Tuberculosis – a Diagnostic Chameleon

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Tuberculosis is well known as a diagnostic chameleon. It is an ancient disease caused by the bacterium Mycobacterium Tuberculosis. It has been around for more than 3000 years probably over 15000 years. Evidence of skeletal abnormalities of TB were found in Egyptian mummies from 2400 BC. The term Phthisis, consumption first appeared in Greek literature. In 460 BC Hippocrates identified phthisis as the most widespread disease defining its symptoms and characteristics as lung lesions. Johann Schonlein coined the term tuberculosis in 1834. Also known as the white plague, the great white plaque captain among the men of death. In 1882 Robert Koch identified the tubercle bacillus as the etiological agent and posted his famous postulates (Koch Henle Postulates). BCG vaccine (Bacille Calmette Guerin) developed by Albert Calmette and Camille Guerin in 1921 revolutionised the immunization programs and tuberculosis control.(1) Infant vaccination has shown an efficacy of 70 – 80% against childhood tuberculosis namely meningitis and military tuberculosis.(2) Every year >10 million people fall ill with tuberculosis, despite being preventable and curable – 1.5 million die each year. TB is the leading cause of death of people with HIV.

The three stages of TB are

1. Primary infection
2. Latent TB infection
3. Active TB disease(3)

Different types of TB

1. Pulmonary TB (lungs)
2. Extrapulmonary - (TB outside the lung)(4) (5)
   a. Pleural TB
   b. Lymphnodes – lymphadenitis
   c. Skeletal TB – spinal column (Potts disease)
   d. Brain tuberculosis – meningitis, tuberculoma.
   e. Genito urinary TB (Bladder and Kidney)
   f. Joint TB (arthritis)
   g. Gastrointestinal infection – any part of the gut can be involved
   h. Miliary – involved lung extensively

Two entities of TB

1. MDR (multidrug resistant)
2. XDR (Extensively Drug Resistant TB)

Treatment – several regimens are recommended. (6) (7) (8)

Case Report:

A 56-year-old male farmer by occupation was admitted to GKNM Hospital on 20th April 2005 with complaints of anorexia and progressive weight loss for 6 months. History of low-grade fever for the 3 past months. No history of cough, blood in the sputum or headache. No comorbid conditions. No history of any addictions. On examination, he was thin built, febrile and pale, weight of 46kg. No icterus, lymphadenopathy, or sternal tenderness. Cardiovascular and respiratory system within normal limits. Abdomen examination - hepatomegaly 7cm and splenomegaly 5cm noted, no free fluid. CNS no deficits were noted. The provisional diagnosis was haematological malignancy. Investigations Hb of 9.3g, Total count 15100 – N 75, L 26, ESR

120mm. RBS and RFT was normal. LFT – ALP of 668 IU, total protein 7 g, albumin 2.4g, PT INR was normal. TFT and BJ protein were normal. Bone marrow? features suggestive of myeloma. USG abdomen multiple hypoechoic lesions in liver and spleen -? lymphoid neoplasm. ECG and CXR were normal. HIV was negative. Serum protein electrophoresis – beta gamma globulin increased, low albumin. Liver biopsy done May 2nd 2005. It showed pre-cirrhotic changes with portal-to-portal bridging and periportal granuloma. In view of features of granulomatous hepatitis on liver biopsy patients started on 4 drugs ATT. The patient tolerated ATT and completed 8 months of ATT (2 months of 4 drugs and 6 months of 2 drugs). The patient progressively improved and became afebrile. Weight gain (53kg) was noted at the time of completion of ATT, no organomegaly. During the follow-up of one year, the patient remained well. The patient was brought to the hospital on 22nd January 2008 by his wife with a history of giddiness and fall, fever followed by generalised tonic-clonic seizures with loss of consciousness. He was taken to a local hospital and referred after treatment and CT brain. His wife also gave a history of multiple abscesses in the leg over the past 6 months treated locally with multiple courses of antibiotics and also underwent skin grafting. Clinically afebrile no pallor, icterus. A tongue bite was noted, no lymphadenopathy and his weight was 46kg. Ulcers are present in the right and left foot. Abdominal examination – Splenomegaly 5 cm, CNS no deficits. Investigations Hb 10g, RFT and RBS were normal, HIV negative. USG abdomen –splenomegaly with multiple focal lesions, chronic portal vein thrombosis. Outside CT Brain showed hypodense lesion in the right posterior region with no post-contrast enhancement. low-grade glioma. MRI brain granulomatous lesions in the right parieto-occipital region suggestive of tuberculoma. In view of the MRI picture suggestive of tuberculoma and past history of granulomatous lesions in the liver and spleen lesion which responded to standard ATT, he was started on anti-epileptics and ATT. He tolerated ATT and had no recurrence of seizures. He was discharged on 25th January 2008. He was regular for follow-up, and given extended ATT (total of 10 months). After completion of treatment, his weight was 55.8kg and clinically no organomegaly. Advised to continue antiepileptics (eptoin 300mg). Last visit in 2016 he was asymptomatic and continuing eptoin. Presenting this case as an uncommon presentation of extrapulmonary tuberculosis, the first abdominal TB proven histologically – treated and recovered. Again presented with recurrence after 2 years as CNS tuberculosis treated with extended ATT with good therapeutic response. (9) (10)

In conclusion, tuberculosis remains a significant global health challenge, with a long history and complex manifestations. The disease's ability to mimic other conditions and its potential for recurrence highlights the importance of thorough diagnosis and appropriate treatment. The development of drug-resistant strains adds further complexity to TB management. Continued efforts in research, prevention, and treatment are essential in the ongoing fight against tuberculosis.

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