

GLAUCOMATOCYCLITIC CRISIS IN EDO STATE, NIGERIA: REPORT OF TWO CASES.

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ABSTRACT

Objective: To report two classical cases of glaucomatocyclitic crisis in tertiary Health Centres in Edo State, Nigeria

Method: Two cases of glaucomatocyclitic crisis seen at Irrua Specialist Teaching Hospital, Irrua, and University of Benin Teaching Hospital, Benin City, both in Edo State, Nigeria are reported. Diagnosis was based on typical history and findings on examination.

Results: The first was a female who presented in December 2002 at Irrua Specialist Teaching Hospital and the second was a male who presented at the University of Benin Teaching Hospital, in March 2004. Both presented with blurring of vision in the affected eye and seeing halos around light. The intraocular pressures in the affected eyes were markedly elevated, there were fine keratic precipitates in the corneal endothelium of the involved eyes and the optic discs and visual fields were normal. Repeated episodes of the symptoms confirmed the diagnosis of glaucomatocyclitic crisis.

Conclusion: This report shows that typical cases of glaucomatocyclitic crisis occur in Nigerians but require a high index of suspicion for the diagnosis.

Key words: Glaucomatocyclitic crisis, Posner-Schlossman syndrome, Nigeria. (*Accepted 7 June 2007*)

Glaucomatocyclitic crisis is a condition with self-limited recurrent episodes of markedly elevated intraocular pressure (IOP) with mild idiopathic anterior chamber inflammation. It is most often classified as secondary inflammatory glaucoma. In 1948, Posner and Schlossman first recognized glaucomatocyclitic crisis and described the features of this syndrome.¹ For this reason, the entity is often termed Posner-Schlossman syndrome (PSS).

It is characterized by recurrent episodes of mild cyclitis, uniocular involvement and duration of attack varying from a few hours to several weeks. There is also a rise in intraocular pressure (IOP) out of proportion to the severity of the uveitis (usually between 40-60mmHg and sometimes to over 80mmHg). There are resultant halos, blurring of vision and ocular discomfort, but no pain. Congestion is almost always absent and the eyes remain white. The angles of the anterior chamber are open, but there is corneal oedema with fine keratic precipitates, aqueous flare, heterochromia with

anisocoria and a large pupil in the affected eye. The visual fields and optic discs are normal.

Almost exclusively, this condition affects individuals aged 20-50years. However, there has been a reported case in a 13year old.² Both eyes may be involved at different times, but very rarely simultaneously.³ Between episodes of attacks, the intraocular pressure, out flow facility and all provocative tests remain normal.

It is a relatively uncommon disorder and most reports are isolated case reports or case series.^{2,4-7}

The pathophysiology of this condition shows episodic changes in the trabecular meshwork leading to impairment of outflow facility with resultant elevation in intra ocular pressure.^{8,9} These changes are accompanied by mild intraocular inflammation.

In the acute phase of Posner-Shlossman syndrome, optic nerve head parameters and retinal flow rates were altered; however, all returned to normal without any permanent damage after resolution of the elevated intraocular pressure. Electro retinogram studies in the acute phase demonstrate a selective reduction in the s-cone b-wave.¹⁰ The

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aetiology of glaucomatocyclitic crisis has remained elusive. Several factors have been postulated as contributors to its development, which include the following: abnormal vascular process, autonomic process, allergic condition, variation of developmental glaucoma, cytomegalovirus and herpes simplex virus.¹¹⁻¹⁷

Glaucomatocyclitic crisis is a relatively rare disorder. Indeed the first documented case report in Nigeria was only in 2004.⁷ It is possible that earlier cases were missed.

Two cases of glaucomatocyclitic crisis are presented in this study; the first, a 30-year old lady and the second, a 32-year old male. The objective of this report is to show that cases of glaucomatocyclitic crisis, though rare, do occur in Nigeria but require a high index of suspicion for the diagnosis.

CASE 1

EO was a 30-year old lady who presented in September 2001 to the eye clinic of Irrua Specialist Teaching Hospital, Irrua with complaints of poor vision and ocular aches of six months duration. She has been using prescribed glasses for more than a year prior to presentation but it got broken in March 2001. Examination revealed a visual acuity of 6/12+2 in the right eye and 6/12+2 in the left eye. The intraocular pressure in the right eye was 13mmHg and 14mmHg in the left eye. Fundoscopy showed normal optic discs in both eyes. Patient was refracted and myopic astigmatic glasses were prescribed.

The patient presented again in December 2002 with complaints of seeing halos around lights and ocular discomfort in the left eye. Her unaided visual acuity on examination was 6/18 in the right eye and 6/12 in the left eye. Aided visual acuity (with her glasses) was 6/6 in both eyes. Intraocular pressure was 14mmHg in the right eye and 39mmHg in the left eye. There was very mild conjunctival injection in the left eye and slit lamp examination showed fine keratic precipitates in the corneal endothelium of the left eye. Fundoscopy revealed normal discs in both eyes. An initial diagnosis of anterior uveitis with secondary glaucoma was made and the patient was placed on Gutt dexamethasone /antibiotic (maxitrol) drops four times daily, Gutt timolol maleate 0.5% twice daily and gutt tropicamide (mydriacyl) 1% two times daily in the left eye. On follow up visit after a week, these symptoms had disappeared. The intraocular pressure in the right eye was 12mmHg and 15mmHg in the left eye.

In January 2003, the patient had another episode of similar attack involving the same left eye with mild

blurring of vision and seeing halos around lights. Fine keratic precipitates were seen in the corneal endothelium. Unaided visual acuity was 6/18 in the right eye and 6/24 in the left eye. With her glasses, her visual acuity was 6/6 in the right eye and 6/6-3 in the left eye. Intraocular pressure was 12mmHg in the right eye and 40mmHg in the left eye. The optic discs in both eyes were normal on fundoscopy. Gonioscopy revealed open angles and automated visual field analysis showed full fields in both eyes. After ruling out other common secondary causes of uveitis and glaucoma such as trauma, toxoplasmosis, tuberculosis, onchocerciasis, acquired immune deficiency syndrome, pseudoexfoliation syndrome and pigment dispersion syndrome, a diagnosis of Posner-Schlossman syndrome was made. Patient was again placed on gutt dexamethasone /antibiotic (maxitrol) drops four times daily, gutt timolol maleate 0.5% twice daily and gutt tropicamide (mydriacyl) 1% 2 times daily in the left eye with resolution of symptoms in a few days. On follow up visit after a week, the symptoms had again disappeared. And the intraocular pressure was 14mmHg in right eye and 16mmHg in left eye.

Patient had recurrence of symptoms in same eye in November 2003. This time, the intraocular pressure was 44mmHg in the involved left eye and 10mmHg in the uninvolved right eye. Aided visual acuity was 6/6 in the right eye and 6/6-2 in the left eye. The same treatment modality was repeated as in previous visits, but with the addition of tabs acetazolamide 250mg three times daily. A week later, on follow up, the left eye was quiet and the intraocular pressure was now 12mmHg; that of the right eye was 11mmHg. Subsequently, the patient remained stable till June 2004 when there was recurrence of symptoms in the same left eye. The treatment modality as in the last attack was again repeated with resolution of symptoms.

After the attack of June 2004, the patient has remained stable. Her glasses were changed in May 2005. She was again seen in December 2005 and was stable. She is still on 6 monthly follow up till date and has remained stable.

CASE 2

Dr. AI was a 32 years old male doctor at the University of Benin Teaching Hospital, Benin City, who presented to the Eye Clinic of the hospital in March 2004 with a history of slight blurring of vision and seeing halos around light in the left eye. He had experienced a similar episode about a year earlier which resolved spontaneously in about a week.

His visual acuity was 6/6 RE and 6/9 LE. The pupil in the left eye was dilated and not reactive to light but the pupil in the right eye was round, central and reactive to light. The optic discs were normal in both eyes. Slit lamp examination revealed slight corneal oedema in the left eye with few scattered fine keratic precipitates and faint flare but no cells. The intraocular pressures were 18mmHg right eye and 58mmHg, left eye. Gonioscopy showed that the angles were open and normal with no pigment deposits. Visual field analysis showed normal fields in both eyes. He was started on gutt timolol 0.5% b.d. LE, gutt latanoprost (xalatan) nocte LE, tabs acetazolamide 250mg 8hrly and gutt fluorometholone tds LE. There was a rapid drop in intraocular pressure in the left eye to 17mmHg within 2 days with resolution of symptoms. Treatment was discontinued in 2 weeks. The intraocular pressures remained below 20mmHg by applanation tonometer for 10 months after discontinuation of therapy. Unfortunately, the patient was lost to follow-up after completion of his residency training programme.

The initial diagnosis by the senior registrar was glaucoma secondary to uveitis. However when the clinical features were reviewed with the consultant, it was agreed that the elevated intraocular pressure was out of proportion to the severity of uveitis. The more common causes of uveitis and secondary glaucoma such as trauma, toxoplasmosis, tuberculosis, onchocerciasis, acquired immune deficiency syndrome, pseudoexfoliation syndrome and pigment dispersion syndrome were eliminated by the history, physical examination and investigations. It was also assumed that the similar experience he had a year earlier may have been an attack of glaucomatocyclitic crisis. A diagnosis of glaucomatocyclitic crisis was then made.

DISCUSSION

Glaucomatocyclitic crisis is a relatively rare self limiting benign condition. This could be the reason why the first documented case of Glaucomatocyclitic crisis in Nigeria was reported only recently from Irrua Specialist Teaching Hospital.⁷ In that case, elevation in intraocular pressure occurred in both eyes with marked glaucomatous optic atrophy and visual field defects. The intraocular pressure elevation was also prolonged requiring trabeculectomy. Although evidence exists that glaucomatocyclitic crisis could be associated with primary open-angle glaucoma (POAG),^{18,19} the cases seen in this report were more typical of glaucomatocyclitic crisis. In the first case, typical symptoms occurred in the

typical age group with unilateral involvement and recurrent episodes, each episode lasting about a week. There was no significant glaucomatous optic atrophy or visual field loss. The rise in intraocular pressure was out of tune with the uveitis. In between attacks, the intraocular pressures were low without medication.

In the second case, symptoms were also typical, occurred unilaterally in the typical age group and was most probably recurrent. The intraocular pressure was markedly elevated with slight corneal oedema and very mild signs of uveitis. After resolution of the crisis, intraocular pressure remained normal without medication.

In these two reported cases, stress could have contributed to the recurrent attacks of glaucomatocyclitic crisis. At the time the first case was having recurrent attacks, she was an applicant and a young unemployed graduate. Attacks suddenly stopped after she got employed. Also in the second case, the resident doctor was preparing for the final fellowship examinations in Pathology at the time of attack. Knox²⁰ has documented a relationship between peptic ulcer, other gastro intestinal disorder, allergy stress and glaucomatocyclitic crisis. The age at presentation of both cases of 30 and 32 years fall within the age group which is usually 20-60 years of age.²

In both cases, the diagnosis of glaucomatocyclitic crisis was missed initially. It took further re-evaluations before the correct diagnosis could be made. This may not be very possible in our busy out-patient clinics unless there is a high index of suspicion. Such cases, though occurring infrequently, could easily be misdiagnosed as glaucoma secondary to acute anterior uveitis. However, the marked elevation of intraocular pressure will be out of tune with the very mild signs of anterior uveitis. This should raise a suspicion of glaucomatocyclitic crisis.

In conclusion diagnosis of glaucomatocyclitic crisis, being a relatively rare condition could easily be missed. This may explain why it is only recently that the first documented case in a Nigerian was reported.⁷ This report shows that cases of glaucomatocyclitic crisis do occur in Nigerians but a high index of suspicion is required for the diagnosis of this condition as it may easily be dismissed as glaucoma secondary to uveitis.

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