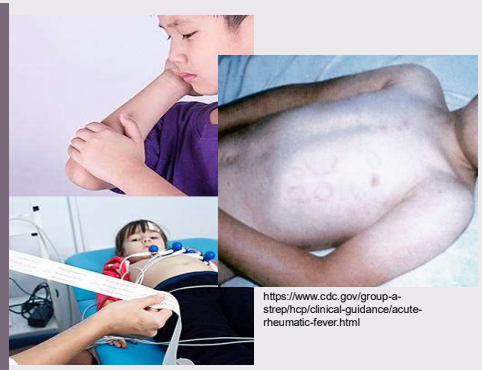
Revision of the Jones Criteria for the diagnosis of acute rheumatic fever in the era of Doppler echocardiography: a scientific statement from the American Heart Association. *Circulation*. 2015

Recognizing Acute Rheumatic Fever

- **Timing**: ~19 d after GAS infection
- **Arthritis**: migratory, polyarthritis involving large joints (knees, ankles, elbows, wrists)
- **Carditis**: wide range of effects – e.g., pericarditis, systolic dysfunction, valvular disease
- **Chorea**: late manifestation; involuntary movements
- **Skin**: Subcutaneous nodules; erythema marginatum (blanches; transient) – rare but specific



https://www.cdc.gov/group-a-strep/hcp/clinical-guidance/acute-rheumatic-fever.html

Karthikeyan G, Guilherme L. Acute rheumatic fever. *Lancet*. 2018.
Principles and Practice of Infectious Disease, 9th ed.

Treatment and Prophylaxis of Acute Rheumatic Fever

Primary Episode	Secondary Prophylaxis
IM benzathine penicillin x 1 OR Oral penicillin x 10 d	IM benzathine penicillin q 4 weeks

Goal: *to prevent rheumatic heart disease*

Duration of ppx: *varies by severity of primary illness*

Contemporary Diagnosis and Management of Rheumatic Heart Disease: Implications for Closing the Gap: A Scientific Statement From the American Heart Association. *Circulation*. 2020
Principles and Practice of Infectious Diseases, 9th ed.

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Duration of Secondary Prophylaxis Following Acute Rheumatic Fever:
Longest if Carditis and Residual Valvular Disease

CATEGORY	DURATION AFTER LAST ATTACK
Rheumatic fever with carditis and residual heart disease (persistent valvular disease[a])	10 yr or until age 40 yr, whichever is longer; sometimes lifelong prophylaxis (see text)
Rheumatic fever with carditis but no residual heart disease (no valvular disease[a])	10 yr or until age 21 yr, whichever is longer
Rheumatic fever without carditis	5 yr or until age 21 yr, whichever is longer

SEE ENLARGED SLIDE FOLLOWING ENTIRE PRESENTATION

Contemporary Diagnosis and Management of Rheumatic Heart Disease: Implications for Closing the Gap: A Scientific Statement From the American Heart Association. *Circulation*. 2020
Principles and Practice of Infectious Diseases, 9th ed

Acute Rheumatic Fever
Take Home Points

- Cause: immune dysregulation following *S. pyogenes* infection
- Epidemiology: children / young adults; rare in US
- Clinical presentation: ~3 weeks following GAS infection
  - Major: *migratory polyarthritis, carditis, chorea, subcutaneous nodules, erythema marginatum*
  - Minor: *fever, arthralgia, elevated ESR/CRP; PR prolongation*
- Diagnosis based on Jones criteria = 2 major OR 1 major + 2 minor (plus e/o prior GAS infection e.g., ASO titer)
- Treatment and secondary ppx with IM Penicillin; duration based on carditis (10 yr or to age 40 if carditis + residual valvular disease)

Question #3

- A 34-year-old male with a history of injection drug use presents to the emergency room with two days of blurry vision and difficulty swallowing. He is also beginning to feel weak in his arm muscles.
- On examination, vital signs are normal, but the patient is noted to have ptosis and sluggish pupillary responses as well as slurred speech.

Question #3

Which of the following treatments are recommended?

A. Plasmapheresis
B. Naloxone
C. Tetanus antitoxin
D. Botulinum antitoxin

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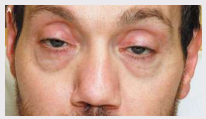
Explanation

Tetanus: sardonic smile



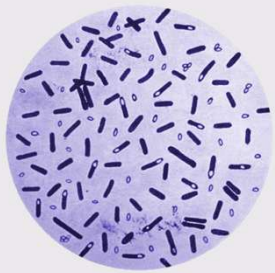
Botulism: ptosis

- ✗ Plasmapheresis – for Lambert-Eaton syndrome, immune attack of neuromuscular junction (chronic; associated with lung cancer)
- ✗ Naloxone – for opioid intoxication (respiratory suppression, *constricted* pupils)
- ✗ Tetanus antitoxin – for tetanus (rigid paralysis)
- ✓ Botulinum antitoxin – for botulism (flaccid paralysis)

SEE ENLARGED SLIDE FOLLOWING ENTIRE PRESENTATION

https://www.thelancet.com/journals/lancet/article/PIIS0140-6736(19)31137-7/fulltex
https://www.nejm.org/doi/pdf/10.1056%2FNEJMicm1003352

Botulism



https://phil.cdc.gov/details.aspx?pid=2107

- Caused by **Clostridium botulinum* (gram positive, strict anaerobe with subterminal spore; found in soil)
- Toxin prevents release of acetylcholine in neuromuscular junction
- Leads to flaccid paralysis of motor and autonomic nerves, beginning with the cranial nerves (*descending* weakness)
- DX: culture or detection of toxin

**other neurotoxin producing species of Clostridium: C. butyricum*, or *C. baratii*

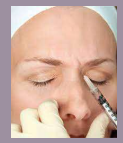
Botulism

Foodborne

Infant

Wound (black-tar heroin)

Iatrogenic



Peak CM, Rosen H, Kamali A, et al. Wound Botulism Outbreak Among Persons Who Use Black Tar Heroin — San Diego County, California, 2017–2018. MMWR 2019.
https://www.cdc.gov/botulism/hcp/clinical-overview/index.html; *Principles and Practice of Infectious Diseases, 9th ed.*

RED FLAGS: symmetric CN palsies and descending / symmetric flaccid paralysis should raise suspicion for botulism

Adverse Effects Linked to Counterfeit or Mishandled Botulinum Toxin Injections

Print

HAN HEALTH ALERT NETWORK

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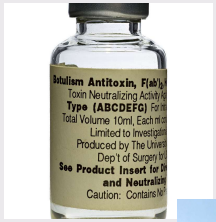
Distributed via the CDC Health Alert Network
April 23 2024, 11:00 AM ET
CDCHAN-00507

https://emergency.cdc.gov/han/2024/han00507.asp

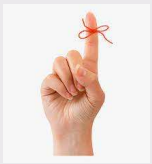
Botulism Treatment

Supportive Care	Antitoxin
• Ventilatory support for respiratory compromise • Wound debridement	• Administer Botulinum anti-toxin (BAT) asap to prevent progression • For infant botulism syndrome, use Botulinum immune globulin (BabyBIG®)



Rao AK, Sobel J, Chatham-Stephens K, Luquez C. Clinical Guidelines for Diagnosis and Treatment of Botulism, 2021. *MMWR Recomm Rep.* 2021. *Principles and Practice of Infectious Diseases, 9th ed.; https://www.cdc.gov/botulism/hcp/clinician-resources/index.html*

Botulism
Take Home Points



- **Cause**: *Clostridium botulinum* toxin impedes acetylcholine release from neuromuscular junction
- **Epidemiology**: food-borne (home-canned veggies, fruits, fish); infant (honey); wound (black-tar heroin); iatrogenic (rare)
- **Clinical features**: *symmetric, descending flaccid paralysis*, starting with *cranial nerves* (ptosis, blurry vision, slurred speech)
- **Diagnosis**: clinical; confirmed by culture or detection of toxin
- **Treatment**: *antitoxin* & supportive care; wound debridement

Question #4

- A 34-year-old motorcyclist is involved in a severe motor vehicle accident, resulting in laceration of the spleen and requiring splenectomy

Question #4

Post-splenectomy, the patient is at increased risk of severe disease due to which of the following microorganisms?

A. Helicobacter pylori
B. Capnocytophaga canimorsus
C. Candida glabrata
D. Clostridium difficile

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A. Helicobacter pylori
B. Capnocytophaga canimorsus*
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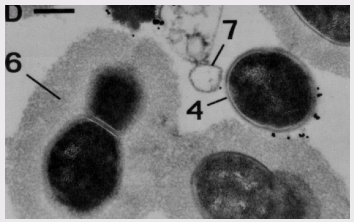
D. Clostridium difficile

Splenectomy and Infection Risk

- **Why:** reduced clearance of encapsulated organisms; impaired humoral immunity



Skov Sørensen et al. (1988) Infect Immun 56: 1890-1896

On the boards, look for…
- *Streptococcus pneumonia*
- *Hemophilus influenza type B*
- *Neisseria meningitidis*
- *Capnocytophaga canimorsus (dog bite)*
- *Babesia microti (tick borne)*
- *Bordetella holmesii*
- *Salmonella typhi*

Rubin LG, Schaffner W. Clinical practice. Care of the asplenic patient. N Engl J Med. 2014

Strategies to Reduce Infection Risk in Asplenia

Vaccination for Encapsulated Organisms
- Pneumococcus
- Meningococcus
- Hemophilus influenza Type B (HiB)

Penicillin Prophylaxis
- Children < 5 years
- Older children/ adults within 1-2 years of splenectomy
- Any age: secondary prevention (lifelong) following sepsis

Rubin LG, Schaffner W. Clinical practice. Care of the asplenic patient. N Engl J Med. 2014; Lee GM. Preventing infections in children and adults with asplenia. Hematology Am Soc Hematol Educ Program. 2020

Infection in Asplenia
Take Home Points

- Increased risk for infection with encapsulated organisms (and others)…
  - *S. pneumoniae; N. meningitidis; HIB; Capnocytophaga; Babesia; Salmonella typhi*
- Reduce risk of infection via:
  - Immunizations
  - PCN ppx if < 5 yrs old; recent splenectomy; h/o sepsis

Question #5

At Clinical Presentation

1 Week After Discharge

- A 19-year-old male presents with **fever**, **tachycardia**, and **hypotension**. On examination, he is found to have two **skin abscesses** in the pubic region, as well diffuse **erythroderma** most notable on the palms.
- Laboratory analyses show **leukocytosis**, **anemia**, **transaminitis**, and **acute kidney injury**. Chest X-ray shows small bilateral **pleural effusions**.
- He is treated with intravenous fluids, inotropic support and empiric broad-spectrum antibiotics. Incision and drainage of the skin abscesses is performed; cultures grow **Methicillin Sensitive Staphylococcus Aureus (MSSA).**
- 1 week after discharge, he develops **skin peeling** on his palms (see images).

J Family Med Prim Care 2022;11:4837-40.

Question #5

At Clinical Presentation

1 Week After Discharge

The patient's clinical syndrome was most likely caused by which of the following?

A. Pyrogenic toxin superantigen
B. Bacterial endotoxin
C. Cereulide toxin
D. Aflatoxin

J Family Med Prim Care 2022;11:4837-40.

Question #5

At Clinical Presentation

1 Week After Discharge

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B. Bacterial endotoxin
C. Cereulide toxin
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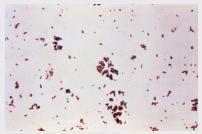
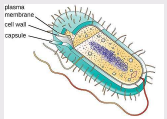
J Family Med Prim Care 2022;11:4837-40.

Explanation

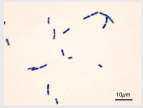
Pyrogenic toxin superantigen - cause of toxic shock syndrome (see next slide)


wwwn.cdc.gov/phil/Details.aspx?pid=5147

Bacterial endotoxin e.g., lipopolysaccharide (LPS) - gram negative cell wall component that can trigger systemic inflammation



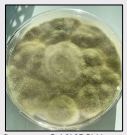
commons.wikimedia.org/wiki/File:OSC_Microbio_03_03_Capsules.jpg



commons.wikimedia.org/wiki/File:Bacillus_cereus_Gram.jpg

Cereulide toxin - produced by *Bacillus cereus*, cause of food poisoning / emesis

Aflatoxin - produced by toxigenic molds e.g., *Aspergillus flavus*, exposure to large quantity can lead to liver injury


commons.wikimedia.org/wiki/File:Aspergillus_flavus_sur_Sab%2BChl.jpg

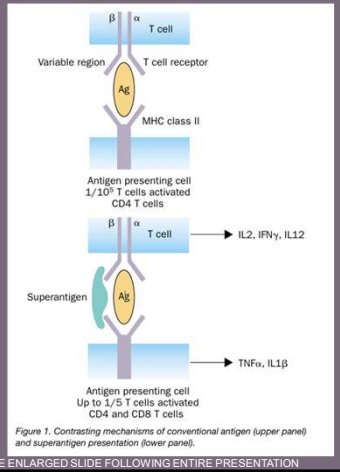


Figure 1. Contrasting mechanisms of conventional antigen (upper panel) and superantigen presentation (lower panel).

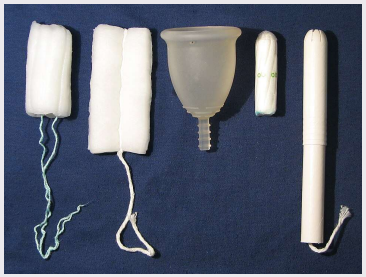
SEE ENLARGED SLIDE FOLLOWING ENTIRE PRESENTATION

Staphylococcal Toxic Shock Syndrome (TSS)

- Bacteria produces **pyrogenic toxin superantigen** (e.g., TSST-1, Staphylococcal Enterotoxin B)
- Superantigen stimulates MHC class II and T cell receptor on antigen presenting cells in a way that bypasses traditional mechanism of activation
- **Activates a large number of T cells at the same time → cytokine "storm"**

Superantigens: microbial agents that corrupt immunity. Lancet Infect Dis. 2002

Risk Factors for Staphylococcal TSS


https://commons.wikimedia.org/wiki/File:Fleurcup_and_tampons.jpg

- Colonization or infection with **toxin-producing strains** (MSSA > MRSA)
- **Menstrual**: use of tampons; also associated with menstrual cups
- **Non-menstrual**: skin infection, burns, surgery

Clinical Features

4 Criteria - *Probable*

5 Criteria - *Confirmed*

***Panel 1*: Staphylococcal toxic shock syndrome clinical case definition**

1 Fever ≥38·9°C
2 Rash—diffuse macular erythroderma
3 Desquamation—1–2 weeks after onset of illness, especially of palms and soles
4 Hypotension—systolic blood pressure ≤90 mm Hg for adults
5 Multi-system involvement—3 or more of the following:
  a Gastrointestinal—vomiting or diarrhoea at the onset of illness
  b Muscular—severe myalgia or elevated creatine phosphokinase
  c Mucous membranes—vaginal, oropharyngeal, conjunctival hyperaemia
  d Renal—blood urea nitrogen or creatinine twice-upper limit of normal
  e Hepatic—total bilirubin twice-upper limit of normal
  f Haematological—platelets ≤100×10^9/L
  g CNS—disorientation or alterations in consciousness without focal neurological signs
6 Negative results on the following tests:
  a Blood, throat, or cerebrospinal fluid culture (blood culture may be positive for *S aureus*)
  b Rise in titre to Rocky Mountain spotted fever, leptospirosis, or measles

Case classification
Probable: case with five of the six clinical findings described
Confirmed: case with all six of the clinical findings described

SEE ENLARGED SLIDE FOLLOWING ENTIRE PRESENTATION

Gram-positive toxic shock syndromes. Lancet Infect Dis. 2009

Treatment

https://en.wikipedia.org/wiki/Clindamycin

Anti-staphylococcal Antibiotic:

- No risk factor for MRSA: **oxacillin** or **nafcillin** or **cefazolin**
- MRSA risk factor: **vancomycin** or **daptomycin**

PLUS

Anti-toxin Agent: **clindamycin** or **linezolid**

PLUS

Source Control

Toxic Shock Syndrome: A Literature Review. Antibiotics (Basel). 2024

Staphylococcal TSS
Take Home Points

- **Epidemiology**: *menstrual* – tampon or menstrual cup use; *non-menstrual* – colonization or infection with toxin-producing strain, burns, surgery
- **Clinical presentation**: fever, rash (erythroderma), desquamation after 1-2 weeks; hypotension; multi-system involvement (suspect in sepsis-like picture)
- **Diagnosis:** clinical; can confirm with isolation of toxin
- **Treatment**: anti-staphylococcal antibiotic plus anti-toxin antibiotic (clindamycin or linezolid) plus source control

Question #6

- WBC 11 x 10^9 / L (high) with 85% neutrophils
- Hgb 13.6 g/dL (normal)
- Platelets 460,000 / mcL (high)
- Cr 1.3 mg/dL (high)
- Na 136 meq/L, K 4.0 meq/L (normal)
- ALT 30, AST 33 U/L (normal)
- Total bilirubin 2.0 mg/dL (high)
- Alkaline phosphatase 190 U/L (high)

- A 65-year-old male undergoes partial colectomy for colon cancer.
- His convalescence is complicated, requiring ICU stay and total parenteral nutrition.
- On hospital day #11, he develops acute onset of fever and hypotension without localizing symptoms. The surgical site is benign-appearing.
- Notable labs are as shown. Blood and urine cultures and drawn, and the patient is started on empiric broad spectrum antibiotics.
- Bedside right upper quadrant ultrasound is performed and shows gall bladder wall thickening without dilation of the common bile duct, and Murphy's sign is negative.

Question #6

Which of the following describes the best next step in management?

A. Computed tomography (CT) abdomen / pelvis
B. CT with PE protocol
C. Hepatic iminodiacetic acid scan (HIDA)
D. Diagnostic laparoscopy

Indian J Dermatol. 2010;55(1):79-85.

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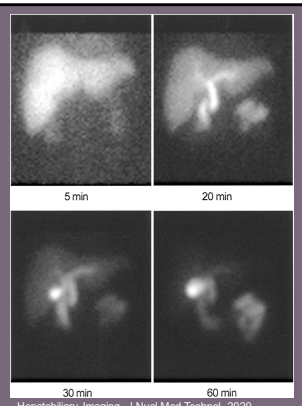

Hepatobiliary Imaging. J Nucl Med Technol. 2020

Normal HIDA Scan Showing Radiotracer In Gall Bladder

SEE ENLARGED SLIDE FOLLOWING ENTIRE PRESENTATION

- **HIDA scan: most sensitive and specific**, non-invasive, safe in renal dysfunction, advised if ultrasound is non-diagnostic
- CT scan: high sensitivity and can rule out other abdominal pathology, but contrast not advised in renal dysfunction
- MRI: most useful if suspect choledocholithiasis but not ideal in renal dysfunction
- Exploratory laparotomy: too invasive

Acute Acalculous Cholecystitis: Epidemiology

- **Pathophysiology**:
  - Fasting → **inhibited emptying of gallbladder** → bile stasis / toxicity
  - Ischemic injury due to vascular constriction or obstruction
- **Risk Factors**:
  - Surgery
  - Trauma
  - Total parenteral nutrition
  - Burn injury
  - Ischemic heart disease
  - Infections

Bacterial: *Brucella, Coxiella, Leptospira, Orientia tsutsugamushi, M. Tuberculosis, Salmonella, Vibrio cholerae*

Fungal: Candida spp

Parasitic: Cyclospora, Microsporidia, Malaria, Schistosomiasis, Echinococcus (obstruction)

Viral: CMV, Dengue

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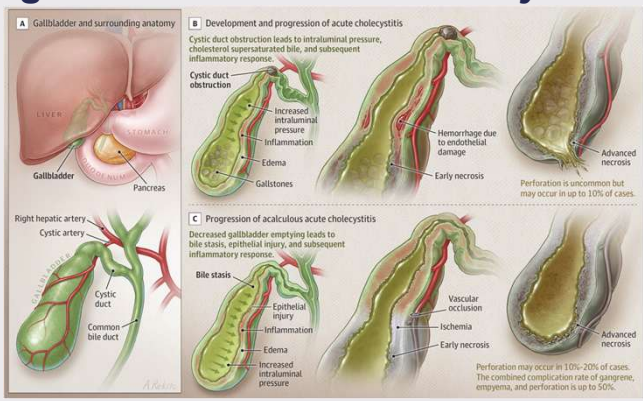
Acute Acalculous Cholecystitis. Curr Treat Options Gastroenterol. 2005

Diagnosis: *Acalculous cholecystitis*



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Acute Cholecystitis: A Review. JAMA. 2022

Clinical Presentation and Treatment

Presentation more **subtle** than for calculous cholecystitis; **RUQ pain** only present in ~25%

Consider diagnosis in critically ill patient with unexplained **fever**, **leukocytosis**, **elevated liver function tests**

Treat with perioperative antibiotics: ampicillin / sulbactam or piperacillin / tazobactam

PLUS laparoscopic **cholecystectomy** or percutaneous **cholecystostomy** (if not surgical candidate)

Acute Acalculous Cholecystitis. Curr Treat Options Gastroenterol. 2005; Acute Cholecystitis: A Review. JAMA. 2022;Tokyo Guidelines 2018: antimicrobial therapy for acute cholangitis and cholecystitis. J Hepatobiliary Pancreat Sci. 2018l Surgical Infection Society Guidelines for Antibiotic Use in Patients Undergoing Cholecystectomy for Gallbladder Disease. Surg Infect (Larchmt). 2022

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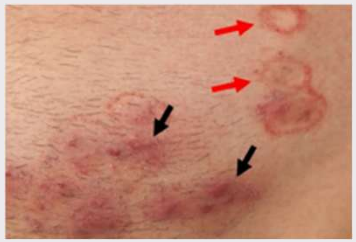
Acalculous Cholecystitis
Take Home Points

- **Risk factors**: surgery, trauma, TPN, burns, ischemia, infections
- **Diagnosis**: subtle presentation; consider if unexplained fever / leukocytosis / elevated LFT's in critically ill patient.
  - Confirm with ultrasound → HIDA scan
- **Treatment**: broad-spectrum antibiotics (ampicillin / sulbactam or piperacillin / tazobactam), then laparoscopic cholecystectomy or percutaneous cholecystostomy (if not surgical candidate)

Question #7



- A 35-year-old male businessman presents with new skin lesions on the trunk, buttocks and extremities. The lesions are pruritic and he is concerned they are spreading.
- He is otherwise healthy. He does not smoke, drinks socially and engages in sexual contact with men; he takes pre-exposure prophylaxis for HIV and uses doxycycline for post-exposure prophylaxis if / when he engages in unprotected intercourse.
- He recently traveled to Paris, France for a work conference.
- Skin lesions are as pictured. Exam is also notable for inguinal lymphadenopathy.

Indian J Dermatol. 2010;55(1):79-85.

Question #7

Which of the following is the most likely diagnosis?

A. Condyloma lata (syphilis)
B. Bacterial folliculitis
C. Trichophyton rubrum
D. Trichophyton mentagrophytes genotype VII

Indian J Dermatol. 2010;55(1):79-85.

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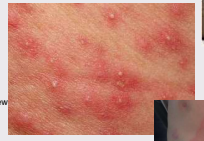
Indian J Dermatol. 2010;55(1):79-85.

Explanation

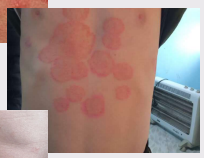
Extensive Condyloma Lata Lesions in Unusual Sites: An Atypical Manifestation of Secondary Syphilis. Case Rep Med. 2025

Condyloma lata (syphilis)



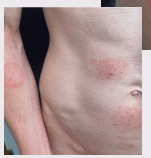
Bacterial folliculitis

https://www.pcds.org.uk/clinical-guidance/folliculitis-an-overview



Trichophyton rubrum: most common cause of tinea corporis

https://commons.wikimedia.org/wiki/File:Tinea_corporis.jpg

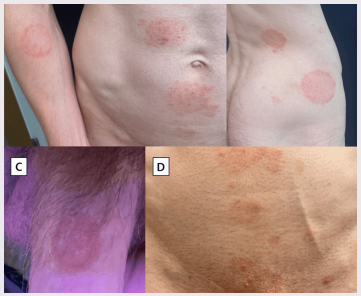


Trichophyton mentagrophytes genotype VII: Sexual transmission, MSM

https://www.cdc.gov/mmwr/volumes/73/wr/mm7343a5.htm

Trichophyton Mentagrophytes Genotype VII (TMVII)

- First described in travelers returning from Southeast Asia; now in Europe, US
- Mostly in men who have sex with men **(MSM) / sexual transmission**
- **Clinical Features**:
  - Lesions located on genital, perianal, perioral / facial, trunk / extremities
  - Superficial or subcutaneous / purulent
  - 1/3 with lymphadenopathy
  - **Pain and itching** common (>75%)
- **Treatment**: oral **terbinafine** until symptoms resolve (up to 3 months); counsel re: transmission risk



Trichophyton mentagrophytes type VII: cohort study on patient characteristics, clinical features, disease course, and treatment. J Dtsch Dermatol Ges. 2025; *Notes from the Field: Trichophyton mentagrophytes* Genotype VII — New York City, April–July 2024. MMWR Morb Mortal Wkly Rep 2024;73:985–988.

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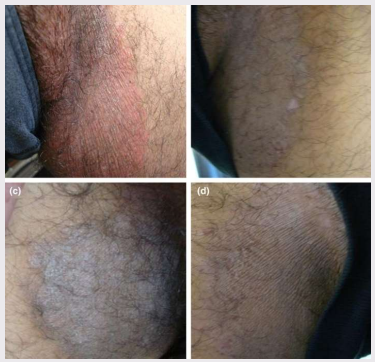
Another Emerging Infection with Reported Sexual Transmission: *Trichophyton indotineae*



- First identified in India, now reported in > 40 countries
- Equally affects men and women
- Person to person contact; sexual transmitted reported (rare)
- Antifungal resistance common; optimal treatment not defined

Trichophyton indotineae: Epidemiology, antifungal resistance and antifungal stewardship strategies. J Eur Acad Dermatol Venereol. 2026

Trichophyton Mentagrophytes Genotype VII (TMVII)
Take Home Points

- **Risk factors**: MSM
- **Diagnosis**: clinical; typically superficial, ringworm-like lesions on **genital** / **perianal** / **perioral** areas but can be subcutaneous / pustular with lymphadenopathy. **Pain**, **itching** common.
- **Treatment**: oral **terbinafine** for ~3 months

Kitchen Sink Summary

Kitchen Sink Summary - 1

Whipple's:
- Classic: arthralgia, diarrhea, weight loss
- Dx with duodenal bx (PAS+, foamy macrophages)
- Or PCR of tissue (heart valve for endocarditis)

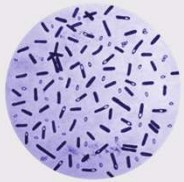
Acute Rheumatic Fever:
- Kids / young adults with migratory polyarthritis, carditis, chorea, subcutaneous nodules, erythema marginatum following GAS pharyngitis
- Monthly IM penicillin prophylaxis for 10 years or to age 40 if carditis + residual valvular disease

SEE ENLARGED SLIDE FOLLOWING ENTIRE PRESENTATION

https://www.cdc.gov/groupastrep/diseases-public/rheumatic-fever.html

Kitchen Sink Summary - 2

Botulism:
- Due to C. botulinum toxin
- Food; infant; wound (black-tar heroin); iatrogenic
- Descending flaccid paralysis (starts with cranial nerves)
- Antitoxin / supportive care



Asplenia:
- Increased risk of infection with encapsulated organisms
- If prompt says asplenia, think…
  - *S. pneumoniae*
  - *N. meningitidis*
  - *H. Influenzae type B*
  - *Capnocytophaga*
  - *Babesia*
  - *Salmonella typhi*
- Prevent infection with vaccines and PCN prophylaxis (< 5 yrs old; recent splenectomy; prior episodes of sepsis)

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52 Kitchen Sink: Syndromes Not Covered Elsewhere

Speaker: Stacey Rose, MD

Kitchen Sink Summary - 3

Staphylococcal toxic shock syndrome

- Menstrual and non-menstrual risk factors
- **Clinical criteria**: fever, rash (erythroderma), desquamation after 1-2 weeks; hypotension; multi-system involvement
- **Treatment**: anti-staphylococcal antibiotic plus anti-toxin antibiotic (clindamycin or linezolid) plus source control

Acalculous cholecystitis

- **Risk factors**: surgery, trauma, TPN, burns, ischemia, infections
- Suspect in critically ill patient with unexplained fever / leukocytosis / elevated LFT's; confirm with ultrasound or HIDA
- **Treatment**: antibiotics + laparoscopic cholecystectomy or percutaneous cholecystostomy (if poor surgical candidate)

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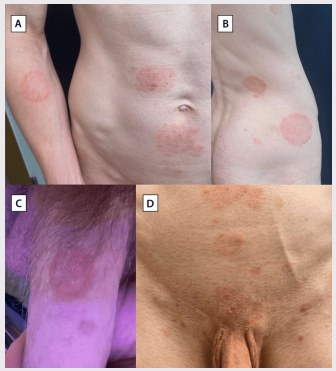
Kitchen Sink Summary - 4



Trichophyton mentagrophytes genotype VII (TMVII):

- Emerging sexually transmitted fungal infection
- Suspect in MSM with painful, itchy "ringworm" lesions in genital / perianal / perioral areas (though can be subcutaneous / pustular with lymphadenopathy)
- Treat with oral terbinafine for ~3 months

SEE ENLARGED SLIDE FOLLOWING ENTIRE PRESENTATION

Questions?

Stacey Rose, MD, FACP, FIDSA
srrose@bcm.edu

52 Kitchen Sink: Syndromes Not Covered Elsewhere
Speaker: Stacey Rose, MD