SEPTIC ARTHRITIS IN CHILDREN

ESSAY

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بسم الله الرحمن الرحيم

"وقل اعملوا فسيرى الله عملكم ورسوله والمؤمنون"

صدق الله العظيم

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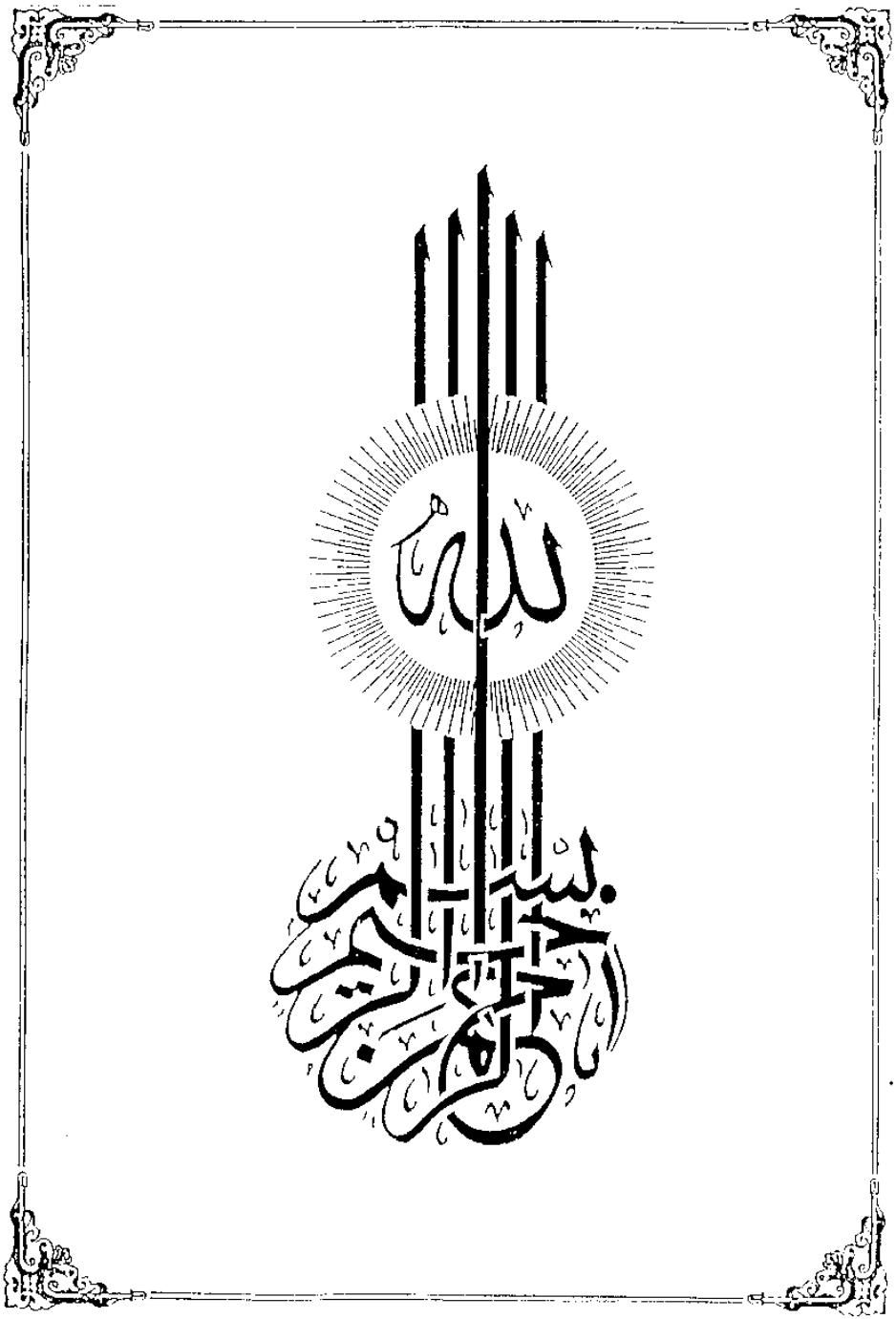

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Introduction

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The term septic arthritis includes all joint infections caused by pyogenic bacteria. Its importance was recognized by ***Smith*** (1874) and pyarthrosis has subsequently been known as "Tom Smith's arthritis." It is not a common condition and the sequelae of delay in diagnosis or inadequate treatment can be crippling, particularly when the hip joint of an infant is involved.

Septic arthritis in the hip of an infant warrants special emphasis, for diagnosis is often delayed, making the prognosis and later management perhaps different from septic arthritis in other joints (***Nade,*** 1983).

Septic arthritis of infancy and childhood is generally a result of haematogenous bacterial seeding of joints (***Jackson and Nelson,*** 1982; ***Greene and Edwards,*** 1989).

If decompression and drainage of the joint space and appropriate antimicrobial therapy is initiated promptly, bacteriological and clinical cure occurs in 80 to 90% of cases (***Badgley et al.,*** 1936; ***Samilson et al.,*** 1958; ***Rotbart and Glode,*** 1985).

Multiple factors, such as patient age less than 6 months, delay in diagnosis beyond 4 days and infection caused by *Staphylococcus aureus* may adversely affect outcome. Neonates in particular are at high risk for sequelae because of their unique epiphysial vascular supply. The most important risk factor for poor outcome, however, may be involvement of the hip or shoulder with osteomyelitis in a contiguous bone (***Dan,*** 1984; ***Syrogianopoulos and Nelson,*** 1988).

The aim of this work is to give an overview of the incidence, the causative organisms, pathogenesis, diagnosis and treatment of septic arthritis in children.

It is of interest to note the finding of ***Kuo et al.*** (1975), who postulated that the variability in presentation of this disease might be a function of immunological competence. In a retrospective study of twelve children known to have pyogenic arthritis in infancy, six still had hypofunction of the antibody-complement-phagocyte pathway. Furthermore, the extent of joint destruction was directly related to the presence of immunodeficiency (***Nade***, 1983).

The Causative Organism

THE CAUSATIVE ORGANISM

The most common causative organisms of septic arthritis in all age groups is *Staphylococcus aureus*. Many other organisms have been isolated from septic joints. In 1966, ***Nelson and Koontz*** drew attention to the increasing importance of *Haemophilus influenzae* as a cause in the infant; this was reinforced by ***Almquist*** (1970). It is important to remember this when choosing antibiotics before the results of bacterial culture and antibiotic sensitivity are known.

Other causative organisms reported include *Streptococcus pyogenes, Streptococcus pneumonia, Escherichia coli, Proteus, Salmonella, Serratia marcescens* (***Martin et al.,*** 1970). *Clostridium welchii* (***Torg and Lammot,*** 1968), *Neisseria, Staphylococcus albus, Aerobacter, Meningococcus, Bacteroides and Paracolon.* However, organisms are not grown from all cases (***Nade,*** 1983).

In the first 2 years of life *H. influenzae* type b and *S. aureus* are the most frequent causes of septic arthritis. Beyond 2 years of age, *S. aureus* is the most likely pathogen but *Streptococcus pneumoniae, Streptococcus pyogenes, Neisseria meningitides* and, up to about 5 years

of age, *H. influenzae* type b all should be considered. The most common causes in neonates are Group B streptococci and *S. aureus*. However, Gram-negative enteric organisms and fungi may infect chronically hospitalized neonates (***Proper,*** 1992).

Nelson and Koontz (1966) summarizing previous reports of ***Samilson et al.*** (1958), ***Obletz*** (1960), ***Baitch*** (1962) and ***Borella et al.*** (1963) found that the causative organisms was unknown in 25 of 133 cases (19 percent), and in 46 of their 117 patients (39 percent). ***Nade et al.*** (1974) did not find a bacterial cause in 19 of 45 patients with a clinical diagnosis of septic arthritis.

Several reasons have been proposed for inability to obtain bacteriological proof of infection, including prior use of antibiotics, inadequate anaerobic cultures, the standard of microbiological laboratories, the changing patterns of the organisms involved and their cultural characteristics (for example, *Haemophilus influenzae*), and failure to obtain blood for cultures or to perform arthrocentesis frequently enough (***Nade,*** 1983).

Cultivation of bacteria such as *H. influenzae* requires media containing X and V factors. Suitable media such as Levinthal and chocolate agar, if employed routinely in culture of septic joints, will

help detect *H. influenzae*. Suspect this organism strongly even if cultures are negative in patients under 2 years of age (***Chung,*** 1986).

Unusual organisms are more likely to appear when there has been penetration of the joint by a foreign body, retention of a foreign body within a joint, systemic disease altering the immunological status of the patient, or treatment with corticosteroids (***Nade,*** 1983).

The Pathogenesis

THE PATHOGENESIS

The inflammatory process in acute septic arthritis starts either within the synovium or within the fluid of a joint effusion, spread by bacteraemia or septicaemia. Spread from adjacent tissues, particularly from a focus of acute osteomyelitis in the metaphyseal end of a long bone, is also important. In those joints in which the metaphyseal portion of the bone is intracapsular especially the hip and shoulder, direct spread from a metaphyseal abscess occurs. The penetration of the joint from without, by diagnostic or therapeutic puncture, as a sequel to femoral venepuncture or following arthrotomy, are other pathways of bacterial infection.

The virulence of the infecting organisms and the resistance of the host determine whether development of the inflammatory process starts in synovium or joint fluid with subsequent suppuration. Infancy, trauma or prior arthropathy reduce the resistance to spread of infection. *In any acute joint disease, infection must be suspected.* "Pus and articular cartilage are incompatible (***Lloyd-Roberts,*** 1971).