Introduction

Endocarditis is referred to as the infection in the endocardium, the inner lining of the heart chambers and heart valves. Infective Endocarditis is associated with substantial morbidity & mortality. Several published studies have reported in-hospital mortality of 15 percent to 20 percent and 1-year mortality of 40 percent. In the United States alone, approximately 15,000 new cases of infective Endocarditis are diagnosed each year. However, Endocarditis caused by Mycobacterium Tuberculosis is very rare. The organism itself is exceptionally resistant to conventional antibiotics, requiring a particular combination of antibiotics (e.g., Isoniazid, Rifampin, Ethambutol, Pyrazinamide). The treatment duration can last for months. Mycobacterium Tuberculosis yet remains a global threat being a major cause of death. It has the potential to infect every organ of the body including heart. The very first cases of Tuberculous Endocarditis (TBE) were reported in 1892. Afterward, many other cases have been described, highlighting the significant morbidity and mortality associated with this manifestation of TB. TBE usually presents with miliary tuberculosis and most early cases were diagnosed on autopsy. With the increasing application of prosthetic valve replacements in the treatment of Infective Endocarditis (IE), TB infections have begun to affect these as well as native valves. With the introduction of TB culture methods and drug therapy, the prognosis has improved. The rarity of this disease and the resistance of the bacteria and treatment duration makes it difficult to treat disease with a high mortality rate.

Case Report:

Mr. M a 5 years old boy presented to our cardiology Department, Rangpur medical college Hospital, Rangpur on 17th February 2020 with a history of fever for 20 days. He had also history of respiratory distress for 10 days, pain in the lower limb for 10 days. General examination revealed that patient is anemic, hepatomegaly present and temperature is raised. Complete Blood Count reveals Hb% 9.8 gm/dl, WBC- 11,000/cu mm, on Chest X-ray shows huge cardiomegaly. Echo finding was – huge pericardial effusion (27mm posteriorly, 13mm anteriorly & 20mm laterally at LV site and 17 mm at RV site) features of early tamponade. Pericardial fluid was drawn and the symptoms improved. Pericardial fluid colour was milky and exudative in nature. ADA for mycobacterium tuberculosis was positive. The patient was given anti tuberculosis drug & improved with time.
also vegetation attached to the upstream site of the anterior and septal leaflets of tricuspid valve and vegetation also attached to the PML of Mitral valve. Pericardiocentesis was done and 300 ml milky coloured pericardial fluid was aspirated and sent for examination. Pericardial fluid report was- ADA- 195 U/L, protein 9 gm/dl, sugar 00 ìg/dl, pus cells – plenty.

So, the final diagnosis was made as tuberculous pericardial effusion with mycobacterial endocarditis. As soon as the pericardiocentesis was done the patient felt comfortable and anti-tuberculosis drugs (four drug regimen) was given as per the regiment for a 5 years child weighing 14.5 kg. With the above regimen, the patient showed good result during follow up.

Discussion:
Laennec, in 1826 recognized and described Cardiac tuberculosis and assigned the heart as the 13th organ affected in order of frequency. In 1906, among 7683 cases of tuberculosis, 49 cases (0.62%) reported myocardial tuberculosis. Pathological post-mortem studies have demonstrated possible tuberculous involvement in all anatomical structures of the heart. Valvular Endocarditis resulting from M. tuberculosis is extremely rare and have only been reported in miliary tuberculosis and after replacement of the aortic valve. Disseminated tuberculosis have been reported with echocardiographic documented aortic valvulitis. These were resolved on antituberculosis therapy.
Most reports of mycobacteria involvement in endocarditis in immunocompromised patients in the context of miliary tuberculosis. Usually patients affected with Human immunodeficiency virus infection or acquired immune deficiency syndrome or patients with glucocorticoid therapy reported endocarditis by M. Tuberculosis. There have been very few cases of tuberculous infection in immunocompetent patients. However, this doesn’t indicate an exception in case in immunocompetent host. Sheikh et al. reported a case of triple valve endocarditis by mycobacterium tuberculosis. The host, a 17-year-old girl was immunocompetent and demonstrated infective endocarditis in the aortic, mitral and tricuspid valves. After developing a right middle cerebral artery stroke, a dual valve replacement surgery and tricuspid repair had been performed. The patient had been assigned an anti-tuberculosis regimen with and an anti-coagulant. To their knowledge this was the first case of mycobacteria involvement in endocarditis in immunocompetent patients.

The case was a first reported case in Bangladesh but well-handled as patient showed signs of recovery and no complications were faced. Further mass spectroscopic analysis of the pericardial fluid protein could have given us a better understanding of the infection but the urgency of treatment was a major focus in this case. Future recommendations can be made to analyze the protein composition of the pericardial fluid.

Conflict of Interest - None.

References: