understood because of the removal of the absorptive surface. The cause of vitamin deficiency in hemicolectomy is uncertain, but it may be related to decreased transit time through the small intestine owing to gut hypermotility. Chronic diarrhoea and the continued use of laxatives have been implicated as the cause of vitamin deficiency.[9]

Our patient did have chronic diarrhoea since the operation, and we feel that gut hypermotility was the cause of his vitamin A deficiency. The reason why the patient only had a fat-soluble vitamin deficiency is that, in mild intestinal absorption disorders, it is the fat-soluble vitamins that are least well absorbed.[10]

In summary, this is the first reported case of Bitot's spots following hemicolectomy.

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Sir,

Bilateral transient amaurosis following *Mycoplasma pneumoniae* infection: a manifestation of acute disseminated encephalomyelitis

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Mycoplasma pneumoniae is an atypical bacterium that can cause a great variety of respiratory infections and can be responsible for ocular involvement such as conjunctivitis, anterior uveitis, and very rarely optic neuropathy. We report a case of bilateral vision loss with optic disc swelling that developed 4 days after starting treatment for atypical pneumonia and was accompanied by multiple demyelinating brain lesions.

Case report

A 24-year-old male presented as an outpatient with a 2-day history of deterioration of vision in both eyes, headache, stiff neck, and general malaise. Owing to febrile illness accompanied by herpes labialis, he had been taking amoxicillin for a week without improvement. An infectologist diagnosed atypical pneumonia 4 days before ophthalmological examination and administered clarithromycin 500 mg b.i.d. The patient had been without fever for the preceding 3 days.

On examination, his unaided visual acuities (VA) were counting fingers (CF)/2 m right, 0.25 left. Pupillary reactions were diminished and relative afferent pupillary defect (RAPD) was absent. Fundoscopy revealed swelling of both optic discs with few splinter haemorrhages peripapillary and mildly dilated veins. Optic papillitis was diagnosed, however, because of the history of fever and meninigeal signs present, he was referred to an infectologist. Laboratory tests and a lumbar puncture were carried out. Serology was positive for *M. pneumoniae* (IgG >200 IU/ml; range 20–30 IU/ml, IgM 149 IU/ml, range 13–17 IU/ml), negative for *Chlamydia pneumoniae*, *Chlamydia psittaci*, *Chlamydia trachomatis* and *Legionella pneumophila*, Herpes simplex, Varicella Zoster and Epstein–Barr virus. A lumbar

puncture was performed and cerebrospinal fluid (CSF) was sent for analysis. The CSF pressure was normal (14 mmH_2O), total proteins were 0.38 g/l (normal range 0.15–0.45) with an increased level of immunoglobulin (Ig) M 2.2 mg/l (less than 0.7) and absent oligoclonal bands.

The patient was then reviewed in the Eye Clinic 4 days after initial examination. His vision deteriorated to no light perception in both eyes. The pupils were nonreactive and fundus appearances were unchanged. The consultant neurologist could not find other cranial nerve abnormalities or motor deficits. The patient was admitted to the Eye Clinic. Magnetic resonance imaging (MRI) demonstrated multiple round lesions located in the cerebellar pedunculum, thalamus, internal capsule, corpus callosum, and corona radiata, showing homogeneous contrast enhancement. Bilateral optic neuritis in the course of acute disseminated encephalomyelitis (ADEM) was diagnosed. Intravenous methyprednisolone (1000 mg/day) was administered for 5 days, followed by oral 60 mg Medrol in tapering doses, while still being treated with clarithromycin 500 mg b.i.d. His VA improved in the next 10 days to 0.7 right and 0.8 left, and 1.5 months later to 1.0 in both eyes. During the last examination 6 months later, VA was the same, the pupils were symmetrical, reactive, RAPD absent, but optic atrophy was noted. Visual fields in the central and nasal part showed slightly decreased retinal sensitivity in both eyes.

Comment

ADEM is a rare inflammatory demyelinating disease of the central nervous system, typically occurring after infections or vaccinations and affecting predominantly children and young adults.[1] Pathogenesis of ADEM is not known; an autoimmune response mediated by autoreactive T lymphocytes with specificity for myelin antigens (such as myelin basic protein) triggered by infection or immunization is considered to be a possible aetiological factor.[2] Characteristic clinical features include sudden onset of multifocal neurological disturbances such as bilateral optic neuritis, aphasia, motor and sensory deficitis, ataxia and signs of an acute meningoencephalopathy with meningismus, seizures, and psychosis.

As in the case highlighted here in, MRI reveals single or multifocal areas of increased signal intensity on T2-weighted sequences predominantly in white matter, but gray matter can also be affected, particularly basal ganglia, thalami, and brainstem. CSF can be normal, but often shows pleocytosis and only rarely intrathecal oligoclonal IgG production. In this case, there was increased intrathecal synthesis of IgM which was also found in 25% of adult patients with ADEM.[3]

Milla *et al*[4] reported a case of severe visual loss following *M. pneumoniae* pneumonia accompanied by single MRI lesion in the white matter with good visual recovery. The authors concluded that optic neuropathy occurring in a young patient within a few days after a febrile illness and especially pneumonia appeared characteristic enough to suggest infection with *M. pneumoniae*. Bilateral optic neuritis has been described as a common finding in ADEM,[5] but was found only in one out of 40 adults diagnosed as ADEM.[3] In the same study, 35% of patients experienced a second clinical episode during the mean follow-up of 38 months and were finally diagnosed as MS. Patients with ADEM were younger, and a preceding infection was present significantly more often than in patients with MS. As fever and infection are also well-known trigger factors for MS attacks, the presence of prior infection cannot reliably distinguish a first episode of MS from monophasic ADEM. Also the MRI findings cannot differentiate ADEM from MS.[5]

Most patients with ADEM had a favourable outcome.[5] At follow-up examinations, 49% were without symptoms, 35% had minor nondisabling symptoms, and 12% had moderate deficits. Among patients with MS (ADEM converting to MS), only two (14%) were without symptoms.

No therapy has been established by controlled clinical trials and spontaneous improvement has been repeatedly noted.[6] Most authors report favourable response to corticosteroids, especially pulsed intravenous steroids with an oral taper.

In this particular case, sudden onset of bilateral neuritis following infection, MRI changes, and complete recovery are indicative for ADEM. For definite diagnosis to confirm a monophasic course, follow-up is necessary, as more than one-third of the patients initially diagnosed as ADEM converted to MS.

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Sir,

Chronic postoperative uveitis—a clinicopathological case report

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The management of chronic postoperative uveitis following uneventful cataract surgery is challenging because of difficulties in isolating low-grade microorganisms from such eyes.[1,2] Often, the initial response to conventional treatment may be poorly sustained, needing more aggressive intervention. With timely intervention, the prognosis can be favourable compared to acute infective endophthalmitis.

Case report

A 72-year-old lady underwent uncomplicated phacoemulsification cataract surgery with implantation of a 6 mm optic Acrysof Intraocular lens (IOL) in her left eye. She was prescribed prednisolone acetate 1% drops and fusidic ointment, and was 'discharged' 6 weeks later with a best visual acuity of 6/12. At 2 months postoperatively, she presented with symptoms and signs of uveitis in the operated eye. The visual acuity was 6/12, and the eye was red with 10–15 cells per high power field (HPF) in the anterior chamber without flare or hypopyon. The patient was recommenced on topical prednisolone acetate 1% drops every 4 h. After 1 week, the inflammation had subsided and the treatment was discontinued. The patient had recurrent episodes of uveitis and was treated at various stages with topical, orbital floor, and systemic steroids. She was referred to our uveitis clinic 5 months following surgery. The visual acuity was 6/36 and N18 with 10–15 cells per HPF in the anterior chamber. Vitreous cells were evident. Clinically, the capsular bag was clear and free of plaques. Fundus fluorescein angiography revealed gross disc oedema and cystoid macular oedema. A vitreous biopsy was carried out together with an intravitreal injection of 2 mg vancomycin. The vitreous biopsy was sterile and the anterior chamber sample showed only inflammatory cells. She was maintained on twice daily topical prednisolone acetate 1% drops. At 12 months postsurgery, she presented with a 6 mm hypopyon. The visual acuity had deteriorated to perception of light. The capsular bag was removed *in toto* together with the IOL. Intravitreal vancomycin 2 mg and ceftazidime 1 mg were injected. The anterior chamber tap revealed Gram positive cocci and bacilli. The histology specimen was made up of a capsular bag ($9 \times 5 \times 3\,mm^3$) enclosing an IOL. The capsular tissues comprised a lens capsule, degenerative cortical lens fibres, an area of capsular fibrosis, and a focus of acute inflammation. This reaction appeared to be located towards the lens equator, in association with one of the IOL haptics, and consisted largely of calcified degenerated polymorphs, calcified material, a microabscess, and some fibrous tissue (Figure 1). No microorganisms were seen. The anterior chamber became quiet within a week. After 12 months, the eye remains free of inflammation with no medication. The visual acuity is 6/36 with an aphakic correction. The view of the fundus is clear, revealing a fine epiretinal membrane secondary to long-standing macular oedema.

Comment

Chronic postoperative endophthalmitis is defined as any inflammation that presents more than 6 weeks after surgery. This definition is arbitrary as the time of onset may be influenced by the postoperative use of steroids and the virulence of the microorganisms involved. Whereas the Endophthalmitis Vitrectomy Study has produced a protocol for treating acute infective endophthalmitis,[3] the management of chronic cases is less certain. Obtaining samples from the anterior chamber and vitreous and intraocular injection of wide-spectrum antibiotics is commonly employed to treat these cases.[4] Further difficulty in the management of these cases arises if there is no response to repeat intraocular antibiotic administration[4,5] and/or microorganisms are not isolated by conventional methods.[6] Identification may need special culture media and prolonged incubation time because of the slow-growing nature of microorganisms such as *Propionibacterium acnes* and *Corynebacterium* species. The persistence of active chronic inflammation also raises the