Case Report

A rare case of tuberculous polyserositis – A case report

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International Archives of Integrated Medicine, Vol. 10, Issue 2, February, 2023.
Available online at http://iaimjournal.com/
ISSN: 2394-0026 (P) **ISSN: 2394-0034 (O)**
Received on: 12-2-2023 **Accepted on:** 20-2-2023
Source of support: Nil **Conflict of interest:** None declared.

How to cite this article: E.A. Ashok Kumar, Siddamshetty Preetham Kumar, Chokkavarapu Renuka Charan, Bachireddy Parimala. A rare case of tuberculous polyserositis – A case report. IAIM, 2023; 10(2): 35-41.

Abstract

Polyserositis is defined as chronic inflammation of several serous membranes with effusions in serous cavities like Pericardial, Pleural and Peritoneal membranes, resulting in fibrous thickening of the serous membranes and sometimes constrictive pericarditis. There are various causes of polyserositis which include autoimmune diseases, neoplasia, endocrine diseases, drug – related causes and infectious diseases such as tuberculosis. Polyserositis in disseminated TB is a very rare presentation. Diagnosis is often delayed due to the non-specific presentation like polyserositis and its unusual nature. We herewith report a rare case of TB polyserositis, involving pleura, pericardium and peritoneum.

Key words

Polyserositis, Constrictive Pericarditis, Pleural Effusion, Peritonitis, Disseminated Tuberculosis.

Introduction

Tuberculosis (TB) is one of the common infectious diseases caused by Mycobacterium tuberculosis. With 28% of cases globally, India has the most active tuberculosis patients. In 2021, an estimated 10.6 million people fell ill with tuberculosis worldwide which is equivalent to 134 cases per 100000 population. India's TB incidence for the year 2021 is 210 per 100,000 population. It also reported the role of nutrition and under nutrition in development of active TB disease [1]. There is substantial evidence that poverty is a determinant of TB, both at the macro-scale and in individual and hierarchical

analyses. Janssens and Rieder documented a linear association between per capita GDP [2] and TB incidence, and Dye found that the country level human development index was a strong predictor of changes in TB incidence over time [3].

TB has various forms of presentation, common being pulmonary and extra-pulmonary forms. Disseminated TB is defined as lymphohematogenous dissemination of tubercle bacilli in two or more non-contiguous sites [4]. The variety of presentations in disseminated TB makes the diagnosis difficult [5, 6]. Polyserositis in disseminated TB is a very rare presentation [7]. Disseminated TB is fatal if diagnosis delayed. The clinical, radiological and histopathological diagnostic pathways are well described.

Polyserositis (PS) is an effusive inflammation of serous membranes, such as the peritoneum, pericardium, and pleura simultaneously [8]. It is commonly seen in autoimmune diseases (especially familial Mediterranean fever), endocrine diseases such as hypothyroidism, infectious diseases such as tuberculosis (TB), neoplasia, and in a drug-associated context (primarily with clozapine and tyrosine kinase inhibitors).[9,10]. TB presenting with serositis, is called serosal tuberculosis, a common extra-pulmonary manifestation of TB, in highly endemic areas [11]. However, Mycobacterium tuberculosis was not isolated frequently with Polyserositis [12, 13]. This makes the diagnosis of TB polyserositis difficult and challenging and this may delay the definitive treatment [14].

Polyserositis is defined as chronic inflammation of several serous membranes with effusions in serous cavities [15], resulting in fibrous thickening of the serous membranes and sometimes constrictive pericarditis. This illness is also called Concato's disease [16].

Case report

A 50 year old female (house wife) was admitted to hospital, who came with complaints of shortness of breath and swelling of both lower limbs, of 3 months duration and also abdominal distension of 15 days duration. Breathlessness was insidious in onset and was more on lying down position along with frequent waking up in the nights. There was history of left sided chest pain which was squeezing in nature and was present along with above symptoms. No history of palpitations, giddiness, and syncopal attacks there was no yellowish discoloration of eyes, burning or increased frequency of micturition no history of any contact with tuberculosis patient.

Past history: Patient had similar complaints of chest pain 5 years ago for which she was admitted and treated symptomatically. Patient was a known case of hypertension since 8 years and NIDDM since 10 years. She suffered a stroke 2 years ago which was diagnosed as CVA with cerebral thrombosis with right MCA territory infarction with left hemiparesis. No history of thyroid disorders, TB, epilepsy, COPD. No significant personal and family history.

Menstrual history: Attained menopause 1 year ago.
Drug history: No history of any drug intake.

General examination: Patient was conscious, coherent, cooperative and uncomfortable in lying down position but was comfortable in sitting position. No Pallor, Icterus, Cyanosis, Clubbing, Koilonychias, Lymphadenopathy. Bilateral pitting pedal edema was present.

Vitals: Temperature - normal, BP=120/70 mm of Hg in sitting position in right arm, (patient is on anti-hypertensive drugs). Pulse=106/min regular in rhythm with no radio-radial or radio-femoral delay.

CVS examination: Engorged neck veins over neck with elevated JVP along with prominent 'x' and 'y' descent. There was no precordial bulge and apical impulse was not visible. On palpation,

apical impulse was felt in left 6th ICS 0.5 cm lateral to mid clavicular line. There was no para sternal heave and palpable murmurs. On auscultation, S1S2 were heard and at mitral area, a pansystolic murmur, which radiates to axilla was heard. There was loud P2 heard.

Abdomen examination: Abdomen was distended with shifting dullness and positive fluid thrill and there was no organomegaly.

Respiratory system examination: Normal vesicular breath sound on auscultation, with decreased breath sounds at bilateral basal areas were present.

CNS examination: Left hemiparesis was present.

Investigations - CUE - normal. ESR – 20 mm/hr. Hb-9.9 g%, RBC count - 3.4 mill/cu mm, WBC count - 4900 cells/cu mm, Platelet count – 3.0 lakh/cu mm. Peripheral smear showing microcytic hypochromic anemia. LFT - total bilirubin - 1.0 mg/dl, SGOT – 19 U/L, SGPT – 10 U/L, ALP – 222 U/L, Total protein - 7.7 g/dl, Albumin - 3.4 g/dl, Globulin - 4.3 g/dl. Lipid profile - Normal. Serum Electrolytes: Na-142 mmol/L, K - 3.6 mmol/L, Cl – 101 mmol/L. RBS – 250 mg/dl. Blood urea – 34 mg/dl. Serum creatinine -1.0 mg/dl. ADA - 4.6 U/L. Serum Iron Conc - 31.8 microgram/dl, TIBC - 288 microgram/dl, Thyroid profile-normal, Anti ds DNA- 10 (ref:12-44), ANA profile – Normal. X-ray Chest PA View (**Figure - 1**) Shows cardiomegaly with pulmonary arterial hypertension. USG abdomen: Coarse echotexture with surface irregularity of liver was seen. Cholelithiasis with 14mm calculi in lumen of gallbladder was present. Moderate ascites with mild right sided pleural effusion was noted. Both kidneys were normal.

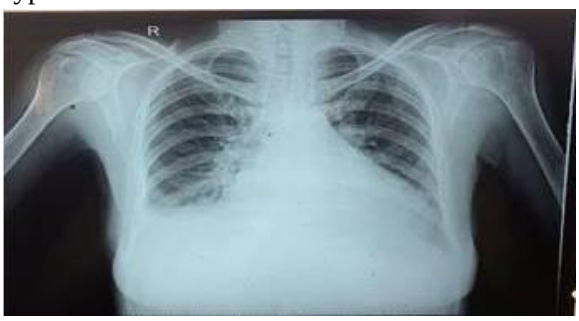
ECG: Rate-100/min, Rhythm –Sinus rhythm, T wave inversion in I, Avl.

Ascitic Fluid Analysis: Protein - 3.8 gm/dl (exudate), Albumin - 1.9 g/dl, LDH - 74.9, Total cell count 200 cells/cu mm with neutrophils 15% and 85% lymphocytes.

Pleural Fluid Analysis: Protein - 2.2 gm/dl, Albumin – 1.2 gm/dl, Sugar – 155 mg/dl, Total cell count 100 cells/cu mm with 5% neutrophils and 95% lymphocytes.

CBNAAT - Negative; 2D ECHO: Mitral valve – MAC +, Aortic valve sclerosis present, EF=30%.Dilated Left Atrium, Right Ventricle, Right Atrium was present. Pericardial thickness-3.0 mm, there was minimal pericardial effusion. Severe left ventricular dysfunction was noted with Grade III Diastolic dysfunction. Moderate Mitral Regurgitation, Mild Aortic Regurgitation were noted. Severe Tricuspid Regurgitation and Pulmonary Artery Hypertension were seen. RVSP 90 mm of Hg was seen.

Figure – 1: X-ray Chest PA View – shows cardiomegaly with pulmonary arterial hypertension.



Diagnosis

A diagnosis of tuberculous constrictive pericarditis along with tubercular peritonitis and tubercular pleural effusion along with HTN and NIDDM and old case of CVA with right MCA infarct with right Cerebral thrombosis and left Hemiparesis was made.

Treatment given

- AKT4 1 strip daily for 2 months followed by AKT3 1 strip daily for 4 months.
- TAB. LASIX 40 mg PO/OD
- TAB. ENAM 2.5 mg PO/OD
- INJ. H. ACTRAPID 4 Units S/C TID
- TAB. ECOSPRIN 75 mg PO/OD
- TAB. STAMLO 5 mg PO/OD
- INJ. OPTINEURON 1 amp iv OD
- TAB. WYSOLONE 5 mg PO/OD x 1 month
- Diabetic diet.

- Mild physical exercise –walking

Discussion

Polyserositis (PS) is characterized by inflammation of multiple serous membranes with effusion in the serous cavities [17]. PS may be part of a wider syndromic presentation of an underlying disease (TB, systemic lupus erythematosus, familial Mediterranean fever, certain malignancies, asbestosis, silicosis), complications of chest surgery, radiation therapy, and certain drugs. Rarely, it is idiopathic [18, 19]. Most reports of serositis in the literature describe cases involving one or two serous membranes, with the majority being cases of pleural effusion, ascites, or constrictive pericarditis secondary to TB or as sequelae of autoimmune diseases such as systemic lupus erythematosus, mixed connective tissue disease, or familial Mediterranean fever [14, 19-24]. We herewith present a case of polyserositis with the simultaneous involvement of all 3 body cavities.

Tuberculosis is one of the common infectious diseases globally. It stands among top ten causes of death [25]. Among different forms of TB, disseminated TB is one of the life-threatening conditions. Exact epidemiology of disseminated TB is not known, but, it is estimated to be less than 2% of all TB cases and up to 20% of extra-pulmonary cases [6]. The definition of disseminated TB involves hematogenous dissemination of Mycobacterium tuberculosis in two or more non-contiguous sites. It can occur through primary focus, reactivation of latent TB (post-primary TB) or rarely through iatrogenic origin. The occurrence of disseminated TB is more with immunocompromised people [4, 6, 26-28]. The exact mechanism for dissemination is not clear. However, dissemination of TB bacilli into the pulmonary vein through the erosion of epithelial layer of alveolar cell is one of the proposed mechanisms for disseminated TB [6, 29]. The disseminated TB presents with non-specific signs and symptoms based on the organs involved. It can range from anorexia, fever with chills and rigor, cough, weight loss, fatigue, lymphadenopathy, ascites, pleural and pericardial effusion and anemia [4, 6]. The involvement of lung, abdomen and heart as polyserositis in disseminated TB is rare as presented in our patient [7].

Polyserositis has been defined as inflammatory membranous thickening lining great serous sacs [30]. Since polyserositis is the presentation in disseminated TB, it can guide the diagnosis of disseminated TB through fluid analysis [31, 32]. The polyserositis due to TB is usually exudative. The liver pathology, especially portal hypertension secondary to liver cirrhosis was ruled out as the clinical and laboratory parameters for liver pathology were normal in our patient. Similarly, the pleural and pericardial fluid analysis based on Light's criteria was against the exudative effusion. But the cells analysis in pleural and ascitic fluid had lymphocytic predominance which was in favor TB pathology. The likely explanation for this kind of result is hypoalbuminemia, which was solely seen in all of the fluid analysis. The link between tuberculosis and nutrition has been studied for many years. The effect of tuberculosis on nutritional state has negative impact with abnormal protein metabolism, so-called anabolic block, due to abnormal cytokine activation [33, 34]. Low albumin level is the consequence of altered nutritional status due to tuberculosis [35, 36]. Hypoalbuminemia has also been found as the predictive factor for prognosis in TB patients [35].

Hence, as in our case the exudative nature of the fluid was due to TB. The evidence of lymphocytic predominance in fluid analysis furthermore confirmed the diagnosis of disseminated TB. Polyserositis in disseminated TB is itself a very rare presentation. In addition to this, the confusing nature of polyserositis makes the diagnosis of disseminated tuberculosis more difficult.

A review of the literature shows that there is a tendency to discuss polyserositis in relation to constrictive pericarditis, particularly of

tuberculous origin [37]. In 1896, Pick quoted by White [38] described 3 cases of constrictive pericarditis with 'pseudocirrhosis' of the liver resulting from chronic adhesive pericarditis involving the mediastinum. Two cases were due to tuberculosis and the third was of unknown aetiology. Osler [39] says: 'In all forms of chronic peritonitis ... polyorrhymenitis, general chronic inflammation of the serous membranes, Concato's disease (as the Italians call it) may occur in this form as well as in the tuberculous variety. The pericardium and both pleurae may be involved.'

In 1942, Harrison and White [40] reviewed a series of 37 cases of constrictive pericarditis; 5 of them were ascribed to tuberculosis and 3 to other infections and in 29 the cause was either - unknown' or 'questionable'. In 1944, Paul Dudley White [38] writes that 'ascites may also be a part of polyserositis (Concato's disease) which forms the background for constrictive pericarditis (Pick's disease)'. He then makes the significant statement that although polyserositis may eventually be responsible for constrictive pericarditis 'these two conditions have often been confused in the past'. Paul Wood [41] described polyserositis as follows: 'Whilst tuberculosis may affect the pleura and peritoneum as well as the pericardium, the term polyserositis (Concato's disease) is usually reserved for a somewhat similar inflammatory process of unknown origin. Large effusions collect in the serous sacs, the fluid being a clear or opalescent, straw-coloured, sterile exudate. When the pericardium is involved, resorption of fluid is followed by total obliteration of the pericardial cavity, and constriction may ensue. The course and prognosis are similar to those of tuberculous pericardial effusion.' In the absence of a known cause of polyserositis as described by Wood, the possibility of a constrictive pericarditis following at a later date again introduces the likelihood of a tuberculous or other infective process as the cause.

The lack of a diagnostic algorithm for PS has led to the development of a wide variety of diagnostic approaches, especially when initial investigations (i.e. serological, biochemical, cytological and microbiological testing) do not allow for a clear diagnosis. Losada in his series, pleura and pericardium were the most common sites of serosal involvement (83%), and the final diagnosis was neoplasm in nearly one third of cases, followed by infectious and autoimmune diseases. Idiopathic PS was diagnosed in 38% of patients. Laboratory tests as the determination of ANA in serum or ADA and LDH levels in fluids have been used in the initial diagnostic approach [14].

Conclusion

Polyserositis is a chronic inflammation of several serous membranes with effusions in serous cavities like Pericardial, Pleural and Peritoneal membranes, resulting in fibrous thickening of the serous membranes and sometimes constrictive pericarditis. There are various causes of polyserositis. While tuberculosis may affect the pleura and peritoneum as well as the pericardium, the term polyserositis (Concato's disease) is usually reserved for a somewhat similar inflammatory process of unknown origin. Large effusions collect in the serous sacs, the fluid being a clear or opalescent, straw-coloured, sterile exudate. When the pericardium is involved, resorption of fluid is followed by total obliteration of the pericardial cavity, and constriction may ensue. There is a tendency to discuss polyserositis in relation to constrictive pericarditis, particularly of tuberculous origin. Polyserositis in disseminated TB is very rare. But in high incidence areas of TB, like India, TB may be the commonest cause of polyserositis. Diagnosis is often delayed due to the non-specific presentation like polyserositis and its unusual nature.

In our case the exudative nature of the fluid was due to TB, as evidenced by lymphocytic predominance in fluid analysis. Polyserositis in disseminated TB is a very rare presentation. In addition to this, the confusing nature of polyserositis makes the diagnosis of

disseminated tuberculosis more difficult. Hence, we herewith report a rare case of TB polyserositis, involving pleura, pericardium and peritoneum.

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