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MYCOBACTERIUM TUBERCULOSIS: An In-depth Discussion for Embalmers Part 2

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CONTINUED: After the 50's, the sanatoriums in the United States were emptying out and effective treatment and prevention was on the horizon and the sanatorium concept died out. Most were abandoned or converted to mental hospitals or other type patient facilities. Tuberculosis was considered conquered and the number of cases had started to drop rapidly. It was assumed that T.B. would never be a threat again - how wrong we were.

THE ORGANISM AND PATHOPHYSIOLOGY: Mycobacteria are gram-positive rods that are acid-fast staining (meaning they retain stain after an acid-alcohol wash during the staining process). They are slender and non-motile and very small (on the order of .5-1 micron). Mycobacteria form no spores or capsules. They have a very thick waxy cell coat that is 40% or greater in lipid content. The cell coat is formed of mycolic acids that are 70-90 carbon chain length fatty acids. This is the reason for their relative high resistance to disinfection and antibiotics. They easily take up residence in macrophage during infection and survive for long periods of time. This is the predominant reason for the very insidious and chronic nature of mycobacterium infection. They are very slow to reproduce, taking 24 hours or more for cell division compared to 20 minutes for most typical bacteria. Colonies are usually not visible until 3-5 weeks of culturing, making the diagnosis of infection very slow. There are greater than 85 different species of mycobacteria known and most are harmless soil-dwellers. However, quite a few infect man and present a definite health hazard under several circumstances.

Tuberculosis, or the T.B. complex, is composed of Mycobacterium tuberculosis, M. bovis (found in cows) and M. africanum (indigenous to west and central Africa). Also included in this category, but not a threat to mankind is M. microti, which infects rodents but does not cause T.B. in humans. In addition, to the T.B. complex, there is M. fortuitum which occasionally causes skin and soft tissue infections in man (some of which can be serious). M. fortuitum can be a serious health hazard due to its resistance to 1st and 2nd tier mycobacterial drugs during treatment. M. haemophilium causes joint infections and skin lesions of a necrotizing nature and other systemic long-term HIV infection. M. kansasii is the cause of a pulmonary disease very similar to T.B. in humans and is the second most found T.B. type mycobacterium in man that causes a pulmonary disease. M. marimum causes skin infections resembling sporotrichosis that is called swimming pool granuloma and is found in tropical fish aquariums, old swimming pools and stagnant pools of water.

An emerging mycobacterium found in Africa (especially Ghana, the Ivory Coast and the western coast of Africa) is M. buruli. This organism is also found in Mexico, India, Peru, Bolivia and southeast Asia. It is occasionally found also in Australia and, in fact, was discovered in Australia in 1947. M. buruli is an impossible to culture mycobacterium that resides in stagnant still waters and is transmitted to humans by unknown means. It causes a horrible, disfiguring skin disease where toxins secreted by the organism destroy flesh and subcutaneous fatty tissue with large areas of dead flesh sloughing away from the sufferer. There is no effective drug treatment - only tissue excision or amputation are options. In some areas M. buruli (buruli ulcer disease) has surpassed leprosy and tuberculosis in frequency of incidence. A microepidemic in Melbourne, Australia in 1997 infected several people and was traced to a golf course pond.

Mycobacterium Avium Complex or MAC is composed of M. avium, which was originally a bird pathogen and M. intracellulare. Both these organisms are indigenous saprophytes found in soil, water, food and occur normally in humans. However, during periods of immunosuppression, such as in HIV infection or AIDS, it can become a serious health hazard. There is very little high-risk AIDS patients can do to stop infection except with long term drug therapy. The disease states are disseminated MAC which is the most common and causes bacteremia and tissue infections and pulmonary MAC which is uncommon, but when present causes chronic progressive pneumonia.

Mycobacterium leprae is the cause of leprosy or Hansen's disease. The transmission of leprosy is uncertain but it is known to be of low infectivity and high chronicity. The incubation period is assumed to be in excess of 3 years. The disfiguring nature of leprosy comes from the chronic infection of skin and lepromatous skin lesions with digit loss possible. Most cases of leprosy occur from long term exposure to an infected individual. It is a very slow growing and impossible to culture mycobacterium. It can infect other animals and is confirmed in armadillos of Texas and Louisiana and New World monkeys. The organism prefers cooler regions of the body and is found in skin, testes and nasal mucosa. There is effective drug therapy through Dapsone and other T.B. type drugs (if the strain of leprae is drug-resistant). It is estimated that greater than 10 million were infected worldwide in 1991 and the numbers are higher now. There is less than 200 cases in the U.S. in a typical year and almost all are imported cases due to immigration.

M. tuberculosis symptoms include fever, chronic cough, night sweats, weight loss and occasional hemopytsis (spitting up of blood). Pulmonary T.B. occurs when the droplet nuclei (less than 10 microns) is inhaled and passes to the alveoli and infects the macrophage by the normal mechanism of phagocytosis. Most cases end here with the infection being successfully resolved. If not, calcified tubercles or granulomas can form and the bacilli travel to the hilar lymph nodes and disseminate throughout the body. The disease can often be subclinical at this point. M. tuberculosis can cause meningitis, skin disease, kidney involvement, bone infiltration and infection and genitourinary disorder (in 15% of all cases one or more of these involvements occur). Progressive primary T.B. can result in necrotizing pulmonary and extrapulmonary disease.

Of all infected with T.B., 5-8% become ill, the rest become carriers and effectively control the infection. Fortunately, carriers are virtually non-infectious. Occasionally, the disease erupts later in life after a previous recovery (e.g. Eleanor Roosevelt) and can reactivate and become a serious infection and health problem.

Infants are especially susceptible to tuberculosis, school age children are resistant, adolescents and young adults (esp. young females) are susceptible, older adults are resistant and the elderly and feeble are highly susceptible.

Reactivation T.B. can occur even years after initial infection in adults with fevers, coughing, night sweats, and upper posterior lobe cavitation or infiltrates. This occurs in up to 10% of carriers. The tuberculin skin test is an IPPD (intermediate strength protein derivative) type test that is only 75% positive in reactivation T.B. and is even less reliable in HIV patients. The typical tuberculin skin test is usually of the HEAF type (which is most familiar in the U.S.) or the mantoux test. Occasionally one will also see the tine test used. Untreated reactivation T.B. is fatal. HIV patients have a 10% lifetime chance of reactivation. T.B. if skin test positive and the greatest risk is within 2 years of pulmonary infection. Unfortunately, until recently, culturing was the only reliable diagnosis, but PCR-DNA tests are being developed and tested, however, the accuracy is not high.

Drug therapy is available through several very effective drug regimens. However, all T.B. drugs are toxic and side effects can occur. The drug regimen can be involved and very lengthy. Typically, INH (isoniazid), rifampin, PZA (Pyrazinamide), ethambutol, streptomycin (sometimes), quinolones (in multi-drug-resistant cases), rifabutin (in rifampin resistant cases) and rifapentine (a new substitute for rifampin) are the drugs of choice. Occasionally, a few others are used in very resistant strain cases. INH is the drug of choice for prophylaxis or prevention in high-risk individuals.

There is a vaccine available but it is not used in the U.S. and has had dubious success in other areas. The BCG (Bacille-Calmette Guerin), a substrain of M. bovis is mainly for children as a protection against the most serious manifestations of tubercular disease such as meningitis and disseminated blood-borne T.B. Protection is only likely for a percentage of recipients and the effects wear off over time. It has never been advocated in the U.S. due to conflicting field reports regarding its effectiveness. Many AIDS patients go into full-blown T.B., even if vaccinated and BCG can also activate in advanced AIDS cases. BCG is only given to skin test negative individuals.

THE NEW EPIDEMIC: After continual declines in the number of tuberculosis cases in the 60's and 70's, starting in 1979, small increases were noted in the relative number of cases. The changes were very small and no one was alarmed. However, in 1985, to everyone's surprise, the statistics reversed and an increase in total number of cases was recorded. By 1990, T.B. cases had climbed to 25,700 per year. This resurgence peaked in 1992-1993 and a slow decline has again occured. In 1997, 7.4 cases/100,000 was reported and in 1998, 6.8 cases/100,000 were documented. This amounted to slightly less than 20,000 total cases in 1997 and even slightly less in 1998. The main reasons for the reversal was too early discharges from treatment clinics and the fact that multi-drug resistant (MDR) strains were starting to appear. In fact, by 1998, MDR strains of M. tuberculosis were reported in 45 states. The early discharges allowed infective people to circulate in society and pose an infection threat. Guinea pig studies have verified infectivity even after several weeks of intensive drug therapy. Most clinics reported that only 1/4th of their patients finished their medications, which are long and involved regimens.

Tuberculosis, worldwide and in the United States, is emerging into the suburbs from innercity areas, the poor and the homeless. There are 8 million new infections every year throughout the world. There were 2.9 million deaths due to T.B. worldwide in 1990. Tuberculosis is the world's number one human pathogen with 10.2 million cases expected in the year 2000. The death toll anticipated in 2000 is 3.5 million. Tuberculosis literally exploded out of sub-saharan Africa in the 1980's. The World Health Organization essentially considers "Africa is Lost", due to the combined impact of HIV, AIDS and T.B.. Over 1/3 of the world's population is skin test positive for tuberculosis, which equals 1.7 billion people. Fortunately, only a small percentage will actually express the disease (3-7%), but the sheer numbers are staggering.

HIV and T.B. feed off each other synergistically in that T.B. is facilitated by the immune suppression that results from infection with HIV and the devastating symptoms of AIDS. HIV infections can actually trigger latent reactivation T.B. or induce a new infection. T.B. hastens the conversion of HIV disease to AIDS with survival times drastically reduced. In the United States minorities account for a disproportionately large number of T.B. cases yearly. For example, in 1995, 54% of all cases were African-Americans and Hispanics, while 17.5 % of all cases were Asians. The other disturbing statistic is that 13-20% of culture-positive T.B. cases were resistant to one or more frontline drugs. In 1995, 39 states reported MDR cases, the number increased to 45 states in 1998.

The continual decreases in T.B. cases up until 1985 was due to improved nutrition, housing, sanitation, improved medical care and effective antibiotics. When the trend reversed and cases again climbed, the major causes were the HIV/AIDS epidemic, increased immigration, continual poverty, increased drug use, increased homelessness, increased alcoholism, over crowded housing, increase in elderly population, poor treatment protocols (resulting in non-compliance with drug regimens), increased prison populations and increased nursing home populations. Even though there are still small declines in total number of cases in 1995 thru 1998, the number of immigrant cases has increased. Most immigrant cases occur with immigration from Mexico, Philippines, Haiti, Vietnam, Korea and India. More Mexican cases are noted due to the much larger immigration numbers from Mexico.

MDR strains are a serious threat to effective treatment and control of tuberculosis in the 1990's and the foreseeable future. Some isolates are resistant to all known anti-T.B. drugs and require a combination of exotic and highly toxic secondary drugs to control the infection with serious side effects. These strains are highly resistant to INH (isoniazid), streptomycin, ethionamide, rifampin, pyrazinamide, cycloserine, rifabutin and ethambutol, in addition to a few other lesser used drugs. Strain W is the most widely known strain that is multi-drug resistant and was first discovered in 1990 in a New York city hospital where it swept through a ward resulting in 70 deaths and 90 additional infections (including the medical staff). The ancestor bacillus of strain W was noted in Ohio six years earlier. By 1995, Denver, Atlanta, Miami and Paris all had reported the presence of strain W.

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