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Congenital Heart Disease Complicated With Pulmonary Tuberculosis: A Rare And Challenging Case For The Clinicians.

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ABSTRACT: Highlighting a case of adult male of 21 years, who reported to us in emergency department with feature masquerading cardiac ailment as well as infectious disease which was later diagnosed as case of bacteriologically confirmed pulmonary tuberculosis.

Keywords: Congenital heart disease, Pulmonary Koch's, ventricular septal defect (VSD), atrial septal defect (ASD).

I. INTRODUCTION:

Pulmonary tuberculosis is 2.5 fold more common in children with congenital heart disease (CHD) as compare to normal population. Children with low flow CHD poses great risk and incidence for pulmonary tuberculosis.[1] more often the symptoms of pulmonary tuberculosis are difficult to diagnose in children as they disguised with the symptoms of congenital heart disease.[2] Approximately 65% of patients are asymptomatic and are diagnosed radiologically.[3] In the present article we discuss the case of 21 year old adult male who was diagnosed with tuberculosis at our institute also had atrial septal defect (ASD -ostium secundum) and had surgical correction done 15 years back.

Case History:

Presenting a case of 21 year old male patient who was known case of congenital heart disease with single ventricle, single AV valve with mild AV regurgitation underwent cardiac surgery in childhood, where median stertnotomy, partial thymectomy, vertical pericardectomy, pericardial stamp, aorto aortal cannulation was done was done and he was put on tab aspirin 150mg from since 15 years. He reported to our institute in emergency department with chief complaints of cough with expectoration from one and half month, also complained of blood in sputum (pink frothy) 02 episodes, shortness of breath since 01 week associated with fever from 04 days all symptoms were associated with history of weight loss, loss of appetite, and abdominal pain since 01 day. He was conscious oriented to time place, person and surroundings. Patient had pallor, cyanosis (both peripheral and central), icterus and grade II clubbing. Bilateral pitting pedal edema was present. On auscultation S_1S_2 with pan systolic murmur was heard. Bilateral wheeze was present in all the auscultatory areas of lung fields. On admission his vitals Bp-100/60 mm of hg, spo2- 92% with 3 liters of O_2, pulse rate 90/ min, regular. Investigation were done and had Hb-16.9gm%, total count- 12000, platelet count- 2.7 lacs, viral markers were negative (i.e HIV, HCV, HbsAg), Pt/Inr-2.94, SGOT-172, SGPT-201 were raised, serum potassium-5.9. On presentation he was complaining of cough with yellow color expectoration, his sputum was sent for cartridge based nucleic acid amplification test (CBNAAT) on obtaining results it was found that in sputum sample Mycobacterium tuberculosis was detected and rifampicin resistance was not detected. Patient was put on alternate tubercular regimen, as his liver function was deranged, as per bodyweight according to NTEP programme. Keeping in view of patient persistent breathless and suspicion of pulmonary embolism CT pulmonary angiography was done and it was reported as complex cardiac congenital anomaly. No evidence of artery thrombosis. Diffusely scattered multiple small well defined centrilobular nodules of varying sizes randomly scattered in both the lungs. Large thick walled cavitory lesion right upper lobe with adjacent nodulo infiltrative changes were seen.

II. DISCUSSION:

Over the year's identification, diagnosis and treatment of tuberculosis has been revamped. Tuberculosis is most prevalent in developing countries due to the issue of poor hygiene, insufficient vaccination, malnutrition and HIV infection. Improper development of fetal heart constitute heterogeneous group of abnormalities resulting in the congenital heart disease with single ventricle (SV) physiology incidence ranging from 3.1-4.9 per 10,000 live births and beyond infancy rate is estimated at 1.6 per 10,000 with SV physiology. Tuberculosis is found to be more common in with patients with cyanotic heart disease (CHD) than acyanotic heart disease patients. Spatiotemporal expression during developmental stages underlies normal heart development and an aberrant expression plays pivotal in the pathogenesis of CHD cases.[1,4] Adult CHD patients falls in two categories i.e operated in childhood and secondly the ones are first diagnosed as adults.[5] Cyanotic CHD (CCHD) being more complex form, bear poor prognosis. CCHD results in the alteration of normal blood flow pattern leading to occurrence low pressure pathways risking co- morbidity, affecting oxygenation, i.e. systemic and pulmonary finally, results in development of reactionary inflammation. Patients with associated CHD i.e. down syndrome, VACTERL association (vertebral defects, anal atresia, cardiac defect, trachea-esophageal fistula, renal anomalies and limb deformity), are more predisposed for bacterial sepsis and thereby causing increase in mortality of CHD patients and by four fold in patients of VACTERL syndrome.[6] Immune system plays an important role through the innate and adaptive immune strategy. CHD advocates surgical correction and because of that thymus removal which ranges from partial to complete removal i.e. thymus size poor surgical correction may range from 0% – 50% of the initial mass. Post thymectomized patients are at greater risk for developing the autoimmune disease, diabetes, hypothyroidism, malignancy, atopic and at higher risk of contracting bacterial and viral infections (63.1% Vs 23.1%) as found in the study by the Laura MW et al.[7] Another theory mentioned by Gunay E et al in their literature states that reduced blood flow in CCHD patients inhibits M.tuberculosis unlike acyanotic CHD patients where high blood flow and normal pulmonary arterial saturation favors and provide suitable environment for the growth of M. tuberculosis like in our present case. Patient diagnosed with tuberculosis, on becoming adults, their findings coincides with non specific pneumonia in disguise secondary to heart failure presentation as similar presentation happened in our case.[8] In present case report patient has congenital heart disease since birth with single ventricle there might have been direct relationship between cardiovascular disease (CVD) and Mycobacterium tuberculosis and vice versa. In a study by Sarode et al in India where autopsy was performed in 1000 patients died in hospital between age group of 13-59 years due to common infections accounted for 46.8% and among those common infection pulmonary tuberculosis accounted for nearly 33.8% of patients.[9] In similar study by Glyone SR et al where 2735 autopsies were performed he found 21 cases of CHD with pulmonary tuberculosis.[10] Therefore pulmonary tuberculosis is almost undermined in disguise of heart disease, specially congenital heart diseases which are rarely detected at times due to prevalent heart condition. Emphasis should be made to examine and investigate the patient for respiratory infection too, specially pulmonary tuberculosis.

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Legends:

Fig 1:

X-ray Chest s/o heterogenous opacity in right middle lobe with nodulo infilterative lesions in b/l upper zone and right middle zone

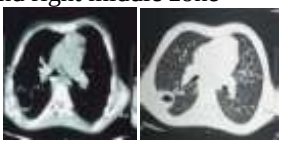
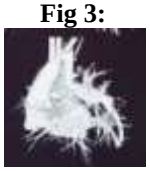


Fig 2: (a) & (b)

HRCT Thorax shows multiple/extensive centrilobular and tree in bud opacities seen scattered in bilateral lung parenchyma with evidence of a cavity in superior segment of right lower lobe with air fluid level (Fig-02 a & b) and also a consolidation in right middle lobe, multiple mediastinal lymph nodes also noted with many of them enlarged with adjacent nodular infiltrative changes were seen.

Fig 3:



CT pulmonary angiography (Fig-03) was done and reported as complex cardiac congenital anomaly. No evidence of artery thrombosis.