An Essay on

CHOLERA
in
INFANCY and CHILDHOOD

Thesis

Submitted in Partial Fulfillment
of M.Sc. Degree

in Pediatrics

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CAIRO , 1985

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ACKNOWLEDGMENT

I am deeply grateful to the eminent Professor Dr. KHALIL ABD ELHADY MOURAD, Professor of Pediatrics, Ain Shams University, who was the actual spirit of this work through his remarkable professorial guidance and elegant supervision. I am cordially indebted to his outstanding and unerring directions. His prints are present everywhere in this work.

To every one who participated in some way or another, to let this work come to such a final picture, I owe my thanks and gratitude.

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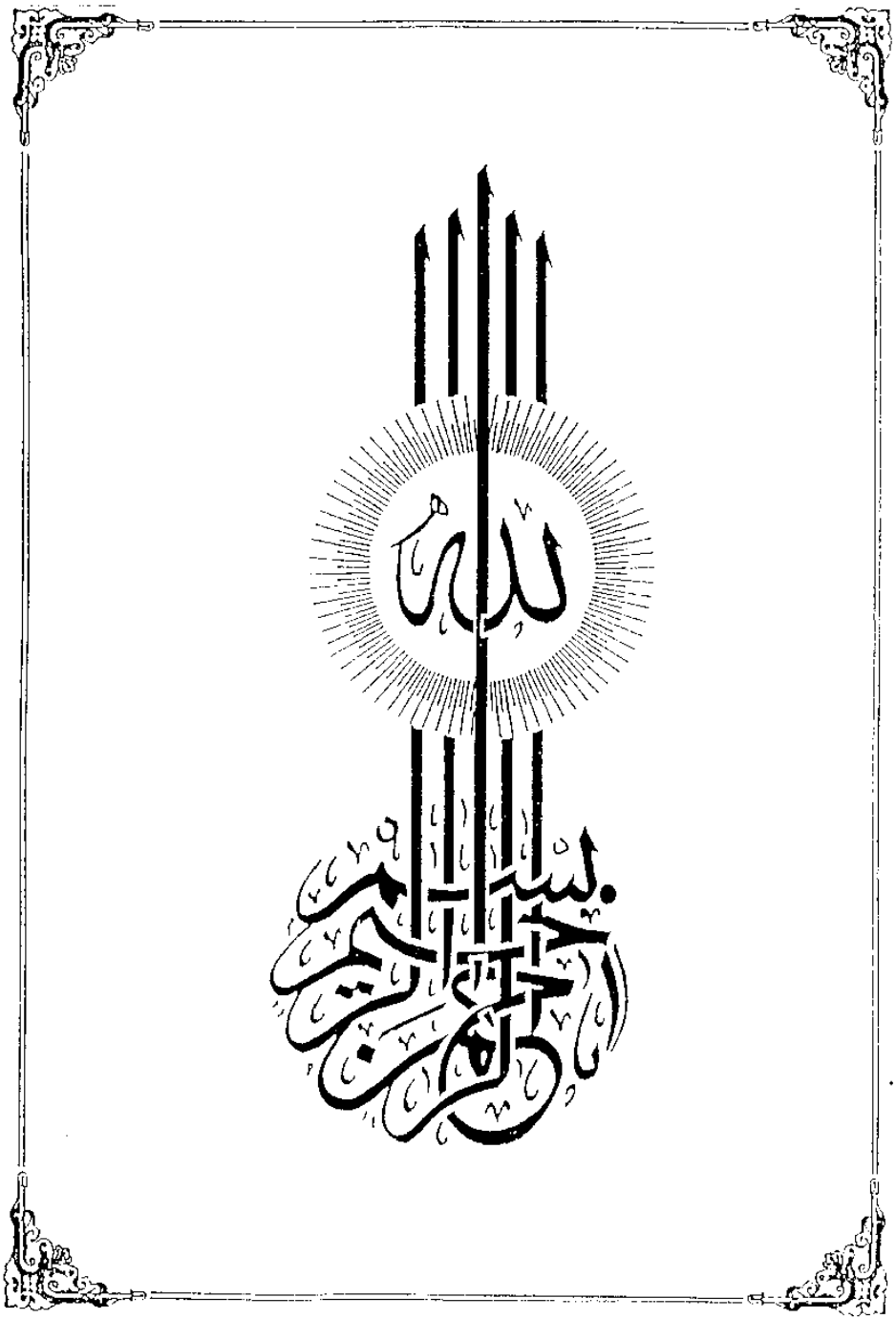

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C O N T E N T S

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INTRODUCTION AND AIM OF THE WORK

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Introduction and Aim of the work

Cholera, in the Greek language means "Flow of bile". It is an acute communicable epidemic disease characterized by profuse purging of a colourless watery material, vomiting, muscular cramps, suppression of urine, algidity, collapse and the presence of cholera vibrios in the intestines (Manson Bahr and Apted, 1982).

In the past centuries cholera was one of the most serious and famous diseases in the world. Even though, it had been vanished from most countries all over the world, eight countries were affected in 1978 and several previously affected countries in Asia and Africa experienced severe recrudescences and extensions of the disease, causing major problems for national health authorities. This present pandemic has stimulated extensive and intensive research on cholera during the last two decades. These researches have made many significant contributions to knowledge of the etiology, epidimilogy, pathogenesis, clinical management and immune mechanisms of cholera, and has made it possible to reduce cholera-related mortality to less than 1% in well equipped treatment centers (W.H.O.Scientific Working Group,1980).

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When cholera strikes a previously unaffected area, the case rate in adults and children are nearly equal; as the disease becomes endemic, the case rate in children exceeds that of adults. For example, when cholera returned to the Philippines in 1961, adults and children were attacked equally. By 1969, the case rate in children was 3 times that of adults. In East Pakistan, where cholera is highly endemic, children between one and five years of age are striken at a rate 10 times that of adults (Carpenter, 1972). This high susceptibility of infants and young children to cholera may be related to low vibriocidal titres, or frequent exposure to infected water while playing (Khan et al,1983).

Epidemic and pandemic cholera is caused by vibrio cholera O group 1 . Two types exist, classical and El Tor . Within each biotype 3 serotypes exist, Ogawa, Inaba and the much less common Hikojima (Levine et al, 1983).

Manson Bahr and Apted (1982), consider that, an endemic area is one in which the total number of months with absence of cholera does not exceed 30 in 32 years or one in which a break of 5 or more months in cholera incidence does not take place.

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Humans are the only documented natural host and victim of cholera (Carpenter, 1983). There is no known animal reservoir of cholera, so they do not play any role in the disease cycle in nature (Felsenfeld 1974; Dorolle, 1974).

The aim of this work is to write an essay about cholera in infancy and childhood. This essay will include :

- History.
- Epidimiology.
- Etiology.
- Pathology.
- Clinical manifestations.
- Investigations.
- Treatment.
- prevention.

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HISTORICAL REVIEW

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History

In 1849, Snow published his pamphlet on the "mode of communication of cholera" in which he showed clearly that the spread of cholera must be due to the passage of a micro-organism carried in excreta,which contaminated water supplies or passed from person to person where elementary personal cleanliness was ignored (Carperter, 1976).

Cholera Vibrios were first described by Pacini who reported on the microscopic and pathologic status of cholera vibrio in 1854. After that it was on the 16 th. of December, 1883 that Koch confirmed comma bacillus as the causative germ of cholera . Thus, Snow preconceived the idea of microbe being involved in the disease process -cholera, and Pacini actually observed vibrio in the stools of cholera patients (Seal, 1983).

History of the Disease :

For the past century and a half, cholera has remained in the delta of the Ganges , with annual epidemics in major population centers in west Bengal and Bangladesh.The disease has made periodic incursions into other portions of South-east Asia, and it has given rise to seven major pandemics since 1817(Carpenter, 1983).

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The first major cholera pandemic began in 1817 in the Ganges River Delta and was associated with high mortality (Rosenberg,1962), after which in 1823 it spread to Russia.By 1831 there had been over one million cases in Russia with an overall mortality of roughly 50% (Arkhangel skii, 1974) .By the time cholera reached Boston and New York in 1832, where it caused a political uproar but was greeted with completely irrational therapy (Carpenter, 1976). In that year the first cardiac catherization was performed by Dieffenbach on a cholera patient who failed to respond well to emetics and cathartics. Dieffenbach,inserted an elastic catheter through the brachial artery into the patient's left ventricle in an attempt to obtain blood from that organ. Dieffenbach became the first advocate of exchange transfusion, arguing for removing the cholera patients' "bad blood" by venesection and replacing it with equal quantities of "good blood" obtained from volunteers (Carpenter, 1976).

In 1849, cholera again swept through the united States, one -tenth of the population of st Louis died of this disease (Rosenberg, 1962). After 15 years, another major pandemic occured, and it was at this time that Austinflint dismissed the theory of contagiousness of cholera despite the earlier demonstration by Snow (1849) that this was clearly a contagious disease spread by water (Carpenter, 1976).

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In 1905 a haemolytic vibrio was isolated from the dead bodies of Mecca pilgrims at the quarantine camp at El Tor in Egypt.

In 1947, cholera broke out in Bengal, India and an epidemic of considerable proportions raged in the Delta of Egypt (Manson-Bahr and Apted, 1982).This occured as a result of dividing the Punjab between Hindustan and Pakistan after declaration of independance of India, where cholera was officially reported among more than five millions emigrants in the Punjab District (Khalil, 1948).

We are now in the seventh great pandemic which started in 1961 spreading from an endemic focus in the Celebes (Indonesia, and by 1965 had invaded 23 countries, among them countries from which cholera had been absent for many decades. By 1970 the pandemic had spread to the Middle East, the USSR and then to Africa via Guinea Cost where it appeared for the first time in the twentieth century in Africa South of the Sahara where it is now endemic in west Africa and has occured in the Southern Sudan, Uganda, Kenya, Zimbabwe, Angola and Mozambique. It has reached Europe and Pacific Islands (Manson Bahr and Apted, 1982).

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It is remarkable that the American continent has escaped so far except for an isolated case in Texas in 1973 (Kelly et al, 1982) and 11 cases on the Gulf Coast of Louisiana in 1978 (Blake et al, 1980).

Vibrio cholerae O group 1, classical biotype, was responsible for the sixth and probably the fourth and fifth pandemics of cholera. These pandemics all originated in west Bengal (Felsenfeld, 1967).

The seventh great pandemic was caused by the EL Tor biotype of V. cholerae.During this seventh pandemic, the classical biotype was completely replaced by the El Tor biotype in Asia (Hug et al, 1980 & Glass et al, 1982).

Recently, a report from the International center for Diarrheal Disease Research, Dacca, indicates that the classical biotype has returned to Bangladesh. Between 1973 and September, 1982, over 5000 Vibrio cholerae O group 1 have been examined in Dacca and only 5 classical strains were cultured from 5 cases occuring in one day in October, 1979. From September, 1982, there has been a gradual transition from the El Tor biotype to the classical biotype, which accounted for over 90 % of cholera occuring in January,1983 (Cook et al ,1983).

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CHOLERA IN EGYPT

Cholera invaded Egypt several times during the last century and during the first half of the present century . Clot Bey had described a cholera epidemic in 1831. He believed that cholera was not infectious at that time (Khalil, 1948).

Another epidemic of cholera was reported in 1865. Rosetta (Rashid) had the highest death rate while Damietta had the second highest figure (42 per 1000) for the death rate. Death rate in Cairo was 32 per 1000 during that epidemic (Khalil 1948).

Two epidemics of cholera during 1883 and 1895 had begun in the same area. Both epidemics began in Damietta and the neighbouring district in the north of Dakhlia province including Mansoura, Manzallah,and other villages lying on the Damietta branch of the Nile . The 1895 epidemic lasted about 5 weeks but returned in a second wave in 1896 (Kalil, 1948).

By the beginning of the present century, a seven months epidemic was reported during 1902 which ended on the 19th of January 1903 (Khalil, 1948). The attack rate was 388 per 100,000 population and the fatality rate was 85% .

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After that epidemic, Egypt remained for about 45 years free from cholera (Kamal, 1948).

In 1947, cholera broke out in Bengal, and an epidemic of considerable proportions raged in the Delta of Egypt (Manson-bahr and Apted, 1982). After declaration of independance of India and division of Punjab between Hindustan and Pakistan, the british military Aeroplanes were evacuating troops from India into the Suez Canal Zone as a station on their way to Britain. Probably the British planes transferred cholera carriers in the second week of September and cases appeared in the British camps in the Suez Canal area about the 11th of September 1947 (Khalil, 1948).

Some of the villagers, in the area surrounding the British camps, were allowed to remove what was known as "Rabbish" which contain the wastes of these camps. Those were thought to be the transmitters of cholera from the camps (Khalil, 1948).

Contaminated dates were considered as the main vehicle of cholera transmission in Korein (near Tel El Kebir where the British camps were present and 8 Kilometers away from Ismalia Canal).

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