

SHORT COMMUNICATION

Case reportOptic nerve lesion following neuroborreliosis:
a case reportC. BURKHARD^{1,3}, M. GLEICHMANN², H. WILHELM³¹Ahaus Eye Hospital, Ahaus²University Neurological Hospital, Tübingen³Dept. of Pathophysiology of Vision and Neuro-ophthalmology, University Eye Hospital, Tübingen - Germany

PURPOSE. *Neuroborreliosis may cause various neuro-ophthalmological complications. We describe a case with a bilateral optic neuropathy.*

CASE REPORT. *A 58-year-old female developed facial paresis six weeks after an insect bite. One week later she developed bilateral optic disc swelling with haemorrhages and nerve fibre bundle defects in the lower visual field of the left eye. In CSF and serum, raised IgM and IgG titres to Borrelia burgdorferi were found. Systemic antibiotic treatment led to improvement of the vision and facial paresis, but not all visual field defects resolved, probably due to ischemic lesions of the optic disc.*

DISCUSSION/CONCLUSIONS. *In optic nerve lesions due to neuroborreliosis it is difficult to distinguish between inflammatory and ischemic lesions. This patient demonstrated features of an ischemic optic nerve lesion. (Eur J Ophthalmol 2001; 11: 203-6)*

KEY WORDS. *AION, Neuroborreliosis, Bannwarth syndrome, Complications, Optic disc, Papilledema*

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INTRODUCTION

Lyme disease is a tick-borne infectious disease due to a spirochetal organism, *Borrelia burgdorferi*, which was discovered in 1982 (1). The clinical manifestations have been divided into three chronological stages, though not all patients present each stage. The first stage comes directly after the bite of a tick and is characterised by local erythema migrans and often unspecific flu-like symptoms. After an incubation period of 3-4 weeks, 15% of patients develop neurological manifestations (stage II). The third stage features chronic polyneuropathy and encephalomyelopathy (2-4).

Case report

At her first visit a 58-year-old female patient reported a short period of eye-pain six weeks earlier. Ophthalmological inspection found nothing remarkable.

Two weeks later she developed facial paresis on the right side, which was treated with oral steroids, but did not improve. An ophthalmological examination because of blurred vision showed bilateral swollen discs. Visual acuity was 20/60 in the right eye and 20/30 in the left. The MRI scan was normal.

Visual acuity increased to 20/30 right eye and 20/25 left eye within one week. At that time the patient complained of seeing a shadow in the lower visual field of the left eye for the first time. Perimetry showed an arcuate defect in the lower half of the visual field.

The next day she was examined at the University Eye Hospital in Tübingen. At the first examination we found visual acuity of 20/30 in both eyes. Pupil, ocular motility and slit-lamp examination were unremarkable. Both eyes had papilledema with hemorrhagic margins (Fig. 1 a and b). Perimetry showed a reduced central differential luminance sensitivity and relative central scotoma in the right eye and reduced central differential luminance sensitivity in addition to the arcuate defects

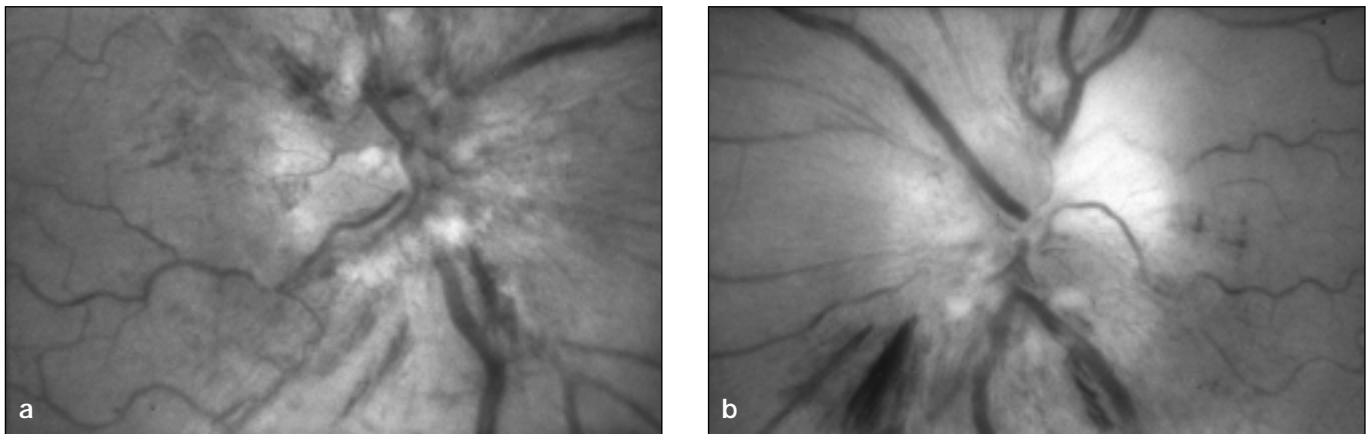


Fig. 1 - Right (a) and left (b) optic discs at the first examination.

in the lower half of the left visual field (Fig. 2).

Neurological examination found facial paresis on the right side, and typical erythema migrans on the back of the right hand, with almost complete healing in the right axilla. Further clinical neurological examination remained inconspicuous. On specific inquiry the patient reported an "insect bite" in her right axilla two months before.

Cerebrospinal fluid (CSF) puncture offered an unequivocal pleocytosis of 240/3 cells. Serological test for syphilis, listeriosis, rickettsiosis, brucellosis and toxoplasmosis on serum and CSF were negative; a serological test for Lyme disease showed a raised IgM titre against *Borrelia* and specific protein bands for IgG and IgM against *Borrelia* in the Western blot. The titre and specific protein bands for IgG and IgM against *Borrelia* were high in CSF. This is considered a typical finding for acute neuroborreliosis.

A 14-day course of intravenous ceftriaxone was administered. The facial paralysis and erythema migrans improved rapidly. Lymphocytes declined to 36/3 cells on the eighth day of therapy.

After four days of therapy visual acuity had improved to 20/20 (right eye) and 20/25 (left eye). The optic discs and visual fields remained unchanged. At the last examination two months after therapy, visual acuity was still 20/20 (right eye) and 20/25 (left eye). A slight relative afferent pupillary defect was present on the left eye but the right optic disc was normal. The left disc had a blurred margin and was slightly atrophic in the upper half. Perimetry still showed an arcuate defect due to nerve fibre loss in the lower half of the visual field (Fig. 3). The facial paresis had resolved completely.

DISCUSSION

This patient suffered from serologically proven neuroborreliosis. She presented with facial paresis, bilaterally swollen discs and vision loss. Treatment with ceftriaxone improved vision and facial paresis markedly, but an arcuate scotoma and sectorial disc atrophy persisted in one eye. The optic disc swelling may be explained in two ways.

First, it could be indicative of bilateral papillitis, completely restored in one eye, but with incomplete recovery in the second eye, probably because of vasculitic vessels occlusion. Second, we might be dealing with a papilledema caused by meningitis, and the visual loss could be seen as a sequela of the edema.

Visual field and disc appearance on the left side are strongly reminiscent of anterior ischemic optic neuropathy (AION). It is questionable whether this was caused by local occlusive vasculitis or was simply a complication of the papilledema. Intracranial pressure was not measured on CSF puncture.

After an incubation period of 3-4 weeks 15% of patients develop neurological manifestations (stage II). The Bannwarth syndrome is the most frequent neurological manifestation, affecting 80% of patients reaching this stage (cardinal symptoms are radicular pain, peripheral paresis, especially facial paresis, and pleocytosis with 10/3 - 1000/3 white cells) (4). All ocular segments can be involved (2, 5) through uveitis and neuro-ophthalmological manifestations are most frequent in stage II (3). Horner's syndrome, idiopathic intracranial hypertension, optic disc edema, optic neuritis, optic neuropathy and optic atrophy are neuro-

Fig. 2 - Right and left 90° visual fields at the first visit.

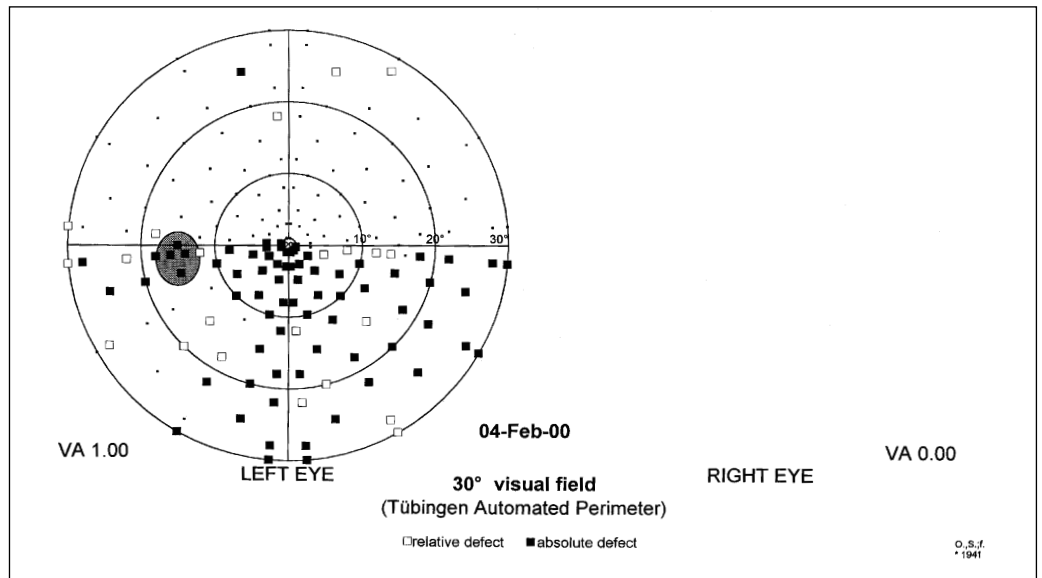
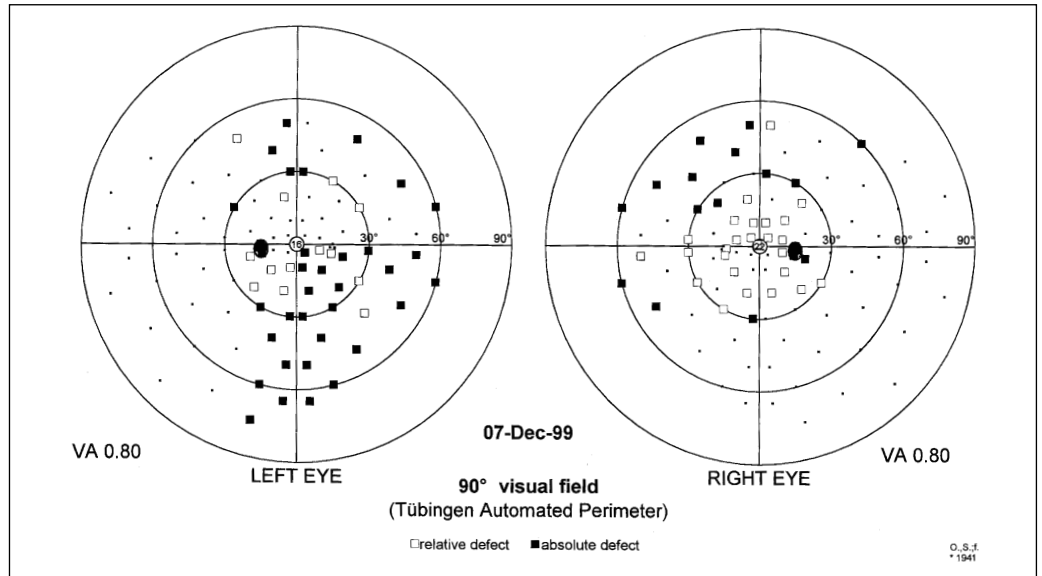


Fig. 3 - Left 30° visual field two months after therapy.

ophthalmological complications (2). AION as a complication of neuroborreliosis has been described in five patients (6-10).

Antibiotic therapy is recommended in all three stages. In the first stage oral treatment is preferred. In stages II and III the drug is best given intravenously. Pfister published a summary in 1998 (4). Oral steroids were tested on radicular pain and had effect so their routine use is not recommended (11). For AION, no therapy is of significant benefit (12).

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