

Unusual association of diseases/symptoms

Tuberculous pericarditis associated with hoarseness of voice due to left recurrent laryngeal nerve paralysis

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Summary

A 16-years-old Egyptian girl presented with massive pericardial effusion, fever, weight loss and hoarseness of voice. Laryngoscopy showed left vocal cord paralysis. Chest CT revealed pericardial effusion, amalgamated mediastinal lymph nodes and clear lung fields. Pericardial fluid analysis revealed a lymphocytic exudate with high adenosine deaminase enzyme level, negative stains and cultures for bacteria and fungi. Despite a negative nucleic acid test for tuberculosis; antituberculous and corticosteroids therapies resulted in resolution of pericardial effusion after 3 weeks but hoarseness of voice persisted. Few cases of vocal cord paralysis with tuberculous mediastinal lymphadenopathy were reported in English literature.

BACKGROUND

Tuberculous pericarditis is a rare cause of pericardial effusion in developed countries, while it is the cause of up to 70% of cases in developing countries. Despite that almost all cases of tuberculous pericarditis are associated with mediastinal lymphadenopathy, only few cases of left vocal cord paralysis with tuberculous mediastinal lymphadenopathy were reported in English literature.

CASE PRESENTATION

A 16-year-old Egyptian girl had fatigue and shortness of breath for the last 6 months. A blood count revealed microcytic anaemia with a haemoglobin (Hb) of 7.6 grams/dl. She was treated with ferrous sulphate tablets. Three months later, she complained of fever, night sweats, bony aches and hoarseness of voice. She lost 23 kilograms over the last year. A large pericardial effusion was found during an investigation done at another facility; the patient was referred to this hospital for further management. On physical examination; she was cachectic with a body mass index of 16. Blood pressure was 130/80mm Hg, pulse rate of 120/min, temperature of 37.4°C and respiratory rate of 20/min. The jugular venous pressure was elevated and there were bilateral firm non-tender upper deep cervical lymph nodes.

INVESTIGATIONS

A flexible direct laryngoscopy revealed normal appearance of vocal cords without any lesions or inflammation, but markedly diminished adduction and abduction of the left vocal cord diagnosing left vocal cord paralysis.

The Hb was 7.6 grams/dl, white blood cell count was 6400/cmm and platelet count was 719000/cmm. The erythrocyte sedimentation rate (ESR) was 150 mm/h. Chest radiograph revealed cardiomegaly. Electrocardiogram showed

sinus tachycardia, and low voltage. Chest CT (figures 1 and 2) revealed large pericardial effusion, clear lung fields and amalgamated mediastinal lymph nodes showing areas of break down. Transthoracic echocardiography (TTE) revealed large pericardial effusion, fibrinous strands in the pericardial cavity (figure 3) and right ventricular diastolic collapse. A 600 millilitres of amber coloured pericardial fluid was drained (2520 cells/cmm, mostly lymphocytes, protein 6.4 g/dl and lactate dehydrogenase 627). Bacterial

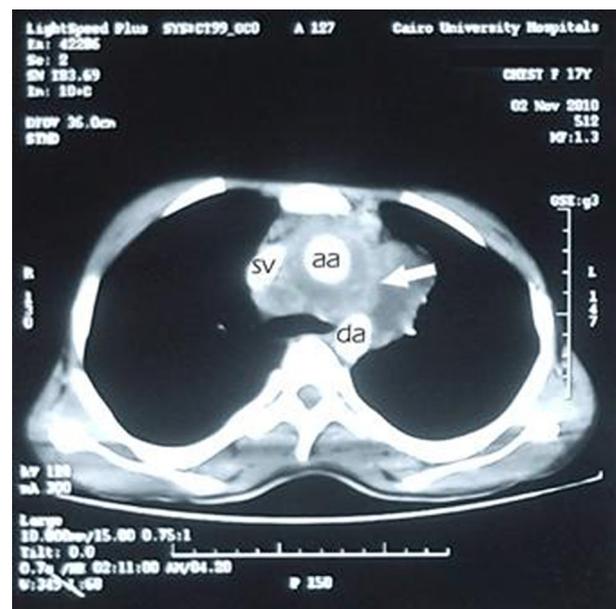


Figure 1 Postcontrast CT scan of the chest at the level of the aorto-pulmonary window. Caseating aorto-pulmonary (block arrow) lymph nodes forming a bulky nodal amalgum. aa, ascending aorta; da, descending aorta; sv, superior vena cava.

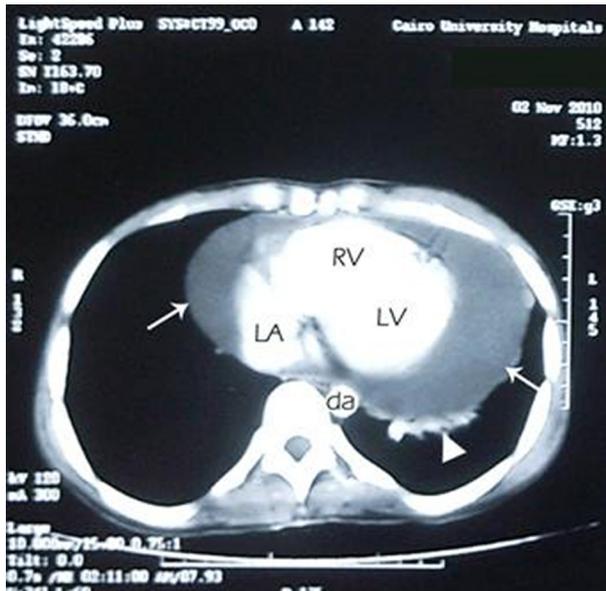


Figure 2 Postcontrast CT scan of the chest at the level of the cardiac chambers. Massive pericardial effusion (arrows) with associated atelectatic changes of the lingula (arrowheads). da, descending aorta; LA, left atrium; LV, left ventricle; RV, right ventricle.

and fungal cultures were negative. Acid fast bacilli and fungal stains were also negative. Adenosine deaminase in the pericardial fluid was high 61.8. The fluid cytology was negative for cancer cells.

DIFFERENTIAL DIAGNOSIS

- ▶ Tuberculous pericardial effusion
- ▶ Purulent pericardial effusion
- ▶ Malignant pericardial effusion.

TREATMENT

A presumptive diagnosis of probable tuberculous pericarditis was made. Treatment with rifampicin, isoniazid, ethambutol and pyrazinamide according to body weight was started in addition to 30 mg of prednisone.

OUTCOME AND FOLLOW-UP

After 3 weeks, she improved dramatically, her appetite improved, and she gained 4 kilograms. The ESR decrease to 30 mm/h, the Hb increased to 12 grams/dl and repeated TTE showed complete resolution of pericardial effusion. The patient continued to have hoarseness of voice.

After 6 months of antituberculous therapy, she has no recurrence of pericardial effusion but she continued to have hoarseness of voice despite speech therapy. She did not decide regarding doing reconstructive vocal cord surgery yet.

DISCUSSION

Tuberculosis causes 70% of cases of large pericardial effusion and most cases of constrictive pericarditis in

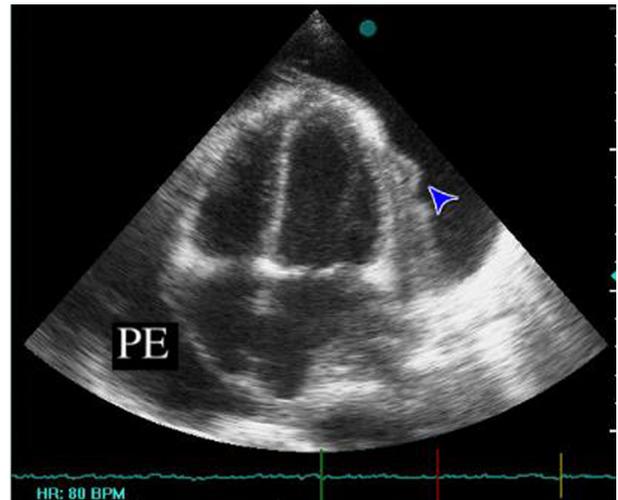


Figure 3 Apical four chambers transthoracic echocardiographic view shows global pericardial effusion (PE) and pericardial adhesions (arrow head).

developing countries, while it accounts for only 4% of cases of pericardial effusion and a smaller proportion of constrictive pericarditis cases in developed countries. Tuberculous pericarditis is a dangerous disease with a mortality of 17% to 40%.¹

Tubercle bacilli reach the pericardium from the adjacent tracheobronchial lymph nodes either directly or via lymphatic channels. Less commonly seeding of the pericardium occurs with miliary tuberculosis and rarely mycobacteria spread directly from pleura or adjacent rib.²

The pericardium drains its lymphatic drainage to the anterior and posterior mediastinal lymph nodes and tracheobronchial lymph nodes. These lymph nodes are the ones involved in tuberculous pericarditis. The hilar lymph nodes are the least to be involved. These enlarged lymph nodes are not seen on chest radiographs but on chest CT they are seen in 100% of cases with caseation and amalgamation (as seen in our case) in more than half of the cases, in addition to a thickened, irregular, non-calcified pericardium with or without pericardial effusion.³

Echocardiography is the test of choice for diagnosing pericardial effusion and tamponade. Fibrinous strands from the visceral pericardium and pericardial thickening are typical but not specific for tuberculous pericarditis. It is also used to guide needle pericardiocentesis.⁴

Left recurrent laryngeal nerve has a unique anatomical location in the aorto- pulmonary window where enlarged tuberculous mediastinal lymph nodes and inflammatory swelling can cause traction and compression on that nerve.

Despite that recurrent laryngeal nerve paralysis is extremely rare in those with tuberculous mediastinal lymphadenopathy, with few cases reported in English literature.⁵ We report a rare case of massive tuberculous pericardial effusion associated with mediastinal lymphadenopathy and left recurrent laryngeal nerve paralysis.

Learning points

- ▶ Tuberculous pericarditis is a common cause of pericardial effusion in developing countries.
- ▶ Negative tuberculin skin test or a pericardial fluid PCR for tuberculosis does not exclude tuberculous pericarditis.
- ▶ Diagnostic pericardiocentesis should be done for any case of suspected tuberculous pericarditis.
- ▶ Lymphocyte predominant pericardial exudate with high adenosine deaminase level suggests tuberculous pericarditis, and response to antituberculous therapy confirms the diagnosis.
- ▶ Left recurrent nerve paralysis is due to a lesion along the course of the nerve from the base of the skull till the level of the aortic arch.

Competing interests None.

Patient consent Obtained.

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