Infectious colitis

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Presentation outline

- Introduction
- Definitions
- Aetiology and epidemiology
- Pathogenesis
- Histopathology
- Clinical presentation
- Evaluation/ diagnosis
- Treatment/ management

Introduction

- Significant global health burden. Highest morbidity and mortality in children < 5 years.
- Estimated 2 to 4 billon episodes of infectious diarrhoea annually.
- Characterised by pathogen-induced inflammation of colon.
- No gender predilection. Male & females affected equally.
- Severity, aetiology and presentation vary drastically by age, geography and socioeconomic status.
- Stool culture helps diagnose less than 50% of patients. Endoscopic examinations usually reveal nonspecific pathological changes.

*Kosek M, Bern C, Guerrant RL (2003). "The global burden of diarrhoeal disease, as estimated from studies published between 1992 and 2000". Bull World Health Organ. **81** (3): 197–204*

Definitions

- Infectious colitis is diagnosed in someone with diarrhoea and one or more of the following:

➢ Fever and/or dysentery.

➢ Stools containing inflammatory markers such as leukocytes, lactoferrin, or calprotectin.

➢ Positive stool culture for an invasive or inflammatory bacterial enteropathogen or colonic inflammation by endoscopy.

- Recent diagnostics.

➢ Multiplex polymerase chain reaction (PCR) followed by guided culture on PCR-positive pathogens can often confirm active infection.

➢ Standard culture methods provide isolates for antibiotic susceptibility testing, subtyping and Whole Genome Sequencing.

Iqbal, T., & DuPont, H. L. (2020). Approach to the patient with infectious colitis: clinical features, work-up and treatment. *Current Opinion in Gastroenterology*, *37*(1), 66–75.

Aetiology/ classification

1. Bacterial infection: C. jejuni, Salmonella, Shigella, Escherichia coli (enterotoxigenic, enteropathogenic, enterohemorrhagic, enteroinvasive, enteroaggregative), Yersinia enterocolitica, **C. difficile** and **Mycobacterium tuberculosis.**

2. Viral infection: Norovirus, Rotavirus, Adenovirus, **Cytomegalovirus(CMV).**

3. Parasitic infection: **Entamoeba histolytica (amoebic colitis).**

4. Sexually transmitted diseases: Neisseria gonorrhoeae, Chlamydia trachomatis (lymphogranuloma venereum), Herpes simplex 1 and 2, and Treponema pallidum (syphilis).

Jewkes J, Larson HE, Price AB, Sanderson PJ, Davies HA. Aetiology of acute diarrhoea in adults. Gut. 1981 May;22(5):388-92.

Infectious mimics of IBD

IBD mimic	Cause(s)	Populations at risk	Diagnostic clues
Bacterial enteropathogens	*Salmonella*, *Shigella*, *Campylobacter*, *Yersinia*, *E. coli*, *Aeromonas*	• Infections are often food- or water-borne	• Positive cultures/PCR
Mycobacterial infections	*M. tuberculosis*, *Mycobacterium avium-intercellulare* complex	• Tuberculous colitis occurs regardless of immune status • Non-tuberculous colitis affects immunodeficient patients	• Positive cultures/PCR • Acid-fast stains have low sensitivity in paraffin-embedded tissue sections
Sexually transmitted proctocolitis	Syphilis (*T. pallidum*), lymphogranuloma venereum (LGV, *C. trachomatis* serotypes L1, L2, L3)	• HIV-infected men who have sex with men	• Positive serologic studies (syphilis) • Positive culture/PCR (LGV)
Amoebiasis	*Entamoeba histolytica*	• All ages affected • Worldwide distribution	• 25–40 nm, PAS + trophozoites containing erythrocytes within adherent exudate • Positive *E. histolytica* serology
Cord colitis	*Bradyrhizobium enterica* (presumably)	• Cord blood hematopoetic stem cell transplant recipients	• Culture-negative, antibiotic-responsive diarrhoea

Prevalence in SSA

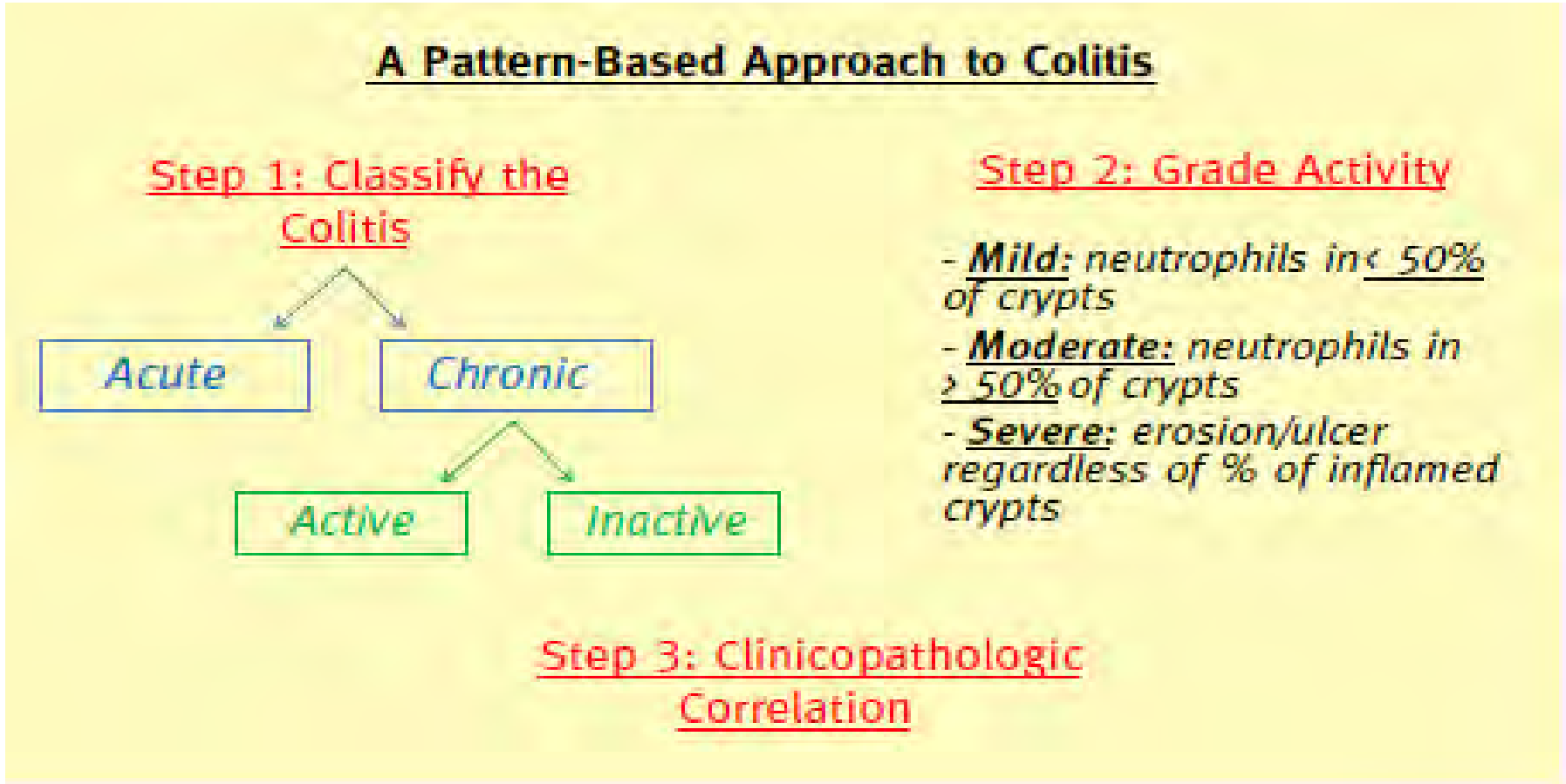
- Systematic review of published articles from the SSA region to determine prevalence and types of diarrhoeal pathogens.
- Based on a search of databases, including EBSCO host, PubMed,Scopus, Science Direct, Google done between September2009 and December 2010.
- Data summarized from 27 studies, with pooled data analysed and reported.
- Pathogens isolated between 26.8-65.6% of cases, overall isolation rate of 55.7%.
- Overall isolation 8% to 99%, heterogeneity testing suggest differences between age groups.
- Mixed E. coli spp(29.95%), Cryptosporidium(21.52%), Cyclospora(18%), Entamoeba(13.8%), Shigella spp(10.49%), Salmonella spp(8.36%), and Campylobacter spp. (8.33%)

Prevalence of gastrointestinal pathogens in Sub-Saharan Africa: systematic review and meta-analysis

Stephanie M. Fletcher,[1] Damien Stark,[1,2] John Ellis[1]

[1]iThree Institute and Department of Medical and Molecular Biosciences, University of Technology, Sydney; [2]Division of Microbiology, St. Vincent's Hospital, Sydney, Australia

Approach to colitis



Adapted from Arnold, Lam-Himlin, and Montgomery. Atlas of Gastrointestinal Pathology. A Pattern Based Approach to Non-Neoplastic Biopsies. (2015) Philadelphia, USA. Wolters Kluwer.

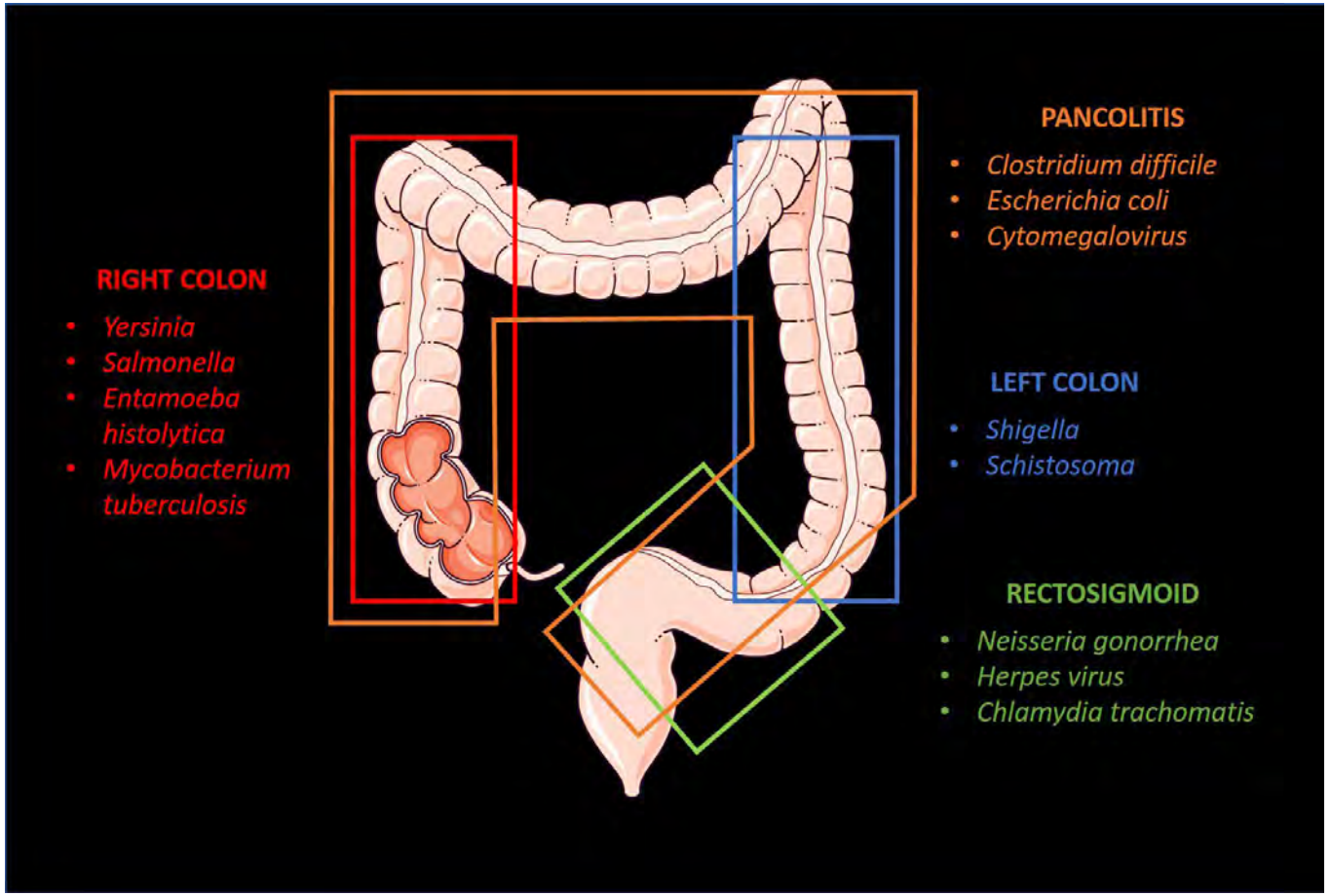
Pattern- based approach

Pattern of injury	Histologic features	Aetiologic considerations
Focal active colitis (FAC)	• Isolated foci of neutrophilic crypt injury: cryptitis ± crypt abscesses • No features of chronicity	• Acute self-limited colitis (infection, medication) • Chemical/irritants • Bowel prep artifact
Acute colitis	• More severe/diffuse than FAC • Cryptitis, crypt abscesses, erosions, ulcerations • No features of chronicity	• Acute viral/bacterial infections • Medications (NSAIDs, Kayexalate, sevelamer, ipilimumab) • Emerging/partially treated IBD
Chronic colitis	• Wide spectrum of morphologic changes: increased lamina propria chronic inflammation, Paneth cell metaplasia, pyloric gland metaplasia, and architectural distortion (a villonodular surface, abnormal crypt configuration, crypt dropout, crypt shortfall, and basal lymphoplasmacytosis)	• IBD • Diverticular disease • Diversion-associated colitis • Medications (NSAIDs, ipilimumab, resins) • Infections • Others (e.g. vasculitis, autoimmune disease, radiation colitis, cord colitis)

Acute self limited colitis

- Defined as a transient, most likely infectious, disorder of the colon.
- Usually resolves completely within 2 - 4 weeks.
- Not always acute or self limited.
- Most commonly bacterial- *Campylobacter jejuni*, *Salmonella*, *Shigella* species, *E. coli* and *Yersinia enterocolitica*.

Differentials at colonoscopy



Infectious colitis. Schematic representation of the distribution of the abnormal part of the colon produced by different offending organisms

Pathogenesis

- Most commonly associated with bacterial enterocolitis
- Bacterial virulence factors include:
  - Adherence to epithelial cells
  - Enterotoxins
  - Invasion factors
  - Cytotoxicity
- Adherence:
  - Via fimbriae or pili
  - Process of adherence destroys the microvilli brush border.
- Enterotoxins:
  - Toxins bind to cell membrane, enter cells, activate massive electrolyte secretion (cholera toxin, *E. coli* heat labile and heat stable toxins
  - No white blood cells in stool
- Invasion factors:
  - *C. jejuni*, *Salmonella* and *Shigella* species, *E. coli* and *Y. enterocolitica* are organisms that invade the tissue and cause epithelial injury
  - Invade via microbe simulated endocytosis, then cause cell lysis and cell to cell spread
  - Cytotoxins work by inhibiting protein synthesis, disrupting tight junctions and depleting adenosine triphosphate (ATP) within cells
  - Endothelial injury results in the activation of the coagulation cascade that ultimately leads to an ischemic colitis pattern of tissue injury
- Cytotoxicity:
  - Shiga toxin, enterohemorrhagic *E. coli*

Nasef NA, Mehta S. Role of Inflammation in Pathophysiology of Colonic Disease: An Update. Int J Mol Sci. 2020 Jul 3;21(13):4748. doi: 10.3390/ijms21134748. PMID: 32635383; PMCID: PMC7370289.

Clinical features

- Abdominal pain, watery or bloody diarrhoea.
- Symptoms within hours (including explosive diarrhoea)
- Sudden onset, early fever, with numerous (> 6) bowel movements daily
- Complications -dehydration, sepsis, perforation occur secondary to potential massive fluid loss and loss of mucosal barrier.
- Inflammatory process usually resolves completely within 2 - 4 weeks

Management

Diagnosis

- Stool MC&S.
- Peripheral blood and faecal leukocyte count
- Colonoscopy with mucosal biopsy
- Imaging studies show nonspecific thickening of the bowel wall

Prognosis

- Depends on specific underlying pathogen.
- Immune status of host.
- Generally good with appropriate therapy.
- Commonly self limited disease (2 - 4 weeks)

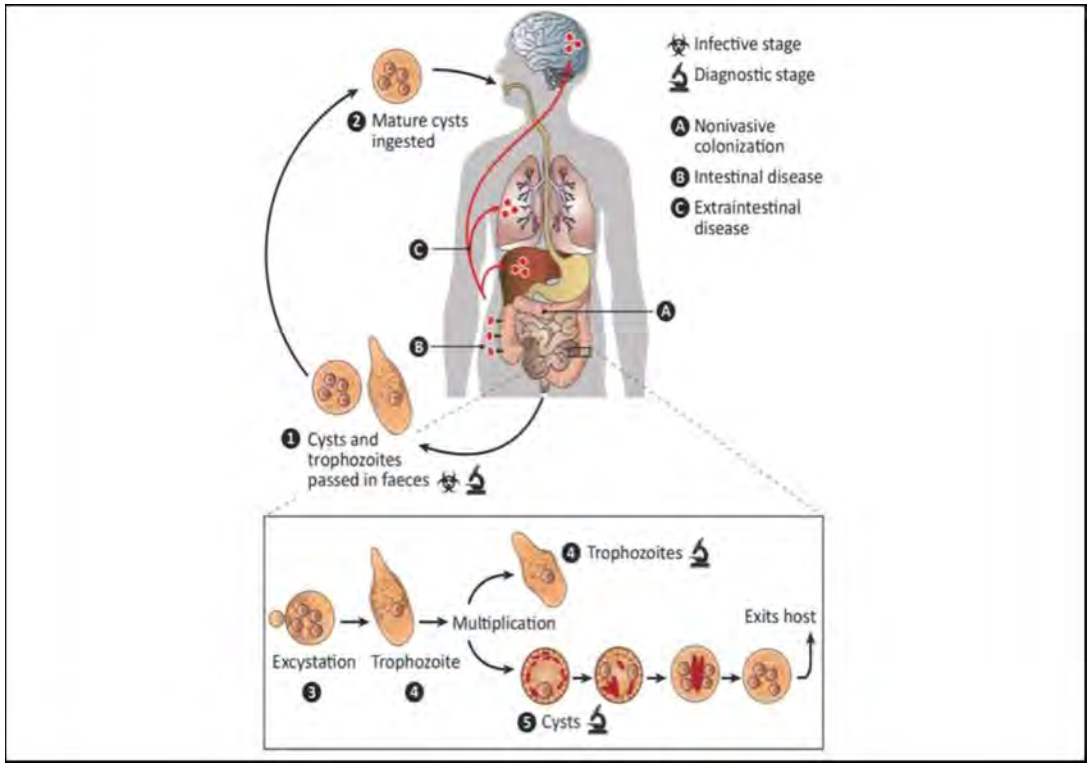
Amoebic colitis

- *E. histolytica*- only species of protozoal genus *Entamoeba* known to cause disease.
- Estimated 50 million people infected annually. 55 000 deaths per year.
- 90% may remain asymptomatic. 10%- amoebic colitis/ liver abscess.
- Majority of infections occur in low income countries (LICs), in areas of contaminated water supply and poor sanitation.
- Mimics IBD and in non-endemic regions particularly, misdiagnosis may lead to significant morbidity and mortality.

Blessmann J, Ali IKM, Nu PAT, *et al*. Longitudinal study of intestinal Entamoeba histolytica infections in asymptomatic adult carriers. *J Clin Microbiol* 2003;41:4745–50.

Amoebic colitis

Life cycle



Risk factors

- Abdominal surgery.
- After use of contaminated colonic irrigation equipment.
- Sexual transmission.
- Males with high alcohol consumption.- Liver abscess
- Immunosuppressive medication- severe complications

Stanley SL Jr. Amoebiasis. *The Lancet* 2003;361:1025–34.

Clinical presentation- Amoebic colitis and ALA

Symptoms

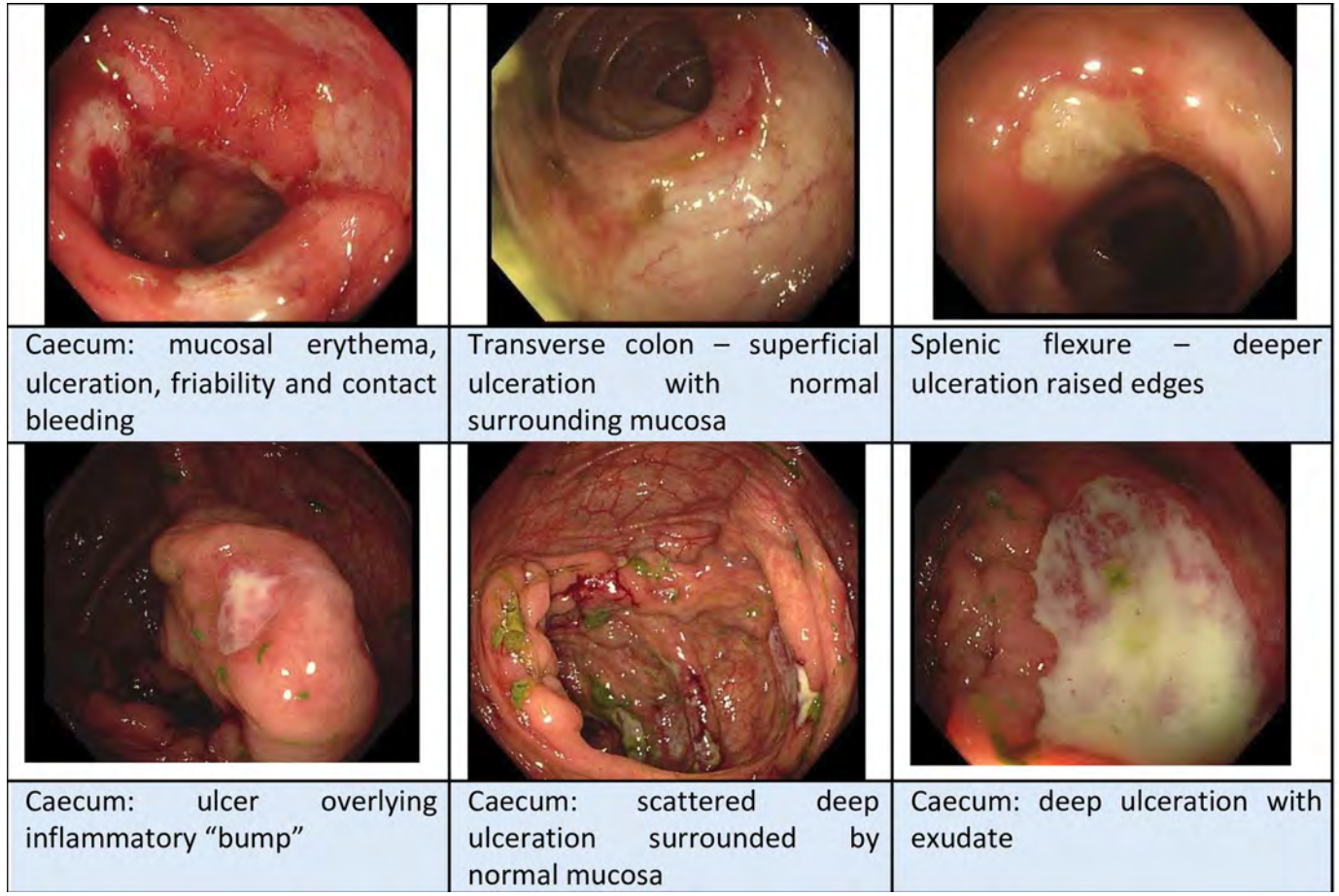
- Asymptomatic -90%
- Abdominal pain- several weeks
- Weight loss
- Watery, mucoid or bloody diarrhoea.
- Median duration of symptoms 14 days to 6 months.
- ALA- well circumscribed abscess with a rim of connective tissue containing anchovy sauce pus.
- RUQ pain with fever.
- Cough and right sided pleural pain- diaphragmatic involvement

Signs

- Abdominal tenderness.
- Distension- Fulminant amoebic colitis with toxic mega colon & perforation.
- Amoeboma-(mimic phlegmon in CD).
- Atypical colonic presentations

❖ Amoebic appendicitis
❖ Large bowel obstruction
❖ Peri anal ulcers/ fistula
❖ Enteric fistula
❖ Gall bladder infiltration

Endoscopic appearances of Amoebic colitis



Caecum: mucosal erythema, ulceration, friability and contact bleeding	Transverse colon – superficial ulceration with normal surrounding mucosa	Splenic flexure – deeper ulceration raised edges
Caecum: ulcer overlying inflammatory "bump"	Caecum: scattered deep ulceration surrounded by normal mucosa	Caecum: deep ulceration with exudate

Cooney J, et al. Frontline Gastroenterology 2025;**16**:37–50. doi:10.1136/flgastro-2023-102554

Fulminant Amoebic colitis

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AOSIS

Page 1 of 4 Case Report

Fulminant amoebic colitis in the era of computed tomography scan: A case report and review of the literature

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Amoebic colitis, caused by ingestion of water or food contaminated with the protozoan Entamoeba histolytica, can progress to a fulminant colitis. Computed tomography (CT) findings reported in the literature on this type of colitis are sparse. We present a 59-year-old male patient with a one-week history of progressive abdominal pain, abdominal distension and associated watery and bloody diarrhoea. A CT scan revealed deep ulcerations with submucosal and intramural tracking of contrast. Colonoscopy and biopsy confirmed a diagnosis of Amoebic colitis. The patient required a laparotomy and demised. Deep ulcerations with submucosal and intramural tracking of contrast on CT are diagnostic of fulminant amoebic colitis. Although not demonstrated at CT in this case, discontinuous bowel necrosis, omental wrapping (seen at laparotomy in our case) and neovascularisation of the bowel wall may be other features to look out for.

Amoebic colitis- FAC

Contrast CT scan



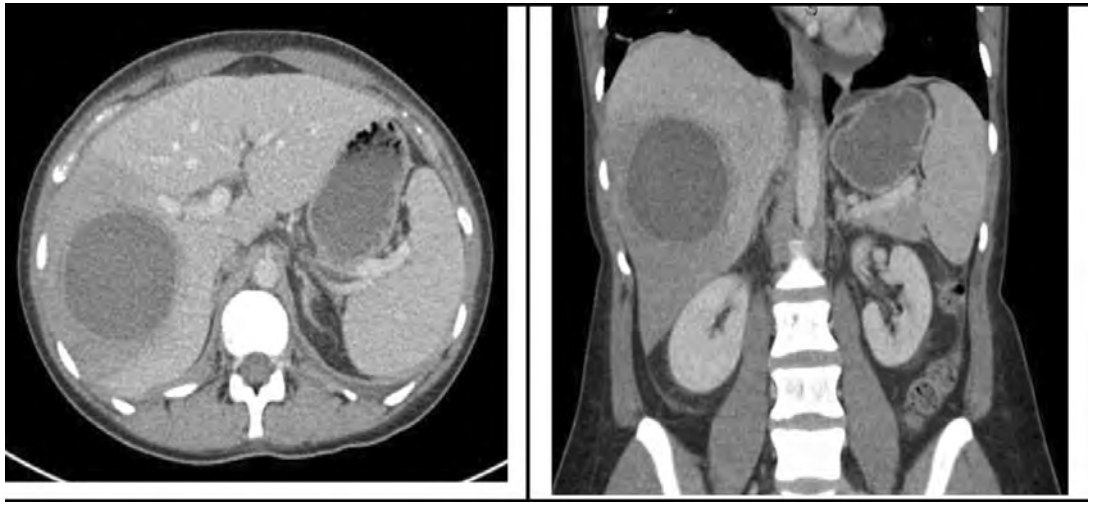
Pathology



Mewa Kinoo S, Ramkelawon VV, Maharajh J, Singh B. Fulminant amoebic colitis in the era of computed tomography scan: A case report and review of the literature. S Afr J Rad. 2018;22(1), a1354

Amoebic colitis- imaging

CT findings



US Liver



Amoebic colitis diagnosis

1. Light microscopy(Sn <60 sp-95)- cyst visualisation. Trophozoites in loose “hot” stools.

- ✓ Stains with iron heamatoxylin – better visualisation of morphology.
- ✓ Iso enzyme analysis by electrophoresis differentiates species(E. histolytica vs E. dispar)

2. Antigen detection(ELISA)- stool/ liver aspirates.

Sn- upto 88, sp >80.

- ✓ No need for fresh stool.
- ✓ Tests for E. histolytica specific antigen.

3. Antibody detection tests(serum)

- ✓ Sen 65-92, Sp >90
- ✓ Intestinal & Extraintestinal infection

4. PCR- conventional, nested, multiplex- 100% sensitivity and specificity. Stool samples.

- ✓ Gold standard- Sn & sp 100.
- ✓ Can differentiate species.
- ✓ Not widely available.

5. Histopathology

- ✓ Operator dependent
- ✓ Cheap
- ✓ Routine practice
- ✓ Biopsies and surgical resections

Shirley D-AT, Farr L, Watanabe K, *et al*. A Review of the Global Burden, New Diagnostics, and Current Therapeutics for Amebiasis. *Open Forum Infect Dis* 2018;5:ofy161.

Treatment

Tissue and luminal amoebicides

1. Amoebic colitis-

- Metronidazole 800 mg three times per day PO for 5 days OR
- Tinidazole 2 g once daily for 3 days followed by a luminal amoebicide
- Paromomycin 25–35 mg/kg/day in three divided doses PO for 7 days OR
- Diloxanide furoate for 10 days.

2. Amoebic liver abscess

- Metronidazole 800 mg three times per day PO for 5-10 days OR
- Tinidazole 2 g once daily for 5 days followed by a luminal amoebicide
- Paromomycin 25–35 mg/kg/day in three divided doses PO for 7 days OR
- Diloxanide furoate for 10 days.

Drainage

- Imminent rupture.
- No clinical response after three or 4 days of medical treatment.
- Abscess diameter >10 cm
- ALA in left lobe of liver

GASTROINTESTINAL TUBERCULOSIS

- ETB accounts for approximately 20% of patients with TB.
- 7%–10% have abdominal TB.
- The clinical, morphological, and histological features of gastrointestinal (GI) TB and CD are similar.
- TB diagnostic criteria. Based on the presence of any of the following three criteria: Sensitivity 30-50 %

➢ Culture of tissue (colonic biopsy, lymph nodes) resulting in growth of *Mycobacterium tuberculosis*

➢ Histological demonstration of typical acid-fast bacilli (AFB)

➢ Histological evidence of necrotizing granuloma.

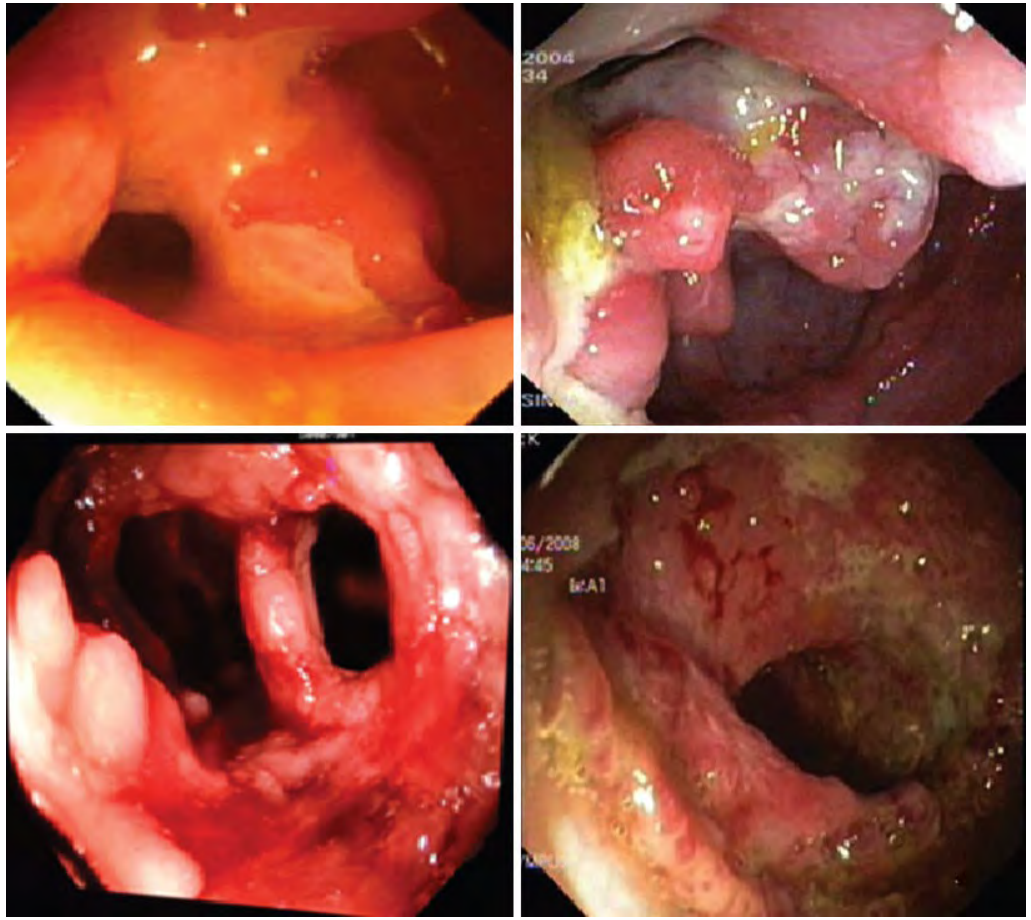
GI TB-Sites of involvement

- Striking tendency for intestinal TB to affect the ileum and ileocecal (IC) area preferentially.
- Colon-frequency of involvement decreases segmentally from the ascending colon to the rectum.
- Rectum and anal canal is uncommon in patients with GI TB.
- Involvement of lymph nodes, peritoneum, and solid viscera either alone or in combination with GIT.
- Appearance of intestinal TB

➢ Ulcerative lesions (60%)- multiple superficial lesions confined largely to the epithelial surface
➢ Hypertrophic lesions (10%)-scarring, fibrosis, and heaped-up mass lesions that can mimic carcinoma.
➢ Ulcerohypertrophic lesions (30%)- mucosal ulcerations are combined with healing and scar formation.

- Histologically, the distinguishing lesion is the granuloma, which is seen in 50%-80%

Endoscopic lesions in intestinal TB



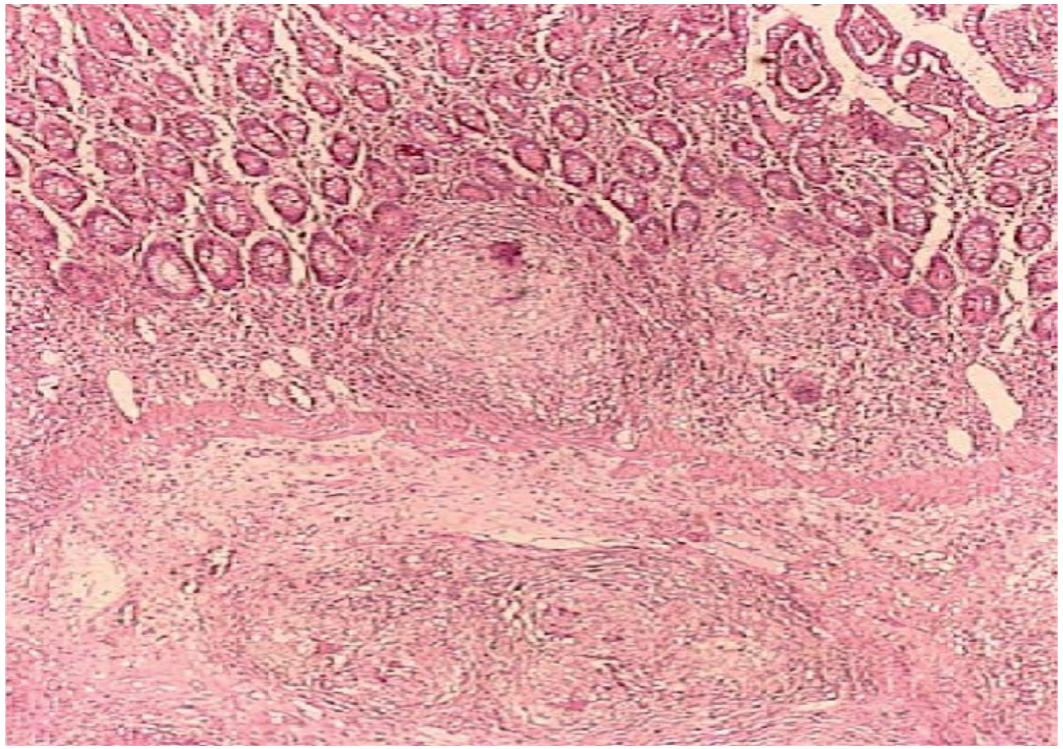
- Ulcers over the ileocecal valve
- Multiple nodules and deep ulcers (with neoplasm-like appearance) in cecum with patulous ileocecal valve
- Fixed patulous ileocecal valve with nodules in cecum and ascending colon
- Circumferential ulcerative lesion

GI TB

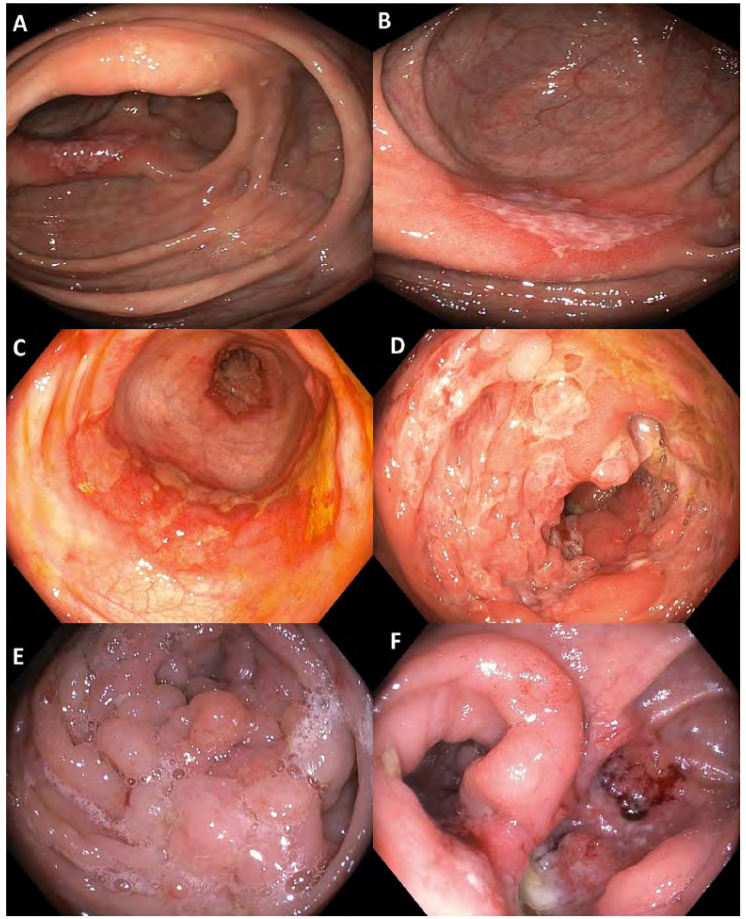
Diagnosis

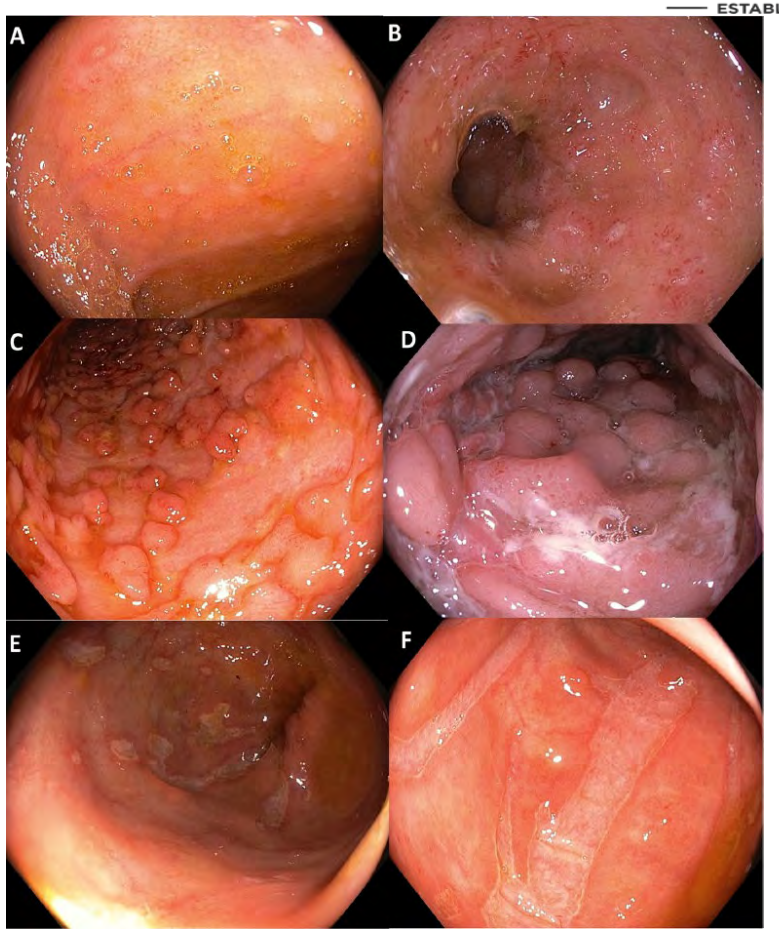
- Colonoscopy with biopsy specimens.
- Histopathology, acid-fast bacilli staining/PCR, and culture (with drug sensitivities).
- TB bacilli are detected with an acid-fast stain in approximately 20% of mucosal samples.
- PCR is more sensitive (65%) and can be performed on tissue or stool.

TB histology



GI TB vs Crohns





Moka P, Ahuja V, Makharia GK. Endoscopic features of gastrointestinal tuberculosis and crohn's disease. J Dig Endosc2017;8:1-11.

GI TB vs Crohns

	Favors Gastrointestinal Tuberculosis	Do not favor a particular diagnosis	Favors Crohn's Disease
Clinical Presentation	Shorter History (< 6 months) **Pulmonary symptoms (Cough, hemoptysis)** **Peritoneal involvement (Ascites)** Abdominal lump	Abdominal Pain Intestinal Obstruction	Chronic diarrhea Hematochezia Perianal symptoms Extraintestinal manifestations
Imaging findings	**Necrotic lymph node** Patulous ileo-cecal valve Short strictures Asymmetric mural thickening Ascites, peritoneal or omental involvement Pulmonary lesions	Mural thickening Strictures Lymph nodes	Mural stratification Fibrofatty proliferation Comb sign Skip lesions Long segment involvement (> 3cm)
Colonoscopic findings	Patulous Ileo-cecal valve Circumferential or transverse ulcers	Ulcers Pseudopolyps Strictures Ulcerated narrowing	Aphthous ulcers Longitudinal ulcers Serpiginous ulcers Cobblestoning Skip lesions
Histopathology	Large granuloma **Confluent granuloma** **Caseating granuloma** **Ulcer lined by histiocytes**	Granuloma Chronic inflammation including distorted architecture Paneth cell metaplasia	Focally enhanced colitis Microgranuloma Sparse granuloma

CLOSTRIDOIDES DIFFICILE

- *Clostridium difficile* has been reclassified and given a new name: *Clostridioides difficile.*
- *Clostridioides difficile (C. difficile)* is a gram positive, anaerobic, spore-forming rod.
- *C. difficile* infection (CDI) is implicated in 10–25% of cases of antibiotic-associated diarrhoea.
- *C. difficile* colonization refers to the detection of the bacteria in an asymptomatic person**.**
- Wide spread antimicrobial use with emergence of increasingly virulent strains.(NAP-1).
- Heightened morbidity and mortality posing a public health challenge.

Czepiel J, Dróżdż M, Pituch H, Kuijper EJ, Perucki W, Mielimonka A, Goldman S, Wultańska D, Garlicki A, Biesiada G. Clostridium difficile infection: review. Eur J Clin Microbiol Infect Dis 2019; 38: 1211-1221 [PMID: 30945014 DOI: 10.1007/s10096-019-03539-6]

Risk factors for C diff

- **Old age (> 65 years)**- Reduction of bifidobacteria.
- **Antibiotic use** (Quinolones, cephalosporins, penicillins, and clindamycin)
- Prolonged hospital stay- High risk for colonisation.
- Immunosuppression (HIV, steroids, chemotherapy, malignancies, CKD, organ transplants)
- Colonic disease (IBD)- stimulate bacterial colonisation.
- PPI use-reduce gastric acid bactericidal effect.

Smits WK, Lyras D, Lacy DB, Wilcox MH, Kuijper EJ. Clostridium difficile infection. Nat Rev Dis Primers 2016; 2: 16020 [PMID: 27158839 DOI: 10.1038/nrdp.2016.20]

Incidence and Prevalence

The *Clostridium difficile* problem: A South African tertiary institution's prospective perspective

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Study highlight

- **Background and objectives.** To report incidence of *Clostridium difficile*-associated disease (CDAD) in a tertiary care hospital and identify risk factors, assess patient outcomes and determine the impact of the hypervirulent strain of the organism North American pulsed-field type 1 (NAP1).

- **Methods.** Adults patients with diarrhoea over 15 months prospectively evaluated for CDAD using stool toxin enzyme immunoassay (EIA). Positive specimens evaluated by PCR. Patient demographics, laboratory parameters and outcomes analysed.

- **Results.** CDAD diagnosed in 59 (9.2%) of 643 patients (median age 39 years, IQR 30 - 55). Recent antibiotic exposure reported in 39 (66%), 27 (46%) hospitalised within 3 months, and 14 (24%) with concomitant IBD. 19 (32%) had CA-CDAD.

Annual incidence HA-CDAD was 8.7 cases/10 000 hospitalisations.2 cases of the hypervirulent strain NAP1 were identified. 7 (12%) patients underwent colectomy.

- **Conclusion.** The incidence of HA-CDAD was far lower than that reported in the West. Antibiotic use was a major risk factor for HA-CDAD while Concurrent IBD was a predictor of CA-CDAD.

Rajabally, N, Pentecost, M, Pretorius, G, Whitelaw, A, Mendelson, M, & Watermeyer, G. (2013). The Clostridium difficile problem: A South African tertiary institution's prospective perspective. *SAMJ: South African Medical Journal*, *103*(3), 168-172. Retrieved October 13, 2026

Pathogenesis

- Toxin A and B are cytotoxic and lead to cell damage by disrupting the cytoskeletal structure of the cell and increasing the release of cytokines, leading to severe inflammation.
- Toxin B is more potent than toxin A.
- Some strains produce only toxin B (toxin A-/B+).
- A hypervirulent strain (*C. difficile* NAP1/BI/027- also called "epidemic strain") is associated with more severe disease, lower cure rates, and higher recurrence rates.
- Mutation in a bacterial gene (*txcD),* which leads to the production of larger quantities of toxins A and B- produces an additional toxin called "binary toxin"

Endoscopy

- Flexible Sigmoidoscopy with biopsy sufficient for diagnosis.

- Classic appearance of pseudomembranous colitis is diagnostic of CDI.

- Histology shows varying degrees of mucosal inflammation.
- Classic "summit" or "volcano" lesion- ulceration of the colonic mucosa with "eruption" of pseudo membranes containing inflammatory cells, fibrin and debris.
- CDI in the setting of IBD

➢ Difficult to differentiate CDI from an IBD flare based on the endoscopic appearance.

➢ Most common endoscopic findings of CDI in a patient with IBD are nonspecific oedema and friability.

➢ Pseudomembranes are uncommon.

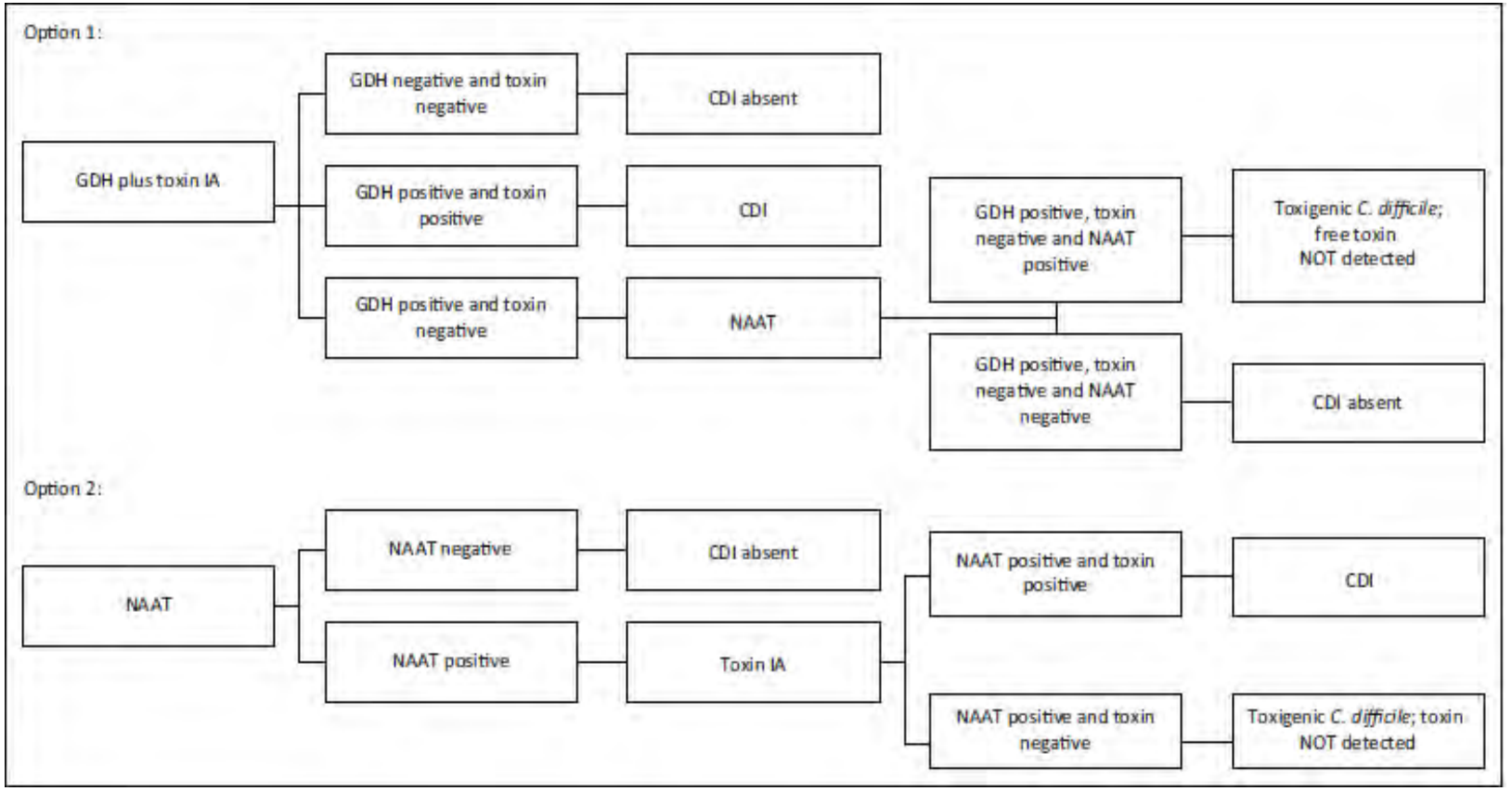
Diagnosis

Test	Characteristics
Polymerase chain reaction (PCR) for *C. difficile* toxins A and B	• High sensitivity and specificity

Enzyme immunoassay (EIA) for *C. difficile* glutamate dehydrogenase (GDH)	• High sensitivity (> 90%)
	  • More sensitive than EIA for toxin A/B
	• High negative predictive value (95-100%)
	  • Useful as an initial screening test
	• Low specificity

Enzyme immunoassay (EIA) for *C. difficile* toxins A and B	• Lower sensitivity (75-95%) and specificity (83-93%) compared to PCR • **Not** recommended as the only test for *C. difficile*

CDI Testing algorithm



General precautions

- Stop the offending antibiotic as soon as possible.
- Patient is receiving PPI therapy, reassess the indication for PPI.
- Deprescribe PPI therapy during treatment for *C. difficile* if the indication for PPI therapy not in category of prolonged use.
- PPI may allow *C. difficile* to remain in the vegetative state-PPI use during treatment for *C. difficile* associated with 42% increased risk of recurrence.
- Confirmed *C. difficile* infection, isolate in a dedicated room with contact precautions to decrease the risk of transmission to other patients.

Treatment of CDI

1. First episode, mild to moderate infection

- Vancomycin PO 125 mg QID x 10 days *or* Fidaxomicin 200 mg BID x 10 days
- Alternative if above not available: Metronidazole 500 mg PO TID for 10 days.

2. First episode, severe infection. WBC >15,000/mm3 or creatinine >1.5 mg/dL.

- Vancomycin PO 125 mg QID x 10 days *or* Fidaxomicin 200 mg BID x 10 days.

3. First episode, severe and fulminant disease: hypotension, shock, ileus, or megacolon (IDSA)or altered mental status, WBC ≥ 35,000/mm3 or < 2,000/mm3, lactate > 2.2 mmol/L, end organ failure, admission to ICU.

- Vancomycin 500 mg PO QID and metronidazole 500 mg IV every 8 hours.
- If ileus or abdominal distension, add vancomycin enema 500 mg in 100 ml of normal saline given via Foley catheter. Repeat every 6 hours.
- Severe and fulminant disease not responding to antibiotics, fecal microbiota transplantation (FMT)should be considered especially if the patient is not a candidate for surgery.

Other considerations

1. *C. difficile* and IBD

- Patients is on immunosuppressive medications, continue medications while on antibiotics.
- No dose increment or new immunosuppressive regimens until the infection is treated.
- Vancomycin PO 125 mg QID is the recommended Treatment.

2. Surgery consideration

- WBC >50,000/mm3, obtain surgical consult for consideration of total colectomy.
- Subtotal colectomy with preservation of the rectum is recommended.
- Alternative surgical approach is performance of a loop ileostomy, colonic lavage, and vancomycin flushed through the ileostomy into the colon.

Recurrent CDI

- Recurrent symptoms of CDI within 8 weeks after the onset of symptoms of the previous CDI episode.

- Recurrence more commonly results from relapse of CDI with same strain, rather than new strain.

- Treatment regimen

➢ Vancomycin pulse then tapered therapy

➢ Vancomycin PO 125 mg QID x 10 days

➢ Rifaximin "chaser" regimen: Vancomycin125 mg PO q.i.d. for 14 days, followed by rifaximin 400 mg b.i.d. for 14 days.

➢ Bezlotoxumab is a monoclonal antibody against toxin B. Shown to lower the recurrence of CDI.

➢ FDA approved as adjunctive therapy for patients on antibiotics for CDI, and who are at high risk for recurrence.

➢ FMT- Recommended for CDI recurrence(third and subsequent recurrence)

Cytomegalovirus colitis

- Double stranded DNA virus and a member of human herpes virus family(herpes virus type 5).

- 3 patterns of CMV infection:

➢Latent infection: Most common, immunocompetent patients

➢Mononucleosis-like syndrome: Immunocompetent patients

➢Tissue invasive disease: Immunocompromised patients

- Tissue invasive disease- Gastrointestinal tract is most commonly involved (30% of tissue invasive cases).

- Most common GI sites- colon and esophagus.

Rafailidis PI, Mourtzoukou EG, Varbobitis IC, Falagas ME. Severe cytomegalovirus infection in apparently immunocompetent patients: a systematic review. Virol J. 2008 Mar 27;5:47. doi: 10.1186/1743-422X-5-47. PMID: 18371229; PMCID: PMC2289809.

CMV terminology

- CMV infection : CMV antigens or antibodies in blood.
- CMV disease: Clinical symptoms and end organ damage.
- CMV colitis
  - Identification of characteristic intranuclear / intracytoplasmic inclusions on H&E sections
  - Identification of CMV specific antigens by immunohistochemistry (IHC) and clinical symptoms

Epidemiology

- 50 - 100% humans worldwide depending on age and race of the population tested- Infects over half of adults by age 40.
- CMV seroprevalence in the United States is 42 - 93%.
- Immunocompetent patients- Mean age is 64 - 75 years.
- Liver transplant patients- Cumulative incidence is 4.9%.
- Allogenic hematopoietic stem cell transplant patients- Incidence is 15 - 25%.
- Inflammatory bowel disease
  - Incidence is 1.5 - 4.5%
  - Ulcerative colitis > Crohn's disease.

1.(CDC: About Cytomegalovirus (CMV) [Accessed 11 November 2021])
2.Bontà J, Zeitz J, Frei P, Biedermann L, Sulz MC, Vavricka SR, Scharl S, Fried M, Rogler G, Scharl M. Cytomegalovirus disease in inflammatory bowel disease: epidemiology and disease characteristics in a large single-centre experience. Eur J Gastroenterol Hepatol. 2016 Nov;28(11):1329-34.

Risk factors

Immunocompromised

- AIDS
- Organ transplantation
- Hematologic malignancy
- Cancer therapy
- Severe UC colitis(High dose steroids)
- RBC transfusion within 1 month of colitis diagnosis

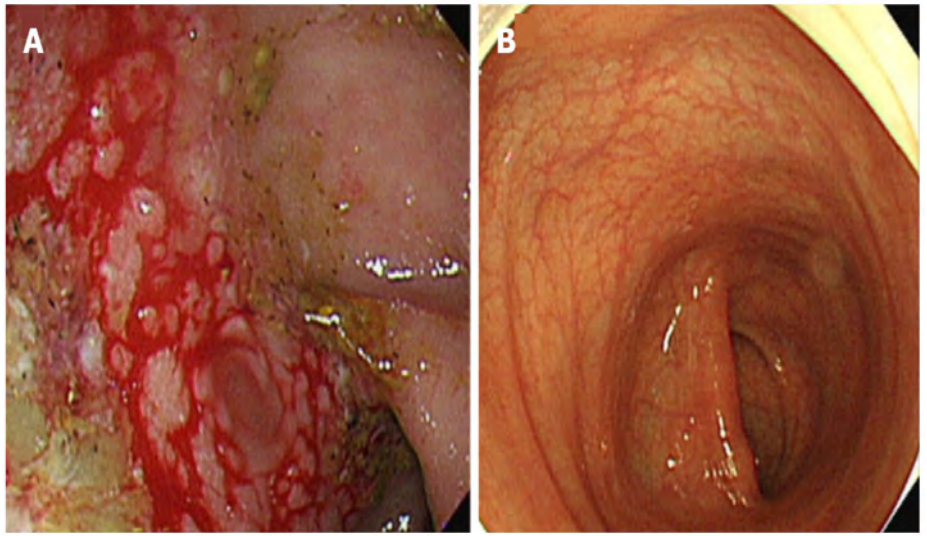
Immunocompetent

- Renal disease
- Haemodialysis
- Neurological disease
- Rheumatic disease
- Exposure to antibiotics and anticids
- Corticosteroid

Ko JH, Peck KR, Lee WJ, Lee JY, Cho SY, Ha YE, Kang CI, Chung DR, Kim YH, Lee NY, Kim KM, Song JH. Clinical presentation and risk factors for cytomegalovirus colitis in immunocompetent adult patients. Clin Infect Dis. 2015 Mar 15;60(6):e20-6. doi: 10.1093/cid/ciu969. Epub 2014 Dec 1. PMID: 25452594.

Endoscopic presentation

- Ulcerations with a well-defined, punched-out appearance.
- Irregular ulceration and a cobblestone-like appearance.
- Friability, loss of vascular patter, mucosal oedema
- Toxic megacolon with pseudomembrane formation.
- Biopsy tissue taken from the base and edge of the ulcers.



Kim BS, Park SY, Kim DH, Kim NI, Yoon JH, Ju JK, Park CH, Kim HS, Choi SK. Cytomegalovirus colitis induced segmental colonic hypoganglionosis in an immunocompetent patient: A case report. *World J Clin Cases* 2021; 9(20): 5631-5636 [PMID: 34307618 DOI: 10.12998/wjcc.v9.i20.5631]

Diagnosis

Serology

- Acute infection
  - CMV IgM antibodies
  - 4 times increase in titer of CMV IgG specific antibodies 2 - 4 weeks apart
- CMV antigenemia
  - Predictor of clinical outcomes
  - Less sensitive for diagnosis of CMV colitis
- Real time polymerase chain reaction (PCR) / CMV DNA quantification
  - Positive in only 50% of patients with biopsy proven CMV colitis / enteritis
- CMV culture
  - High sensitivity and specificity for diagnosis of CMV colitis
  - Takes longer to obtain results (1 - 3 weeks)
  - May delay diagnosis and timely treatment

Histology

- Histology: Gold standard test.
- IHC for CMV- greater sensitivity than hematoxylin & eosin (H&E).
- CMV infected cells larger than normal, containing intranuclear or intracytoplasmic inclusions.
- Owl's eye: Large ovoid or pleomorphic nucleus with basophilic intranuclear inclusions (Cowdry bodies) surrounded by a clear halo

Landry ML, Ferguson D. Comparison of quantitative cytomegalovirus antigenemia assay with culture methods and correlation with clinical disease. J Clin Microbiol. 1993 Nov;31(11):2851-6. doi: 10.1128/jcm.31.11.2851-2856.1993. PMID: 8263166; PMCID: PMC266143.

Management

Treatment

- Intravenous (IV) ganciclovir (5 mg/kg twice daily)
- After 3 - 5 days of IV ganciclovir, oral valganciclovir (900 mg, twice daily)
- Foscarnet in ganciclovir resistant or tolerant cases

Prognostic factors

- Excellent overall prognosis
- Factors associated with poor prognosis and higher mortality (immunocompetent patients)
  - M > F
  - Age > 55 years
  - Patients requiring surgery
  - CMV colitis reactivation with ulcerative colitis.
- Timely diagnosis and treatment greatly improves clinical outcomes

Jones A, McCurdy JD, Loftus EV Jr, Bruining DH, Enders FT, Killian JM, Smyrk TC. Effects of antiviral therapy for patients with inflammatory bowel disease and a positive intestinal biopsy for cytomegalovirus. Clin Gastroenterol Hepatol. 2015 May;13(5):949-55.

Sexually Transmitted Diarrhea Is Exploding in The US. And It's Growing Resistant to Medicine

HEALTH 08 October 2026 By CARLY CASSELLA



Sexually Transmitted Infections

Common aetiology

- HSV 1&2 colitis
- Lympogranuloma venerum(Chlamydia Trachomatis L1, L2 L3)
- Gonorrhea colitis
- Syphilitic colitis

Risk factors

- HIV
- MSM
- Co- infection with other STIs
- Immunosuppression

Clinical presentations

- Proctalgia
- Pruritis
- Constipation
- Mucopurulent or bloody discharge
- Tenesmus
- Abdominal pain
- Fever

- Classic herpetic lesions (oral, genital, anal)
- Groove sign & Anorectal syndrome, strictures, fistulae(chlamydia)
- Disseminated disease(HSV)
- May be complicated by bowel perforation
- Endoscopic findings: ulcers, necrotic mucosa

Diagnosis and Treatment

- **Chlamydia-** NAATs performed on rectal swabs or anogenital lesions- 91 - 100% sensitivity, > 95% specificity. Doxycycline for 7 days OR Azothromycin for 3 days

- **HSV-** HSV culture. HSV nucleic acid testing; high viral load suggests systemic infection.

Acyclovir/ valacyclovir, famcyclovir- Prophylaxis for frequent recurrences

- **Syphilis-** Tissue biopsy or resection of mass-like lesions- *T. pallidum* spirochetes by IHC.
  Nontreponemal assays (serum)
  - Venereal disease research laboratory (VDRL), rapid plasma reagin (RPR)
    - Sensitivity depends upon disease stage (higher in secondary syphilis)
- Treponemal assays (serum): detect antibodies to *T. pallidum*
- Benzathine benzylpenicillin.

Papp JR, Park IU, Fakile Y, Pereira L, Pillay A, Bolan GA. CDC Laboratory Recommendations for Syphilis Testing, United States, 2024. MMWR Recomm Rep. 2024 Feb 8;73(1):1-32. doi: 10.15585/mmwr.rr7301a1. PMID: 38319847; PMCID: PMC10849099.

Take home points

- Inflammatory diarrhoea is commonly infectious.
- Infections mimic other inflammatory bowel pathologies.
- Judicious assessment- clinical, endoscopic, stool analysis & histology is crucial in diagnosis.
- Empiric therapy should be initiated in severe presentations pending confirmatory tests.

References

- Iqbal, T., & DuPont, H. L. (2020). Approach to the patient with infectious colitis: clinical features, work-up and treatment. *Current Opinion in Gastroenterology*, *37*(1), 66–75.

- *S.M. Fletcher et al., 2011 Journal of Public Health in Africa 2011; 2:e30 doi:10.4081/jphia.2011.e30.*

- Golden MP, Vikram HR. Extrapulmonary tuberculosis: An overview. Am Fam Physician 2005;72:1761-8.

- Landry ML, Ferguson D. Comparison of quantitative cytomegalovirus antigenemia assay with culture methods and correlation with clinical disease. J Clin Microbiol. 1993 Nov;31(11):2851-6. doi: 10.1128/jcm.31.11.2851-2856.1993. PMID: 8263166; PMCID: PMC266143.

- Jones A, McCurdy JD, Loftus EV Jr, Bruining DH, Enders FT, Killian JM, Smyrk TC. Effects of antiviral therapy for patients with inflammatory bowel disease and a positive intestinal biopsy for cytomegalovirus. Clin Gastroenterol Hepatol. 2015 May;13(5):949-55.

- Papp JR, Park IU, Fakile Y, Pereira L, Pillay A, Bolan GA. CDC Laboratory Recommendations for Syphilis Testing, United States, 2024. MMWR Recomm Rep. 2024 Feb 8;73(1):1-32. doi: 10.15585/mmwr.rr7301a1. PMID: 38319847; PMCID: PMC10849099.