Research & Reviews: Journal of Medical and Health Sciences ISSN: 2319-9865

Pathophysiology of Rheumatic Fever: Signs and Symptoms

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Opinion Article

Received: 05-May-2023, Manuscript No. JMAHS-23-97992; **Editor assigned:** 09-May-2023, Pre QC No. JMAHS-23-97992 (PQ); **Reviewed:** 23-May-2023, QC No. JMAHS-23-97992; **Revised:** 30-May-2023, Manuscript No. JMAHS-23-97992 (R); **Published:** 06-Jun-2023, DOI: 10.4172/2319-9865.12.2.005.

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Citation: Zhang H, Pathophysiology of Rheumatic Fever: Signs and Symptoms. RRJ Med Health Sci. 2023;12:005.

DESCRIPTION

An inflammatory condition known as Rheumatic Fever (RF) can affect the heart, joints, skin, and brain. The illness ordinarily creates two to four weeks after a streptococcal throat disease.

Rheumatic fever might happen following a contamination of the throat by the bacterium *Streptococcus pyogenes*. Rheumatic fever can affect up to 3% of people if the infection is not treated. Determination of RF is in many cases in view of the presence of signs and side effects in blend with proof of a new *streptococcal* contamination. The risk of developing rheumatic fever is reduced when people who have strep throat are treated with antibiotics like penicillin. To keep away from anti-infection abuse this frequently includes testing individuals with sore throats for the disease; however, the developing world might not have access to testing. Other preventive measures incorporate superior disinfection. Some people with rheumatic fever and rheumatic heart disease are advised to take antibiotics for a long time. After an attack, it's possible to gradually return to normal activities. It is more difficult to treat RHD once it develops. Periodically valve substitution medical procedure or valve fix is required.

Signs and symptoms

The disease typically manifests itself two to four weeks after a throat infection. The signs include: fever, muscle developments, and infrequently a trademark non-irritated rash known as erythema marinate. About half of the

cases involve the heart. Rheumatic Heart Disease (RHD), or damage to the heart valves, usually occurs after multiple attacks, but it can also happen after one. Heart failure, atrial fibrillation, and valve infection are all possible outcomes of damaged valves.

Pathophysiology

Rheumatoid arthritis is a systemic disease that affects the connective tissue surrounding arterioles. It can happen after an untreated strep throat infection caused by *Streptococcus pyogenes*, a *Group a Streptococcus* (GAS) bacteria. Type II hypersensitivity reactions can occur due to similarities between *Streptococcus pyogenes* antigens and numerous cardiac proteins. Without T cell co-stimulation, self-reactive B cells typically remain anergic in the peripheral region.

Antibodies produced by the immune system against the M protein may cross-react with the protein myosin, which is found in heart muscle cells, glycogen, and arteries' smooth muscle cells, resulting in the release of cytokines and the destruction of tissue. However, the only cross-reaction that has been demonstrated is with connective tissue that is perivascular. This inflammation is caused by the direct attachment of complement and the recruitment of neutrophils and macrophages through the Fc receptor. Light microscopy reveals the swollen eosinophilic collagen of typical Aschoff bodies surrounded by lymphocytes and macrophages. Larger macrophages may develop into Aschoff giant cells. Due to the presence of T-helper cells and macrophages, rheumatic valvular lesions may also involve a cell-mediated immune response.

These lesions can occur in any layer of the heart in rheumatic fever, resulting in various forms of carditis. The irritation might cause a serofibrinous pericardial exudate depicted as "meat and potatoes" pericarditis, which normally settle without sequela. Wart formation and fibroid necrosis typically follow the closure of the left-sided heart valves when the endocardium is involved. Warty projections emerge from the affidavit, while subendocardial injuries might initiate unpredictable thickenings called MacCallum plaques.