HSR (LONDON),
HOUGHTON STREET REVIEW JOURNAL
ISSN: 2058-5519|Impact factor-15.9
http://houghtonstreetreview.com/index.php/hsr

A THOROUGH OVERVIEW OF THE PATHOPHYSIOLOGY, DIAGNOSIS, AND PREVENTION OF RHEUMATIC FEVER AND RHEUMATIC HEART DISEASE

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Doi: https://doi.org/10.5281/zenodo.21236665

Abstract: Following infection with Group A beta-hemolytic streptococcus (GABHS), rheumatism is a systemic inflammatory connective tissue disease that primarily affects the cardiovascular system, particularly in children and adolescents. An autoimmune cross-reactivity between human tissues and streptococcal antigens causes the illness. Fibrotic and degenerative alterations in the myocardium and heart valves are part of the pathophysiology of rheumatic heart disease (RHD), which results in persistent cardiac dysfunction. To avoid problems and irreversible valve damage, early identification and treatment are crucial.

Through prompt antibiotic medication, anti-inflammatory treatment, and long-term prophylaxis, the general practitioner plays a critical role in the diagnosis, treatment, and prevention of rheumatic fever and RHD. Living circumstances, overcrowding, and restricted access to healthcare are examples of social and environmental determinants of health that significantly affect the prevalence of rheumatic fever. With a focus on the significance of preventive care in primary health practice, this article provides an overview of the epidemiology, pathophysiology, diagnostic standards, clinical symptoms, and treatment approaches to rheumatic fever and rheumatic heart disease.

Key words Rheumatic fever; Rheumatic heart disease; Group A beta-hemolytic streptococcus; Autoimmune response; Carditis; Polyarthritis; Chorea; Prevention; Penicillin prophylaxis; General practitioner.

Introduction

Rheumatism is a systemic inflammatory connective tissue disease that mostly affects the cardiovascular system. After an acute infection with group A beta-hemolytic streptococcus (GABHS), it manifests in vulnerable individuals, primarily children and teens. Primary rheumatism is quite rare among the elderly. Women are affected 2.5-3 times more often than men. According to Stollerman, rheumatism is a post-infectious complication of A-streptococcal pharyngitis (tonsillitis) in susceptible people because of the development of an autoimmune reaction to streptococcal epitopes and cross-reactivity with similar epitopes in human tissues (skin, joints, heart, and brain).

The present understanding of the pathophysiology of rheumatism serves as the foundation for this definition. This position influences the most current classification of rheumatic heart disease (RHD) by the American Rheumatology Association (ARA) as a group of reactive syndromes associated with infection. Although it can be fatal, produce rheumatic heart disease, or be the only clinical sign of a painful process that was previously present, heart inflammation is by no means a required indication of this condition. In certain situations, fibrotic alterations in the valve leaflets without a formed heart defect can be found by specialised (echocardiography) investigations, and the murmur of the valve defect will go away after a few years.

Heart valve distortion and scarring, together with fibrous and degenerative alterations in the myocardium, are the causes of rheumatic heart disease (RHD), a chronic ailment. Primary valve

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damage not only results from rheumatic heart disease but also acts as the trigger for further secondary circulatory system rearrangements. Primary valve injury or subvalvular changes, each of which is impacted by distinct intracardiac haemodynamic factors, dictate the rate at which fibrosis and calcification of the leaflets progress. Any recurrence of rheumatic activity is considered rheumatic heart disease, and its severity is assessed.

Given the current challenges in understanding the pathophysiology of rheumatic heart disease, the general practitioner (GP) has a crucial role in the timely diagnosis, treatment, and prevention of this condition. For a watchful observer, each case of the sickness serves as a life experiment, exactly the quality that general practitioners must develop. This makes it possible to comprehend, from an immunogenetic viewpoint, the relative importance of the pathogenic microbe, GABHS, its biological pathogenic features, and the host's genetic qualities that promote the development of the illness. Although the connection between rheumatic heart disease and an upper respiratory tract infection caused by GABHS is widely known, several points need to be made clear.

1. Table: Mechanisms of Rheumatic Fever Pathogenesis

Step	Mechanism	Result
1	Infection with GABHS (pharyngitis/tonsillitis)	Streptococcal antigens enter body
2	Immune response against bacteria	Antibodies and T-cells formed
3	Molecular mimicry between bacterial and heart/joint tissues	Cross-reactive autoimmune reaction
4	Inflammation in target organs	Carditis, arthritis, chorea, skin lesions
5	Healing with fibrosis and scarring	Valve deformities → Rheumatic heart disease

For example, several papers report that 0.3-3% of people develop rheumatoid arthritis after acute streptococcal pharyngitis. 40% of all streptococcal infections occur between the ages of 2 and 6, despite the fact that children between the ages of 5 and 15 are most likely to develop rheumatic. Explaining why GABHS is commonly seen in the upper respiratory tract and why it doesn't appear to be able to induce rheumatoid arthritis is equally important. This unique relationship between Streptococcus and its host should be explained by the characteristics of the macroorganism.

Along with the importance of various human genetic features, age, gender, and environmental factors are examined. Rheumatoid arthritis is a disorder that develops in combination with an infection, although it does not fit within the category of infectious pathology. The causative agent, one of the most common pathogenic bacteria, causes a number of infectious diseases, including sinusitis, rhinitis, tonsillitis, and acute pharyngitis. The genetic programming of the immune response does not determine whether the sickness is fatal when streptococcal antigen stimulation takes place. In this situation, it is crucial to search for other factors influencing the chance of contracting an illness. Social factors (overcrowding and unfavourable living conditions;

The presence of streptococcal infection foci (chronic tonsillitis, sinusitis, dental caries, kidney disease; decreased reactivity; climatic and geographical factors; socioeconomic factors; hereditary predisposition) greatly influences the pathophysiology of RL and determines the main areas of preventive work for general practitioners. clinic as well as diagnostics. A.A. Kisel, TD Jones, and A.I. Nesterov's primary diagnostic criteria are carditis, polyarthritis, chorea, annular erythema, and subcutaneous rheumatic nodules; minor criteria include proof of previous streptococcal infection (GABHS culture from the pharynx), detection of streptococcal antigen, elevated titer of

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antistreptococcal antibodies, and laboratory testing (positive "acute phase" tests). In accordance with the 1992 change made by the WHO expert committee,

The previous criterion, which said that the diagnosis of rheumatic fever may be made with just two major or one major and two minor clinical criteria, was changed to include evidence of a previous GABHS infection. Chorea and late-onset carditis are the two exceptions to this rule; in these cases, the patient's initially elevated antistreptococcal antibody titer may revert to normal by the time they seek medical attention. Rheumatic heart disease, which often affects both the myocardium and endocardium, is present in 90 to 95 percent of individuals with acute rheumatic fever. Myocarditis manifests as palpable heart pain, dyspnoea, weakening of the first heart sound, apical murmur, and abnormalities in conduction (prolonged PQ) and ECG rhythm.

Endocarditis manifests as dilated cardiac borders, cardiac arrhythmia, thromboembolic syndrome, and a louder systolic murmur at the apex and in the Botkin zone, which is linked to the original heart sound and is not affected by breathing or changes in body posture. Functional murmurs, which are different from organic murmurs due to their shorter duration, milder tone, and lack of connection to the initial heart sound, are experienced by healthy individuals, especially adolescents and teens. Depending on the respiratory phase and body position, these irregular murmurs might have different characteristics.

Two-thirds of cases of first acute respiratory disease bouts are caused by rheumatic polyarthritis, which is characterised by exudative inflammation. The knee, ankle, and elbow are the most often affected joints. All signs of acute inflammation, including joint pain, swelling, hyperaemia (redness), and dysfunction, are present in symmetrical lesions with a pain pattern that is often transient. Chorea minor, which is associated with involvement of the striatum, subthalamic nuclei, and cerebellum, is a frequent symptom of ARF (6-30% of patients). Girls make up the bulk of those affected. Chorea is characterised by bad handwriting, slurred speech, awkward movements, and unpredictable, uncontrolled twitching of the limbs and facial muscles (hyperkinesia). The patient is unable to perform coordination tests, such as the finger-to-nose test. Symptoms often completely disappear when you sleep.

Annular erythema is a common yet rare sign of ARF (4-17% of patients). It is characterised by a rapidly fading pink rash. Erythematous areas are primarily seen in the trunk and proximal extremities. They are rarely seen in person. Squeezing erythematous areas causes them to blanch; erythema is transient, migratory, and heat-induced. It doesn't cause irritation or thickening of the skin.
rheumatic nodules under the skin. They are located over bony prominences in the knee and elbow joints, above the occipital bones, and in periarticular tissues near tendon attachments. Typically, they are quite small, hardly larger than a pinhead. They manifest as painless swellings. They only afflict children (1-3%), usually after the first acute rheumatic fever episode, and go away entirely two to four weeks after the sickness starts.

Stage III activity can include fever, acute rheumatic polyarthritis, myocarditis, raised ESR (40 mm/h), high titers of antistreptococcal antibodies, pancarditis, and serositis. Stage II activity is characterised by subfebrile body temperature, chorea, polyarthralgia or mono-oligoarthritis, and severe heart damage symptoms; a full blood count reveals leukocytosis and an ESR of 20-40 mm/h. Stage I activity is distinguished by monosyndromicity (mild chorea or carditis). The lab's parameters are either minimally changed or normal. Specialised research and labs: high ESR, high CRP, and neutrophilic leukocytosis are observed in the initial days following the acute onset of the illness. The latter two indicators often persist long after the clinical signs of LC have diminished.

A general urine analysis is often considered normal. Occasionally, minor proteinuria or microhematuria is seen. Antistreptococcal titers, such as antistreptolysin Q AT to DNase, in a titer

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greater than 1:250, were raised in two-thirds of patients. A smear is analysed bacteriologically to find GABHS. Serial cultures are more helpful when streptococci are detected since they generate streptococci in 20-45% of cases.

To detect cardiac anomalies and pericarditis, echocardiography is advised.

An ECG is essential to comprehending the nature of heart rhythm disturbances and the broad principles of treatment. The comprehensive treatment of ARF includes rehabilitative methods in addition to etiotropic, pathogenetic, and symptomatic treatments. non-pharmacological treatment. All ARF patients get the following medications: a diet high in protein and vitamins and low in salt and carbohydrates; hospitalisation with bed rest for the first two to three weeks.

etiotropic therapy. Eliminating GABHS is the aim of antimicrobial therapy. Medication containing penicillin is prescribed. Adults and teenagers: Benzylpenicillin should be administered intramuscularly at four dosages of 1.5-4 million U/day for 10 days. Children: Give four intramuscular doses of 400-600 thousand U of benzylpenicillin for 10 days. After that, they start taking long-acting drugs such as benzathine penicillin. pathogenetic therapy. The main goals of anti-inflammatory treatment are to limit the activity of the rheumatic process in patients with primary rheumatic carditis in order to avoid the development of heart abnormalities. For those who have experienced recurrent episodes of ARF, this type of treatment attempts to promote general health and prevent the development of pre-existing cardiac problems.

Acute cases of the disease with polyserositis, maximum and moderate inflammatory activity (ESR 30 mm/h or greater), and severe and moderate carditis (pancarditis) are treated with GCS. Adults and teenagers: Take 20 mg of prednisolone orally in the morning after meals until a therapeutic response is achieved (usually within 2 weeks). Children:

Take prednisolone orally in the morning after meals at a dose of 0.7-0.8 mg/kg until a therapeutic effect is achieved. After that, the dosage is gradually reduced until it is stopped entirely. The duration of therapy is between one and two months. NSAIDs are recommended for rheumatic arthritis without carditis, mild rheumatic carditis, low process activity (ESR less than 30 mm/h), the need for long-term treatment after stopping high activity and GCS, and recurrent ARF in the context of RPS. Adults and teenagers: Take 75-150 mg of diclofenac orally in three sessions for a month. Children: Take three oral doses of 2-3 mg/kg of diclofenac for a month. If necessary, the diclofenac course of treatment is extended for a maximum of three to five months, or until there are no more indications of inflammatory process activity.

Prevention of acute respiratory infections. The cornerstone of primary prevention is antimicrobial therapy for both acute and chronic recurring GABHS infections of the upper respiratory tract (tonsillitis, pharyngitis). Benzathine benzylpenicillin IM 2.4 million U/day once (adults); IM 0.6 million U/day once (children under 25 kg); IM 1.2 million U/day once (children over 25 kg); or Amoxicillin IM 0.5 g three times daily for ten days (adults); or Phenoxymethylpenicillin IM 0.5 g three times daily for ten days (adults); or Cefadroxil IM 0.5 g twice daily for ten days (children over 25 kg);

1.2 million U/day for kids weighing more than 25 kg. Phenoxymethylpenicillin is primarily recommended for the treatment of children due to the availability of a suspension dosage form. Benzathine benzylpenicillin should be prescribed in the following circumstances: there is a possibility that the patient will not adhere to the prescribed oral antibiotic regimen; the patient or close relatives have a history of acute respiratory illness; unfavourable living and social circumstances; outbreaks of GABHS infection in preschools, schools, boarding schools, colleges, military units, etc.

If an individual is intolerant to P-pactam antibiotics, they can take Roxithromycin orally 0.3 g/day in two doses for ten days (adults); Midecamycin orally 1.2 g/day in three doses for ten days

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(adults); oral 50 mg/kg in two doses for ten days (children); or Azithromycin orally 0.5 g/day on the first day, then 0.25 g/day for six days. or erythromycin orally (1.5 g/day in 3 doses, 10 days for adults; 40 mg/kg/day in 3 doses, 10 days for children) or intramuscularly (0.5 g/day in 2 doses, 10 days for adults; 15 mg/kg in 2 doses, 10 days for children).

Ten days for adults; two intramuscular injections of three million units each day for children. A person should take either Clindamycin (0.6 g/day in 4 doses, 10 days for adults), Lincomycin (1.5 g/day in 3 doses, 10 days for adults), or Lincomycin (30 mg/kg/day in 3 doses, 10 days for children) if they are intolerant to macrolides and β-lactam antibiotics. Oral amoxicillin/clavulanate 1.875 g/day in three doses for ten days; oral clindamycin 0.6 g/day in four doses for ten days (adults); oral lincomycin 1.5 g/day in three doses for ten days (adults); oral 30 mg/kg/day in three doses for ten days; oral 0.5 g/day in three doses for ten days (adults); oral 20 mg/day in two doses for ten days. secondary prevention of ARF. In those who have had ARF. Secondary prophylaxis of ARF includes regular administration of long-acting penicillin (benzathine benzyl penicillin), which attempts to stop recurrent attacks and the development of illness in those who have had ARF. Adults and teenagers should get 2.4 million units of benzathine benzylpenicillin intramuscularly once daily for three weeks; children weighing more than 25 kg should receive 1.2 million units once daily; and children weighing less than 25 mg should receive 600,000 units once daily.

The duration of secondary prophylaxis, which should begin while the patient is still in the hospital, is determined for each patient. It is typically at least five years for people who have had ARF without carditis (arthritis, chorea); it is more than five years (or lifetime) for people who have had a primary or recurrent episode of ARF with cardiac injury (especially if there are signs of developing or preexisting heart disease). Features of congestive heart failure therapy for those with RPS and ARF: Since high dosages of prednisolone (40-60 mg/day) might obviously have a therapeutic effect, cardiotonic drugs are not recommended for treating primary rheumatic carditis, which frequently only affects youngsters.

More research is required to determine whether using ACE inhibitors in patients with rheumatic carditis against the background of RPS is appropriate because concurrent use of NSAIDs and ACE inhibitors can weaken the latter's vasodilatory effect; nitrates worsen the prognosis in patients with RPS; and GCS should not be used in patients with RPS without obvious signs of rheumatic carditis due to the risk of increasing myocardial dystrophy. Prediction: The development of ARF usually seldom poses an immediate risk to life, with the exception of extremely uncommon adolescent cases of pancarditis. The state of the heart (the presence and severity of the abnormality, the degree of heart failure) largely determines the prognosis.

The start of treatment is important since postponing drug therapy greatly increases the risk of developing. RHD develops as a result of rheumatic carditis. The highest frequency of RHD development (75%) occurs within three years after the disease's onset. Defects occur in 20-25% of toddlers, 33% of adolescents, and 39-45% of adults after an initial ARF attack. Aortic and isolated mitral insufficiency are more common in children, and 7-10% of them have mitral valve prolapse. Isolated mitral valve insufficiency is the aetiology of half of adolescent cases. Adult problems have special characteristics because adults are more likely (93%) to develop recurrent ARF in the context of pre-existing cardiac abnormalities. While 12% and 13% of patients had discrete mitral and aortic valve insufficiency, respectively, 42% of cases had concomitant mitral valve failure.

We provide a real-world example: B-naya D., who was born in 1966, complains of palpitations and dyspnoea during physical activity. She is on the "D" registry and has had rheumatism since 1983. EchoCG Timely detection of some heart problems can guarantee their successful surgical treatment,

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even though high-resolution imaging for mitral and aortic stenosis is insufficiently useful in situations of valve insufficiency.

On June 22, 2002, her rapidly deteriorating condition led to a referral to the National Center for Cardiology and Therapy in Bishkek. Rheumatism n/f. Significant cardiac stenosis was the diagnosis. Aortic orifice stenosis; insufficiency of the mitral valve stenosis. 2-12. insufficient aortic valve performance. The tricuspid valve is inadequate (relative). fluttering atrial fibrillation. Heart failure, FC 212. Surgery is recommended. CBC: Hb-89 g/l, Er-3 300000, Colour p. -0.8, leukocytes-6200, p/y-6%, s/y-48%, eosinophils-1%, mon-10%, lymphocytes-35%, and ESR-43 mm/h. The urine was found to be yellow, sl/turbid, acidic, and to have a specific gravity of 1016. It also included units of leukocytes and oxalates, three to four epithelial densities in the field of vision, and no protein.

Rheumatoid tests: ASL-313 units, CRP-negative. A coagulogram is absent. HbE-antigen and RW-negative. ECG: horizontal EOS and atrial fibrillation in a minute at a heart rate of 85. The ventricles' early repolarisation. QT is 0.36 seconds. EchoCG before surgery: The aorta remains unaltered, with a diameter of 2.83 cm, fibrous cusps, a marginal focal change, an incomplete opening of 1.6-1.7 cm, an FC of 2.2 cm, a gradient of 24-28 mm Hg, and regurgitation (+). Mitral valve: unidirectional movement, marginal focal thickening, fibrous alteration of the cusps, and fusion along the commissures. AMO: 1.2 cm, FC: 5.2 cm, gradient: 9.8 mm Hg, regurgitation: (++). Tricuspid cusps regurgitate and are moveable (+++). There is a dilated pulmonary artery.

LA-4.86 cm, LV-5.36x3.86 cm, EF-54%, RV-1.49 cm; aortic position: prosthesis; systolic gradient: 18.2 mmHg; regurgitation: no; mitral position: prosthesis; gradient: 12 mmHg; regurgitation: minimal; the pulmonary artery is dilated; mean PBP: 30 mmHg; tricuspid regurgitation is harmful; the lungs' haemodynamics are within normal; the heart rests widely on the diaphragm, flattened waist, and has an enlarged left atrium that casts a shadow over the peripheral airway level.

A prosthesis was implanted using nine "P" + 4 "Z" + 6 interrupted sutures; the aortic valve was fibrotic, shortened, and fused along the commissures. After surgery, wound healing was sufficient. The patient was discharged following therapy. Consequently, social factors determine the main emphasis of a general practitioner's prophylactic efforts and have a major impact on the pathophysiology of rheumatic fever.

CONCLUSION

Particularly in areas with low socioeconomic resources, rheumatic fever and its aftereffect, rheumatic heart disease, continue to be serious worldwide health issues. In genetically susceptible people, the illness manifests as a post-streptococcal autoimmune reaction that mostly affects the heart, joints, and central nervous system. Preventing persistent cardiac problems requires prompt identification using the Jones criteria and laboratory proof of past streptococcal infection.

Antimicrobial eradication of GABHS, anti-inflammatory medication, and ongoing secondary prophylaxis to avoid recurrences are all part of comprehensive care. Implementing community-based preventive initiatives, identifying early signs, and guaranteeing adherence to long-term benzathine penicillin treatment all depend on general practitioners. In order to lower the prevalence of rheumatic fever, it is also essential to improve living conditions, reduce overcrowding, and provide health education.

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