pulsations may be very useful for the ophthalmic assessment whether the intracranial pressure is normal, or if spontaneous vein pulsation cannot be detected, whether the intracranial pressure is likely to be elevated.¹⁻⁴

Case report

Our clinical observational case series included 199 patients (384 eyes; mean (standard deviation (SD)) age, 63.9 (12.3) years; range, 7–90 years) attending the hospital because of cataract ($n = 66$), age-related macular degeneration ($n = 37$) or other diseases ($n = 96$) not affecting the optic nerve or primarily the inner retina. Intraocular pressure ranged between 10 and 21 mm Hg. The mean (SD) refractive error was -0.15 (3.04) D (range, -15.5 to $+6.50$ D). Using tropicamide and phenylephrine 5% eye drops, the pupil was dilated. The optic disc was examined using a slit lamp and a hand-held 78-D ophthalmoscopic lens for at least 30 s. Any small pulsatile synchronous movement of the central retinal vein or its major branches inside the optic disc was noted as spontaneous pulsations. The non-contact examination was part of a routine ophthalmoscopic examination of the fundus of the patients. The medical ethics committee approved the study protocol, fulfilling the criteria of the Declaration of Helsinki.

Spontaneous central retinal vein pulsations were detected in 343 (89.3%; 95% confidence interval (CI), 7.6 to 13.8) eyes of 186 (93.5%; 95% CI, 3.1 to 10) patients. The presence of a spontaneous central retinal vein pulsation was statistically independent of age ($p = 0.57$; 95% CI, 0.98 to 1.04) and gender ($p = 0.78$; odds ratio (OR): 1.19; 95% CI, 0.39 to 3.68). Frequency of detected spontaneous central retinal vein pulsations was significantly associated with hyperopic refractive error ($p = 0.017$; 95% CI, 1.08 to 2.19).

Comment

Confirming the findings of previous studies,^{5,6} the results of the present study (relatively high frequency of ophthalmoscopically detected spontaneous pulsations of the central retinal vein), suggest that about 10% of otherwise normal people do not have ophthalmoscopically detectable spontaneous central retinal vein pulsations. Our results as well as the previous findings may be important for several reasons: in a patient with an increased intracranial pressure, the retrobulbar cerebrospinal fluid pressure rises, eventually equalling the intraocular pressure. When this occurs, there is no longer a fluctuating intravascular pressure gradient between the intraocular retinal veins and the retrolaminar retinal vein. The venous blood flow becomes constant, and spontaneous central retinal vein pulsations cease.⁴ Based on the results of previous studies and the present investigation a lack of a spontaneous central retinal vein pulsation in a patient with a presumed increase in intracranial pressure may suggest with a probability of 10% a normal finding and with a probability of about 90% an abnormal situation. To cite an example, by Levine searched for spontaneous retinal venous pulsations in 218 subjects in a previous investigation.⁶ Spontaneous venous pulsations were present in 87.6% of 146 unselected subjects 20–90 years of age, and were absent in 100% of 33 patients with raised intracranial pressure without papilloedema and in 10 patients with papilloedema. Lumbar puncture in nine patients with raised intracranial pressure established the upper level at which spontaneous pulsations

disappear at 190 mm H₂O, and no pressure above 180 mm H₂O was found in 29 patients with venous pulsations present before lumbar puncture. Some normal subjects with no pulsations showed definite pulsations on subsequent examinations. Levine's findings show that presence of spontaneous venous pulsations is a reliable indicator of an intracranial pressure below 180–190 mm H₂O. They suggest, like the results of the present investigation, that pulsations may be absent in normal subjects and absence of pulsations is, therefore, not a reliable guide to raised intracranial pressure.

In conclusion, ophthalmoscopic assessment of spontaneous pulsation of the central retinal vein is useful for the estimation of increased intracranial pressure; in a similar manner, it may be useful for the diagnosis of glaucomatous optic neuropathy and retinal vein occlusions, in which increased central retinal vein pressure has been reported.⁷

Bjoern Harder, Jost B Jonas

Department of Ophthalmology, University of Heidelberg, Mannheim, Germany

Correspondence to: Dr J B Jonas, Universitäts-Augenklinik, Theodor-Kutzer-Ufer 1–3, 68167 Mannheim, Germany; Jost.Jonas@augen.ma.uni-heidelberg.de

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Late-stage inclusion body conjunctivitis: trachoma?

Chlamydia trachomatis is the most commonly found infectious agent in chronic conjunctivitis. Diagnosis is often delayed due to the oligosymptomatic course of the disease and the difficulty of obtaining enough infected cells in the conjunctival swab to confirm the diagnosis. These difficulties often lead to a prolonged course of the infection and possibly to scarring when infection occurs owing to chronic inflammation. We report on a 30-year-old Swiss patient having trachomatous entropion with trichiasis of the upper eyelid requiring corrective surgery, but with no history of travelling to trachoma endemic areas.

Figure 1 Cicatricial changes of the palpebral conjunctiva. Informed consent was obtained for publication of this figure.

Case report

A 34-year-old patient was seen in consultation for trichiasis involving both upper eyelids causing keratopathy. According to the patient, the development of entropion was preceded by a 1-year episode of recurrent conjunctivitis. Except for hay fever there was no other relevant ophthalmic or medical history, especially no history of possible infection elsewhere. On eversion of the eyelid, there were cicatricial changes and inflammation involving the entire palpebral conjunctiva with marked papillae (fig 1). Infection with *C trachomatis* was suspected and confirmed by a polymerase chain reaction. The patient was treated with a 2×100 mg doxycycline regimen for 2 weeks, with topical ofloxacin ointment treatment for 4 weeks. Owing to advanced entropion and trichiasis, a lid margin rotation (Trabut) procedure was performed (fig 2). The post-operative course was uneventful, with successful anatomical correction of the upper eyelids.

Comment

Chlamydiae are obligate intracellular Gram-negative eubacteria that lack cytochromes and the ability to synthesise their own ATP. *C trachomatis* has been a source of human disease especially in the eye and urogenital system for millennia, thriving in poor, crowded and unsanitary conditions. It has been known as a blinding disease for more than 3500 years.¹ Trachoma was rampant throughout Europe and North America in the 1800s, but disappeared in England before the Second World War. In the 1950s, trachoma also disappeared in Italy and the Soviet Union. This was mainly due to improved hygiene standards.

In the developing world, trachoma is still the leading infectious cause of blindness. The World Health Organisation estimates that 70

Figure 2 Lid margin rotation procedure. Informed consent was obtained for publication of this figure.

million people are infected with trachoma. Five million people are infected with its late stages and two million are blind because of it.

The trachoma biovar of *C. trachomatis* can be subdivided into 15 serovars which are designated by the letters A–K based on the polymorphism in the sequence of the major outer membrane protein.^{2,3} Serovars A–C can usually be isolated from patients with clinical trachoma in regions where trachoma is endemic.

Oculogenital infection caused by serovars D–K is common in developed countries and leads to inclusion conjunctivitis, also called paratrachoma. Up to 90% have concurrent urogenital infections.

Although the different groups of serovars display unique tissue tropisms, they are not tissue selective, and serovars D–F, J and K have all been isolated from conjunctival swabs taken from individuals with typical clinical signs of active trachoma.^{4–7} However, all these reports are from areas where trachoma is endemic.

Therefore, the traditional distinction between ocular and genital strains may have to be reconsidered.

Clinically, it is difficult to diagnose the beginning of trachoma or inclusion body conjunctivitis, and it is only by laboratory testing that the diagnosis can be confirmed. Among the available assays, nucleic acid amplification tests have proven superior in detecting, quantifying and genotyping *C. trachomatis*.⁸ Because chlamydia is an intracellular organism, the correct swab technique is very important to obtain a positive test result. This includes firm rubbing with the swab in the fornix, which is unpleasant for the patient. Because chlamydial infection is frequently oligosymptomatic and routine laboratory screening is seldom not readily available for the ophthalmologist practitioner, the diagnosis of the disease is often delayed or even missed. Additionally, repeated reinfection occurs when the sexual partner is not treated as well.

These difficulties lead to a prolonged course of the infection and possibly to scarring due to chronic inflammation and repeated reinfections. Unfortunately, we were not able to differentiate the serovar of the *C. trachomatis* isolate in our case, as serotyping is not routinely performed in Switzerland. However, the advanced clinical findings leading to entropion necessitating surgery emphasise not only the need for early diagnosis and treatment of this disease but also the need for thorough clinical examination including eversion of the upper lid and inspection of the tarsal conjunctiva in any patient with conjunctivitis.

Martin Zinkernagel, Elena Catalano, Dagmar Ammann-Rauch, Martin Zinkernagel, Elena Catalano, Dagmar Ammann-Rauch
Department of Ophthalmology, Cantonal Hospital St Gallen, St Gallen, Switzerland

Correspondence to: Dr M Zinkernagel, Department of Ophthalmology, Cantonal Hospital St Gallen, 9007 St Gallen, Switzerland; martin.zinkernagel@kssg.ch

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CORRECTIONS

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Several errors occurred in the paper titled, Linezolid-induced optic neuropathy: a mitochondrial disorder? (*Br J Ophthalmol* 2007;**31**:111–5). A fully corrected pdf is available online at <http://bjo.bmj.com/>. The journal apologises for these errors.

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In the paper titled, Plasma apolipoproteins and risk for age-related maculopathy (*Br J Ophthalmol* 2006;**90**:1028–33) previous literature has been incorrectly cited within the text.

In second paragraph of the Discussion section (page 1030, right column), the authors refer to "large associations between neovascularisation and plasma cholesterol (OR = 4.1) in the Eye Disease Case Control Study (EDCCS)⁴⁸ and neovascularisation and apoB (OR = 9.2) in the Beaver Dam Eye Study (BDES).⁴⁹" Further on in the same paragraph, we state, "It is not clear why the EDCCS and BDES obtained the strong effects they did".

In both cases these results were incorrectly attributed to the BDES. These two sentences should have instead cited Reference 79, which pertains to data from the NHANES III study. Citations to References 49 (BDES) and 79 (NHANES II) are correct in the 3 tables.