a journal for clinicians

The Pediatric Infectious Disease Journal

Volume 7 Number 7 July 1988

Pediatr Infect Dis J, 7:514–516, 1988
0891-3668/88/$02.00/0

Vol. 7, No. 7
Printed in U.S.A.

Ocular tuberculosis in a five-month-old

MELVIN L. MORSE, MD, DANIEL J. KARR, MD AND PAUL M. MENDELMAN, MD

In recent years the few cases of ocular tuberculosis reported have been primarily in immigrant populations and in third world nations.[1–4] The common association of ocular tuberculosis with miliary disease was well-known nearly 50 years ago[5] but is not generally appreciated today. In contrast to miliary disease the incidence of ocular tuberculosis with active pulmonary disease (although controversial at the turn of the century) remains rare.[5–7] We present a case of choroidal tuberculoma in a 5-month-old, nonimmigrant American male without evidence of miliary disease who presented with lymphohematogenous tuberculosis.[8]

Case report. A 5-month-old, previously healthy Caucasian male presented with acute irritability and a 2-week history of fever. Of epidemiologic significance was the fact that the 24-year-old paternal uncle, who has lived in the household since his return from military service in Korea 2 years earlier, developed an increasing cough over the previous 4 months and was diagnosed with tuberculous pneumonia coincident with the patient's illness. Tuberculin skin tests were placed on all members of the household and all were positive. The chest radiographs of the parents were normal and they were given monotherapy with isoniazid. The chest radiograph of the 17-month-old female sibling showed hilar adenopathy and she was treated with isoniazid and rifampin. Our patient had 32 mm of induration in response to the standard tine test and hilar adenopathy on his chest radiograph.

Physical examination showed an alert, intermittently irritable child in no respiratory distress. Temperature was 104.4°F rectally. Respiratory rate was 28 and vital signs were normal. Examination of ears, nose and throat were noncontributory. No retractions of the chest wall were noted and the chest was clear to auscultation. Cardiovascular examination was normal. A hard spleen tip was palpated 1 cm below the costal margin. The liver span was 3.5 cm at the midclavicular line. The remainder of the physical examination was unremarkable; there was no peripheral adenopathy and the neurologic examination was normal.

The infant's past medical history was normal. He was the full term product of a healthy pregnancy. His growth indices were in the 50 to 70th percentile and he received regular well child care and routine immunizations. The infant's parents are middle class Americans who live in the Seattle suburbs. His mother works in a neighborhood delicatessen and his father is a salesman.

Initial white blood cell count was 21 900 with 65% neutrophils, 26% lymphocytes and 9% monocytes. Hemoglobin was 8.9 g/dl. Mean corpuscular volume was 78.6 μm^3. Platelet count was 549 000. The alanine aminotransferase level was 38 units/ml and the aspartate aminotransferase level was 40 units/liter. Cerebrospinal fluid showed a glucose of 62 mg/dl, protein of 21 g/dl, 90 red blood cells and 4 monocytes. Urinalysis, serum electrolytes, urea nitrogen and creatinine concentrations were normal. Cultures of gastric washings, spinal fluid, urine and blood were sterile. Subsequent chest radiographs showed increasing hilar adenopathy with compression of the bronchus intermedius on the right and the mainstem bronchus on the left as well as patchy parenchymal densities in the right hilum and the right middle lobe.

The infant was presumptively diagnosed as having lymphohematogenous tuberculosis and was treated with isoniazid, pyrazinamide and rifampin. He defervesced 5 days after the initiation of therapy. As part of his work-up for tuberculosis an ophthalmology consultation was obtained. Visual acuity response by fixation pattern was central, steady and maintained. The anterior segment and gross motility examinations were normal. Dilated examination of the right eye was within normal limits. The left eye was normal with the exception of a perifoveal lesion which was slightly raised and yellow-white. The lesion was deep to the retinal tissue and best localized in the choroid. The size was ¼ disc in diameter. There was no inflammatory change or opacification of the surrounding vitreous. The lesion did not extend into the fovea and there were no other lesions found in the left eye. Photo-

Accepted for publication March 16, 1988.
From the Department of Pediatrics, Valley Medical Center (MLM), Renton, WA; the Departments of Ophthalmology (DJK) and Pediatrics (DJK, PMM), the University of Washington School of Medicine, Seattle, WA; and the Department of Surgery (DJK) and the Division of Infectious Diseases (PMM), Children's Hospital and Medical Center, Seattle, WA.

Address for reprints: Paul M. Mendelman, M.D., Division of Infectious Disease, Children's Hospital and Medical Center, 4800 Sand Point Way N.E., Seattle, WA 98105.

graphic documentation was not possible until 8 days after the initial evaluation. By this time the center of the lesion had changed character and become more white. The overall size of the lesion was larger than previously noted but remained perifoveal in position. After triple drug therapy for 3 weeks the choroidal lesion quadrupled in size. The antimicrobial susceptibility of the isolate from the uncle was not available and a fourth drug, ethambutol, was added to the regimen because of the increasing size of the lesion while receiving triple therapy. After treatment for 2 months the lesion appeared to stabilize in size (Fig. 1) and no new lesions were seen. Three months later the patient's private ophthalmologist referred him for re-evaluation with the concern that the lesion had increased in size. Visual acuity by the Teller Acuity Cards demonstrated right eye 20/80 and left eye 20/600 (normal for age, 20/80 to 20/300). Examination confirmed the choroidal lesion to be 2 disc diameters in size with extension into the central fovea (Fig. 2). A sensory retinal detachment surrounded the lesion with extension to the region of the optic nerve. The vascular pattern of the mass suggested choroidal neovascularization. This could not be confirmed because it was elected not to perform fluorescein angiography under general anesthesia in view of the already damaged fovea.

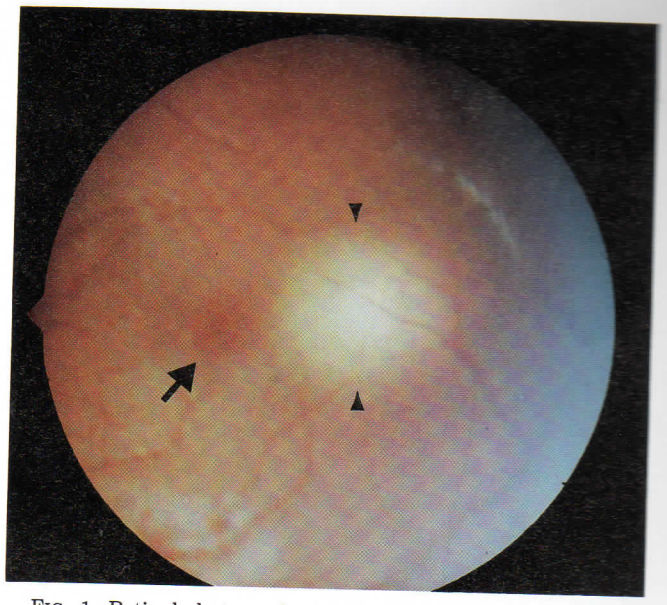

FIG. 1. Retinal photograph of the macular region of the left eye in our patient at 7 months of age. The fovea is marked by the *large arrow*. The optic nerve is to the left out of the photographic field. *Small arrows* delineate the choroidal tuberculoma. Retina and retinal vessels course over the elevated choroidal lesion.

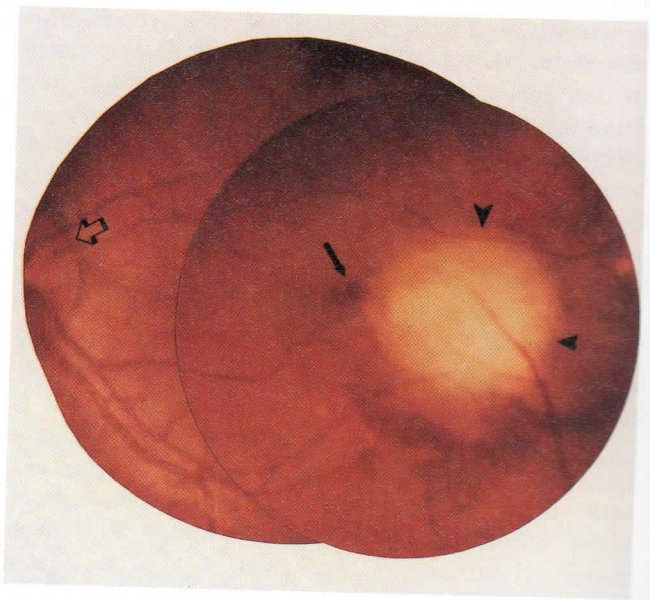

FIG. 2. Montage of the retina of the left eye of our patient at 10 months of age. For orientation, *open arrow* is at temporal border of optic nerve, *solid arrow* is pointing to fovea and *arrowheads* are at superior and temporal borders of the tuberculoma. The presence of subretinal blood (pigmented area under the tuberculoma) and subretinal exudate located at 2:30 o'clock from the tuberculoma are very suggestive of subretinal neovascularization.

DISCUSSION

In the United States ocular tuberculosis occurs primarily in malnourished immigrants[3] and its incidence in association with active pulmonary disease is reportedly rare; a figure of 1.4% has been described.[6, 7] In association with miliary tuberculosis and meningitis, choroid involvement has been reported in up to 90% of cases but only in a minority of patients with tuberculous meningitis without miliary disease.[5, 7] Excluding miliary disease intraocular disease is nearly always secondary to inactive or quiescent disease elsewhere in the body, is believed to be caused by intermittent episodes of bacillemia with seeding to the eye and occurs commonly in otherwise healthy people who are not systemically ill.[7] In contrast our patient had active and evolving (but nonmiliary) tuberculosis. The pathogenesis appeared to follow the usual pattern of acute tubercular infection of lung with lymphatic spread to regional lymph nodes followed by a transient bacteremia with spread of tubercle bacilli to a distant focus, the choroid, just before the development of hypersensitivity (manifested by systemic signs of disease and the positive tine test). Thus the necessary event preceding all cases of tuberculosis of the choroid appears to be bacteremia.

The tubercle bacillus may infect any part of the eye including the eyelids, conjunctivae, cornea, sclera, orbital structures (lacrimal gland, extraocular muscles), retina, optic nerve and the uvea (iris, ciliary body and choroid); the choroid is the most common site of involvement. Choroidal tubercles may be the first sign of disseminated disease during the initial several weeks and are almost always found early in the course of illness (at the time of the first or second ophthalmic examination[5, 9–11]). Thus examination of the fundi in

all cases of primary tuberculosis may help to establish an early diagnosis. Eye examination in our patient confirmed the disseminated nature of the disease and prompted aggressive chemotherapy which interrupted the progression of disease; the course of patients with protracted lymphohematogenous tuberculosis often ended tragically with tuberculous meningitis.[8]

Repeated fundal examination may be useful prognostically because the evolution of new tubercles in the choroid or failure of resolution of previously detected lesions while receiving therapy have been reported as ominous signs.[5] In contrast resolving lesions would confirm adequacy of treatment. The healing tuberculoma of the choroid becomes larger, paler and more distinct with time, with the healed lesion becoming parchment-white with sharp borders and usually pigmentation.[5,7] In our patient a fourth drug was added to the regimen because the natural occurrence of lesion enlargement with resolution of disease was not appreciated. Because of the perifoveal location of choroidal tuberculomas, sequelae may include significant loss of vision.

In visually immature children a choroidal tuberculoma in the macular region of the retina represents a potential cause for permanent vision loss even when the central macula (fovea) is spared. Transient disuse of one eye as a result of distortion of vision from elevation of the macula by an underlying choroidal lesion or from inflammation and opacification of the vitreous could produce amblyopia even when there is documented complete resolution of the choroidal lesion. Visual acuity assessment and fundus evaluation must be followed closely with early amblyopia therapy when necessary. Choroidal tubercles producing significant scarring sequelae of the central macular region (as in our patient) would result in loss of vision usually not retrievable with amblyopia therapy.

The differential diagnosis of choroidal granulomatous disease includes syphilis, toxoplasmosis, toxocariasis, histoplasmosis, brucellosis, sarcoidosis, melanoma, metastatic tumor, retinoblastoma and tuberculosis. Histopathologic confirmation is usually not possible unless tumor is suspected and the eye is enucleated. Enucleation for preoperative suspicion of retinoblastoma has occurred in a tuberculous eye,[4] prompting several authors to recommend the tuberculin skin test in the work-up of such ocular lesions.

More commonly, as in our case, a presumptive diagnosis is made based on associated clinical findings. Fluorescein angiography studies would have provided important additional information, especially in view of our concerns for choroidal neovascularization in the tubercle. Although not diagnostic the fluorescein angiographic findings in an isolated choroidal tuberculoma help to distinguish the lesion from other choroidal lesions such as melanoma.[11] Additionally fluorescein angiographic documentation of neovascularization would have provided useful new information because a recent review of choroidal neovascularization in children did not include tuberculosis as a cause.[12]

Our particular case appears unique in that it occurred in the absence of miliary disease in association with lymphohematogenous tuberculosis and active pulmonary disease in a nonimmigrant Caucasian middle class family and serves as a reminder of the protean manifestations of this ancient disease.

ACKNOWLEDGMENTS

We thank Arnold L. Smith for the recommendation of a consultation with ophthalmology.

REFERENCES

1. Cangemi FE, Friedman AH, Josephberg R: Tuberculoma of the choroid. *Ophthalmology* 87:252–258, 1980
2. Saini JS, Mukherjee AK, Nadkarnf N: Primary tuberculosis of the retina. *Br J Ophthalmol* 70:533–555, 1986
3. Dinning WJ, Marston S: Cutaneous and ocular tuberculosis: A review. *J R Soc Med* 78:576–581, 1985
4. Ayanru JO, Alli AF, Elebesunu MI: Tuberculoma of the eye: A case report. *Trop Geogr Med* 38:301–304, 1986
5. Illingworth RS, Wright T: Tubercles of the choroid. *Br Med J* 4572–4580, 1948
6. Donahue HC: Ophthalmologic experience in a tuberculosis sanitorium. *Am J Ophthalmol* 64:742–748, 1967
7. Duke-Elder S, Perkins ES: Diseases of the uveal tract. *System of Ophthalmology.* St. Louis, Mosby, 1966, vol 9, pp 246–285
8. Smith MHD, Marquis JR: Tuberculosis and other mycobacterial infections. *Textbook of Pediatric Infectious Diseases.* Edited by RD Feigin, JD Cherry. Philadelphia, WB Saunders, 1987, pp. 1342–1387
9. Cargill LV, Mayou S: A case of miliary tuberculosis in adults. *Trans Ophthalmol Soc UK* 26:101–107, 1906
10. Olazdbal F: Choroidal tubercles: A neglected sign. *JAMA* 200:104–107, 1967
11. Jabbour NM, Faris B, Trempe CL: A case of pulmonary tuberculosis presenting with a choroidal tuberculoma. *Ophthalmology* 92:834–837, 1985
12. Wilson ME, Mazur DO: Choroidal neovascularization in children: Report of 5 cases and literature review. *J Pediatr Ophthalmol Strabismus* 25:23–29, 1988