LOW TENSION IN ACUTE GLAUCOMA*

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It is universally accepted that the cardinal sign of primary closed-angle glaucoma is a raised intra-ocular pressure. It might even be considered that it would be a contradiction in terms to make a diagnosis of acute primary closed-angle glaucoma in a soft eye. It is, however, the purpose of this article to show that, in some rare cases, such a diagnosis can be made in spite of this finding; for, apart from the low tension, most of the typical signs and symptoms of acute glaucoma are present. These include pain and blurring of vision, followed by signs of ocular congestion—ciliary and conjunctival injection, haziness of the cornea, and dilatation and oedema of the pupil. But whereas the ocular tension in a classical case of acute glaucoma may rise to about 50 to 60 mm. Hg (Schiötz), in these exceptional cases the tension is normal (between 20 and 25 mm. Hg) or low.

Confirmation of the diagnosis can be obtained by investigations and provocative tests carried out after the acute phase has passed. A typical case is described in detail below and others exhibiting the same phenomenon are briefly mentioned. A hypothesis is put forward which may account for the clinical findings, and a name is suggested for this syndrome which, to my knowledge, has not before been fully described.

Case Report

A woman aged 80, who had been under treatment at home for bronchopneumonia, began to complain of frontal headaches, vague pains in the eyes, and increasing blurring of vision in the right eye. A more detailed history could not be obtained, owing to the patient's senility and poor intelligence. She attributed the onset of her symptoms to a fall on the stairs.

Clinical Findings.—The corrected visual acuity was 6/18 in the right eye and 6/6 in the left.

The right eye showed some conjunctival and ciliary hyperaemia. The cornea was oedematous and the pupil was semi-dilated and fixed. The anterior chamber was shallow and the iris almost bombé in type, but the optic disc was normal.

The left eye showed a shallow anterior chamber, but no other signs of glaucoma.

Both eyes showed some degree of iris atrophy, and there were fairly advanced bilateral nuclear cataracts.

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The intra-ocular pressure was low: 10 mm. in the right eye and 12 mm. in the left, using the 5.5 g. weight (Schiötz).

Diagnosis.—Acute glaucoma was diagnosed in spite of the low intra-ocular pressure which could not be attributed to dehydration, as there was nothing in the history or clinical findings to suggest this. Treatment with miotics was given and improvement was rapid; after only a few instillations of pilocarpine 2 per cent. the right eye started to whiten, the corneal oedema cleared, and the symptoms were gradually relieved.

Investigations.—When the oedema had cleared, the cornea was examined under the microscope. The extent of the iris atrophy was greater than was at first apparent, and in the right eye a remarkable picture was seen (Figs 1–3). Numerous filamentous processes from the iris were seen to extend into the anterior chamber, some near the pupillary margin reaching to the corneal endothelium.

FIG. 1.—Case 1, artist's drawing of right iris, showing early atrophic changes in lower temporal sector.

FIG. 2.—Drawing of optical section of this sector, showing atrophic elements in contact with cornea.

FIG. 3.—Gonioscopic view of the same region, also showing projecting elements. Unfortunately the numerous white peripheral anterior synechiae are not well shown.

Gonioscopy showed that these processes were very numerous in the angle, being ranged in a row between the last fold of the iris and the trabecular region. The angles were narrow, with only half of the trabecular region visible.

24-hour phasing of the intra-ocular pressure showed little variation in either eye.

Visual Fields.—These were difficult to record accurately due to the patient's poor rapport, but seemed normal to confrontation.

Tonography.—The rate of aqueous outflow in each eye was well above the minimum normal level, producing a coefficient of 0.3 in 4 minutes.

Provocative Tests.—These all failed to elicit any rise in tension:

DARK-ROOM TEST: There was no rise in tension in the right eye after one hour, and only a very insignificant rise in the left eye.

MYDRIATIC TEST: After a drop of 2 per cent. homatropine with cocaine had been instilled, the tension actually fell in the right eye from 14 to 12 mm. Hg (Schiötz), and it rose slightly, from 12 to 15 mm. Hg, in the left eye.

WATER-DRINKING TEST: This had to be omitted, as the patient was suffering from congestive heart failure, and it was not possible to subject her to a large fluid intake.

PRISCOL TEST: This was carried out as originally described by Leydhecker (1954) and more recently by Primrose (1958). The tension in each eye remained quite unchanged for 90 minutes after a subconjunctival injection of 4 mg. Priscol.

Clinical Course and Management.—The patient soon became symptom-free and the ocular tension settled at a level of 16 mm. Hg in each eye (using the 5.5 g. weight—Schiötz). Miotics were withheld for 2 weeks while the investigations were carried out, and the tension remained controlled. However, the patient was discharged on a prophylactic dose of pilocarpine 0.5 per cent. twice daily in each eye.

Discussion

The term "low-tension" glaucoma has been applied to cases of chronic simple glaucoma in which the resting intra-ocular pressure may be normal, but rises at certain periods of the day or in response to provocative tests. Here we are concerned with low tension in acute or closed-angle glaucoma. The fact that the provocative tests failed to precipitate a rise in tension might suggest that the cause of the original attack had been relieved, though possibly only temporarily. If the attack had been relieved this could not have been done by relieving the obstruction at the angle, because the angle was seen to be extensively blocked by atrophic iris filaments forming goniosynechiae; nor was the tension adversely affected by stopping miotics. Various causes for glaucoma in iridoschisis have been postulated (and these may also contribute to the raised ocular tension in cases of iris atrophy):

(1) Blocking of the trabecular meshwork by scattered pigment, after trauma (Loewenstein, Foster, and Sledge 1948). In the present case there were no abnormal amounts of scattered pigment, and though there was a history of trauma, this had not involved the eyes.

(2) Hypersecretion of aqueous caused by irritation of the ciliary processes (McCulloch, 1950). In this case the tensions were low rather than high.

(3) Dilatation of the pupil in an eye with a narrow angle (Carter, 1953). In this case neither the dark-room nor the mydriatic test produced a rise in tension, so that this was probably not the main cause of the glaucoma.

(4) Angle-closure by contact between the abnormal iris and cornea, so that the periphery of the anterior chamber becomes separated from the rest of the compartment. The aqueous in the angle is absorbed through the trabeculae but is not replaced because of the block, and the resultant negative pressure in the angle causes the periphery of the iris to adhere completely round the angle. This mechanism was described by Posner (1953) and Duke-Elder (1940), but it is unlikely to have operated in this case as the fragmented iris would not have formed a very good partition between the central and peripheral parts of the angle.

(5) A further hypothesis is now put forward, which explains most of the features in this case. This is that the attack of acute glaucoma impaired the vitality of the ciliary body, so that aqueous production was thereafter reduced, producing a state of low tension and a negative response to provocative tests. A normal facility of aqueous outflow indicates later recovery of secretion.

Several other cases of low tension in acute glaucoma, but without such marked iris atrophy, have recently been seen. In all of these, however, although the presenting tension was low, a rise was elicited by provocative tests, either a dark room or mydriatic test, after the acute phase had settled.

The damaging effect of glaucoma on the iris producing atrophy is well known, and has been especially well described by Gorin and Posner (1957). It seems reasonable, therefore, that damage to the ciliary body may also be caused with impairment of its secretory capacity. This impairment may be almost complete, as in the case described above, or it may be incomplete as in several other cases mentioned above, in which the tension was normal but one or more provocative tests were positive.

The mechanism of damage is probably vascular, with occlusion of afferent vessels from the *circulus iridis major*. This may be caused by congestion of the ciliary body, which is precipitated by the factors causing primary closed-angle glaucoma:—anatomically shallow-angle, large lens, and the effort of accommodation which is needed in hypermetropes and early presbyopes (Sugar, 1941). As the *circulus iridis major* is situated in the anterior part of the ciliary body, it is only reasonable that the ciliary body should be damaged as well as the iris in episodes of acute glaucoma as both structures have the same blood supply.

Various observers have reported changes in the ciliary processes and ciliary body due to congestive glaucoma. von Fieandt (1949) reported significant changes in the appearance of the ciliary processes after iridectomy for acute glaucoma. The processes were lighter in colour and further apart than usual and in some cases they were atrophic. Trantas found that the ciliary processes in many cases of acute glaucoma were colourless, whereas they usually appear violet, and that there were often yellow atrophic spots in the ciliary body. In fact, Sugar (1941) suggested that, in cases in which the anatomical and physiological conditions predispose to glaucoma, an attack may be induced by a congested ciliary body, which pushes the root of the iris forward and completely closes a narrow angle. In the event of structural damage to the ciliary body in acute glaucoma being so clearly shown, it

TABLE
FOUR CASES OF "CILIARY SHOCK"

Case No.	Eye	Acute Attack + (Visual Acuity Corrected)	Initial Tension (mm. Hg Schiötz)	State of Angle of Anterior Chamber	Response to Miotics Only (for Acute Attack)	Dark-Room Test Rise in Tension (mm. Hg Schiötz)	Mydriatic Test Rise in Tension (mm. Hg Schiötz)	Treatment	Tension after 6 mths (Visual Acuity Corrected)
1	Right	(Subacute) (6/18)	10	Narrow with P.A.S.	Good	0	0	Maintained on pilocarpine	Normal (6/18)
	Left	(6/18)	12	Narrow with P.A.S.	—	4	3	Maintained on pilocarpine	Normal (6/18)
2	Right	(6/6)	18	Narrow but open	—	16	—	Drainage operation (Scheie)	Normal (6/6)
	Left	(H.M.) ⁺	22	75 per cent. closed	Good	10	—	Peripheral iridectomy	Normal (H.M.)
3	Right	(After L.E.) (6/24) ⁺	18	Narrow (50 per cent. open)	Poor	6	6	Drainage operation (Scheie)	Normal (6/18)
	Left	(H.M.) ⁺	15	Almost 100 per cent. closed	Poor	—	—	Drainage operation (Scheie)	Normal (6/18)
4	Right	(C.F.) ⁺	14	Narrow (50 per cent. open)	Good	10	—	Peripheral iridectomy	Normal (6/24 ptly)
	Left	(6/6)	26	Narrow —open	—	8	—	Peripheral iridectomy	Normal (6/12)

seems reasonable to suppose that its functions may be impaired, and it is suggested that such a condition might be described as "ciliary shock".

The relevant details in four cases mentioned in this article have been summarized in the Table (above). Case 1, which has been described in detail, was that in which the lowest tensions of the series were recorded. It was also the only one which showed a noticeable degree of iris atrophy of the type caused by primary closed-angle glaucoma. This suggests that severe damage to the iris may be accompanied by quite extensive damage to the ciliary body or by ciliary shock. That the mechanism of these two conditions is primarily the same explains the presence of one or even both in acute glaucoma.

The response to miotics was so good that, considering the patient's age, it was judged quite satisfactory to maintain her on pilocarpine. In the other cases operative treatment was undertaken and the type of operation was chosen mainly according to the response to miotics and the state of the angle of the anterior chamber. Thus, each patient was given one drop of eserine 0.25 per cent. to the affected eye on admission. The dose was repeated after an hour and the condition assessed after one hour. In all except case three, there was relief of pain, good miosis, some clearing of the cornea, and lessening of vascular injection. The angle of the anterior chamber was narrow in

each, but it remained at least 25 to 50 per cent. open, and peripheral iridectomy was performed and found to control the condition quite satisfactorily (Table). However, it was considered necessary to perform a drainage operation (Scheie, 1959) for the second eye of Case 2, because of pathological cupping of the optic disc with early field loss, and also for both eyes of Case 3, because of the poor response to miotics alone, which necessitated the use of Diamox in the acute phase of each attack.

All these cases have been followed up for 6 months until the present time and in all the ocular tensions have been well controlled and vision maintained or even improved. In Case 2, the lack of visual improvement is due to the very long history of visual loss with consequent permanent optic damage.

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