TOXEMIA OF PREGNANCY PIGMENT EPITHELIOPATHY MASQUERADING AS A HEREDOMACULAR DYSTROPHY*

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INTRODUCTION

SOME PATIENTS WITH TOXEMIA OF PREGNANCY DEVELOP AN EXUDATIVE RETINAL detachment caused by multifocal areas of fibrin-platelet occlusion of the choriocapillaris and infarction of the pigment epithelium. This usually occurs just prior to or immediately following delivery. It may or may not be accompanied by signs of hypertensive retinal changes. The detachment resolves spontaneously soon after delivery and return of vision is complete in most cases. Permanent and peculiar changes in the pigment epithelium usually occur and if not discovered until later may be mistaken for those caused by heredomacular dystrophies, diffuse tapetoretinal dystrophies, or other diseases.[1]

The purpose of this paper is to illustrate these pigment epithelial changes that are usually sufficiently characteristic to suggest the correct diagnosis.

CASE REPORTS

EARLY ACUTE EXUDATIVE STAGE

CASE 1

A 42-year-old Haitian woman gravida 3, para 1, was admitted to the hospital in the 35th week of gestation because of tonic and clonic seizures. Examination revealed

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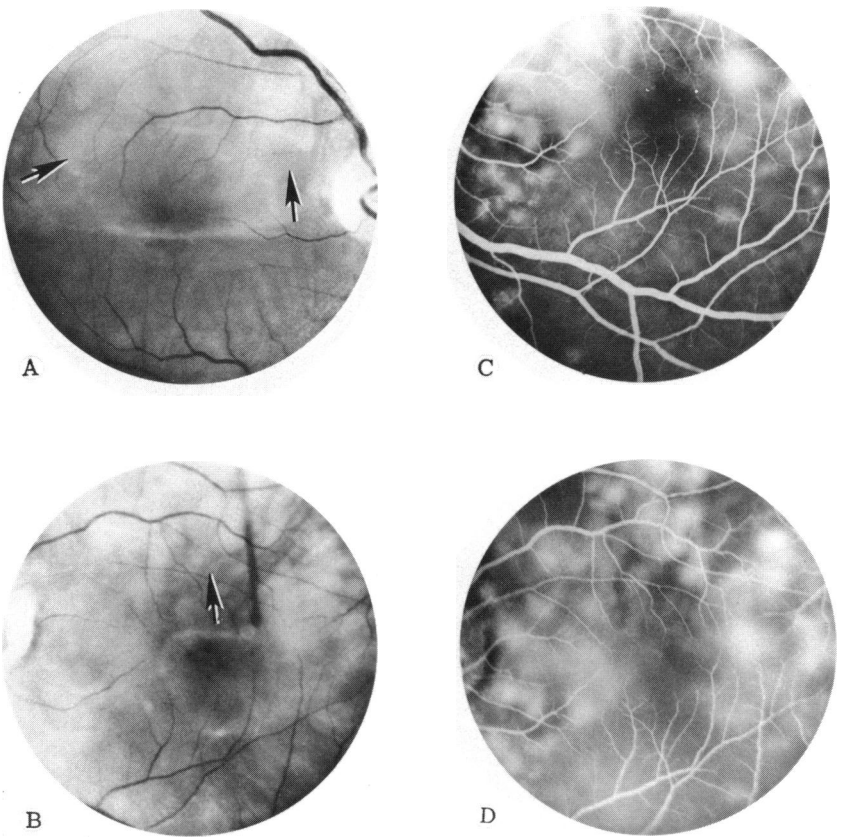

FIGURE 1
Case 1: Bullous retinal detachment in both eyes with ischemic white pigment epithelial lesions (A and B, *arrows*). Angiogram of macular region in both eyes showing multifocal areas of staining at level of pigment epithelium (C and D).

a lethargic obese woman with 2+ pitting edema below the knees and a blood pressure of 150/100 mm Hg. Urinalysis revealed 4+ proteinuria, white blood cells, red blood cells, and casts. Her platelet count was 131,000 (normal, 140,000 to 440,000), prothrombin time 10.2 (normal, < 14), partial thromboplastin time was 34 (normal, < 45), and fibrinogen was 470 mg/dl (normal, 200 to 400 mg/dl). She was treated with intravenous magnesium sulfate and her blood pressure was reduced to 135/90 mm Hg, just prior to a cesarean section. On the third postoperative day, she complained of blurred vision. Examination revealed a visual acuity

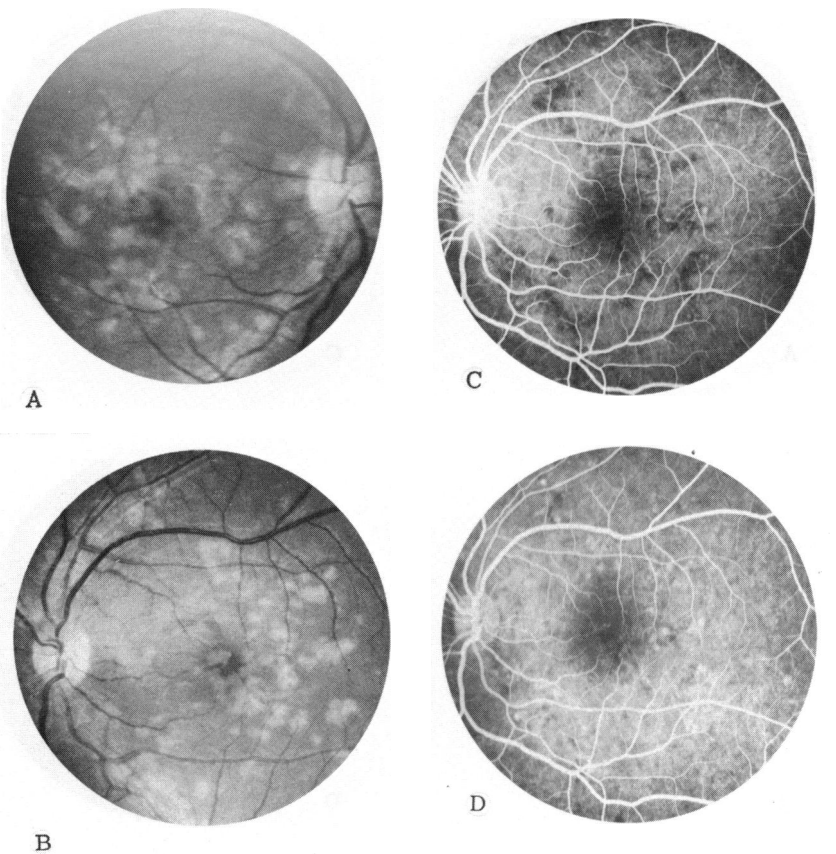

FIGURE 2
Case 2: Multifocal ischemic white pigment epithelial lesions (A and B). Note branched pattern of nonfluorescence (C) and early multifocal areas of punctate staining (D).

of 20/400 in both eyes and bullous retinal detachment with shifting subretinal fluid and multiple yellow-white patches at the level of the pigment epithelium in the posterior fundus of both eyes (Fig 1A). The anterior ocular segments were normal. The vitreous was clear. Fluorescein angiography revealed some delay in the appearance of the choroidal fluorescence, multifocal areas of staining at the level of the pigment epithelium, and late staining of the subretinal exudate (Fig 1B to D). Following discharge from the hospital, she failed to return for follow-up examination. When contacted by telephone 10 months later, she reported that

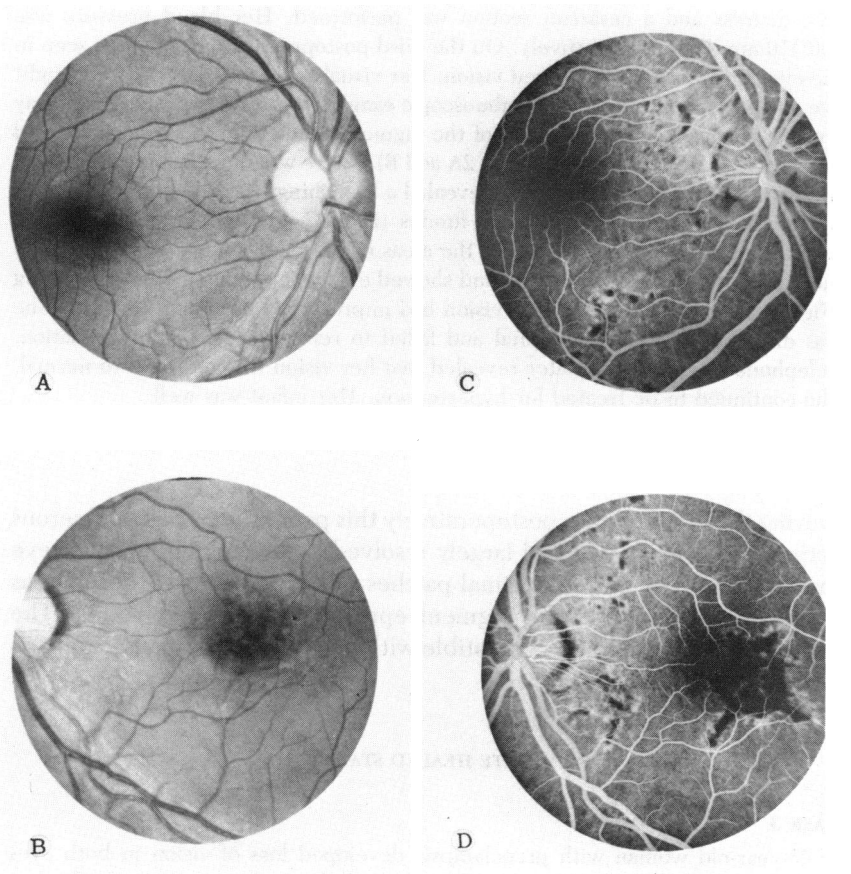

FIGURE 3
Case 3: Note branching pattern of nonfluorescent pigment clumping that is more evident angiographically than ophthalmoscopically (Elschnig's spots) (A-D).

her vision had returned soon after leaving the hospital and she refused further examination.

CASE 2

A 29-year-old black woman gravida 4, para 1, was hospitalized at 35 weeks' gestation because of chronic hypertension and superimposed preeclampsia. Her blood pressure was 192/104 mm Hg. There was 3+ pitting edema, hyperactive deep tendon reflexes, and proteinuria. Her platelet count, prothrombin time, and

partial thromboplastin time were within normal limits. There was evidence of fetal distress and a cesarean section was performed. Her blood pressure was 190/110 mm Hg postoperatively. On the third postoperative day, she was seen in the eye clinic because of blurred vision. Her visual acuity was 20/200 in the right eye and 20/70 in the left. Ophthalmoscopic examination revealed multifocal gray areas of color change at the level of the pigment epithelium in the macular and peripapillary areas in both eyes (Fig 2A and B). There was questionable subretinal exudate. Fluorescein angiography revealed a branching pattern of linear zones of hypofluorescence in the posterior fundus (Fig 2C and D). These zones that included, but were not confined to the areas of gray subretinal patches, became more fluorescent after 29 seconds and showed evidence of some punctate staining (Fig 2C and D). Two days later, vision had improved to 20/60 in both eyes. She was discharged from the hospital and failed to return for further examination. Telephone contact 1 year later revealed that her vision had returned to normal. She continued to be treated for hypertension. Her infant was well.

Comment

During the first few days postoperatively this patient may have had serous retinal detachment that had largely resolved by the time of her first eye examination. The gray subretinal patches probably represent focal areas of ischemic damage to the pigment epithelium and outer retina. The angiographic findings are compatible with patchy areas of choriocapillaris obstruction.

LATE HEALED STAGE

CASE 3

A 25-year-old woman with preeclampsia developed loss of vision in both eyes secondary to bullous detachment of the retina 2 days prior to delivery. She was moderately hypertensive but did not have convulsions. Following delivery, she experienced rapid return of vision and became normotensive. Approximately 1 month postpartum, her visual acuity was 20/20 in both eyes. She had multifocal areas of alteration in the pigment epithelium in the macular region of both eyes (Fig 3A and B). There was no exudative detachment. Angiography showed evidence of a branching pattern of linear hypofluorescent areas surrounded by evidence of depigmentation of the surrounding pigment epithelium (Fig 3C and D).

CASE 4

A 56-year-old white woman was examined by her local ophthalmologist because of asthenopia. A peculiar pattern of pigment epithelial change was noted and she was referred to the Bascom Palmer Eye Institute with the diagnosis of macular

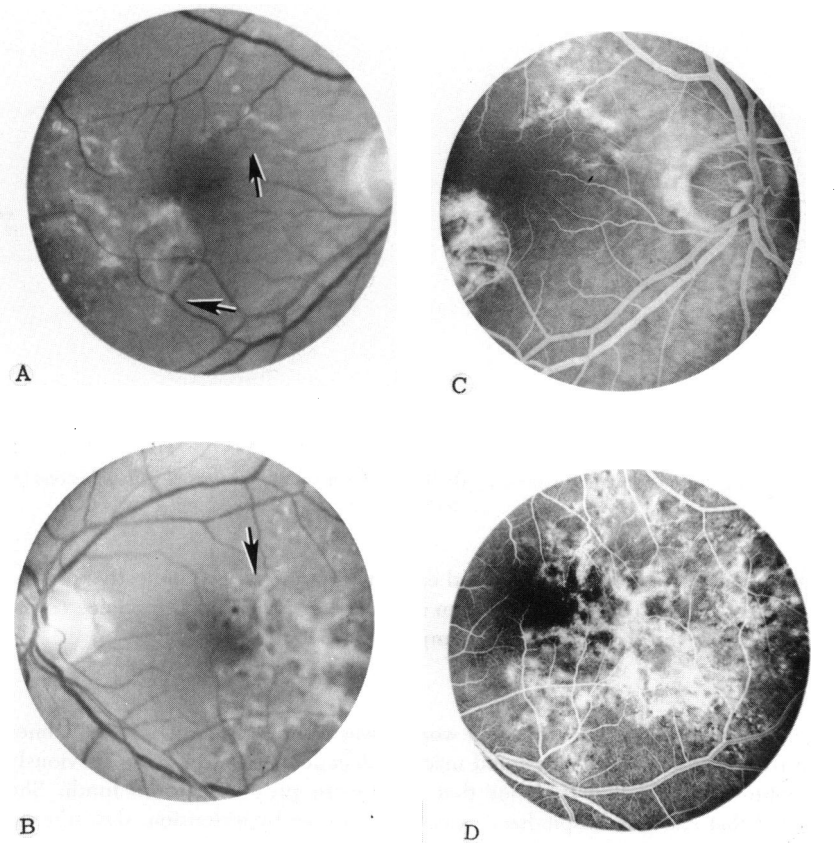

FIGURE 4
Case 4: Branching pattern of yellow lesions and pigment clumps (*arrows*) (A and B). Angiograms of same eyes (C and D).

dystrophy. She gave a history that at age 22 years she had hypertension, generalized seizures, and bilateral retinal detachment just prior to delivery of a stillborn infant. She recovered normal vision and blood pressure soon afterward. Hypertension was diagnosed when she was age 51 years. Her past medical history and family history were otherwise unremarkable. Her blood pressure was 150/80 mm Hg. Her visual acuity was 20/20 in both eyes. The positive ocular findings were confined to ophthalmoscopic examination that revealed a branching pattern of irregular linear yellow lesions and multiple focal hyperpigmented spots at the level of the pigment epithelium in the macula and justapapillary area of both eyes

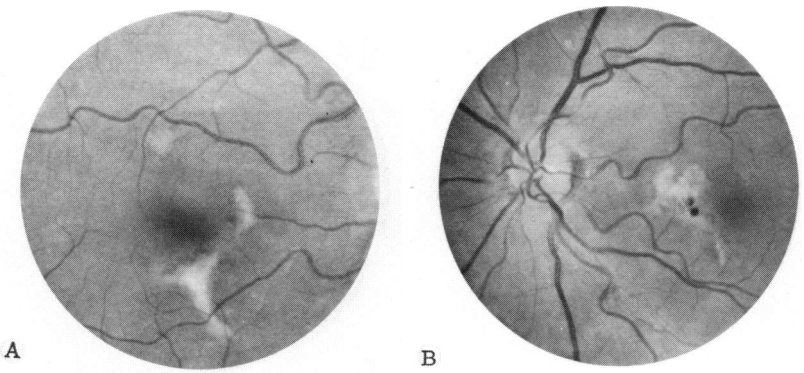

FIGURE 5
Case 5: Irregular mottling of pigment epithelium and branching pattern of yellow lesions (A and B).

(Fig 4A and B). Angiography revealed early hyperfluorescence indicative of window defects in the pigment epithelium and focal areas of nonfluorescence corresponding with the round pigment clumps (Fig 4C and D).

CASE 5

An asymptomatic 63-year-old white woman was referred to the Bascom Palmer Eye Institute because of suspected macular degeneration. Ten years previously an ophthalmologist had told her that scars were present in both fundi. She recalled that she was hospitalized because of severe hypertension, dysesthesia, and weakness just prior to the birth of her second child. She did not develop seizures and could not recall visual loss. She had been treated for hypertension, however, since that pregnancy. Her family history was negative. Her visual acuity was 20/20 in both eyes. She had a branching pattern of linear yellow lesions and some irregular areas of mottling of the pigment epithelium in the macular region of both eyes (Fig 5A and B).

Comment

Although the pattern of pigment epithelial changes is compatible with that caused by toxemia, the absence of a history of visual loss suggests that a pattern-type dystrophy of the pigment epithelium is also possible. Unfortunately, no other family members were available for examination.

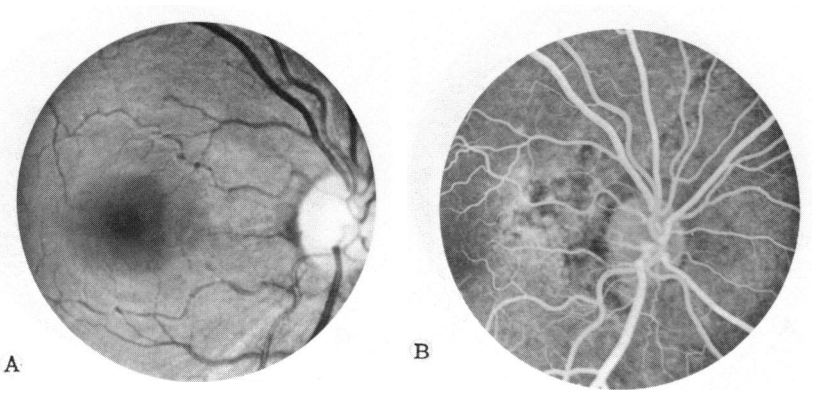

FIGURE 6
Case 6: Juxtapapillary changes in pigment epithelium seen best angiographically (A and B).

CASE 6

A 25-year-old black woman had a cesarean section at 28 weeks gestation because of severe preeclampsia. Eighteen hours postoperatively she complained of visual loss. Visual acuity in the right eye was counting fingers and in the left eye was 20/60. She had exudative detachment of the retina that was confined to the posterior fundi. Ten days later her visual acuity had improved to 20/40 in the right eye and 20/30 in the left. The subretinal exudate had resolved and the fundi were interpreted as normal (Fig 6A). Angiography revealed window defects in the pigment epithelium in the papillomacular bundle region of both eyes but no evidence of staining (Fig 6B).

Comment

This patient demonstrates the rapidity with which the integrity of the pigment epithelium is restored after delivery.

CASE 7

A 50-year-old black woman was examined for glasses. Corrected visual acuity was 20/20 in both eyes. Funduscopic changes in both eyes were interpreted as either senile macular degeneration or a patterned dystrophy of the retinal pigment epithelium. Several years later, one of us (JDMG) examined the photographs and raised the question as to whether the retinal changes might have been caused by toxemia of pregnancy. The patient was contacted and reported that she had

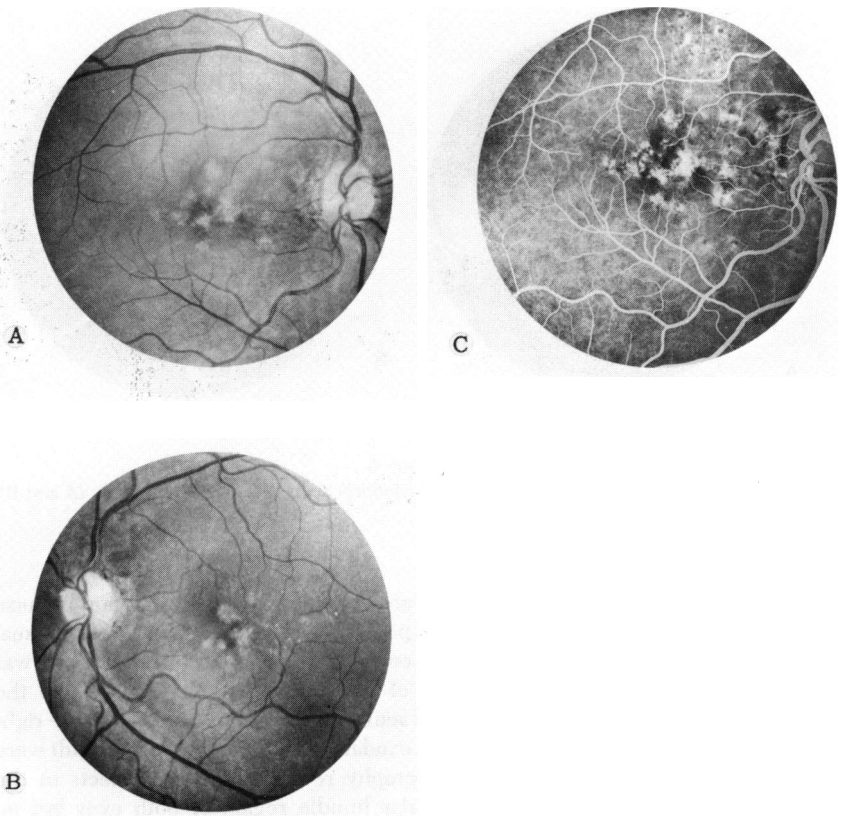

FIGURE 7
Case 7: Irregular patchy depigmented areas in both maculas (A and B). Angiogram showing hypofluorescent spots and triradiate pattern of hyperfluorescence (C).

developed high blood pressure and swelling of the extremities at the end of each of her seven pregnancies. The last two pregnancies were complicated by loss of vision in both eyes and loss of hearing just prior to delivery. Her vision slowly returned each time over a period of a few months. She continued to have chronic hypertension. Her family history was negative. The fundus photographs revealed numerous, irregular, patchy, depigmented areas measuring about ⅛ to ¼ disc diameter in size (Fig 7A and B). Five to ten patches were seen in the macula of both eyes. Fluorescein angiography showed findings very similar to those found in the patterned dystrophies of the retinal pigment epithelium. There were

several discrete, round, hypofluorescent spots at the level of the retinal pigment epithelium with a surrounding triradiate pattern of transmitted hyperfluorescence from the choroid (Fig 7C). Similar changes were present in the peripapillary areas.

CASE 8

A 37-year-old black woman was examined because of loss of vision that had occurred at age 18 years, when she was hospitalized for severe preeclampsia. She recalled severe headaches but denied seizures. Immediately after delivery she noted total blindness in both eyes. Within several months she recovered part of her vision that remained the same thereafter. Her infant died 6 days after delivery. She has continued to have chronic hypertension. She had 12 siblings and there was no family history of eye disease. Her visual acuity at the time of examination at the Bascom Palmer Eye Institute in 1974 was 20/400 in both eyes. Visual fields were constricted to approximately 10° in both eyes. Ophthalmoscopic examination revealed marked degenerative changes of the pigment epithelium throughout the eyes. There were numerous round clumps of black pigment and irregular areas of depigmentation in the macular region of both eyes (Fig 8A). There was extensive bone spicule migration of pigment into the retina peripherally (Fig 8B). Angiography revealed evidence of extensive window defects in the pigment epithelium, as well as multiple focal nonfluorescent spots corresponding with areas of pigment clumping (Fig 8C). Electroretinography showed markedly abnormal rod and cone responses. The patient was followed at intervals until 1981 with no change in the visual function or appearance of the fundi.

CASE 9

A 54-year-old Latin woman was examined because of episcleritis in the right eye. She gave a history of bilateral loss of vision and retinal detachment associated with severe preeclampsia at age 28 years. Her vision recovered within several weeks after delivery that was done by cesarean section. She continued to receive treatment for chronic hypertension. Her family history was negative. Visual acuity in both eyes was 20/25 and J-1. Ophthalmoscopic examination revealed a reticular pattern of depigmentation of the pigment epithelium in the macular region of both eyes. This was associated with a branching and linear arrangement of calcified drusen (Fig 9A). There was a large wedge-shaped area of atrophy of the pigment epithelium extending from the optic disc into the peripheral fundus inferiorly. Within this area there were round and bone-spicule clumps of pigment (Fig 9B). There was temporal pallor of the left optic disc. There was some narrowing of the retinal vessels in the wedge-shaped area of hypopigmentation inferiorly. Angiography revealed multifocal irregular areas of early hyperfluorescence, each surrounded by a round central area of nonfluorescence in the macular region bilaterally (Fig 9C). Similar round spots of nonfluorescence were apparent within a large zone of early hyperfluorescence inferior to the optic disc in both eyes.

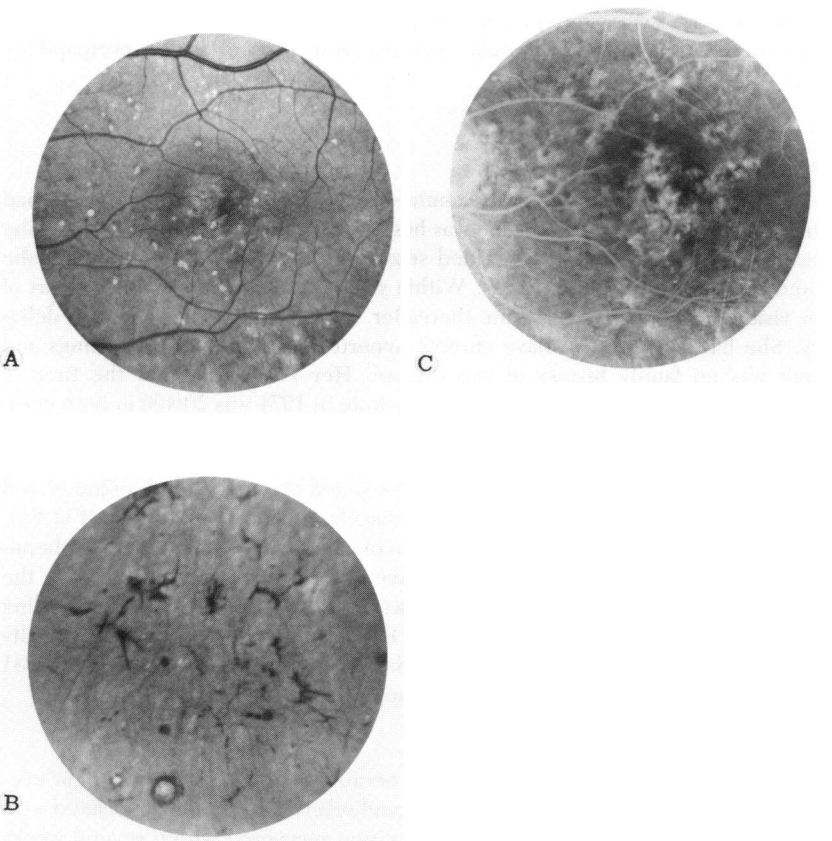

FIGURE 8
Case 8: A branching pattern of pigment epithelial changes associated with calcified drusen (A). Periphery of fundus showing narrowed retinal vessels and bone spicule pattern of pigment migration into retina (B). Angiogram showing triradiate pattern of pigment epithelial attenuation (C). Identical changes were present in other eye.

COMMENT

Toxemia of pregnancy is characterized by the development during the third trimester of hypertension, proteinuria, edema, and excessive weight gain. When severe, it may be accompanied by cerebral and visual disturbances. Signs of retinal arterial spasm (focal narrowing, cotton-wool patches, retinal hemorrhages, and optic disc edema) may occur in as high

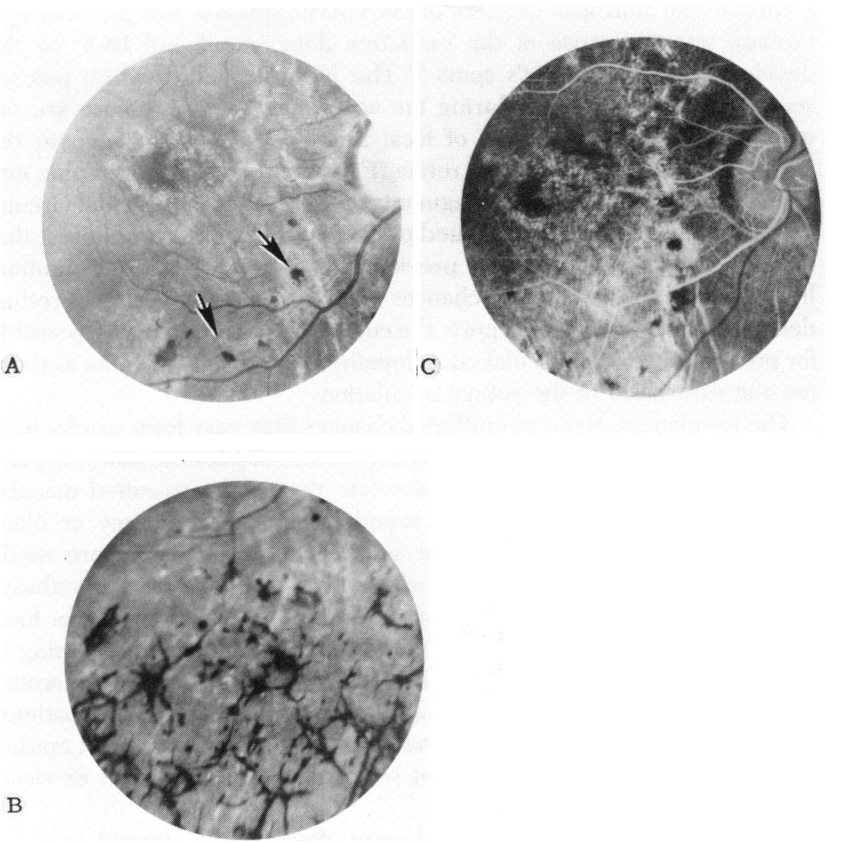

FIGURE 9
Case 9: Severe degeneration of pigment epithelium, narrowed retinal vessels, and intraretinal pigment migration. Note branched pattern of changes in papillomacular bundle region and Elschnig's spots (*arrows*) (A and B). Angiogram of same eye (C). Identical changes were present in other eye.

as 70% of patients. Only 1% to 2% of patients develop secondary retinal detachment.[2,3] This incidence rises to 10% in those patients who develop seizures (eclampsia).[3] While many early reports suggested that retinal vascular changes were the cause for the retinal detachment, Verdehame[4] and others[5-10] suggested its origin was from the choroidal vessels. After the development of fluorescein angiographic techniques, there was convincing evidence of this.[1,11-14] Histopathologic findings demonstrated

evidence that fibrinoid necrosis of the choriocapillaris and pigment epithelium was the cause of the exudative detachment and later for the development of Elschnig's spots.[15] The multiple yellow-white patches seen ophthalmoscopically during the acute stage of the disease are the ophthalmoscopic counterpart of focal areas of ischemic change to the pigment epithelium and outer retina (Figs 1 and 2).[13] Angiographic evidence of delay of perfusion of segments of the posterior choroidal circulation (Fig 2) has been demonstrated previously.[13,14,16] It is of interest that in cases 1 and 2 and in several previously reported cases, no or minimal hypertensive retinal vascular changes were present at the time of retinal detachment.[11,14,17] This supports the concept that the factors responsible for precipitating intravascular coagulopathy may not be the same as those causing vasospasm in the retinal circulation.

The permanent pigment epithelial changes may vary from patchy mottling[10,13,17-19] to an organized branching pattern of pigment clumping and yellowish discoloration that may simulate that of the so-called macular patterned dystrophies.[1,20,21] The round focal clumps of grey or black pigment (Elschnig's spots) that in some patients may be more easily detected as focal nonfluorescent spots angiographically are nearly always part of the pigment epithelial change. While not pathognomonic for focal choroidal infarction, the presence of this pattern of pigment clumping in the macula and juxtapapillary region in a female patient should arouse suspicion of prior toxemia of pregnancy. It is probable that some patients with preeclampsia develop focal areas of infarction of the pigment epithelium without developing significant serous retinal detachment or visual symptoms (case 5).

Patients with severe occlusive disease affecting the choroid may develop intrachoroidal, as well as massive subretinal hemorrhage in addition to exudation. It is probable that cases 8 and 9 had severe and extensive occlusion that produced the late ophthalmoscopic picture simulating a tapetoretinal dystrophy. The nonprogressive nature of these changes stresses the importance of not mistaking them for a dystrophy.

The predilection for fibrin-platelet intravascular coagulopathy to occur in the macular and juxtapapillary areas may be related to the rapid deceleration of blood flow as it empties from the short posterior ciliary arteries into the large sinusoidal network of the choriocapillaris.[22] Experimentally the intraarterial injection of small emboli produces multiple focal infarcts in the pigment epithelium and secondary retinal detachment that occurs primarily in the macular area.[23,24]

The histopathologic changes of fibrin-platelet occlusion of the choroidal arteries and choriocapillaris has been referred to in ocular pathology texts

as fibrinoid necrosis. It may occur as part of disseminated intravascular coagulopathy (DIC) in patients with a variety of disease states that cause unusual activation of fibrinogen. These states include in addition to toxemia, abruptio placentae, fetal demise, amniotic fluid embolization, malignant hypertension, collagen vascular diseases, tissue transplantation, idiopathic thrombocytopenic purpura, thrombotic thrombocytopenic purpura, Goodpasture's syndrome, sepsis, severe burns, drug reactions, cancer (especially leukemia), and antibody reactions.[22,25] The mechanism which causes this occlusion in toxemia is unknown. A small percentage of patients with toxemia and preeclampsia demonstrate evidence of DIC.[26-28] Some have stressed DIC as a major mechanism of disease in eclampsia,[26] where as other believe that when coagulation changes are present, that they are the result of, rather than the cause of the eclampsia.[29] The occlusion of the choroidal vessels by platelet thrombi in some patients with toxemia may be caused by increased stickiness of the capillary walls induced by the spastic arteriolar changes induced by hypertension rather than by increased levels of thromboplastin. The fact that some toxemic patients develop detachment in the presence of modest elevations of the blood pressure, however, suggests that stimulation of the fibrinogen system by release of products of conception or other mechanisms is important in the pathogenesis of choroidal vascular occlusion.[13] The findings in our case 5 suggest that toxemic patients who develop retinal detachment are prone to develop chronic hypertension.

SUMMARY

Patients with toxemia of pregnancy may develop permanent alterations of the pigment epithelium that if first discovered in later life may be mistaken for a heredomacular dystrophy, a diffuse tapetoretinal dystrophy or other diseases. These nonprogressive changes are caused by multifocal areas of fibrous platelet occlusion of the choriocapillaris that usually occur just prior to or following delivery and is usually associated with a transient period of exudative retinal detachment. The pattern of pigment epithelial derangement is often sufficiently characteristic to suggest the correct diagnosis.

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DISCUSSION

DR MELVIN L. RUBIN. In 1977, Doctor Gass, in his superb *Atlas of Macular Diseases* suggested that there was a relationship between toxemia of pregnancy and some pigmented macular changes that occur later. In today's presentation, Doctors Gass and Pautler offer additional cases to further affirm that original suggestion. The macular changes they show are found late—in the healed phase of the disease. The authors feel that the pigmentation pattern is sufficiently discrete to point to toxemia as the cause of the pigmentation and not a geographic macular degeneration. I cannot help but agree with their analysis. In this current paper, the authors point out also that similar macular changes occur in a large variety of other diseases that include a disseminated intravascular coagulopathy (DIC), which is somehow related to a hyperactivation of fibrinogen. Obviously, then, these latter diseases need to be ruled out before toxemia is implicated as the cause of unusual macular pigmentation.

Anyway, in toxemia, the retina itself may or may not be significantly involved. If it is, it may show hypertensive vascular changes, which can occur with or without prominent signs of vascular occlusion. (The retina may even show a Purtcher's type retinopathy, as Doctor Gass also has previously described.) Of importance, though, is that the retinal changes are independent of those involving the choroid, choriocapillaris, and pigment epithelium. As the authors have described, it is the latter changes that are responsible for the pigmented fundus findings in the healed stage of toxemia. What could account for the difference between involvement of retina and choroid? Perhaps the explanation relates to the fact that the retinal circulation is *auto*regulated, whereas the choroidal circulation is regulated by the sympathetics. We might conjecture that in young patients with malignant hypertension, the sympathetic mechanism is much more likely to be affected than is autoregulation.

In any case, I would like to ask the authors two questions. They have shown us clear fundus and angiographic evidence of what the acute lesions look like, as well as photographs of the late lesions. But, these were not in the same patients. Have the authors followed any individual patients long enough to see fundus changes progress from the acute stage to the late pigmented stage?

My second question is, can the authors give us any clue—any clinical sign or any laboratory test—in the acute phase that might help us predict which patients with toxemia are more likely to develop subretinal exudates and retinal detachment?

I would like to thank the authors for allowing me to review their complete paper and photographs well ahead of this meeting and appreciate the opportunity to open the discussion on this interesting paper.

DR THOMAS P. KEARNS. Doctor Gass, I too, would like to thank you for bringing this entity to the attention of the Society. I would have entitled this paper, "The Retinopathy of Toxemia Revisited." When I first went to the Mayo Clinic about 36 years ago I had the great pleasure of working with Doctor Henry Wagener who

some of the older members here will remember. One of the first things that Doctor Wagener taught me was about the retinopathy that Doctor Gass has described to us today. I recall seeing this retinopathy only once or twice at least 20 to 25 years ago and feeling very pleased in recognizing it. However, I have not seen an example of it since. I believe that the reason I have not seen it is the increasing rarity of toxemia of pregnancy. Like other diseases such as tabetic atrophy of syphilis and gonorrheal ophthalmia the toxemia of pregnancy is disappearing. I believe that Doctor Gass pointed out that toxemia is the condition in which a seizure may occur (eclampsia). The term preeclampsia is used to signify the period preceding any actual seizure activity. A patient with eclampsia may have two different types of blindness. The patient often becomes blind shortly preceding the actual seizure. This is a cortical blindness, presumably caused by cerebral edema secondary to the hypertension. The second type is the blindness secondary to a serous detachment of the retina. The retinopathy described by Doctor Gass is the residual of the serous detachment.

I do not mean to degrade Miami but you are not going to see this retinopathy in most private practices as the patients are going to receive good obstetrical care. I believe that the incidence of this retinopathy is inversely proportional to the quality of obstetrical care the patient receives. I believe that Doctor Gass will agree that the type of patients he is reporting just don't go to the physician until they are almost ready to deliver. However, this entity is still around and I am glad that Doctor Gass has brought it to our attention. I enjoyed the paper very much.

DR J. DONALD M. GASS. I would like to thank the discussors for their comments. I couldn't agree more that poor obstetrical prenatal care is responsible for virtually all of these cases. Follow-up of these patient is difficult because this socioeconomic group that seeks obstetrical care for the first time after development of toxemia are also the same ones who don't like to return to the clinic for postnatal care, much less another fluorescein angiogram. We tried repeatedly to induce the first two patients to return for reexamination. One of our patients was observed during the period with retinal detachment, as well as 1 month later after resolution of the detachment. In regard to this question, is it the hypertension only that explains the detachment. Although most toxemic patients who develop visual symptoms have significant hypertension, that is not always the case. In some patients with only modest evaluation of the blood pressure, retinal detachment may occur and may be associated with marked activation of the thromboplastin-fibrinogen system. Once this occurs the patient is probably predisposed to choroidal vascular occlusion even in the presence of minimal vascular damage caused by the hypertension. I don't think there is any reliable way to predict which of these patients with severe toxemia is going to develop retinal detachment.