INTRODUCTION

Pericarditis refers to inflammation of the pericardium, two thin layers of a sac-like tissue that surround the heart, hold it in place and help it work. A small amount of fluid keeps the layers separate so that there’s no friction between them. Primary pericarditis is unusual and almost always from neoplasms arising in remote sites, or a surgical procedure on the heart. Pericardial inflammation is usually secondary to a variety of cardiac diseases, thoracic or systemic disorders, metastases from neoplasms arising in remote sites, or a surgical procedure on the heart. Primary pericarditis is unusual and almost always of viral origin. The major causes of pericarditis evoke an acute pericarditis, but a few, such as tuberculosis and fungi, produce chronic reactions. Tuberculous pericarditis, caused by *Mycobacterium tuberculosis*, is found in approximately 1% of all autopsied cases of TB and in 1% to 2% of instances of pulmonary TB. It is the most common cause of pericarditis in Africa and other countries in which TB remains a major public health problem.

Here, I present a case report of a 36 year old male who had chest pain and breathlessness since 1 day and expired. He was a known case of pulmonary tuberculosis since 3 months and was on irregular treatment. During autopsy, Pericardium was thickened and adherent to the heart at places. Greyish white thick granulomatous patches were present over the heart at places. On histopathological examination of the heart, it was diagnosed as tuberculous pericarditis due to the presence of caseating granulomas over heart surface which also showed fibrosis and presence of chronic inflammatory cells like lymphocytes.

**ARTICLE INFO**

**Article History:**
Received 15th February, 2020
Received in revised form 7th March, 2020
Accepted 13th April, 2020
Published online 28th May, 2020

**Key Words:**
Pericarditis, Tuberculous pericarditis, Pericardial inflammation, Caseating granulomas over heart surface, *Mycobacterium tuberculosis*, Primary pericarditis, Acute pericarditis, Chronic pericarditis.

**ABSTRACT**

Pericarditis refers to inflammation of the pericardium, two thin layers of a sac-like tissue that surround the heart, hold it in place and help it work. A small amount of fluid keeps the layers separate so that there’s no friction between them. Pericardial inflammation is usually secondary to a variety of cardiac diseases, thoracic or systemic disorders, metastases from neoplasms arising in remote sites, or a surgical procedure on the heart. Primary pericarditis is unusual and almost always of viral origin. The major causes of pericarditis evoke an acute pericarditis, but a few, such as tuberculosis and fungi, produce chronic reactions. Tuberculous pericarditis, caused by *Mycobacterium tuberculosis*, is found in approximately 1% of all autopsied cases of TB and in 1% to 2% of instances of pulmonary TB. It is the most common cause of pericarditis in Africa and other countries in which TB remains a major public health problem.

Here, I present a case report of a 36 year old male who had chest pain and breathlessness since 1 day and expired. He was a known case of pulmonary tuberculosis since 3 months and was on irregular treatment. During autopsy, Pericardium was thickened and adherent to the heart at places. Greyish white thick granulomatous patches were present over the heart at places. On histopathological examination of the heart, it was diagnosed as tuberculous pericarditis due to the presence of caseating granulomas over heart surface which also showed fibrosis and presence of chronic inflammatory cells like lymphocytes.

**INTRODUCTION**

Pericarditis refers to inflammation of the pericardium, two thin layers of a sac-like tissue that surround the heart, hold it in place and help it work. A small amount of fluid keeps the layers separate so that there’s no friction between them.¹ Pericardial inflammation is usually secondary to a variety of cardiac diseases, thoracic or systemic disorders, metastases from neoplasms arising in remote sites, or a surgical procedure on the heart. Primary pericarditis is unusual and almost always of viral origin. The major causes of pericarditis evoke an acute pericarditis, but a few, such as tuberculosis and fungi, produce chronic reactions.

The causes of pericarditis are as follows

2. **Immune mediated**- Rheumatic fever, Systemic lupus erythematosus, Scleroderma, Post cardiotomy, Post myocardial infarction (Dressler) syndrome, and Drug hypersensitivity reaction.

3. **Miscellaneous**- Myocardial infarction, Uremia, Following cardiac surgery, Neoplasia, Trauma, and Radiation.

Acute pericarditis is of different types e.g. Serous pericarditis, Fibrinous pericarditis, Serofibrinous pericarditis, Purulent or suppurative pericarditis, Hemorrhagic pericarditis and Caseous pericarditis.

Chronic or Healed pericarditis include Adhesive mediastino pericarditis and Constrictive pericarditis. Healed pericarditis is sometimes called as “Soldier’s plaque”.²³ Tuberculous pericarditis, caused by *Mycobacterium tuberculosis*, is found in approximately 1% of all autopsied cases of TB and in 1% to 2% of instances of pulmonary TB.⁴ It is the most common cause of pericarditis in Africa and other countries in which TB remains a major public health problem.⁵ The incidence of tuberculous pericarditis in sub-Saharan Africa is increasing as a result of the human
immunodeficiency virus (HIV) epidemic, and this trend is likely to appear in other parts of the world where the spread of HIV is leading to a resurgence of TB.  

Pericardial involvement usually develops by retrograde lymphatic spread of *M. tuberculosis* from peritracheal, peribronchial, or mediastinal lymph nodes or by hematogenous spread from primary tuberculous infection.  

**Case report**

A 36 years old male was brought to the Victoria hospital, Bangalore Medical College and Research Institute, Bangalore with chief complaints of chest pain and breathlessness since 1 day. He was a known case of pulmonary tuberculosis since 3 months and was on irregular treatment. He was a laborer but since few months he was unable to do any work due to his illness. History of fever on and off was present. History of cough and weight loss was also present. On further asking he gave history of similar episodes of chest pain and breathlessness previously on few occasions. He collapsed after sometime in hospital and could not be revived, and declared dead after some time. There was no history of any alcohol intake or smoking habits. There was no history of Diabetes, hypertension, heart disease or any other disease except pulmonary tuberculosis. There was no family history of Diabetes, Hypertension, tuberculosis or any congenital heart disease. Autopsy was conducted at the mortuary of Victoria hospital, Bangalore Medical College and Research Institute, Bangalore. On external examination the dead body measured 152 cm in length, weighing 50 kg, moderately built and poorly nourished. Body was emaciated. Rigor mortis was present all over the body. Post mortem staining was seen over the back of the body. There were no external injuries on the body. On internal examination, Pericardium was thickened and adherent to the heart at places. Heart weighed 350 gms. Greyish white thick granulomatous patches were present over the heart at places. Right ventricle wall thickness measured 0.8 cm. Left ventricle wall thickness measured 1.5 cm. Interventricular septum thickness measured 1.7 cm. Heart valves were normal. All coronaries were patent. Pleura were adherent to the lungs and chest wall on both sides over apical region. Apical lobes of both Lungs were consolidated and on cut section showed fibrocavitary lesions filled with caseous material and pus. Other internal organs were congested. On histopathological examination of the heart, it was diagnosed as tuberculous pericarditis due to the presence of caseating granulomas over heart surface which also showed fibrosis and presence of chronic inflammatory cells like lymphocytes microscopically. Histopathological examination of the lungs was suggestive of pulmonary tuberculosis.  

**DISCUSSION**

Chronic constrictive pericarditis results when the healing of an acute fibrinous or serofibrinous pericarditis or the resorption of a chronic pericardial effusion is followed by obliteration of the pericardial cavity with the formation of granulation tissue.  

In India, tuberculosis accounts for nearly two-thirds of all cases of constrictive pericarditis. Constrictive pericarditis is a serious sequel of tuberculous pericarditis, developing in approximately 60-80% of the patients despite receiving treatment with antitubercular drugs and prednisolone.  

Wanjari K et al reported a case of tuberculous pericardial effusion in a 30-year-old male who presented with fever, chills, and dry nonproductive cough since one month. The case was diagnosed by radiological findings, which were suggestive of pulmonary tuberculosis, followed by acid fast staining and culture of the aspirated pericardial fluid. The patient was responding to antitubercular treatment at the last follow up. Pannu AK et al, in 2018 described a patient who presented with prolonged fever and was subsequently diagnosed as pericardial TB. He was successfully treated with ATT and adjuvant steroid therapy.  

Faria D et al, in 2018 reported a case of tuberculous pericarditis in a 29-year-old woman who presented with fever, dyspnea, and hypotension and was found to have a large pericardial effusion with extensive fibrin strands.  

Mastroianni A et al reported two cases of tuberculous pericarditis in adult patients with AIDS occurred over a 12-month period at their clinic. In the first patient pericardial effusion represented the AIDS-defining illness and was an expression of a disseminated tuberculous disease. The second patient developed a fatal pericarditis due to a multiple-drug resistant *Mycobacterium tuberculosis* infection.  

In the present case, tuberculous pericarditis was found during autopsy on histopathological examination of the heart, in a 36 year old male who had chest pain and breathlessness since 1 day and expired. He was a known case of pulmonary tuberculosis since 3 months and was on irregular treatment. History of fever on and off was present. History of cough and weight loss was also present. History of similar episodes of chest pain and breathlessness was also present previously on few occasions. There was no history of any alcohol intake or smoking habits. There was no history of Diabetes, hypertension, heart disease or any other disease except pulmonary tuberculosis. There was no family history of Diabetes, Hypertension, tuberculosis or any congenital heart disease.  

**CONCLUSION**

In this case report a young male who was a laborer, had chest pain and breathlessness since 1 day. He was brought to the hospital where he collapsed after sometime and could not be revived, and declared dead after some time. He was a known case of pulmonary tuberculosis since 3 months and was on irregular treatment. History of fever on and off was present. History of cough and weight loss was also present. History of similar episodes of chest pain and breathlessness was also present previously on few occasions. During autopsy, Pericardium was thickened and adherent to the heart at places. Greyish white thick granulomatous patches were present over the heart at places. On histopathological examination of the heart, it was diagnosed as tuberculous pericarditis due to the presence of caseating granulomas over heart surface which also showed fibrosis and presence of chronic inflammatory cells like lymphocytes microscopically. Pleura were adherent to the lungs and chest wall on both sides over apical region. Apical lobes of both Lungs were consolidated and on cut section showed fibrocavitary lesions filled with caseous material and pus. Histopathological examination of the lungs was suggestive of pulmonary tuberculosis. This case emphasises the requirement of essential investigations to detect this cardiac...
pathology and proper treatment to prevent death in young individuals. It also highlights the role of meticulous autopsy and histopathological examination to detect this condition.

Acknowledgements: I would like to thank and acknowledge Dr. P. K. Devadass, Retd. Professor of Forensic Medicine, Ex. Dean & Director, and Dr. Dayanand S. Biligi, Professor & Head, Dept. of Pathology, Bangalore Medical College & Research Institute, Bangalore for their guidance, constant support and encouragement.

Conflict of Interest: None.

Financial Assistance: None.

References

How to cite this article:
DOI: http://dx.doi.org/10.24327/ijrser.2020.1105.5357

******