Response of Human Lymphocytes To Measles Virus After Natural Infection.

THESIS

SUBMITTED FOR PARTIAL FULFIL MENT
FOR THE REQUIREMENT
OF (M.Sc.) DEGREE IN
(PEDIATRICS)

By

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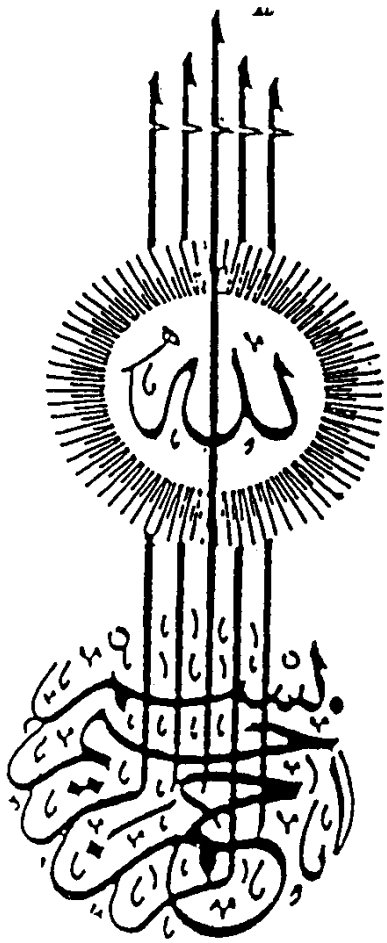
1984



سبحانك

قالوا

لا علم لنا إلا ما علمتنا إنك أنت العليم الحكيم

صدق الله العظيم

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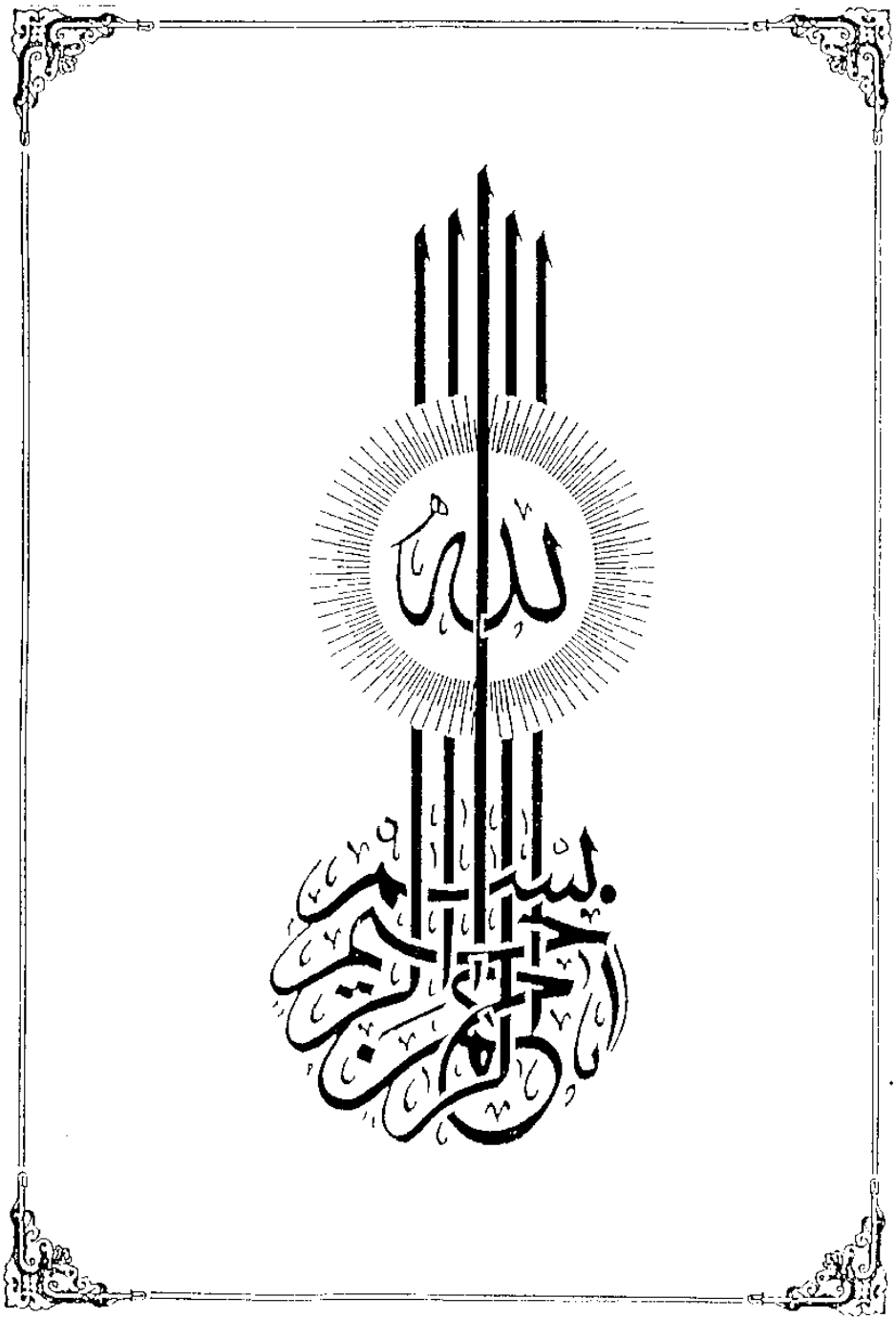

ACKNOWLEDGEMENT

I would like to express my deepest gratitude and appreciations to Professor Dr.Karima Abd El-Khalik, assistant professor of Pediatrics, Faculty of Medicine, Ain Shams University for her generous assistance, unlimited support and helpful criticism and guidance throughout this work.

I also would like to thank Dr. Mohammed Farid, Lecturer of Pediatrics, Faculty of Medicine, Ain Shams University for his kind advice and help.

I owe special thanks and regards to Dr. Amani Saleh and Dr.Laila El-Shawarby, Lecturers of Clinical Pathology, Faculty of Medicine, Ain Shams University for their sincere help and cooperation in taking care of the practical part of this work.

Finally, my thanks are also extended to all who have helped me in the final preparation of this work.

TO

MY HUSBAND

AND SON

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ABBREVIATIONS

CMI	Cell mediated immunity
CTL	Cytotoxic T-lymphocytes
HAI	Hemagglutination inhibition.
ISG	Immune serum globulin.
NK	Natural killer.
PBL	Peripheral blood leucocytes.
PHA	Phytohemagglutinin
SSPE	Subacute sclerosing panencephalitis.

INTRODUCTION

AND

AIM OF WORK

Introduction

Measles is a universal disease, endemic in urban communities and epidemic in rural areas. Epidemic cycles have been noted every two to three years, presumably whenever a sufficient number of susceptibles are available. The epidemiology of measles have been dramatically changed in recent years by the introduction of the vaccine. The antiviral immune response is categorised into antibody-mediated and cell-mediated , each of which may operate through several different mechanisms. The cellular mechanisms involve T-cells which may lyse virus- infected cells directly or through the release of mediators (lymphokines and interferon), which trigger other cells including macrophages and natural killer (NK) cells.

Measles virus replicates in lymphoid organs and in human peripheral blood-leukocytes of B and T-cell origin and also in monocytes (Perrin et al.,1977). T-cells appear to have a greater susciptibility to infection by measles virus than either macrophages or B-cells (Whittle et al., 1978).

Transient, widespread depression of cell-mediated immunity occurs during the acute phase of measles infection

(Osunkoya et al.,1974; Fraser et al.,1978). The effect of natural measles infection on various systemic and Laboratory parameters is on interest. It is found that there is a decline in peripheral esinophilia in measles and following measles vaccine. (Fireman et al., 1969). There is a decrease in absolute Leukocyte count (Black and Sheridan, 1967).

Aim of the work:

The aim of the work is to confirm the relation between measles infection and the number of lymphocytes and T-cells and profil for the prognosis.

REVIEW OF LITERATURES

Measles

Epidemiology:

Measles is a common infectious disease. The epidemiology of measles has been changed in recent years. The relative incidence of the disease appears to be related to the degree of immunization. It is apparent that cases found in the United States are seen in children or adolescent who have not been immunized or who have been improperly immunized. (Schiff et al.,1975). The incubation period of the disease is approximately fourteen days, but an individual is contagious only two to four days prior to the onset of symptoms and for five to six days after the appearance of the rash. Transmission is usually direct and occurs by droplet infection. Contagion is very high, with equal susceptibility in both sexes, and immunity appears to be lifelong (Laurence D. et al., 1982).

The virus:

Measles virus, a paramyxovirus, is 120-200 nm in diameter and irregular in morphology. Measles virus particles are believed to contain six or seven polypeptides (Perrin and Oldstone, 1977; Wechsler and Fields, 1978). The coat consists of a flexible membrane from which fine surface projections protrude. The measles

surface projections include the hemagglutinating or hemadsorption activity, but not the neuraminidase activity. The antigens are responsible for the production of hemagglutination - inhibiting (HAI) and neutralizing (N) antibody. Another function of the measles viral envelope is to facilitate cell membrane fusion, which underlies the characteristic cytopathic effects caused by the virus in infected cells.

Pathology and natural history:

In man, measles virus infects and multiplies primarily in the cells of the mucous membranes of the respiratory tract (Wenner and Lou, 1963). Following infection, the virus can be recovered from the nose and throat, and thereafter from the blood and urine. The viremia indicates that the virus is not limited to its target organ, but causes generalized infection. Clinical and pathological changes are primarily limited to the skin and mucous membranes. During the two to four days of prodromal period, the virus replicates in the respiratory tract, and later involves reticuloendothelial and lymphoid tissues. The formation of multinucleated respiratory epithelial giant cells is the characteristic of this infection (Rake,1965). Giant cells with intranuclear and intracytoplasmic inclusions are also found primarily in the lungs of immunodeficient children who may not demonstrate a rash. The