

Example of essay on tuberculosis

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Tuberculosis is one of the oldest diseases known to cause medical illness in humans, and is the second most common cause of death due to infection worldwide (Riley, 2013). The bacteria mycobacterium tuberculosis (M. Tuberculosis) causes the disease; and while it is widely known that it affects the lungs of the individual, other organ systems may be affected in nearly one-third of cases (Harrison's, 2008). Proper treatment in drug-susceptible strains leads to a cure; however, drug resistant strains or improper treatment has a 5-year survival rate between 50-65% of cases (Harrison's, 2008). Transmission is through respiratory droplets passed from infectious patients that suffer from pulmonary tuberculosis.

Members of the M. tuberculosis complex cause the actual disease, tuberculosis and besides M. tuberculosis includes the bacteria, M. bovis, M. africanum, M. microti, M. canetti, M. caprae, and M. pinnipedii (Riley, 2013). The cell envelope of these bacteria separates it from other gram-negative bacteria in that there is no true outer membrane. In fact the outer membrane is composed of three macromolecules, peptidoglycan, arabinogalactan, and mycolic acid, covalently linked to each other. These components of the cell wall give mycobacterium their unique staining properties. The organism stains positive with gram stain, however it is its ability to resist destaining by alcohol that leads to the term acid-fast bacillus. Staining and detection of this acid-fast bacillus using the Ziehl - Neelsen stain is the most commonly used method of detecting infection. The bacteria is characteristically slow growing on artificial growth media, taking between 20-24 hours (Riley, 2013).

Initial infection leads to the bacilli being taken up by macrophages. As the

bacteria multiply in the macrophages and these cells rupture releasing chemo-attractants that bring in other cells, ultimately leading up to the activation of cell mediated and humoral immune system (Harrison's, 2008). The initial stages of the infection are generally asymptomatic; however, following a period of approximately 2-4 weeks two important host responses occur; the macrophage response which activates the cell mediated immune system, and a tissue damaging response (Harrison's, 2008). The macrophage response leads to the activation of T-cells, which activate macrophages that are able to kill and digest bacilli. The tissue damaging response is a result of a delayed type hypersensitivity reaction wherein it destroys macrophages that are not activate and contain bacilli that are actively multiplying; the problem is that it also destroys surrounding tissue leading to the characteristic pathological appearance of caseous necrosis (Harrison's, 2008).

Clinically speaking, the disease can manifest as pulmonary, extra-pulmonary, or may have symptoms of both. The presence of extra-pulmonary symptoms is mainly seen in HIV infected individuals, while in approximately 80% of cases in patients that are HIV negative, have an infection limited to the lung. The pulmonary infection can be categorized as a primary infection or a secondary infection. Because most inspired air travels to the middle or lower lung zones, these areas are most commonly affected in primary disease. Lesions are usually associated with accompanying lymphadenopathy and may regress spontaneously. They are sometimes evident on chest radiographs as small-calcified nodules and are termed Ghon lesions (Harrison's, 2008). Secondary infection is accomplished by reactivation of a

latent infection that is characteristically seen in the apex of the lungs; where the higher oxygen tension, as compared to the middle and lower zones, leads to multiplication of the bacteria (Harrison's, 2008). The extent of parenchymal involvement in this stage varies greatly depending on the individual; in severe cases extensive cavitory lesions may be seen, which contains liquefied necrotic contents that discharge into the airways leading to satellite lesions in the lung (Harrison's, 2008). In the early stages of the disease symptoms include mainly fever and night sweats. Weight loss, anorexia, and a general feeling of being unwell. As time goes on a cough always develops which at first is unproductive but later becomes productive as well as bloody (Harrison's, 2008). Massive hemoptysis may develop as erosion of the blood vessels occurs. In extra - pulmonary disease, the most common sites of infection, in order of frequency, include the lymph nodes, pleura, genitourinary tract, bones, joints, meninges, peritoneum, and pericardium (Harrison's, 2008).

Diagnosis of infection involves microscopy and culture as described above. Other methods of diagnosing the infection involve a tuberculin skin test; which is positive 3-6 weeks following infection but remains positive for the entire life of the patient, even after treatment (Shingadia, 2012). Following a positive tuberculin skin tests patients are sent for chest radiography where it is possible to detect lung parenchymal damage and lymphadenopathy (Shingadia, 2012).

The four major drugs that are considered the first line treatment in the fight against tuberculosis infection are, isoniazid, rifampin, pyrazinamide, and ethambutol (Harrison's, 2008). The treatment regimen involves a 2-month

initial, or bactericidal, phase of the use of all four drugs, with a following 4-month continuation regimen, or sterilizing, phase involving only the use of isoniazid and rifampin (Harrison's, 2008). The most common reason for not being cured is not sticking to the treatment plan; this is also the reason why drug-resistant strains have developed in the past decades (Harrison's, 2008).

Prevention can be accomplished by quick recognition and the prompt administration of treatment. However, other forms of prevention include the use of the BCG vaccination as well as treatment of latent infection in patients at high risk of developing active disease.

Works Cited

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