



ARVO

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1466 — 27

THE PATHOLOGY OF OCULAR CONGENITAL LYME BORRELIOSIS.
R. Spector, V. Hummel, R. Folberg University of Iowa, Iowa City, IA.

Purpose: This report describes the clinical, histopathological, and ultrastructural findings in the eyes of a 7 year old boy who died because of cerebral complications of congenital Lyme Borreliosis. **Methods:** Both eyes from this patient were obtained at autopsy and fixed in 10% neutral buffered formalin and postfixed in 2.5% glutaraldehyde for transmission electron microscopy. Pathologic findings were compared with extensive collections of antemortem fundus photographs. **Results:** The patient was diagnosed with Lyme disease in the neonatal period proven by serology (ELISA 1:512) and polymerase chain reaction positive identification of *Borrelia burgdorferi* in placental tissues; the mother had been treated for serologically proven Lyme disease over a period of one year before pregnancy. Histologically, the retinal pigment epithelium (RPE) was focally atrophic and hypertrophic, rendering a "salt and pepper" appearance to the fundus. Focally, the RPE migrated into the retina. The optic nerve was atrophic. A non-granulomatous choroiditis was identified. The Warthin Starry stain revealed rare spirochetes in the choroid at the posterior pole, and transmission electron microscopy confirmed their presence in the vascular endothelium of the choroid. Spirochetes had also been detected in the heart and kidneys at autopsy. **Conclusions:** The histologic findings suggest that pigmentary changes in the fundus in congenital Lyme disease are similar to those described in congenital syphilis. Spirochetes (*Borrelia burgdorferi*) were most likely transmitted from the mother to the fetus transplacentally.

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None

1467 — 28

OCULAR TOXOPLASMOSIS IN THE FETUS:
IMMUNOHISTOCHEMISTRY AND DNA AMPLIFICATION A.P. Brézin,
L. Kasner, *P. Thulliez, Q. Li, *F. Daffos, R.B. Nussenblatt, and C.-C. Chan Laboratory of Immunology, National Eye Institute, NIH, Bethesda, MD, U.S.A. and *Laboratoire de la Toxoplasmose, Institut de Puériculture de Paris, France

Background: Most cases of ocular toxoplasmosis are the results of congenital infection. The study of early stages of the infection may provide new insights into the mechanism of chorioretinal tissue damage. **Methods:** The eyes from four 24-27.5 week-old fetuses with *Toxoplasma gondii* (T. gondii) infection were studied by means of routine histopathological and immunohistochemical techniques. Two of the four were also examined by means of polymerase chain reaction (PCR). **Results:** The eyes were grossly and histopathologically normal in two cases with mild cerebral involvement, while marked retinal necrosis was present in the other two, bilaterally in one and unilaterally in the other. Although no toxoplasmic cysts were identified by routine histopathology, antigens from tachyzoites were detected by immunohistochemistry in the areas of retinal necrosis. PCR confirmed the presence of T. gondii in one of the cases with ocular lesions. Large numbers of infiltrating cells reacting with anti-pan T cell (CD3) antibodies were observed in the retinal lesions and the adjacent choroid. **Conclusion:** The presence of ocular lesions correlated with the severity of central nervous system pathology. Results suggest that T lymphocytes play an important role in the immune response to ocular toxoplasmosis from the early stages of the infection.

None

1468 — 29

EXPERIMENTAL MODEL OF ACQUIRED OCULAR TOXOPLASMOSIS IN MICE USING ME49 AND S2C9 *TOXOPLASMA GONDII* STRAINS.
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Purpose: A high incidence of both acquired toxoplasmosis and ocular manifestations in the general population (18%) have been reported in Erechim, RS, southern Brazil. We have developed an acquired ocular model of toxoplasmosis in mice. Using this model we compared eye and brain pathology induced by a Brazilian strain (S2C9) with a standard USA strain (ME49). **Methods:** The S2C9 strain was isolated in southern Brazil from a chicken whereas the ME49 strain was isolated in USA from a lamb. Female Swiss-Webster mice were inoculated by the subcutaneous (SC) or intraperitoneal (IP) route with 10 cysts of strain ME49 (25 animals) or strain S2C9 (16 animals). Mice were sacrificed on days 7, 14, 21, 28 and 42 after inoculation and the eyes and brains were examined histopathologically. **Results:** By day 14, 100% of mice developed cysts in the brain. Retinal inflammation was also noted in 100% of the animals, however, inflammation was initially seen on day 7 in the S2C9 inoculated mice and on day 14 in the ME49 inoculated mice. Moreover, the intensity of retinal inflammation in S2C9 inoculated mice was much greater than was seen in the ME49 inoculated mice. Chorioretinal scars were also observed in mice inoculated with both strains of *Toxoplasma gondii*. Retinal cysts were found in mice 28 days after inoculation with ME49 strain. **Conclusions:** This study identifies an animal model of ocular toxoplasmosis characterized by retinal inflammation, chorioretinal scarring, retinal disorganization and cyst formation. Using this model we show that the S2C9 strain, isolated from an area of high incidence of ocular toxoplasmosis, triggers a more rapid and intense ocular disease than the ME49 strain.

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1469 — 30

HISTOCHEMICAL AND FLUORESCCEIN CONJUGATED LECTIN (FCL) STUDY OF OCULAR CYSTICERCOSIS.
Jyotiirmay Biswas, Lingam Copal, G Sitalakshmi and S S Badrinath, Vision Research Foundation, Madras, India.

Cysticercosis is a common ocular parasitic infection in developing countries like India. We studied twelve specimens of ocular cysticercosis which includes intravitreal (7), subretinal (2), subconjunctival (2) and whole eyeball (1) with dissecting microscope, light microscopy using routine & special histochemical stains and lectin binding using a panel of seven commercially available FCLs. The cyst size varied from 4mm to 10mm. The body of the cysticercus comprised of a single invaginated protoscolex in the cavity of a fluid filled cyst which showed sucker, hooklets and a branched tract in protoscolex. The cyst wall was composed of three layers i.e. outer ciliated cuticle, middle muscular layer and inner tegumental cells with multiple calcareous corpuscles. Hooklets (two rows) showed auto fluorescence on polarised light. The surrounding tissue showed chronic inflammatory cells, pigment laden macrophages (in subretinal membrane) and extensive necrosis. Histochemistry revealed outer cyst wall as well as the corpuscles staining positive with alcian blue, PAS and Verhoeff-van Geison stain, the muscular layer delineated by trichome stain and corpuscles alone stained with Gomori's methanamine silver stain. Brightest degree of fluorescence (4+ to 3+) was observed with wheat germ agglutinin and concanavalin A in the outer layer of cell wall and calcareous corpuscles. Mild (1+) to no staining was observed with other five FCL's. This study describes the histochemical and FCL staining pattern of ocular cysticercus along with clinicopathological correlation.