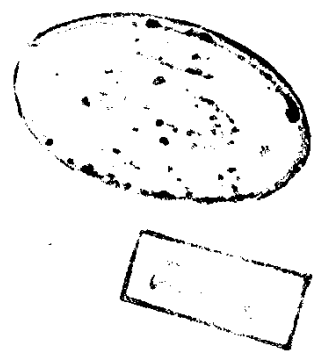
ELECTRO-CARDIOGRAPHIC CHANGES IN EXANTHEMATA

THESIS

SUBMITTED FOR PARTIAL FULFILMENT

OF THE MASTER DEGREE OF

(PEDIATRICS)



By

Fawzy Abdel Hamid Mohamad

M.B., B.Ch.

UNDER THE SUPERVISION OF

Prof. Dr. Yehia El Gamal

Prof. of Pediatrics

Faculty of Medicine

AIN SHAMS UNIVERSITY

Dr. Galila M. Kamal

Lec. of Pediatrics

Faculty of Medicine

AIN SHAMS UNIVERSITY

Dr. Wagdy Abdel Hamid

Lec. of Cardiology

Faculty of Medicine

AIN SHAMS UNIVERSITY

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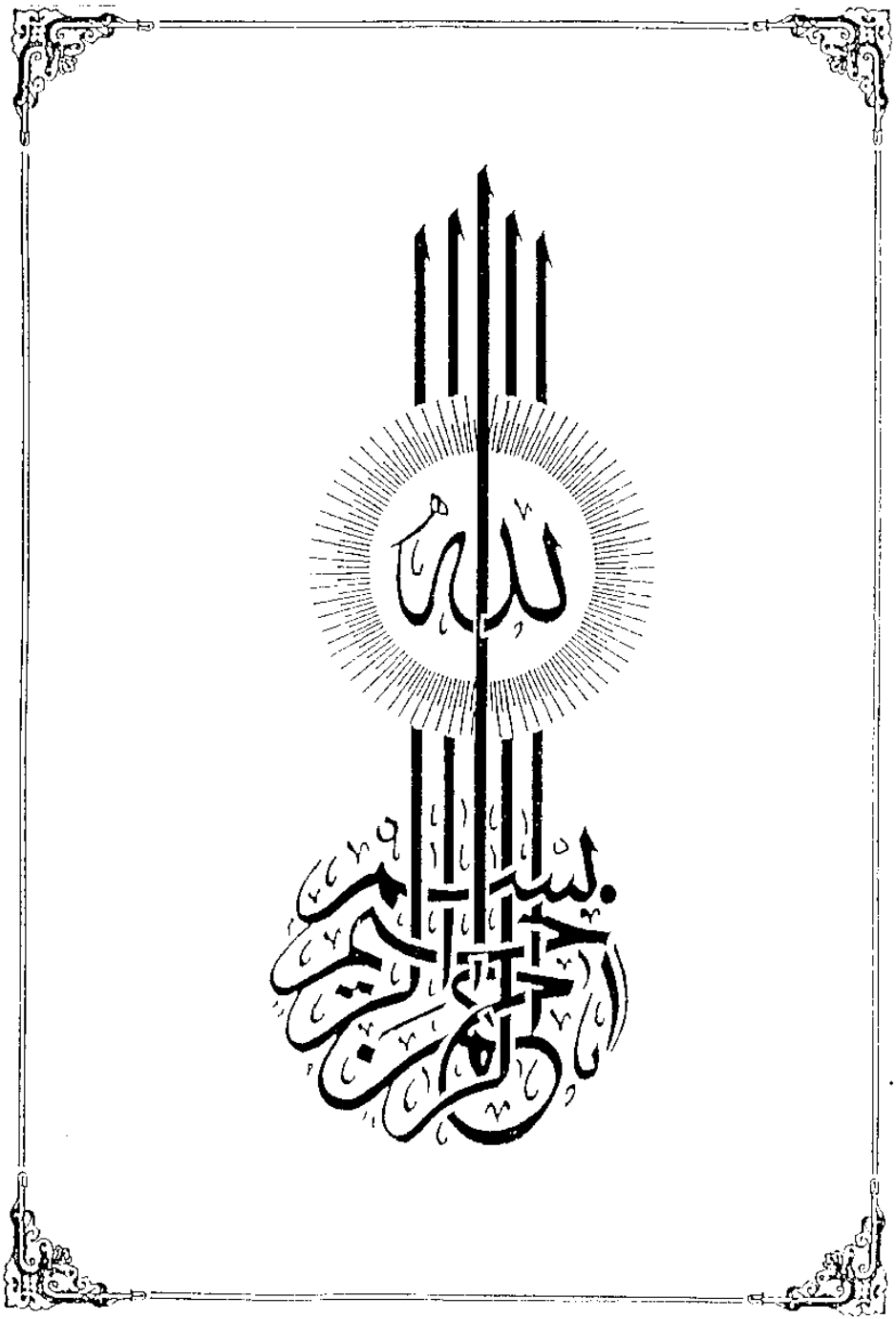
○ قَالُوا سُبْحَانَكَ لَا عِلْمَ لَنَا إِلَّا مَا عَلَّمْتَنَا
إِنَّكَ أَنْتَ الْعَلِيمُ الْحَكِيمُ ○

صدق الله العظيم



TO

MY PARENTS AND WIFE

ACKNOWLEDGMENT

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INTRODUCTION AND AIM OF STUDY

Exanthematus diseases are febrile illnesses associated with rash. (Duncan, 1977).

They are quiet common in children. Regarding the aetiology, they may be due viruses, bacteria, chlamydia, rickettsia, fungi, protozoa or parasites. Most common of them are those of viral aetiology. The commonest and clinically important exanthematous diseases in pediatrics are measles (rubeola), german measles (rubella or three-day measles) and chickenbox (varicella).

Many complications have been being reported including different body systems. These complications may be: respiratory, gastrointestinal, nervous and lastly - which we are concerned with - the cardiac complications. The route of entery to the body is commonly through the nasopharyngeal route. The respiratory complications are pharyngitis, pharyngo-laryngitis, laryngo-bronchitis and pneumonia. Gastrointestinal complications include stomatitis, gastroenteritis and protein energy malnutrition. The complications conserning the nervous system may be drowsiness, irritability, convulsions or encephalitis.

Cardiac affection is rare. This affection may be in the form of myocarditis, pericarditis, transient electro-

cardiographic changes or other complications. Electro-cardiographic tracing studies have shown rare cases of bundle branch block, conduction abnormalities, congestive heart failure which is a poor prognestic sign and often including a fatal outcome. (Branwold, 1980).

On rare occasions sudden death may occur, viral myocarditis may be severe specially in children. So the electrocardiographic changes varie from simple transient changes up to fatal ~~congestive heart~~ myocardits failure.

So our present study aim at examining the detailed ECG changes in exanthematous diseases of viral aetiology; particularly those commonly ~~delt~~ met with in pediatrics namely measles, chickenpox and german measles. On doing that we hope to correlate these findings to each disease , so as to diagnose and treat as early as possible, to avoid complications.

Review Of Literature

Exanthematous Diseases

Rashes accompany many infectious diseases. They may be diagnostic of some diseases, but the skin lesion produced my be common to many infections. Skin lesion may result from direct inoculation (e.g. anthrax), blood dessimination of microorganisms (e.g. meningococcemia or septicemia due to other bacteria); direct spread (e.g. herpetic lesion). Skin may reflect the effect of toxins (e.g. scarlet fever), or delayed type of hypersensitivity to the infecting agent e.g. (erythema nodosum). Rashes can be classified as macular eruption, erythematous maculopapular eruption, papulovesicular or bullous eruptions, petechial or haemorrhagic eruptions, ulcerative eruptions and nodular eruptions. The best way to classify the disorder is as being viral, bacterial or rickettsial infections., (Duncan, 1977 - Krugman et al., 1977).

Diseases associated with macular rash include the echovirus 2,4,5,14, 17, 18, 19, 30, Epstein Barr,Coxackie virus B_1, B_2 , B_5 , enteroviruses 71, staph. aureus, strept. pyogenes and salmonella. Maculopapular rash; which in accordance with aetiology may be viral (e.g.

coxsackie viruses, measles, E.B. virus, hepatitis B, influenza, parainfluenza, rubella, varicella-zoster (chickenpox , herps zoster) and small pox), presumed viral (e.g. roseola infantum and fifth disease), chlamydial (e.g. psittacosis), rickettsial e.g. endemic typhus and epidemic typhus, rocky mountain spotted fever, bacterial (e.g. erysipelothrix , leptospirosis, mycobacterial leprae, salmonella and streptococcus pyogenes (scarlet fever and rheumatic fever), fungal (e.g. tinea vercicolor), protozoa (e.g. malaria), collagen disease (e.g. juvenile rheumatoid arthritis and systemic lupus erythematosus). Vesicular eruptions; which in accordance with aetiology; may be : viral (e.g. coxackie, herps simplex, smallpox and chickenpox), bacterial (e.g. bacillus anthracis, mycobacterium tuberculosis and staphylococcus pyogenes) and rickettsial vesicular eruptions like that in rickettsia tsutsugamushi.

Viral Exanthematous Diseases

Commonest viral infections associated with rash are: measles, rubella, varicella, respiratory syncthytial virus infection (RSV), non polio enteroviruses infections (Coxsackie and echoviruses), infectious mononucleosis (Epstein Barr virus infection), exanthem subitum (roseola infantum), erythema infectiosum (fifth disease) and influenza viral infections. They will be discussed in some details.

Measles (Morbilli, Rubeola):

Measles is a highly contagious acute disease. It occurs almost exclusively in children. The disease is caused by the measles virus. The virion is a roughly spherical particle 120 - 250 nm in diameter, made of a coiled helical RNA. The virus has been recovered from the throat, conjunctiva, urine, blood and other infected tissues. On rare occasions the virus has been isolated from the cerebrospinal fluid. The half life of the virus is about 2 hours at 37°C. It belongs to the paramyxovirus group. Spread occurs via direct contact with infected droplets or contaminated fomites. No carrier state has been documented. Period of communicability begins during the prodromal stage until 4-5 days

after onset of rash. The attack rate in susceptible patients is 90 % or more. Measles has been described in neonates born in susceptible mothers, it is rarely seen in infants under 6 months because the transplacentally acquired maternal antibodies. (Marcy et al.,1977).

Inoculation of the upper respiratory tract or conjunctival sac with measles is followed by a period of viral replication in the mucosa and in the reginal lymph nodes. Shortly thereafter, on the second day of illness, primary viremia disseminates the virus to lymphoid tissues throughout the body. A more extensive secondary viraemia on the fifth day onword is responsible for the wide spread, focal inflammation of the tissues. Multiplication of the virus and inflammation with necrosis progress in the involved organs. By the eleventh day the rash appears. Viremia ceases and symptoms begins to abate. (Fenner, 1948).

The skin lesion may be caused by direct viral damage to the epithelium and vascular endothelium or as a result of general, delayed hypersensitivity to virus antibody

complexes. (Suringa et al., 1970).

The age of peack incidence is 5 - 10 years, since the wide spread use of vaccine most cases are seen in adolescent and young adults who didn't receive the vaccine or were immunized under 15 mounthes of age (Cherry, 1980).

The incubation period is 10 - 14 days. A prodromal phase lasts 3 - 5 days with moderate fever, cough, coryza and conjunctivitis. These almost preceed koplick spots by 2 - 3 days. They are pathognomonic sign, usually present as grayish white dots opposite the lower molars or buccal mucosa. The temperature rises as the rash appears. It starts as macules on the back of the neck, behind ears then lesion becomes maculo papular and spread over the entire face, neck, upper arms, chest, abdomen, entire arms and thigh and finally the feet on second to third day. (Nelson, 1983).

A typical measles is associated with prior adminstration of killed measles vaccine. The illness starts