SPONTANEOUS ANTERIOR CHAMBER HEMORRHAGE FROM THE IRIS: A UNIQUE CINEMATOGRAPHIC DOCUMENTATION*

BY *Robert B. Welch,* MD

INTRODUCTION

ACCORDING TO DUKE-ELDER[1] ANTERIOR CHAMBER HEMORRHAGE FROM THE IRIS is a rare event if trauma is excluded. His list of causative factors apart from trauma include 1) over-distention of the vessels, such as from hypotony during intraocular surgery, 2) fragility of vessel walls, such as seen in iridocyclitis associated with herpes or gonorrhea, 3) a derangement of the blood itself affecting the vessel wall, as in hemophilia, leukemia or scurvy, 4) neovascularization as seen in diabetic rubeosis and 5) highly vascularized tumors such as juvenile xanthogranuloma or melanoma. Although he lists no idiopathic category for spontaneous anterior chamber hyphema, Duke-Elder seems to anticipate such a category when he alludes to the fact that "the older writers spoke at some length of spontaneous hemorrhages into the anterior chamber" and states that the origin of many "still seem enigmatic."

In recent years a new form of iris vascular abnormality has been recognized which occurs at the pupillary border and has been variously referred to as vascular tufts,[2] microhemangiomas,[3] or neovascular iris tufts.[4] It is perhaps lesions of this type that account for apparent spontaneous idiopathic anterior chamber hemorrhages that have been reported on occasion over the past 170 years (Table I).

BACKGROUND INFORMATION

Duke-Elder[1] lists a number of references to reports in the early literature of spontaneous anterior chamber hemorrhage including one as early as 1808 by John Bell.[5] However, it was not until Fechner's paper in

*From The Retina Service of The Wilmer Ophthalmological Institute, The Johns Hopkins Hospital, Baltimore, Md.

TABLE I: CAUSES OF ANTERIOR CHAMBER HEMORRHAGE EXCLUDING TRAUMA
DUKE-ELDER'S CLASSIFICATION AND RECENT UPDATE

Cause	Example
Over-distention of vessels	Hypotony during intraocular surgery
Fragility of vessel walls	Iridocyclitis with herpes or gonorrhea
Blood derangement	Hemophilia, leukemia or scurvy
Iris neovascularization	Diabetic rubeosis
Highly vascularized tumors	Juvenile xanthogranuloma
Related to surgical procedure	Cataract wound vascularization with hyphema (Swan[21]; Watzke[22])
Idiopathic	Engimatic spontaneous hemorrhage from the iris—Duke-Elder (older literature)
	Pupillary tufts, microhemangiomas, or neovascular tufts (recent literature)

1958[6] that a good documentation of such a case became available in the modern literature. This report described a 42-year-old woman with prominent radial iris vessels and an aneurysmal abnormality at the pupillary border of the right eye which was the site of bleeding into the anterior chamber. The left eye showed a similar finding but no hyphema. Fechner found no associated ocular or systemic disease and postulated a congenital abnormality. In 1965 Riffenburgh[7] reported a 59-year-old woman who suffered recurrent spontaneous hemorrhages from the iris on 10 to 12 occasions in a period of five months. One of these was observed and seemed to emanate from the surface of the iris in the region of the colarette in a pulsatile fashion synchronous with the pulse. No ocular or systemic abnormalities were found and in spite of repetitive hemorrhage no complications ensued.

In 1968 Jensen and Lundback[8] in Denmark stimulated a new interest in the iris vasculature when they introduced a technique of iris fluorescein angiography using a Zeiss fundus camera. They studied nondiabetics, early onset and longstanding diabetics and found that although both nondiabetics and early onset diabetics showed fluorescent dots at the pupillary border, it was the longstanding diabetics who showed leakage from the pupillary border. Bagesen[9] in 1969 followed up on their study and modified the photographic technique with a Zeiss Photo-Slit-Lamp. He found that both nondiabetics and diabetics were capable of demonstrating leakage from the pupillary border of the iris and that this was unrelated to fundus abnormality. None of the patients in either of these reports showed clinically apparent abnormal pupil vasculature nor were there any reports of hyphema. During the same period (1968), Cobb[2] in London presented his observations on 44 patients with vascular tufts at the pupillary margin which he had collected over a nine-month period through observation at the slit lamp.

He noted the following characteristics of the tufts: 1) They protruded forward from the iris in the form of single or multiple loops. 2) They were vascularized. 3) They were adjacent to the pupillary ruff when this was present. 4) They were separate from each other and did not form a vascular network. A large proportion of his patients, both diabetics and nondiabetics, had bilateral tufts without retinal abnormality, while a few of the cases showed a mixture of tufts and flat neovascularization of the iris. Twelve patients showed unilateral tufts. It was of interest that those patients with many and prominent tufts showed either an elevated 2-hour blood glucose without retinopathy, severe vascular or respiratory disease or a retinopathy in the absence of the other conditions. In a later publication Cobb et al[10] also associated the occurrence of vascular tufts in patients with myotonic dystrophy. None of these patients suffered a hyphema by history or observation. However, in 1969 Rosen and Lyons[3] did report a patient with such abnormalities who presented with a spontaneous unilateral hyphema. They chose to designate such lesions as microhemangiomas at the pupillary border. Their patient showed no other abnormality other than some evidence of hypertensive vasculopathy in the fundus. An additional two cases of hyphema from pupillary tufts or microhemangiomas were reported in 1972 by Sellman[11] in patients without apparent systemic disease. During the next five years an increasing number of cases of spontaneous hyphema were reported[12-15] most of which seemed related to vascular tufts at the pupillary border.

In 1977 Coleman, Green and Patz[16] presented a case of bilateral pupillary tufts with observation of active bleeding from a tuft at 12 o'clock in the right eye. This lesion was subsequently treated with the Argon laser. Because the patient had a cataract in the left eye an intracapsular cataract extraction was performed on this eye with a sector iridectomy to include a clinically apparent pupillary tuft. Histopathologic study of the iris obtained revealed neovascularization at the pupillary margin and mild chronic inflammatory cell infiltration. In the same year Perry et al[17] presented a patient who developed acute glaucoma following a spontaneous hyphema from iris tufts while Krarup[18] reported recurrent spontaneous hyphemas in a patient with congenital cyanotic heart disease who showed both microhemangiomas and flat neovascularization of the iris. Most recently, Mason and Ferry[19] reported a bilateral spontaneous hyphema arising from what they termed iridic microhemangiomas while in a later publication Mason[20] suggested that a more appropriate name would be iris neovascular tuft. The reports to date are summarized in Table II.

TABLE II: IDIOPATHIC SPONTANEOUS ANTERIOR CHAMBER HEMORRHAGE FROM THE IRIS
A REVIEW OF CASE REPORTS

Report and reference	Date	Patient age	sex	Anterior segment observation & iris abnormalities: Right eye	Left eye	Correlation with angiography	Systemic disease
Fechner	1958	42	F	Peripupillary ovoid pinkish sac—active bleeding seen	Peripupillary swelling prominent radial vessels	No	None
Riffenburgh	1965	59	F	Spurts of blood from iris surface synchronous with pulse	No description	No	None
Rosen and Lyons	1969	73	M	Cluster of blood vessels at pupillary border—called microhemangiomas	Anterior chamber blood clot from 1 o'clock, cluster of vessels at pupillary border	Yes	Hypertension Chronic bronchitis
Manor and Sachs	1972	58	F	None specifically stated	Blood clot formed at 12 o'clock following dilated exam with 10% phenylephrine counteracted with pilocarpine 2%. No abnormalities seen	No	Hypertension A-V fundus changes
Sellman	1972	45	F	Pupillary vascular tufts	Vascular tuft at 6 o'clock with hyphema & blood in aqueous	No	None
	1972	76	F	Hyphema—blood clot at 6 o'clock	Vascular tuft 9 o'clock	No	Dyspnea Cardiac sx
Magauran	1973	62	M	Hyphema—recurred 7 yrs later with glaucoma circumpupillary vascular tufts	Circumpupillary vascular tufts	Yes	None
	1973	61	F	Hyphemia with tufts	Not stated	Yes	Not reported

	1973	69	M	Vascular tufts with active bleeding	Not stated	Yes	Not stated
Israel and Lorenzetti	1974	60	F	Microhemangiomas	Microhemangiomas observed—3 weeks later blood in AC	Yes	Osteoarthritis GTT negative
Savir and Manor	1975	47	F	Dilated arteries temporal side of iris. Pupillary dots. Hyphema from temporal side. Site not defined	Not stated	Yes	Right orbital hemangioma
Perry, Mallen and Sussman	1977	69	F	Microhemangiomas Periph. iridectomy later	Hyphema, acute glaucoma & microhemangiomas. Peripheral iridectomy later	Yes	Emphysema
Coleman, Green and Patz	1977	71	F	Acute bleeding—many vascular tufts—Argon laser	Vascular tufts. Iridectomy spec. = new vessels at pupillary border	Yes	Not stated
Krarup	1977	36	F	Neovascular net at pupillary border. Cluster pupillary tufts. Recurrent hyphema—active bleeding seen	Neovascular net at pupillary border. Cluster pupillary tufts. Hyphema	No	Congenital cyanotic heart disease with venous stasis retinopathy
Mason and Ferry	1979	81	M	Hyphema. Active bleeding from pupillary tuft at 3 o'clock	9 o'clock pupillary tuft. Hyphema following dilation	Yes	Hypertension Parkinsons disease
Welch	1980	54	F	Active bleeding from pupillary border 11 o'clock subtle whitish tuft	Filmy pupillary membrane remnant 11 o'clock. Red dot in pupillary ruff at 6:30	Yes	Diabetes diet-controlled

It is the purpose of this paper to present a case of idiopathic spontaneous anterior chamber hemorrhage from the iris, document its occurrence with cinematography and provide a stimulus for the evaluation of the etiologic, morphologic and pathologic aspects of this entity.

CASE REPORT

A 54-year-old white female was referred by her internist on September 13, 1979 for emergency consultation because of an anterior chamber hemorrhage in the right eye. The patient had been previously seen by me for routine ophthalmic examination on March 4, 1969, November 2, 1972 and January 20, 1975. On these occasions her best corrected vision in each eye was 20/15 and her near vision was Jaeger #1 with an appropriate reading addition. Her refractive error was minimal and the 1975 glasses prescription was OD +.75

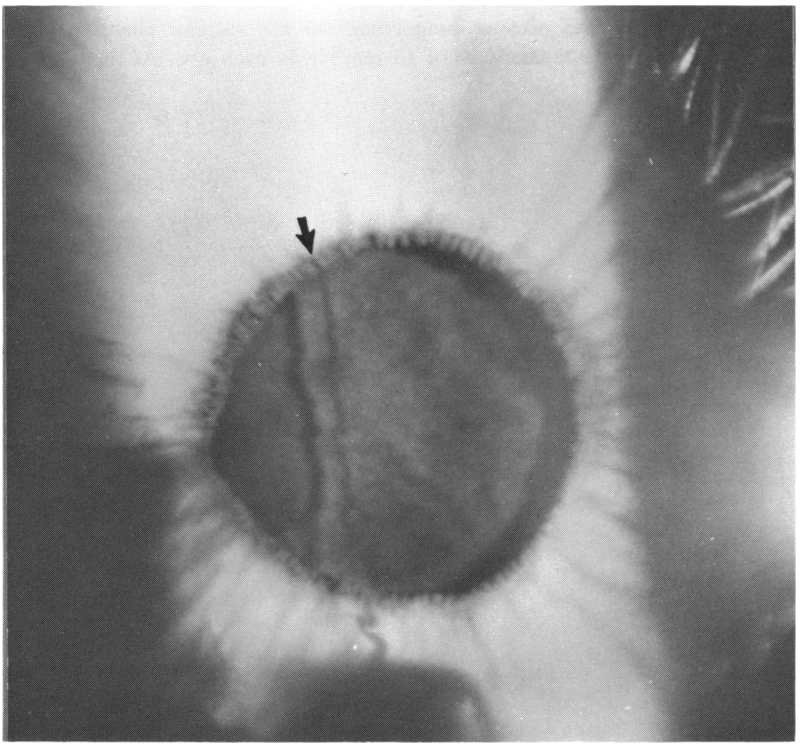

FIGURE 1
Slit lamp photograph of the right eye showing active bleeding from the iris border at 11 o'clock (arrow). Dark vertical stripe to the left is a shadow from the blood column on the anterior lens capsule.

+.50 × 155; OS +.25 add +2.00. Family history was significant because her grandmother, father, brother and sister all had diabetes. The patient herself was diagnosed as diabetic in 1973 but had been controlled on diet alone. The remainder of her past medical history was negative. Her previous ocular examinations had been unremarkable except for presbyopia and a small choroidal nevus superotemporal to the left optic disc. Her anterior segments were always recorded as normal.

On presentation to the Retina Service on September 13, 1979 the patient stated that two days previously she had moved large cans of clay weighing approximately 250 pounds while at work in her school art class and subsequently noted cloudiness of vision in her right eye. One day previously she noted redness of the right eye and continued cloudiness of vision. On the morning of the day of her referral she suffered acute pain in the right eye while in the sunlight of the school yard during a fire drill and promptly consulted her internist who immediately referred her to me.

Examination revealed a visual acuity of 20/25 in the right eye and 20/20 in the left eye. There was obvious hemorrhage in the anterior chamber of the right eye. Applanation tensions were 15 mm/Hg in each eye. At the slit lamp

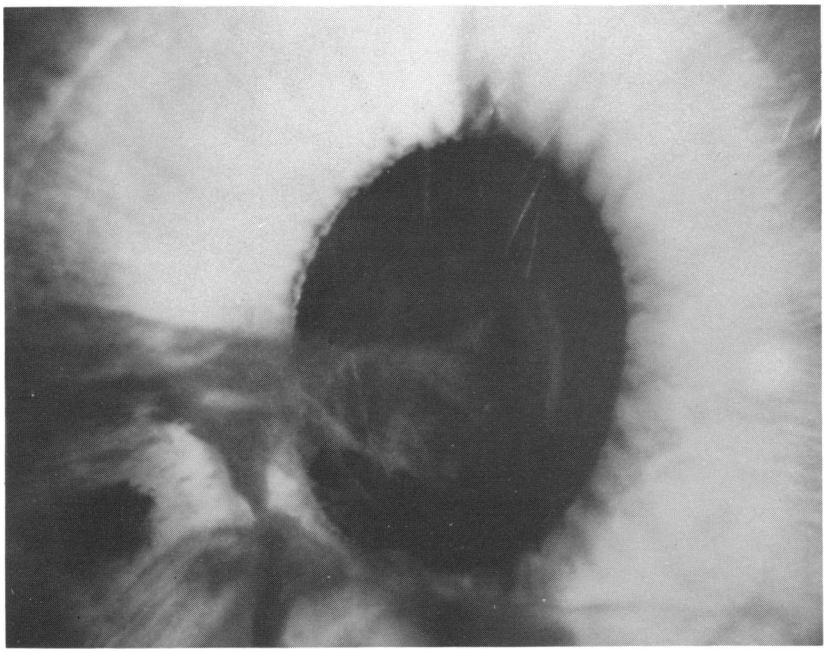

FIGURE 2
Slit lamp photograph of the right eye following instillation of homatropine. The pupil is partially dilated, bleeding has stopped and residual blood remains in the anterior chamber.

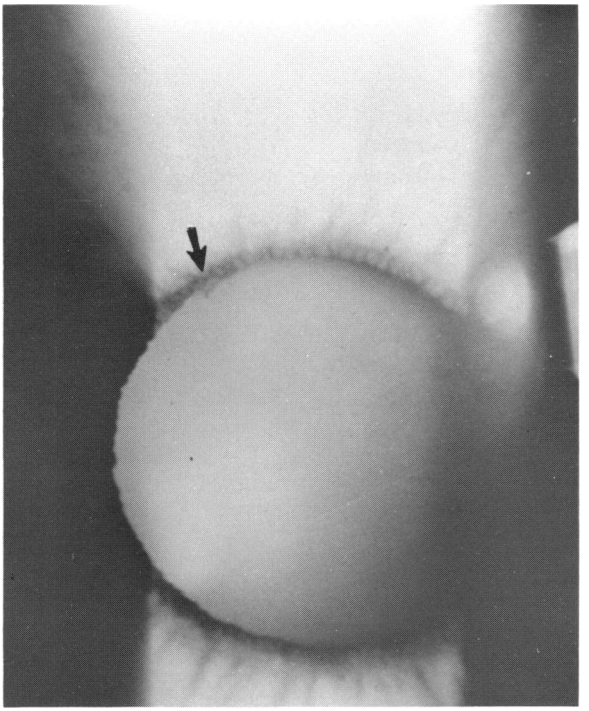

FIGURE 3
Slit lamp photograph of the right eye following absorption of hemorrhage from the anterior chamber seen in Fig 1 and Fig 2. A barely perceptible whitish pupillary tuft is seen at 11 o'clock (arrow).

the patient showed active bleeding from the pupillary border of the right eye at 11 o'clock with blood cascading down across the pupil onto the inferior iris and forming a small inferior hyphema (Fig 1). No obvious rubeosis could be seen and the left anterior segment appeared normal. The patient was placed at rest in the supine position and told to keep both eyes closed. Over the next few minutes the anterior chamber hemorrhage did not appear to increase. However, repeat biomicroscopy showed continued bleeding and cinematography was obtained to document the event. Because of no evidence of diminution of blood flow from the iris it was decided to instill homatropine 5% with the hope that dilation of the pupil would tamponade the offending vessel. Within several minutes the bleeding stopped (Fig 2) and the patient was sent home on bed rest and binocular eye pads. When seen the following day almost all the blood had resorbed. Examination now showed a small white pupillary tuft at 11 o'clock but no evidence of abnormal blood vessels (Fig 3). Subsequent iris angiography showed leakage from the pupillary tuft at 11 o'clock and

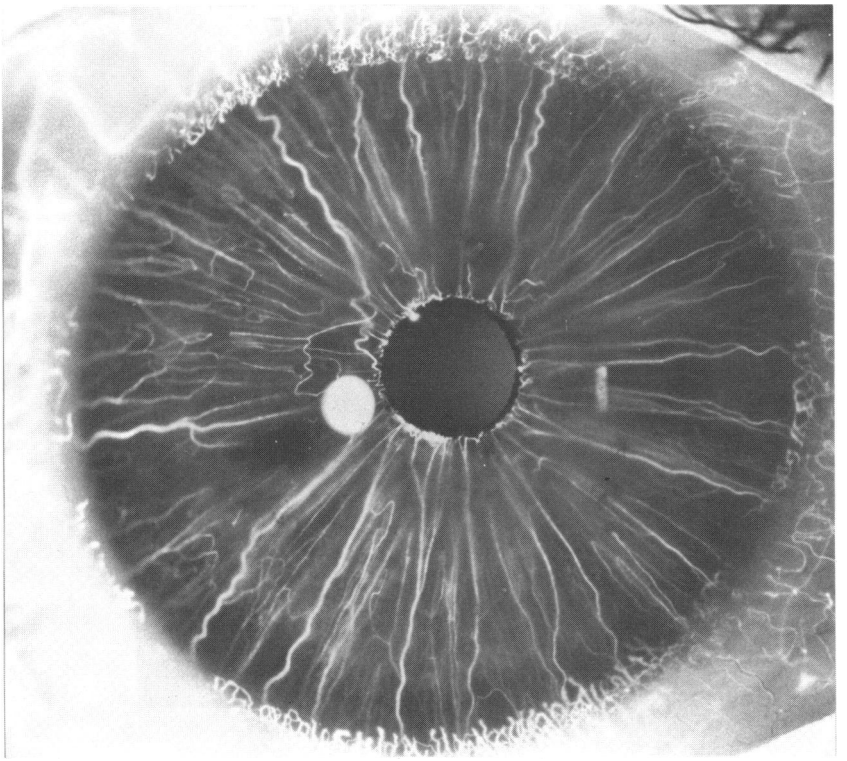

FIGURE 4
Iris angiography of the right eye shows fluorescein leakage from the pupillary tuft at 11 o'clock as well as from other areas of the pupillary border.

from focal areas at 12 and 3 o'clock as well as from the inferior iris border (Fig 4). No obvious pupillary vascular abnormalities were noted in these regions. The patient showed no fundus abnormalities or gonioscopic evidence of rubeosis. The left eye appeared normal (Fig 5) although a reddish vessel was seen in the pupillary ruff at 6:30. Iris angiography of the left eye (Fig 6) showed peripupillary fluorescent areas with some leakage. Gonioscopy of the left eye was unremarkable.

DISCUSSION

When assessing the cause of an apparent spontaneous anterior chamber hemorrhage unassociated with trauma one should always be alert to certain possible causes enumerated by Duke-Elder[1] and others[12] and referred to earlier in this paper. It is of note that even within the

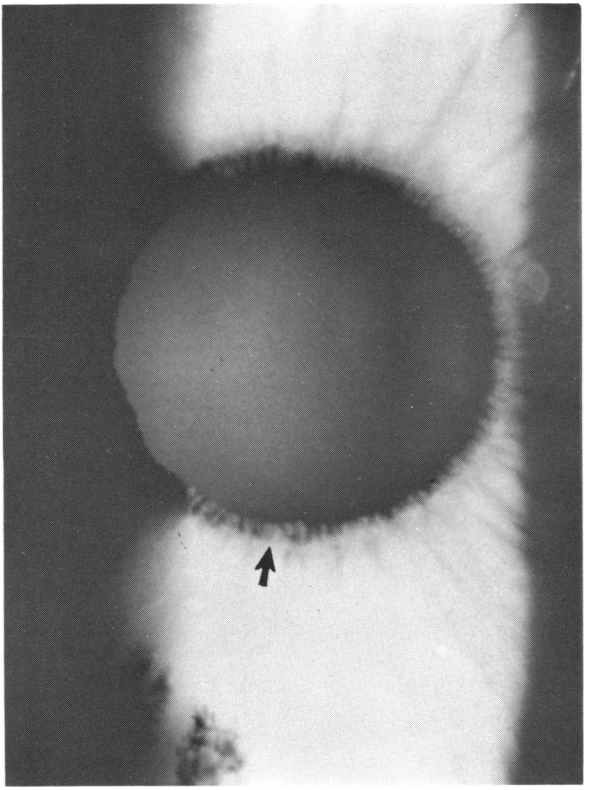

FIGURE 5
Slit lamp photography of the left eye appears normal. High magnification showed a subtle reddish dot in the pupillary ruff at 6:30 (arrow).

framework of this group new clinical entities may still be recognized as illustrated by the observations of Swan[21] and Watzke[22] in recent years on recurrent hyphema from wound vascularization following cataract surgery. However, from a review of the literature we have seen that the majority of cases of spontaneous idiopathic anterior chamber hemorrhage are secondary to bleeding from the iris itself and most often from the pupillary border. In most of these reports gross vascular abnormalities were described and variously referred to as vascular tufts, microhemangiomas or iris neovascular tufts. The cause of these lesions to date remain unknown but have been attributed to congenital lesions, a form of rubeosis or secondary to some unrecognized systemic factor by various authors.

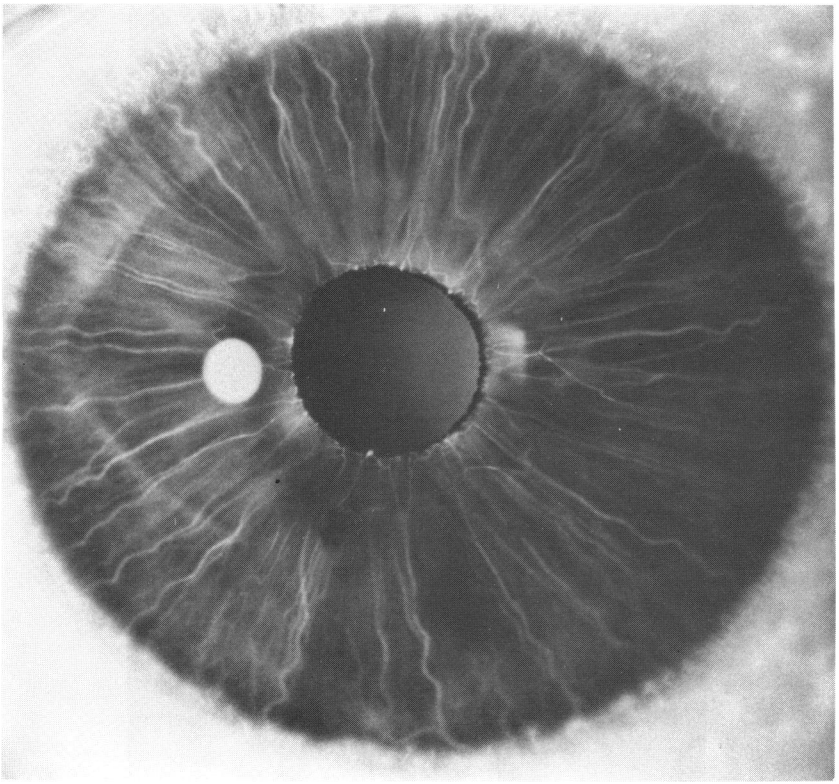

FIGURE 6
Iris angiography of the left eye shows fluorescence from the area of the reddish dot seen in Fig 5 at 6:30, as well as from other areas of the pupillary border above.

It is certainly feasible that some cases could represent congenital lesions such as persistent pupillary membrane, corpora nigra or vascular hamartoma, yet the tufts described in the majority of case reports are not found in young individuals and are anatomically unrelated to pupillary remnants. The terminology microhemangiomas, therefore, is probably not the best term to use since hemangioma connotes a congenital lesion and Ferry[23] has questioned whether true hemangiomas of the iris exist at all. Furthermore, Green[16] has shown histopathologically that a typical iris tuft is indeed neovascular in nature. Thus it is probably best to refer to these lesions as pupillary vascular tufts as originally suggested by Cobb[2] or iris neovascular tufts as recently proposed by Mason.[4]

The question remains to explain the etiology of such lesions. We have noted the fact that vascular tufts occur in patients with no obvious systemic abnormality yet we are also aware that they occur in patients with diabetes, congenital heart disease, hypertension, central vein thrombosis and myotonic dystrophy. A search for a common denominator seems indicated. Mason[20] has postulated that the common factor in myotonics and adult onset diabetics is pancreatic B-cell dysfunction with resultant high serum insulin and that a refractive hormone-receptor interaction may play a causative role. In addition to hyperinsulinemia he also speculated on hypotony as a cause, since he points out that myotonics often show hypotony while ocular hypotension may occur after retinal vein occlusion and may precede rubeosis and neovascular glaucoma in eyes with proliferative diabetic retinopathy. Although pupillary tufts are neovascular in nature they do not behave like typical rubeosis iridis either morphologically or prognostically and have not been observed to change with time in the reports to date. Since we know that certain diabetics as well as so-called normal individuals show pupillary leakage of fluorescein without evidence of tufts, it may be that it is these patients who go on to develop the full blown picture. Continued observation of such patients may hopefully provide some information in the future.

In the present case report there was no obvious vascular abnormality of the iris when the patient presented with active bleeding from the pupillary border. Of note was the fact that I had personally followed this patient for over ten years without notation of anterior segment abnormality. After the hemorrhage had cleared I was able to see a subtle whitish tuft from the pupillary border at 11 o'clock in the right eye, while the left iris appeared normal, although there was a tiny reddish dot in the pupillary ruff at 6:30. Iris angiography confirmed these areas and showed leakage of dye in these and other areas. No clinical neovascular tufts were observed. It is of interest that several colleagues were asked to examine this patient's irides and all stated that there were no abnormalities. A recent in-depth medical evaluation of this patient revealed no abnormalities with the exception of a slightly elevated endogenous insulin level and a positive glucose tolerance test.

This case would seem to represent an intermediate state between pupillary border leakage seen on angiography in some apparent normal individuals and frank pupillary neovascular tufts reported in normal individuals as well as in a variety of conditions from myotonic dystrophy to cyanotic heart disease. Whether all of the reported cases represent a continuum of a fundamental abnormality remains to be seen.

SUMMARY

A 54-year-old white female was observed with an apparent spontaneous idiopathic anterior chamber hemorrhage from the pupillary border of the iris. This event was documented by cinematography. A review of the literature concerning anterior chamber hemorrhage is presented and reports of spontaneous hyphema enumerated. The relationship of the entity of pupillary vascular tufts to the present report are discussed and etiologic factors considered. It is apparent that closer scrutiny of the pupillary border should be performed and iris angiography obtained in a variety of eyes to delineate normal and abnormal variants.

REFERENCES

1. Duke-Elder S: *System of Ophthalmology*. St Louis, CV Mosby Co, 1966, vol IX, pp 19-21.
2. Cobb B: Vascular tufts at the pupillary margin; a preliminary report on 44 patients. *Trans Ophthalmol Soc UK* 88:211, 1968.
3. Rosen E, Lyons D: Microhemangiomas at the pupillary border. *Am J Ophthalmol* 67:846, 1969.
4. Mason GI: Iris neovascular tufts. *Arch Ophthalmol* 97:2346, 1979.
5. Bell J: *The Principles of Surgery*. London, 1808, vol 3, p 269.
6. Fechner PV: Spontaneous hyphema with abnormal iris vessels. *Br J Ophthalmol* 42:311, 1958.
7. Riffenburgh RS: Recurrent spontaneous iris arterial hemorrhage. *Am J Ophthalmol* 59:319, 1965.
8. Jensen VA, Lundback K: Fluorescence angiography of the iris in recent and long term diabetes. *Diabetologia* 4:161, 1968.
9. Baggesen LH: Fluorescence angiography of the iris in diabetics and non-diabetics. *Acta Ophthalmol* 47:449, 1969.
10. Cobb B, Shilling JD, Chisholm IH: Vascular tufts at the pupillary margin in myotonic dystrophy. *Am J Ophthalmol* 69:573, 1970.
11. Sellman A: Hyphema from microhemangiomas. *Acta Ophthalmol* 50:58, 1972.
12. Manor RS, Sachs W: Spontaneous hyphema. *Am J Ophthalmol* 74:293, 1972.
13. Magauran DM: Unilateral spontaneous hyphema. *Br J Ophthalmol* 57:945, 1973.
14. Israel MP, Lorenzetti DWC: Bilateral microhemangiomas of the pupillary border with later hyphema. *Can J Ophthalmol* 9:138, 1974.
15. Savir H, Manor RS: Spontaneous hyphema and vessel abnormality. *Arch Ophthalmol* 93:1056, 1975.
16. Coleman SL, Green WR, Patz A: Vascular tufts of pupillary margin of iris. *Am J Ophthalmol* 83:881, 1977.
17. Perry HD, Mallen FJ, Sussman W: Micro haemangiomas of the iris with spontaneous hyphema and acute glaucoma. *Br J Ophthalmol* 61:114, 1977.
18. Karup JC: Atypical rubeosis iridis in congenital cyanotic heart disease. *Acta Ophthalmal* 55:581, 1977.
19. Mason GI, Ferry AP: Bilateral spontaneous hyphema arising from iridic microhemangiomas. *Ann Ophthalmol* 11:87, 1979.
20. Mason GI: Iris neovascular tufts. *Arch Ophthalmol* 97:2346, 1979.
21. Swan KC: Hyphema due to wound vascularization after cataract extraction. *Arch Ophthalmol* 89:87, 1973.

22. Watzke RC: Intraocular hemorrhage from wound vascularization following cataract surgery. *Trans Am Ophthalmol Soc* 72:242, 1974.
23. Ferry AP: Hemangiomas of the iris and ciliary body: Do they exist? A search for a histologically proven case. *Int Ophthalmol Clin* 12:117, 1972.

DISCUSSION

DR ROBERT C. WATZKE. Doctor Welch has presented a beautifully documented and extensive review of an entity whose most apt title is iris neovascular tufts. I'd like to show a movie of a similar case and also make three comments.

This is a movie of a patient seen seven years ago with spontaneous hyphema from a neovascular anomaly at the mid-iris. The patient was 65-years-old, not diabetic by the usual standards and had no significant ocular or systemic disease. He presented with episodes of blurred vision due to hemorrhage from the iris as demonstrated in the film. An iris angiogram demonstrated diffuse leakage from both mid-stromal and pupillary vessels. Unfortunately, he was lost to follow-up and his records are now unobtainable.

These patients seem to fall into two groups. First are those who have some ocular or systemic cause for iris neovascularization. Diabetes and myotonic dystrophy are examples. In a recent article, Mason states that 25 of 62 reported cases had some associated disease. Many were occult diabetics and had abnormal glucose tolerance curves or abnormally high serum insulin levels after glucose challenge.

One could argue that, in spite of the morphologic differences, iris neovascular tufts are only a variant of iris rubeosis. Note that this case and many others have diffuse leakage from many iris vessels rather than just the point of hemorrhage alone.

Yet there is another group of patients with this entity who have no other ocular or systemic disease. Until we know the basic cause of iris neovascularization, we shall have to consider this an entity separate from the usual rubeosis.

Finally, it is intriguing that this occurred in a diabetic without diabetic fundus changes. This is difficult to explain by the current hypothesis of diffusion (toward the iris) of a vasoformative factor from an ischemic retina. Perhaps a retinal angiogram might demonstrate occult ischemia.

I wish to thank Doctor Welch for sending his manuscript well in advance and will close with one question: "If homatropine dilatation had not stopped the bleeding, what would have been your next move?"

DR WILLIAM H. SPENCER. At the request of Doctor Melvin Rubin, I would like to show photographs taken by Doctor Rubin of a remarkably similar case of idiopathic spontaneous bleeding from the pupillary margin. (slide) Under this power you can see a small bleed appearing at the pupillary margin. A slightly higher power view demonstrates the apparent bleeding site. (slide) Doctor Rubin elected to treat this with a laser. This photograph was taken one minute

after a 50 μ 200 mw laser burn placed to the bleeding site. At a slightly higher power (slide) the site of application of the laser burn is visible in this location. According to the notation on the slide, three such spots were placed. Twenty-four hours later the hyphema had cleared and in the final slide it is quite difficult to detect the site of the laser application. I would like to add one comment on my own. My experience with spontaneous bleeding into the anterior chamber has been related to iris melanomas and iris nevi. It is not uncommon to find varicosities on the surface of focal tumors of the iris. The paper by Rones and Zimmerman in 1958 mentioned that approximately 20% of iris melanomas had spontaneous bleeds. It would be of interest to review some of the cases that have been described as spontaneous bleeds from the iris to see whether or not they were associated with iris neoplastic processes.

DR THOMAS P. KEARNS. Doctor Welch told me that he wanted me to discuss this paper. I don't know how he knew that I had seen such a patient, but I have. About 25 years ago I saw an elderly woman from a local nursing home. The nurse asked me to see her because she had seen some blood in her eye.

One of our residents saw this lady first and then came to me saying that she had a very strange finding. He said that she had an anomalous blood vessel running across the anterior chamber. I looked and the "vessel" was anomalous. It had no vessel wall! There was a thin column of blood streaming down from the 12:00 o'clock position to the 6:00 o'clock position across the iris. She had a small hyphema that slowly increased in size as we watched it.

We didn't know just what we should do so we admitted her to the hospital. Fortunately the bleeding stopped spontaneously. We did not have a light coagulator or laser in those days so we were thinking about doing an iridectomy. I have not seen such an example since, but I would think that treatment with the laser would be the preferred form of therapy now.

I would like to thank Doctor Welch for showing us this interesting patient.

DR WILLIAM GLEW. About a month ago one of our residents called me at the end of the day to demonstrate a case just like you've seen here. It's a very dramatic thing and stimulates people to take pictures, but unfortunately, our photographer had already gone home. We did however deliver two bursts from the argon laser to the pupillary margin. Because of the location of the bleeding vessel at the pupillary iris margin, one should be careful to avoid missing the iris and hitting the macula. Therefore we used 0.10 of a second instead of a longer exposure and the two applications successfully stopped the bleeding.

DR PAUL LICHTER. This is probably belaboring a small point because these cases are so rare. However, I get the idea from the report and discussion that the impression might be left that these hemorrhages are bad things and ought to be treated. I had one of these in a glaucoma patient on maximum medical

therapy. We just left it alone and the bleeding stopped quite quickly. I would like to ask Doctor Welch whether, in his review of the literature, he came up with any incidence of recurrence of the bleeding that would suggest that one needs to treat these patients. My single anecdotal case would suggest that you don't need to treat them.

DR JOHN WOODWORTH HENDERSON. May I add one comment. I've seen one of these occur, but in a patient of Doctor Fralick's. I was a beginning resident and responsible for removing silk sutures from a post-operative cataract. I watched the "rainbow falls" trickle down through the anterior chamber, layer up into a hyphema, and finally stop.

DR ROBERT B. WELCH. I would like to thank Doctor Watzke for his dicussion of this paper. His case of anterior chamber hemorrhage is most interesting since it depicts bleeding which seems to emanate from the mid-stroma rather than the pupillary border. There are two reports in the literature which also showed bleeding from the mid-stroma, one by Riffenburgh in 1965 and the other by Savir and Manor in 1978. The latter report is especially interesting because the patient also had an orbital hemangioma.

In response to Doctor Watzke's question, "If homatropine had not worked, what would you have done next?" I can only answer in a retrospective way. Initially I placed the patient at bed rest and when the bleeding continued I thought of the laser. However when the instillation of homatropine drops worked my dilemma was ended. I suppose my next step might have been laser therapy had the bleeding continued.

I would like to thank Doctor Spencer for presenting Doctor Rubins' case which appears similar to mine. I would also like to thank him for emphasizing that iris melanomas may be the basis for anterior chamber hemorrhage.

I know that Doctor Kearns has seen everything and that is why I asked him to comment on this paper. I appreciate his report.

Doctor Glew has demonstrated still another case where the laser was effective in stopping the bleeding.

I appreciate Doctor Lichter's comments for they appeal to my conservative philosophy. In the literature there was only the case of Doctors Coleman, Patz and Green that received the laser treatment. All of the other cases showed cessation of bleeding from the iris. However, a number of the reports indicate recurrent hyphema and in the report by Perry, Mallen and Sussman acute glaucoma was precipitated by the anterior chamber hemorrhage.

Whatever the basic cause of spontaneous bleeding from the pupillary border of the iris it would seem that the pathologic focus is neovascular in nature. Thus I would endorse the term vascular tuft or neovascular tuft of the iris rather than microhemangioma. It is hoped that this paper will stimulate the closer scrutiny of the pupillary border of the iris in all patients.