

ARTERIAL ANASTOMOSIS WITH SCHLEMM'S CANAL: A RARE CAUSE OF SECONDARY OPEN-ANGLE GLAUCOMA*

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INTRODUCTION

BLOOD IS ORDINARILY VISIBLE IN THE CANAL OF SCHLEMM BY GONIOSCOPY ONLY when episcleral venous pressure exceeds intraocular pressure (IOP). The three most common reasons are ocular hypotony, high episcleral venous pressure, and compression of the episcleral veins by the examiner's gonioscopes. In this paper, I will describe four patients in whom gonioscopic findings suggest a fourth reason for blood in the canal of Schlemm: an anastomosis of a deep limbal artery with the canal.

In each of these patients, blood was visible behind the trabecular meshwork, apparently within the canal of Schlemm, for a quadrant or more of the anterior chamber angle of one eye. When IOP was raised moderately with a suction cup or by external compression, the blood remained. Only when IOP approached systolic artery pressure was the blood expelled. Just before the blood finally disappeared, it could be seen to pulsate into and out of the eye with each beat of the heart.

CASE REPORTS

CASE 1

A 75-year-old white woman complained of a gradual loss of vision during the past year. She had worn glasses for myopia and astigmatism for most of her life, but had no history of ocular redness, pain, inflammation, or trauma. No relatives had glaucoma. Her health was good with the exception of one episode of congestive heart failure accompanying pneumonia 6 years earlier. Treatment with methyl-dopa, hydrodiuril, and furosemide provided relief of her pulmonary symptoms. Her blood pressure was normal.

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Visual acuity was 20/60 in both eyes with symmetrical spectacle corrections of $-7.00 + 4.00 \times 90$ degrees. Nuclear cataracts accounted for some of the visual loss. However, a 0.6 log unit relative afferent pupillary defect was present in the left eye. The visual field in the right eye was normal. In the left eye there was a nonspecific overall depression of sensitivity. IOPs were: right eye 19 mm Hg and left eye 28 mm Hg. The optic discs were asymmetrically cupped, with horizontal cup/disc ratios of right eye 0.4 and left eye 0.6.

The anterior chamber angles were wide open and normal except for what appeared to be blood in Schlemm's canal in the temporal angle of the left eye. The blood was present in the same place during a number of examinations, even when a Zeiss gonioscope which has no flange to compress the episcleral veins was used for the examination. Episcleral venous pressure, measured with a pressure chamber device,¹ was 9.6 mm Hg in both eyes in all veins measure. The episcleral veins appeared normal, without engorgement or hyperemia.

Firm finger pressure applied to the globe did not expel the blood from the angle. A suction cup connected to a suction ophthalmodynamometer² was applied to the paralimbal tissues. The IOP at any degree of suction deformation of the globe was measured directly with a Goldmann applanation tonometer at the Zeiss slit lamp, using the tonometer calibration bar as a counterweight when measuring pressures over 80 mm Hg. Only when IOP was raised to 91 mm Hg could the blood be expelled from the canal. Ophthalmic artery pressure, measured with the same instrument, was 116/40 mm Hg.

As the IOP approached the level at which the blood completely disappeared, the segment of blood became shorter and finally narrowed to a single pulsatile point that filled and emptied with each heart beat. This point was in the same location each time the experiment was repeated.

We thought these findings were consistent with an anastomosis between a deep limbal artery and Schlemm's canal. We prescribed timolol which lowered the left IOP to a level of 20 mm Hg to 22 mm Hg on six subsequent measurements during the next 2 years. The pressure in the untreated right eye varied from 14 mm Hg to 18 mm Hg. Blood continued to be present in the temporal angle of the left eye. The optic nerve did not deteriorate further.

CASE 2

A 42-year-old white woman was referred to us for an opinion regarding her need for glaucoma surgery. She had been found to have unilateral open-angle glaucoma in her right eye when she was 29 years old. However, she had noted somewhat prominent blood vessels on the surface of that eye even earlier, when she was 19 years old.

At the time of her original diagnosis, she and her mother were evaluated at the National Eye Institute. Her mother was found to have bilateral open-angle glaucoma with elevated episcleral venous pressure. The patient was found to have unilateral IOP elevation, blood in Schlemm's canal, and an episcleral venous pressure of 18 mm Hg to 21 mm Hg (measured with an applanating force transducer).³ No cause for the episcleral venous pressure elevation could be found in

either the patient or her mother. This family was subsequently reported as a rare example of familial idiopathic elevation of episcleral venous pressure.⁴

She was followed at the National Eye Institute for several years. Episcleral venous pressure, measured with a pressure-chamber device, varied from 10 mm Hg to 17 mm Hg depending on the vein selected. Outflow facility measured by tonography ranged from 0.08 to 0.14 $\mu\text{l}/\text{mm Hg}/\text{min}$ in the right eye and from 0.19 to 0.26 $\mu\text{l}/\text{mm Hg}/\text{min}$ in the left eye.

When we examined her, 13 years after the original diagnosis, her visual acuity remained 20/20 in both eyes. Her right eye had a superior arcuate scotoma and glaucomatous optic disc cupping. The left visual field and optic disc were normal. The episcleral veins were somewhat prominent in the superotemporal region of the right eye.

IOPs were, right eye 23 mm Hg and left eye 15 mm Hg. Around-the-clock measurements at three hourly intervals disclosed a variation in IOP of 23 mm Hg to 33 mm Hg in the right eye and 10 mm Hg to 16 mm Hg in the left eye.

Episcleral venous pressure in the right eye varied from 9 mm Hg to 12 mm Hg, depending upon the vein selected for measurement, with the highest readings in the prominent veins adjacent to the superotemporal limbus. The pressure in the same veins was lower a few millimeters behind the limbus. In the left eye, the episcleral venous pressure was 7 mm Hg in all veins measured. By gonioscopy with a flangeless goniolens, the anterior chamber angles were wide and symmetrical and differed in the two eyes only in that blood was visible behind the meshwork in the temporal, superior, and inferior portions of the right chamber angle. Hertel exophthalmometry measurements were right eye 9 mm and left eye 8 mm. The right orbit was found to be normal by echography: there was no evidence of soft tissue thickening or superior ophthalmic vein dilatation.

Para-limbal suction was used to raise the IOP in steps. The extent of the blood reflux gradually shortened, but the blood did not completely disappear until the IOP reached 56 mm Hg. It reappeared in an intermittent pulsatile fashion, synchronous with the heart beat, at an IOP of 52 mm Hg. Intravenous fluorescein angiography revealed no obvious abnormalities on the surface of the right globe.

We interpreted these findings as being consistent with a pathological anastomosis between a deep limbal artery and the canal of Schlemm in the right eye. We thought the dilated episcleral veins and borderline high episcleral venous pressures in the superotemporal quadrant were secondary to the large amount of flow away from the anastomosis. We recommended a trabeculectomy at the site of the presumed anastomosis, both for IOP control and to provide a biopsy for histologic confirmation. The patient returned to the care of the referring physician and subsequently had a filtering operation, but this was done in the superonasal quadrant away from the possible anastomosis. We have no further follow-up information.

CASE 3

This 80-year-old white man was blind in his right eye from an old hunting accident and complained of blurred vision in his left eye of 18 months' duration. The

physician who later referred him to us for consultation found cataract and "mild" glaucoma. He treated the patient with epinephrine for 16 months and then with timolol for 2 months. The patient was also being treated for atherosclerotic heart disease with digoxin and Peritrate. His sister was said to have glaucoma.

When we examined the patient, we found that he was unable to see light with his right eye. The visual acuity in his left eye was 20/25. The left visual field had a superior arcuate scotoma. Both eyes had pseudoexfoliation, mild lenticular nuclear sclerosis, and iridoschisis. Both optic discs had large cups, and the right disc was pale.

IOPs were right eye 11 mm Hg, left eye 15 mm Hg. The anterior chamber angles were wide open and symmetrical except for blood behind the trabecular meshwork in the inferior portion of the left chamber angle. This blood, which was visible with a Zeiss gonioscope as well as with lenses with flanges, was visible in the same location on four subsequent examinations. It did not fade with IOP elevation until the pressure reached approximately ophthalmic artery level. Like the previous two patients, with increasing levels of IOP the extent of the blood reflux diminished to a single pulsatile point before disappearing.

We thought that the cause of this patient's glaucoma was probably pseudoexfoliation, and that the IOP had been reduced to normal by the timolol treatment. The role of the possible anastomosis of an artery with Schlemm's canal in the left eye was unclear.

CASE 4

This 79-year-old white man was referred for evaluation of macular degeneration. He had been treated for open-angle glaucoma for the past 3 years with pilocarpine and timolol. He had no family history of glaucoma and no history of trauma, inflammation, or corticosteroid use. His health was otherwise good.

His visual acuity was right eye 20/500 and left eye 20/20. The right eye had disciform macular degeneration, and the left eye had extensive macular drusen. The optic discs appeared healthy with horizontal cup/disc ratios of 0.3.

IOPs were right eye 38 mm Hg and left eye 32 mm Hg. By gonioscopy, again using a flangeless lens, the anterior chamber angles were open and appeared normal except for an area in the temporal angle of the right eye in which blood was seen behind the trabecular meshwork. As in the previous three patients, the blood could not be expelled from the angle until IOP was raised with a suction ophthalmodynamometer to levels above diastolic ophthalmic artery pressure. As the IOP approached this level, the extent of blood reflux gradually shortened until it diminished to a single pulsatile point.

During the examination, the patient developed a corneal abrasion, and he refused further testing including the conversion of the suction readings to IOP measurements.

Our diagnosis was severe ocular hypertension in both eyes with the additional finding of an apparent anastomosis of an artery with Schlemm's canal in the eye with the higher IOP. We wondered if the anastomosis contributed to the asymmetry of IOPs.

DISCUSSION

The canal of Schlemm is ordinarily invisible by gonioscopy, because it is filled with clear aqueous and is hidden behind the translucent trabecular meshwork. However, the canal of Schlemm is a venous channel and is connected to blood-carrying episcleral veins by the collector channels and aqueous veins. In certain circumstances blood can reflux from the episcleral veins into the canal, and in an eye with open-angle glaucoma this may provide an important clue to the cause of glaucoma.

The reason the canal is usually filled with aqueous rather than blood is that the IOP is ordinarily about 5 mm Hg or 6 mm Hg higher than the pressure in the episcleral veins. Thus, there is a pressure gradient that maintains flow of aqueous from the eye and prevents blood in the episcleral veins from refluxing into the eye.

If this normal pressure gradient becomes reversed, blood will reflux into the canal. One reason for reversal of the normal pressure gradient is ocular hypotony, which may follow injury, inflammation, or cyclodialysis. Another reason is high episcleral venous pressure, which may result from Sturge-Weber syndrome,⁵ carotid-cavernous fistula,⁶ dural-cavernous fistula,^{7,8} orbital-arteriovenous fistula,⁹ and superior vena-caval obstruction.¹⁰ A third reason, the most common, is artifact: during the examination a flange of the goniolens occludes the episcleral veins in one quadrant, causing pressure to build up in those veins and blood reflux to occur in that quadrant of the angle. This artifact occurs only when a goniolens with a flange, such as the Allen-Thorpe or Goldmann lens, is used. It does not occur with the flangeless Zeiss lens. This examination artifact has been used to advantage to study blood reflux in various conditions.¹¹⁻¹⁶

Several findings in the present four cases indicate that the blood observed behind the trabecular meshwork was not due to one of these more common causes of reflux of blood from the episcleral veins into the canal of Schlemm. First, episcleral venous pressure was normal (≤ 12 mm Hg) in two of the cases, and in the other two, in which it was not measured, there was no engorgement of the episcleral veins to suggest high episcleral venous pressure. Second, the blood was visible through a flangeless goniolens and, thus, was not an artifact of the examination. Third, it was not easily expelled from the eye by moderate elevation of IOP, in contrast to conditions with elevated episcleral venous pressure such as dural-cavernous fistula or Sturge-Weber syndrome in which the blood is easily expelled from the eye with only a slight elevation of IOP. In fact, in the present cases it was only when the IOP was elevated above diastolic ophthalmic artery pressure that the blood was expelled. Most important-

ly, when the IOP was raised gradually, the blood narrowed down to a single pulsatile entry point before completely disappearing.

I believe that the findings in these four cases indicate an arterial source for the blood. Several anatomists have described small terminal branches of the anterior ciliary artery running adjacent to the canal of Schlemm for short distances.¹⁷⁻²³ Friedenwald,¹⁷ studying serial sections, described six to eight small afferent arterioles to Schlemm's canal, too small in diameter for red blood cells to pass through but large enough for plasma flow. He believed he could trace these vessels, which had no muscular walls, to larger arterial vessels. However, subsequent investigators, who used serial sections^{18,20} and injection molds^{12,21-23} to confirm Friedenwald's findings of arterioles near the canal, were unable to confirm his finding of anastomoses between these arterioles and the canal, and the consensus today is that no connections are present between arteries and the canal of Schlemm in normal primate eyes.

I cannot be certain if the blood visible behind the trabecular meshwork in these four patients was an artery running parallel to the canal or was the result of a pathologic anastomosis between an artery and the canal, but the blood's appearance favored the latter interpretation. The red stripe was broad and even in color. It had a sharp anterior border and an ill-defined posterior border, as does blood in the canal in other conditions. In contrast, arteries elsewhere in the eye or even in the trabecular meshwork are usually narrower, uneven in caliber, and somewhat tortuous in course, and are bright red in the center with a burnished edge.

The question will be unresolved until a biopsy is done, but in the meantime it is interesting to speculate on the consequences of an arterial anastomosis with the canal. Pressure in the canal would be raised at the point of anastomosis. Away from the anastomosis, the pressure would dissipate, but the average pressure in the canal would most likely be higher than normal. This, in turn, could raise the IOP to higher-than-normal levels.

The importance of finding blood in Schlemm's canal during the evaluation of a patient with open-angle glaucoma is that glaucoma secondary to high episcleral venous pressure is resistant to usual glaucoma treatment. IOP cannot be reduced to a level below that of the episcleral venous pressure. Drugs that reduce aqueous formation and drugs or laser treatment that increase aqueous outflow facility can only narrow the gap between the IOP and the elevated episcleral venous pressure. Only filtration surgery, which allows aqueous outflow to bypass the episcleral veins, will bring the IOP to normal,^{5,6} but the potential hazards of surgery in eyes with high venous pressure²⁴ must be kept in mind. If the

hypothesis I offer here is correct, ie, that glaucoma can result from high pressure in the canal of Schlemm due to a pathologic anastomosis of the canal with an artery, it is likely that this form of glaucoma would also be unusually resistant to medical treatment.

SUMMARY

In four patients with open-angle glaucoma, blood was present in the region of Schlemm's canal. The blood had four characteristics that distinguished it from other more common causes of blood in Schlemm's canal: (1) it was observed with a Zeiss gonioscopes, a lens that has no flange, and thus was not an artifact of the examination technique; (2) the blood could not be expelled from Schlemm's canal by raising IOP with para-limbal suction unless the pressure in the eye exceeded the diastolic ophthalmic artery pressure level; (3) as IOP approached this high level, the amount of blood in Schlemm's canal progressively decreased, eventually diminishing to a single point of reflux that was always in the same location; and (4) at this point the blood pulsed in and out of Schlemm's canal with each beat of the heart.

These findings suggest a pathological anastomosis between a deep limbal artery and Schlemm's canal. The high arterial pressure transmitted into Schlemm's canal could, in turn, lead to an elevation of IOP.

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DISCUSSION

DR MANSOUR F. ARMALY. It is a pleasure to discuss this provocative presentation. The hypothesis that this is a secondary glaucoma rests on the assumption that an open and free communication exists between an arterial vessel and the lumen of the canal of Schlemm, resulting in pressure elevation within the canal which is transmitted faithfully and equally to the otherwise normal level of ocular pressure.

Is there a free anastomosis and free blood in the lumen, or, is one looking at and describing a segment of an arterial vessel in the wall of the canal? Doctor Phelps favors the former based on his assessment of the blood column. I shall, on the other hand, be the devil's advocate and point out that an arterial vessel is more consistent with the findings and has not been ruled out.

The fixed location of the blood column over years of observation, the limited and fixed extent on repeated examination, being less than a quadrant in three of the four cases, its behavior on compression, all these speak in favor of a blood

column within an arterial vessel rather than free blood in the canal lumen. Why, if free, did the blood column remain of the same extent on repeated examination, repeated compression of the eye, and of the episcleral veins? Why, if connected to the high pressure of an arterial feeder, did a free blood column fail to fill most if not all of the circumference of the canal? Why was there no evidence of leakage of blood cells or proteins into the anterior chamber? Why has the force of gravity failed to influence the distribution of the blood column over the many years of its existence? Why did episcleral vein compression or inhibition of aqueous formation fail to influence the extent of the blood column? All these speak against a free anastomosis and in favor of a closed arterial vessel with fixed location and length.

Ashton and later on Jocson et al demonstrated a circumferential almost complete arterial circle intimately related to, but not connected with, the lumen of the canal. Such arteries were shown to lie just beneath the endothelial layer and bulge into the lumen. They too will have a sharp anterior boundary. A useful test in this regard is to observe the appearance and time course of intravenously injected fluorescein in this blood column, in the canal, and in the anterior chamber; it can help distinguish an open communication with the canal from a closed arterial vessel.

The ability of a free anastomosis to raise the pressure in the canal lumen is dependent upon its ability to provide enough blood flow rate to exceed the capacity of the collector channels and the episcleral veins. With 25 to 35 large collector channels, raising the pressure in the canal by 18 mm Hg (as in the first case) above pressure in the episcleral veins would require a significant blood flow rate that should involve all the collector channels and all the circumference of the canal with signs of increased flow into the episcleral veins. Such findings were notably absent, thus reducing greatly the likelihood that in these cases an open arterial anastomosis with the canal was responsible for the elevation of ocular pressure. If this were the case, why was the tonographic C-value (reported only for the second case) significantly reduced to 0.08 in the so-called affected eye if the mechanism is operative? Why was timolol therapy so successful in lowering ocular pressure? In two of the four cases glaucoma was bilateral and the asymmetry between the two eyes was not beyond that frequently encountered in open-angle glaucoma or exfoliation glaucoma to require a separate explanation or support a different mechanism.

To specifically test the author's hypothesis, one should, when the occasion arises, perform a sinusotomy or unroofing of the canal at a site distant from that of the anomaly. If the hypothesis is correct, such a procedure should promptly normalize ocular pressure level and result in the rapid movement of the blood column to the open site in free flow. This functional test will, I believe, be more informative than a biopsy of the anomaly and should precede it.

Finally, I wish to congratulate Doctor Phelps on bringing this possible mechanism to our attention and will look forward to more specific testing in future cases now that we have been alerted.

DR MAX FORBES. Doctor Phelps has developed an extremely intriguing concept and I found this analysis of these cases to be quite persuasive. Since the offending arterial anastomosis can be localized, I wonder if it could be occluded by laser photocoagulation thereby relieving the glaucoma. A favorable outcome would provide additional evidence to support the mechanism proposed by Doctor Phelps, and I would like to ask him if he has considered trying that method of treatment.

DR MARVIN SEARS. I was very pleased to hear Doctor Phelps personal observation in these four cases of glaucoma and was particularly gratified to hear Doctor Armaly's very thorough and persuasive discussion of his objection to the concept of connections of an artery to the canal. It is certainly true that Friedenwald made the hypothesis based on the rabbit eye—I think it was in the 1940s—that there might be a connection between an artery and Schlemm's canal. His actual hypothesis was not that the pressure in this eye would be raised but that it would be lowered because the osmotic effect of the proximity of the artery to the canal would suck water out of the canal. However, later studies, particularly by Ashton, and then later by a fellow named Grafton who worked at Hopkins, showed that in the human eye there were extensive A-V anastomoses in the bulbar conjunctiva and episclera but that there were no arterial connections to the canal. Several years later we became interested in this phenomenon as a possible mechanism for regulation of pressure, and abandoned the idea, but I did bring along three slides to show you how in some 70 odd monkeys, both rhesus and cynomolgus, studied by Doctor Jocson and Doctor Gaasterland, there were extensive A-V anastomoses, but we never found any connections of arteries to the canal. I would subscribe to Doctor Armaly's hypothesis that what has actually been seen is a vessel which is visible on gonioscopy but not directly in communication with the canal nor does it represent the canal itself. May I just show these three slides? This is from a rhesus monkey and it shows the under surface of the inferior rectus. This is the artery here and you see direct segmental filling in the vein down below here. You can see there is a distinction between the vessel on the left and on the right. The inside one is the artery and the outside is the vein. You get segmental filling under the muscle. In this second slide, you see this vessel running over here is a connector between artery and vein—there is a little branch in there. And finally, this shows a study with india ink and the muscular branch of the artery is right over here and here you see these fine connecting A-V anastomostic twigs and this vessel running horizontally here and then branching here perpendicularly is a vein and you can see that black stuff is entering the vein at these points which represents filling from this artery through these A-V anastomoses. So these anastomoses are very common in both the monkey and the human but we have not found any artery going into the canal. If there was a really close approximation you might expect as Friedenwald hypothesized over 40 years ago that there might be some sucking osmotic effect to decrease rather than increase the pressure.

DR CLEMENT McCULLOCH. This is a thought-provoking presentation. I wonder if the phenomenon described is not just an extension of a normal finding on routine gonioscopy. If one does not handle the gonioscopic lens lightly, blood will appear in Schlemm's canal in one quadrant, go away when pressure on the lens is withdrawn. The blood usually does not appear equally in the total extent of Schlemm's canal, but comes and then goes in a quadrant, as if that segment of the circulation has been embarrassed.

DR BRIAN R. YOUNGE. I think they are both right, perhaps for the following reason. The disc in a patient with a unilateral retinal vein occlusion has engorged veins that will not collapse with ophthalmodynamometry until pressures approach diastolic pressure. I think this is good evidence that what Doctor Phelps is seeing is really arterial pressures. The fact that Schlemm's canal is not necessarily continuous from one part to the other may permit this to be an arterIALIZED segment of Schlemm's canal. One can actually measure the pulsations and pressure with the ophthalmodynamometer and maybe because of this discontinuity the two ideas are both compatible.

DR CHARLES PHELPS. I thank all the discussants, and I'm glad I was able to provoke a little thought among the audience this early in the morning. Let me consider your questions in reverse order. First, Doctor Younge, I think there probably is circumferential flow in Schlemm's canal. Doctor McCulloch, I think we ruled out artifactual blood reflux into Schlemm's canal by using a Zeiss lens that doesn't have a flange to compress the episcleral veins. Doctor Sears, you were unable to find a connection between an artery and Schlemm's canal in 60 to 80 monkeys, but I think a rare monkey or a rare person could still develop such an anastomosis pathologically. Certainly the incidence in people of such an anastomosis is far more rare than 1:60. Doctor Forbes, we haven't considered photocoagulation seriously, because the glaucoma in our patients didn't seem severe enough to warrant it, but it is an interesting idea that I will keep in mind if we ever get backed into a corner.

I've been discussing glaucoma with Mansour Armaly since 1967, when I was assigned to work for him before I started my residency. I rarely have the opportunity to have the last word in these discussions, so I appreciate the format of this meeting. I'm not going to try to answer all of his points, which I think were all very good ones. I would just note that if the blood we observed was in a normal vessel, such as those that have been demonstrated histologically by many investigators, it is difficult to understand why we do not see it gonioscopically in most normal eyes and yet do see it in these four eyes. The normal vessels that run alongside Schlemm's canal in most normal eyes must ordinarily be invisible. Why could we see the blood so plainly in these cases? A second point is that when we compressed the eyes and raised the intraocular pressure, the blood column contracted from both ends to a single point. One could interpret this as indicating an artery that branched in two directions from its point of entry, but I think an

equally reasonable interpretation is that the point represented an anastomosis where an artery entered into the canal of Schlemm with the blood travelling into the canal both ways from there. Gravity had some effect; the filling extended further inferiorly than superiorly from the point of presumed anastomosis. In one patient we tried injecting fluorescein to see if we could actually demonstrate the anastomosis, but we were unable to see the fluorescein enter with sufficient clarity to make a judgement. In the one patient who underwent surgery, I suggested that the area of presumed anastomosis be excised during the trabeculectomy. Unfortunately, the surgeon who did the operation chose another quadrant, so we still don't have an answer.

Let me once again thank all the discussants.