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Uncal Herniation: Early Recognition and Emergency Management by Prehospital Care Providers-An Updated Review

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Abstract

Background: Uncal herniation is a rapidly progressive and potentially fatal neurological emergency caused by displacement of the medial temporal lobe through the tentorial notch as intracranial pressure rises. It may result from traumatic brain injury, intracranial hemorrhage, malignant cerebral edema, tumors, hydrocephalus, and other space-occupying intracranial lesions.

Aim: This article reviews the anatomical basis, causes, pathophysiology, clinical manifestations, diagnostic evaluation, treatment approaches, complications, and prognosis of uncal herniation.

Methods: A narrative review was undertaken using established neurocritical care concepts and published literature addressing transtentorial herniation, intracranial hypertension, clinical neurological assessment, neuroimaging, emergency management, and neurosurgical intervention.

Results: Uncal herniation develops when compensatory intracranial mechanisms fail and pressure gradients force the uncus against the tentorial edge. Compression of the ipsilateral oculomotor nerve can cause a dilated, poorly reactive pupil, while cerebral peduncle compression may produce hemiparesis. Progressive midbrain and brainstem involvement can lead to declining consciousness, abnormal posturing, respiratory failure, coma, and death. Urgent non-contrast computed tomography is essential for identifying hemorrhage, mass effect, cisternal compression, hydrocephalus, and midline shift. Immediate management includes airway protection, optimization of oxygenation and cerebral perfusion, osmotherapy, controlled ventilation when indicated, cerebrospinal fluid diversion in selected cases, and urgent evacuation or decompression of the causative lesion.

Conclusion: Uncal herniation requires rapid clinical recognition and coordinated emergency, critical care, radiological, and neurosurgical management. Early treatment is central to reducing mortality and limiting permanent neurological disability.

Key words: Uncal herniation, intracranial pressure, transtentorial herniation, traumatic brain injury, anisocoria, neurocritical care, emergency management.

Introduction

Abdominal tuberculosis represents a clinically important manifestation of extrapulmonary tuberculosis, characterized by infection involving one or more intra-abdominal structures. The disease may affect the gastrointestinal tract, peritoneum, solid visceral organs, mesenteric structures, and intra-abdominal lymph nodes, either as isolated sites of involvement or in various combinations. Although abdominal tuberculosis may coexist with active pulmonary tuberculosis, a considerable proportion of affected individuals present without demonstrable pulmonary disease, which may further complicate clinical recognition and diagnostic assessment. As a result, the absence of respiratory symptoms or radiological evidence of pulmonary tuberculosis should not exclude the possibility of abdominal involvement in patients presenting with compatible clinical manifestations. Globally, abdominal tuberculosis constitutes a relatively uncommon form of tuberculosis, accounting for approximately 1% to 3% of all reported tuberculosis cases. However, its epidemiological distribution varies substantially across countries and populations, largely reflecting

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regional differences in the prevalence of extrapulmonary tuberculosis and the overall burden of Mycobacterium tuberculosis infection [1]. The proportion of extrapulmonary tuberculosis represented by abdominal involvement also demonstrates marked geographical variation, with estimates ranging from approximately 13% of extrapulmonary tuberculosis cases in India to around 6% in the United States [2][3]. Such variation may be influenced by differences in disease prevalence, demographic characteristics, immune status, diagnostic capacity, healthcare accessibility, and the recognition and reporting of extrapulmonary manifestations.

The diagnosis of abdominal tuberculosis remains a significant clinical challenge because its manifestations are frequently nonspecific and may resemble a wide range of inflammatory, infectious, and malignant disorders. Patients may present with chronic abdominal pain, weight loss, fever, anorexia, altered bowel habits, abdominal distension, ascites, or symptoms related to intestinal obstruction. The considerable overlap between these manifestations and those associated with other gastrointestinal and intra-abdominal diseases often results in delayed diagnosis. Furthermore, laboratory investigations and radiological findings may provide supportive but non-definitive evidence, necessitating careful integration of clinical, microbiological, histopathological, endoscopic, and imaging findings to establish an accurate diagnosis. Despite these diagnostic challenges, abdominal tuberculosis generally demonstrates a favorable response to appropriate standard antituberculous pharmacotherapy when identified and treated at an early stage. Medical treatment remains the primary therapeutic approach, whereas surgical intervention is typically reserved for patients who develop significant complications, including intestinal obstruction, strictures, perforation, fistula formation, or other conditions that fail to respond adequately to conservative management. Therefore, maintaining a high level of clinical suspicion is essential, particularly among individuals from regions with a substantial tuberculosis burden or those with relevant clinical risk factors. Early recognition, timely initiation of antituberculous therapy, and coordinated management through an interprofessional healthcare approach are fundamental to minimizing diagnostic delay, preventing disease-related complications, and reducing associated morbidity and mortality [2][3].

Etiology

Abdominal tuberculosis is caused predominantly by members of the Mycobacterium tuberculosis complex, a group of closely related pathogenic mycobacterial species responsible for human and animal tuberculosis. Among these organisms, Mycobacterium tuberculosis remains the principal causative agent of abdominal tuberculosis in most populations, while Mycobacterium bovis may also contribute to disease, particularly in settings where the consumption of unpasteurized dairy products remains prevalent. The development of abdominal involvement reflects the ability of tuberculous bacilli to disseminate beyond the primary site of infection and establish infection within the gastrointestinal tract, peritoneum, abdominal lymphatic system, or solid intra-abdominal organs. The pathogenic mechanisms underlying abdominal tuberculosis are therefore heterogeneous and may differ according to the patient's initial site of infection, immune response, route of exposure, and epidemiological circumstances. Several routes of infection have been implicated in the pathogenesis of abdominal tuberculosis. One important mechanism involves the swallowing of infected sputum by individuals with active pulmonary tuberculosis. Following ingestion, viable Mycobacterium tuberculosis bacilli may reach the gastrointestinal tract and penetrate the intestinal mucosa, particularly in anatomically susceptible regions. Abdominal involvement may also develop through hematogenous dissemination from a distant tuberculous focus, allowing bacilli to reach abdominal organs through the systemic circulation. Similarly, lymphatic spread from infected lymph nodes can facilitate the extension of infection to mesenteric and intra-abdominal lymphatic structures. Direct extension from an adjacent infected organ or tissue represents another potential pathway, particularly when tuberculosis affects structures located in close anatomical proximity to the peritoneum or gastrointestinal tract. In addition, ingestion of dairy products contaminated with Mycobacterium bovis, especially raw or unpasteurized milk, has historically represented an important route of primary abdominal infection [4].

Based on the presumed mechanism of acquisition, some investigators have categorized abdominal tuberculosis into primary and secondary forms. Primary abdominal tuberculosis is generally associated with the direct ingestion of Mycobacterium bovis, resulting in infection of the gastrointestinal tract without an identifiable pulmonary focus. In contrast, secondary abdominal tuberculosis is more commonly associated with the dissemination or extension of infection originating from active pulmonary tuberculosis or another distant tuberculous site [5]. Although this classification provides a useful conceptual framework, the precise route of infection may not always be identifiable in clinical practice, particularly because pulmonary lesions may have healed or become clinically silent before abdominal manifestations develop. The terminal ileum, ileocecal region, and peritoneum represent the anatomical sites most frequently affected by abdominal tuberculosis. Their susceptibility is associated with several local anatomical and physiological characteristics. The relatively narrow intestinal lumen, prolonged

physiological stasis, reduced digestive activity, and substantial concentration of lymphoid tissue within these regions may collectively facilitate the contact, uptake, and persistence of tuberculous bacilli [5][6][7]. The abundance of lymphatic tissue, particularly within the ileocecal region, provides a favorable environment for the localization and multiplication of organisms after penetration of the intestinal mucosa. This predilection contributes to the frequent clinical presentation of abdominal tuberculosis with ileocecal inflammation, intestinal thickening, strictures, masses, or obstructive complications. Following gastrointestinal involvement, tuberculous bacilli may subsequently disseminate to solid abdominal organs through vascular pathways, including the portal and hepatic arterial circulations. This mechanism may result in infection of organs such as the liver, spleen, and pancreas. Hematogenous dissemination from distant tuberculous lesions and lymphatic spread may similarly establish infection within multiple abdominal sites. Furthermore, direct extension to the peritoneum may occur from infected gastrointestinal structures or from adjacent sites, including infected adnexal structures and paraspinal muscles [8]. The diversity of these pathogenic pathways explains the wide anatomical distribution and variable clinical manifestations of abdominal tuberculosis and highlights the importance of considering the disease as a systemic infectious process rather than an isolated gastrointestinal disorder [5][6][7][8].

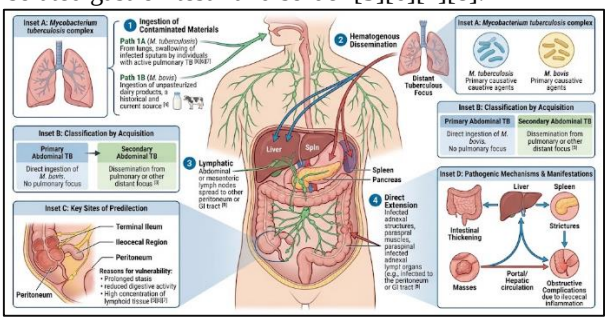

Fig. 1: Etiology of Abdominal Tuberculosis.

Epidemiology

Extrapulmonary tuberculosis represents an important component of the global tuberculosis burden and accounts for approximately 12% of all tuberculosis cases. Within the spectrum of extrapulmonary disease, abdominal tuberculosis constitutes an estimated 6% to 13% of cases, although its reported frequency varies across populations according to differences in tuberculosis prevalence, demographic characteristics, socioeconomic conditions, diagnostic practices, and access to healthcare services [9]. Abdominal tuberculosis may occur as an isolated extrapulmonary manifestation or in association with pulmonary disease. Estimates indicate that approximately 6% to 38% of individuals diagnosed with abdominal tuberculosis may have concomitant pulmonary tuberculosis, emphasizing the importance of evaluating patients for both pulmonary and extrapulmonary involvement during the diagnostic process [10][11]. The wide range reported across studies may reflect differences in study populations, disease definitions, diagnostic methods, and the prevalence of tuberculosis within individual geographic regions. The epidemiological distribution of abdominal tuberculosis is strongly influenced by socioeconomic and health-related factors. The disease occurs more frequently among populations experiencing lower socioeconomic conditions, where factors such as overcrowding, limited access to healthcare, nutritional deficiencies, delayed diagnosis, and increased exposure to tuberculosis may contribute to disease transmission and progression [3][9]. In addition to these environmental and socioeconomic determinants, several host-related conditions increase susceptibility to abdominal tuberculosis. Individuals living with human immunodeficiency virus are at increased risk because impaired cellular immunity reduces the body's capacity to contain Mycobacterium tuberculosis infection. Immunosuppressive therapy represents another important risk factor, particularly among patients receiving prolonged treatment that compromises cell-mediated immune responses [3][12].

Several chronic medical conditions have also been associated with an increased likelihood of abdominal tuberculosis. Female sex has been identified as a potential epidemiological risk factor, while end-stage renal disease may increase susceptibility through alterations in immune function and the underlying disease burden. Liver cirrhosis represents another recognized risk condition, with impaired immune responses and altered host defenses potentially contributing to increased vulnerability. Diabetes mellitus is also associated with an elevated risk of tuberculosis and may facilitate disease progression through impaired immune mechanisms [13][14][15]. The epidemiological profile of abdominal tuberculosis differs between industrialized countries and regions with a high tuberculosis burden. In industrialized settings, abdominal tuberculosis occurs predominantly among individuals with HIV-TB coinfection and among immigrants originating from countries where tuberculosis remains endemic. Within immigrant populations, reactivation of previously acquired latent tuberculosis is considered the most common mechanism responsible for disease development [16]. Consequently, demographic background, previous exposure to tuberculosis, immune status, comorbid conditions, and migration from high-endemic regions remain important considerations when assessing the epidemiological likelihood of abdominal tuberculosis.

Pathophysiology

Tuberculosis is an infectious disease capable of affecting virtually any organ system, and abdominal involvement represents one of the major manifestations of extrapulmonary tuberculosis. Abdominal tuberculosis can affect the entire gastrointestinal tract, extending anatomically from the oral cavity to the anal canal, as well as the peritoneum, mesenteric lymph nodes, and intra-abdominal solid organs. The pathophysiological process depends largely on the route through which Mycobacterium tuberculosis reaches the abdominal compartment. Primary abdominal tuberculosis may develop following ingestion of infected sputum in individuals with pulmonary disease or consumption of unpasteurized milk contaminated with Mycobacterium bovis. Secondary abdominal disease may develop through lymphatic dissemination or hematogenous spread from a distant focus of infection, or through direct contiguous extension from an infected structure within the abdominal cavity. Abdominal tuberculosis may therefore represent either progression of a primary infection or reactivation of a previously contained latent infection. The intestine and peritoneum constitute the most frequently affected anatomical compartments [17]. After entering the gastrointestinal tract, tubercle bacilli interact with the intestinal mucosa and underlying lymphoid tissues. The organisms may be taken up by macrophages and transported through regional lymphatic channels, producing localized granulomatous inflammation. The host's cellular immune response plays a central role in determining the subsequent course of infection. Activated macrophages and T lymphocytes attempt to contain the organisms through granuloma formation, while persistent infection may lead to caseous necrosis, mucosal ulceration, tissue destruction, fibrosis, and subsequent anatomical distortion. These processes account for the broad clinical spectrum of abdominal tuberculosis. Patients may experience prolonged fever, malaise, anorexia, weight loss, abdominal pain, abdominal distension, or changes in bowel habits. Because these manifestations are not specific to tuberculosis, the underlying pathological process may progress before the diagnosis is established. Tuberculosis of the oral cavity represents an uncommon manifestation of abdominal or gastrointestinal tuberculosis. The mandible, buccal mucosa, lips, gingiva, and tongue may become involved through direct inoculation or extension of infection. Lesions can present as ulcers, nodules, fissures, or granulomatous lesions and may be either painful or painless. Their clinical evolution may be acute or chronic, depending on the underlying mechanism and host response. The nonspecific appearance of oral lesions may result in diagnostic confusion with malignant, inflammatory, or other infectious conditions. Confirmation requires tissue assessment because clinical examination alone cannot reliably establish the diagnosis. Histopathological examination, acid-fast staining, polymerase chain reaction, and mycobacterial culture contribute to definitive identification of the causative organism [18].

Esophageal tuberculosis is also rare and most frequently develops through contiguous extension from tuberculous mediastinal lymphadenopathy. Primary infection of the esophagus is exceptionally uncommon because the anatomical and physiological characteristics of the esophagus provide substantial protection against direct infection. When esophageal involvement occurs, the inflammatory process can produce mucosal ulceration, nodular lesions, and progressive tissue infiltration. Progressive dysphagia represents a characteristic clinical manifestation and may closely resemble esophageal malignancy. This similarity creates an important diagnostic challenge, particularly in patients presenting with progressive swallowing difficulty, weight loss, and an esophageal mass or ulcerative lesion. Histological examination of biopsied tissue may demonstrate caseating or noncaseating granulomas. Acid-fast staining, PCR testing, and culture should be performed on biopsy specimens to establish microbiological confirmation and distinguish tuberculosis from malignant and other granulomatous disorders [19]. Gastric and gastroduodenal tuberculosis occur infrequently. Several physiological mechanisms may contribute to the relative resistance of these regions to tuberculous infection. Gastric acidity may impair the survival of ingested bacilli, while the stomach contains comparatively limited lymphoid tissue and demonstrates rapid movement of its contents. When infection develops, however, the inflammatory process may produce mucosal ulceration, hypertrophic changes, and progressive tissue fibrosis. Patients may present with anorexia, weight loss, abdominal discomfort, vomiting, or symptoms associated with gastric outlet obstruction. Endoscopic assessment may reveal ulcerative or hypertrophic lesions, while progressive fibrosis and scarring may eventually result in pyloric stenosis. Involvement of regional lymph nodes can also produce extrinsic compression of the duodenum, generating obstructive symptoms without direct primary mucosal disease [15][8].

The small intestine and large intestine are among the most clinically important sites of gastrointestinal tuberculosis. The terminal ileum and ileocecal region demonstrate particular susceptibility because of their abundant lymphoid tissue and relatively slow intestinal transit. These characteristics may prolong exposure of the mucosa to tuberculous organisms and facilitate bacterial uptake and lymphatic dissemination. The resulting inflammatory response can produce several distinct morphological patterns. The ulcerative form represents the most frequently described pattern and is characterized by superficial transverse ulcers, particularly within the

small intestine. The hypertrophic form develops when a marked proliferative inflammatory response surrounds an ulcerated area, producing a thickened inflammatory mass and occurring more frequently in the cecal region. Some patients demonstrate a combined ulcero-hypertrophic pattern in which mucosal ulceration and tissue proliferation coexist. A further pathological outcome involves progressive fibrosis and formation of fibrous strictures. These strictures narrow the intestinal lumen and may ultimately produce partial or complete intestinal obstruction [11][20]. The pathological progression of intestinal tuberculosis explains many of its major complications. Persistent inflammation can result in transmural involvement, adhesions, fibrosis, luminal narrowing, and obstruction. In advanced disease, tissue destruction may progress to intestinal perforation, although this complication occurs less frequently. The clinical and pathological resemblance between intestinal tuberculosis and Crohn's disease represents a major diagnostic problem. Both conditions can produce abdominal pain, altered bowel habits, weight loss, ulceration, strictures, bowel-wall thickening, lymphadenopathy, and granulomatous inflammation. Radiological, endoscopic, and histological findings may therefore overlap substantially. Establishing the diagnosis requires integration of clinical and epidemiological information with endoscopic tissue sampling and microbiological investigation. Demonstration of mycobacteria through appropriate staining, PCR, or culture can provide critical evidence for tuberculosis [8][15][21]. Tuberculosis involving the rectum and anal canal is uncommon. When present, the disease may produce mucosal ulceration, hypertrophic lesions, abscess formation, or fibrotic strictures. Hematochezia is among the more frequently reported manifestations and may prompt evaluation for inflammatory bowel disease, malignancy, anorectal infection, or other causes of lower gastrointestinal bleeding. The pathological process reflects granulomatous inflammation and tissue destruction resulting from local infection or extension from adjacent structures. Because the clinical presentation is nonspecific, microbiological and histopathological confirmation remains essential. Identification of mycobacteria through tissue histology, PCR, or culture supports the diagnosis and helps distinguish tuberculosis from other inflammatory or infectious disorders affecting the anorectal region [22][3].

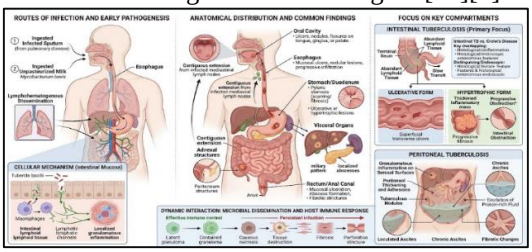

Fig. 2: Pathophysiology of Abdominal Tuberculosis.

Abdominal tuberculosis can also involve visceral organs, including the liver, gallbladder, spleen, pancreas, kidneys, and adnexal structures. Infection of these organs may develop through lymphohematogenous dissemination or direct contiguous extension from neighboring infected tissues. Hematogenous dissemination is particularly relevant in disseminated tuberculosis, where bacilli reach multiple organs through the bloodstream and establish granulomatous lesions. Direct extension can occur when infection spreads across anatomical boundaries between adjacent intra-abdominal structures. The clinical manifestations of visceral tuberculosis are often nonspecific and may include persistent fever, malaise, weight loss, abdominal pain, jaundice, and ascites. This broad symptom profile may delay recognition, particularly when no obvious pulmonary focus is present. Imaging may demonstrate multiple small miliary nodules, reflecting disseminated disease. In some cases, however, tuberculosis produces a localized lesion or tuberculous abscess within an individual organ rather than a diffuse miliary pattern [20][23]. Peritoneal tuberculosis represents another important manifestation and frequently occurs alongside intestinal or lymphatic abdominal disease. It may develop following rupture of necrotic mesenteric lymph nodes, direct extension from an infected abdominal organ, or reactivation of latent tuberculosis within the peritoneal compartment. Reactivation of previously latent infection represents the most common proposed mechanism for peritoneal tuberculosis. Once the peritoneum becomes infected, granulomatous inflammation develops along the serosal surfaces and may produce exudation of protein-rich fluid, peritoneal thickening, adhesions, and formation of tuberculous nodules. Chronic ascites without an alternative explanation such as advanced liver disease represents the most frequent clinical manifestation [24]. The character of tuberculous ascites can vary considerably according to the extent and stage of peritoneal involvement. Fluid may remain free within the abdominal cavity, become loculated through the formation of adhesions, or coexist with fibrotic changes affecting the peritoneal surfaces. Persistent inflammation can cause intestinal loops to adhere to one another or to the abdominal wall, producing mechanical restriction and contributing to intestinal obstruction. Peritoneal nodules may also develop as manifestations of granulomatous infection and can resemble peritoneal metastatic deposits on imaging or during surgical exploration [23]. These pathological similarities explain why peritoneal tuberculosis can mimic intra-abdominal malignancy and other chronic inflammatory disorders. Overall, the pathophysiology of abdominal tuberculosis reflects a dynamic interaction between microbial dissemination and the host's immune response. The route of infection

determines the initial anatomical site, while lymphatic, hematogenous, and contiguous spread can subsequently produce multifocal disease. Granuloma formation, caseous necrosis, ulceration, fibrosis, adhesions, strictures, and tissue destruction represent major pathological processes that shape the clinical presentation. The resulting disease may range from localized mucosal lesions to extensive intestinal, peritoneal, lymphatic, and visceral involvement. Understanding these mechanisms is essential for interpreting the diverse clinical and radiological manifestations of abdominal tuberculosis and for distinguishing it from diseases with overlapping presentations, including Crohn's disease, gastrointestinal malignancies, and other chronic granulomatous disorders.

Histopathology

Histopathological examination plays a central role in the diagnosis of gastrointestinal tuberculosis, particularly in patients with nonspecific clinical manifestations and inconclusive radiological findings. Tissue specimens obtained through endoscopic biopsy or surgical sampling may demonstrate a characteristic granulomatous inflammatory response within the mucosa, submucosa, or deeper intestinal layers. The principal histological findings include caseating and noncaseating granulomas, chronic cellular inflammation, and lymphatic hyperplasia. Caseating granulomas reflect organized collections of epithelioid histiocytes surrounding areas of central necrosis and represent an important pathological feature supporting tuberculous infection. However, noncaseating granulomas may also occur and therefore cannot independently establish the diagnosis. The detection of acid-fast bacilli using Ziehl-Neelsen staining is influenced by the experience and persistence of the examining pathologist because gastrointestinal tuberculosis is frequently paucibacillary, resulting in a relatively low organism burden within tissue specimens [25]. The histological appearance of abdominal tuberculosis may closely resemble Crohn's disease, creating a significant diagnostic challenge [25]. Nevertheless, certain pathological findings provide stronger support for tuberculosis, particularly caseating granulomas, confluent granulomas, and ulcers containing a prominent histiocytic lining. The identification of granulomatous inflammation within intra-abdominal lymph nodes in the absence of significant intestinal inflammatory changes may also favor tuberculosis over primary inflammatory bowel disease [17]. Accordingly, histopathological interpretation should always be integrated with clinical, radiological, epidemiological, and microbiological findings rather than considered in isolation. Hepatic involvement may similarly demonstrate caseating or noncaseating granulomas. However, granulomatous hepatitis has an extensive differential diagnosis that includes infectious, inflammatory, autoimmune, and drug-related conditions [15]. Consequently, hepatic granulomas alone are insufficient to confirm tuberculosis. Acid-fast bacilli staining may provide direct evidence of mycobacterial infection, but its sensitivity remains relatively low because of the paucibacillary nature of the disease. Moreover, staining performance is partly dependent on the operator's experience and the thoroughness of microscopic examination. Therefore, when tuberculosis is suspected, histopathology should be complemented by microbiological culture and molecular methods such as PCR whenever adequate tissue is available.

History and Physical

Abdominal tuberculosis may develop without any previous personal or family history of tuberculosis or a clearly identified exposure. Therefore, the absence of recognized risk factors should not exclude the diagnosis, and a high clinical index of suspicion is often required for timely recognition. In some patients, abdominal involvement may occur concurrently with active pulmonary tuberculosis, although this association is not consistently present [15]. The clinical presentation is frequently nonspecific and develops gradually, contributing to delayed diagnosis. Common constitutional manifestations include fever, malaise, anorexia, fatigue, and progressive weight loss. Gastrointestinal symptoms may include epigastric or generalized abdominal pain, nausea, vomiting, abdominal distension, and changes in bowel habits. The clinical picture varies considerably depending on the organs and anatomical sites involved, as abdominal tuberculosis may affect the intestine, peritoneum, lymph nodes, liver, or multiple sites simultaneously. The typically indolent course may further obscure the diagnosis because symptoms can persist for weeks or months before becoming sufficiently severe to prompt extensive investigation [3][26]. Physical examination findings are similarly variable and depend on the extent and anatomical distribution of disease. Abdominal tenderness, ascites, hepatosplenomegaly, jaundice, abdominal distension, or a palpable abdominal mass may be identified. Intestinal involvement can produce obstructive manifestations resulting from inflammatory strictures, extrinsic compression caused by adjacent inflammatory processes, or enlarged intra-abdominal lymph nodes [17]. In some cases, fever of unknown origin may represent the principal or only clinical manifestation, making diagnosis particularly challenging. Importantly, no individual symptom, physical finding, or clinical presentation is pathognomonic for abdominal tuberculosis. Consequently, clinical assessment should be integrated with appropriate laboratory investigations, imaging studies, microbiological testing, and histopathological examination to establish the diagnosis and distinguish abdominal tuberculosis

from other infectious, inflammatory, and malignant conditions.

Evaluation

Laboratory evaluation is an important component of the assessment of suspected abdominal tuberculosis and should be interpreted together with the clinical presentation, imaging findings, microbiological investigations, and histopathological results. Patients with symptomatic abdominal tuberculosis commonly demonstrate nonspecific laboratory abnormalities that reflect chronic inflammation, nutritional impairment, or systemic disease. Anemia and hypoalbuminemia are frequently observed, while inflammatory markers such as C-reactive protein (CRP) may be elevated [27]. These abnormalities are not specific for tuberculosis but can provide supportive evidence in patients presenting with prolonged constitutional and gastrointestinal symptoms. CRP, erythrocyte sedimentation rate (ESR), and fecal calprotectin may also have value in monitoring the inflammatory response and assessing improvement during anti-tuberculosis treatment (ATT), although they should not be used as isolated indicators of treatment response [28]. In patients with ascites, diagnostic evaluation of the ascitic fluid can provide important supportive information. Tuberculous peritonitis typically produces an exudative fluid with a relatively high protein concentration and a predominance of lymphocytes. Ascitic fluid examination should include appropriate biochemical, cytological, microbiological, and, when indicated, molecular investigations. Cytological and culture studies are particularly useful in excluding alternative causes of peritonitis, including malignant and other infectious conditions. Chest radiography should also be performed as part of the overall evaluation because pulmonary tuberculosis may coexist with abdominal disease or provide evidence supporting the diagnosis.

Mycobacterium-Specific Testing

Specific microbiological testing is essential when abdominal tuberculosis is suspected, but its diagnostic performance is affected by the paucibacillary nature of the disease. Because the organism burden in gastrointestinal and peritoneal specimens is often low, conventional acid-fast bacilli (AFB) staining and mycobacterial culture have relatively limited sensitivity, although a positive result is highly specific [29]. Polymerase chain reaction (PCR)-based methods may provide greater sensitivity and can detect *Mycobacterium tuberculosis* DNA in clinical specimens [30]. Therefore, molecular testing may serve as an important adjunct to conventional microbiological and histopathological assessment. Tuberculin skin testing and interferon-gamma release assays (IGRAs) may support evidence of previous exposure to *M. tuberculosis*, but neither test reliably establishes active abdominal tuberculosis. False-negative results may occur, particularly in patients with advanced disease, immunosuppression, or other conditions affecting cellular immunity [31]. Consequently, negative tuberculin or IGRA results should not be interpreted as evidence against active abdominal tuberculosis when clinical suspicion remains high. Measurement of adenosine deaminase (ADA) in peritoneal fluid is particularly useful in suspected tuberculous peritonitis. Elevated ADA concentrations can provide strong supportive evidence for peritoneal tuberculosis and should be interpreted in conjunction with the patient's clinical and laboratory findings [32][17]. In contrast, the diagnostic role of IGRA performed directly on peritoneal fluid has not been adequately established and is therefore not considered a definitive diagnostic approach [17].

PCR testing for *M. tuberculosis* in clinical specimens may be used as an adjunct to the initial diagnosis but is not appropriate for monitoring treatment response. This limitation results from the inability of PCR to reliably distinguish viable organisms from nonviable bacterial DNA. Consequently, PCR may remain positive after successful completion of anti-tuberculosis therapy [33]. Molecular testing may nevertheless be particularly useful when distinguishing intestinal tuberculosis from Crohn's disease, especially when conventional cultures are negative [34]. Multiplex PCR assays capable of detecting multiple *M. tuberculosis*-specific genetic targets may provide improved diagnostic performance and may also contribute to differentiation between tuberculosis and Crohn's disease [35][36]. Rapid molecular diagnostic methods are increasingly important in the contemporary evaluation of tuberculosis. The World Health Organization (WHO) recommends nucleic acid amplification testing for the diagnosis of tuberculosis, with certain automated PCR-based platforms capable of providing results within approximately two hours. These assays can simultaneously identify *M. tuberculosis* and detect genetic markers associated with rifampicin resistance. Detection of rifampicin resistance is clinically important because it may indicate the presence of multidrug-resistant tuberculosis and should prompt further drug-susceptibility testing and appropriate treatment planning.

Imaging Studies

Imaging plays a central role in evaluating suspected abdominal tuberculosis because it can demonstrate the anatomical distribution, extent, and complications of disease and can identify suitable sites for tissue or fluid sampling. No imaging finding is completely specific for tuberculosis, and radiological appearances should therefore be interpreted alongside microbiological and histopathological evidence.

Computed tomography scan

Computed tomography (CT) is considered the principal imaging modality for evaluating the extent and anatomical pattern of abdominal tuberculosis [37]. It provides detailed assessment of the bowel, mesentery, peritoneum, lymph nodes, and solid abdominal organs. Intestinal tuberculosis commonly involves the ileocecal region and may appear as asymmetric circumferential wall thickening involving the terminal ileum, cecum, or ileocecal valve. Necrotic or centrally hypoattenuating lymph nodes may accompany intestinal disease and can provide an important radiological clue. Chronic inflammation may result in fibrosis and stenosis, producing a contracted, irregular cecum and potentially contributing to intestinal obstruction [38]. Solid-organ involvement may be demonstrated by multiple small hypoattenuating nodules distributed over the organ surface or within the parenchyma. Hepatic and splenic involvement may occur as part of disseminated abdominal disease. CT may also demonstrate characteristic abnormalities associated with tuberculous peritonitis. High protein and cellular content within peritoneal fluid may produce relatively increased attenuation compared with simple fluid. The plastic or fibrous form of peritoneal tuberculosis may be characterized by thickening of the peritoneal and mesenteric surfaces, associated with loculated ascites. The nodular or dry form may demonstrate irregular or nodular mesenteric thickening and extensive fibrous adhesions, potentially producing an appearance resembling peritoneal carcinomatosis [39]. CT also has an important procedural role because CT-guided aspiration or biopsy can be performed when an accessible lesion, lymph node, or fluid collection is identified. Obtaining tissue or fluid enables microbiological, molecular, and histopathological examination and may substantially improve diagnostic certainty.

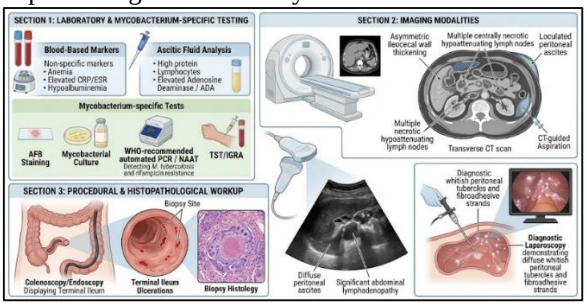

Fig. 3: Diagnostic Evaluation of Abdominal Tuberculosis.

Ultrasound

Ultrasonography is frequently used as an initial imaging investigation because it is widely available, does not expose patients to ionizing radiation, and can provide rapid assessment of abdominal structures. It may demonstrate ascites, bowel-wall thickening, peritoneal abnormalities, enlarged lymph nodes, and other features suggestive of abdominal tuberculosis. Ultrasound is particularly useful for identifying and characterizing free or loculated abdominal fluid and can guide diagnostic aspiration when required. Ultrasound-guided aspiration and assistance during laparoscopic biopsy may facilitate the acquisition of appropriate specimens for microbiological and pathological analysis [40][41]. However, its diagnostic accuracy is operator-dependent, and CT is generally more comprehensive for assessing the full extent of intra-abdominal disease.

Laparoscopic Evaluation

Diagnostic laparoscopy provides direct visualization of the peritoneal cavity and allows targeted tissue sampling when noninvasive investigations remain inconclusive. Typical laparoscopic findings suggestive of peritoneal tuberculosis include diffuse or focal peritoneal thickening, multiple small light-yellow or whitish tubercles, and fibroadhesive strands involving the peritoneal and visceral surfaces. Laparoscopy may therefore provide both visual evidence and tissue specimens for histopathological and microbiological confirmation [17]. Despite these advantages, the procedure is invasive and carries potential complications, including bleeding, visceral injury, and bowel perforation. Its use should therefore be considered according to the clinical condition and the diagnostic information obtained from less invasive investigations.

Gastroenterology Procedures

Endoscopic evaluation, particularly colonoscopy, has an important role in suspected intestinal tuberculosis. Colonoscopy may occasionally identify asymptomatic disease when performed for other clinical indications [42]. The ileocecal region is one of the most frequently affected intestinal sites, making careful inspection of the terminal ileum, ileocecal valve, and cecum particularly important. Endoscopic abnormalities may include ulceration, mucosal irregularity, edema, narrowing, and other inflammatory changes. Although these findings are not pathognomonic, their distribution and morphology may assist in differentiating intestinal tuberculosis from Crohn's disease [17]. Multiple biopsies should be obtained from abnormal areas whenever intestinal tuberculosis is suspected. Histopathological examination can identify granulomatous inflammation, while microbiological culture and molecular testing may provide additional evidence of infection. Colonoscopic biopsies have been reported to achieve diagnostic accuracy of up to approximately 80%, although diagnostic yield varies according to sampling technique, disease distribution, and the presence of bacilli within the tissue [43]. Obtaining multiple tissue specimens during colonoscopy may increase the yield of mycobacterial culture and improve the overall diagnostic assessment.

When conventional diagnostic investigations remain inconclusive despite a high clinical suspicion of abdominal tuberculosis, an empiric therapeutic

trial of anti-tuberculosis treatment may occasionally be considered. Clinical improvement following ATT has historically been proposed as supportive evidence for the diagnosis [28][44]. However, therapeutic response should be interpreted cautiously because symptomatic improvement is not specific to tuberculosis and may occur in other inflammatory or infectious gastrointestinal disorders. Reported diagnostic accuracy of therapeutic trials has varied considerably among studies, with estimates ranging from approximately 16% to 29% [45][29]. Although improvement may become clinically apparent within approximately two weeks in responsive patients, the timing and magnitude of response can vary according to disease severity, anatomical involvement, nutritional status, and treatment adherence. Therefore, an empiric therapeutic trial should not replace appropriate microbiological, molecular, histopathological, and radiological evaluation whenever these investigations are feasible. A structured diagnostic approach is preferable because establishing microbiological or pathological confirmation can prevent inappropriate treatment and is particularly important when the differential diagnosis includes Crohn's disease, malignancy, or other chronic intra-abdominal infections.

Treatment / Management

The primary treatment for abdominal tuberculosis is appropriate anti-tuberculosis therapy (ATT), with the regimen selected according to the site and extent of disease, drug susceptibility, and the patient's clinical condition. For drug-susceptible disease, the standard initial regimen consists of four first-line agents: isoniazid, rifampicin, pyrazinamide, and ethambutol. These drugs are administered during the intensive phase, traditionally for the first two months, followed by a continuation phase consisting of isoniazid and rifampicin for an additional four months [46]. Thus, a total treatment duration of six months is generally recommended for uncomplicated luminal intestinal tuberculosis. Healing of intestinal ulceration may begin relatively early, with endoscopic evidence of mucosal improvement sometimes observed by the end of the initial two-month phase [44]. Most contemporary treatment guidelines support a six-month treatment course for luminal abdominal tuberculosis. Prospective randomized controlled studies and systematic review evidence have demonstrated favorable cure rates with six months of therapy compared with longer nine-month regimens, while the shorter course offers potential advantages in terms of lower treatment costs and improved adherence [47][48]. Nevertheless, treatment duration should be individualized. Prolonged therapy may be considered in patients with disseminated tuberculosis, extensive disease, slow clinical response, complications, or other factors that may increase the risk of treatment failure or relapse [49][50]. Management should therefore be guided by the anatomical extent of infection, microbiological findings, drug susceptibility, nutritional status, comorbidities, and clinical response. Consultation with an infectious disease specialist may be appropriate, particularly in complex or drug-resistant cases. Clinical and radiological improvement generally accompanies effective treatment, and intestinal ulcers may heal substantially following ATT. However, structural abnormalities caused by chronic inflammation and fibrosis may persist despite microbiological control. Strictures, polyps, and hypertrophic lesions may remain after treatment, and these abnormalities can continue to produce gastrointestinal symptoms [44]. Importantly, intestinal obstruction may occasionally worsen during therapy as inflammation resolves and fibrotic scar tissue develops, resulting in further luminal narrowing [51]. Therefore, patients with pre-existing strictures require careful clinical monitoring during treatment. The routine use of adjunctive corticosteroids in abdominal tuberculosis has not been clearly established, and their role remains uncertain [17].

Endoscopic treatment may be considered in selected patients who develop symptomatic intestinal strictures despite appropriate ATT. Endoscopic balloon dilatation has been used particularly for accessible ileal and duodenal strictures and may provide a minimally invasive alternative to surgery in carefully selected patients [52][53]. The decision to perform dilatation should consider the length, severity, and anatomical location of the stricture, as well as the presence of active inflammation, ulceration, perforation, or other complications. Endoscopic intervention requires appropriate expertise because dilatation of severely diseased bowel carries a risk of perforation and bleeding. Surgical intervention is generally reserved for patients who develop complications or structural disease that cannot be adequately managed with medical or endoscopic therapy. Important indications include intestinal obstruction, perforation, fistula formation, severe or persistent strictures, and complications associated with extensive intestinal involvement [54]. Surgery should ideally be performed in conjunction with effective ATT to control active infection and reduce the risk of recurrent disease. Surgical approaches can broadly be divided into bypass procedures, radical resection, and conservative procedures. Bypass operations, such as entero-enterostomy or ileotransverse colostomy, are generally avoided when possible because they may be associated with blind-loop syndrome, fistula formation, persistent or recurrent tuberculosis, and disease involving the remaining bowel segments. Radical resection involves removal of the affected intestinal segment followed, when feasible, by primary anastomosis. This approach is particularly relevant in tuberculous bowel perforation and can be

combined with appropriate ATT to control residual infection. However, extensive surgery may be challenging because many patients with abdominal tuberculosis have significant malnutrition and impaired nutritional reserves, which can increase perioperative morbidity and compromise postoperative recovery.

Conservative surgical procedures may provide an alternative when preservation of bowel length is desirable. Strictureplasty, for example, may be considered for selected strictures causing substantial luminal compromise, particularly when multiple strictures are present and extensive resection could result in significant loss of functional intestine. Surgery may also become necessary when a clinically significant stricture persists despite adequate ATT or when multiple strictures are unlikely to resolve with medical therapy alone [46][47][55]. Overall, management should follow a multidisciplinary approach that integrates medical treatment, nutritional optimization, endoscopic intervention when appropriate, and carefully selected surgical procedures according to disease severity and complications.

Differential Diagnosis

Abdominal tuberculosis is frequently described as a "great mimicker" because its clinical, radiological, endoscopic, and histopathological manifestations can closely resemble a wide range of gastrointestinal, hepatobiliary, inflammatory, and malignant disorders. The differential diagnosis should therefore remain broad, particularly in patients presenting with chronic abdominal pain, constitutional symptoms, weight loss, ascites, bowel abnormalities, or unexplained intra-abdominal lesions. Abdominal tuberculosis has been reported to resemble esophageal and gastric malignancies, esophageal and gastric ulceration, colorectal cancer, hepatic abscesses, hepatic granulomatosis, and other intra-abdominal malignancies [56][57]. Depending on the anatomical site and acuity of presentation, it may also be clinically difficult to distinguish from spontaneous bacterial peritonitis, acute appendicitis, colitis, acute cholecystitis, and even necrotizing fasciitis [58][59][60]. Other infectious diseases should also be considered because several infections can produce caseating granulomatous inflammation. Conversely, noncaseating granulomas may occur in conditions such as sarcoidosis and berylliosis, further complicating histopathological interpretation [3]. Imaging findings may also be misleading because tuberculous lesions can demonstrate enhancement following administration of CT contrast media and increased metabolic activity on positron emission tomography, potentially producing appearances similar to malignancy [61].

Differentiation from Crohn's Disease

One of the most clinically important differential diagnoses is Crohn's disease, particularly in patients with intestinal involvement. Intestinal tuberculosis and Crohn's disease share numerous clinical, radiological, endoscopic, and histological characteristics because both can produce chronic granulomatous inflammation, ulceration, bowel-wall thickening, strictures, and lymphadenopathy [62]. Consequently, distinguishing between the two conditions can be challenging, and diagnostic errors or misclassification have been reported [63]. This distinction is particularly important because the treatment approaches are fundamentally different. Immunosuppressive or immune-modulating therapy used for Crohn's disease may be inappropriate or potentially harmful in patients with active tuberculosis. Tuberculin skin testing and interferon-gamma release assays have limited value in definitively distinguishing intestinal tuberculosis from Crohn's disease. Although several clinical, endoscopic, radiological, and histological criteria have been proposed, substantial overlap remains between the two disorders, limiting the reliability of any single feature [21]. Certain findings may favor tuberculosis, including caseating or confluent granulomas, involvement of regional lymph nodes, and characteristic ileocecal disease. However, the absence of these findings does not exclude tuberculosis. Microbiological confirmation remains particularly important when the distinction is uncertain. Multiple biopsies should be obtained from affected intestinal areas to maximize the likelihood of detecting *Mycobacterium tuberculosis*. AFB staining has low sensitivity in abdominal tuberculosis because the disease is frequently paucibacillary, and therefore a negative smear cannot reliably exclude infection. Culture sensitivity has been reported to range from approximately 7% to 79%, while PCR sensitivity varies according to the molecular targets and assay methodology used [30]. Multiplex PCR assays may improve diagnostic sensitivity while simultaneously identifying genetic markers associated with drug resistance. When definitive microbiological or histopathological confirmation cannot be achieved, an appropriately supervised therapeutic trial of anti-tuberculosis treatment may occasionally be considered. However, empiric initiation of immune-modulating therapy for presumed Crohn's disease should be avoided until tuberculosis has been reasonably excluded. This precaution is particularly important in regions with high tuberculosis endemicity, where abdominal tuberculosis should be carefully excluded before initiating immunosuppressive treatment for suspected Crohn's disease [21].

Conclusion:

Abdominal tuberculosis remains a clinically challenging condition because of its diverse anatomical involvement, nonspecific manifestations, and considerable overlap with inflammatory, infectious, and malignant gastrointestinal disorders. The disease may develop without an identifiable history of tuberculosis and often follows an indolent

course, resulting in delayed recognition. A high index of clinical suspicion is therefore essential, particularly in patients with persistent abdominal symptoms, constitutional manifestations, unexplained ascites, bowel abnormalities, or intra-abdominal lymphadenopathy. Accurate diagnosis requires a multidisciplinary approach integrating clinical assessment with laboratory investigations, imaging, endoscopy, histopathology, microbiological culture, and molecular testing. CT provides valuable information regarding disease extent and complications, whereas tissue and fluid sampling can establish microbiological or pathological evidence. Particular attention is required when differentiating abdominal tuberculosis from Crohn's disease because of substantial diagnostic overlap and important differences in treatment. Standard multidrug anti-tuberculosis therapy is highly effective in most uncomplicated cases, while endoscopic or surgical intervention may be required for persistent strictures, obstruction, perforation, or fistula formation. Early diagnosis and individualized management remain essential to reduce complications, prevent unnecessary interventions, and improve clinical outcomes.

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