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BOOK OF ABSTRACTS

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CARDIOVASCULAR DISEASES
CVD-IA

Integrated Approach From Basic,
Clinical Science, Public Health and Bioethics

Yogyakarta, May 14-17, 2016

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BOOK OF ABSTRACTS
INTERNATIONAL CONFERENCE
CARDIOVASCULAR DISEASES

Integrated Approach from Basic, Clinical Science, Public Health and Bioethics

Editor

Rahma Yuantari

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Tri Suwarno

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dr. Eka Ginanjar, Sp.PD-KKV, FIBASIM, FACP, FICA

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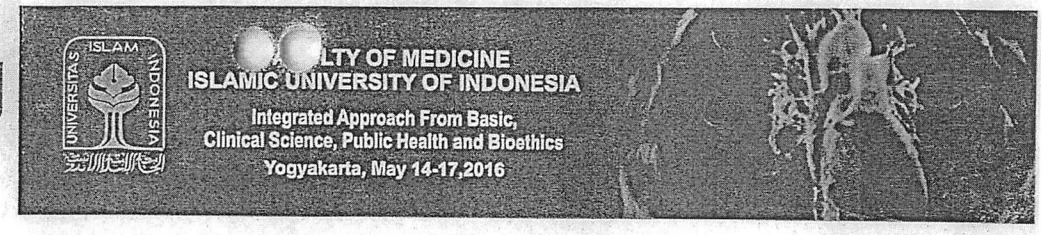
BACTERIA THAT CAUSED RHEUMATIC HEART DISEASE

Farida Juliantina Rachmawaty
Microbiology Department, Faculty of Medicine, Islamic University of Indonesia

Rheumatic Heart Disease (RHD) is a disease of the heart as a result of residual symptoms (sequelae) of Rheumatic Fever (RF), which marked the occurrence of heart valve defects. Rheumatic fever is an autoimmune disease that attacks the multi system due to infection of *Streptococcus* group A â-hemolyticus of the pharynx (pharyngitis). These bacteria usually attack children and young adults. Rheumatic fever causes inflammation that usually occurs in the heart, skin and connective tissue. In endemic areas, 3% of patients with pharyngitis by *Streptococcus* develop rheumatic fever within 2-3 weeks after the infection of the upper respiratory tract. Approximately 40-60% of patients experience acute rheumatic fever that develops into Rheumatic Heart Disease. The bacteria are believed as *Streptococcus* â-hemolyticus group A. This bacterium is a gram-positive bacteria, cocci shaped with the trademark arranged like a chain on microscopic examination. The length of the chain is highly variable and influenced by environmental factors. The chain will be longer in liquid medium rather than in solid medium. The bacteria can do hemolysis to the blood perfectly so that the seed so that the blood look like a clear area around the colony. Protein M, virulence factors found in the cell walls of *Streptococcus* group A â-hemolyticus, has similarities with the structural protein in the myocardium, heart valves, synovial, skin and sub thalamic that causes cross-reactions.

Keywords: Rheumatic Heart Disease, rheumatic fever, *Streptococcus* â-hemolyticus



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has succesfully participated in :

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INTERNATIONAL CONFERENCE ON CARDIOVASCULAR DISEASES
INTEGRATIVE APPROACH FROM BASIC, CLINICAL SCIENCE, PUBLIC HEALTH AND BIOETHICS (CVD-IA)
FACULTY OF MEDICINE, ISLAMIC UNIVERSITY OF INDONESIA
YOGYAKARTA, MAY 14-17, 2016

Faculty of Medicine, Islamic University of Indonesia
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Bacteria That Caused Rheumatic Heart Disease

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Rheumatic Heart Disease (RHD) is a disease of the heart as a result of residual symptoms (sequelae) of Rheumatic Fever (RF), which marked the occurrence of heart valve defects. Rheumatic fever is an autoimmune disease that attacks the multi system due to infection of Group A *Streptococcus* β-hemolyticus (GAS) of the pharynx (pharyngitis). These bacteria usually attack children and young adults. Rheumatic fever causes inflammation that usually occurs in the heart, skin and connective tissue. In endemic areas, 3% of patients with pharyngitis by *Streptococcus* develop rheumatic fever within 2-3 weeks after the infection of the upper respiratory tract. Approximately 40-60% of patients experience acute rheumatic fever that develops into Rheumatic Heart Disease.

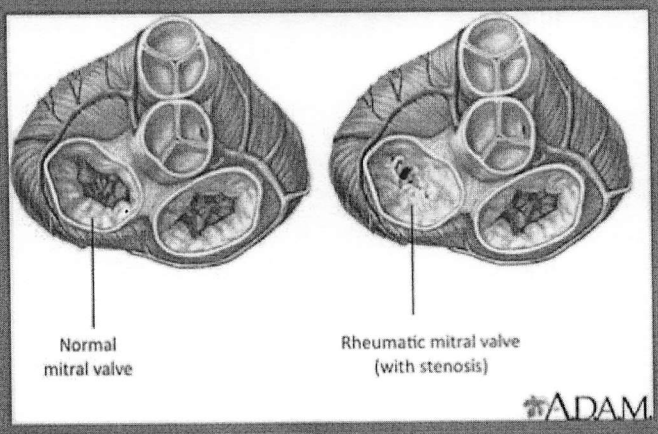

Figure 1. Rheumatic mitral stenosis. Cross-sectional view of the base of the heart showing the 4 valves. The mitral valve (a left-sided heart valve; identified by arrows) has 2 leaflets around an orifice through which it allows blood flow from the upper chamber (atrium) to the lower chamber (ventricle). On the left-hand side is a normal mitral valve with normal leaflets, whereas on the right-hand side a mitral valve narrowed as a result of rheumatic heart disease is depicted. Note how the orifice is severely narrowed as a result of abnormal thickening and fusion of the valve leaflets. Obstruction to blood flow through the narrowed orifice results in symptoms as heart failure or stroke. Reproduced with permission from A.D.A.M. Inc. (Mirabel, 2014)

The bacteria are believed as Group A *Streptococcus* β-hemolyticus. This bacterium is a gram-positive bacteria, cocci shaped with the trademark arranged like a chain on microscopic examination. The length of the chain is highly variable and influenced by environmental factors. The chain will be longer in liquid medium rather than in solid medium. The bacteria can do hemolysis to the blood perfectly so that the seed so that the blood look like a clear area around the colony.

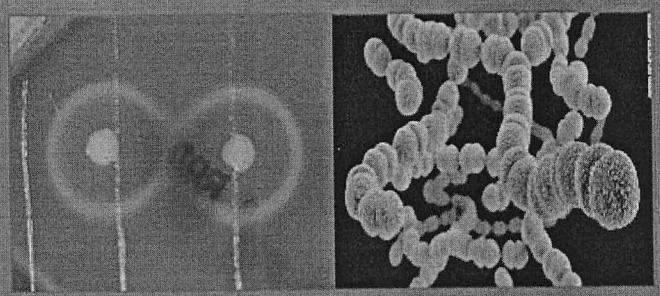

Figure 2. Group A *Streptococcus* β-hemolyticus. A. *S. pyogenes* on Columbia agar with 5% defibrinated sheep blood, 24 hours in an aerobic atmosphere, 37°C. Colonies are **surroundend by a wide zone of beta-hemolysis**. B. *Streptococcus* is seen with an electron microscope.

M-protein, virulence factors found in the cell walls of Group A *Streptococcus* β-hemolyticus, has similarities with the structural protein in the myocardium, heart valves, synovial, skin and sub thalamic that causes cross-reactions. M-protein contributes to invasiveness through its ability to impede phagocytosis of streptococci by human polymorphonuclear leukocytes (PMNL). Conversely, type specific antibody against the M-protein enhances phagocytosis. Following infection with a particular M-type, specific antibody confers resistance to challenge with viable GAS of that M-type. Recently, Boyle has shown that GAS protease cleaves the terminal portion of the M-protein, rendering the organism more susceptible to phagocytosis by normal serum but more resistant to phagocytosis in the presence of type specific antibody. While M types 1 and 3 strains have accounted for the vast majority of strains isolated from cases of **Streptococcal Toxic Shock Syndrome** (StrepTSS), many other M types.