

Abdominal Tuberculosis: A Histopathological Study with Special Reference to Intestinal Perforation and Mesenteric Vasculopathy

Alakananda Dasgupta, Navjeevan Singh, Arati Bhatia

Department of Pathology, University College of Medical Sciences and Guru Teg Bahadur Hospital, Delhi, India

Address for correspondence: Dr. Alakananda Dasgupta, E-mail: alakanandadasgupta@gmail.com

ABSTRACT

Background: Along with the increased incidence of pulmonary tuberculosis in parallel with the increase in population in various parts of the world, in recent years, the incidence of abdominal tuberculosis has also increased. The pathogenetic events in intestinal tuberculosis, which culminate in ulcer formation, perforation, and stricture, still have to be identified.

Aim: To correlate the gross and microscopic features in intestinal tuberculosis, in particular tuberculous perforation with changes in mesenteric vasculature.

Patients and Methods: A one-year prospective study of excised/resected tissues from patients with abdominal tuberculosis requiring surgical intervention was conducted. Tissues from fifty-six patients were included in the study—of which 36 were resected intestinal segments and 20 were intestinal and lymph node biopsies. Hematoxylin and Eosin and Ziehl-Neelsen stains were used for histopathological examination.

Results: Tuberculous enteritis was found to be present in 49 of the 56 patients (87.5%) (ileum being the site most commonly affected), while nodal involvement was seen in 39 (69.6%) patients. Perforations were present in 39 out of 49 (79.6%) intestinal tissues; most being solitary and ileum was the commonest site. Typical epithelioid cell granulomas were seen in the intestine and lymph nodes, with caseation being more prevalent in the latter. The mesenteric vasculature was frequently involved by granulomatous inflammation, with intravascular organizing thrombus being present in 30% of the resected specimens with perforation. Acid fast bacilli were demonstrated in the tissue sections of 37.5% of the patients. AFB positivity was higher in caseating granulomas.

Conclusion: Involvement of mesenteric vasculature by granulomatous inflammation was commonly associated with the ulcerative type with perforation, suggesting that ischemia caused by vascular thrombosis is responsible for tissue breakdown. This implies that vasculitis plays an important role in the natural history of abdominal tuberculosis.

Keywords: Abdominal tuberculosis, intestinal perforation, mesenteric vasculopathy, acid fast bacilli

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INTRODUCTION

Tuberculosis (TB) is a chronic granulomatous disease caused by *Mycobacterium tuberculosis*. The usual site of infection is the lung, but other organs may be involved.

Abdominal tuberculosis represents the sixth most frequent form of extra-pulmonary tuberculosis after lymphatic, genitourinary, bone and joint, miliary, and meningeal tuberculosis.^[1-3] Tuberculous bacteria reach the gastrointestinal tract via hematogenous spread, ingestion of infected sputum, or contiguous spread from adjacent organs.^[1,2,4-9] Almost all cases of abdominal TB are caused by *Mycobacterium*

tuberculosis.^[2,4,6,9] The predilection of the bacillus for the ileocecum, is attributed mainly to three factors: Relative physiological stasis of the area, the high rate of absorption, with more complete digestion (permitting free contact of the organism with the mucosal lining), and the abundance of lymphoid tissue at this site.^[1,6,8,10,11] There are three gross morphological forms of tuberculous enteritis: Ulcerative, hypertrophic, and ulcerohypertrophic. The ulcerative type, which commonly affects the ileum and jejunum, is characterized by a single or multiple transverse ulcers, the healing of which leads to stricture formation, and may perforate, bleed, or form fistulas. The hypertrophic and ulcerohypertrophic types commonly affect the ileocecum and cause obstruction

or present as a mass.^[1,2,4,8,10-13] Grossly, peritoneal tubercles and enlarged, matted, caseous mesenteric lymph nodes may be seen.^[1,2,4] Microscopically, numerous, large, confluent granulomas of variable size, composed of epithelioid cells, with a peripheral zone of lymphocytes and Langhan's giant cells with central caseous necrosis, and surrounding fibrosis are seen.^[12,14] These 'caseating granulomas' are a characteristic histological feature of tuberculosis. Lesions are seen mainly in the submucosal and serosal layers. Sometimes, granulomas with caseation are seen only in the regional lymph nodes.^[8]

Perforation is a serious complication of abdominal TB, associated with high morbidity and mortality.^[4,6,8,15-17] The low incidence of tuberculous perforation is due to reactive fibrosis of the peritoneum.^[6,15,16,18-21] However, in recent years, intestinal perforation, which was relatively rare in the past, has been reported more frequently. The cause of this remains unknown.

Vasculitis is a well-established feature of tuberculosis of the central nervous system (CNS),^[22,23] lungs,^[24] and kidneys,^[25] but scarcely described in intestinal tuberculosis. Few studies have evaluated the role of mesenteric vasculitis in the natural history of intestinal tuberculosis.^[26,27] Ischemic changes have been said to underlie perforation and stricture formation. However, an in-depth analysis into the cause of intestinal changes in abdominal tuberculosis and the association between macroscopic and microscopic features (i.e., perforation, ulceration, and stricture formation) with changes in mesenteric vasculature has not been done previously. This study attempts to elucidate the changes in mesenteric vessels and their association with perforation, and also document various gross morphological types of intestinal TB and their correlation with the microscopic features.

PATIENTS AND METHODS

Patients

A prospective study was conducted. The study population comprised of 56 patients with abdominal tuberculosis, who underwent emergency or elective laparotomy for obstruction or perforation over a period of one year. Of these, the number of resected specimens of intestine was 36 and intestinal biopsies that included perforation edge biopsies and strictures were 13. In addition, there were 14 lymph-node biopsies. In seven cases, both intestinal and lymph node biopsies were sent. The total number of cases of perforation (resected specimens and biopsies) was 39 (69.6%), of which 30 (53.5%) were resected specimens of intestine.

Histopathological evaluation

Gross findings recorded were length of the intestine, number of strictures, perforations, and ulcerations, circumference of the stricture compared to the circumference of the intervening normal intestine, relationship of the perforation to the stricture, form of lesion (ulcerative, proliferative, or ulceroproliferative), draining lymph nodes, serosal tubercles, and mesenteric vasculature.

Microscopic features recorded were granulomatous inflammation without necrosis, granulomatous inflammation with necrosis, and necrosis with acid fast bacilli positivity (AFB positivity), in sections from the intestine, lymph nodes, and mesenteric vasculature. Sections were stained by Hematoxylin and Eosin (H and E) and Ziehl-Neelsen (ZN) stain for acid fast bacilli. A known positive control section was used to ensure that correct differentiation had been achieved.

RESULTS

Of all the surgically removed tissues from patients with abdominal tuberculosis, during the study period, intestinal resection was the most common procedure followed by lymph node and intestinal biopsy. The total number of cases with perforation (resected specimens and biopsies) was 39 of 56 (69.6%), of which 30 (76.9%) were resected specimens of the intestine.

Clinical presentation

Perforation peritonitis was the most frequent clinical presentation followed by intestinal obstruction. There was a wide age range, with the majority between the ages of 10 and 30. The mean age was 25 years and the male: Female ratio was 0.9:1.9. Associated pulmonary tuberculosis was present in only two patients. In the absence of active lung disease, abdominal tuberculosis was probably due to reactivation of latent infection. Two patients developed perforation and one intestinal obstruction, while on chemotherapy.

Macroscopic features

Tuberculous enteritis was seen in 49 of the 56, (87.5%) patients, ileum being the most commonly affected site. Colonic and appendiceal involvement was the least. Perforation also occurred most frequently in the ileum [26 of 30 (86.7%) resected specimens]. Associated nodal and peritoneal involvement (serosal tubercles) were present in 25 (69.4%) and 11 (30.6%) resected specimens, respectively.

The lymph nodes involved were those along the mesentery and those of the ileocecal region. In all, lymph nodes were involved in 39 (69.6%) patients, and caseation (which was confirmed microscopically) was present in 33 (84.6%) of these. Pure nodal involvement was seen in 4 (11.1%) and multiple sites of involvement were seen in 29 (80.6%) of the resected specimens. The circumference of normal versus strictured segments was 4-8cm and 0.5-4cm, respectively.

Of the total 49 cases (36 resected intestinal specimens and 13 intestinal biopsies), perforation was seen in 39 (79.6%), of which 30 (76.9%) were in resected specimens. Most were solitary (23 out of 30 or 76.7%), in the ileum (26 out of 30 or 86.7%) and at the site of the stricture, along with superficial transverse ulcers, exudates on the external surface, miliary tubercles and lymphadenopathy being present (in the resected specimens).

Strictures were mostly present in the ulcerative lesions with perforation (26 out of 30 or 86.7%); of which 10 or 38.5% were multiple. Among the three types of gross morphological forms, the ulcerative type was the most common followed by ulcerohypertrophic and hypertrophic types, among the resected specimens. Seventy percent (21 out of 30) of the perforations were seen in the ulcerative type. Perforation was seen in the absence of strictures in four (19.1%) cases, with ulcerative lesions.

Microscopic features

In the intestine, epithelioid cell granulomas were seen in 33 out of 36 (91.7%) cases in the submucosa and serosa, with caseation in 18 (54.5%) of these [Figure 1]. Multiple confluent granulomas and fibrosis around the granuloma were seen in the resected specimens. In three resected specimens and one biopsy, in the absence

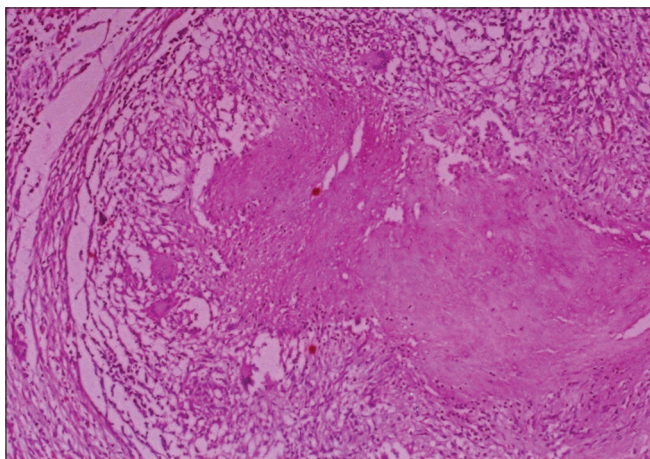


Figure 1: Caseating granuloma: typical epithelioid cell granuloma with central caseation and a peripheral part composed of lymphocytes and Langhan's type of giant cells is a diagnostic finding; surrounding fibrosis is also seen. H and E, $\times 200$

of intestinal granulomas, the diagnosis was based on caseating granulomas in the lymph node. Superficial ulcers and organized serositis were present in the cases with perforation. Thickening of the wall in the hypertrophic and ulcerohypertrophic types was mostly due to extensive granulomatous inflammation and, in a few instances, due to submucosal fibrosis, edema, and serosal fibrosis.

Tissue reaction that is typical of tuberculosis was seen more often in the lymph nodes than in intestinal lesions. Caseation (in 22 of the 25 or 88% of lymph nodes in the resected specimens and 11 out of 14 or 78.6% of the lymph node biopsies), fibrosis around granulomas, and multiple confluent granulomas were seen more often in the lymph nodes.

It is considered that intestinal lesions with caseation, with reaction similar to the draining lymph nodes, are sufficiently characteristic of supporting a diagnosis of TB, even in the absence of bacteriological proof. However, in this study, this was the case in 10 patients only.

Acid fast bacilli (AFB) could be demonstrated in 21 (37.5%) tissue sections [Figure 2]. Of these, 15 (71.4%) were resected specimens and six (28.6%) were biopsies. Of the resected specimens, six (40%) showed positivity in the intestine, six in the lymph node, and three (20%) in both. Of the six biopsies, one intestinal biopsy (16.7%), and four lymph node biopsies (66.7%) were positive. In one case, both intestinal and lymph node biopsies were positive. AFB were seen more often in specimens with perforation (40%). AFB positivity in the intestine was present in 11 (24.4%), in the lymph nodes in 14 (35.9%), and in the mesentery in 5 (23.8%) patients [Table 1]. AFB positivity was higher in caseating granulomas.

Mesenteric vasculopathy

Changes in the mesenteric vasculature were observed in

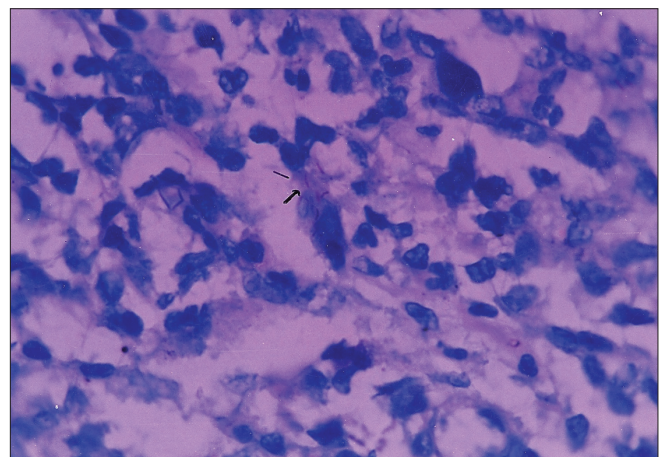


Figure 2: Acid fast bacilli in a granuloma. ZN $\times 1000$

32 (88.9%) resected specimens and 7 (53.9%) intestinal biopsies. The mesenteric vasculature (medium and small vessels mostly) was frequently involved (36/45 i.e., 80%) by granulomatous inflammation, which included intravascular organizing thrombus/intravascular granuloma, leading to occlusion of the lumen [Tables 2-4 and Figures 3 and 4].

Acid fast bacilli could be demonstrated by Ziehl Neelsen stain in the vessel wall in only a single resected specimen (with perforation).

DISCUSSION

Along with the increase in population and increased incidence of pulmonary TB, that of abdominal TB has also increased. This has been attributed to the emergence of multi-drug resistant strains of *Mycobacterium tuberculosis*, infection with human immunodeficiency virus (HIV) or Acquired immunodeficiency syndrome (AIDS), along with factors like poverty, overcrowding, inadequate public health measures, partial anti-tuberculous therapy, and influx of migrants, prevailing in different parts of the world.^[1,2,4,8,15,16,28,29]

Clinical presentation

In this study, cases of abdominal tuberculosis that required surgical intervention were common (in cases with obstruction and perforation). Perforation peritonitis was more common than that reported previously. In our geographical region, where there is a high incidence of tuberculosis, pulmonary disease was uncommon in patients with tuberculous enteritis.

Macroscopic and microscopic features

The ulcerative form of gross morphological type, with strictures, and tuberculous perforation, and mesenteric lymph node involvement, along with microscopic features of caseating granuloma and AFB positivity were the most common combination of findings.

Perforation

Reactive fibrosis of the peritoneum and formation of adhesions with adjacent tissues accounts for the low incidence (0-11% in adults, 3-4% in children, 2.5-6% at autopsy, and 20% of all non-appendiceal perforations) reported in literature.^[6,15,16,18-20] However, in the present

Table 1: Acid fast bacilli positivity in caseating and non-caseating granulomas in the intestine, lymph nodes, and mesentery

Granuloma	AFB positivity				
	Intestine		Lymph node		Mesentery (%) (n = 21)
	Resected (%) (n = 33)	Biopsy (%) (n = 12)	Resected (%) (n = 25)	Biopsy (%) (n = 14)	
Caseating	8/18* (44.4)	2/6 (33.3)	8/22 (36.4)	5/11 (45.5)	3/10 (30.0)
Non-caseating	1/15 (6.7)	0/6 (0.0)	1/3 (33.3)	0/3 (0.0)	2/11 (18.2)
Total	9/33 (27.3)	2/12 (16.7)	9/25 (36.0)	5/14 (35.7)	5/21 (23.8)

*In one patient, acid fast bacilli were demonstrated in the vessel wall

Table 2: Number of cases with granuloma in and around the vessel wall

Granuloma	No. of cases (%) (n = 45)*	Resected (%) (n = 33)*	Biopsies (%) (n = 12)*
In the vessel wall	13 (28.9)	12 (36.4)	1 (8.3)
Near the vessel wall	23 (51.1)	19 (57.6)	4 (33.3)

*Three resected specimens and one intestinal biopsy did not show any granuloma

Table 3: Relation between changes in mesenteric vessels with macroscopic features

Type of vascular involvement	Stricture (%) (n = 29)	Perforation (%) (n = 30)	Ulcer (%) (n = 34)
Intravascular granuloma	9 (31.0)	9 (30.0)	11 (32.4)
Granuloma near vessel	15 (51.7)	17 (56.7)	18 (52.9)
Perivascular cuff	20 (69.0)	22 (73.3)	24 (70.6)

Table 4: Vasculitis in specimens of intestinal tuberculosis

Type of vascular involvement	All cases (n = 45)* (%)						Cases with perforation (n = 39) (%)					
	Resected (n = 33)*			Biopsies (n = 12)*			Resected (n = 30)			Biopsies (n = 9)		
	S	M	L	S	M		S	M	L	S	M	
Granuloma in and near vessels	31 (93.9)			5 (41.7)			26 (86.7)			4 (44.4)		
	13 (41.9)	16 (51.6)	2 (6.5)	5 (100)	0 (0)		13 (50.0)	12 (46.2)	1 (3.9)	4 (100)	0 (0)	
Perivascular cuff	26 (78.8)			5 (41.7)			22 (73.3)			4 (44.4)		
	26 (100)	0 (0.0)	0 (0.0)	5 (100)	0 (0)		22 (100)	0 (0.0)	0 (0.0) (0)	4 (100)	0 (0)	

*Three resected specimens and one intestinal biopsy did not show any granuloma; S - small, M - medium, L - large sized vessels

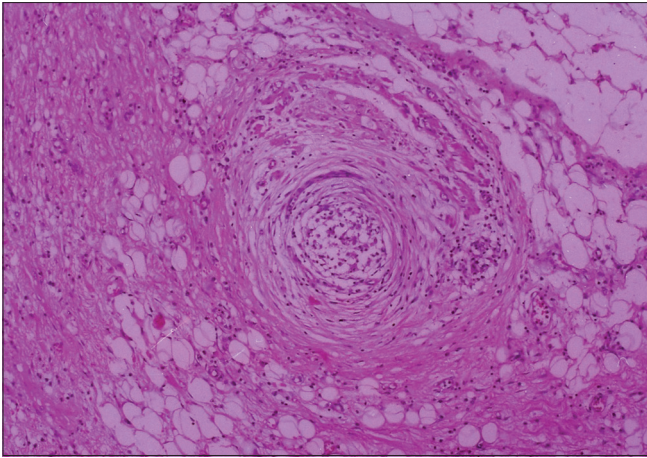


Figure 3: Intravascular granuloma: organizing thrombus due to granulomatous inflammation causing occlusion of a muscular artery (medium sized vessel). H and E \times 300

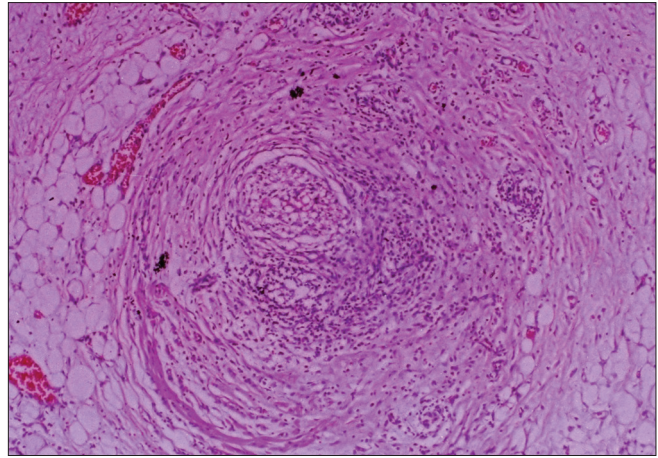


Figure 4: Intravascular granuloma or organizing thrombus in a medium sized vessel. H and E \times 100

study, perforation was more common than that reported previously (79.6% of all cases and 83.3% of resected specimens). This observed discrepancy can partly be explained by the fact that 71% of the patients presented with an acute abdomen and that surgery is reserved mainly for acute complications such as perforation and obstruction or in cases where there is diagnostic uncertainty.

Even then, ileal perforation, which is a relatively rare complication in abdominal TB and is mostly attributed to typhoid, seems not to be as uncommon, as shown by the results. Perforation is reported to be more frequent in immunocompromised patients, such as those with AIDS, as the reduction in immunity leads to decreased fibrosis and hence reduced healing.^[2] An increased incidence of perforation has been observed in patients who are taking antituberculous treatment, due to reduction of inflammatory response by therapy, before adequate fibrosis has occurred, leading to poor healing of the ulcers or a drug induced paradoxical reaction (caused by local tissue immunity alterations in relation to mycobacterial metabolic products).^[4,15,16] This was seen in two patients with perforation.

As most patients were from a low socioeconomic strata, it is possible that malnutrition (and the resultant decrease in cell mediated immunity, fibrosis, and healing) was a factor in the pathogenesis and increased frequency of perforation in abdominal TB in this study, as compared to a study of patients from affluent areas.

Most of the perforations were solitary and located in the ileum. Perforations were present both proximal to and at the site of the stricture, as reported.^[4,15,17,18] Multiple perforations occur in 40% of the patients and are associated with a poor prognosis.^[15]

Acid fast bacilli by Ziehl Neelsen stain

The total number of patients in whom AFB could be demonstrated by ZN stain was 21 of 56 (37.5%). Demonstration of AFB in the ZN-stained sections in the necrotic debris or within the granuloma provides a definitive diagnosis.^[2,4,6,8,11,12,14] Therefore, any lesion of the bowel showing epithelioid cell granulomas should be carefully searched for tubercle bacilli.^[14] Reports of AFB positivity in intestinal TB in literature are quite variable; from as low as 6-8%^[4,7] to 53.4%.^[15] Except for a few cases in which a large number of tubercle bacilli were present, most cases showed positivity only after a prolonged search.

AFB positivity was higher in caseating granulomas. Some studies, however, report that the amount of caseation bears no relationship to the ease with which bacilli are demonstrated.^[14] In one resected specimen with caseation in the intestine, numerous mycobacteria were seen only in granulomas without necrosis, and none in the caseous areas.

Mesenteric vasculopathy

Vasculitis is a well documented feature of tuberculosis of the CNS,^[22,23] lungs,^[24] and kidney.^[25] However, few studies have evaluated the role of mesenteric vasculitis in the natural history of intestinal tuberculosis.^[26,27] One such study observed changes in the large, medium, and small mesenteric vessels, in patients with histologically proven intestinal tuberculosis presenting with obstruction and perforation.^[30] Presence of granulomas in or adjacent to the vessel wall (suggesting direct involvement), thrombus formation in large and medium vessels, and subintimal fibrosis were also observed. In addition, perivascular cuffing was present in the intramural and subserosal vessels. Based on the above

findings, it was postulated that changes in the vessel wall may lead to gut ischemia, which may contribute to the development of ulcers, perforation, fibrosis, and strictures.^[31]

Enderteritis of submucosal vessels due to intravascular granuloma, a feature seen in this study, has been reported earlier and may have caused the mucosal ulceration and perforation.^[14] Organizing thrombus has been observed in a case report.^[28] Acid fast bacilli have been demonstrated in the media and adventitia.^[32]

Involvement of mesenteric vessels in intestinal tuberculosis and their role in the pathogenesis of intestinal changes has been reported in a few studies only. In the present study, the mesenteric vessels in the resected specimens were dissected and observed for vascular changes. Intramural blood vessels were also specifically studied for the presence of granulomatous inflammation and acid fast bacilli. This was done in order to determine the role and type of vascular involvement in gastrointestinal tuberculosis.

Granulomas were seen in and near medium and small-sized vessels, suggesting a direct involvement. Intravascular organizing thrombus, due to granulomatous inflammation, leading to occlusion of the lumen of the vessel, was present in the medium (muscular) arteries. Perivascular cuffing was, however, noted in the intramural and subserosal (small) vessels only and is probably a non-specific finding.

In conclusion, involvement of mesenteric vasculature by granulomatous inflammation was commonly associated with the ulcerative type with perforation, suggesting that tissue breakdown due to ischemia, caused by vascular thrombosis, plays a role in the pathogenesis of perforation in intestinal tuberculosis.

This, therefore, implies that vasculitis plays an important role in the natural history of abdominal tuberculosis.

REFERENCES

1. Marshall JB. Tuberculosis of the gastrointestinal tract and peritoneum. *Am J Gastroenterol* 1993;88:989-99.
2. Aston NO. Abdominal tuberculosis. *World J Surg* 1997;21:492-9.
3. Kapoor VK. Abdominal tuberculosis: The Indian contribution. *Indian J Gastroenterol* 1998;17:141-7.
4. Kapoor VK. Abdominal tuberculosis. *Postgrad Med J* 1998;74:459-67.
5. Das P, Shukla HS. Clinical diagnosis of abdominal tuberculosis. *Br J Surg* 1976;63:941-6.

6. Bhansali SK. Abdominal tuberculosis: Experiences with 300 cases. *Am J Gastroenterol* 1977;67:324-37.
7. Prakash A. Ulcero-constrictive tuberculosis of the bowel. *Int Surg* 1978;63:23-9.
8. Horvath KD, Whelan RL. Intestinal tuberculosis: Return of an old disease. *Am J Gastroenterol* 1998;93:692-6.
9. Tandon HD. The pathology of intestinal tuberculosis and distinction from other diseases causing stricture. *Trop Gastroenterol* 1981;2:77-93.
10. Paustian FF, Bockus HL. So-called primary ulcerohypertrophic ileocecal tuberculosis. *Am J Med* 1959;27:509-18.
11. Paustian FF, Marshall JB. Intestinal tuberculosis. In: Berk JE, editor. *Bockus Gastroenterology*. 4th ed. Philadelphia: WB Saunders: 1985. p. 2018-36.
12. Tandon HD, Prakash A. Pathology of intestinal tuberculosis and its distinction from Crohn's disease. *Gut* 1972;13:260-9.
13. Prakash A, Tandon HD, Nirmala L, Wadhwa SN, Prakash O, Kapur M. Chronic ulcerative lesions of the bowel. *Indian J Surg* 1970;32:1-14.
14. Howell JS, Knappton PJ. Ileocaecal tuberculosis. *Gut* 1964;5:524-9.
15. Talwar S, Talwar R, Prasad P. Tuberculous perforations of the small intestine. *Int J Clin Pract* 1999;53:514-8.
16. Seabra J, Coelho H, Barros H, Alves JO, Rocha-Marques A. Acute tuberculous perforation of the small bowel during anti-tuberculosis therapy. *J Clin Gastroenterol* 1993;16:320-2.
17. Wig JD, Malik AK, Chaudhary A, Gupta NM. Free perforations of tuberculous ulcers of the small bowel. *Indian J Gastroenterol* 1985;4:259-61.
18. Chitkara N, Garg P, Tehlan RN, Dheer V. Multiple ileal perforations. *Postgrad Med J* 1997;73:757-8.
19. Bagga D, Taneja SB. Perforated tuberculous enteritis of childhood: A ten year study. *Indian J Pediatric* 1990;57:713-6.
20. Chaudhary SK. The perforation of tuberculous lesion of the intestine is extremely rare. *J Indian Med Assoc* 1997;95:59,63.
21. Arunabh AS, Kapoor VK, Chattopadhyay TK. Tuberculous perforations of the small intestine. *Indian J Tuberculosis* 1986;33:190-1.
22. Damjanov I, Linder J. Mycobacterial diseases, mycobacterial vasculitis. In: *Anderson's Pathology*. 10th ed. Vol. 1. USA: Mosby-Year Book, Inc.; 1996. pp. 843-50 and 1142-3.
23. Lalitha VS, Dastur DK. Histopathology of blood vessels in neurotuberculosis. In: *Proceedings of the symposium on tuberculosis of the central nervous system*. New Delhi: Indian Academy of Medical Sciences; 1973. pp. 97-116.
24. Bamberg P, Bharati B, Dutta BM. Pulmonary vasculitis and the Indian perspective. *Semin Resp Med* 1991;12:115-23.
25. Gupta KL, Sakhuja V, Katariya S, Malik AK. Tuberculous arteritis in renal tuberculosis. *Indian J Tuberculosis* 1989;36:181-3.
26. Shah P, Ramakant R. Role of vasculitis in the natural history of abdominal tuberculosis: Evaluation by mesenteric angiography. *Indian J Gastroenterol* 1991;10:127-30.
27. Bhusnurmath SR, Nagi B. Significance of vasculitis in gut tuberculosis. *Indian J Gastroenterol* 1991;10:125-6.
28. Ahmed FB. Grand rounds- Hamersmith hospital: Tuberculous enteritis. *BMJ* 1996;313:215-7.
29. Dolin PJ, Raviglione MC, Kochi A. Global tuberculosis incidence and mortality during 1990-2000. *Bull World Health Organ* 1994;72:213-20.
30. Kuwajerwala NK, Bapat RD, Joshi AS. Mesenteric vasculopathy in intestinal tuberculosis. *Indian J Gastroenterol* 1997;16:134-6.
31. Sharma MP, Bhatia V. Abdominal tuberculosis. *Indian J Med Res* 2004;120:305-15.
32. Malik AK, Bhasin DK, Pal L, Wig JD, Singh K, Mehta SK. Does vasculitis occur in abdominal tuberculosis? *J Clin Gastroenterol* 1992;15:355-6.

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