Clinical Images

Tree-in-Bud Sign

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Clinical summary:

A 45-year-old male who was alcoholic and sero-positive for HIV 1 antibody presented with nonproductive cough, low grade fever and weight loss for two months. He was noncompliant with anti-retroviral therapy. On clinical examination he was anemic, emaciated, tachypneic and hypertensive. Examination of the respiratory system revealed bilateral vesicular breath sounds and bilateral inspiratory crackles



Fig.-1: HRCT scan of thorax revealed small well defined centrilobular nodules of soft tissue attenuation connected to linear Y or V shaped branching opacities resembling Tree-In-Bud (TIB) appearance.

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His hemoglobin level was 9.4 g/dl, fasting plasma glucose 96 mg/dl, serum creatinine 0.8 mg/dl and he had normal liver function test. HRCT scan of thorax revealed small well defined centrilobular nodules of soft tissue attenuation connected to linear Y or V shaped branching opacities—thus resembling Tree-In-Bud (TIB) appearance (Figure 1). Induced sputum was positive for acid fast bacillus. He was put on short course anti tuberculous regimen consisting of two months therapy with isoniazid, rifampicin, ethambutol and pyrazinamide followed by four months therapy with isoniazid and rifampicin.

Hence, the diagnosis was sputum smear positive pulmonary tuberculosis with bronchogenic spread.

Discussion:

Each secondary pulmonary nodule is supplied by a lobular bronchiole and a lobular artery that are located in the centre of the lobule. Normally they are not visible either in chest radiograph or CT scan of thorax. TIB pattern represents bronchiolar luminal impaction with mucus, pus or fluid which demarcates branching course of the peripheral airways. Dilatation and thickening of bronchiolar wall along with peribronchiolar inflammation also contribute to the visibility of the bronchioles. This results in Y and V shaped branching pattern which together with centrilobular nodule forms the TIB appearance. Inflammation of the small pulmonary arterioles can sometimes produce this sign.

Though initially described in patients of endobronchial tuberculosis, it is also found in several other conditions including infective disorders like Mycobacterium avium-intracellulare, viral, fungal (aspergillosis, *Pneumocystis jirovecii*), bacterial (*H. influenzae*, *Staph.aureus*); diffuse aspiration syndrome; congenital disorders like cystic fibrosis, primary ciliary dyskinesia; connective tissue disorders like rheumatoid arthritis, Sjögren's syndrome; diffuse panbronchiolitis; neoplasms like primary alveolar cell carcinoma, primary pulmonary lymphoma, and carcinomatous endarteritis.²

Classical cause of TIB appearance is post primary pulmonary tuberculosis. It is characteristic, but not pathognomonic, of active and probably contagious disease. It suggests bronchogenicspread in which active organism Tree-in-Bud Sign JM Vol. 17, No. 1

spreads via the airways after caseous necrosis of abronchial wall. The terminal tufts of the TIBpattern may representlesions in the bronchioles and alveolar ducts, whereasthe stalk may represent a lesion affecting the last-order bronchus in the secondary pulmonary lobule. After a full course of anti-tuberculous therapy these lesions disappear. However few small nodules and areas of hyperlucency may be present.

References:

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