POLYMORPHONUCLEAR LEUKOCYTE FUNCTON IN MEASLES

THESIS

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BY

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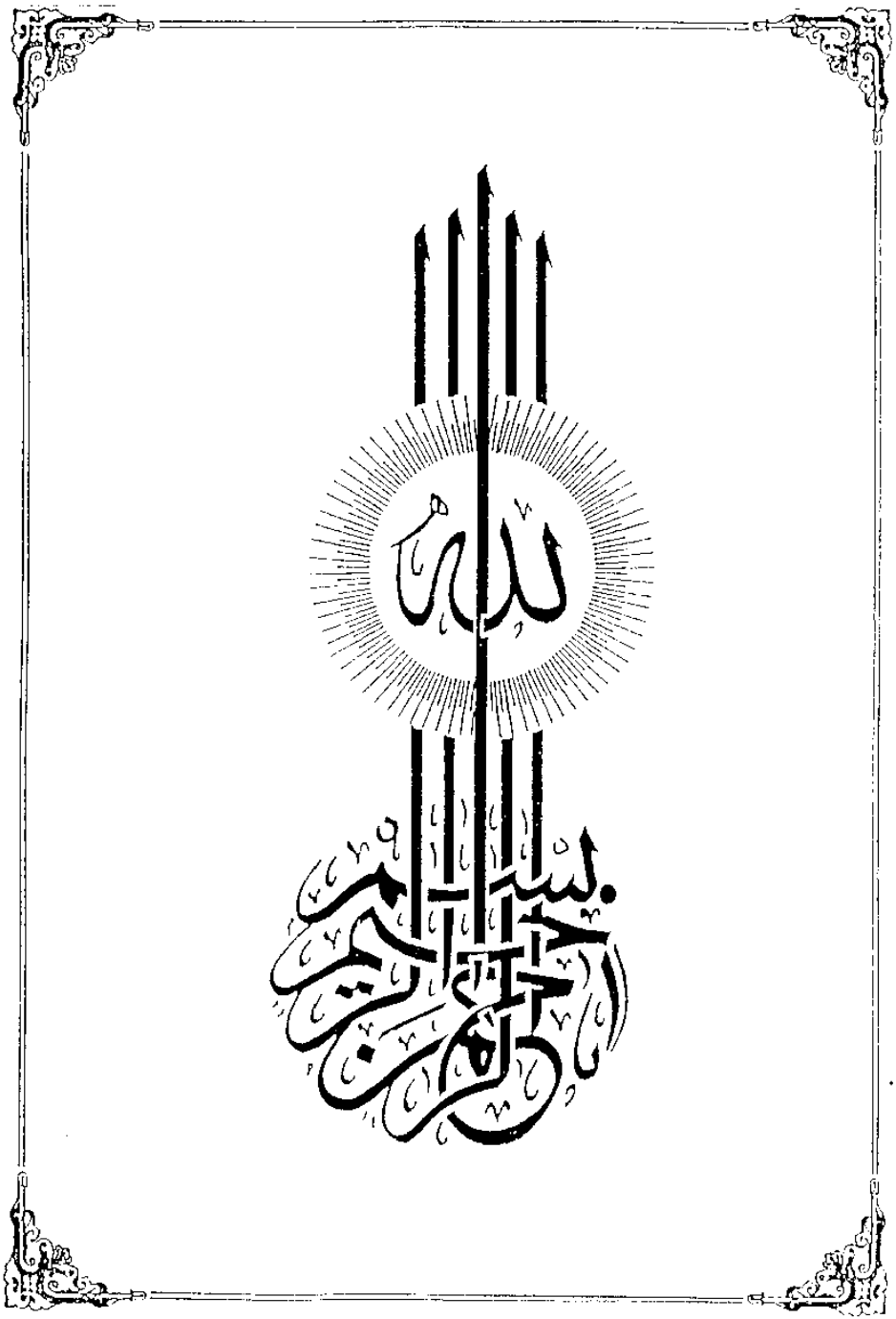

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Measles is a highly infectious virus disease which spreads rapidly wherever there are sufficient numbers of susceptible people. Measles complications are common and sometimes severe. The most frequent complications are bronchopneumonia and otitis media attributable to secondary invasion by bacteria, (KRUGMAN, et al, 1965).

Polymorphonuclear phagocytosis represents one of the most important lines of body defence against invasion by microorganisms, (LARSON and BLADES, 1976). The necessity of sufficient quantities of phagocytic cells for bacterial resistance has been established from clinical experience from a variety of causes, (QUIE, et al, 1967). A small number of patients however suffer recurrent bacterial disease inspite of vigorous leukocyte response to infection. Adequate bactericidal function of phagocytic cells, as well as the quantity of available leukocytes, appears to be a critical factor in bacterial resistance, (QUIE, et al, 1967).

Quantitative and qualitative disorders of polymorphonuclear leukocytes (PMNs) are detected in many viral diseases. Among viruses which produce leukopenia and neutropenia are varicella, rubella and infectious hepatitis, (HAVENS and MARCK, 1946 & CRAFT, et al, 1976). Qualitiatively, it has

been found that some viruses including influenza, (LARSON and BLADES, 1976) and chickenpox, (SHUKRY, et al, 1980), could affect PMN function.

Aim of the work

The aim of the present work is to study PMN phagocytic activity in measles patients in view of explaining why bacterial infection often accompanies measles infection in children, since these cells are agreed to be important defence against such bacteria.

Review Of Literature

MEASLES

The word measles most probably comes from the latin term "misellus" or "misella" , itself a diminutive of the latin "miser" , meaning miserable. Before the 17th century, the disease was confused with smallpox, and though the Arabian physician, Rhazes, is generally credited of having drawn a distinction between the two diseases, demarcation between them was becoming clearer, however, by the beginning of the 17th century,(WILSON, 1962).

Mortality Rate:

The mortality caused by measles was varied greatly during the past 3 centuries. In the London epidemic of 1674 it caused more deaths in the first 6 months of the year than smallpox. In the 18th century in Britain, it was a comparatively mild disease, but early in the 19th century it again became more serious, probably due to discovery of smallpox vaccination, (WILSON, 1962).

In Nigeria , in 1962, mortality rate from measles was 5 % , (MORLEY,et al, 1963). In Valée de Sénégal; in 1956 - 1957, measles accounted for 24 % of all deaths in children under the age of 15 years, and diarrhea complicating measles accounted for a further 13 % , (GANS, et al, 1961).

Now measles remains one of the leading causes of childhood mortality in countries where malnutrition, poor sanitation, and inadequate medical care are prevalent, (MARCY and KIBRICK, 1972).

Attack Rate:

The attack rate in measles is higher than for any other infectious disease. In virgin population that have not experienced a previous visitation, susceptibility appears to be complete, (PHILIPS, 1979).

In populations such as those in Europe and the United States that have been in contact with the disease for some centuries, the secondary attack rate on susceptible members over 1 year of age is usually 80 % - 90 % . The difference between the 100 % in virgin populations and the 85 % attack rate in more civilized populations raises the question of genetic immunity, (WILSON, 1962).

Epidemiology:

Epidemiologically, measles has been considered to be a respiratory disease since Panum, (1846) first assumed that infectious droplets of nasopharyngeal secretions from a patient land upon the respiratory epithelial cells of the new host, and initiate the infection and the chain of events resulting in disease.

However, the portal of entry and the subsequent fate of the virus are matters of conjecture. The traditional belief that virus gains access through the respiratory mucosa has been challenged by experiments in which susceptible children were exposed to measles while wearing goggles and dit not subsequently develop the disease, (EMOND, 1976).

Before the widespread use of measles vaccine, measles epidemics lasting 3 - 4 months were traditionally observed every 2 - 5 years. For a measles epidemic to occur, an adequate number of susceptibles must be in close contact with each other. Therefore, the larger the population, the more frequently epidemics will occur, (GERSHON, 1979).

Pathology:

Inoculation of the upper respiratory tract or conjunctival sac with measles virus is followed by a period of viral replication in the mucosa and in the regional lymph nodes. Shortly thereafter, on the second or third day of illness, a primary viremia disseminates the virus to lymphoid tissues throughout the body, where it multiplies to higher titer. A more extensive and prolonged secondary viremia, from the fifth or sixth day onward, is responsible for the widespread, focal infection of the tissues that later show involvement. Multiplication of the virus and inflammation with necrosis progress in involved organs, and by the eleventh day prodromal symptoms are generally evident. On or about the 14th day, the rash appears.

From 24 to 48 hours later, coincident with the appearance of measurable amounts of circulating antibody, viremia ceases and symptoms begin to abate (MARCY and KIBRICK,1972).

During the biphasic viremia, the measles virus seems to be dessiminated mainly within leukocytes. Measles has been transmitted to volunteers through the transfer of washed leukocytes, (but not plasma) obtained from patients 6 and 8 days before the onset of the rash. Viral multiplication within leukocytes is believed to account for leukopenia and the increased incidence of chromosomal breaks seen in measles,(GRESSER and CHANY, 1963).

Clinical Manifestations:

Incubation period:

It is approximately 10 - 12 days if the first symptoms are selected as the time of onset , or approximately 14 days if the appearance of the rash is selected; rarely it may be as short as 6 - 10 days (PHILIPS, 1979).

The prodromal phase:

This phase is lasting 2 - 4 days and characterized by fever, malaise, conjunctivitis, rhinitis and tracheobronchitis. It is during this period that koplik's spots, generally regarded as pathognomonic of measles,

are noted on the buccal mucous membranes and sometimes on the soft palate, (ROBBINS, 1962).

Koplik's spots in words of koplik are; "small, irregular spots, of a bright red color. In the centre of each spot, a minute bluish white speck appears in strong daylight, (KOPLIK, 1962). They have also been likened to grains of sand on a red background and without examination of the buccal mucosa in good light, they may be overlooked. Most often they appear on the mucosa opposite the second molars. However, in severe cases the entire mucous membrane of the mouth may be involved. This enanthem persists for several days and begins to slough as the rash appears, (GERSHON, 1979).

The conjunctival inflammation and photophobia during the prodromal period, lead one to suspect measles before koplik's spots appear. In addition, a transverse line (Stinson's line) of conjunctival inflammation, sharply demarcated along the eyelid margin, may be of diagnostic assistance. As the entire conjunctiva becomes involved, the line disappears, (PHILIPS, 1979).

The rash:

It is usually first evident as faintly pink macules located behind the ears or on the forehead near the hairline. The rash quickly becomes maculopapular and

spreads rapidly downwards over the face, neck, trunk and extremities during the next 3 days. As the rash progresses, the areas initially involved develop additional lesions that tend to coalesce, especially on the face. At its height, the eruption has generally deepened to reddish purple, and it may be associated with edema of the skin, (MARCY and KIBRICK, 1972).

The patient with measles is usually most ill during the first or second day of the rash. Several days after the appearance of the rash the fever abates, and the patient begins to feel better. The entire uncomplicated illness from late prodrome to resolution of fever and rash lasts 7 - 10 days, (GERSHON, 1979).

The rash fades from above downward in the same sequence as that of its appearance. As clearing occurs, the lesions become brown, and a fine, branny desquamation often appears, usually sparing the hands and feet and thus distinguishable from the heavier flaky desquamation seen in scarlet fever. Although the early rash blanches on pressure, the brown staining that is the residual from the capillary hemorrhages does not, (MARCY and KIBRICK, 1972).