A DETAILED REVIEW OF TUBERCULOSIS

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Abstract:

Mycobacterium tuberculosis complex organisms are the cause of the airborne infectious disease known as tuberculosis (TB). Although M. tuberculosis is primarily a pulmonary pathogen, it can infect practically any area of the body and lead to disease. M. tuberculosis infection can progress from a state of containment in the host, where the bacteria are isolated within granulomas (latent TB infection), to a contagious state, in which the patient will exhibit symptoms such as coughing up blood, having a fever, sweating excessively at night, and losing weight. Contagious pulmonary TB is the only active kind. Drug-resistant TB is a major concern in many contexts, and it continues to be a leading cause of morbidity and mortality in many low- and middle-income nations. Although several innovative TB diagnostics, such as quick molecular testing, have been created, there is still a demand for more straightforward point-of-care tests. Several antimicrobials must typically be taken for a long period throughout treatment, prompting efforts to create shorter medication regimens. The Bacillus Calmette-Guérin (BCG) vaccine is widely used, mostly to protect newborns and young children from TB that can be fatal, but it has not been successful in containing the global TB epidemic. Thus, attempts are being made to create novel vaccinations with greater efficacy. This review article was created after a thorough analysis of the literature to better understand and raise awareness about tuberculosis. This is a real effort to assist tuberculosis to be eradicated from the pit lanet in the not-too-distant future.

Keywords: Mycobacterium tuberculosis, Tuberculosis, Active Tuberculosis, Latent Tuberculosis

Introduction

By inhaling minute droplets from an infected individual's cough or sneeze, a person can get tuberculosis (TB). Although the lungs are the primary organs affected, the stomach (abdomen), glands, bones, and neurological system may also be impacted. Lungs are typically impacted by the bacterial infection known as tuberculosis (TB). A condition that is caused by a certain type of bacteria and is transmitted from person to person through the air. Still, tuberculosis (TB) is the major worldwide health threat that results in morbidity and mortality. Infection with Mycobacterium tuberculosis (M. tuberculosis) is thought to affect between 2 and 3 billion individuals worldwide, or one in three, with a lifetime risk of active TB disease of 5–15%. An estimated 9.6 million individuals were diagnosed with TB in 2014, and 1.5 million died from it, including 1.1 million HIV-negative people and 400,000 people living with HIV. While tuberculosis is found in all nations, the majority of TB patients reside in low- and middle-income nations, particularly in areas like Sub-Saharan Africa and South East Asia. Multiorgan disease active tuberculosis is brought on by primary infection or by the reactivation of latent tuberculosis. Hence, original TB or reactivation tuberculosis could both be considered active tuberculosis. When the immune system is unable to fight off the infection caused by the Mycobacterium tuberculosis bacterium (MTB), primary tuberculosis develops. Reactivation TB, as its name suggests, is the reactivation of a previously contained mycobacterial infection. Reactivation With 90% of cases, Tb is the most prevalent kind of active tuberculosis. The most frequently affected organ is the lung, but other organ systems such as the gastrointestinal tract, musculoskeletal system, lymphoreticular system, skin, liver, and reproductive system are also frequently impacted. Before a more recent change, patients with active TB infection were formerly thought to have latent TB, which has since been altered to TB infection. Whereas TB disease is the term used to describe patients with active disease. A patient's lifetime chance of getting TB disease is 5–10% in those with TB infection, and this risk rises in various immunodeficiency stages to up to 16% annually in HIV patients. An estimated 10 million new jobs were added in 2019. Individuals with TB infection have a 5–10% lifetime chance of getting TB disease; this risk rises to 16% annually in HIV patients in various immunodeficiency states depending on their level of immunodeficiency. An estimated 10 million new incident cases of active TB illness were reported globally in 2019.

The following factors are necessary for effective TB treatment:

- Timely diagnosis of TB and identification of medication resistance;
- Encouraging and guaranteeing patient adherence to the regimen;
- Thorough contact tracing and treatment of contacts as a preventative measure; and
- TB infection screening in high-risk groups.

Much research is being done to create quick, accurate ways to identify medication resistance even in settings with limited resources. Many less harmful, effective drugs are now being developed. Moreover, strategies for encouraging and ensuring treatment adherence are being examined. The development of effective vaccinations to stop the spread of this lethal illness is another important area of proactive TB prevention where important research is now being conducted.© 2023 IJRTI | Volume 8, Issue 5 | ISSN: 2456-3315

Causative organism:

- Mycobacterium tuberculosis = Human
• Mycobacterium Bovis = Animals

CLASSIFICATION OF TUBERCULOSIS:

<table>
<thead>
<tr>
<th>Class</th>
<th>Type</th>
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| TB-0  | -No TB exposure  
|       | -Not Infected   | No history of exposure. Negative reaction to the tuberculin skin test. |
| TB-1  | -TB exposure  
|       | -No evidence of infection | History of exposure. Negative reaction to tuberculin skin test. |
| TB-2  | -TB infection  
|       | -No disease | Positive reaction to the tuberculin skin test. Negative bacteriologic studies (if done). No clinical or radiographic evidence of TB. |
| TB-3  | -Current TB Disease | M. tuberculosis cultured (if done) or both a positive reaction to tuberculin skin test and clinical and/or radiographic evidence of current disease. |
| TB-4  | -Previous TB disease | History of the episode(s) of TB, abnormal stable radiographic findings in a Person with a positive reaction to the tuberculin skin test, negative Bacteriologic studies (if done), and no clinical or radiographic evidence of Current disease. |
| TB-5  | -TB suspect | Diagnosis pending (a patient should not be in this class for more than 3 months). |

Table 1: Classification of Tuberculosis

ETIOLOGY:
Airborne droplet nuclei are airborne particles that spread tuberculosis (spread when infected people sneeze, laugh, speak, sing, or cough). TB is brought on by M. tuberculosis. An alcohol and acid-fast bacillus, M. tuberculosis. It belongs to a collection of organisms known as the M. tuberculosis complex. Mycobacterium africanum, Mycobacterium bovis, and Mycobacterium microti are additionally included in this category. Non-tuberculous or atypical mycobacterial organisms are the majority of other mycobacteria species.[4] The organism differs from other bacteria in several ways, including the existence of various lipids in the cell wall, including Wax-D, cord factor, and mycolic acid. The following characteristics of M. tuberculosis infection are hypothesized to be influenced by the high-fat content of the cell wall:
• The ability to survive under extreme conditions, such as extreme acidity or alkalinity, low oxygen situations, and intracellular survival;
• Resistant to various antibiotics;
• Difficulty staining with Gram stain and several other stains; (within the macrophage)[5]

HISTORY:
For a large portion of recorded human history, tuberculosis has taken its victims. During the 18th and 19th centuries, it spread like an epidemic over Europe and North America, earning the moniker "Captain Among these Men of Death." Then it started to go downhill. Beginning with Théophile Laennec's research at the beginning of the 19th century, understanding of the pathogenesis of tuberculosis was furthered by Jean-Antoine Villemin's 1865 demonstration of the transmissibility of Mycobacterium tuberculosis infection and Robert Koch's 1882 identification of the tubercle bacillus as the etiologic agent. The tuberculin skin test was created in 1907 by Clemens von Pirquet, who used it three years later to show that asymptomatic children had a latent tuberculosis infection. Sanatoria were created in the late 19th and early 20th century to cure tuberculosis sufferers. In addition to the rest that was offered there, pulmonary collapse techniques were used to rest infected lung tissue and plug cavities. Following the identification of the bacterial origin of tuberculosis, public health initiatives to prevent its spread were developed. The BCG vaccine was widely used after World War I. The discovery of streptomycin in 1944 and isoniazid in 1952 marked the beginning of the modern age of tuberculosis treatment and control.[6]

Types of tuberculosis:
There are two types of tuberculosis (TB): active disease and latent infection. The most prevalent form of active TB is a lung disease, but the so-called "extrapulmonary TB" can also spread to other organs.[7]
Active tuberculosis:

Multiorgan disease active tuberculosis is brought on by primary infection or by the reactivation of latent tuberculosis. Hence, original TB or reactivation tuberculosis could both be considered active tuberculosis. When the immune system is unable to fight off the infection caused by the Mycobacterium tuberculosis bacterium (MTB), primary tuberculosis develops. Reactivation TB, as its name suggests, is the reactivation of a previously contained mycobacterial infection. In of cases, reactivation Tb is the most prevalent kind of active TB. The lung is the organ that is most frequently involved, but other organ systems such the gastrointestinal system, musculoskeletal system, lymphoreticular system, skin, liver, and reproductive system are also frequently impacted. According to estimates from the World Health Organization (WHO), each year over 8 million people worldwide get active tuberculosis, and nearly 2 million people pass away from the condition. One M. tuberculosis patient in ten may get an active infection at some point in their lifespan. According to a 2017 WHO analysis, since 2000, the projected annual worldwide incidence rate of tuberculosis has fallen by 1.5%. Nonetheless, despite these notable successes and the aggressive international efforts to eradicate tuberculosis, the illness continues to be a major cause of morbidity and mortality globally. The highest rates of sickness and mortality are found in developing nations like India, Pakistan, the Philippines, China, South Africa, Indonesia, and Nigeria. According to WHO, these nations collectively were responsible for 64% of all tuberculosis-related fatalities in 2016. A combination of medications, including an intensive phase and a continuation phase, are needed to treat an active tuberculosis infection. To lower the possibility of the mycobacterium developing antibiotic resistance, monotherapy should never be used for active illness.

The most widely used regimens for treating active tuberculosis include those that involve first-line drugs, such as:

- Isoniazid: It prevents neuropathies when combined with vitamin B6.
- Rifampicin is hepatotoxic; hence patients should undergo baseline and follow-up liver function tests.
- Ethambutol: Children whose visual acuity cannot be assessed and monitored should not use ethambutol since it can lead to optic neuritis.
- Pyrazinamide: Patients should undergo routine chest X-rays, serum uric acid testing, sputum cultures, and liver function tests every two to three months during their treatment.

Following a two-month intensive phase in which a four-drug combination (isoniazid, rifampin, ethambutol, and pyrazinamide) is given, a four-month continuation phase using an isoniazid and rifampin combination is given. Patients receiving treatment are advised to use directly observed therapy. After the first two weeks of daily medication, patients on the aforementioned regimens could switch to 2 to 3 times per week with this sort of therapy. Those who take medication twice a week must not skip any doses. Patients taking self-administered medication should be prescribed daily therapy.

Among second-line drugs are:

- Kanamycin, amikacin, and streptomycin are injectable aminoglycosides.
- Capreomycin and viomycin are injectable polypeptides.
- Fluoroquinolones, including Levofoxacin, Gemifloxacin, Ofloxacain, and Moxifloxacin
- Other: terizidone, linezolid, prothionamide, cycloserine, para- amino salicylic acid, and thioacetazone.

Third-line anti-tuberculosis medications are drugs with variable but unproven efficacy against the disease. They are the last resort for total drug-resistant tuberculosis infections and include:

- Amoxicillin/clavulanic acid
- Clarithromycin
- Clofazimine
- Linezolid
- Imipenem/cilastatin

Latent tuberculosis:

When a person has Mycobacterium tuberculosis infection but no active tuberculosis, it is referred to as latent tuberculosis (LTB), also known as latent tuberculosis infection (LTBI). A large reservoir of infected people who may later acquire tuberculosis disease has been created by latent tuberculosis infection. The two billion people who have latent tuberculosis infection, often known as those who have had tuberculosis but do not now have active disease, represent a sizable reservoir of future active cases. Clinical features of this group include evidence of tuberculosis infection as shown by a positive response to the TST, a normal chest X-ray, and the lack of any symptoms or indicators of active tuberculosis. Future instances of active tuberculosis will develop from this population. Immune responses to Mycobacterium tuberculosis infection are what define latent tuberculosis infection (LTBI), which is absent of any clinical signs of active tuberculosis (TB). Mycobacterium TB infection affects an estimated one-third of people worldwide. The majority of infected people show no symptoms of TB disease and are not contagious, but they are at risk of becoming contagious if they develop active TB disease. A proven LTBI contains a 5–15% lifetime risk of reactivation TB, with the majority of cases resulting in TB disease within the first five years of the first infection. However, the likelihood that LTBI may develop into active TB depends on the bacteria, the host, and the environment. Preventive treatment can stop the reactivation of TB. The effectiveness of the LTBI treatment regimens now on the market ranges from 60% to 90%, and the protection they provide can last up to 19 years. The likelihood of adverse drug-related occurrences must be carefully weighed against the potential benefit of treatment. The predicted advantages are typically larger than the possible risks for infected persons in population groups with a high likelihood of progression to active illness. So, it’s crucial to determine which groups would profit the most.

In the US, several treatment plans are suggested for latent TB infection. The following are some of the drugs used to treat latent TB infection:

- Rifapentene (RPT);
- Isoniazid (INH);
• Rifampin (RIF)
  Due to the issues with INH mentioned above, there is a great deal of interest in discovering LTBI treatments that are quicker, safer, and just as effective. This curiosity has led to several observational studies and randomized trials looking into the acceptability, safety, and efficacy of various alternative LTBI treatment regimens. Briefly, preliminary studies in a mouse model showed the potential efficacy of three short-course regimens containing rifampin, with or without auxiliary drugs. 110 This research inspired several clinical studies of these three regimens:
  • 2 months rifampin-pyrazinamide (2RZ)
  • 3–4 months INH-rifampin (A few studies have investigated 3 months INH- rifapentine taken once weekly)
  • 4months rifampin[13]

Drug resistance
• Polydrug resistance - Resistant to two TB drugs (but not both isoniazid and rifampin);
• Multidrug resistance - Resistant to both isoniazid and rifampin;
• Extensively drug-resistant - Resistant to both isoniazid and rifampin, as well as resistance to any fluoroquinolone and one of the three injectable second-line drugs

Any patient who develops MTB resistance should be referred to an expert in infectious diseases. Infections with tuberculosis that are multi-drug resistant are more frequent. This disorder is being treated using a high-dose mixture of first- and second-line medications.[2]

TB signs and symptoms:
Infection with tuberculosis (TB) occurs when the TB germs persist and grow in the lungs. One of the three stages of TB infection may be present. Each stage has a particular set of symptoms.

Primary TB infection: The initial infection is the first stage. Immune system cells locate and seize pathogens. The germs might be eliminated by the immune system. Yet, some caught pathogens could still live and proliferate. For the most part, primary infection is symptomless. Some individuals may exhibit flu-like symptoms, such as:
  • Low fever;
  • Fatigue;
  • Cough.

Latent TB infection: The stage known as latent TB infection typically follows primary infection. Immune system cells surround lung tissue harboring TB germs with a wall. If the immune system manages to keep the bacteria in check, they cannot cause any more damage. But the germs survive. Latent tuberculosis infection is symptomless.[35]

Active TB disease: Active tuberculosis is a condition that develops when the immune system is unable to suppress infection. Infectious agents spread sickness throughout the body, including the lungs. TB infection may become active as soon as the initial infection is over. However, it typically occurs when a latent TB infection has existed for months or years. Lung TB illness symptoms typically start mildly and get worse over a few weeks. They may consist of:
  • Cough
  • Coughing up blood or mucus
  • Chest pain
  • Pain with breathing or coughing
  • Fever
  • Chills
  • Night sweats
  • Weight loss
  • Not wanting to eat
  • Tiredness
  • Not feeling well in general

Active TB disease in children
There are different signs of active TB disease in youngsters. Symptoms by age may often include the following:
  • Teenagers - Symptoms are comparable to those in adults.
  • 1 to 12 years old: younger children may experience weight loss and a fever that won't go away.
  • Infants: The baby doesn't develop or put on weight as anticipated. A baby may also experience the following signs of fluid swelling around the brain or spinal cord:
    o Sluggishness or inactivity.
    o Extremely fussy.
    o Vomiting.
    o Inadequate nutrition.
    o soft spot bulging on the head.
    o Slow reflexes [36]

Risk Factors of Tuberculosis:
An immunocompromised person is at an increased risk of developing TB. High-risk factors of tuberculosis include:
  o Infants and children have not fully developed immune system
Chronic kidney disease or diabetes
- HIV/AIDS patients
- Organ transplant recipients
- Cancer patients undergoing chemotherapy
- Autoimmune diseases like rheumatoid arthritis or Crohn’s disease[14]

Figure 1: Risk factors for Tuberculosis infection and disease.

Causes of tuberculosis:
The only way to contract tuberculosis is by breathing in polluted air because it is disseminated through the air. Speaking, coughing, sneezing, or talking while they are currently ill can spread TB. Because the germs cannot survive on surfaces, you cannot contract TB by:
- Shaking hands
- Using a toilet
- Sharing drinking glasses or eating utensils
- Touching other surfaces

Transmission of tuberculosis
M. tuberculosis is dispersed in airborne droplet nuclei, which have a diameter of 1 to 5 microns. When people with pulmonary or laryngeal TB illness cough, sneeze, shout, or sing, infectious droplet nuclei are produced. These tiny particles may float in the air for several hours, depending on the surroundings. M. tuberculosis spreads via airborne rather than surface-to-surface contact. When a person inhales droplet nuclei carrying M. tuberculosis, the droplet nuclei travel through the mouth, nose, upper respiratory tract, and bronchi to reach the lungs' alveoli. This is known as transmission.
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**Pathophysiology:**
Aerosolization, phagocytosis, phagolysosome blocking and replication, T-helper response, granuloma formation, clinical symptoms, and ongoing disease and transmission round out the pathophysiology of this illness. The summaries of each of these processes are provided here.

1. **Aerosolization**

   The transfer of infectious bacteria is where the story of the tuberculosis pathophysiology brought on by M. tuberculosis begins and ends. Several processes and criteria make up the TB transmission cascade. These aerosolizations are produced by the source through vigorous expiratory movements like coughing, sneezing, screaming, or singing. The infectious particles' sizes, however, range from 0.65 to greater than 7 m. The bacteria settle there after they get to the alveolar sacs.

   The bacteria travel to the alveolar sacs and settle there.

2. **Macrophage phagocytosis**

   The bacilli will come into contact with alveolar macrophages, sometimes known as dust cells in this relative anatomical capacity, monocytes, and dendritic cells once M. tuberculosis has established itself as a resident in the alveolar sacs. Since they function in surfactants, alveolar macrophages, the predominant cell type in tuberculosis, are thought to have only modest bactericidal action. The mannose receptor attracts Grb2 once M. tuberculosis binds to it, activating the Rac/Pak/Cdc-42 pathway of M. tuberculosis uptake. The absorption of M. tuberculosis is associated with the Rac/Pak/Cdc-42 pathway, which also attracts protein tyrosine phosphatase 1 (Ptp1) that contains the Src homology 2 (SH2) domain (SHP-1). SHP-1 restricts the activity of the trafficking phospholipid phosphatidylinositol 3-phosphate (PtdIns3P), which in turn restricts the fusion of the phagosome and the lysosome. In terms of pathophysiology, the aforementioned actions signify the establishment of the bacteria and, thus, the start...
of bacteremia and the innate inflammatory response. The dust cells’ chemokines will draw in gamma delta (T) cells, neutrophils, monocytes, and natural killer cells. \[20\]

3. **Phagolysosome blockage & replication**

After inhibiting the union of the phagosome and lysosome, **M. tuberculosis** reproduces intracellularly within the macrophages. Asymmetric cell division is a particularly special type of cell division found in **M. tuberculosis**. When an invader enters the lung parenchyma or interstitial space, the immune system starts to create a granuloma, also known as a tuberculoma in this case, to enclose it. The bacteria enter the logarithmic phase of growth and need to be contained as the granuloma develops along with the concurrent recruitment of monocytes and immune cells. The anatomical shift to the lung parenchyma is pathologically linked to lung inflammation. As previously said, the bacilli proliferate intracellularly. As a result of this growth, the macrophage will eventually be destroyed through apoptosis, pyroptosis, necroptosis, ferroptosis, and destruction associated with extracellular traps. Apoptosis and pyroptosis, on the other hand, limit **M. tuberculosis** development as a whole, but necroptosis and ferroptosis are advantageous to the bacteria's survival and success.

4. **T-helper response**

Major Histocompatibility Complex (MHC) class II proteins and IL-12 are used to activate T-cells in the local and regional lymph nodes by dendritic cells and monocytes from earlier in the tale. Within the first three weeks of infection, during which **M. tuberculosis** would have greatly increased its population and may be expanded to other organs, this cluster of differentiation 4 (CD4+) response takes place. The first is a type IV hypersensitivity reaction, which is pathophysiological the reason the pure protein derivative tuberculin glycerol extract had a positive Mendel-Mantoux test result. The release of IFN, which further activates macrophages with enhanced bacitracin characteristics to fight the invader more, is the second key consequence. The development of the granuloma, which happens as a result of numerous macrophages being drawn to the initial lesion, will be the third effect. TH2 cells secrete IL-4, IL-5, IL-10, and IL-13, which promote humoral immunity and are thought to have a less significant function in tuberculosis. \[16\]

5. **Granuloma formation**

As an analogy, a granuloma is a bacterial jail designed to confine germs within a barrier of immune cells. IFN from the TH1 response will lead the macrophages to create nitric oxide through nitric oxide synthase, mature the phagolysosome, and trigger autophagy. TNF alpha (TNF) will be released by the activated macrophages because they are no longer able to get rid of the pathogen. To contain **M. tuberculosis**, TNF stimulates the development of monocytes into epithelioid histiocyte cells, which create caseating granulomas. Giant cells can be created when some of these epithelioid histiocyte cells combine. Sadly, the necrotic pool in the granuloma acts as a nutrition supply and a barrier of defense for this disease. Moreover, the developing vasculature increases the bacteria's access to nutrients.

6. **Clinical manifestations**

Regarding clinical importance, there are two forms of TB: primary tuberculosis and secondary tuberculosis. Primary tuberculosis, or first-time **M. tuberculosis** infection, is a new infection. This infection (primary) develops when the immune system is unable to handle the initial infection. Immunocompromised people are typically affected by this illness. One of the story’s resolutions is the principal infection. At this point, the infected person can release infectious aerosols of **M. tuberculosis** and infect the subsequent susceptible person. The lymph nodes, genitourinary, gastrointestinal, pleura, and skeletal systems are all affected by extrapulmonary illness (with the latter resulting in tuberculosis spondylitis). Mild tuberculosis is a condition in which the granuloma has spread throughout the body and tuberculomas are seen all over the body. Because the tuberculoma can liquefy and drain following bacterial reactivation (cavitation) and the bacilli are aerosolized via the airways, secondary tuberculosis may also be the end of the narrative. As a result of **M. tuberculosis** infection and the pathophysiology of the illness known as tuberculosis, clinical outcomes might be affected in either a primary or secondary manner. \[22\] \[24\] \[27\]

Clinical features:

Clinical characteristics: EPTB is harder to diagnose clinically and less frequently encountered by doctors than PTB.

1. **Military TB**

Clinical signs, which can include fever, weight loss, night sweats, anorexia, and weakness, are typically non-specific. Fever, wasting, hepatomegaly, pulmonary findings, lymphadenopathy, and splenomegaly are the physical findings, listed in decreasing order. The presence of granulomas in the retinal choroid is a very suggestive sign of disseminated TB. \[26\]

2. **T-cell lymphoma**

It appears as an uncomfortable swelling in the cervical region. (supra-clavicular fossa). The procedure is typically bilateral, and as the disease progresses, the lymph nodes merge and become commented. The skin that lies on top becomes irritated, and eventually, swollen lymph nodes rupture through the inflammatory skin, creating a sinus tract. By squeezing the bronchi or causing bronchiectasis, which is prevalent in children, intra-thoracic adenopathy may result in atelectasis. \[27\]

**Pleural TB**
The quantity of germs that have infected the pleural space determines how tubercular pleurisy presents. Few MTB bacilli entering the pleural space trigger a hypersensitive reaction that causes a pleural effusion. The process may end on its own or it may result in a massive effusion that brings on symptoms including fever, pleuritic discomfort, dyspnea, and weight loss. Tubercular empyema results from a large number of MTB bacilli due to a cavity rupture or an adjacent parenchymal fistula. HIV-positive individuals with pleural TB present with chronic disease and extra symptoms like tachypnea, night sweats, lethargy, and diarrhea, as well as higher hepatomegaly, splenomegaly, and lymphadenopathy than HIV-negative patients.[28]

1. Abdominal TB

   Depending on the site of involvement, as TB can affect any area from the mouth to the anus, the clinical presentation will vary. The terminal ileum or caecum is the most typical location of involvement, and symptoms commonly include abdominal pain, a palpable mass, weight loss, a fever, and decreased appetite. The characteristic symptoms of tubercular peritonitis include a doughy belly, ascites, abdominal pain, and fever. Additional signs of esophageal TB include dysphagia, odynophagia, and retrosternal pain or discomfort. Additionally, the patient has potentially fatal consequences such as broncho-esophageal fistula and hematemesis. Due to the acidic pH, lack of lymphoid tumors in the mucosa, and fast stomach emptying, gastric TB is uncommon. Dyspepsia, duodenal blockage, and duodenal ulcers are symptoms of duodenal TB. Perforation, fistulae, and obstructive jaundice are further reported consequences. Hematochezia is the typical rectal TB presenting symptom, followed by constitutional symptoms and complications. As an anal fissure, fistula, or perirectal abscess, among other manifestations.[29]

CNS TB

Meningitis (95%) is the most prevalent symptom of CNS TB, followed by tuberculomas (2%) and abscesses (1%). Clinical signs include headache, vomiting, decreased level of awareness, stiff neck, and, in the absence of medical attention, coma, and death. They also include signs associated with cranial nerve involvement.[30]

2. Skeletal TB

The most frequent presenting feature is pain. With or without edema, the affected joints have a restricted range of motion. The patient may have sinus tract symptoms. Chronic back pain, fever, and more than 50% of patients experience neurological symptoms as a result of spinal cord compression when the spine is involved. Due to severe, irreversible neurological consequences, such as paraplegia, and spinal deformities, a delayed diagnosis could exacerbate the situation.[31]

3. Genito-urinary TB

Local symptoms like dysuria, hematuria, flank pain, and an increased frequency of micturition are how patients typically present. In men, the most typical presentation is scrotal swelling/mass with or without discomfort. In women, genital involvement manifests as pelvic pain, menstrual abnormalities, and infertility. Depending on the affected place, symptoms of prostatitis, orchitis, or epididymitis may also appear.[32]

Diagnosis of tuberculosis:

Mycobacterium tuberculosis complex bacilli isolated from body fluids are used to diagnose active tuberculosis. Any patient who is thought to have active tuberculosis poses a transmission risk to the public health system and should be isolated with airborne protections. Initial testing for pulmonary tuberculosis comprises a chest X-ray and sputum analysis. Acid-Fast Bacilli smear (AFB smear), mycobacterial culture, and nucleic acid amplification assays are all included in the assessment of sputum. (NAAT). Occasionally, the inability to produce sputum can be a problem; in this case, nebulized hypertonic saline can be utilized to induce sputum. X-rays are used in radiology to make a TB diagnosis. Chest radiograph abnormalities can be used to rule out pulmonary TB even if they are never diagnostic of the disease.

Chest X-ray:

An X-ray of the chest can detect lung damage, a sign of pulmonary tuberculosis. Further testing is required to demonstrate if TB is the cause of the damage discovered.

CT scan:

Your doctor might recommend a CT scan if a chest X-ray does not provide an accurate or clear image. For clear images of the bones and soft tissues in your body, a series of X-rays are taken at various angles. More mild symptoms of tuberculosis that may be present can be found with a CT scan.

Tuberculin skin testing:

The Mantoux test is a two-part procedure that involves injecting a pure protein derivative into the skin for one milliliter and checking for induration 48–72 hours later. The size of the induration and the risk of exposure are then used to divide the patients into three groups. These three teams consist of:

* **Low risk**: There is very little chance that these patients may become infected with tuberculosis. Only a substantial induration of 15 mm or more after the intradermal injection of a pure protein derivative qualifies as a positive Mantoux test.
* **Intermediate risk**: The likelihood that these patients may get tuberculosis is intermediate. Their test is deemed successful if the measured induration is greater than 10 mm.

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• **High risk:** There is a high chance that these patients may get tuberculosis. Their test is deemed successful if the measured induration is greater than 5 mm.[2]

**Blood Test:**
A reliable test for tuberculosis is a blood test. Results can be obtained with just one clinic visit. A positive test, however, does not reveal whether an infection is dormant or active. This needs to be clarified through additional testing. QuantiFERON and T-SPOT are the two types of blood test technology now available.

**Sputum culture for TB**
For diagnosis, mycobacterial culture is the gold standard. Both solid and liquid media should be used for mycobacterial culture. The gold standard for bacterial detection is liquid media culture, which can identify very low bacterial loads. Testing for drug susceptibility requires culture.[3] Solid media costs less, but it takes longer for the organism to grow. Although more sensitive and costly than solid media, liquid media can produce organisms as early as 10–14 days.[34] Culture can distinguish between MTB and NTM.

**Tuberculosis complication:**
Infection with tuberculosis can result in problems like:
- Joint damage
- Lung injury
- Bone, spinal cord, brain, or lymph node infections or damage
- Kidney or liver issues
- Inflammation of the heart's surrounding tissues[35]

**Side Effects of Treatment**
Immediately notify your doctor if you experience any of the following signs:
1. Fever for 3 or more days
2. Pain in the lower abdomen
3. Itchiness or a rash
4. Nausea, vomiting, or no appetite
5. Yellowish skin or eyes
6. Dark or brown urine
7. Fatigue
8. Tingling, burning, or numbness of the hands and feet
9. Easy bruising or bleeding
10. Dizziness[36]

**Nursing management of tuberculosis:**

**Assessment:**
1. **The extent to which the patient comprehends the illness**
The ability of the patient to adhere to treatment programs will be shown by their knowledge of the disease process and how it is spread.
2. **Family members or close friends.**
People beside the patient may contract the disease as well. They can potentially expose the patient to additional germs in a similar way.
3. **The way of life of the sufferer.**
The risk of contracting TB and other infectious diseases might be increased by personal risk factors like smoking, drinking, and drug misuse.
4. **Assess the patient's cooperation level.**
To effectively treat TB, a multidrug regimen must be strictly followed.

**Interventions:**
1. **Place an airborne precaution**
   A negative-pressure isolation chamber must be provided for the patient while they are a patient, and airborne measures must be taken. Teach the patient how to practice good hygiene, such as wearing masks and frequently washing their hands, to help prevent the spread of the bacterium to others.
2. **Inform others about the dosage schedule.**
   It could take six months of treatment for TB to be cured. Inform the patient that to effectively eliminate the bacteria, their prescription must be taken exactly as directed.
3. **Stress the value of follow-up visits and routine sputum testing once more.**
   To make sure that therapies are working, it is crucial to track the disease's growth or decline.
4. **Track your symptoms.**
The most typical signs of infection include fever, tachycardia, and changes in sputum production.
5. **Promote a healthy, balanced diet**
   Loss of appetite and weight loss may be brought on by TB. If patients are unable to stomach larger meals, teach them how to consume small, frequent snacks. Malnutrition can be avoided with a nutrient-rich diet.

6. **Report to the appropriate health authorities.** TB is a reportable disease, and healthcare professionals are required to report cases and potentially exposed individuals to the local health department within 24 hours in most states.

7. **Check liver function studies (ALT/AST)**
   Since the treatment plan includes a months-long multi-drug regimen, the liver may be affected.

**Conclusion:**

Tuberculosis remains one of the deadliest infectious diseases and has claimed millions of lives for many years. While significant progress has been made toward controlling the global burden of TB over the past decade, more efforts are still needed. Emerging issues such as multidrug resistance threaten to revert the progress made regarding TB care and control. The knowledge base for TB remains a rapidly expanding area and global guidelines are continually being refined for instance to incorporate new anti-tubercular drugs to tackle issues of resistance. Health professionals, policymakers, patients, and the general public must keep up-to-date with current TB management and control trends. This will be essential for the efficient adoption of global guidelines to the country-level situation, particularly taking into consideration issues such as disease burden, health system structures, and available resources.

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