

Case Report


A rare case of TB colitis of descending colon with miliary tuberculosis - A case report

E.A. Ashok Kumar^{1*}, Prudhvi Raj Marapelly²

¹Professor, ²Post Graduate

Department of General Medicine, Malla Reddy Institute of Medical Sciences, Hyderabad, Telangana, India

*Corresponding author email: ashokedla@gmail.com

	International Archives of Integrated Medicine, Vol. 8, Issue 5, May, 2021.	
	Available online at http://iaimjournal.com/	
	ISSN: 2394-0026 (P)	ISSN: 2394-0034 (O)
	Received on: 14-04-2021	Accepted on: 23-04-2021
	Source of support: Nil	Conflict of interest: None declared.
How to cite this article: E.A. Ashok Kumar, Prudhvi Raj Marapelly. A rare case of TB colitis of descending colon with miliary tuberculosis - A case report. IAIM, 2021; 8(5): 80-90.		

Abstract

Tuberculosis (TB) is still common in many countries and there has been a resurgence of TB in the developed nations. Gastrointestinal system TB is one of the extra-pulmonary manifestation of TB disease. Small bowel TB is the most commonly affected gastrointestinal tract, but colon TB is also described frequently in the literature. The diagnosis of colon TB is a challenge for physicians due to its diverse clinical manifestations that mimic other infectious, autoimmune, and neoplastic disorders, and is thus rarely considered as the causative agent of disease. Therefore, a high index of suspicion is essential to reach the correct diagnosis. Colonic tuberculosis, in the presence of pulmonary disease, is a relatively common clinical entity. The manifestations are nonspecific with diarrhea, abdominal pain, weight loss, fever, and abdominal mass being most common. Although the radiographic features of colonic tuberculosis are well described, it is difficult to differentiate from other diseases causing ulceration, stricture, or mass effect such as carcinoma and Crohn's disease. TB of the colon predominantly affects the cecum and the ascending colon. TB colitis of descending colon is rare. Ulceration, nodularity, and stricture are the prominent findings. Majority of the lesions heal with anti-TB treatment. Our Patient had old healed lesions of Pulmonary Tuberculosis and multiple tuberculous lymphadenitis at multiple sites. Hypotension, significant loss of weight, S. Cortisol levels were low and Barium enema revealed TB colitis of descending colon. Patient could not afford costly, sophisticated investigations. We herewith present a case report, diagnosed as Miliary Tuberculosis + Addisons Disease + TB lymphadenitis in multiple locations + TB colitis of descending colon with old healed lesions of Pulmonary Tuberculosis. Response to anti TB treatment and steroids was good.

Key words

Miliary Tuberculosis, Addison's Disease, TB Lymphadenitis, TB Colitis - descending colon.

Introduction

According to WHO, world-wide TB is one of the top 10 causes of death and the leading cause from a single infectious agent (above HIV/AIDS). In 2019, an estimated 10 million people fell ill with tuberculosis (TB) worldwide. 5.6 million men, 3.2 million women and 1.2 million children. TB is present in all countries and age groups. But TB is curable and preventable. In 2019, the 30 high TB burden countries accounted for 87% of new TB cases. Eight countries account for two thirds of the total, with India leading the count, followed by Indonesia, China, the Philippines, Pakistan, Nigeria, Bangladesh and South Africa. [1]. The World Health Organisation (WHO) TB statistics for India for 2019 give an estimated incidence figure of 2.64 million cases. This is a rate of 193 per 100,000 population [2]. The most common form of the disease is tuberculosis of the lungs, but the intestines, bones and joints, the skin, and the genitourinary, lymphatic, and nervous systems may also be affected. Extra pulmonary TB constitutes 15%, gastro intestinal TB constitutes 3%.

There has been increase in incidence of TB in many developed countries secondary to migrant population, deteriorating social conditions, cutbacks in public health services and increasing prevalence of immuno-suppressed individuals [3–9]. Co infection of TB and HIV was reported to be as high as 73% in one study [6]. It is a major concern in patients receiving biological agents for treatment like rheumatoid arthritis or inflammatory bowel disease [3, 10]. Immuno-suppressants given on misdiagnosis of inflammatory bowel disease can result in systemic spread of TB with serious complications. TB of the gastrointestinal tract is not that common as pulmonary TB. In GI tract terminal ileum is the most commonly affected organ. This is due to the high presence of lymphoid tissue in the terminal ileum and a

prolonged contact time of luminal contents. Ulcerations and luminal narrowing are the most common lesions found in the small bowel. Apart from the terminal ileum, several small case series have described TB affecting the colon [11–21]. TB of the colon is rare and most of time it is misdiagnosed as colon cancer, inflammatory bowel diseases, ischemic colitis or infectious colitis (also endemic in areas with high prevalence of TB). In view of the anatomic (lymphoid tissue) and physiological (contact time with luminal contents) differences between the small bowel and the colon, the resolution pattern of lesions in the colon after anti-TB treatment is not well known as routine colonoscopy and other modalities of investigations are not done after completion of the treatment [22]. The incidence of intestinal tuberculosis has declined since the introduction of pasteurization of milk and milk products, improvements in living conditions, and the introduction of anti-tuberculous drugs [23]. Its clinical manifestations are nonspecific [24, 25]. Intestinal tuberculosis typically affects the ileocaecal region. Isolated tuberculous colitis is extremely rare and only affects less than 3% of patients with documented intestinal tuberculosis. The most commonly observed pattern is concentric thickening of the ileum and caecum. There may be associated ascites and mesenteric lymphadenopathy in the majority of patients. Although the lymphadenopathy may be discrete, matted and cavitating lymph nodes are more frequent. Cavitating nodes have an anechoic central area. The ascites may be clear or may contain septations and debris. Mesenteric and omental thickening is also present in tuberculous ileocolitis. Chronic tuberculous ileocolitis may lead to a cicatrization and fibrosis of the bowel and patients may present with ileocolic strictures, mesenteric retraction and bowel obstruction [26]. Most patients with intestinal tuberculosis are asymptomatic. The most common complaint is chronic, nonspecific abdominal pain. Weight loss, fever, diarrhea or

constipation, and blood in the stool may be present. An abdominal mass, commonly located in the right lower quadrant of the abdomen, is appreciated in about two thirds of patients. Complications include gastrointestinal bleeding, obstruction, perforation, fistula formation, and malabsorption [27].

Pathology

Intestinal tuberculosis manifests itself in three pathological forms: ulcerative, fibrous, and hyperplastic. The first, the most frequent, is a complication of pulmonary tuberculosis, occurring in most of adults having pulmonary tuberculosis have intestinal involvement. The second is evidently a sequel to the ulcerative class, a result of the healing of the ulcers by the usual process of fibrous tissue formation, occasionally forming a stricture. The occurrence of such strictures, is exceedingly rare, as the general constitutional involvement at this stage is overwhelming. The third variety, hyperplastic tuberculosis of the intestine, a chronic inflammation and infiltration of the bowel walls, and an overproduction of fibrous tissue [28].

Diagnosis of colon TB.

Initial diagnosis of “confirmed colon TB” was made if the biopsy specimen showed caseating granulomas and/or acid-fast bacilli. Initial diagnosis of “suspected colon TB” was made in patients on clinical grounds: pyrexia of unknown origin, weight loss, anemia, active or previous pulmonary TB, history of TB in close contacts and biopsy showing non-caseating granuloma and chronic inflammation. Although patients with noncaseating granulomas could have inflammatory bowel disease, empiric immunosuppressive therapy was avoided and anti-TB treatment was started as all patients came from areas endemic for TB [22].

Diagnosis of colon TB was made by assessing response to treatment as per Paustian criteria with Logan’s modification [29]. Response to anti-TB treatment as a criteria to confirm TB has also been used in other studies [30]. Additional tests like Purified Protein Derivative (PPD) have

limited value in regions endemic to TB and are not recommended by the Revised National Tuberculosis Control Program guidelines from India for diagnosis of TB [31]. Because of insufficient data, QuantiFERON test is relatively contraindicated in the evaluation of active TB as active TB can be associated with suppressed interferon-g responses [32, 33]. Culturing intestinal biopsy specimens is expensive, and has been shown to be of low yield in previous studies. None of the 62 patients studied by Singh, et al. [34] were positive on culture and only 3 out of 50 intestinal biopsy specimens in the study by Shah, et al. [20] showed positive cultures.

Case report

A 24 year old male patient was admitted to the hospital with chief complaints of pain in the left flank of 2 days duration, loose stools of 2-3 episodes per day for 2 months, generalized weakness of 6 months duration.

History of present illness; Patient was apparently asymptomatic 6 months ago, then he developed generalized weakness which was insidious in onset which was initially on exertion later weakness gradually increased which was noticed as difficulty in walking and difficulty in getting up from squatting position. Patient developed loose stools of 2-3 episodes per day which did not contain blood, mucus or pus, non-foul smelling. Patient then developed pain in the left flank of 2 days duration which was insidious in onset and progressive, colicky type, not associated with nausea or vomiting with no aggravating or relieving factors. There was a significant history of decreased appetite of 1 year duration. There was a significant history of weight loss, 15 kg in 6 months (**Figure - 1** October, 2020 and **Figure - 2** March, 2021). No History of cough, shortness of breath, chest pain, palpitations, orthopnea, PND.

Past history: about 12 years ago patient suffered from tuberculous cervical adenitis on right side. The abscess over it ruptured for which he was treated at local hospital. After 3 years of this

episode, he developed similar swellings in the left axilla which got ruptured after forming an abscess for which he was started on ATT, but the patient stopped medication after 2 months. After 2 years of this episode, he developed a TB cold abscess on the right side of neck, incision and drainage was done. FNAC of the swelling showed caseating granuloma, AFB and CBNAAT was negative, he was started on ATT empirically. But he took treatment for 1 month, and stopped again. After 1 year the cold abscess recurred on right cervical region, again incision and drainage was done and started ATT. The patient used ATT for 4 months and stopped. There were no similar complaints in the family history.

Figure -1: October, 2020. **Figure -2:** March, 2021.



Personal history: Appetite: decreased, Diet: mixed, Digestion: normal, Sleep: sound, Bowel: the frequency of increased, loose stools, since 3 months, bladder: Regular, Addictions: occasional alcoholic, non-smoker, not a known diabetic, hypertensive.

General examination: Patient was conscious, coherent and cooperative. Thinly built and malnourished. Oral cavity: Teeth: stained. Cervical lymphadenopathy present with multiple enlarged lymph nodes largest measuring 2.5x2 cm, matted. Draining sinuses with Scrofuladerma were present on the right side of near the right ear lobe. Neck: multiple old TB scars (Scrofuladerma) are present on both sides of the neck, (**Figure - 3, 4, 5**) and Lt. axilla: healed scars were present on the left axilla. Severe Pallor was present, No icterus, Not cyanosed, Universal Clubbing (grade 3) was present, mild pedal edema present. VITALS; Temperature: afebrile; Pulse rate: 112 / minute, regular, low volume, not

collapsing, all peripheral pulses felt. Blood pressure: 70/50 mm Hg. Respiratory rate: 18 /min.

Figure - 3:

Lt. side of the neck.



Figure - 4:

Rt. side of the face



Figure - 5:

Rt. side of the neck.



Figure - 6: X Ray Chest PA View on 01/09/2012 – NAD.



Gastro intestinal system: Inspection: Shape of abdomen – scaphoid, No visible masses, no visible pulsations, no engorged veins, umbilicus normal in shape; Palpation: soft, severe tenderness noted extending from the left renal angle to left lumbar region anteriorly with

guarding and rigidity was present, there was no rebound tenderness, there was no organomegaly, All hernial orifices were normal; Percussion: no fluid thrill and no shifting dullness; Auscultation :normal bowel sounds were heard.

Other systems; C N S; Cardio Vascular System; Respiratory Systems exam was normal.

Figure – 7: X-Ray Chest PA View on 24/03/2021 – Shows mediastinal lymph node enlargement on Lt. side, with multiple healed parenchymal calcified lesions on both sides and solitary calcified lymph node Lt. axillary region.



Figure – 8: Plain X-Ray Abdomen reveals multiple para aortic lymph node calcifications.



Figure – 9: Barium enema reveals, edematous mucosal folds, mucosal ulcerations and nodularity, segmental luminal narrowing in descending colon.



Investigations

Complete blood picture: Hb-5.8 gm%, RBC Count - 2.6 mill/cumm, WBC Count-8,300 cells/cumm, Platelet Count -1.5 lakhs/cumm; Blood Picture – Microcytic, hypochromic anemia with relative neutrophilia; ESR - 110mm/hr; Random Blood Sugar – 90 mg/dl; B.Urea – 25 mg/dl, S. Creatinine - 0.9 mg/dl, LFT - Total Bilirubin - 0.4 mg/dl, Direct Bilirubin - 0.1 mg/dl, Indirect Bilirubin - 0.3 mg/dl, AST – 32 U/L, ALT - 21U/L, ALP – 318 U/L, Total Proteins - 4.3 g/dl, S. Albumin-1.2 g/dl, S. Globulin-3.1g/dl, A/G ratio-0.4. S. Electrolytes- Na^+ - 135 mEq/L; K^+ - 2.6 mEq/L; Cl^- - 101 mEq/L; Serum cortisol (between 7-9 AM) – 4.29 ug/dl (ref.4.30 – 22.4); HIV, HBsAg, HCV – Negative, CUE-Normal, Stool Examination-No Ova and Cysts were found, Stool for c/s – Negative; Stools for Occult Blood – Negative; Stools for AFB – Negative; Sputum

for AFB and CB NAAT - negative, X Ray Chest PA View on 01/09/2012 – NAD (**Figure - 6**), X Ray Chest PA View on 24/03/2021 – Shows mediastinal lymph node enlargement on lt. side, with multiple healed parenchymal calcified lesions on both sides, and solitary calcified lymph node lt. axillary region. (**Figure - 7**), Plain X Ray Abdomen reveals multiple para aortic lymphnode calcifications (**Figure - 8**),USG abdomen - :Liver shows increased echotexture - suggestive of fatty changes, Multiple peripancreatic, para aortic, mesenteric lymph nodes enlargement noted largest measuring 3.2x2.3 cm. Wall thickening of descending colon measuring 5.3mm in diameter with adjacent mesenteric fat stranding and omental caking noted. Minimal interbowel free fluid noted -p/o kochs etiology. Barium enema reveals, oedematous mucosal folds, mucosal ulcerations and nodularity, segmental luminal narrowing in descending colon. (**Figure - 9**), USG neck – Evidence of multiple matted lymph nodes noted largest measuring 2.7x1.5cm.Bilateral cervical lymphadenopathy. FNAC of cervical lymph node – revealed caseating tuberculous lymphadenitis. Patient was planned for colonoscopy but he didn't give consent for colonoscopy and also he could not afford sophisticated costly investigations like CT Scan of the abdomen.

Diagnosis

Patient had old healed lesions of Pulmonary Tuberculosis and multiple tuberculous lymphadenitis at multiple sites. Hypotension, significant loss of weight, S. Cortisol levels were low and Barium enema revealed TB colitis of descending colon. Patient could not afford costly, sophisticated investigations.

Diagnosed as Miliary Tuberculosis + Addison's Disease + TB lymphadenitis in multiple locations + TB colitis of descending colon with old healed lesions of Pulmonary Tuberculosis.

Treatment planned

Standard protocol of ATT, with Steroids.

Treatment given

- 1) Tab.AKT – 4; 1 strip daily
- 2) Tab. Wysolone 10mg T.I.D
- 3) Inj. Optineuron 1 amp iv O.D
- 4) inj. Buscopan 1Amp I.V. B.D
- 5) I.V.FLUIDS

Response

At the end of one month the response was good. Pain subsided, Gained 3 Kg of weight. BP increased to 100/70 from 70/50 mm of Hg.

Discussion

Colonic tuberculosis, without pulmonary tuberculosis is uncommon. The symptoms and signs are nonspecific with diarrhea, abdominal pain, weight loss, fever, and abdominal mass being most common [35]. The radiographic features of colonic tuberculosis are well described, but precise differentiation from other disorders producing ulceration, stricture, or mass effect like carcinoma and Crohn's disease is difficult [36]. Many patients with colonic TB have widespread tuberculosis in addition to the large bowel lesion [37]. In an Indian series, rectal bleeding was present in 30% of patients in addition to the other features of abdominal TB [34]. Colonic TB can appear as segmental ulcers, generalized colitis, mucosal nodules, polyps and strictures [34, 38, 39]. There are also reports of patients with colonic involvement presenting with perforation [40], and fistulae [41]. Barium studies in colonic TB may demonstrate strictures, colitis, perforations and less frequently, polypoid lesions [17], none of which are specific to colorectal TB. Colonoscopy is a successful diagnostic tool in 60–80% of patients with suspected colonic TB with the pattern of distribution, gross appearance, biopsy histology and microbiological culture contributing to the final diagnosis [12]. Colonic disease is usually segmental with two or more segments involved in 28– 58% of patients [42]; it is far less common to find a pancolitis (4–8%) [20].The most common colonoscopic features are ulcers of variable size (few mm up to 2 cm) and mucosal nodules [20]. Ulcers are usually superficial, transverse and circumferential, with sharp,

irregular margins surrounded by hyperaemic mucosa. The ulcer bases are often granular and covered with necrotic slough. Variable sized mucosal nodules (2– 6 mm) are friable and may be scattered or densely packed [20]. Strictures may be associated with mucosal nodules and ulcers in a third of patients and mucosal cobblestoning or pseudopolyps may also be present [20]. Multiple biopsy samples may confirm granulomas which are typically located in the submucosa [23]. Granulomas have been identified in 41– 48%, whereas caseation is found in 19–38% of biopsies [20]. Fine needle aspiration has been advocated to increase granuloma detection deeper in the submucosa. Culture of biopsy samples yield positive results in 6–69% of patients [20, 43]. Despite negative histology or microbiological culture, it is recommended that early anti-tuberculous therapy be initiated in patients with suggestive features at endoscopy [20].

Gastrointestinal TB, although not as commonly seen as pulmonary TB, can be a source of significant morbidity and mortality as it can mimic other conditions and tends to be diagnosed late. Less than 25% of patients with gastrointestinal TB have concomitant pulmonary TB [9]. Hence diagnosing gastrointestinal TB requires a high index of suspicion. Some of these patients are misdiagnosed as having inflammatory bowel disease and may get immunosuppressant medications resulting in dissemination of TB. The intestinal tract is usually affected by swallowed bacterium, spread from adjacent organs or via hematogenous spread. Secondary to abundant lymphoid tissue and the relatively longer contact time of intestinal contents with the mucosa, the terminal ileum has a higher predilection for TB infection [9, 11, 14]. Hence, in an appropriate clinical setting (for example, an immunosuppressed patient or immigrant population), TB is always in the differential diagnosis if there is an ulcer, stricture or an inflammatory mass in the terminal ileum. In a study by Nagi, et al. [17] only 10.8% of the 684 cases with abdominal TB had TB affecting the colon. Case reports and small

retrospective case series have been published describing the endoscopic features of TB of the colon. [11 - 18, 20, 21, 41, 44 - 50].

These features can be non-specific and mimic inflammatory bowel disease or malignancy. As follow-up colonoscopy is not routinely performed after completion of anti-TB treatment, there are limited data on predicting the outcomes of TB lesions, namely, resolution of, lack thereof, or scarring or stricturing of the lumen after healing. In their study, they report on one of the largest cohorts of patients with colon TB who were followed prospectively with colonoscopy examination after completing anti-TB treatment. Similar to previously reported case series [11, 21, 48, 51] majority of the patients had non-specific symptoms, with weight loss and abdominal pain being the predominant symptoms in over 80% of the patients. Less than half of the patients in this study had fever. Abdominal mass on physical examination was noted in only 6.4% of the patients. The number is significantly less compared with the study by Palmer et al. [52] in which 43% patients developed abdominal mass comprising either inflammatory intestinal lesions or enlarged mesenteric lymph nodes. The difference is likely due to the fact that their series had predominantly small bowel involvement in 40 out of 42 patients compared with only 4.7% showing isolated TB affecting the colon. Bleeding per rectum was observed in 20% of patients in the present series, but none of these was severe enough to require surgical or angiographic intervention. In the series by Misra, et al. [48] 10% of patients required surgery for massive lower gastrointestinal bleeding. Similar to previously published series [11, 21, 48], in their study, the predominant lesions identified on colonoscopy were ulceration and nodularity. The right colon (ileocecal valve, cecum, and ascending colon) was significantly more affected compared with the left. Nagi, et al. [17], on the other hand found the transverse colon to be the most commonly affected site and strictures the predominant lesion. However, their study was based on radiological evaluation and not colonoscopy. On the basis of the above, the

symptoms and the endoscopic findings of colon TB can be non-specific and hence diagnosing TB of the colon can be difficult. They had six patients with previous pulmonary TB as seen on chest X-ray and one patient with ascites had positive adenosine deaminase test. Histology was supportive of TB in 73% of the patients. Three patients showed positive acid-fast bacillus on histology; granulomas were the predominant finding in other patients, but as reported previously [48], caseating granulomas were rarely seen. In the current series, caseating granulomas were present in two patients and in all the five resected specimens of those who underwent surgery. Majority of the patients presented with suspected TB and diagnosis was confirmed by response to anti-TB treatment. Response to anti-TB treatment as a criterion to confirm TB has also been used in other studies [30]. Although it will be difficult to justify empiric anti-TB treatment for patients in developed countries, this has been an acceptable approach in countries where TB is endemic. Park, et al. [53] compared the outcomes of anti-TB treatment in patients with non-specific ileocecal ulcers, and showed that 3 months of trial of anti-TB treatment can be sufficient to assess response to treatment and thus differentiate between colon TB and Crohn's disease.

In our case, the patient is an old case of TB lymphadenitis not on regular treatment, his ESR was 110mm/hr, Barium enema revealed, edematous mucosal folds, mucosal ulcerations and nodularity, segmental luminal narrowing in descending colon. TB Colitis of descending colon is rare, S Shah, et al. [20], in his study, found colonic tuberculosis in descending colon in two patients out of 50 patients. Patient was planned for colonoscopy but he didn't give consent for colonoscopy and also he could not afford sophisticated costly investigations like CT Scan of the abdomen. Based on these probable findings we thought it as TB colitis with miliary TB and was started on ATT and steroids.

Conclusion

Usually the patients with TB of the colon present with nonspecific symptoms and hence diagnosis involves a high index of suspicion (migrant population, immuno-suppressed individuals). Abdominal pain and weight loss are the predominant symptoms. Ulceration, nodularity, and luminal narrowing are the prominent endoscopic findings mostly affecting the right colon. Histology is helpful in ruling out other conditions, but TB-specific findings like caseating granuloma and acid-fast bacilli are rarely seen. Tuberculous ulcers are morphologically different from Crohn's ulcers in that they are predominantly linear, fissured, transverse, or circumferential and the mucosa surrounding these ulcers is abnormal. Majority of the colon lesions resolve with anti-TB treatment, including strictures, thereby suggesting that strictures are more inflammatory rather than fibrotic in nature. Follow-up colonoscopy is not required in those who have symptomatic improvement after anti-TB treatment [22].

Our Patient had old healed lesions of Pulmonary Tuberculosis and multiple tuberculous lymphadenitis at multiple sites. Hypotension, significant loss of weight, S. Cortisol levels were low and Barium enema revealed TB colitis of descending colon. Patient could not afford costly, sophisticated investigations. Diagnosed as Miliary Tuberculosis + Addisons Disease + TB lymphadenitis in multiple locations + TB colitis of descending colon with old healed lesions of Pulmonary Tuberculosis. On follow up after one month, response to anti TB treatment and steroids was good.

References

1. WHO - Global Tuberculosis Report, 2020.
2. WHO - TB statistics for India, 2019.
3. Karagiannis S, Papaioannou D, Goulas S, et al. Intestinal tuberculosis in a patient on infliximab treatment. *Gastrointest Endosc.*, 2008; 67: 1178–1179 (discussion 1179).

4. Pop M, Pop C, Homorodean D, et al. Abdominal miliary tuberculosis in a patient with AIDS: a case report. *Rom J Gastroenterol* 2003; 12: 231–234.
5. Robles RR, Marrero CA, Reyes JC, et al. Risk behaviors, HIV seropositivity, and tuberculosis infection in injecting drug users who operate shooting galleries in Puerto Rico. *J Acquir Immune Defic Syndr Hum Retrovirol.*, 1998; 17: 477–483.
6. Heunis JC, Wouters E, Norton WE, et al. Patient- and delivery-system factors related to acceptance of HIV counseling and testing services among tuberculosis patients in South Africa: a qualitative study with community health workers and program managers. *Implement Sci.*, 2007; 6: 27.
7. Albalak R, O'Brien RJ, Kammerer JS, et al. Trends in tuberculosis/human immunodeficiency virus comorbidity, United States, 1993–2004. *Arch Intern Med.*, 2007; 167: 2443–2452.
8. Burzynski J, Schluger NW. The epidemiology of tuberculosis in the United States. *Semin Respir Crit Care Med.*, 2008; 29: 492–498.
9. Horvath KD, Whelan RL. Intestinal tuberculosis: return of an old disease. *Am J Gastroenterol.*, 1998; 93: 692–696.
10. Singh JA, Wells GA, Christensen R, et al. Adverse effects of biologics: a network metaanalysis and Cochrane overview. *Cochrane Database Syst Rev*; (2): CD008794.
11. Alvares JF, Devarbhavi H, Makhija P et al. Clinical, colonoscopic, and histological profile of colonic tuberculosis in a tertiary hospital. *Endoscopy*, 2005; 37: 351–356.
12. Bhargava DK, Kushwaha AK, Dasarathy S et al. Endoscopic diagnosis of segmental colonic tuberculosis. *Gastrointest Endosc.*, 1992; 38: 571–574.
13. Bhargava DK, Tandon HD, Chawla TC et al. Diagnosis of ileocecal and colonic tuberculosis by colonoscopy. *Gastrointest Endosc.*, 1985; 31: 68–70.
14. Das HS, Rathi P, Sawant P et al. Colonic tuberculosis: colonoscopic appearance and clinico-pathologic analysis. *J Assoc Physicians India*, 2000; 48: 708–710.
15. Medina E, Orti E, Tome A et al. Segmental tuberculosis of the colon diagnosed by colonoscopy. *Endoscopy*, 1990; 22: 188–190.
16. Morgante PE, Gandara MA, Sterle E. The endoscopic diagnosis of colonic tuberculosis. *Gastrointest Endosc.*, 1989; 35: 115–118.
17. Nagi B, Kochhar R, Bhasin DK et al. Colorectal tuberculosis. *Eur Radiol.*, 2003; 13: 1907–1912.
18. Namisaki T, Yoshiji H, Fujimoto M, et al. Two cases of colonic tuberculosis presenting with massive melena. *Int J Clin Pract.*, 2004; 58: 1162–1164.
19. Pulimood AB, Peter S, Ramakrishna B, et al. Segmental colonoscopic biopsies in the differentiation of ileocolic tuberculosis from Crohn's disease. *J Gastroenterol Hepatol.*, 2005; 20: 688–696.
20. Shah S, Thomas V, Mathan M, et al. Colonoscopic study of 50 patients with colonic tuberculosis. *Gut*, 1992; 33: 347–351.
21. Villanueva Saenz E, Martinez Hernandez Magro P, Fernando Alvarez-Tostado Fernandez J, et al. Colonic tuberculosis. *Dig Dis Sci.*, 2002; 47: 2045–2048.
22. Saurabh Mukewar, Shrikant Mukewar, Raghvendra Ravi, Arun Prasad, and Kulwinder S Dua; Colon Tuberculosis: Endoscopic Features and Prospective Endoscopic Follow-Up After Anti-Tuberculosis Treatment. *Clinical and Translational Gastroenterology*, 2012; 3: e24. doi:10.1038/ctg.2012.191.
23. Marshall JB. Tuberculosis of gastrointestinal tract and peritoneum. *Am J Gastroenterol.*, 1993; 88: 989-99.

24. Tabrisky J, Lindstorm RR, Peters R, Lachman RS. Tuberculous enteritis, review of a protean disease. *Am J Gastroenterol.*, 1975; 63: 49-57.
25. Paustian FF, Marshal JB. Intestinal tuberculosis. In: Berk JE. 4th edition, Bockus Gastroenterology. Vol 3. Philadelphia: WB Saunders; 198,. p. 2018-36.
26. Rakesh Sinha; CHAPTER 20 - Appendix, colon and rectum; Volume 1, 2011, Pages 388-410; In *Clinical Ultrasound (Third Edition)*; published by Churchill Livingston.
27. Davidson H. Hamer, Christopher J. Gill, Roma Chilengi. Intestinal Infections: Overview. *International Encyclopedia of Public Health (Second Edition)*, 2017, Pages 322-335.
28. G. Milton Linthicum. Tuberculosis of the large intestine; *American Journal of Surgery*; New Series Vol. XVI, No. 3; JUNE, 1932; 471 – 477
29. Logan VS. Anorectal tuberculosis. *Proc R Soc Med.*, 1969; 62: 1227–1230.
30. Makharia GK, Srivastava S, Das P, et al. Clinical, endoscopic, and histological differentiations between Crohn's disease and intestinal tuberculosis. *Am J Gastroenterol Clin.*, 2010; 105: 642–651.
31. Treatment of Tuberculosis: Guidelines for National Programmes. Fourth Edition. WHO Press: Geneva, Switzerland, 2010.
32. Streeton JA, Desem N, Jones SL. Sensitivity and specificity of a gamma interferon blood test for tuberculosis infection. *Int J Tuberc Lung Dis.*, 1998; 2: 443–450
33. Mazurek GH, Villarino ME. Guidelines for using the QuantiFERON-TB test for diagnosing latent Mycobacterium tuberculosis infection. Centers for Disease Control and Prevention. *MMWR Recomm Rep.*, 2003; 52: 15–18.
34. Singh V, Kumar P, Kamal J et al. Clinicocolonoscopy profile of colonic tuberculosis. *Am J Gastroenterol.*, 1996; 91: 565–568.
35. Balthazar EJ, Bryk D. Segmental tuberculosis of the distal colon: Radiographic features in 7 cases. *Gastrointest Radiol.*, 1980;5:75-80.
36. Carrera GF, Young S, Lewicki AM. Intestinal tuberculosis. *Gastrointest Radiol.*, 1976; 1: 147-55.
37. Chen WS, Leu SY, Hsu H, Lin JK, Lin TC. Trend of large bowel tuberculosis and the relation with pulmonary tuberculosis. *Dis Colon Rectum.*, 1992; 35: 189–92.
38. Kalvaria I, Kottler RE, Marks IN. The role of colonoscopy in the diagnosis of tuberculosis. *J Clin Gastroenterol.*, 1988; 10: 516–23.
39. Breiter JR, Hajjar JJ. Segmental tuberculosis of the colon diagnosed by colonoscopy. *Am J Gastroenterol.*, 1981; 76: 369–73.
40. Gupta NM, Motup T, Joshi K. Isolated colonic tuberculous perforation as a rare cause of peritonitis: report of a case. *Surg Today*, 1999; 29: 273–5.
41. Patel M, De I. Segmental tuberculosis of the colon with entero-colic fistula. *Br J Radiol.*, 1972; 45: 150–2.
42. Chawla S, Mukerjee P, Bery K. Segmental tuberculosis of the colon (a report of ten cases). *Clin Radiol.*, 1971; 22: 104–9.
43. Sakai Y. Colonoscopic diagnosis of the intestinal tuberculosis. *Mater Med Pol.*, 1979; 11: 275–8.
44. Carkman S, Ozben V, Aytac E. Cecum perforation due to tuberculosis in a renal transplant recipient: a case report. *J Med Case Reports*, 2009; 3: 132.
45. Das K, Puri S, Puri AS. Gastrointestinal: multiple colonic strictures caused by tuberculosis. *J Gastroenterol Hepatol.*, 2006; 21: 476.
46. Devanesan JD, Sable RA, Pitchumoni CS et al. Segmental tuberculosis of the

- colon mimicking carcinoma. Arch Surg., 1980; 115: 90–91.
47. Madani TA. Colonic tuberculosis clinically misdiagnosed as anorexia nervosa, and radiologically and histopathologically as Crohn's disease. Can J Infect Dis., 2002; 13: 136–140.
48. Misra SP, Misra V, Dwivedi M et al. Tuberculous colonic strictures: impact of dilation on diagnosis. Endoscopy, 2004; 36: 1099–1103.
49. Steer D, Essa A, Clarke DL et al. Transverse colon tuberculosis presenting as colonic obstruction. S Afr J Surg., 2009; 47: 31–32.
50. Wadhwa N, Agarwal S, Mishra K. Reappraisal of abdominal tuberculosis. J Indian Med Assoc., 2004; 102: 31–32
51. Chong VH, Lim KS. Gastrointestinal tuberculosis. Singapore Med J., 2009; 50: 638–645 (quiz 646).
52. Palmer KR, Patil DH, Basran GS, et al. Abdominal tuberculosis in urban Britain—a common disease. Gut, 1985; 26: 1296–1305.
53. Park YS, Jun DW, Kim SH, et al. Colonoscopy evaluation after short-term anti-tuberculosis treatment in nonspecific ulcers on the ileocecal area. World J Gastroenterol., 2008; 14: 5051–5058.