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Perspective

Pathogenesis and Treatment of Tuberculous Pericarditis

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DESCRIPTION

The pericardium, which is composed of two layers, pleural and visceral, keeps the heart in place and serves as a barrier against infections. They are of viral, fungal, and bacterial origin, and they can be caused by Mycobacteria.

The inflammation of the pericardium caused by Koch's bacillus is known as tuberculous pericarditis. It is accessed *via* three mechanisms: (1) Lymphatic (2) Hematogenous spread, which occurs primarily in immunocompetent patients, and (3) Direct contact from adjacent structures such as the lung and pleura.

The condition is typically paucibacillary in immunocompetent patients, manifesting at the level of a single organ, whereas in immunocompromised patients, the rate of bacterial replication is high. Tuberculosis (TB) is a disease that has yet to be eradicated. Despite the fact that pulmonary involvement is confirmed in the vast majority of cases, a large number of patients experience complications from other organs. If tuberculous pericarditis is suspected, it is critical to make an early diagnosis in order to begin appropriate treatment as soon as possible.

Pathogenesis of tuberculous pericarditis

Tubercle bacilli enter the pericardium through three routes: (1) Retrograde lymphatic spread from mediastinal, paratracheal, and peribronchial lymph nodes, (2) Hematogenous spread (predominant in immunocompromised hosts), and (3) Direct contiguous spread from adjacent structures like the lungs, pleura, and spine (infrequent).

Tuberculous pericardial disease is limited to the pericardial space when the host is immunocompetent. Tubercle proteins typically cause an important cell-mediated hypersensitivity response with T-helper cell (subtype 1) predominant cytokine release, resulting in an inflammatory exudative effusion and its hemodynamic sequelae in a paucibacillary condition. The morbidity associated with tuberculous pericarditis is caused by the immune response to viable acid-fast bacilli penetrating the pericardium.

Tuberculous pericarditis has four pathological stages: (1) Fibrinous exudation, initial polymorphonuclear leukocytosis, abundant mycobacteria, and early granuloma formation with loose organisation of macrophages and T cells; (2) Sserosanguineous effusion with a predominantly lymphocytic exudate with monocytes and foam cells; (3) Absorption of effusion with organisation of granulomatous caseation and; (4) Constrictive scarring. The fibrosis formed between the visceral and parietal pericardiums can calcify and adhere to the myocardium, forming a cuirass around the heart, preventing proper diastolic filling, and resulting in the clinical syndrome of constrictive pericarditis.

Treatment

Even in HIV-positive patients, pharmacological treatment improves survival in tuberculous pericarditis. In extrapulmonary tuberculosis, a regimen of rifampicin, isoniazid, pyrazinamide, and ethambutol for at least 2 months, followed by rifampicin and isoniazid (up to 6 months) proved effective. Treatments that last longer than 6 months do not produce better results, increasing cost and decreasing tolerance.

Corticosteroid treatment would not be justified at this time, given the lack of evidence supporting its use. Although the results are inconclusive, adding corticosteroids to treatment may reduce morbidity and relapses, but randomised controlled trials with a large enough sample size of HIV-positive and HIV-negative patients are required.

If pericarditis is associated with severe pericardial effusion and hemodynamic compromise, drainage via subxiphoid puncture or minimal thoracotomy is the first treatment option associated with antituberculous treatment.

CONCLUSION

Tuberculous pericarditis is uncommon in developed countries but common in developing countries. Because of the low bacteriological and histological results, the diagnosis is difficult. In patients with torpid pericarditis, the utility of indirect diagnostic methods should be considered. The presence of HIV-positive serology can alter the clinical course and outcome of tuberculous pericarditis. There is still insufficient evidence to support the use of systemic corticosteroids in this pathology.

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