

Abdominal tuberculosis: a panoramic view

A Sunkara, D Wagh, S Singhal

Citation

A Sunkara, D Wagh, S Singhal. *Abdominal tuberculosis: a panoramic view*. The Internet Journal of Surgery. 2009 Volume 22 Number 2.

Abstract

Tuberculosis can involve almost any part of the gastrointestinal tract and the latter is considered the sixth most frequent site of extrapulmonary involvement. The most common site of involvement of gastrointestinal tuberculosis is the ileocaecal region. Ileocaecal and small bowel tuberculosis presents with a palpable mass in the right lower quadrant and/or complications of obstruction, perforation or malabsorption, especially in the presence of stricture. Useful modalities for investigating a suspected case include small-bowel barium meal, barium enema, ultrasonography, computed tomographic scan and colonoscopy. Ascitic fluid examination reveals straw-coloured fluid with high protein, serum/ascites albumin gradient less than 1.1g/dl, predominantly lymphocytic cells, and adenosine deaminase levels above 36 U/l. Laparoscopy is a very useful investigation in doubtful cases. Management is with conventional antitubercular therapy for at least 6 months. The recommended surgical procedures are conservative and a period of preoperative drug therapy is controversial.

INTRODUCTION

Tuberculosis of the gastrointestinal tract is the sixth most frequent form of extra-pulmonary site; after lymphatic, genitourinary, bone and joint, miliary and meningeal tuberculosis.¹ Five percent of TB patients may have abdominal tuberculosis² and the most common site of involvement of gastrointestinal tuberculosis is the ileocaecal region. HIV sero-positivity prevalence in patients of abdominal tuberculosis is significantly higher (16.6%) compared to pulmonary tuberculosis (6.9%)³ and with the emergence of multi-drug resistant bacilli, HIV/AIDS have posed newer threats and added a new dimension to the control of tuberculosis.

BACKGROUND

Tuberculosis (TB) has existed from the very dawn of civilization with records of TB in mummies found in Egypt as far as 5000 years ago. Extra-pulmonary tuberculosis was also known since antiquity. Pott's disease was described in paintings and statues of ancient Egyptians. Hippocrates had noted intestinal involvement in pulmonary tuberculosis in 5 B.C. He said "Diarrhea attacking a person with phthisis is a mortal symptom".⁴ Ebn Sina, the famous Arab Scientist (980-1037) described tuberculosis (Al-Sol) in detail in his book "Al-Kanoun". He described abdominal distention, diarrhea and borborygmi. Lubeck, in 1930, furnished convincing proof that the gastrointestinal tract can be the seat of primary tuberculosis by ingestion of tubercle bacilli.⁴

Armstrong (1952) stated that abdominal tuberculosis may be said to comprise infection by *Mycobacterium tuberculosis* of (i) the intestinal tract (ii) the mesenteric lymph glands (iii) the peritoneum.⁵ Wig (1972) is of the opinion that it should include disease of the stomach, duodenum, small bowel, large bowel, intra-abdominal lymph nodes, liver, spleen, pancreas and the peritoneum.⁶

PATHOGENESIS

Mycobacterium tuberculosis is the pathogen in most cases, more common being *Mycobacterium bovis* in some parts of the world with no pasteurization of milk.⁸ *Mycobacterium avium intracellulare* has become a major pathogen in HIV patients. In the UK and USA, primary abdominal tuberculous infection by ingestion of infected milk from tuberculous cows (bovine infection) was quite common at one time. However, bovine tuberculosis is rare among Indians, because of the habit of boiling milk before consumption which renders it safe, even if infected.

TYPES

- Intestinal
- Ulcerative
- Hyperplastic, plastic form- whole intestine plastered
- Strictures

- Perforative
- Peritoneal
- Wet type: ascites – generalized or loculated
- Dry plastic: mesenteric thickening, caseous lymph nodes and fibrous adhesions
- Fibrotic fixed type: mass formation of omentum, matting of bowel loops
- Acute primary peritonitis
- Mesenteric involvement
- Mass
- Abscess
- Nodal
- Solid organ
- Liver, spleen, pancreas
- Localised abscess
- Multiple miliary form

MECHANISM

Tubercle bacilli reach the gastrointestinal system via; (i) hematogenous spread from the primary lung focus in childhood, with later reactivation; (ii) ingestion of bacilli in sputum from an active pulmonary focus; (iii) direct spread from adjacent organs or (iv) through lymph channels from infected nodes. In the intestinal type, bacilli in the depth of the mucosa cause inflammatory reaction and are carried to Peyer's patches which form tubercles and undergo necrosis. Submucosal tubercles enlarge causing endarteritis, oedema, sloughing, ulcer formation and thus stenosis.⁹ Then the inflammatory process in the submucosa reaches to the serosa via lymphatics; however, lymphatic obstruction of mesentery and bowel causes a thick fixed mass. Spread may occur to the gland in the mesenteric root to the para-aortic glands and on to the cisterna chyli and then to the thoracic duct resulting in miliary dissemination. Caseous glands may invade the peritoneum or may soften and breakdown forming a localised abscess. Sometimes intestinal obstruction may result from pressure of glands or kinking through adhesions and their healing is by fibrosis or calcification leading to "napkin ring strictures".⁹ In the peritoneal type, there is extension from caseating and breaking-down glands from

loculated areas of peritonitis by direct continuity, by lymphatic spread or by haematogenous spread. However, the subacute type may be a part of polyserositis; the peritoneal tubercles are larger, loops of intestine are inflamed and tumid. It may resolve or may become chronic. The chronic type of peritonitis is usually benign, but it may be caseating or ulcerative, ascitic and fibrotic.

CLINICAL FEATURES

Because of its varied presentation and its ability to mimic a variety of other abdominal conditions, a high index of suspicion is required¹⁰, mainly in individuals aged 25-44 years who are mostly affected.¹¹ The disease commonly presents insidiously with abdominal pain, fever, night sweats, weight loss, anorexia, nausea and vomiting, diarrhea or constipation.⁹ It may also present as an enterocutaneous fistula after bowel surgery, an umbilical abscess, a discharging sinus or as non-healing surgical wound. On examination, pallor, ascites, hepatomegaly or abdominal masses due to enlarged lymph nodes, adherent bowel loops or a cold abscess may be noted.¹² The classical doughy abdomen is considered non-specific.¹³ Common complications are obstruction, perforation, fistulae and malabsorption.⁹

DIAGNOSIS

The diagnosis of abdominal tuberculosis is indeed challenging. Even in highly endemic regions the accuracy of clinical diagnosis is 50%.¹¹ A clinical suspicion should lead to an investigative workup in which raised erythrocyte sedimentation rate, anemia and hypoalbuminaemia are commonly seen.¹² Erythrocyte sedimentation rate is a common hematology test which is a non-specific measure of inflammation. Sputum examination for acid-fast bacilli (AFB) using Ziehl-Neelson staining may provide indirect evidence of abdominal tuberculosis. The Mantoux test may be used as a screening test but is of limited value in endemic areas of high false-positive rates.¹⁴

Examination of ascitic fluid will have all characteristics of an inflammatory exudate. Tubercle bacilli can sometimes be demonstrated by direct smear examination of the centrifuged deposit but culture examination will reveal them more often. The ascitic fluid in tuberculosis is straw-coloured with protein >3g/dl, and a total cell count of 150-4000/ μ l, consisting predominantly of lymphocytes (>70%). The ascites to blood glucose ratio is less than 0.965 and the serum/ascites albumin gradient is less than 1.1g/dl.¹⁵ Adenosine deaminase (ADA) is an aminohydrolase that

converts adenosine to inosine and is thus involved in the catabolism of purine bases. ADA is increased in tuberculous ascitic fluid due to the stimulation of T-cells by mycobacterial antigens. Serum ADA level above 54 U/l, ascitic fluid ADA level above 36 U/l and a ascitic fluid to serum ADA ratio >0.985 were found suggestive of tuberculosis.¹⁵ Serological tests are based on detection of specific antibodies to mycobacterial tuberculosis. ELISA enables rapid diagnosis. The IgG component has high specificity for abdominal tuberculosis⁷, an optical density (OD) of 0.81 on ELISA and a fluorescent coefficient of 2.56 on soluble antigen fluorescent antibody (SAFA).¹⁶

Chest x-ray is an important first investigation due to the fact that active pulmonary lesions may be present in up to 60% of patients with abdominal disease.¹⁸ Evidence of tuberculosis in a chest x-ray supports the diagnosis but a normal chest x-ray does not rule it out. Plain x-ray of the abdomen shows enteroliths, features of obstruction and evidence of ascites; perforation or intussusception. In addition, there may be calcified lymph nodes, calcified granulomas and hepatosplenomegaly. Small-bowel barium meal shows features like accelerated intestinal transit, hypersegmentation of the barium column (chicken intestine), precipitation, flocculation and dilution of barium, stiffened and thickened folds, luminal stenosis with smooth but stiff contours (hour-glass stenosis); multiple strictures with segmental dilatation of bowel loops may also be found as well as fixity and matting of bowel loops.¹⁵ Barium enema shows (i) early involvement of the ileocaecal region manifesting as spasm and oedema of the ileocaecal valve. Thickening of the lips of the ileocaecal valve and/or wide gaping of the valve with narrowing of the terminal ileum (Fleischner or inverted umbrella sign). (ii) fold thickening and contour irregularity of the terminal ileum (iii) conical caecum, shrunken in size and pulled out of the iliac fossa due to contraction and fibrosis of the mesocolon (iv) loss of normal ileocaecal angle and dilated terminal ileum, appearing suspended from a retracted, fibrosed caecum (goose neck deformity) (v) purse string stenosis, localized stenosis opposite the ileocaecal valve with a rounded-off smooth caecum and a dilated terminal ileum (vi) Stierlin's sign which is a manifestation of acute inflammation superimposed on a chronically involved segment and is characterized by lack of barium retention in the inflamed segments of the ileum, caecum and a variable length of the ascending colon, with a normally configured column of barium on either side. It appears as a narrowing of the terminal ileum with rapid emptying into a shortened, rigid or

obliterated caecum and (vii) String sign which is a persistent narrow stream of barium indicating stenosis.^{15,19} Both Stierlin and String signs can also be seen in Crohn's disease and hence are not specific for tuberculosis.

Ultrasound is very useful for imaging peritoneal tuberculosis. Intra-abdominal fluid may be free or loculated, and clear or complex (with debris and septae); even fluid collections in the pelvis may have thick septa and can mimic ovarian cysts. The club-sandwich or sliced-bread sign, localized fluid between radially oriented bowel loops due to local exudation from the inflamed bowel (interloop ascitis), is visible. Lymphadenopathy may be discrete or conglomerated (matted) and the echotexture is mixed heterogenous, in contrast to the homogeneously hypoechoic nodes of lymphoma. Small discrete anechoic areas representing zones of caseation may be seen within the nodes. With treatment, the nodes show a transient increase in size for 3-4 weeks and then gradually reduce in size. Calcification in healing lesions is seen as discrete reflective lines. Both caseation and calcification are highly suggestive of a tubercular etiology, neither being common in malignancy-related lymphadenopathy. Bowel-wall thickening is best appreciated in the ileocaecal region and is uniform and concentric as opposed to the eccentric thickening at the mesenteric border found in Crohn's disease and the variegated appearance of malignancy. The pseudo-kidney sign is the involvement of the ileocaecal region which is pulled up to a subhepatic position.^{20,21} Ileocaecal tuberculosis is usually hyperplastic and well evaluated on CT scan. In early disease, there is slight symmetric circumferential thickening of caecum and terminal ileum. Later, the ileocaecal valve and adjacent medial wall of the caecum is asymmetrically thickened. In more advanced disease, gross wall thickening, adherent loops, large regional nodes and mesenteric thickening together can form a soft-tissue mass around the ileocaecal junction. Caseating lymph nodes are seen as having hypodense centers and peripheral rim enhancement. Mesenteric disease is seen on CT scan as a patchy or diffuse increase in density, strands within the mesentery, and a stellate appearance. A fibrous wall can cover the omentum, developing from long-standing inflammation, and is called omental line. An omental line is less common in malignant infiltration. Computed tomography (CT) enteroclysis is emerging as the gold standard radiographic test for diagnosing small-bowel disease.²² MRI depicts para-aortic, aortocaval and mesenteric nodes effectively. Macronodular tubercular liver abscess appears on MR imaging. CT scan can also pick up ulceration

or nodularity within the terminal ileum, along with narrowing and proximal dilatation. Other areas of small and large bowel involvement manifest as circumferential wall thickening, narrowing of the lumen and ulceration. In the colon, involvement around the hepatic flexure is common. Complications of perforation, abscess, and obstruction are also seen.²³

Upper GI endoscopy is used to detect oesophageal, stomach and duodenal tuberculosis. Endoscopic brush or needle biopsy is often diagnostic.²⁴ Recently, capsule endoscopy is used to record images through the digestive tract, the capsule camera is primarily used to visualize the small intestine.²⁵ Colonoscopy is an excellent tool to diagnose colonic and terminal ileal involvement but is still often under-utilized. Biopsies should be taken from the edge of the ulcers. A combination of histology and culture of the biopsy material can be expected to establish the diagnosis in over 60 per cent of cases.²⁴ Punch biopsy of peritoneum is a safe and valuable procedure for the diagnosis of abdominal tuberculosis; but is risky in the absence of fluid.²⁶ Genetic tests are rapid, sensitive, specific and inexpensive methods of diagnosis and results are available in few hours. TB-nested polymerase reaction has the ability to detect as little as 8 fg pf mycobacterial DNA or 1-2 bacilli. It is a sensitive and specific test to detect early TB.¹⁷

Paustian, in 1964, stated that one or more of the following four criteria must be fulfilled to diagnose abdominal tuberculosis,

- histological evidence of tubercles with caseation necrosis
- a good typical gross description of operative findings with biopsy of mesenteric nodes showing histologic evidence of tuberculosis
- animal inoculation or culture of suspected tissue resulting in growth of *M. tuberculosis*
- histological demonstration of acid-fast bacilli in a lesion.¹

Peritoneal biopsy can be obtained by blind needle biopsy, mini-laparotomy, laparotomy and laparoscopy. Laparoscopy is considered most appropriate because of its ability to visualize the peritoneal cavity in detail and take biopsies from the suspected lesions, at the same time being minimally invasive and less morbid.²⁷ The laparoscopic findings in

peritoneal tuberculosis can be grouped into 3 categories; thickened peritoneum with tubercles, thickened peritoneum without tubercles, and fibroadhesive peritonitis with markedly thickened peritoneum and multiple thick adhesions fixing the viscera.²⁸ Although other diagnostic methods of TB such as imaging, culture of ascites and polymerase chain reaction (PCR) are used today, laparoscopy with tissue biopsy provided rapid and correct diagnosis of abdominal tuberculosis.²⁸

The differential diagnosis of TB includes Crohn's disease, non-Hodgkin lymphoma, ulcerative colitis, amoebic colitis, disseminated carcinoma, chronic liver disease, sarcoidosis, peritoneal mesothelioma and appendicitis.^{19,23}

MANAGEMENT

Pathological diagnosis may not always be obtained. Hence, with given clinical, radiological and colonoscopy findings, a therapeutic trial may be given. All patients should receive conventional antitubercular therapy for at least 6 months including two initial months of rifampicin, isoniazid, pyrazinamide and ethambutol.²⁹ If there is recurrent disease, drug resistance or serious illness, the regimen is two initial months of rifampicin, isoniazid, pyrazinamide and ethambutol followed by 5 months of rifampicin, isoniazid and ethambutol. Some clinicians add oral corticosteroids in a dosage of 20 to 40mg. The assumption is that the late complications resulting from fibrosis and cicatrization are minimized. In cases of hepatocellular dysfunction, a combination of streptomycin and ethambutol is considered safe. Nearly 80% of patients respond to chemotherapy. Supportive measures like good nutrition and multi-vitamins help to correct deficiencies, which are often present. Concomitant administration of pyridoxin (vitamin B6) reduces the chances of INH neuro-toxicity.³⁰

Surgery is not recommended, either for confirming the diagnosis or as the first-line approach to the management of uncomplicated abdominal tuberculosis. However, patients with acute and sub-acute intestinal obstruction, who do not respond to conservative measures, must be treated surgically.³¹ Diseased segments of bowel with adequate free margins are removed, avoiding extensive resection. Surgery is also needed in patients with a free perforation or perforation associated with abscess formation.³² If ascites is present, it is evacuated and the abdomen is closed without leaving drains.³¹ If the clinical, radiographic and endoscopic data are consistent with the diagnosis of abdominal tuberculosis, and are adequate to rule out other common

diseases, e.g. cancer, non-specific inflammatory bowel disease and other specific infections, it is considered appropriate to give a trial of anti-tuberculosis chemotherapy.³³

Sometimes one may come across plastered intestine due to omental and bowel adhesions. In such cases, little more than exploration can be done and tissue for diagnosis is taken as extensive adhesiolysis may lead to postoperative fistulation leading to increased morbidity and mortality. Recurrent fistula in ano and abscesses are common in tropical countries and respond to antitubercular treatment.⁷

CONCLUSION

Abdominal tuberculosis is one of the commonest forms of extra-pulmonary tuberculosis, it is ill understood and is being neglected all too often by clinicians and researchers. There is resurgence of abdominal TB due to multidrug resistance and co-existing HIV/AIDS. As long as tuberculosis remains uncontrolled, abdominal tuberculosis will be commonly met with intestinal obstruction and intestinal perforation. Symptoms and signs are very variable and clinical diagnosis is almost impossible. A raised E.S.R. with symptoms, with or without a positive chest x-ray and with positive tuberculin test, is suggestive of abdominal tuberculosis. The convincing way to prove the diagnosis is to do bacteriological and histopathological examination of the biopsy specimens, or by genetic tests being more rapid and specific. Antimicrobial treatment is the same as for pulmonary tuberculosis and surgery is occasionally required. The first country to eliminate tuberculosis will be the one which regards the disease as a serious problem, right to the end - as recently the slogan for the World TB Day, March 24, 2008, was "I Am Stopping TB". It marked the start of a 2-year campaign that belongs to people everywhere who are doing their part to "Stop TB". It reflects the reality that every one of us health leaders, health workers, patients, families and community members has a role to play to stop TB.

References

1. Paustian FF. Tuberculosis of the intestine. In: Bockus HL, editor. *Gastroenterology*, vol.11, 2nd ed., Philadelphia W.B. Saunders Co.; 1964
2. Marshall JB. Tuberculosis of gastrointestinal tract and peritoneum. *Am J Gastrointest* 1993;88:989-99.
3. Rathi PM, Amarapurkar DN, Parikh SS, et al. Impact of human immunodeficiency virus infection on abdominal tuberculosis in western India. *J Clin Gastroenterol* 1997;24:43.
4. Pimparkar BD, Donde UM. Intestinal tuberculosis. Clinical and radiological studies. *J Assoc Phy India* 1974;22:205-218 and II *Gastrointestinal Absorption studies. J Assoc Phy India.* 1974;22:219-228.
5. Armstrong Basil W. Abdominal Tuberculosis. *Modern Practice in Tuberculosis* Ed. by T. Holmes Sellorand, J.H. Livingston, Butterworth & Co. (Publishers), 1952, Vol. II, pp.267-302.
6. Wig KL, Bawa YS. *Ind J Tuber* 1953;1:6.
7. Sunil Kumar et al. Abdominal tuberculosis. *Recent Advances in Surgery*, Vol. 28, p.49.
8. Collins CH, Grange JM. A review. The bovine tubercle bacillus. *J Appl Bacteriol* 1983;55:13-29.
9. Rangabashyam N, Anand BS, Om Prakash R. Abdominal TB. In: Morris PJ, Wood DWC, eds. *Oxford Textbook of Surgery*, Vol. 3, 2nd ed., pp.3237-3249.
10. Badaoui E, Berney T, Kaiser L. Surgical presentation of abdominal tuberculosis: a protean disease. *Hepatogastroenterology* 2000;47:751-755.
11. Sheer TA, Coyle WJ. Gastrointestinal tuberculosis. *Curr Gastroenterol Rep* 2003;5:273-278.
12. Al Muneef M, Mermish Z et al. Tuberculosis in the belly: a review of forty-six cases involving gastrointestinal tract and peritoneum. *Scand J Gastroenterol* 2001;36:528-532.
13. Sheer TA, Coyle WJ. Gastrointestinal tuberculosis. *Curr Gastroenterol Rep* 2003;5:273-278.
14. Hassan I, Brilakis ES, Thompson RL, Que FG. Surgical management of abdominal tuberculosis. *J Gastrointest Surg.* 2002;6:862-867.
15. Sharma MP, Vikram Bhatia. Abdominal tuberculosis. *Indian J Med Res* 2004;120:305-315.
16. Chawla TC, Sharma A, Kiran U, Bhargava DK, Tandon BN. Serodiagnosis of intestinal tuberculosis by enzyme immunoassay and soluble antigen fluorescent antibody tests using a saline extracted antigen. *Tubercle* 1986;67:55-60.
17. Chan CM et al. Single tube nested PCR in the diagnosis of tuberculosis. *J Clin Pathol* 1996;49:290-294.
18. Joy B, Gulshan B et al. Gastrointestinal tuberculosis: an eighteen patient experience. *J Clin Gastroenterol* 2000;30:397-402.
19. Akhan O, Pringot J. Imaging of abdominal tuberculosis. *Eur Radiol* 2002;12:312-323.
20. Batra A, Gulati MS, Sarma D, Paul SB. Sonographic appearances in abdominal tuberculosis. *J Clin Ultrasound* 2000;28:233-45.
21. Malik A, Saxena NC. Ultrasound in abdominal tuberculosis. *Abdom Imaging* 2003;28:574-9.
22. Suri S, Gupta S, Suri R. Computed tomography in abdominal tuberculosis. *Br J Radiol* 1999;72:92-8.
23. Buxi TBS, Sud Seema, Vohra Rakesh. CT and MRI in the diagnosis of tuberculosis. *Indian J Paediatr* 2002;69:965-972.
24. Leung VK, Tang WL, Cheung CH, Lai MS. Importance of ileoscopy during colonoscopy for the early diagnosis of ileal tuberculosis: report of two cases. *Gastrointestinal endoscopy* 2001;53(7):813-5.
25. Reddy DN, Sriram PV, Rao GV, Bhaskar Reddy D. Capsule endoscopy appearances of small-bowel tuberculosis. *Endoscopy* 2003;35:99.
26. Shukla HS, Naitrani YP et al. Peritoneal biopsy for diagnosis of abdominal tuberculosis. *Postgrad Med J* 1982;58(678):226-228.
27. McLaughlin S, Jones T, Pitcher M, Evans P. Laparoscopic diagnosis of abdominal tuberculosis. *Aust NZ J Surg* 1998;68:599-601.
28. Bhargava DK, Kushwaha AKS, Dasarathy S, Shrinivas, Chopra P. Endoscopic diagnosis of segmental colonic tuberculosis. *Gastrointest Endosc* 1992;38:571-4.
29. Balasubramanian R, Ramachandran R, Joseph PE, Nagarajan M, Thiruvengadam KV, Tripathy SP, et al. Interim results of a clinical study of abdominal tuberculosis.

Indian J Tuberc 1989;36:117-21.

30. Balasubramanian R, Nagarajan M, Balambal R, Tripathy SP, Sundararaman R, Venkatesan P. Randomised controlled clinical trial of short course chemotherapy in abdominal tuberculosis: a five-year report. *Int J Tuberc Lung Dis* 1997;1:44-51.

31. Sircar S, Taneja VA, Kansra U. Epidemiology and clinical presentation of abdominal tuberculosis - a retrospective study. *J Indian Med Assoc* 1996;94:342.

32. Schwartz SI. *Principles of Surgery*. McGraw Hill, 1984; pp.1152-1153.

33. Singh V, Jain AK, Agrawal AK, Gupta S, Khanna S, Khanna AK, Gupta JP. Clinicopathological profile of abdominal tuberculosis. *Br J Clin Pract* 1995;49:22-24.

34. Dhar P, Kulshrestha P. Fiberoptic examination of the colon: A review of 328 colonoscopies. *J Assoc Physicians India* 1988;36:214-215.

35. Oksoy G, Duren E, Uygun M. Gastrointestine tuberculosis: 10 years' experience in a surgical university clinic. *Chirurg* 1994; 65: 546.

36. Peda Veerraju H. Abdominal tuberculosis. In: Satya Sri S, editor. *Textbook of Pulmonary and Extrapulmonary Tuberculosis*. 3rd ed. New Delhi: Interprint; 1998; p.250-2.

37. Huang JH, Kao PN, Adi V, Ruoss SJ. *Mycobacterium avium-intracellulare* pulmonary infection in HIV-negative patients without preexisting lung disease. *Chest* 1999;115:1033-1040.

Author Information

Anil Sunkara, MBBS

Resident, Department of Surgery, Jawaharlal Nehru Medical College

D. D. Wagh, MS

Professor, Department of Surgery, Jawaharlal Nehru Medical College

Sameer Singhal, M.D

Professor, Department of Chest and Tuberculosis, Jawaharlal Nehru Medical College