CERTAIN EFFECTS OF TOXOPLASMA INFECTION ON SOME EXPERIMENTAL ANIMALS

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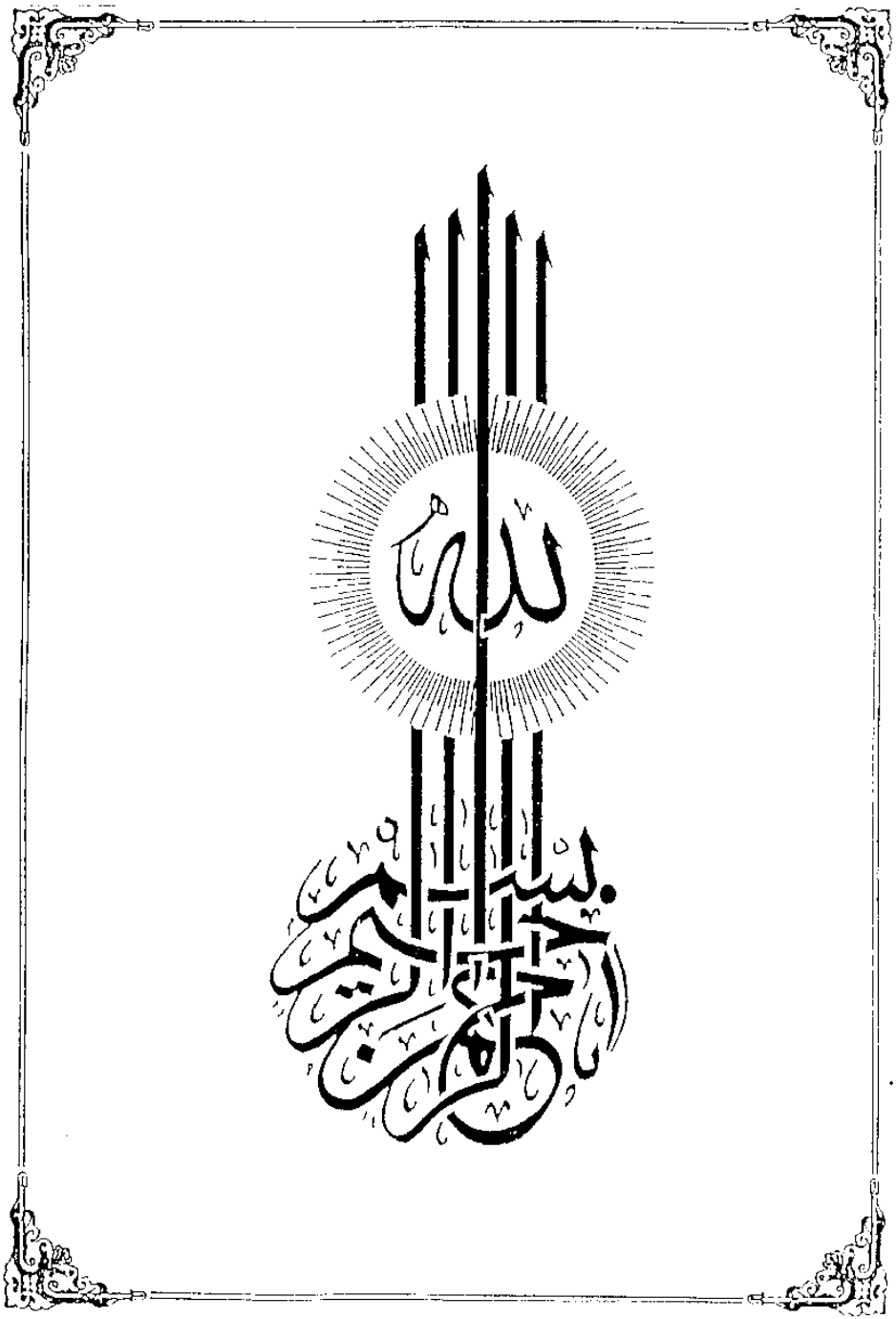

Dedicated to

my parents

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INTRODUCTION AND REVIEW OF LITERATURE

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Toxoplasma is a protozoan parasite which can infect all mammals and man. It was first described by Nicolle and Manceaux (1908) from the small North African rodent Ctenodactylus gondi. The name was derived from the Greek toxon, bow or arc due to its lunate shape .

Sabin and Olitsky (1937) stated that Toxoplasma gondii is an obligatory intracellular parasite . Frankhauser (1951) found dogs, cats and rabbits infected naturally with Toxoplasma gondii pigs, sheep and cattle were also found naturally infected (Jacobs et al., 1960). Moreover, Toxoplasma gondii in man was detected by its isolation by mouse passage (Alexander and Callister, 1955).

Hutchison el al. (1969) and Frenkel et al. (1970) proved that the definitive host of Toxoplasma was the cat.

The clinical picture of toxoplasmosis is usually in the form of abortion in pregnant women, delivery of a child with chorio-retinitis,

hydrocephalus, encephalitis or severe nervous manifestations (Desmants et al., 1965). In adults, acquired toxoplasmosis causes lymphadenitis and relapsing fever in acute cases (Siim, 1951).

The aim of the present work is to study the changes in liver functions, minerals and histopathology induced by Toxoplasma gondii in experimentally infected rabbits and mice.

Toxoplasmosis is a disease which affects both human beings and animals. The cat acts as the only final host. The rest of animals act as intermediate hosts.

I. Morphology, biology and life-cycle of Toxoplasma gondii:

Beaver (1984) stated that there are five main stages in the life cycle of T. gondii. Although all the five stages occur in the cat, only two stages are found in man, other mammals, and in birds. These two stages are: (1) the intracellular trophozoites or proliferative form (tachyzoites), usually seen during the early acute stage of infection, and (2) an encysted form, which is found during chronic or latent infection. Reproduction is by endodyogeny, a process of division in which two daughter trophozoites are formed within the parent cell, which disintegrates when the young trophozoites are released. The trophozoites in stained films are crescentic in shape, 4 to 8 μm in length, 2 to 3 μm in breadth, and have one end that is more rounded than the other.

Evident in Giemsa-stained preparations of various types of smears are a delicate azure cytoplasm and a reddish, spherical, or ovoid nucleus, that is usually nearer to the blunt end of the parasite.

Electron micrographs reveal a complex system of organelles that clearly demonstrate the taxonomic relationship of this organism to the Apicomplexa. One of these structures is a rather short, truncate, and hollow conoid that is located at the more pointed (anterior) end. In histological sections, the trophozoites often appear to be ovoid. The cysts that occur in chronic infections are formed when the parasites multiply and produce a wall within a host cell. The cyst wall is eosinophilic, argyrophilic, and weekly PAS - positive; the organisms within the cyst are strongly PAS - positive. During acute infections, a group of proliferative stages may be seen in a wide range of host-cell types. These have been termd "pseudocysts" and "terminal colonies" and can be differentiated from true cysts in that the organisms are at most only slightly PAS-positive and the cyst membrane is neither argyrophilic nor PAS-positive. Frenkel (1973) proposed the term "tachyzoites" for the rapidly dividing, proliferative forms of the trophozoite seen in acute infections, and the term "bradyzoites" for the slowly multiplying forms contained in true cysts (Beaver, 1984).

During a primary infection, and for a period of about two weeks, the cat sheds unsporulated oocysts that measure approximately

10 by 12 μm. The latter contain a sporoblast, and are not infective. Sporulation at room temperature requires 3 to 4 days. During this time, the primary sporoblast divides into two sporoblasts, and four sporozoites are formed within each of them. The ripe, infective oocyst thus contains two sporocysts, each with four sporozoites. These oocysts are relatively tolerant of environmental conditions and may remain infective in the soil for at least 1 year. Ingestion of the infective oocysts by a susceptible bird or mammal initiates an acute infection that usually subsides to a chronic infection, or the initial infection may be relatively mild and passes unrecognized. During the acute stage, proliferative forms occur in various tissues, but in the chronic phase of the disease, it is the cyst form that is found in the muscles and various other tissues, and is most readily demonstrated in the central nervous system (Beaver, 1984).

The cycle that occurs in cats includes both asexual multiplication (schizogony) and sexual reproduction (gametogony) within the mucosal epithelial cells of the small intestine. The final product of the sexual phase of the cycle is the fertilized macrogamete or zygote, which then protects itself with a thin but remarkably resistant wall before elimination in the feces as an unsporulated

oocyst. If a susceptible cat ingests sporulated oocysts and develops an intestinal infection, the animal will pass oocysts in 21 to 24 days. However, if a cat is fed an acutely ill mouse with proliferative forms of T. gondii in its tissues, oocysts will appear in the cat's feces in 9 to 11 days. Finally, when a chronically infected mouse with cysts in its tissues is fed to a cat, oocysts are shed in the cat's feces after only 3 to 5 days.

These observations suggest that reproduction in the proliferative stage and in the cyst stage in the tissues of non-felines that ingest oocysts fulfills an essential part of the life cycle of the parasite; hence, when a cat ingests tissues infected with one or the other of these, the cycle is completed in a much shorter time. It has been reported that the cat may also have an extraintestinal infection-proliferative or cyst forms in various tissues that are infective to animals eating the cat (Dubey, 1977).

II. Epidemiology of toxoplasmosis in man:

A. Distribution:

Toxoplasmosis is widely disseminated in most parts of the world

except extremely cold or dry climates; its distribution being dependent on the distribution of cats. The highest prevalence rates in humans are found in tropical, moist climates where cats are present. On some pacific islands, however, where cats were absent antibody to Toxoplasma was also absent or nearly so (Frenkel, 1989).

Infection is permanent in individuals and thus cumulative in populations. The yearly incidence is as high as 10% in some tropical climates. Serologic studies in children in central America indicated that up to 30% were infected in the first 5 years of life and 70% of the population in the first 25 years. High prevalence rates for infection in childhood are known from Hawaii and other Pacific islands, Japan, tropical Africa, and Brazil. In the U.S.A only 0.5% to 1% of the population becomes infected each year, and by age 25 the cumulative infection rate varies from 10% on the Pacific coast to 25% in the southeast. The incidence of congenital infections in countries where it has been studied is 7 in 1000 to 1 in 10,000 live births.

B. Epidemiology and transmission of Toxoplasma:

Feldman (1963) reported on dye-test results on sera from United States army recruits in which the percentage of those with