AMEBIASIS IN INFANTS AND CHILDREN

Essay

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By

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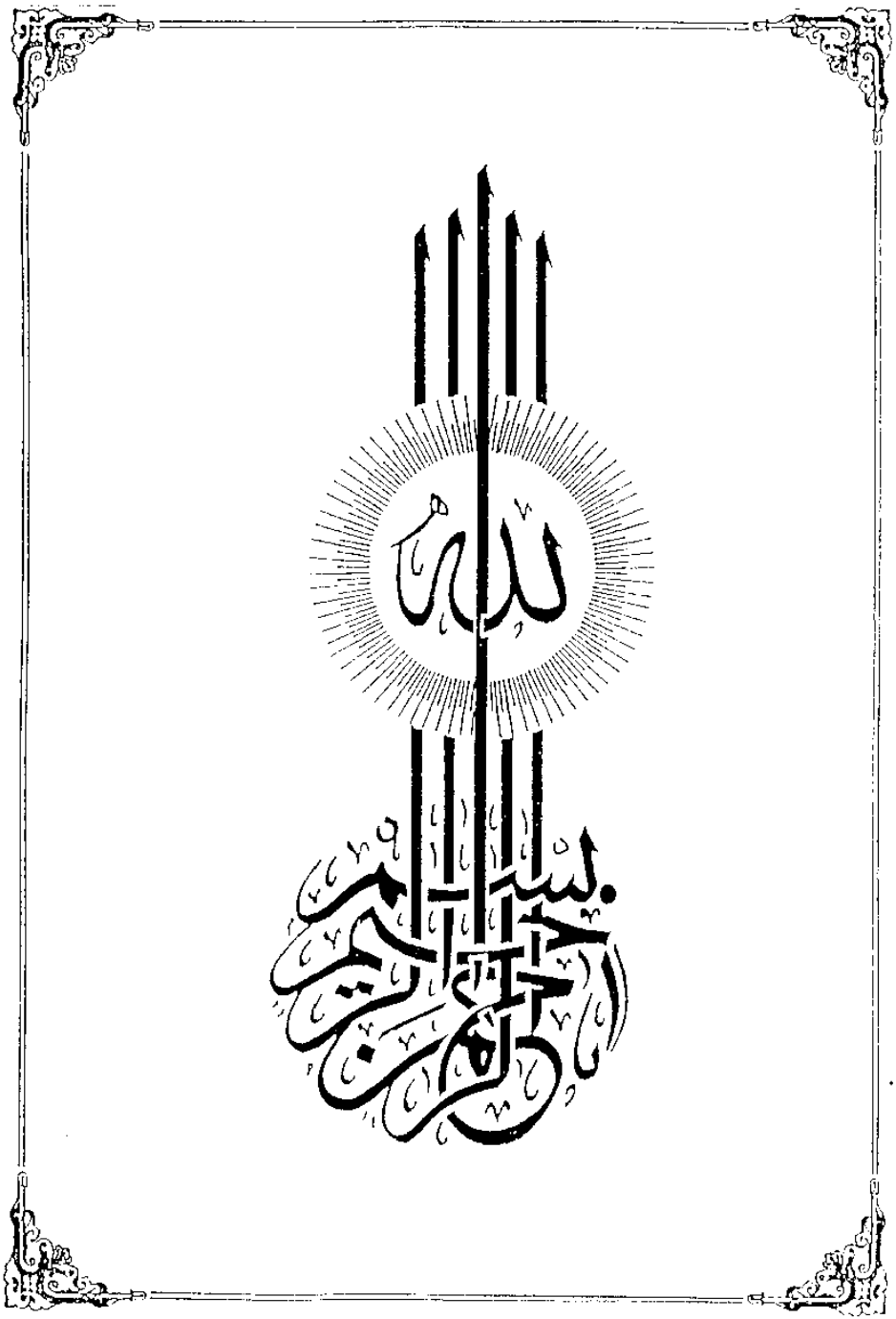

CONTENTS

INTRODUCTION

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Amebiasis is a world-wide parasitic infestation, but is more prevalent and virulent in warm countries. It is marked where sanitation is poor.

The lesions produced by E. Histolytica are primarily intestinal, and secondarily extra-intestinal. Infection with E. Histolytica has been classified as asymptomatic, and symptomatic infection. Most infected individuals are asymptomatic, the only evidence of infection is the cysts found in their faeces.

Symptomatic infections are frequent in tropical climates especially in rural areas where standards of living are low, and hygiene is poor, so that faecal oral transfer of the parasite is facilitated. Children are most at risk from faecal oral contamination. Amebiasis when occurs in the young, it tends to be commoner below the age of six years with a peak incidence between 1 - 3 years.

The prevalence of amebic infection varies from 5 - 81 percent in different parts of the world. Amebic dysentery due to the invasion of intestinal mucosa is found in approximately 1 - 7 percent of infected subjects. Dissemination of the parasite to internal organs such as the liver occurs in an even smaller fraction of infected individuals [about 5 percent].

AIM OF THE ESSAY

Our aim is to write an essay about amebiasis in infants and children. The following points will be discussed:

* *Pathogenesis and epidemiology*
* *Pathology*
* *Clinical picture*
* *Diagnosis, and*
* *Treatment*

REVIEW OF LITERATURE

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The term amebiasis is usually limited to mean infection with E. Histolytica.

Discovery of E.Histolytica

In 1875 Lösch discovered E. Histolytica in the feces of a Russian with severe dysentery; he also experimantally produced intestinal lesions in a dog. However, the association of the parasite with dysentery was not definitely established until the investigations of Kartulis in 1887. In 1901 Councilman and Lafleur made their important study of the pathology of amebic dysentery and hepatic abscess. Schaudinn, of spirochete fame, differentiated E. Histolytica from E. Coli in 1903. In 1913 Walker and Sellards definitely established the pethogenicity of E. Histolytica by feeding cysts to volunteers, thus furnishing the basis of our present concept of its host-parasite relation in respect to clinical infection.

Morphological Characters

There are 3 stages in the life cycle of E. Histolytica.

1. *The trophozoite*
2. *Precyst, and*
3. *Cyst*

* *The living trophozoite, vary in size from about 12 to 60 microns in diameter. The trophozoites are usually actively motile. They move by means of pseudopodia. Motility is usually progressive, and directional rather than apparently aimless as in other amebae [markell, and voge 1981].*

The wide, clear, refiactile, hyaline ectoplasm sharply separated from the endoplasm, constitutes about one third of the entire surface. The finely granular endoplasm usually contain no bacteria, or foreign particles, but sometimes include red blood cells in various stages of disintegration. There is a single eccenteric nucleus, staining with hematoxylin reveals a clearly defined nuclear membrane. The small deeply staining centrally located karyosme consists of several granules in a halo-like capsule, from which a linin network of fine fibrils radiates toward the periphery of the nucleus.

* *The precyst amoebae are colorless round or oval cells, that are smaller than the trophozoites, but larger than the cysts. They are devoid of food inclusions. Pseudopodial action is sluggish, and there is no progressive movement.*

* *The cysts are round or oval slightly asymmetrical hyaline bodies 10 to 20 microns in diameter with a smooth refractile non-staining wall. The cytoplasm of the young cyst contain vacuoles with glycogen, and dark staining refractile sausage shaped bars, with rounded ends. These chromatid bodies tend to disappear as the cyst matures. The immature cyst has a single nucleus about one third of its diameter, while the mature infective cyst contains four smaller nuclei.*

The habitat of E. Histolytica trophozoites is the wall, and lumen of the colon especially in the cecal and sigmoidorectal regions. They multiply by binary fission, the nucleus is dividing by a modified mitosis [Brown and Neve, 1983].

E. Histolytica has been considered an anaerobe because it grows best under reduced oxygen tension. Trophozoites are more easily destroyed than cysts, they survive up to 5 hours at 31°C, and 96 hours at 5°C. In contrast the resistant cysts can survive for 2 days at 31°C, and up to 60 days at 0°C.

There are different strains of E. Histolytica, some are pathogenic, while others such as Laredo are non pathogenic for humans, the virulence of amebal strains

may be related to surface properties of the organism as evidenced by agglutinability with the Lectin and the lack of electrophoretic surface charge [Sargeaunt et al., 1978]. Amebae have also been found to harbor viruses, but these amebal viruses have not been linked to virulence. There are many unknown factors that determine pathogenicity of E. Histolytica such as the intestinal flora and diet as well as virulence of strains [Abdel-Aziz et al., 1979].

Life Cycle:

* *Mature cysts of the amebae are ingested in food contaminated by a subclinical case or a carrier of amebiasis or in water polluted by sewage.*

* *The cysts pass intact from the stomach to the small intestine*

* *Alkaline digestive juices in the small intestine activate the cysts, and mobile trophozoites emerge, and are carried in the fecal stream to the cecem.*

* *The active trophozoites in the caecum multiply by binary fission, and invade the intestinal wall ulcerating it to the submucosa by cytolytic action.*

* *The trophozoites enter the faeces, and they travel along the colon, they secrete a cell wall, and become cysts.*

* *The cysts are excreted in the feces, and infect new hosts by contaminating food or water [Tartakow and Vorperian, 1981].*

PATHOGENESIS
&
EPIDEMIOLOGY

PATHOGENESIS AND EPIDEMIOLOGY

Amebiasis in children is a common, and serious problem in some tropical countries, but is rare where sanitation is good. E. Histolytica organisms are sometimes classified on the basis of size as large race [more than 10 microns in diameter], or small race [less than 10 microns in diameter]. Small race amebae are usually regarded as non pathogenic [Moffet, 1982].

Incubation Period:

The incubation period of amebiasis is from 5 days to several months, commonly 3 to 4 weeks [Tartakow and Vorperian, 1981].

Mode of Transmission:

Amebiases is a world wide parasitic infestation, but is more prevalent and virulent is warm countries [Adeyemo et al., 1984]. As with other infectious diseases of the intestinal tract, amebiasis is marked where sanitation is poor [Hart et al., 1984].

Symptomatic infections are frequent in tropical climates especially in rural areas where standards of living are low and hygiene is poor, so that faecal-oral transfer of the parasite is facilitated [Bonilla et al., 1982]. Children are most at risk from faecal-oral contamination but have a natural resistance to invasive amebiasis although the carrier state is common. However, severe of fatal disease may develop

at any time in these children, so that cysts in the stools should be regarded as potentially dangerous [Rubidge, 1984]

The main source of infection is the cyst-passing chronic patient or asymptomatic carrier. Acutely ill patients are not important since they pass the non-infective trophozoites, and reservoir hosts play a negligible role. Cysts reach human through water and vegetables contaminated with ifective faeces, through food contaminated by flies or the hands of infected food handlers of by direct transmission by cyst carriers [Brown and Neva, 1983]. Major common source out breaks of amebiasis have occured as a result of water supply contamination [Harrison et al., 1979].

Person-to-person transmission, has been important, with infection rates correlating with crowding, poor sanitation, and use of common toilet facilities. It is important to note that, amebic infection in the community tends to cluster in house hold foci with person-to-person transmission playing the major role in endemic disease. Spencer and others [1977], showed infection to be endemic, with significantly higher rates of stool and seropositivity seen in household with crowding and poor sanitation [Harrision et al., 1979]. The infants infections are probably acquired from their mothers who are passing cysts in their stools. In the case of the too youngest children, infection must have been acquired very soon after birth to account for the onset of illness at eary weeks of age [Hart et al., 1984]