بسم الله الرحمن الرحيم

وعلمك ما لم تكن تعلم

وكان فضل الله عليك عظيما

صدق الله العظيم
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EXANTHEM IN INFANT AND CHILDREN

SUBMITTED IN FULFILMENT FOR THE REQUIRMENTS OF THE DEGREE OF

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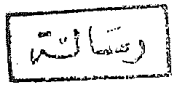

DERMATOLOGY AND VENEROLOGY

PRESENTED BY

DR . WAFAA EL - SHARAAN

SUPERVISED BY

DR . NADIA SALEH
DOCTOR. ASSISTANT OF
DERMATOLOGY

DR . HESHAM ZAHER
DOCTOR. ASSISTANT OF
DERMATOLOGY

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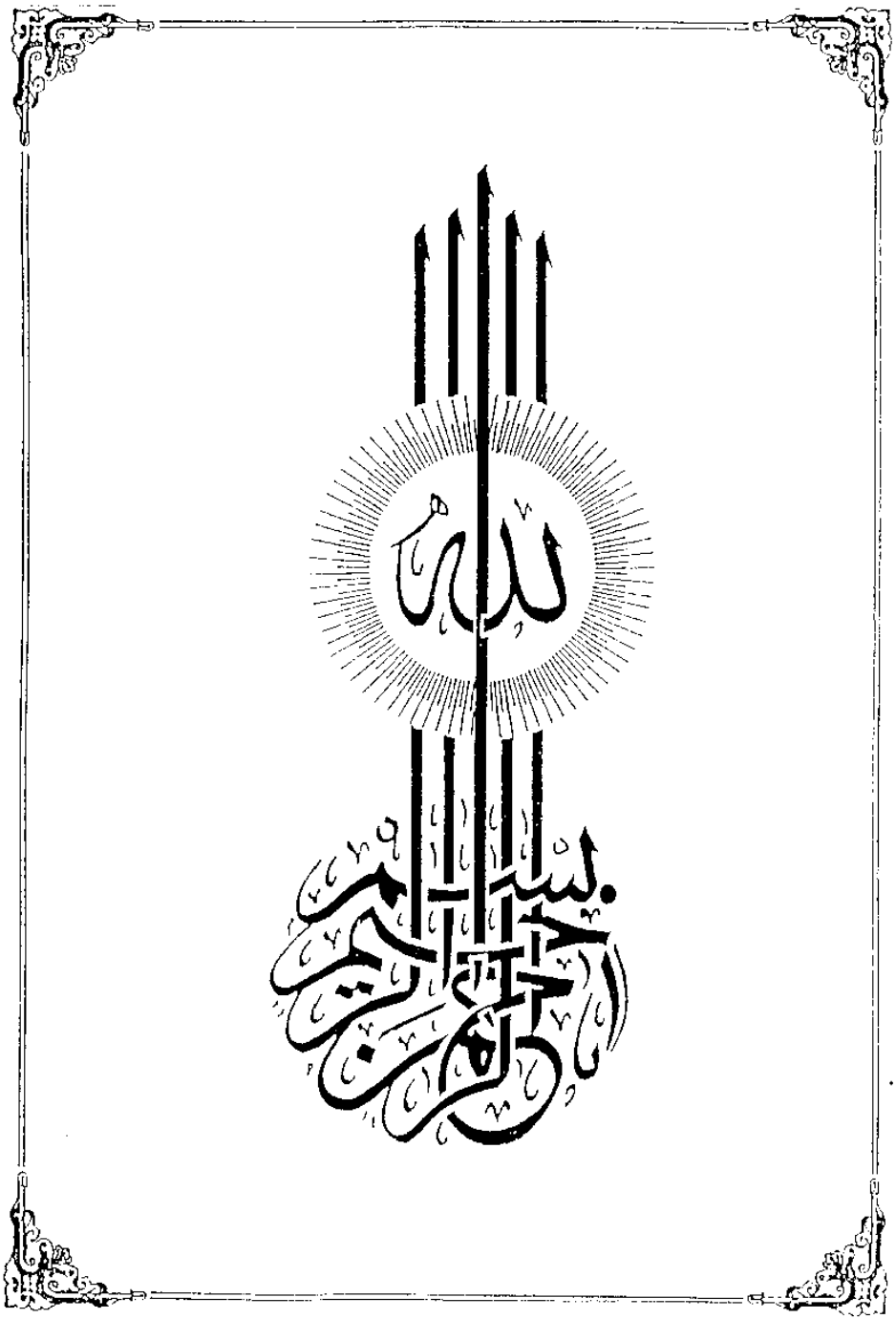

TO MY

"PARENTS",

MY

HUSBAND,

MY

CHILDREN:

Yara, Mohamed and Tamara

I would like to express my deep thanks to their support.

ACKNOWLEDGEMENT

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WAFAA SHARRA'N

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INTRODUCTION AND AIM OF THE WORK

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Exanthem is a term sometimes used to describe a rash associated with systemic illness, especially with fever. Exanthems are a common cause of generalized rashes in infant and children. Because of the diversity of their clinical presentation, they form a diagnostic problem to even the most experience physician. The morphology, distribution, and associated signs and symptoms are sometimes specific enough for a definitive etiologic diagnosis. However, non specific clinical findings often make this impossible. Advances in laboratory techniques, new antiviral drugs and vaccines, epidemic of old exanthems, and the recognition of new clinical syndromes have stimulated renewed interest in exanthems.

Boalecki et al. (1989) mentioned that the six classic exanthems of childhood have many similar physical findings. Familiarity with them, as well as with important laboratory data, allows early and accurate diagnosis of these often confusing diseases. Thus the first disease was measles, second scarlet fever and third rubella. The specific disease described as fourth disease, so called fillatow. Dukes disease, is no longer accepted as a distinct clinical entity, with some authors speculating that it represented staphylococcal scalded skin syndrome, and others speculating that it was concurrent infection with both scarlet fever and

rubella. Fifth disease is erythema infections and sixth disease is roseola infantum.

Frieden and Resnick (1991), described more and more infantile and childhood exanthems and differentiated from one another. More than 50 viral agents and several bacterial and rickettsial infections are now known to cause infantile and childhood exanthems. As chicken pox, enteroviral exanthems, adenovirus exanthems, epstein barr exanthems, staph scalded skin syndrome, toxic shock syndrome, meningococcemia, rocky mountain spotted fever, gianotti corsti syndrome, kawasaki disease.

The aim of this work is to discuss the most common and clinically significant exanthems, with special emphasis on new findings, causes, epidemiology, clinical manifestations, diagnosis, differential diagnosis and management.

MEASLES (RUBEOLA)

Epidemiology:

Reported incidence rate is 98% below that of the pre vaccine era. Seasonal variation of measles continues; the highest number of cases occur in spring. (U.S.public health service, 1987).

The explanations for continued measles out breaks include vaccination before 15 months of age, vaccination with improperly stored vaccine, immunoglobulin given concurrently with vaccine or waning immunity because of vaccination with killed virus. Live measles vaccine is approximately 95% efficacious if given to children older than 15 months of age. Lower efficacy rates n children younger than 15 months of age are most likely due to persistence of transplacentally acquired maternal antibody **(Orenstein et al., 1986).**

Lower efficacy rates in children younger than 15 months of age are most likely due to persistence of transplacentally acquired maternal antibody (Red Book of the American Academy of pediatrics, 1986).

Studies of measles outbreaks between 1985 and 1986 reveal the highest rate of transmission to be in primary and secondary schools, followed by colleges and universities. The out breaks among preschool age children indicate deficiencies in the implementation of the national measles elimination strategy. School based out breaks have been reported

despite a greater than 96% immunity among students (**Morkowitz et al., 1989**).

The incidence of measles in the united states is now less than 1% of what it was before the introduction of a vaccine. Despite the adoption and enforcement of school immunization requirement in every state, however out breaks continue to occur (**Gustafson et al., 1987**).

Measles virus in spread via respiratory droplets, direct face to face contact with a person infected with measles was noted by most of these secondary cases (**Istre et al., 1987**).

Etiology:

Measles is caused by an RNA virus of the paramyxovirus group. The spherical viral particles are approximately 100 to 200 nm in diameter and contain one single strand of RNA as their genome. The virus has an outer envelop that contains hemagglutinin. Measles virus is highly labile, which results in a very short survival time on fomites (**Cherry 1980**).

Extracutaneons clinical manifestations:

The incubation period of measles is 10 to 12 days. A prodrome follows with fever and malaise, accompanied by coryza, conjunctivitis, and cough virtually always precedes the onset of the exanthem. Defervescence of fever usually occurs on second or third day of the rash, also the coryza and conjunctivitis typically resolve at the same time. Cough may persist for five to ten days after the onset of the exanthem.

An infected person is contagious at the onset of the prodrome and remains contagious until day of the rash **(Horstmann 1978)**.

The most common complication of measles is secondary bacterial infection. In a study of Air Force recruits, pneumonia complicated measles in 106 of 3220 cases (3.3%) **(Germillion et al., 1981)**.

Bacterial pathogens include Haemphilus influenza, Neisseria meningitides, and streptococcus pneumonia. On radiographic examination the most common finding was a fine multilobar, reticulonodular infiltrate of the lower lobes **(Cherry 1987)**.

Horstmann (1987) stated that other common complication of measles is post infectious encephalomyelitis. This demyel-inating disease complicates 1 in 1000 measles infections and carries mortality rate of 10% to 20%. The onset is usually between days two and six of the rash and is characterized by fever, vomiting, and generalized seizures, A rare complication of measles is subacute sclerosing panencephalitis which is fatal, progressive, neurologic disease.

Atypical measles, this syndrome was first noted in 1965 but did not receive much attention until 1967, until **Fulginiti, et al (1976)** reported a number of cases. It is characterized at first by acute onset of fever, headache, abdominal pain, and myalgia; two to three days later, a yellowish red, maculopapular, and frequently vesicular and petechial skin rash appears, beginning distally and then spreading centrally.

In many patients, the extremities are edematous, and pneumonia occurs in almost all. The illness has been described as lasting one to three weeks, but many reports suggest increasing duration and severity. Radiographic findings in these patients are almost uniformly positive for diffuse or lobar infiltrates. During the disease course, the infiltrates become nodular and these nodules may persist for years.(**Mitrick 1980**).

Dermatologic findings:

Koplik spots, the enanthema of measles, appear on the buccal mucosa during the prodromal period, approximately two days before the rash. Koplik spots are punctate, blue white spots on a reddish base. A gradual increase in the number occurs, resembling grains of salt sprinkled on a red back ground. Resolution is usually complete by the peak of the exanthem (**Horstman 1978**).

The exanthem of measles appears approximately three to five days after the start of the prodromal illness. It begins as prominent erythematous macules and papules at the hair line, fore head, behind the ears, and on the upper part of the neck. The exanthem then spreads downward to involve the neck, trunk and extremities by the third day. As the rash spreads, lesions of the initially involved areas may coalesce. By the third day, clearing occurs that follows the same down ward progression and leaves a brownish staining. The early exanthem blanches on diascopy, but the brown pigmentary changes resulting from capillary hemorrhage do not (**Lang 1988**).

The exanthem of typical measles is often variable. The exanthem usually begins as erythematous macules and papules on the distal extremities around the palms, wrists, soles and ankles. This centripetal pattern also occur in Rocky Mountain spotted fever and meningococcal sepsis. It then spreads to involve the trunk and face. The lesions may continue in this pattern or progress to vesicles or petechial lesions with purpura. The vesicles appear singly or in crops with erythematous bases, mainly over the trunk, and resemble the rash of varicella. The petechial rash, when present, appears predominantly on the trunk and extremities. Koplik spots have not been observed in a typical measles. **(Martin et al., 1979).**

Pathologic findings:

It is believed that measles virus initially gains access to the respiratory tract. By the onset of the prodrome of measles, the virus is widely distributed in the body. During this time multinucleated giant cells can be recovered from urinary sediment, septum, nasal secretions and various lymphoid tissues **(Wenner, 1973).**

During the prodromal period rubeola virus may be found readily in the oropharynx, and in many organs as well principally those of the reticuloendothelial system. In immunologically impaired children, multinucleated cells and inclusion-bearing cells have been found in proximal collecting tubules of the kidney, and in epithelial cells of the renal pelvis, ureter and bladder **(Lipsey and Boland 1967).**

Rubeola virus may be found in urine for 4 days after onset of rash. In contrast viremia ends shortly after the rash appears and recovery of virus from pharynx or tissues is progressively difficult. Particles identical with rubeola virus have been seen in reactive lesions of the skin and in koplik's spots **(Suringa et al., 1970).**

Virus reaches the skin through the superficial vessels of the dermis. Histologic examination of the cutaneous eruption reveals epidermal hyperplasia, focal parakeratosis, spongiosis, ballooning, necrotic keratinocytes, multinucleated kerationcytes and a superficial perivascular lymphohistocytic inflammatory cell infiltrate. Parakeratotic cells along the margins of involved areas of the skin may contain acidophilic intranuclear inclusions **(Horstmann 1978).**

Koplik spots demonstrate focal areas of epidermal necrosis especially within the basal call layer, with associated serum exudates in submucous glands. Loss of the roof of the subsequent vesicles prodnces the characteristic appearance of the spots **(Lang 1988).**

Laboratory findings:

Recovery of the measles virus is difficult. Verification of infection is best performed. By serologic testing. A four fold or greater increase in either neutralizing haemagglutinis- inhibition or complement fixing antibodies between acute and convalescent serum samples is diagnostic of measles. Antibodies first appear One to Two days after the rash, and peak titires are reached two to four weeks later **(Cherry, 1980).**