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Anterior diffuse scleritis diagnosed as conjunctivitis

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Abstract

This article presents a case of anterior diffuse scleritis that initially was diagnosed as conjunctivitis. Anterior diffuse scleritis (ADS) is a potentially vision-threatening inflammation of the sclera whose etiology may include autoimmune and systemic conditions such as rheumatoid arthritis and tuberculosis. The signs and symptoms of ADS include pain, tearing, tenderness, redness, painful sensitivity to light and decreased visual acuity. Ocular and physical examinations including blood tests to rule out underlying causes are important. Medications such as corticosteroids, non-steroidal anti-inflammatory drugs and possibly immune-suppressants are used in the management of ADS. If care is not taken, ADS can be mis-diagnosed as conjunctivitis because the redness is similar in both conditions. Such mis-diagnosis can be sight-threatening and therefore it is essential that primary eye care practitioners are cautious in all diagnoses of red eye conditions. (*S Afr Optom* 2012 **71**(1) 51-54)

Key Words: Anterior diffuse scleritis, conjunctivitis, poor vision, ocular pain, photophobia, tearing

Introduction

Scleritis is a potentially blinding inflammatory disease that affects the sclera[1]. The condition can result from several autoimmune and systemic diseases[1, 2]. Recently described subsets of T-helper lymphocytes, known as Th-17, have emerged as key factors in the pathogenesis of scleritis[1, 2]. Scleritis is usually associated with autoimmune diseases such as rheumatoid arthritis, systemic lupus erythematosis, polyarteritis nodosa, Wegener's granulomatosis, ankylosing spondylitis, giant cell arteritis and gout[3, 4]. Recently, scleritis has also been associated with erythema elevatum diutinum, a chronic and rare dermatosis that is considered to be a variant of leukocytoclastic vasculitis [5]. Other systemic conditions such as tuberculosis, herpes zoster ophthalmicus and syphilis have been reported to be associated with scleritis and sometimes the cause is unknown [6].

Scleritis is accompanied by severe pain of gradual onset that often radiates to the peri-orbital region [7-9]. Other symptoms include photophobia, tearing, and decreased vision (if other ocular tissues are involved)[7-9]. The most important ocular signs of scleritis are globe tenderness on palpation, sectorial or diffuse scleral erythema, thinning with bluish hue, oedema and possible nodules or necrosis[7-9]. Scleritis is usually confined to one eye, but may affect both eyes and there may be possible corneal or intraocular inflammation[7-9]. If left untreated, scleritis may cause perforation of the eyeball, leading to vision loss[7-9]. This article describes the case of a patient with ADS that was previously treated as bacterial conjunctivitis by ophthalmic nurses.

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Epidemiology

Although scleritis is not common, its exact incidence is unknown[7-9]. Scleritis can also be attained through disorders of menstruation[7-9]. It is for this reason that scleritis occurs more frequently in women aged 30 to 60 years and is rare in children[7-9]. Scleritis is more common in the fourth decades of life, with a peak incidence in the fifth decade[7-9].

Classification

Scleritis may be classified into anterior and posterior[7-9]. There are several different sub-types of anterior scleritis: diffuse, nodular, necrotizing scleritis with inflammation and necrotizing without inflammation (scleromalacia perforans)[7-9]. Diffuse scleritis is the most common causing generalized inflammation, nodular causes the affected area of the sclera to be confined to small nodules and necrotizing scleritis is the most severe[7-9]. Posterior scleritis is rare and is characterized by flattening of the posterior aspect of the globe, thickening of the choroid and sclera, and retrobulbar oedema[7-9]. Posterior scleritis usually presents with poor or double vision, severe pain and proptosis[7-9].

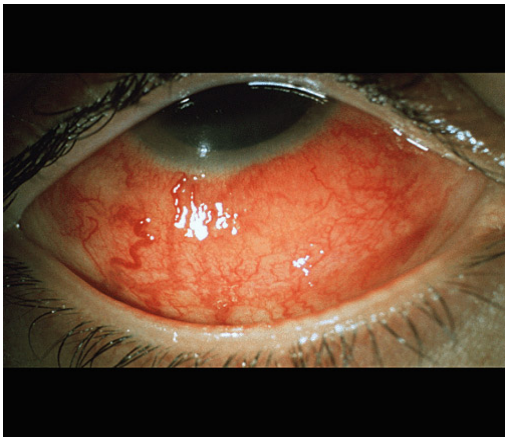

Figure 1 Anterior diffuse scleritis (source: http//www.redatlas.org).

Management

Differentiating infectious scleritis from noninfectious scleritis is important because corticosteroid therapy (often used in noninfectious autoimmune scleritis) and immune-suppressive scleritis are generally contraindicated in active infections[1]. The mainstay initial therapy of noninfectious scleritis includes a corticosteroid such as prednisone and the use of nonsteroidal anti-inflammatory agents for pain relief1. Diffuse scleritis is the form that is most responsive to therapy[1, 7-9]. In more aggressive cases of scleritis, chemotherapy (such as immunosuppressive therapy with such drugs as cyclophosphamide or azathioprine) may be used to treat the disease[1, 7-9]. The most severe form, necrotizing scleritis has poor visual outcomes and requires surgery to repair damaged corneal or scleral tissue to preserve the patient's eye[1, 7-9]. Blood tests play an important role in ensuring that there is no underlying cause of scleritis[1, 7-9]. If there is involvement of the back of the eye, neuro-imaging tests such as computed tomography (CT) scan, magnetic resonance imaging (MRI) or ultrasonography of the eye may be ordered[1, 7-9].

Case report

SM, a 46 year old Black female cleaner, presented to the Optometry clinic of the University of KwaZulu-Natal on the 25th of May 2011 at 14:00 pm. She complained of a left red eye over the preceding eight weeks, photophobia, tearing and constant severe ocular pain. She also reported that the pain was so severe that it prevents her from sleeping at night. She did not report any history of trauma. SM reported that she visited her local community clinic, where an ophthalmic nurse told her she had conjunctivitis and placed her on chloromycetin eye ointment. She, however, said that despite the ointment given, the redness and pain were getting worse. Previous ocular, medical and family histories were unremarkable. Besides the chloromycetin ointment, she reported not using any other medication or having any allergies to the same. Blood pressure for the right arm while seated measured 112/76 mmHg.

Clinical findings

Presenting unaided visual acuity (VA) measured with the Snellen chart was 6/6 in the right eye and 6/9 in the left eye. Pupils were round and reactive to light and accommodation with no afferent pupillary defect in either eye. Intraocular pressure (IOP) measured with the Nidek Tono-Pachymeter NT530P was 13 mmHg in the right eye and 12 mmHg in the left eye

at 15:45 pm. Direct ophthalmoscopy revealed nothing significant with both lens and vitreous clear. Fundus assessment revealed well-perfused optic nerve heads with cup to disc ratios of 0.5H/0.5V in the right eye and 0.4H/0.4V in the left eye. No abnormalities were noted in the peripheral fundi and macular regions of both eyes.

Anterior segment evaluation of the left eye with a slit lamp biomicroscope revealed diffuse redness of the sclera and bulbar conjunctiva. However, the palpebral conjunctiva was normal. Areas of sub-epithelial haze were noted in the cornea of the left eye but the iris was still visible. Cover tests did not reveal any tropia or phoria and extraocular muscles were not restricted in all positions of gaze. Confrontation visual field was full in both eyes. Refraction was not done on the first visit as she was experiencing severe pain. Palpation of the eye revealed some slight tenderness and the sclera looked oedematous. Digital manipulation of the overlying tissue revealed minimal mobility of the vessels thereby indicating that the significant hyperemia was scleral in origin. Also, instillation of eye gene drops (naphazoline) in the left eye did not produce blanching. SM was referred to an ophthalmologist in Addington hospital for confirmation of diagnosis of ADS and to initiate medical treatment.

SM reported back to the Optometry clinic on the 7th of July 2011 for a follow-up with a report from the ophthalmologist. The report confirmed the diagnosis of ADS (without any associated disease) and showed that SM was given prednisone and amitriptyline. Unaided VA in the left eye was 6/6. Conjunctival and scleral congestion and chemosis had completely resolved. External and internal ocular structures were normal. SM reported that she was no longer experiencing any pain. She was given a six weeks appointment for follow up examination.

Discussion and conclusion

The diagnosis of ADS is often confused with that of conjunctivitis due to similarities in some of the signs and symptoms of the two conditions and also the relative non-familiarity of the eye care professional with scleritis[10, 11]. Both ADS and conjunctivitis are causes of red eye, conjunctivitis being the most common cause[12]. Conjunctivitis refers to inflammation of the conjunctiva, and is usually associated with blepharitis, recurrent styes or meibomianitis[12]. This condition is generally treated with the application of topical antibiotics and good eyelid hygiene[13, 14]. It often presents in one eye after direct contact with the microbe, the second eye becoming involved soon after[13, 14]. During examination, no papillae or follicles were observed in SM, reflecting absence of conjunctivitis. Although there may be an irritation and photophobia with conjunctivitis, patients do not usually complain of severe pain[13, 14]. SM had severe peri-orbital pain, scleral oedema and tenderness and reduced VA in the left eye, which are not typical features of conjunctivitis but suggest more serious underlying ocular disease processes such as acute angle-closure glaucoma or uveitis. However, the IOPs and optic nerves were normal and the anterior chamber was quiet. This ruled out the possibility of acute angle-closure glaucoma and uveitis. Tearing or photophobia without mucopurulent discharge also occurs in patients with ADS[15]. SM's visual acuity of the left eye was affected due to the corneal involvement.

The distinction between episcleritis and ADS can also be difficult to make due to the similarity in the pattern of hyperemia between the conditions[16]. Furthermore, episcleritis often co-exists with ADS[16]. However, episcleritis is generally a self limiting condition, not associated with the severe peri-orbital pain and with limited complications[16]. Topical phenylephrine 2.5% or 10% will constrict the vessels of the conjunctiva and episclera but not the deeper scleral vessels and this may help the clinician in the differential diagnosis of conjunctivitis/episcleritis and ADS[16]. Anterior diffuse scleritis must also be differentiated from preseptal or orbital cellulitis where there is proptosis and impairment of eye movements[17]. Similarly, posterior scleritis presents with proptosis, eyelid oedema and restriction of ocular movements with periscleral inflammation spreading to the orbit and extraocular muscles and increased IOP[11, 18]. These clinical findings were not found in SM, therefore making preseptal cellulitis, orbital cellulitis or posterior scleritis very unlikely.

The treatment option for SM was prednisone and amitriptyline, which yielded good results. Amitriptyline has been reported to be an effective option for pain relief which might otherwise require immunosuppressants[19]. However, it should be used with cau-

tion as it may cause coma-shaped bullous lesions in the lower extremities[20].

An accurate diagnosis of ADS is important as ocular complications may occur. These complications include keratitis, cataracts, uveitis and glaucoma[7-9]. Therefore, proper assessment of VA, measurement of IOP, slit lamp examination and ophthalmoscopy are important in making an accurate diagnosis of ADS. The visual outcome in ADS also depends on the time interval between diagnosis and initiation of treatment and therefore, prompt referral to the ophthalmologist is important[7-9]. Further, the correct diagnosis and appropriate therapy can halt the progression of ocular and systemic processes in patients with ADS associated with systemic conditions[7-9].

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