CHRONIC OPEN-ANGLE GLAUCOMA SECONDARY TO RHEGMATOGENOUS RETINAL DETACHMENT

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IN RETINAL DETACHMENT SURGERY GLAUCOMA IS A COMMONLY ENCOUNTERED condition, which may occur preoperatively or postoperatively. Sometimes the two diseases appear to be unrelated and simply occur coincidentally in the same eye. In other patients there may be a possibility of a predisposition to both diseases which may be caused by a yet undetermined genetic factor. In still other patients surgical treatment of retinal detachment may induce postoperative angle-closure glaucoma. Conversely, in some instances, glaucoma therapy with strong miotics can initiate vitreoretinal changes which result in retinal detachment.[1] I have encountered a small number of patients with unilateral open-angle glaucoma and retinal detachment in whom the history and postoperative course indicate that the untreated retinal detachment preceded and actually caused the secondary open-angle glaucoma.

This paper describes 11 patients who incurred glaucoma as a result of untreated retinal detachment. Following surgical retinal reattachment the glaucoma resolved in all patients.

The following two histories exemplify the development and course of glaucoma occurring secondarily to retinal detachment .

CASE 1

A 28-year-old male presented a diagnostic problem of intractable unilateral glaucoma. He had an eight-month history of low-grade iridocyclitis in his right eye. Three months after the onset glaucoma developed in that eye, and the intraocular pressure ranged from 40–60 mm Hg Schiotz, despite extensive medical therapy with miotics, epinephrine, and Diamox®. Celestone® (12 tablets daily for six weeks) was initiated one month after the onset of glaucoma, and three months later Meticorten® (40 mg daily for two weeks) and two subconjunctival cortisone injections. These medications had no effect on the glaucoma or iridocyclitis, and intraocular pressure persisted in the 40–60 mm Hg range.

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The visual acuity was 20/30 in the right eye and 20/15 in the left eye. Intraocular pressure was 42 mm Hg Schiotz in the right eye and 18 mm Hg in the left.* The anterior chamber depth of the right eye was 3+, with 1+ flare and 2+ cells, but no KP. There were a few small posterior subcapsular lens opacities, and the posterior hyaloid was detached. The retina was shallowly detached nasally from the midperiphery to the ora serrata with three small retinal holes near the ora serrata between the 1:30 and 2:30 o'clock positions. The visual fields were contracted temporally to 30°. Gonioscopy indicated a 3+ deep angle without abnormality. Tonography on three different occasions gave a coefficient of outflow of 0.03, 0.07, and 0.05. The coefficient of outflow values in the left eye at these times were 0.26, 0.25, and 0.29.

The anterior segment, lens, vitreous, retina, visual field, and gonioscopy examinations were all normal in the left eye.

The diagnosis was retinal detachment of the right eye, rhegmatogenous, and low-grade uveitis with open-angle glaucoma, probably secondary to the retinal detachment.

At surgery a short segmental buckle to cover the retinal breaks was performed under lamellar scleral flaps. Diathermy was applied under the buckle, and the subretinal fluid was drained.

The intraocular pressure on the first postoperative day was 37 mm Hg Schiotz, with readings of 35 mm Hg on the second, 22 mm Hg on the third, 16 mm Hg on the fourth and twenty-first postoperative days, and 14 mm Hg one year later.

Three weeks postoperatively there were minimal flare and cells in the anterior chamber, and at three months the anterior chamber was clear. Three weeks postoperatively "C" values on tonography were 0.28 right eye and 0.27 left eye. One year after surgery "C" values were 0.26 right eye and 0.24 left eye.

CASE 2

A 7-year-old male had been struck in the left eye with a stick four years prior to the present examination. There were no ensuing ocular complaints until one year ago when, at the age of six years, decreased vision, low-grade iridocyclitis, and glaucoma were noted. The full range of medical therapy failed to control either the iridocyclitis or the glaucoma.

When the patient was examined at the age of seven years, his visual acuity was 20/20 in the right eye and light perception in the left. The intraocular pressure was 16 mm Hg Schiotz, right eye, and 51 mm Hg Schiotz, left eye. The anterior segment, lens, vitreous, fundus, and anterior chamber angle were normal in the right eye.

The anterior chamber of the left eye was 3+ deep, with 2+ flare and 2+ cells. The lens was subluxated nasally and superiorly. The retina was totally detached with a small dialysis inferotemporally between the five and six

*Tonometry and tonography corrected by graph method for abnormal scleral rigidity coefficient in all patients reported in this paper.[2]

o'clock positions. Numerous tiny white spots, which were scattered throughout the retina, as well as a moderate sized intraretinal macrocyst inferotemporally, indicated long-standing retinal detachment. Gonioscopy was normal with a 3+ deep open angle. Tonography "C" values were 0.30 in the right eye and 0.02 in the left.

The diagnosis was posttraumatic rhegmatogenous retinal detachment in the left eye, with iridocyclitis and open-angle glaucoma, probably secondary to retinal detachment.

Therapy consisted of a scleral buckle, performed under lamellar scleral flaps between the two and seven o'clock positions, utilizing an encircling polyethylene tube, diathermy, and drainage of subretinal fluid.

On the fifth postoperative day the intraocular pressure was 20 mm Hg in the right eye and 14 mm Hg in the left. Three weeks postoperatively it was 14 mm Hg in the right eye and 10 mm Hg in the left. Three weeks postoperatively tonography "C" values were 0.25 in the right eye and 0.18 in the left. Twelve years postoperatively visual acuity was 20/20 in the right eye and 20/400 in the left. Intraocular pressure readings were 14 in the right eye and 17 in the left. The coefficient of outflow values were 0.28 in the right eye and 0.24 in the left.

The 11 patients in the series are summarized in Tables 1 and 2.

DISCUSSION

Studies of the intraocular pressure in eyes with retinal detachment have indicated that approximately 60 to 65 per cent are hypotensive, 30 per cent normotensive, and 5 to 10 per cent hypertensive.[3] In studies using intravenous fluorescein Dobbie[4] showed that hypotension in an eye with retinal detachment is caused by decreased aqueous secretion, rather than by an increased retrograde outflow of aqueous through the retinal break. Becker[5] confirmed Dobbie's observation by tonography studies of a group of retinal detachment eyes, in which a common finding was a decreased "F" value, sometimes associated with a decreased "C" value. Therefore normotension is manifested in two circumstances: (1) normal "F" and "C" values, and (2) correspondingly decreased "F" and "C" values.

Before a diagnosis of open-angle glaucoma secondary to retinal detachment is made, two other groups should be differentiated. The first and most common group is that of retinal detachment occurring in an eye with preexisting chronic simple glaucoma. The clues to this diagnosis are the bilaterality of the glaucoma, the presence of typical glaucomatous nerve fiber bundle visual field defects, and the presence of glaucomatous cupping of the optic disks.

In another group glaucoma is secondary to subacute or chronic uveitis. Retinal detachment is not primary in this group, but rather develops after these disease processes, as an independent disease. The retinal detachment may be non-rhegmatogenous and directly caused by the uveitis. It may also be rhegmatogenous and unrelated to the uveitis, although in some patients of this group the uveitis may play a marginal role in the rhegmatogenous retinal detachment by inducing vitreous degeneration. If uveitis is the primary process (later followed by glaucoma and retinal detachment), keratic precipitates, iris atrophy, or visible foci of active uveitis are likely to be present.

Occasionally the time sequence of uveitis, glaucoma, and retinal detachment is not clear, and diagnostic conclusions may have to await the findings in the postoperative course. If uveitis subsides promptly after retinal reattachment surgery, and the coefficient of outflow and intraocular pressure readings normalize within a few weeks after retinal reattachment, the evidence would suggest that retinal detachment had been the prime etiologic factor in both the uveitis and the glaucoma. If, on the contrary, the glaucoma and abnormal aqueous dynamics persist, it is more likely that the glaucoma is either a chronic simple open-angle type or that it has occurred secondarily to a condition other than retinal detachment. In chronic simple glaucoma or glaucoma secondary to a cause other than retinal detachment there may be a temporary decrease in the "F" value and a lowering of intraocular pressure following surgical retinal reattachment, but the "C" value does not return to normal.

In most patients the intraocular pressure measured by the Schiotz tonometer is decreased following retinal reattachment surgery, and remains so for several months. In a minority of patients this decrease persists for several years. In this series of 11 patients, the hypotony as measured by Schiotz tonometry has been found to be the result of decreased "F", as calculated from tonography values, associated with a variable decrease of scleral rigidity, as calculated by graph.[2] "F" usually returns to normal within a few months, but reduced scleral rigidity may persist much longer, thus giving a false low value for intraocular pressure readings by single-weight Schiotz tonometry. In this series the postoperative scleral rigidity coefficient, "E", varied from 0.010 to 0.020 (average normal is 0.0215).

Several other interesting points were noted in these 11 patients. The age range was younger than that of the usual group of retinal detachment patients, varying from 6 to 53 years of age. The fact that all but one of the patients in this small series were males is unexplained. A history of trauma directly to the eye was elicited in approximately half

TABLE 1

Case	Sex	Age	History of/or findings of direct eye injury	Duration of retinal detachment (estimated)	Retinal detachment distribution	Retinal break			Lens status
						Location	Size	No.	
1	M	28	None	8 months	RE: nasal half; midperiphery onward	Peripheral upper nasal	Small	3	Phakic
2	M	7	Yes; lens subluxation	1 yr	Total LE	Inf. temp. dialysis	1 hour	1	Phakic
3	M	6	Yes	>1 month	Total LE	Temp. dialysis	3 hours	1	Phakic
4	M	35	Yes	1 yr	RE: nasal half	Upper nasal equator	Small	1	Phakic
5	M	28	Yes	>1 month	RE: nasal half	Upper and lower nasal equator	Small	4	Phakic
6	M	50	No	6 months	RE: inferior half	Inferior equator	Small	4	Aphakic
7	M	53	Yes; choroid rupture and A.C. angle recession	1 wk	LE: upper nasal quadrant	Upper nasal equator	Small	1	Phakic
8	M	14	No	3 months	RE: inferior half	Inf. temp. dialysis	3 hours	1	Phakic
9	F	12	No	6 months	LE: inferior half	Inf. dialysis temp. and nasal	5 hours	1	Phakic
10	M	49	No	2 days	LE: superior half	Superior ora	Small	1	Phakic
11	M	48	No	6 months	Total LE	All four quadrants	Small	4	Aphakic

TABLE 2

Case	Intraocular pressure (mm Hg)*		"c" values*		"E" coefficient scleral rigidity		Type of surgery	Result	Visual acuity	
	Preop	Postop	Preop	Postop	Preop	Postop			Preop	Postop
1	R: 42	16	0.05	0.26	0.0220	0.0170	Segmental scleral	Cure	20/30	20/20
	L: 18	16	0.26	0.24	0.0250	0.0230	buckle, diathermy		20/15	20/15
2	R: 16	14	0.30	0.25	0.0230	0.0240	Encircling scleral	Cure	20/20	20/20
	L: 51	10	0.02	0.18	0.0210	0.0140	buckle, diathermy		L.P.	20/400
3	R: 48	14	0.03	0.23	0.0290	0.0200	Scleral imbrication,	Cure	L.P.	20/200
	L: 13	15	0.32	0.28	0.0310	0.0290	cryosurgery		20/20	20/20
4	R: 46	21	0.07	0.20	0.0270	0.0170	Segmental scleral	Cure	20/100	20/20
	L: 20	17	0.22	0.23	0.0320	0.0340	buckle, diathermy		20/20	20/20
5	R: 29	17	0.10	0.18	0.0240	0.0160	Segmental scleral	Cure	20/20	20/20
	L: 17	17	0.21	0.24	0.0200	0.0230	buckle, cryosurgery		20/15	20/15
6	R: 36	14	0.06	0.31	0.0190	0.0100	Encircling scleral	Cure	C.F. — 1″	20/80
	L: 14	17	0.34	0.25	0.0220	0.0230	buckle, cryosurgery		20/100	20/100
7	R: 25	26	0.16	0.17	0.0290	0.0290	Encircling scleral	Cure	20/25	20/25
	L: 55	12	0.03	0.20	0.0270	0.0130	buckle, cryosurgery		20/70	20/70
8	R: 29	16	0.12	0.23	0.0190	0.0160	Scleral imbrication,	Cure	20/60	20/60
	L: 15	14	0.36	0.34	0.0200	0.0210	cryosurgery		20/25	20/25
9	R: 21	21	0.18	0.20	0.0180	0.0190	Encircling scleral	Cure	20/80	20/80
	L: 47	10	0.04	0.26	0.0190	0.0120	buckle, diathermy		20/25	20/25
10	R: 21	19	0.20	0.22	0.0290	0.0270	Segmental scleral	Cure	20/20	20/20
	L: 48	12	0.05	0.28	0.0250	0.0190	buckle, diathermy		C.F.	20/200
11	R: 16	15	0.27	0.29	0.0200	0.0270	Encircling scleral	Cure	20/25	20/25
	L: 39	21	0.08	0.19	0.0230	0.0120	buckle, cryosurgery		C.F.	20/200

*Intraocular pressure and "c" values are corrected for altered scleral rigidity.[2]

of these patients, whereas in the author's over-all retinal detachment series no more than 10 per cent give a history of direct trauma to the eye. The percentage of aphakia in this series differed only slightly from that in the usual distribution of retinal detachment patients, namely 20 per cent.

Linner[3] noted that hypotony in long-standing retinal detachments tends to increase with time. Retinal detachment in seven patients in this series was considered to be long-standing on the bases of either history or ophthalmoscopic findings, such as friction lines, intraretinal white spots, or retinal macrocyst formation. Dobbie[4] found that the larger the area of the retinal detachment, the greater the decrease in intraocular pressure. In this series of glaucoma eyes the extent of detachment did not seem to be related to the intraocular pressure. The distribution of the retinal detachment was predominantly inferior, nasal, or total. None of the patients showed an isolated upper temporal detachment. The significance of this finding is unclear.

As for the type and distribution of retinal breaks, 4 of the 11 patients showed an inferior temporal dialysis, with all but one of the remainder showing nasal breaks. The number and size of the breaks did not differ significantly from the usual incidence expected in any group of retinal detachment patients. In the affected eyes intraocular pressure ranged from 29 to 55 mm Hg, with the other eye being normotensive. (In one patient the pressure and "c" values were borderline in the fellow eye.)

Although corticosteroids were sometimes given for the concomitant uveitis, they cannot be implicated as the cause of glaucoma, since it existed before corticosteroid therapy. It is significant that this medication did not relieve either the glaucoma or the uveitis. Medical control in general was ineffective in controlling the glaucoma.

Postoperatively scleral rigidity was lowered and, as mentioned earlier, this decrease persisted for several months and sometimes years. This was especially true in the eyes with encircling scleral buckles, in which the ocular deformity was likely to be permanent, with a correspondingly permanent decrease in scleral rigidity. To evaluate accurately the intraocular pressure dynamics it is necessary to correct for altered scleral rigidity, either by paired weights on Schiotz readings or, preferably, by applanation and Schiotz comparisons.

All 11 retinal detachments in this series were cured by reattachment surgery, with resolution of the iridocyclitis and the glaucoma. The intraocular pressure rapidly returned to normal within a few days. The coefficient of aqueous outflow, "c", returned to normal within three weeks and remained normal for the duration of follow-up in all patients.

Of the 11 reattachment procedures, 5 were encircling scleral buckles, 4 were segmental scleral buckles, and 2 were scleral imbrications. Five patients were treated by diathermy and 6 by cryosurgery.

Four patients had a prolonged delay before the retinal detachment was discovered and proper treatment instituted. During this delay there was extensive and ineffectual treatment for both uveitis and glaucoma. Several factors contributed to this delay in making an accurate evaluation of the patients' status. Naturally complete ophthalmoscopic evaluation can be made only through a dilated pupil and fairly clear media. If anterior uveitis is prominent and the media hazy, it may be difficult to visualize the retina, a phenomenon which discourages thorough evaluation. Once miotic therapy is started for the glaucoma, the ophthalmologist's orientation may be so channeled that ophthalmoscopic examination subsequent to this diagnosis is focused almost entirely on the optic disk. Thus the retinal detachment, especially if it is confined to the periphery, is not seen. This experience makes us stress the dictum that any patient with unilateral glaucoma should have the benefit of complete mydriatic ophthalmoscopic examination as well as visual field study to evaluate fully the problem.

Visual field examination can be another pitfall. Once glaucoma is diagnosed, it is common for the ophthalmologist to confine his examination of the visual field to the central 30 degrees on the tangent screen. Thus any detachment which does not invade the retina within 30° of the fixation point will not be reflected in visual field loss on the tangent screen. A small contraction in the field in one area may be ignored or misinterpreted as glaucoma by the casual perimetrist. It cannot be overly stressed that, if the visual field defect is not of the typical nerve fiber bundle type, the suspicions of the ophthalmologist should be aroused. It is imperative to perform a full visual field examination extending to the most peripheral limits as tested on the Goldmann or Ferree-Rand perimeter. It may be noted that even a full visual field on such an instrument does not always exclude a peripheral retinal detachment. Since the superior and nasal visual field limits are only to 50–60°, or approximately the retinal equator, if the retinal detachment were confined to the area between the equator and the ora serrata inferiorly or temporally it would not result in visual field constriction even on these instruments.

A common teaching is that retinal detachment with associated glaucoma is suggestive of intraocular tumor. This probably relates to the pathologist's finding that, in some eyes which have glaucoma and in which the retina cannot be visualized, enucleation and pathologic ex-

amination subsequently reveal an intraocular melanoma. This does occur, but the incidence of this condition in the pathologist's laboratory leads to erroneous conclusions about the incidence of this problem in clinical practice with tumors and retinal detachment. In reviewing the charts of retinal surgery patients we found 35 intraocular tumors associated with retinal detachment in the total series. In 18 of these patients the intraocular pressure in the affected eye was normal, and in 17 it was hypotonic. In no instance was the intraocular pressure elevated in this group. The tumors ranged in size from 3 to 12 disk diameters, all had associated retinal detachment, and all were histologically identified as malignant tumors. Therefore I would like to stress that elevated intraocular pressure associated with retinal detachment is not necessarily a positive sign of an intraocular tumor. And, conversely, in the presence of retinal detachment with no visible break a concomitant normotensive or hypotensive eye does not exclude the possibility of tumor.

In managing the patients in this series it was considered prudent to limit preoperative medical therapy for elevated intraocular pressure to Diamox and epinephrine and thus to avoid using miotics even if the pressure could not be reduced to normal limits. The type of retinal reattachment surgery was selected on the basis of retinal and vitreous findings and was totally independent of the concomitant presence of glaucoma. Following reattachment surgery, intraocular pressure returned rapidly to normal, and no other therapy was needed for glaucoma.

The remarkable finding in this series was that, even when the glaucoma had been present for a prolonged time (as much as a year or more), reattachment surgery resulted in normalization of intraocular pressure within a few days.

Although the mechanism of secondary glaucoma is not fully understood, it has been demonstrated clearly that the presence of retinal detachment frequently results in a decrease of aqueous secretion ("F") and a decrease in the coefficient of aqueous outflow ("C").[5]

In most patients the decrease in "F" is greater than that in "C", so that frequently the result is hypotony. In approximately one-third of patients there is either no decrease in "F", or the decrease is approximately equalled by a corresponding decrease in "C", so that the intraocular pressure is normal. In the patients reported in this series the reduction of "C" was greater than that of "F", resulting in secondary glaucoma.

These cases would suggest a mechanism whereby retinal detachment induces cyclitis, varying in degree, although usually slight. This is reflected in the frequent aqueous flare, and decreased secretion of

aqueous. It would seem likely that the cyclitis is accompanied by a mild inflammation in the anterior chamber trabecular region causing some degree of blockage of outflow facility as measured by "c" on tonography. The exact site of the change in the trabecular region and the reason for its lack of beneficial response to antiinflammatory agents is not known. What is remarkable is the effect of reattachment surgery in rapidly reversing this change in the outflow mechanism.

SUMMARY

An eye which is normotensive can develop a rhegmatogenous retinal detachment with subsequent unilateral glaucoma in that eye. The glaucoma, which is associated with a decreased coefficient of outflow of aqueous from the anterior chamber, can persist for the duration of the retinal detachment. The signs and symptoms of glaucoma may be so prominent that the etiologic retinal detachment may be overlooked, sometimes for a prolonged time. Following surgical cure of the retinal detachment, the open-angle secondary glaucoma rapidly and permanently resolves.

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DISCUSSION

DR WILLIAM EVERETT. It has been a stimulating experience to study Dr Schwartz's paper and speculate along with him as to the possible mechanisms of chronic open-angle glaucoma with iridocyclitis which responds so remarkably to an anatomical reattachment of the retina, however minor the surgical procedure.

I believe those of us who deal in considerable numbers of retinal detachments see a somewhat larger group with iridocyclitis in which the tension may be elevated, normal, or hypotensive. The author has carefully and thoroughly

documented that group in which the intraocular tension is elevated. I believe it is fair to state that some degree of iridocyclitis is present in this group, and to speculate that the iridocyclitis may well be the cause of the pressure rise.

In assessing this group of patients the factors of young age, preponderance of males, and an unusually high incidence of nasal retinal breaks all seem to be in keeping with the high incidence of trauma of 50 per cent in the group. I would concur that one should anticipate no more than a 10 per cent incidence of trauma in the usual retinal detachment series. I believe trauma answers Dr Schwartz's question as to the nasal distribution of tears in this group, while asking another question, namely, what, if any, role does shock of trauma play in the causation of iridocyclitis and increased intraocular pressure in these cases of retinal detachment?

Certainly one does not usually see either of these two findings of iridocyclitis and increased ocular pressure in routine retinal detachment patients. Angle recession was found in one patient but was not seen in the remaining ten patients. I am inclined to interpret the decreased "c" factor on the basis of the iridocyclitis; however I would appreciate the opinion of one of our glaucoma specialists in this matter.

I have observed Dr Schwartz's work in retinal detachment for nearly twenty years and find here, as usual, a careful, thorough, conservative, yet provocative, approach to an interesting problem.

DR C. MCCULLOCH. I just wonder about this matter of trauma in relation to uveitis, glaucoma, and retinal detachment. I am reminded of a patient, a man aged 50. This gentleman came in with a retinal detachment, with a hole in the temporal periphery. There were flare and cells in the anterior chamber which were quite marked. The man was highly myopic. I took the ocular pressure and found it to be elevated. I did a retinal detachment procedure, which was successful. Rapidly the man's uveitis disappeared, and his glaucoma disappeared. Now, five years later, his retina is in good shape.

About three years ago he came in with exactly the same situation in the other eye. He had glaucoma, flare, cells, retinal detachment, and I found a hole in the retina. Again I repaired the retina and today both of his eyes are excellent.

I can't help but think that Dr Schwartz is pointing out to us a new syndrome, a new entity. I don't understand Dr Schwartz's syndrome, but I did not see in my patient any relationship to trauma. The disease was in both eyes. I think we should keep an open mind and watch for further patients like those described by Dr Schwartz.

DR MANSOUR F. ARMALY. I would like to make a comment on the definite improvement of ocular hypertension following the reattachment of the retina and possibly on some of the tonographic findings in this very interesting collection of cases.

It seems to me that Dr Schwartz leaves us with very little doubt that reattachment of the retina in these cases has definitely controlled ocular hypertension and that the corrected C value is consequently improved. One may speculate that the resulting improvement in uveitis may have been the mechanism of influencing ocular hypertension and outflow facility.

The problem of low ocular rigidity is a finding that can persist after a scleral buckle and is not easily corrected for in tonography. It is much easier to suspect abnormal rigidity by several weight measurements, but is not as simple to correct for it quantitatively so that one has the same comparative C value. I think that, having used the applanation tonometer, Dr Schwartz convincingly demonstrated an improvement in true ocular pressure and is not portraying a false effect due to a reduced ocular rigidity.