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INTERNATIONAL JOURNAL OF ADVANCED RESEARCH (IJAR)

Article DOI:10.21474/IJAR01/18645
DOI URL: http://dx.doi.org/10.21474/IJAR01/18645

RESEARCH ARTICLE

A RARE CAUSE OF ACUTE RESPIRATORYFAILURE: CYSTICPULMONARYTUBERCULOSIS

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Manuscript Info

Manuscript History
Received: 29 February 2024
Final Accepted: 31 March 2024
Published: April 2024

Abstract

Tuberculosis continues tobe a major cause of death. It is a preventable and curable disease. Themost commonbacteria likely to cause a cystic pneumoniaare Staphylococcusaureus, streptococcuspneumonia and Escherichiacoli. The cystic pulmonary tuberculosis is exceptional and shoulddefinitized be from other cystic lung disease.Wepresent a case report of immunocompetent patient with acute respiratoryfailurePulmonarytuberculosiswithunusualcystic change in an immunocompromised host.

Introduction:-

Tuberculosisischaracterized by a clinical and radiologicalpolymorphism the diagnosisiseasy in the typicalform but presents a real problem of differentialdiagnosis in countries endemic for tuberculosis.

Acute respiratorydistress syndrome (ARDS) isknown to be one of the complications of miliarytuberculosis [1]. Cystic and bullouslung tissue changes caused by tuberculosis are an exceedingly rare condition withonly a limitednumber of cases reported. This specialform of TB infection featuresrapidlydeveloping multiple cystic-like lesions in bothlungs, mostly in the upper lobes, alongwithseverecliniсalsymptomssuch as respiratorydistress and/or recurrent pneumothorax, leadingoften to a fatal outcome [2].

Case Report

A59-year-old male patient presented in emergency roomwith the complaints of fever for 1 month duration and shortness of breath for 10 days. On examination, patient's pulse rate was 130 beats/min,oxygen saturation was 77blood pressure was90/60 mmHg, respiratory rate was30/min. On auscultation chestrevealedleftcrepitations.Chest X-ray showedleftdiffuse reticular shadows (figure 1). A CT scann of chest (Figure 2)showeda multiplethinwalledcysts in theleftlung; diffuse ground glass opacities and centrilobular nodules in leftlung.

The WBC count was 5800/mm3, CRP: 83 mg/L, Procalcitonin: 0.12. A serological test for HIV wasnegative. The patient'ssputumwas positive for acid-fast bacilli, and a culture was positive for Mycobacterium tuberculosis.

A diagnosis of respiratoryfailurefrominterstitiallungdiseaseassociatedwithtuberculosiswas made and treatment with corticosteroids and the standard TB drugsrifampicin (R), isoniazid (H), ethambutol (E) and pyrazinamide (Z) (known as the 2RHZE/4RHE regimen) wasinitiated, allowinrapidimprovement of the respiratoryfailure.

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Discussion:

Tuberculosis continues to be a major public healthproblemcausingillhealth to approximately 10 million people eachyear. Tuberculosis (TB) is a preventable and usually curabledisease. Yetin 2022, TB was the world's second leadingcause of deathfrom a single infectious agent, after coronavirus disease (COVID-19), and causedalmosttwiceasmanydeaths as HIV/AIDS [3].

The mostcommon CT findings of reactivationpulmonary TB are centrilobular small nodules, branching linear and nodularopacitiespresent as 'tree-in-bud' sign, patchy or lobular areas of consolidation, and cavitation. Theriskfactorsof atypicalforms of tuberculosis are HIV, diabetes, substance misuse, advancedkidneydisease, malnutrition and treatmentwithcorticosteroids or immunosuppressant.

A lung cystis defined as a well-circumscribed air-filled structure thatislocalizedwith in the lungparenchyma, is>1 cm in diameter and has a definable epithelial or fibrouswallthatisusually<1 mm thick, but thatmaybeupto 2 or 3 mm thick.[4]

Cysts arising as a complication of pulmonary tuberculosis have been veryrarelyreportedand are amongst the rarestpresentation of thiscommondisease [5].

A variety of lungdiseases can cause or mimic thin-walled air-containing cysts in the lung. Cystsmaybeclassified as congenital and acquired. The more commoncongenital causes for cysts in lungdiseasesinclude central and peripheral bronchogeniccysts, intralobarpulmonarysequestrations, congenitalcysticbronchiectasis, cysticadenomatoid malformation of lung and tracheobronchialpapillomatosis [6].

Severalmechanisms have been suggested for the pathogenesis of cysticlunglesions due to tuberculosis: a) a check-valve mechanism due to the granulomatousinvolvement of bronchioles and the excavation of caseousnecroticmaterial by bronchial drainage;b) the communication of tuberculouslesionscontainingcaseousnecrosiswith the bronchi, resulting in the excavation of necroticmaterial and cystic changes; and c) the cysticlesionsrepresentation of areas of dilated bronchioles. Immunohistochemicalstudies and electronmicroscopicexaminations have revealedthat the proteinasessecretedfrom the inflammatorycells of peribronchiolargranulomas are partlyresponsible for the degradation of elasticfibersalong the bronchioles, alveolar ducts, and alveolarwalls.[7]

Cause of hypoxemia in non-miliarytuberculosisis a result of direct injury to alveolarepithelialcellsfromtubercularantigensthroughliquefied, caseouslesions. Theseeffectsmayfurtherbeaccentuated by bronchogenic spread. A smallamount of bacillaryantigenisenough to evoke an exudativeresponse in the host and is an important determinant of direct injury [1].

Conclusion:-

Tuberculosiscysticwithrespiratoryfailure has a good prognosis,atypicalforms of tuberculosis must beknown for rapiddiagnosis and the diagnosisisbased on clinic, radiology and microbiologyearlydiagnosis and treatment are imperative to prevent not onlymorbidity and mortality but alsodisease transmission.

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