TUBERCULOSIS IN ORTHOPAEDICS

Tuberculosis is a multisystemic disease with a wide range of presentations. It carries the highest mortality and morbidity of infectious diseases worldwide. It is most common in South Eastern Asia with the highest rates of mortality in sub-Saharan Africa. The major factor of large disease burden is HIV/AIDS. Other factors like poverty and social deprivation have led to congestion in slums and prisons and limited access to health care. Other conditions that lead to an immunosuppressive state like chronic renal failure, substance abuse and malnutrition have also contributed to the prevalence of tuberculosis worldwide[1].

WHO reported 9.6 million people infected with TB worldwide with 1.5 million deaths. Out of those affected 95% were from low and middle income countries[2]. It was reported as the top 5 cause of death in those aged between 15 and 44 years old. One out of three HIV deaths was due to tuberculosis and 480,000 people developed multidrug resistant TB. The millennium development goals have been met in 2015 with the incidence falling by 1.5% annually from 2000[2]. Tuberculosis death rates have fallen by 47% from 1990 to 2015 with 43 million lives saved between 2000 and 2014 by TB diagnosis and treatment[2].

Tuberculosis dates back to antiquity. Signs of skeletal TB (Pott's disease) have been reported from as early as 8000 BC in Europe from Neolithic remains, 1000 BC in ancient Egypt and the pre-Columbian new world[3]. TB was recognized as a contagious disease around 400 BC at the time of Hippocrates when it was termed "phthisis" derived from the Greek word phthinein which means to waste away. In English, pulmonary TB was referred to as "consumption". *Mycobacterium tuberculosis* the organism responsible for TB infection was first isolated by German physician Robert Koch in 1882[3]. With increase in population and urbanization around the 17th century the incidence of TB also increased so that by the industrial revolution in Europe in 1750 TB was responsible for over 25% of adult deaths. The incidence further increased through the 18th and 19th centuries. The situation improved in 20th century with improved identification, screening, chemotherapeutic measures and infection control methods like isolation. A resurgence of TB was noted from 1985 due to the emergence of HIV/AIDS. Globally, co-infection with HIV is highest in South Africa, India and Nigeria. People with AIDS are 20-40 times more likely to develop active TB than immunocompetent people and TB is the leading cause of death in people with HIV/AIDS[3].

Tuberculosis can be transmitted either via inhalation (*Mycobacterium Tuberculosis*) or ingestion (*Mycobacterium Bovis*). The most common primary site is the lungs (85%). Extrapulmonary TB can occur as part of primary or late, generalized infection. Most common sites of extrapulmonary disease include mediastinal, retroperitoneal and cervical lymph nodes, vertebral bodies, adrenals, meninges and GI tract. Classical clinical features of active pulmonary TB include cough, hemotypsis, fever, night sweats, weight loss/anorexia, chest pain and fatigue[4].

Out of the patients with extrapulmonary disease, bone and soft tissue TB accounts for 10-15%, and 1-2% of the total TB cases. Tuberculous spondylitis is responsible for 40-50% of cases with musculoskeletal involvement. Musculoskeletal complaints include swelling, stiffness and pain. Osseus involvement is associated with localized warmth, swelling and tenderness. Articular involvement is associated with soft tissue swelling, pain and restriction in involved joint motion. Patients with spinal disease may have pain, kyphotic deformity and neurologic deficit[4].

Tuberculous osteomyelitis represents around 5% of ostearticular tuberculosis. Abscess formation may occur and sinuses are common. Symptoms may take days to months to develop and less commonly is visceral disease associated. The tuberculous sinus tends to have a bluish discoloration at the periphery with sero-sanguinous discharge, undermined edges, matted lymph nodes and fixation to underlying bone. The most common radiographic presentation is a solitary lytic lesion usually with a sclerotic rim. The lesion may be diaphyseal or metaphyseal and may extend to the adjacent joint. Unusual forms of skeletal tuberculosis include multiple cystic tuberculosis, disseminated skeletal tuberculosis (immunocompromised), closed multiple diaphysitis (Immunocompromised children) and tuberculous dactylitis. Biopsy is recommended to establish the diagnosis. Treatment involves anti-tuberculous drugs and curettage with or without bone grafting[1,5].

Tuberculous arthritis develops by either direct penetration from a metaphyseal focus or seeding of the synovium by bacilli. This results in development of an effusion and synovial hypertrophy. There is proliferation of synovial granulation tissue initially at the joint periphery progressing centrally, leading to marginal erosion, destruction of articular cartilage and further bone erosion. This subsequently leads to joint destruction which may be associated with subluxation or dislocation and finally bony ankylosis. Clinical and radiographic findings, treatment and expected outcome are correlated by Tuli classification[6]. Chemotherapy is recommended in all patients with active disease. Treatment of synovitis and early arthritis (stage I, II) involve chemotherapy, rest, range of motion exercise and splinting. Late disease (stage III, IV, V) treatment involves chemotherapy, osteotomy, arthrodesis and arthroplasty. Ideally there should be a disease free interval between completion of treatment and joint

replacement. Prophylactic chemotherapy for weeks to months may allow early joint replacement and chemotherapy can salvage a reinfection of a prosthetic joint[6].

Tuberculous spondylitis commonly involves the lower thoracic and thoraco lumbar spine but can affect any level of the spine. Rarely, skip lesions may occur. It commonly presents with back pain, kyphotic deformity and neurological dysfunction. It presents radiologically as destruction of adjacent end plates in two or more vertebra. The prognosis for neurological dysfunction is good if symptoms develop gradually, are of short duration and in "active" rather than "healed" phase of the disease. Tissue diagnosis is difficult to achieve, even under the best circumstances[7]. Treatment options include chemotherapy, surgical decompression and arthrodesis. Traditionally chemotherapy was admitted for up to 18 months, but currently can be administered up to 9 months. Surgical treatment is suggested for an increase in size of a paravertebral abscess despite adequate chemotherapy, involvement of the posterior elements, lack of clinical response after 3 to 6 months of chemotherapy (neurologically normal), lack of neurologic recovery or progression of neurologic deficits after 3 to 4 weeks of chemotherapy, recurrence of disease, mechanical instability, or an uncertain diagnosis[8].

It is very vital to detect and treat tuberculosis early in order to maximize the outcomes and reduce potential complications.

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