FLUORESCEIN STUDIES OF PATIENTS WITH MACULAR EDEMA AND PAPILLEDEMA FOLLOWING CATARACT EXTRACTION*

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IN 1953, IRVINE[1] described the syndrome of spontaneous rupture of the hyaloid face following uneventful cataract extraction with the formation of vitreous adhesions to the wound, irritability of the eye, and reduction of visual acuity secondary to vitreous opacities and macular degeneration. Since recent improvements in sutures, instruments, techniques, and antibiotic therapy have lowered the incidence of many postoperative complications, loss of central vision secondary to changes in the macula[1-7] and optic nerve[2,6,8] following uneventful cataract extraction has been recognition as a major complication of cataract surgery. The funduscopic changes, although characteristic, may be easily overlooked, and the failure of the patient to refract to normal vision may be mistakenly attributed to a variety of other factors, e.g., senile macular degeneration, uveitis, optic neuritis, irregular astigmatism, or hazy media. The true incidence, natural course, etiology, pathogenesis, treatment, and prevention of this disease process are unknown.

The purpose of this report is to present the fluorescein angiographic findings in these patients. The macular lesion exhibits a pattern of fluorescence which is sufficiently characteristic and easy to see with the ophthalmoscope and slit-lamp to permit early and accurate diagnosis. Fluorescein studies demonstrate that the intraretinal accumulation of fluid occurring in the macular region and in the area of the optic nerve head is secondary to alterations in the capillary permeability of the intraretinal and papillary vessels.

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METHODS AND MATERIALS

Forty-four patients developing macular edema and/or papilledema following cataract extraction were studied with fluorescein angiography as described by Novotny and Alvis[9] and modified by Sever and Justice.[10] These patients were obtained from the authors' private practice, the outpatient department of the Jackson Memorial Hospital, and referrals from local ophthalmologists. Periodic photographs were made during the one-hour period following injection of the dye. Biomicroscopic examination of the vitreous and fundus by one or both authors was done in most of these patients, the Hruby lens being used in most instances in order to preserve corneal clarity for fluorescein angiography. Selected patients were also studied with the Goldmann three-mirror contact lens with particular attention to the relationship between vitreous attachments and the macular and optic nerve changes. Funduscopy utilizing the cobalt blue filter in the indirect ophthalmoscope and slit-lamp was done at periodic intervals during the course of fluorescein angiography. Stereophotographs of the fundus made before and during fluorescein angiography were available for the study in some of these patients.

FINDINGS

Figures 1 through 4 depict angiographically the characteristic sequence of events which can be observed directly through the ophthalmoscope and slit-lamp following the intravenous injection of fluorescein in these patients. Leakage of dye usually appears initially within the retina in the perifoveal area in an irregular circular or wreath-like pattern (Figures 1C and 2D) and progresses both centrally and peripherally at a variable rate. As the dye converges on the foveal area, a remarkable and diagnostic geometric dark stellate figure develops centrally on the background of fluorescein staining (Figures 1F, 2F, 2E, and 4F). The outer margins of the area of fluorescence are typically irregular and splotchy. The accumulation of intraretinal fluorescein lies deep to the larger paramacular vessels, which stand out as dark lines against the background of pooled fluorescein (Figures 1E and 6C). The time required for development of the characteristic macular pattern of staining is variable. In most it is well developed in five to fifteen minutes, whereas in others, it becomes evident only after thirty minutes or more. It is important to observe

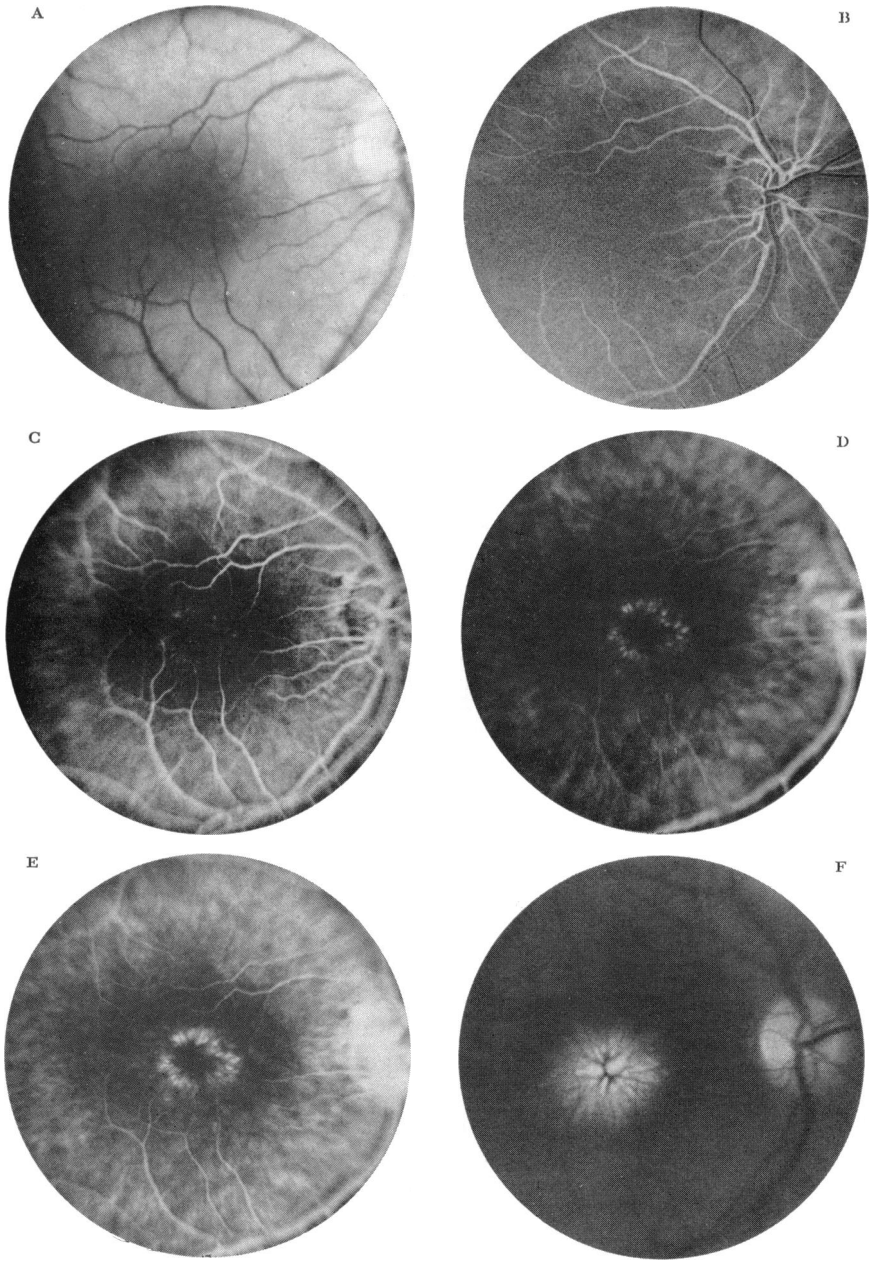

FIGURE 1. CASE 1.

A, fundus right eye, stellate change indicative of cystoid edema of macula is barely visible. B, early arteriovenous phase. C, early leakage of dye in perifoveal area, about one minute post-injection. D and E, progressive leakage of dye into macular region. F, one hour post-injection showing central dark stellate figure surrounded by fluorescein pooled with intraretinal cystoid spaces.

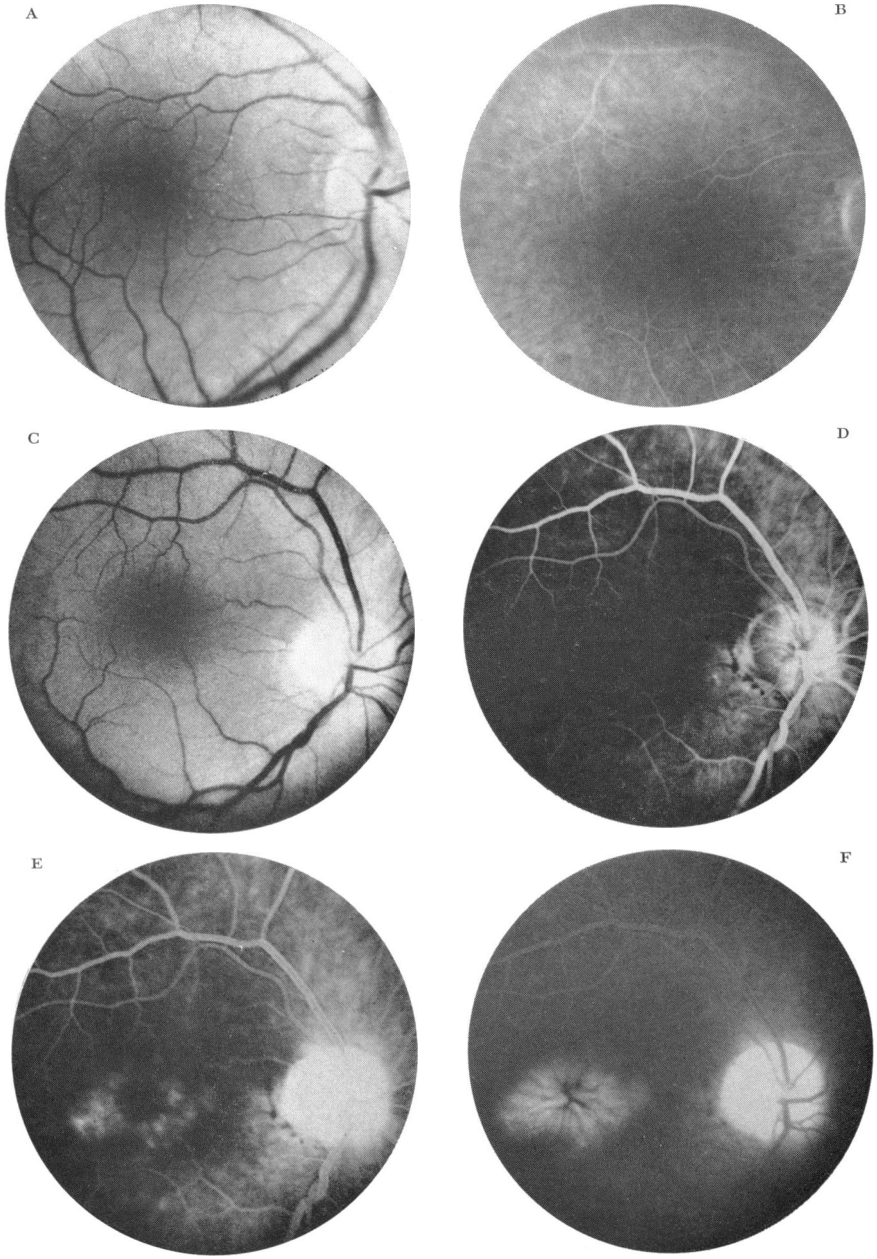

FIGURE 2. CASE 1. FLUORESCEIN STUDY RIGHT EYE FOLLOWING RESOLUTION OF MACULAR EDEMA.

A, fundus right eye; note perisistence of small subretinitic deposits on nasal side of macula. B, approximately 15 minutes post-injection showing absence of intraretinal staining. CASE 2. C, left fundus appears remarkably normal. D, venous phase. E, about two minutes post-injection. F, macular staining 45 minutes post-injection.

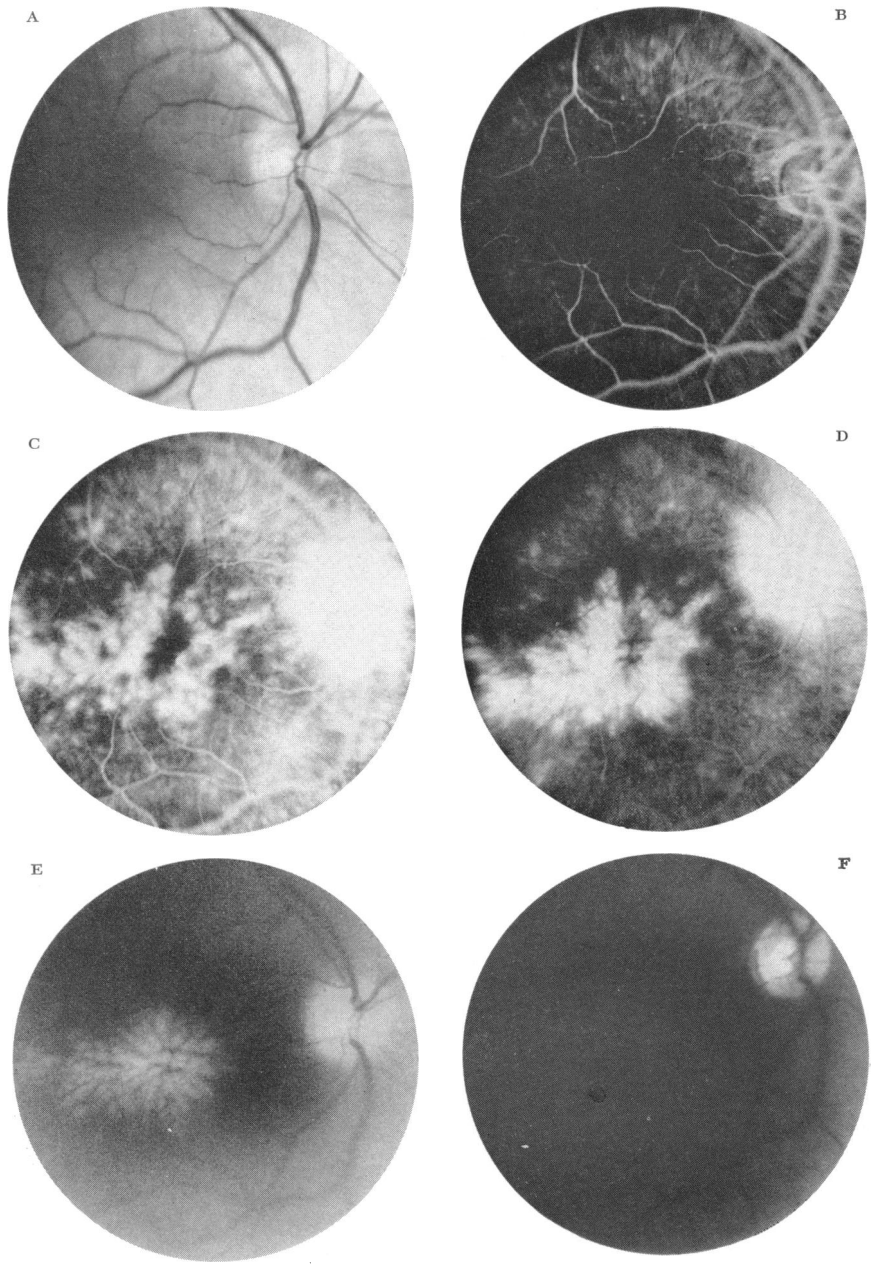

FIGURE 3. CASE 3.

A, right fundus; note peripapillary retinal folds and slight blurring of disc margins. B, early re-circulation phase. C, about five minutes post-injection; note widespread splotchy leakage of dye from posterior retina and from optic disc. D, about ten minutes post-injection. E, One hour post-injection; note apparent rapid diffusion of dye away from optic disc (compare with D and E). F, eleven weeks later; one hour post-injection photograph showing no fluorescein staining.

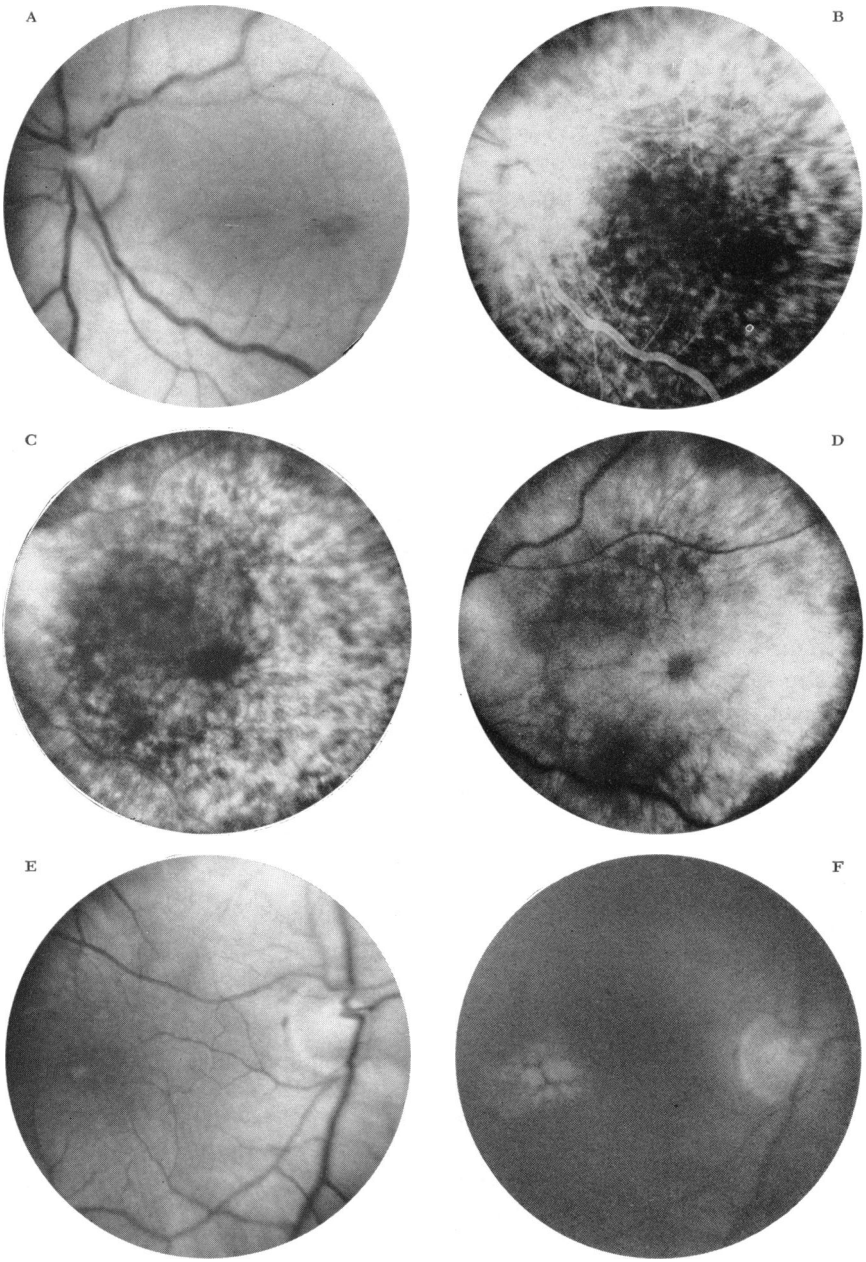

FIGURE 4. CASE 4.

A, fundus left eye; note papilledema with small hemorrhages near superior disc margin. B, one minute post-injection; note marked splotchy intraretinal fluorescein staining and leakage of dye from optic disc. C, about ten minutes post-injection. D, one hour post-injection; in this case, dye has not extended into central cystoid space. E, fundus right eye; note slightly irregular spot in foveal area. F, one hour post-injection showing pooling of dye in cystoid spaces of retina.

the patient at periodic intervals during the first fifteen minutes following injection because in some patients excessive extravasation of the dye into the vitreous and aqueous humor anteriorly may obscure the fundus details relatively early. This leakage of dye anteriorly accounts for the haze so often present in the angiograms made in the later phases of the study (Figure 3E). In some patients there may be rather widespread splotchy leakage of dye into the retina in the posterior pole of the eye (Figures 4A–4D). Leakage of dye into the optic nerve head and surrounding retina occurs in patients with papilledema (Figures 3 and 4). This is most apparent approximately ten to fifteen minutes after injection of the dye. Rapid diffusion of dye from the nerve head into the vitreous makes leakage there less apparent during the later stages of the study (compare Figures 3C and 3D with Figure 3E).

Resolution of fluorescein leakage into the retina and optic nerve generally parallels the clinical resolution of the edema of the macula and optic disc. The patient's vision and the appearance of the fundus may revert to complete normalcy and all fluorescein leakage disappear (Figures 2A, 2B, and 3F). In those patients developing permanent cystic macular changes, leakage of fluorescein into the cystic spaces may or may not be demonstrable.

The fluorescein findings in these patients, although quite characteristic, are not pathognomonic. A similar fluorescein staining pattern has been noted in a variety of ocular diseases associated with cystoid edema and degeneration of the macula, e.g., chronic chorioretinitis (Figures 5A–5C), central retinal vein occlusion, chronic papilledema, retinal vasculitis, and in the retina overlying old disciform lesions (Figures 6A–6C).

REPORT OF CASES

CASE 1

A 60-year-old Caucasian male had an uncomplicated round pupil intracapsular cataract extraction on January 12, 1965. He developed a hyphema on the third postoperative day. This cleared completely by the tenth postoperative day. By February 18, 1965, vision in the right eye was 20/30 and it remained at this level until May 5, 1965, when it decreased to 20/60. At that time, cystoid macular edema was noted for the first time. There was also a serous detachment of the macula associated with multiple yellow dot-like subretinitic precipitates. The anterior hyaloid face was intact. Some cells were present in the vitreous. He was referred to the Bascom Palmer Institute for fluorescein study which revealed leakage of dye into the intraretinal cystoid spaces (Figure 1). The patient received

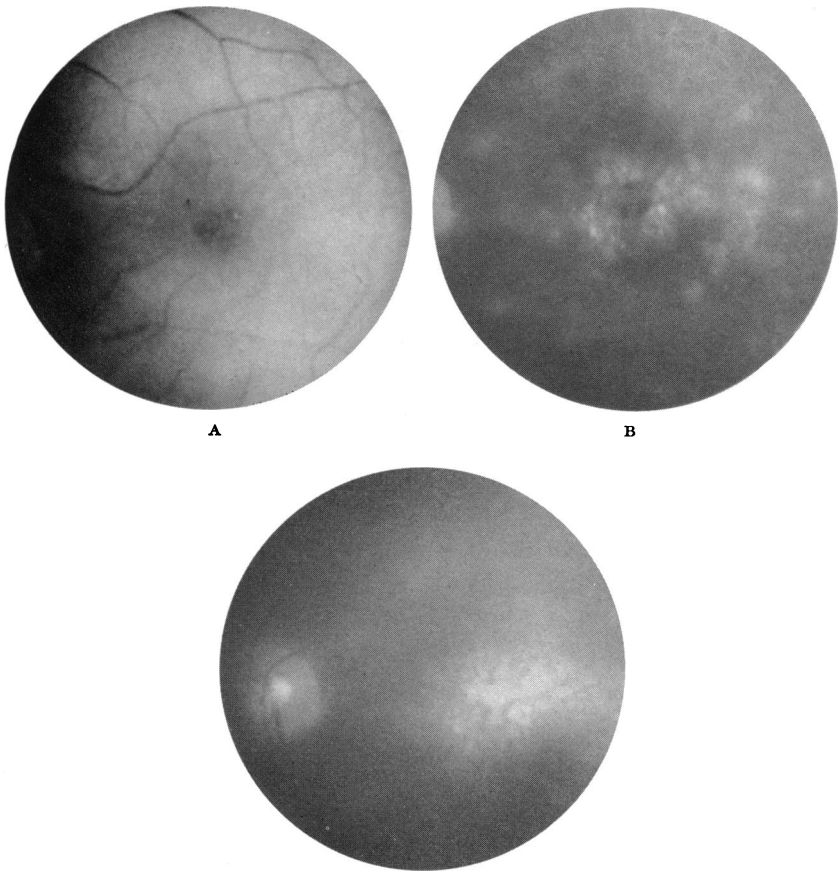

FIGURE 5

A, elderly phakic patient with cystoid macular changes secondary to long-standing smoldering peripheral chorioretinitis; vitreous reaction blurs fundus details. B, approximately 15 minutes post-injection: early filling of cystoid spaces as well as widespread and patchy intraretinal leakage of dye. C, one hour post-injection: extensive filling of intraretinal cystoid spaces. Identical findings were present in the opposite eye.

prednisone, 80 mg. daily for ten days without improvement. The cystoid macular edema persisted for six months, but in November, 1965, it began to subside and the patient's visual acuity improved to 20/30. At that time, a complete extracapsular cataract extraction was done in the left eye. The postoperative course was uneventful. By January 12, 1966, all cystoid

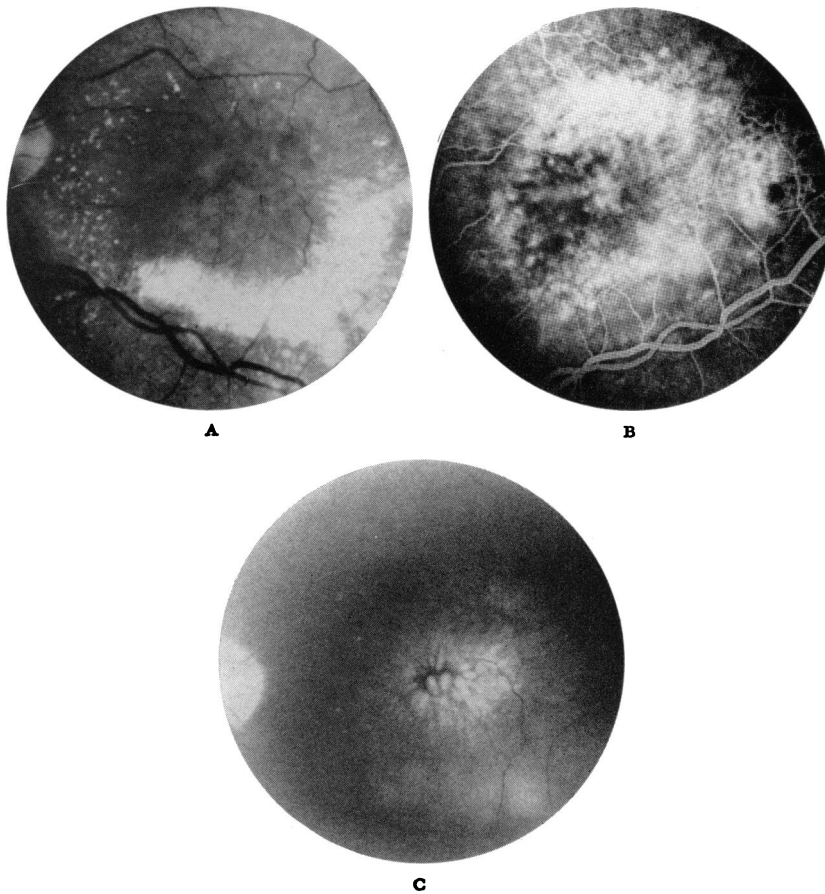

FIGURE 6

A, elderly phakic patient with cystoid macular changes secondary to prolonged serous disciform detachment of the retina and pigment epithelium. B, approximately two minutes post-injection: extensive paramacular fluorescence is primarily choroidal fluorescence transmitted through altered pigment epithelium; mottled fluorescence centrally represents early intraretinal leakage of dye. C, one hour post-injection: typical pattern of fluorescein pooling within intraretinal cystoid spaces overlying background of fluorescein-stained subretinal fluid.

edema had disappeared in the right eye and no fluorescein staining was demonstrable (Figures 2A and 2B). In the left eye, however, the patient had developed cystoid macular changes which stained prominently with fluorescein. By January 28, this had subsided and vision in the left eye was 20/20.

CASE 2

A 67-year-old Caucasian male had an uneventful round pupil intracapsular cataract extraction in the right eye on September 20, 1965. During the early postoperative period the anterior hyaloid face was intact. By the ninth postoperative day, a multilobulated mass of vitreous protruded into the anterior chamber. On October 5, 1965, visual acuity was 20/25+3. At that time the pupil was peaked and a strand of vitreous extended to the wound. The fundus was normal. On December 1, 1965, a round pupil intracapsular cataract extraction was done on the left eye and the postoperative course was virtually identical to that in the right eye. By January 8, 1966, vision in the left eye was 20/20. The anterior hyaloid face was ruptured and the pupil was peaked. The macula was normal. In the right eye, however, vision had dropped to 20/30− and cystoid edema of the macula was present. Vitreous strands could be traced into the posterior part of the eye but no attachment to the macula could be demonstrated. By January 27, 1966, vision in the right eye was 20/40−3 and in the left eye was 20/30. Bilateral cystoid macular edema was present. A moderate number of vitreous inflammatory cells and vitreous strands moved freely just anterior to the macular region in both eyes. The anterior retinal surface appeared smooth and undistorted. No attachment of the vitreous to the macula could be seen. Figures 2C–2F show the fluorescein angiographic findings in the right eye. Similar changes were present in the left eye. The patient received three weeks of systemic steroid therapy. On March 30, visual acuity was 20/20 in the right eye and 20/25 in the left eye. The right macula appeared normal. There was minimal cystoid change in the left eye. On May 6, visual acuity was 20/20 bilaterally, the fundi were normal, and no fluorescein staining was demonstrable.

CASE 3

A 51-year-old Caucasian female had an uneventful round pupil intracapsular cataract extraction in the right eye on May 6, 1965. Visual acuity was normal in the left eye. On May 25, corrected vision in the right eye was 20/25. On June 8, the corrected vision had decreased to 20/70−. Examination at that time revealed minimal conjunctival injection. The wound was well healed. The cornea was clear. The anterior chamber was deep. The pupil was round and centrally located. No cells or ray were present. The anterior hyaloid face was intact. There were scattered inflammatory cells in the posterior vitreous. The foveal depression was obliterated by the presence of multiple translucent intraretinal cystoid spaces. The surface of the thickened macula was smooth. There was no evidence of vitreous attachment to the macula or unusual light reflexes in the posterior pole. There was some edema of the optic disc associated with concentric retinal folds on the temporal side of the disc (Figure 3A). Figures 3B–3E show the fluorescein angiographic findings on June 8, 1965. The patient received a small dose of prednisone for a one-week period. The macular

edema and visual acuity improved rapidly and by August 3, 1965, visual acuity had returned to 20/15, the fundus appeared normal, and there was no fluorescein staining (Figure 3F).

CASE 4

A 48-year-old Caucasian woman with a history of essential hypertension had a round pupil intracapsular cataract extraction in the left eye on September 8, 1964, and in the right eye on October 7, 1964. She corrected to 20/15 in both eyes following surgery. In early November, 1964, she began to experience blurred vision in both eyes. Edema of the macula was noted bilaterally. The patient was treated with systemic steroids and vitamins. Vision in the right eye, reduced to 20/50 at its lowest level, soon improved. The vision in the left eye remained low. She was referred for evaluation and fluorescein study on September 21, 1965. Vision in the right eye was 20/30 and in the left eye was 20/300. Both eyes were non-inflamed. The corneas were clear. The anterior chambers were deep. The pupils were round. Transillumination revealed atrophy of the iris pigment epithelium. The iridectomies were opened. The vitreous face was intact bilaterally. Inflammatory cells were present in the vitreous of both eyes. Vitreous strands could be traced posteriorly but no attachment to the macular region could be demonstrated. Cystoid macular edema was present bilaterally. This was quite extensive in the left eye. Papilledema associated with several small hemorrhages was present in the left eye. There was extensive leakage of fluorescein into the posterior retina and optic disc in the left eye (Figure 4). Cystoid edema of the right macula was also demonstrated angiographically (Figures 4E–4F).

COMMENT

The loss of central vision in these patients has been attributed to vitreous traction on the macula,[1,2,7] iritis,[1] chorioretinal vascular instability,[3,4] choroidal vascular instability[2] hypotony,[5] papilledema,[2] and optic neuritis.[6,8] The results of this study provide evidence that the pathogenesis of the visual disturbance involves leakage of serous exudate from the capillary network within the macular portion of the retina and/or the optic nerve head. This extravasation into the macular region, which may be associated with small intraretinal hemorrhages, produces a cystoid swelling of the retina which may be completely reversible or which, if it persists, may lead to permanent cystic changes and loss of macular function. The extravasation of exudate into the optic nerve head results in the clinical picture of papilledema. This may also be only a transient phenomenon, but in some cases optic atrophy and reduction in visual acuity may ensue. We have been unable to demonstrate any fluorescein angiographic evidence of alterations in the permeability of the choriocapillaris in this syndrome.

The cause of the capillary leakage is not understood. Tolentino and Schepens[7] reported "vitreoretinal traction as the only indisputable pathogenic factor in edema of the posterior pole after cataract extraction." Our findings do not agree with theirs. We have been unable to demonstrate to our satisfaction vitreous adherence to the macular region in those patients studied with the Goldmann lens. Often the vitreous haze is such that a detailed view of the macula cannot be obtained. In many cases, however, visibility is good, and in these, vitreous strands surrounded by inflammatory cells can typically be seen moving freely immediately in front of the macular region. In a few cases, unusual light reflexes emanating from the retinal surface suggest the presence of vitreoretinal interface changes, but in most cases, the inner retinal surface appears undisturbed in the macular region. If vitreous traction were present, it seems likely that the remarkably similar and rather symmetrical configuration of the dark geometrical figure produced in these patients by fluorescein injection would be distorted. In patients, usually phakic, who have developed loss of central vision secondary to easily visible vitreous traction on the macula, we have usually been unable to demonstrate evidence of fluorescein leakage, and when leakage has been demonstrated, it has occurred in an irregular pattern and one unlike that seen in the typical patient following cataract extraction.

Although vitreous alterations following cataract extraction probably play an important role in the pathogenesis of this syndrome, we do not believe that direct traction by the vitreous on the macula and optic disc is a constant or necessarily an important factor in producing the edema of the macula and/or optic disc. This is not to deny the possible significance of vitreous abnormalities in these patients. The frequent occurrence of delayed rupture of the anterior hyaloid face with vitreous strands leading to the wound area in these patients as well as the high incidence of macular changes following vitreous loss cannot be ignored. The infrequency of this disease following other operations, e.g., retinal detachment and glaucoma procedures, further suggests that vitreous alterations following removal of the lens are of importance in the pathogenesis of this lesion.

The frequent mild irritability of the eye, the almost invariable presence of significant numbers of inflammatory cells, particularly in the posterior vitreous, and the apparent response to corticosteroids in some cases suggest that inflammation probably plays an important role in the abnormal capillary permeability.

The occurrence of similar structural and inflammatory alterations in the vitreous of many eyes following cataract extraction, the relative

infrequency of the development of edema of the macula and optic disc, and the tendency for the disease to be bilateral suggest that some inherent defect in capillary integrity may be present prior to cataract extraction in patients developing this syndrome. To date, however, we have no evidence of unusual systemic vascular disease in these patients.

While many questions regarding the etiology, pathogenesis, and treatment of this syndrome remain unanswered, the use of fluorescein has provided us with additional information concerning pathophysiological changes in the macula as well as a new and useful tool for the diagnosis of this syndrome, which undoubtedly occurs more frequently than the reported incidence of less than 2 per cent.[2,7]

SUMMARY

1. Macular edema and/or papilledema should be suspected in any postoperative cataract patient where visual acuity either fails to improve to normal or suddenly decreases following cataract extraction.

2. In the postoperative cataract patient the pattern of fluorescence following intravenous fluorescein injection is diagnostic and provides the clinician with a valuable means of detecting the lesion which may be difficult to visualize by other means.

3. Fluorescein studies demonstrate that the pathogenesis of the macular and optic nerve lesions involves leakage of fluid from the retinal and optic nerve head capillaries.

4. Although the cause of this leakage is unknown, biomicroscopic examination of these patients implicates vitreous inflammation, rather than direct vitreous traction on the macula, in the pathogenesis of this disease.

ACKNOWLEDGMENT

The authors wish to extend thanks to the many physicians who provided patients for this study, and to Mr. Johnny Justice who did the fluorescein fundus photography.

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DISCUSSION

DR. GEORGE N. WISE. I would like to thank Drs. Gass and Norton for permitting me to see their manuscript well ahead of this meeting. Our own fluorescein study of this lesion is too meager for comment except to state that in no case have we seen a choroidal leak as in central serous retinopathy.

With the contact lens and slit-lamp the macular lesion has two appearances: (1) a fuzzy, poorly outlined macular elevation with loss of the foveal reflex and (2) a more clearly seen cystic elevation of the macular area with loss of foveal reflex and a vague border which blends with more normal surrounding retina thereby differing from central serous retinopathy. Probably the turbidity of the intraretinal fluid in the first case accounts for this difference in appearance, for some lesions which appear fuzzy at first later become clearly cystic. Its honeycombed appearance is best seen by retroillumination.

Having carefully searched for it, I am in full agreement with the authors in not once having seen a vitreous traction band at the macula. Such a traction band at the macula in conjunction with posterior vitreous detachment has been postulated as a cause of this macular pathology. In almost 100 cases of fresh spontaneous posterior vitreous detachment which I have been studying over the past few years, no case has shown such a maculo-vitreous adhesion or, for that matter, any macular pathology and only one showed any pathology about the disc—a small superficial retinal hemorrhage.

There is clinical and now fluorescein evidence that this lesion is due to a retinal vascular aberration.

This honeycombed cystic change in the macula is a common lesion following obstruction of any vein draining the macular area. It has been shown by Verhoeff to be due to a collection of fluid just beneath the deep capillary bed in the internuclear layer. For some unknown reason the cystic change becomes exaggerated at the macula and may form a larger foveal cyst whose rupture gives rise to the so-called macular "hole." A lesion clinically identical to the one under discussion is more common in the venous and capillary

obstructive diseases such as Eales' disease, diabetes, and vein obstruction and is the usual lesion seen at the macula when the latter is secondarily involved in cases of chorioretinitis.

Here, in 1960, when I called attention to the similarities in some of the fundus findings in partially compensated vein obstruction and uveitis, citing the cystic macula in each and pointing out the probability of the macular lesion in uveitis being due *not* directly to the toxic action of the uveitis as then thought, but indirectly to its action in slowing the blood flow at the capillary-vein level, I was much taken to task by my discusser. I am therefore pleased to see that the authors have demonstrated by fluorescein the similarity of pattern in the lesion under discussion and the macular lesions of vein obstruction, retinal vasculitis, and chronic chorioretinitis—a pattern which incriminates the retinal perifoveal capillaries in its etiology.

There is at times a definite relation between vascular lesions of the macular and peripheral retina. Both are points of terminal circulation and I have wondered if this played any part in their associated pathology. Two of my own cases showed peripheral retinal edema and hemorrhages at the 6 o'clock position suggesting too vigorous a manipulation of counterpressure in this region at the time of lens delivery. The peripheral lesion was not recognized until after the onset of macular edema. Both areas tended to clear together. Have the authors noted any untoward peripheral retinal lesions in their Goldmann 3-mirror lens study?

Certainly no one can doubt that I have thoroughly enjoyed this beautiful presentation.

DR. JOHN C. LOCKE. Dr. Gass and Dr. Norton are to be congratulated on their excellent studies. I have been interested in the subject of macular disturbances after cataract extraction for eight years, not by fluorescent funduscopic examination, but by a technique which I have called "after-image scotometry" (A.I.S.). This is a simple but extremely sensitive test, which is specific for flat separations of the macula. Using this method we have found that such separations exist in almost all cases of so-called "macular edema" after cataract surgery.

The technique and the results were reported in detail in my thesis for this Society, three years ago (Tr. Am. Ophth. Soc., 61: 682–769, 1963). Very briefly it consists of adapting the macula for 15 seconds to the bright white light of an electric ophthalmoscope. This produces an after-image scotoma which normally disappears within two minutes. Any separation of the layer of rods and cones from the choriocapillaris causes a delay in the recovery of macular function, with an abnormal persistence of the light-induced scotoma—in all cases for longer than three minutes. The macular separation may be so slight as to be subclinical—that is, it cannot be seen by slit-lamp or ophthalmoscope and does not cause a measurable scotoma under ordinary conditions of illumination. In carrying out this test, the macular field is plotted first before and then three minutes after dazzling.

In serous detachment of the macula after cataract extraction, the persistent light-induced scotoma when plotted at this time is characteristically large (10 to 12 degrees in radius); it is round and pericentral [slide].

In a series of 92 eyes, in which this test was carried out three weeks after cataract extraction, 72 eyes or 78.3 per cent showed a positive test. Thirty-seven eyes or 50 per cent of those with positive test results became negative later—at varying times up to sixteen weeks after operation. There was a direct correlation between the age of the patient and the time at which the test results became negative. Patients in whom the test was negative three weeks after surgery were the younger ones with an average age of 54.

Thirty-five of the eyes (or 38 per cent) remained positive longer than 16 weeks. It was in this group that the vitreous adhesion syndrome described by Dr. Irvine occurred. These eyes showed vitreous degeneration on slit-lamp examination, and in all cases where it had been possible to do the test prior to operation, the test results had also been positive preoperatively. It was in this group that a sudden late decrease in central vision occasionally occurred in some eyes. These results indicate that what has generally been looked upon as a delayed onset of macular edema after cataract extraction is, in most instances, an exacerbation of a pre-existing subclinical macular disturbance, rather than a new event.

The development of uveitis in aphakic eyes, whose macular function had previously been found normal, invariably gave positive test results. The persistent after-image scotoma due to uveitis, however, was much smaller (three to five degrees in radius) than the case already described. I have not been able to postulate the reason for this interesting difference which characterizes the macular response in uveitus.

May I suggest to the authors that they consider combining this approach with their continued fluorescein studies, because of its simplicity and its sensitivity. It might help to add even more to the valuable light which their present studies are shedding on this important problem.

DR. IRA S. JONES. I would like to contribute to the discussion so that anyone who happens to read the paper by Drs. Gass and Norton will not be unaware of the contribution of Reese, Jones, and Cooper on the same subject.

I believe this excellent paper adds an important dimension to the description of this macular syndrome. I feel, however, that the paper adds more to description than it does to etiology. Nothing that was said is incompatible with that which we have said regarding the nature of this syndrome, and the only difference is in the etiology. It seems to me quite suggestive that in the fluorescein studies the abnormalities were noted around the optic disc and at the macula, areas which we feel are the site of vitreous traction or vitreous support. I would like to suggest to Drs. Gass and Norton that they continue to observe these patients carefully in the hope of finding vitreous traction or vitreous support.

DR. MICHAEL J. HOGAN. I have been interested in the relationship between the vitreous and the retina for 7 or 8 years and have published one paper on this subject. Since this publication, a considerable number of gross and histologic studies have been made on human eyes.

In a study of some 500 to 600 fairly normal eyes I have yet to encounter an adhesion between the retina and the vitreous posterior to an area about 5 mm. from the ora serrata. It would seem that if vitreous traction were of importance in the development of macular degenerations following cataract extraction, an occasional adhesion would be found between the vitreous and the retina in a normal eye or even in a diseased eye enucleated for some other purpose.

It seems difficult to believe that direct vitreous traction is important in the causation of this disease, and it appears more likely that vitreous traction through another mechanism, probably in the periphery of the eye, is more important than traction directly on the macular area. We have evidence in uveitis eyes, for example, that changes in the region of the ora serrata can lead to shrinkage of the vitreous base followed by resulting secondary changes in the posterior eye.

I have two questions to ask Dr. Gass and Dr. Norton. One is this: I am not quite clear why they mentioned the small whitish area in the fovea which was not the foveal reflex. Is this an area of leakage, or does this represent an area of intense edema? Secondly, can they, by their studies, evolve a prognosis based on the fluorescein pattern? In other words, is there a type of pattern that could be used to indicate a good prognosis, or one that would indicate a poor prognosis?

One final comment in regard to Dr. Locke's statements has to do with some work that is being done by one of our residents in regard to photostress testing, which is similar to what Dr. Locke described. He has found that in normal individuals, commencing at age 40, there is gradual deterioration of a person's ability to recover from photostress testing. It is a perfectly normal phenomenon in individuals, and it becomes worse with increasing age. This suggests that something is going on in the macular region which may predispose to this type of change following cataract surgery.

We have a paper which will be published in the coming A.M.A. meeting which would seem to explain his findings. The study suggests a histologic cause for the degeneration of the retina in elderly individuals.

DR. J. DONALD GASS. I certainly appreciate the comments of Drs. Wise, Long, Jones and Hogan.

In answer to Dr. Wise's question, we have not noted peripheral lesions of any specific type in these patients.

Dr. Locke brings up some very interesting suggestions; and as a matter of fact we plan to incorporate similar studies for macular function in our evaluation of patients with macular disease.

In regard to Dr. Jones' comment, we certainly do not know the etiology of this syndrome. I think it is interesting how different people can look at the same patients and see different things, and I believe it points up the difficulty in examination of the posterior part of the eye. We certainly feel that vitreous must play a very important role in the development of this lesion. We also admit that by a process of traction in a few cases, vitreous can cause loss of macular function. In none of our aphakic patients studied to date, however, have we been able to demonstrate vitreous traction on the macular region.

The reason I mentioned the yellow spot in the foveal area is because it can be misinterpreted as the foveal reflex in a fundus which at times may superficially appear quite normal. I do not know the nature of this spot. It may be exudate on the back of the retina in the foveal area. I have placed some stereophotographs of one of the patients whom I presented here at the back of the room. I think the yellow spot shows very nicely, and if any of you have ideas about what it is I would like to hear from you.

In regard to prognosis, it is my impression that there is really no way to gain any prognostic information from a single study or from repeated studies. It has been our experience that most of these patients over a period of several months will show a gradual decrease in the amount of fluorescein leakage which closely parallels the progressive increase in their visual acuity. Not all of these patients get better. For almost two years now, we have followed some who still show fluorescein staining and who have not improved one iota.