Pre- and postnatal AQP4 immunolocalization in iris and ciliary epithelial cells

No significant AQP4–IR was noted in the iris or ciliary body through perinatal stages up to P0 (Fig. 4a,b; prenatal period not shown). At P7, faint AQP4–IR was first observed on NPE cells of the immature pars plicata (Fig. 4c). By P14, AQP4–IR was more intense and extensive, and AQP4–IR in NPE cells in the pars plicata was unambiguously observed (Fig. 4e). At both P7 and P14, the two layers of iris epithelial cells were unstained (Fig. 4d,f,g).

At PW8, AQP4–IR was observed throughout the pars plicata. AQP4–IR in PE cells was negligible, and only NPE cells were stained (Fig. 5a). AQP4–IR in NPE cells was observed only in the basolateral plasma membrane and not on the apical side (Fig. 5d,e) or in the pars plana (Fig. 5a). In the iris (Fig. 5a–c), only the iris posterior region continuous with NPE cells displayed weak AQP4–IR (Fig. 5a).

Pre-adsorption of anti-AQP1 or anti-AQP4 antibody with antigenic peptide in PW8 rat eyes

When the primary antibodies against AQP1 and AQP4 were pre-adsorbed with the corresponding immunogenic peptides and then applied to PW8 anterior eye samples, all AQP1–IR and AQP4–IR observed at the ciliary body, iris, and cornea was completely abolished (AQP1, Fig. 3f–i; AQP4, Fig. 5f,g). Similarly, when E18 anterior eye samples were incubated with anti-AQP1 antibody pre-adsorbed with immunogenic AQP1 peptide, AQP1–IR both at the anterior tip of the optic cup and cornea was completely abolished (not shown).

Discussion

The present immunohistochemical study has revealed that iris and ciliary epithelial cells in the adult rat eye express both AQP1 and AQP4 with characteristic distribution patterns. Whereas AQP1–IR is localized to the entire iris and anterior ciliary body, AQP4–IR is localized solely to the ciliary body. On the basis of these AQP1 and AQP4 expression patterns, iris and ciliary epithelial cells can be divided into three regions (Fig. 6): region 1, including the major part of the iris, and expressing only AQP1; region 2, including the posterior iris and anterior pars plicata, expressing both AQP1 and AQP4; and region 3, the medial and posterior pars plicata, expressing AQP4 alone. These expression patterns may offer some insights into the mechanisms controlling intraocular fluid transport. First of all, the present study has confirmed and further extended the findings of previous investigations that AQP is present on iris epithelial cells (Nielsen et al. 1993; Hamann et al. 1998). The results of our study support the idea that iris epithelial cells, in addition to ciliary epithelial cells, are
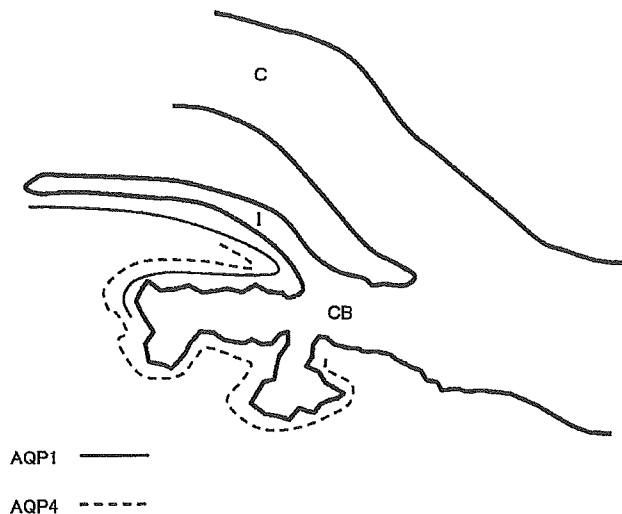
Fig. 6 Representation of expression patterns for AQP1 (*solid line*) and AQP4 (*dotted line*) in iris and ciliary epithelial cells in PW8 rat eyes. Iris and ciliary epithelia are divided into three regions depending on AQP1 and AQP4 expression patterns. Region 1 includes the major part of the iris and expresses only AQP1. Region 2 includes the posterior region of the iris and anterior pars plicata and expresses both AQP1 and AQP4. Region 3 includes the medial and posterior pars plicata and expresses AQP4 alone (*C* cornea, *CB* ciliary body, *I* iris)

involved in the secretion of the aqueous humor (Green and Pederson 1973). Since the precise role of the iris in regulating aqueous humor volume remains unclear, the present findings are expected to provide clues for further analysis of this issue. Secondly, the characteristic AQP1 and AQP4 expression patterns raise the possibility that ciliary epithelial cells in regions 2 and 3 differ with respect to their ability to secrete aqueous humor, although ciliary epithelial cells in these two regions appear morphologically indistinguishable. Studies have yet to determine the way that each of these two regions contributes to aqueous humor secretion within the ciliary body, in which aqueous humor is most actively secreted. Thirdly, no significant AQP1 or AQP4 expression has been detected in the pars plana in this study, consistent with the localization of Na–K–ATPase and Na–K–Cl–cotransporter, both of which are considered to be closely associated with aqueous humor secretion. Both Na–K–ATPase and Na–K–Cl–cotransporter are reportedly more abundant in the pars plicata than in the pars plana (Ghosh et al. 1991; Dunn et al. 2001). The present study thus provides a molecular basis for aqueous humor secretion through an analysis of AQP distribution. Fourthly, the localization of AQP1 and AQP4 also differs significantly at the cellular level. Our results for AQP1 expression on both the apical and basolateral plasma membranes of both layers of iris epithelial cells and ciliary NPE cells confirm the findings of Hamann et al. (1998). Recent studies have shown that cultured NPE cells actively transport liquid in an apical–to–basolateral direction in the absence of PE cells (Patil et al. 2001). AQP1 on both apical and basolateral plasma membranes may play important

roles in transporting water within ciliary NPE cells. In contrast, the present findings on AQP4 expression differ from those of previous studies (Hamann et al. 1998), in which AQP4 reactivity has been demonstrated on both the apical and basolateral plasma membranes of NPE cells. Although the reasons for this discrepancy remain unclear, variable expression of AQP isoforms as described above may contribute to the precise volume regulation of aqueous humor. AQP1–IR has also been found in the cornea from E15 through to adulthood. The observation of AQP1–IR at PW8 in the present study is consistent with that reported previously (Hamann et al. 1998).

The essential role of AQP molecules in regulating aqueous humor balance has been demonstrated by the generation of mice lacking AQP1 and/or AQP4 (Zhang et al. 2002). Significant decreases in IOP have been seen in these mice. Interestingly, aqueous humor secretion is significantly decreased in mice lacking AQP1 alone and also in mice lacking both AQP1 and AQP4, indicating a major role for AQP1 in fluid transport in the murine eye (Zhang et al. 2002). In contrast, humans with complete hereditary deficiency of AQP1 show no significant IOP abnormalities and instead display only a disorder in maximal urinary concentrating ability (King et al. 2001). This may be attributable to the presence of various compensatory mechanisms among AQP isoforms, such as functional redundancy, at least in the eye. Species differences may also warrant consideration.

AQP molecules in iris and ciliary epithelial cells thus appear to play important roles in regulating aqueous humor turnover and thereby in regulating IOP. In future, the manipulation of aqueous humor secretion may be achievable by regulating the kinetics of AQP molecules by using factors regulating AQP1 and AQP4 activity (Patil et al. 1997b; Han et al. 1998; Han and Patil 2000). The present study may represent a starting point for designing novel therapies to treat disorders caused by impaired IOP regulation, such as glaucoma.

The present study also examined the expression of AQP1 and AQP4 in developing rats. AQP1–IR was detected as early as E15, whereas AQP4–IR was first observed at P7. AQP4–IR density increased with development, possibly reflecting increases in the requirement of aqueous humor secretion in the area. Taken together, developing iris and ciliary epithelium expressed only AQP1 during the embryonic and early postnatal stages, but expressed both AQP1 and AQP4 after birth. AQP1 is known to be abundant in the choroid plexus throughout fetal development in the rat, indicating the presence of AQP1-mediated water transport in cerebrospinal fluid secretion during embryonic stages (Bondy et al. 1993). Similarly, AQP1 in the eye appears to play an important role in water transport during the prenatal period. Interestingly, when fold formation of the ciliary body is still immature during embryogenesis, AQP1 appears to be more abundantly expressed in the iris than in the ciliary epithelial primordium. This pattern contrasts with that in the adult eye, where the ciliary body is the dominant site of AQP expression. These results indicate that the iris epithelium is

more active in secreting aqueous humor compared with ciliary epithelium during embryonic life. Indeed, one previous study has suggested that AQP1 expression in iris epithelium is related to the regulation of aqueous humor volume (Hasegawa et al. 1994).

IOP in the early prenatal stage has been considered to be maintained primarily by the growing vitreous body (Beebe 1986). If this is really the case, active fluid transport by the ciliary epithelium may play little if any role in this regard (Beebe 1986). The present results, however, suggest that AQP1 found in the inner plate is involved in fluid secretion, even in the prenatal period. Indeed, in chicks, early secretion of aqueous humor from the embryonic stages has been demonstrated (Latker and Beebe 1984; Linser and Plunkett 1989; Reichman and Beebe 1992). Secretion of aqueous humor during the embryonic stages is further supported by experiments on mouse embryos in which the administration of carbonic anhydrase inhibitors suppressed aqueous humor secretion and caused microphthalmia (Scott et al. 1984). The presence of functional tight junctions in rat ciliary epithelium as early as E18 also supports its involvement in secretion (Arguillere et al. 1986). The findings of the present study thus provide a molecular basis for aqueous humor secretion from the iris and ciliary epithelial primordium during the prenatal period.

In conclusion, the study reported here has confirmed that AQP molecules play important roles in the regulation of aqueous humor secretion in rats, in both mature and developing eyes. These results provide a basis for understanding the molecular mechanisms underlying the regulation of normal intraocular fluid balances and offer insights into the pathophysiology of disorders involving the impairment of intraocular fluid balance.

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眼科医のための病理学

48.　前衛と後衛—未熟児網膜症の病理

小　幡　博　人[1]　平　形　明　人[2]
Alan D. Proia[3]　青　木　真　祐[4]

　未熟児網膜症 retinopathy of prematurity（以下，ROP）は，網膜血管の発達の未熟性を基盤にして血管新生や網膜剥離を起こす疾患である。出生時体重が 1,500 g 未満の極低出生体重児や在胎週数が 32 週以下の早産児に発症することが多い。一般に，出生時体重 2,500 g 未満のものを低出生体重児というが，体重が少なくても母体内で 10 カ月近く経過している場合は ROP が問題となることはない。問題となるのは，妊娠 7〜8 カ月以前に出産してしまう早産児である。近年，周産期医療の進歩によって，超低出生体重児（出生体重 1,000 g 未満）の生存率が向上しているため，ROP の治療率が増加傾向にあることが問題となっている。昨年の平岡らの論文によると，超低出生体重児 122 例中，ROP は 105 例（86.1%）に発症しており，そのうち 50 例（41.0%）にレーザー治療が施行されたとしている[1]。

　ROP の病態を理解する前に正常な網膜血管の発生を理解する必要がある。網膜血管の発生は胎生 4 カ月頃から，血管新生の原基となる細胞集団が視神経乳頭から網膜内層をはうように網膜周辺部へと広がることによって始まる[2,3]。これを vasculogenic wave という。胎生 8 カ月頃に網膜血管の発達はかなりの完成をみるが，鼻側はほぼ鋸状縁まで達するものの，耳側はまだ赤道部付近までで，鋸状縁まで達するのに胎生 10 カ月を要する。なぜならば，耳側周辺部は鼻側に比べて乳頭からの距離が遠いためである。この耳側周辺部の網膜血管の未熟性ゆえに，ROP は耳側周辺部に発生することが多い。言うまでもなく，8 カ月以前に出生すれば，鼻側やより後極側に ROP が生じることがある。ROP は網膜血管の発達が未熟な眼に発症するものであり，網膜血管の発達が成熟した眼には発症しない。

　ROP の症例の眼底写真を提示する。図 1 A は在胎 26 週で生まれた症例の stage 1（国際分類，以下同）の眼底写真である。左眼の耳側に境界線 demarcation line と広範な無血管帯が観察される。境界線は，血管のある網膜と血管のない網膜との境界である。無血管帯とは血管のない網膜であり，感覚網膜は存在している。図 1 B は在胎 30 週で出生した症例の stage 3 の眼底写真である。Ridge とよばれる隆起と，その後極側から硝子体中に立ち上がる線維血管組織がみられる。図 1 C は在胎 24 週で生まれた症例の stage 4 B の眼底写真である。左眼耳側に黄斑部を含む広範な網膜剥離が認められる。

　1) Hiroto OBATA　自治医科大学眼科学講座，講師
　2) Akito HIRAKATA　杏林大学医学部眼科学教室，教授
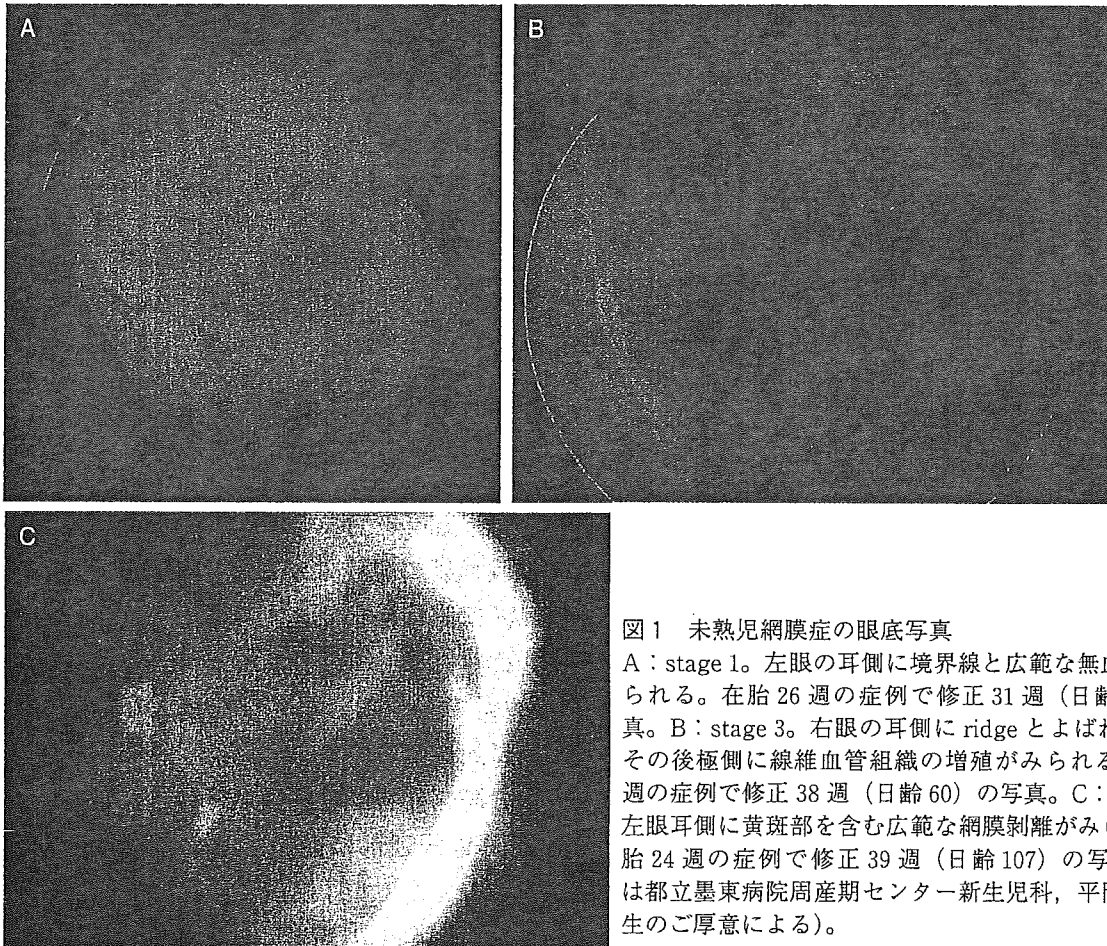　3) Alan D. PROIA　Duke 大学医学部病理学教室，教授
　4) Shinsuke AOKI　自治医科大学眼科学講座

図1　未熟児網膜症の眼底写真
A：stage 1。左眼の耳側に境界線と広範な無血管帯がみられる。在胎 26 週の症例で修正 31 週（日齢 38）の写真。B：stage 3。右眼の耳側に ridge とよばれる隆起と，その後極側に線維血管組織の増殖がみられる。在胎 30 週の症例で修正 38 週（日齢 60）の写真。C：stage 4 B。左眼耳側に黄斑部を含む広範な網膜剥離がみられる。在胎 24 週の症例で修正 39 週（日齢 107）の写真（A〜C は都立墨東病院周産期センター新生児科，平岡美依奈先生のご厚意による）。
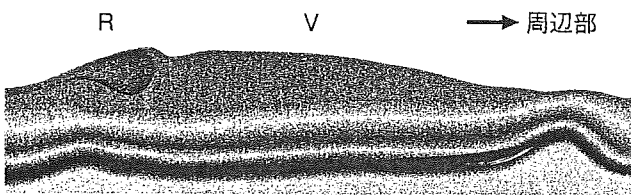

図2　網膜血管の発達における前衛と後衛のシェーマ
網膜血管の原基は網膜内層を周辺部に向かって進展する。その先端部には，前衛（V）とよばれる紡錘形細胞の集団があり，その後方に後衛（R）とよばれる原始的な血管内皮細胞の集団がある。本図は stage 2 の ridge の組織像をシェーマにしたもので前衛，後衛ともに肥厚している（文献5）を参考に作製）。
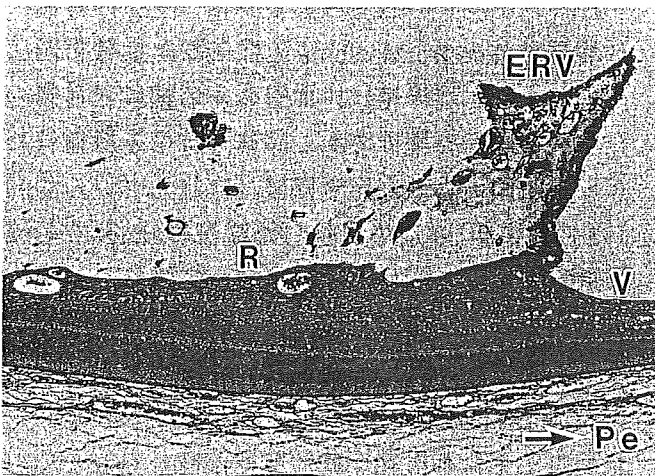

図3A　stage 3 の病理組織像（弱拡大）
前衛（V）の後極側にある後衛（R）の細胞が増殖し肥厚している。後衛から網膜外血管増殖（ERV；extraretinal vascularization）が硝子体へ立ち上がっている。Pe：網膜周辺部。HE 染色，原倍率 18 倍。

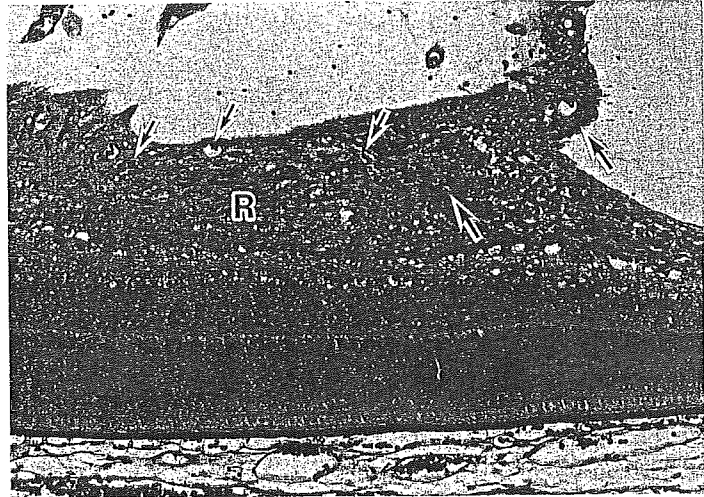図3B　stage 3 の病理組織像（強拡大）
肥厚した後衛（R）のなかに増殖する蒼白な核
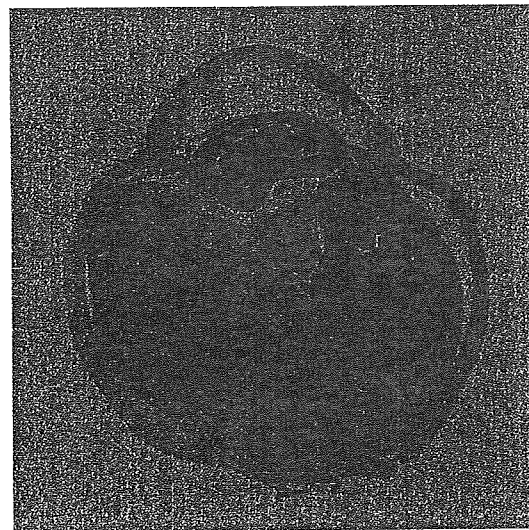をもつ細胞は血管内皮細胞である（大矢印）。
後衛のなかに新生血管がみられる（小矢印）。
HE 染色，原倍率 44 倍。

図4A　stage 5 の病理組織像（HE 染色標本のルーペ像）
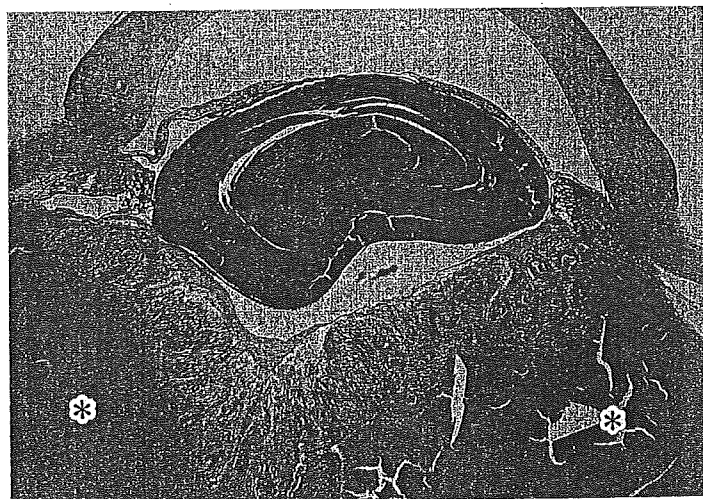網膜は全剝離し，漏斗状になっている。多量の網膜下液はエオ
ジンでピンクに染まっている。

図4B　stage 5 の病理組織像（retrolental fibro-
　　　plasia）
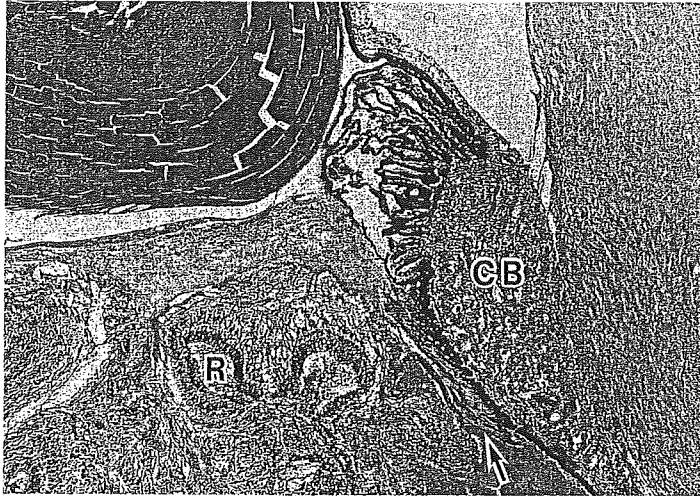水晶体後面に非常に強い線維血管増殖組織が形
成され，水晶体が前方へ偏位している。＊：網
膜下液。HE 染色，原倍率 3.12 倍。

図4C　stage 5 の病理組織像（retrolental fibro-
　　　　plasia と毛様体）
Retrolental fibroplasia は水晶体後面と毛様体
（CB）の高さに位置している。強い牽引によっ
て剥離した網膜（R）はロール状になり，毛様
体無色素上皮も剥離している（矢印）。水晶体
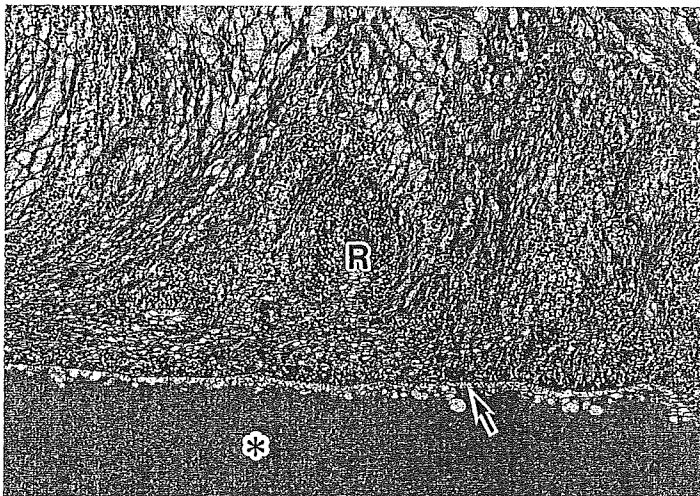後面の硝子体は寒天の断面に類似しており，ゲ
ルが凝集していることがわかる。HE 染色，原
倍率 12 倍。

図4D　stage 5 の病理組織像（剥離した網膜
　　　　と線維血管増殖）
剥離した網膜（R）は層構造が乱れ，線維血管
増殖との境界は判別が困難である。視細胞外節
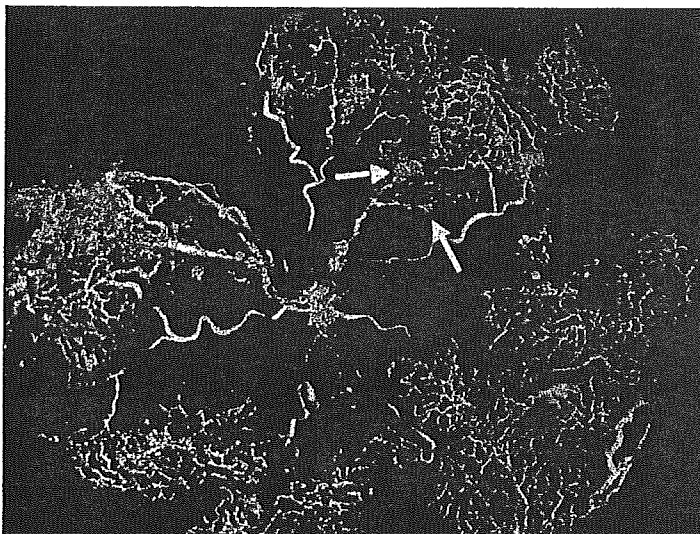は変性し消失している（矢印）。＊：網膜下液。
HE 染色，原倍率 25 倍。

図5　未熟児網膜症のマウスモデル
Fluorescein-dextran による蛍光造影であるが，
後極に無血管領域と新生血管（矢印）がみられ
る。

ROP の病理に関しては，Foos による研究が詳しい[4]~[6]。まず vasculogenic wave の先端はどうなっているのであろうか？ 組織学的に観察すると，そこには2つの細胞集団が存在する（図2）。最も先端，すなわち周辺部には前衛 vanguard とよばれる紡錘形細胞の集団があり，その後方に，後衛 rear guard とよばれる原始的な血管内皮細胞の集団がある。前衛や後衛という言葉は，テニスなどのダブルスや軍隊で用いられる言葉であるが，ROP の病態を理解する際に必要な言葉である。たとえて言うならば，前衛として紡錘形細胞の部隊がまず鋸状縁に向かって進行し，その後衛に血管内皮細胞という部隊が血管を構築する，という作業が巧みに行われている。この部隊の作業に外から種々の障害（高濃度酸素など）が加わると秩序が乱れ，血管の異常な増殖が起こる。これが ROP である。

各病期と病理組織所見を対応させて述べる。Stage 1 は境界線の形成がみられる時期であるが，組織学的に境界線は前衛における原始的な紡錘形細胞の増殖である。Stage 2 は ridge とよばれる隆起が観察される状態である。組織学的に，前衛のみならず後衛の細胞が増殖し，肥厚している状態である。Stage 3 は ridge の上あるいは後方から網膜外へ線維血管増殖がみられる状態である。組織学的に，後衛の細胞が増殖し，後衛から網膜外血管増殖（ERV；extraretinal vascularization）が硝子体中へ立ち上がっている像が観察される（図3A）。肥厚した後衛のなかに増殖する蒼白な核をもつ細胞は血管内皮細胞である（図3B）。Stage 4 は，部分的網膜剝離（あるいは限局性網膜剝離）が起こっている状態であり，4A は黄斑部が剝がれていないもの，4B は黄斑部も剝がれているものである。

Stage 5 は網膜が全剝離している状態で，剝離した網膜は漏斗型を呈する（図4A）。漏斗型の形状については前方と後方に分け，それぞれの部位で open か narrow かに分ける。網膜下には多量の漿液が存在する。水晶体後方には retrolental fibroplasia（以下，RLF）の形成がみられ，臨床的には白色瞳孔として観察される。ROP が初めて記載されたのは，1942 年 Terry が RLF として報告したのが最初とされる[7]。ROP という概念や名称が確立したのはのちになってからであり，RLF はしばらくの間 ROP の代名詞として使われていた[8]。RLF は水晶体後面に形成された非常に強い線維血管増殖組織である（図4B）。RLF は網膜周辺部における線維血管増殖に，この時期の硝子体の特性（ゲルの性質，後部硝子体剝離がない，など）という要因が重なり形成されると考えられる。水晶体は RLF に押されるように前方へ偏位し，前房が浅くなる。図4C をみると，増殖組織とその牽引によって剝離した網膜が前方へ巻き込まれてロール状になり，毛様体無色素上皮までもが剝離している。水晶体後面の硝子体は寒天の断面に類似しており，硝子体のゲルが凝集していることがわかる。剝離した網膜の視細胞外節は変性し，消失している（図4D）。剝離した網膜と網膜上の線維血管増殖組織の境界は不明瞭であり，網膜硝子体境界面の細胞反応が強いことがわかる（図4D）。これらの組織像をみていると，stage 5 の硝子体手術はいかに困難であるかが容易に想像される。たとえ手術で網膜復位を得ても網膜視機能の予測は非常に難しく，手術の難易度と手術時期が課題であることが推測される。

ROP の発症因子は多因子であるといわれているが，外的要因としては高濃度酸素投与による血管の収縮・閉塞という因子が有名である。ROP の動物モデルは，マウス，イヌ，ネコ[9]などを用いたものがあったが，新生血管の誘導と

いう点で十分なものではなかった。1994年
Smith らは，ROP のマウスモデルの論文を発
表した[10]。その方法は次のごとくである。C 57
BL/6Jマウスの子を，生後7日目から5日
間，75% の高濃度酸素下に授乳のため母親と
一緒に飼育させる。12日目に通常の room air
における飼育に戻し，17日目に眼球を摘出す
ると，後極を中心に無血管領域と新生血管が生
じる（図5）。このマウスモデルは，無血管領
域が後極にできるという点と，このまま room
air で飼育すると網膜の異常は自然に消退し，網
膜剥離は生じないという点がヒトの ROP と異
なっている。しかし，このマウスモデルを用い
て近年多くの研究が行われている[11)~13)]。

　ROP の軽症例は自然治癒するが，進行例は
適切な時期に適切な治療をしないと失明する疾
患である。本年，国際分類の改訂版が発表され[14)]，
多施設研究においてより定量的なアプローチが
期待される。今回，米国の早産児の剖検例にお
ける摘出眼球のプレパラートを多数観察する機
会を得た。いずれも 1970 年代から 1980 年代前
半にかけてのものである。どのプレパラートに
も摘出眼球の大きさなどの外観に関するデータ
と，詳細な病理組織所見がタイプされたレポー
トが存在する。その多くは stage 5 のもので
あった。この貴重な財産を数十年の時を経て，
そして海を越え，今ここに紹介することにより，
ROP は一部の眼科医が診療にあたる疾患とい
う認識を払拭できれば幸いである。

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読書評価が診断と治療に有効であった心因性視覚障害の1例

西脇　友紀[1]，田中恵津子[1]，平形　明人[1]，小田　浩一[1)2)]，気賀澤一輝[1]，樋田　哲夫[1]

[1]杏林大学医学部杏林アイセンター，[2]東京女子大学現代文化学部コミュニケーション学科

要　約

目　的：成人の心因性視覚障害への具体的な対応は難しいことが多い．読書評価を基にした対応が診断と治療に有効であった心因性視覚障害の1例を報告する．

症　例：近視性黄斑変性症の 37 歳男性．初診 3 か月後，眼底所見は不変であったが，急激な視力低下および重度求心性視野狭窄を示した．管状視野，視力検査法による視力値の乖離，読書検査および他覚検査結果，行動パターンから心因性視覚障害と診断し，眼科と精神科を併診させた．ロービジョン外来では，視機能評価，読書環境の整備，書字・パソコン訓練，社会復帰に向けた情報提供を行い，経過観察した．その中で，読書検査結果に基づく補助具の使用や読材料の選択の指導が視環境改善に有用であった．9 か月後，各種検査結果は改善，患者の再就職への積極的な姿勢もみられた．

結　論：読書検査は成人の心因性視覚障害の診断および本人の視覚障害の訴えに対する具体的対応に有用であった．(日眼会誌 109：761—765, 2005)

キーワード：心因性視覚障害，読書検査，ロービジョン

A Case Report Illustrating the Effectiveness of Reading Performance Assessments in the Diagnosis and Treatment of Psychogenic Visual Disturbances

Yuki Nishiwaki[1], Etsuko Tanaka[1], Akito Hirakata[1], Koichi Oda[1)2)]
Kazuteru Kigasawa[1] and Tetsuo Hida[1]

[1]Department of Ophthalmology, Kyorin University School of Medicine, Kyorin Eye Center
[2]Tokyo Woman's Christian University

Abstract

Purpose: An optimal strategy for treating psychogenic visual disturbances in adults has not been established. We report a patient with psychogenic visual disturbances who recovered his visual acuity and showed an improvement in his reading performance after undergoing training based on a reading performance assessment.

Case: A 37-year-old man who had been diagnosed as having myopic macular degeneration was referred to our clinic. Three months after his initial diagnosis, no changes in his fundi were observed, but his visual acuity had significantly decreased and his peripheral field of vision had become severely restricted. In view of his tunnel vision, the discrepancy among the visual acuity results obtained by different test methods, the results of a reading assessment, objective eye examination data, and his behavioral patterns, we diagnosed a psychogenic visual disturbance in the patient and referred him to an ophthalmologist and a psychiatrist for follow-up care. In our low vision clinic, we assessed his visual function, including reading performance, and developed a training program including reading, writing, and computer skills. We also provided information to help the patient find a job. The training program included instructions on how to manipulate reading aids and how to select reading materials to maximize his vision ; these instructions were effective. Nine months after his rapid decrease in visual acuity, the results of his visual function tests showed an improvement. The patient also became motivated to find a job.

Conclusion: Reading assessments are a useful tool for diagnosing psychogenic visual disturbances in adults and for coping with functional vision impairment.

Nippon Ganka Gakkai Zasshi(J Jpn Ophthalmol Soc 109：761—765, 2005)

Key words : Psychogenic visual disturbances, Reading assessment, Low vision

I　緒　　言

心因性視覚障害への対応については，これまでに多くの報告があり，学童期小児の発症要因はわかりやすい場合が多い．成人例では，発症要因が複雑あるいは不明なものが多いためか，その対応に苦慮するのが現状である．

今回，ロービジョン外来における読書評価を基にした対応が診断と治療に有用であった成人の心因性視覚障害の1例を経験したので報告する．

II　症　　例

患　者：37歳，男性．

初　診：2002年5月28日．

主　訴：右眼の変視症．

現病歴：2週間前から右眼に歪みが出現したため近医を受診したところ，近視性黄斑変性の疑いと診断され，精査目的で当科を紹介された．

既往歴：両眼高度近視．

家族歴：特記すべきことはない．

初診時所見：視力は右眼 0.01(0.3×−20.0 D◯cyl−1.0 DAx 30°)，左眼 0.01(0.3×−20.0 D)，眼圧は右眼14 mmHg，左眼 15 mmHg，両眼とも対光反射は正常，前眼部，中間透光体に異常はなかった．両眼底検査で，高度近視性の豹紋状眼底，軽度の黄斑部萎縮性変化がみられた．蛍光眼底造影検査では，脈絡膜新生血管，黄斑出血などはみられず，光干渉断層法(optical coherence tomography：以下，OCT)でも黄斑部に異常はなかった．以上の所見から，頭蓋内病変，acute idiopathic blind spot enlargement や acute zonal occult outer retinopathy(AZOOR)などの正常眼底で視野障害を呈する網脈絡膜疾患，心因性視覚障害，詐病などを鑑別疾患として考え，経過観察を行った．

III　経　　過

初診約1か月後の Goldmann 視野検査の結果を図1 A に示す．両眼とも約20度のマリオット盲点の拡大，中心比較暗点，高度近視眼にみられる軽度の求心性視野狭窄を示した．その後，経過観察を続けていたが，約3か月後に両眼の変視が悪化したと訴え診察予約日前に来院した．視力は右眼(眼前 50 cm 手動弁)，左眼(0.01)，両眼近見視力は(0.006)と急激な低下を示した．しかし，眼底検査，OCT 検査では初診時所見と変化なく，パネル D-15 テストでの色覚検査は正常範囲内，白色閃光網
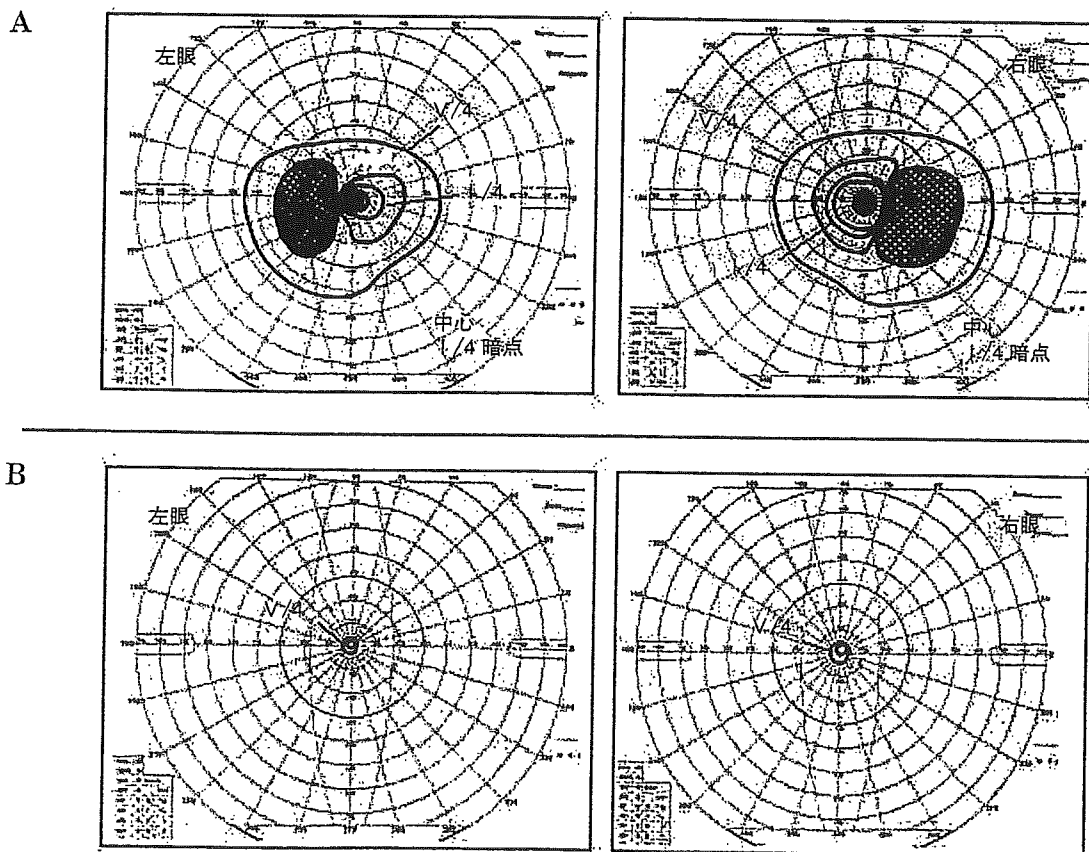

図1　Goldmann 視野検査(A：初診約1か月後，B：初診約3か月後).

A：両眼とも約20度のマリオット盲点の拡大，中心比較暗点，高度近視眼にみられる軽度求心性視野狭窄を示した．

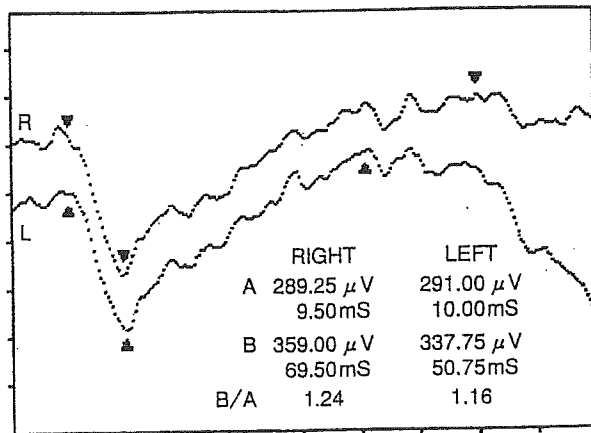B：両眼とも V/4 イソプターが5度以内と眼底所見とは異なる重度求心性視野狭窄を示した．

図 2　白色閃光網膜電図(ERG).
a 波，b 波，op 波に明らかな異常はみられなかった.

図 3　読書検査(初診約 8 か月後の結果と予想される読
　　書プロファイル).
　　この時は時間をかけて課題文を拾い読む状態であり，
　　最大読書速度は右眼 41 文字/分(━◯━)，左眼 33 文字/
　　分(━□━)であった. 視野の形状から予想される読書プ
　　ロファイル(点線グラフ)と読書検査の結果(実線グラ
　　フ)とは異なっていた.

膜電図(electroretinogram：以下，ERG)では，a 波，b
波，op 波に明らかな異常はみられず(図 2)，頭部 mag-
netic resonance imaging(MRI)でも異常はなかった.
一方，Teller acuity cards(以下，TAC)を使った縞視
力検査では，両眼(0.13)と，遠見視力値と一致しなかっ
た. また，Goldmann 視野検査では，両眼とも V/4 イ
ソプターが 5 度以内と重度の求心性視野狭窄を示し(図
1 B)，対座法視野検査では管状視野を示した. 限界フ
リッカ値は，右眼は赤 3.3，緑 2.7，黄 2.0(Hz)，左眼
は赤 1.7，緑 1.7，黄 1.7(Hz)で，著しい低値を示した.
　診察室や検査室内における患者を観察すると，手で障
害物を探索する姿勢はとるものの検出は困難ではない様
子がみられたり，約 4 m 離れた場所で待っている家族
に向かって直進していた. これは，著しい視機能低下を
示した検査結果，あるいは逆に詐病者にみられる作為的
な行動とは一致しないものであった. また，診察時の会
話から，患者の仕事面・家庭面での問題の存在が窺われ
た. 患者からは，視覚障害の身体障害者手帳申請の希望
が出された.
　このような他覚的検査の経時的所見から，頭蓋内病変
や網脈絡膜疾患よりも心因性要素が主因であると考えら
れた. また，疾病利得と考えられる手帳申請の希望が出
されたことから詐病も鑑別となるが，他覚的検査間の乖
離と行動パターンおよび管状視野の特徴から，心因性視
覚障害と診断した. 眼科での定期的な診療を行うととも
に，当院の精神科に受診依頼した. 手帳申請は見合わ
せ，ロービジョン外来では，視力検査(ランドルト環，
TAC)，視野検査(アムスラーチャート，対座法，Gold-
mann 視野)，読書検査(MNREAD-J)により視機能を評
価し，視覚補助具の選定，社会復帰をめざしたサポート
で経過観察した.
　初診約 8 か月後には，視力は右眼(0.09)，左眼(0.06)
と改善がみられたが，視標提示後から応答までは 30 秒
以上の時間を要し，視覚障害の自覚症状の変化は聞かれ

なかった. TAC は視力検査ではないと説明して行い，
両眼縞視力は(0.25)であった. この時の読書検査の結果
を図 3 に示す. 時間をかけて課題文を拾い読む状態であ
り，最大読書速度は右眼 41 文字/分，左眼 33 文字/分で
あった. この結果は，約 5 か月前に呈した重度求心性視
野狭窄(図 1 B)の形状から予想される読書プロファイル
(図 3 内点線グラフ)と読書検査の結果(図 3 内実線グラ
フ)とは異なっており，心因性視覚障害を示唆した.
　このように，読書検査の結果は重篤な求心性視野狭窄
の結果や眼底所見と異なるものであったが，実際に記録
された結果に合わせ，拡大補助具を紹介し「拡大すれば
見える」ことを患者に確認させた. 臨界文字サイズ，す
なわち最大の速度がでる限界の文字サイズの読材料を利
用するための拡大補助具の選択や方法を指導し，「読
む」という行為に慣れるよう促した. 拡大文字で印刷さ
れている読材料は補助具なしで読めることも認知させ，
弱視者団体の機関誌や，中途視覚障害者のリハビリテー
ション体験記などを貸し出した.
　一方，社会復帰への情報提供として，リハビリテー
ションを終えた患者の就職・復職例を伝え，職業安定
所，職業訓練校，障害者対象の集団説明会，視覚障害リ
ハビリテーション施設，中途視覚障害者の復職支援団体
などの就職相談窓口への相談計画を立てるなどした.
　精神科では，一般的な心理テストは視力が低いために
行うことができなかったが，カウンセリングにより「身
体表現性障害」と診断され，定期的な通院精神療法が続
けられた.

その後，ロービジョン外来の来院頻度は2週間ないじ4週間間隔とし，読書，書字，パソコン作業を行った．具体的内容は，貸し出していた拡大補助具のチェック，筆記用具の選定，宅急便の宛名や就職申込書類の記入作業，視覚以外の感覚（聴覚・触覚）利用を中心としたパソコン作業であった．自宅での練習にはノートパソコンを貸し出した．

初診約12か月後には，9か月前に呈した著しい視機能低下と比べ，顕著な改善がみられた．視力は右眼(0.2)，左眼(0.1)と向上し，視標提示後から応答までの時間も数秒となり，格段に早くなっていた．TAC での縞視力は右眼(0.25)，左眼(0.2)であり，視力と近い結果を示した．読書検査では，回を重ねる毎に改善傾向を示していたが，この時は，最大読書速度が右眼238文字/分，左眼208文字/分と，各々初回の5.8倍，6.3倍に上昇していた(図4)．臨界フリッカー値も，右眼は赤
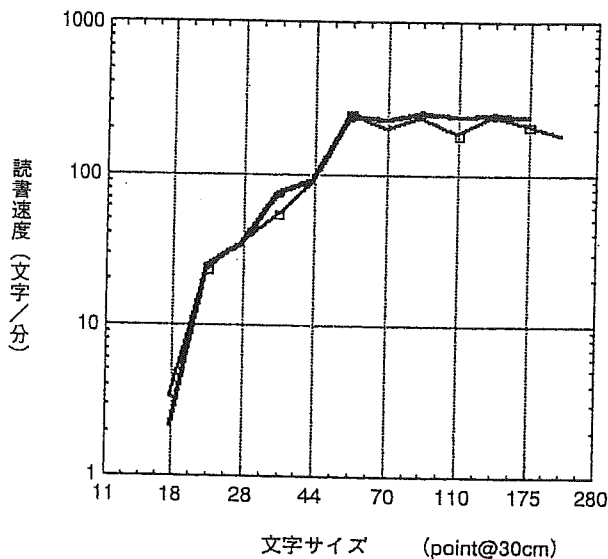
36.7，緑41，黄40(Hz)，左眼は赤30.2，緑31.5，黄34.3(Hz)と上昇し，Goldmann 視野検査の結果も，初診約1か月後の結果とほぼ同程度に改善した(図5)．再就職に対し前向きに取り組む姿勢もみられるようになり，患者自身から「一番見えにくかった半年前に比べて，比較にならないほど見やすくなり生活しやすくなった．精神的にも落ち着いている．」「ロービジョン外来は，自分の視覚障害の訴えに対し，視環境改善に具体的に対応してくれたので有効だった．」という言葉が聞かれた．

IV　考　按

心因性視覚障害の成人例の場合，特に詐盲との鑑別が困難である[1]2)．詐盲は，器質的異常がみられず疾病利得がある場合に疑われるものであり，本症例は，眼底所見は不変であるにもかかわらず，急激な視力低下を示したこと，それに相応する行動がみられなかったこと，および失職した状態で身体障害者手帳の取得希望があったことから，詐盲である可能性があった．精査の結果，やはり相応する器質的異常がみられなかったこと，心因性視覚障害に特徴的な視野であったこと[2]~7)，疾病利得(手帳申請)には拘泥せずに視環境改善に対して積極的な姿勢がみられたこと，精神科で身体表現性障害として通院精神療法が続けられたことから，詐盲ではなく心因性視覚障害であると診断された．

診断に際し有用であった検査としては，TAC と読書検査が挙げられる．心因性視覚障害における TAC の有用性については既に報告[8]~10)されているように，本症例でも遠見視力と TAC での縞視力が大きく乖離し，実際の視力はさほど低下していないことを推察させる一因となった．一方，読書検査は，従来ロービジョン患者の最多ニーズである読書のパフォーマンスを測り，拡大補助具の選定に役立つ検査である[11]~13)が，本症例では，視野検査結果と読書プロファイルの不一致から，実際の視野が狭窄していないことを推察させ，心因性視覚障害の

図4　読書検査(初診約12か月後)．
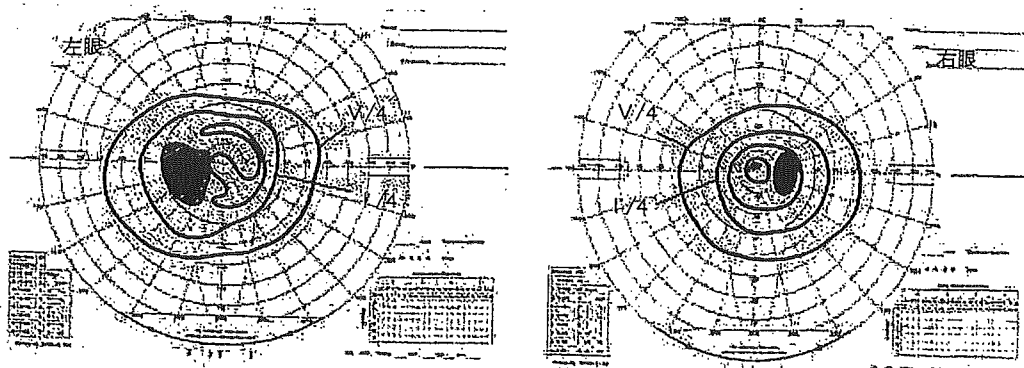最大読書速度は右眼238文字/分(●)，左眼208文字/分(□)と，各々初回の5.8倍，6.3倍に上昇していた．

図5　Goldmann 視野検査(初診約12か月後)．
両眼とも初診約3か月後に示した重度求心性視野狭窄はみられず，初診約1か月後の結果とほぼ同程度に改善を示した．

診断に役立った．通常，求心性視野狭窄でみられる読書検査の結果は狭窄した視野に収まる適切な文字サイズの場合には十分な速度で読め，文字サイズが大きくなった時に速度が落ちるという山形のものである[14]が，本症例の結果は異なっていたからである．さらに今回は，検査の学習効果が影響しない間隔で複数回読書検査をしたところ，回を重ねる毎に最大読書速度が速くなっていることがわかった．これは，本人が読書検査を通して，自己の見え方の改善を自覚しやすいという利点があった．

心因性視覚障害の原因については，眼科診察時の会話や，精神科でのカウンセリングから，家庭面・仕事面での問題の存在が窺われたが，判然としていなかった．そのため，心因性視覚障害の小児例で試みられるような対応，例えば，親子あるいは兄弟関係における問題の解決，塾通いの回数軽減，眼鏡装用への憧れに対し眼鏡処方といった解決は困難であった．本症例については，両眼約－20 D という高度近視であったことが，心因性視覚障害の背景の一因であった可能性も考えられる．9 歳以上では－8.25 D 以上の屈折度，矯正視力 0.6 以下の視力を病的近視とする診断基準によれば[15]，本症例は病的近視に分類される．Takashima ら[16]の病的近視患者の quality of life (QOL) 調査の結果によると，病的近視患者の多くは，将来の視力低下に対する不安を常に抱えている．本症例においても，仕事面・家庭面の問題が病的近視特有の視力低下のストレスを増悪させた可能性もある．

そのため本症例に対しては，心因性視覚障害の原因については拘泥せず，眼科・精神科で経過観察を行った．ロービジョン外来では，視機能を多面的に評価し，重篤な求心性視野狭窄の結果や眼底所見から予想される結果より悪い検査結果であっても，その結果に合わせて拡大補助具を選定した．また，その視環境を自宅でも再現できるよう，拡大補助具や，患者の臨界文字サイズに合った読材料を提供した．こうした見やすい最適条件の具体的提示と日常的な読書環境の改善は，患者が感じていた「見えない」という消極的な現実認識を「拡大すれば見える」という積極的な現実認識に変えたのかもしれない．同時に，社会復帰に向けての情報提供は，重度な視覚障害を負っても社会復帰可能であることを認識させた．患者の症状の改善は複数の要素に起因していると考えられるが，ロービジョン外来で行った読書評価を基にした，本人の視力低下の訴えに対する具体的な作業は，患者の不安を緩和し心理的抑圧から解放する一種の作業療法的な効果をもたらしたのではないかと思われた．心因性視覚障害は，身体表現性障害に含まれる精神的な疾患であるが，精神科との協調のもとで，身体科による作業療法的関与が治療効果の向上に寄与するものと考えられた．

本症例の経験から，読書評価は，拡大補助具の選定のみならず，心因性視覚障害の診断および治療に有用な場合があると考える．

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Intraocular Lens Implantation after Atopic Cataract Surgery Decreases Incidence of Postoperative Retinal Detachment

Makoto Inoue, MD,[1] Kei Shinoda, MD,[1] Susumu Ishida, MD,[1] Atsuro Uchida, MD,[1] Daijiro Kurosaka, MD,[1] Hiroshi Katsura, MD,[2] Kazuo Tsubota, MD[1]

Purpose: To investigate the efficacy of intraocular lens (IOL) implantation in reducing the incidence of postoperative retinal detachment after cataract surgery in patients with atopic dermatitis.

Design: Retrospective review.

Participants: One hundred sixty-nine eyes of 126 patients who underwent cataract surgery for atopic cataract were followed for more than 1 year. None of the eyes previously had a retinal detachment or retinal detachment surgery.

Methods: The eyes were divided into 132 eyes of 95 patients with an IOL implantation (IOL group) and 37 eyes of 31 patients without an IOL implantation (aphakia group). The postoperative visual acuity and incidence of postoperative retinal detachment were compared between the 2 groups. The effects of the location of the causative retinal breaks, intraoperative posterior capsule rupture, and postoperative posterior capsulotomy on the incidence of retinal detachments were evaluated.

Main Outcome Measures: The postoperative corrected visual acuity, incidence of postoperative retinal detachment, and influence of intraoperative posterior capsule rupture on the retinal detachment.

Results: The final visual acuity was better than or equal to 20/20 in 128 eyes (97.0%) of the IOL group and in 29 eyes (78.4%) of the aphakia group ($P = 0.0007$). Retinal detachment after an uncomplicated cataract surgery occurred in 3 eyes (2.3%) of the IOL group and in 8 eyes (25.8%) of the aphakia group ($P<0.0001$, Mantel–Cox). Two of 3 eyes (66.7%) in the IOL group and 1 of 8 eyes (16.7%) in the aphakia group that later developed a retinal detachment had an intraoperative posterior capsule rupture. Posterior capsulotomy by yttrium–aluminum–garnet laser did not seem to alter the incidence of postoperative retinal detachment in either the IOL (2.0%) or the aphakia group (25.0%).

Conclusions: The rate of postoperative retinal detachment in eyes with IOL and no intraoperative posterior capsule rupture seems to be low. Intraocular lens implantation with capsular bag fixation may reduce the incidence of postoperative retinal detachment triggered by lens surgery for atopic cataract. *Ophthalmology* 2005;112:1719–1724 © 2005 by the American Academy of Ophthalmology.

The ocular complications in patients with atopic dermatitis include keratoconjunctivitis, keratoconus, cataract, and retinal detachment. Atopic cataracts develop in approximately 12% to 24% of patients, and retinal detachment is reported in 8% of patients with atopic dermatitis predominately between the ages of 10 to 30 years.[1–3] The retinal detachments associated with atopic dermatitis are usually rhegmatogenous, and the most prominent feature is the presence of causative retinal tears in the extreme periphery near the ora serrata and in the pars plana or pars plicata of the ciliary body.[4,5] Fragility of tissues at these locations is most likely a predisposing factor, although the exact cause of the retinal detachment has not been determined. A relatively high incidence of giant breaks and proliferative vitreoretinopathy is also a characteristic of the eyes of patients with atopic dermatitis.[5,6]

Because of the high risk for retinal detachment in atopic dermatitis patients, the implantation of an intraocular lens (IOL) has tended to be avoided after cataract extraction in patients with atopic dermatitis. However, it is sometimes very difficult for atopic patients to wear contact lenses because of keratoconjunctivitis.

In some of the aphakic patients with a retinal detachment, traction on the epithelium of the ciliary body by contraction of the residual lens capsule has been suggested to play a role in the formation of retinal tears.[7] Thus, we have hypothesized that IOL implantation in the capsular bag will reduce the risk of postoperative retinal detachment by blocking the contraction of the residual lens capsule. In an

Table 1. Preoperative Conditions in the Intraocular Lens and Aphakia Groups

	Intraocular Lens Group	Aphakia Group	P Value*
Number of eyes (cases)	132 (95)	37 (31)	
Patient age (mean ± SD) (yrs)	27.3 ± 9.4	24.6 ± 7.1	0.164
Age range (yrs)	13–50	14–46	
Gender (female/male)	34/61	11/20	
Preoperative VA (log MAR) (mean ± SD)	1.08 ± 1.00	1.56 ± 1.03	0.099
High myopia (axial length >27 mm)	14	5	0.840†
Durations of follow-up (mean ± SD) (mos)	54 ± 32	105 ± 34	<0.001

SD = standard deviation; log MAR = the logarithm of the minimum angle of resolution; VA = visual acuity.
*Significance calculated by unpaired t test.
†Significance calculated by chi-square test.

earlier study with a small number of patients, none of the 36 eyes that underwent IOL implantation after cataract surgery for atopic cataracts developed a retinal detachment.[8] The purpose of this study was to investigate a larger number of eyes retrospectively to determine whether IOL implantation affected the incidence of postoperative retinal detachment and whether the presence of a posterior capsule rupture (PCR) or posterior capsulotomy seemed to alter the incidence of retinal detachment.

Patients and Methods

The medical records of 169 eyes of 126 patients, who underwent cataract surgery for atopic cataract at Keio University Hospital between 1990 and 2001 and were followed for more than 1 year, were reviewed. An IOL had been implanted in 132 eyes of 95 patients (IOL group) and had not been implanted in 37 eyes of 31 patients (aphakia group) during the cataract surgery. These 2 groups were determined consecutively, according to the change of the surgical procedure from surgery without IOL implantation in the former period (before 1992) to surgery with IOL implantation as a routine procedure in the later period (after 1992). Patients with a retinal detachment, a history of retinal reattachment surgery, incomplete in-the-bag fixation of the IOL, or lensectomy with complete removal of the lens capsule by a vitreous cutter with scleral indentation were excluded.

The postoperative corrected visual acuity, the incidence of retinal detachment, and the location of the retinal breaks were reviewed in patients with or without intraoperative PCR or posterior capsulotomy.

The cataract surgery was performed by using standard procedures and was begun by a local retrobulbar injection of 2.5 ml of 2% lidocaine, a conjunctival incision, and the creation of a corneoscleral tunnel. After anterior capsulotomy with continuous curvilinear capsulorhexis, phacoemulsification, and/or lens aspiration was performed. After cataract extraction, the fundus was examined by indirect ophthalmoscopy with scleral indentation. When breaks were found in the retina or ciliary body, cryopexy was performed.

A single-piece, polymethyl metacrylic acid lens was then implanted with complete in-the-bag fixation in the IOL group. An IOL was not implanted in the aphakia group. If a retinal detachment was identified intraoperatively, scleral buckling and/or vitrectomy were performed, but these cases were excluded from the study.

An unplanned rupture of the posterior capsule occurred intraoperatively in 6 eyes of the aphakia group and 3 eyes of the IOL group, and anterior vitrectomy was performed. In 6 eyes of the aphakia group with PCR, an IOL implantation had been planned preoperatively in 3 eyes and not planned in 3 eyes. In another 3 eyes of the IOL group with PCR, the IOLs could still be inserted with in-the-bag fixation after anterior vitrectomy.

The Mantel–Cox test and Kaplan–Meier analysis were used to evaluate the significance of the differences in the incidence of postoperative retinal detachments. The unpaired t test and chi-square test were also used for statistical analysis.

Table 2. Postoperative Vision and Retinal Detachment after Cataract Extraction in the Intraocular Lens and Aphakia Groups

	Intraocular Lens Group	Aphakia Group	P Value*
Postoperative VA (log MAR; mean ± SD)	0.05 ± 0.57	0.08 ± 0.44	0.782*
Final visual acuity 20/20 or better	128/132 (97.0%)	29/37 (78.4%)	0.0007†
Final visual acuity 20/25 or better	121/132 (91.7%)	33/37 (89.2%)	0.42†
Improved VA (eyes)	130/132 (98.5%)	36/37 (97.3%)	0.53†
Intraoperative or postoperative complications (eyes)			
Posterior capsule rupture	3/132 (2.3%)	6/37 (16.2%)	0.375†
Retinal detachment without PCR	3/129 (2.3%)	8/31 (25.8%)	<0.0001‡
Retinal detachment after PCR	2/3 (66.7%)	1/6 (16.7%)	0.114‡
Requirement of posterior capsulotomy	51/129 (39.5%)	12/31 (38.7%)	0.933†
Retinal detachment after capsulotomy	1/51 (2.0%)	3/12 (25.0%)	0.0152‡
Retinal detachment without capsulotomy	2/78 (2.6%)	5/19 (26.3%)	0.0108‡

log MAR = the logarithm of the minimum angle of resolution; SD = standard deviation; PCR = posterior capsule rupture; VA = visual acuity.
*Significance calculated by unpaired t test.
†Significance calculated by chi-square test.
‡Significance calculated by Mantel–Cox test.

Results

Preoperative Conditions

The differences in the age ($P = 0.164$) and preoperative vision of the logarithm of the minimum angle of resolution ($P = 0.099$, unpaired t test) were not significant between the IOL group and the aphakic group (Table 1). Highly myopic eyes (axial length >27 mm or postoperative correction in aphakic eyes <+6 dioptor [D]) were 14 eyes (10.6%) in the IOL group and 5 eyes (13.5%) in the aphakia group ($P = 0.840$, chi-square test). No patients in the aphakia group were implanted with IOLs, because the IOL of target power was not commercially available for being highly myopic. Postoperative correction in the aphakia group was from +4.25 D to +12.0 D. The follow-up period, however, was significantly longer in the aphakia group than in the IOL group ($P < 0.001$). The eyes excluded from this study included 16 eyes with a history of retinal reattachment, 2 eyes with incomplete in-the-bag fixation of the IOL (bag/sulcus), 5 eyes with lensectomy, and 27 eyes with a coexisting retinal detachment, for which simultaneous surgery to repair the retinal detachment was performed.

Postoperative Visual Acuity

The mean postoperative vision of the logarithm of the minimum angle of resolution was 0.05 in the IOL group and 0.08 in the aphakia group, and this difference was not significant ($P = 0.782$, unpaired t test; Table 2). The number of eyes with postoperative vision better than or equal to 20/20 was 128 of 132 eyes (97.0%) in the IOL group and 29 of 37 eyes (78.4%) in the aphakia group. This difference was highly significant ($P = 0.0007$, chi-square test). However, the incidence of postoperative vision better than 20/25 was not significantly different in the 2 groups ($P = 0.42$, chi-square test).
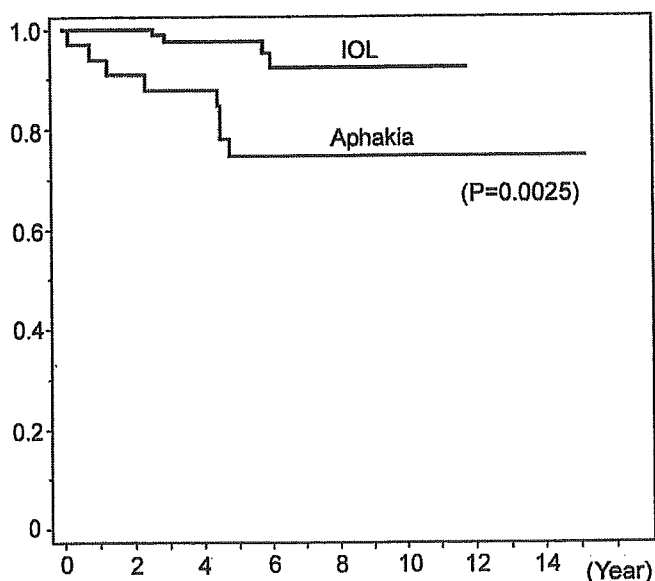

Figure 1. Incidence of postoperative retinal detachment in the intraocular lens (IOL) and aphakia group without intraoperative posterior capsule rupture. The incidence of postoperative retinal detachment was significantly lower in eyes with IOL implantation and an intact posterior capsule. IOL = IOL group; aphakia = aphakia group.
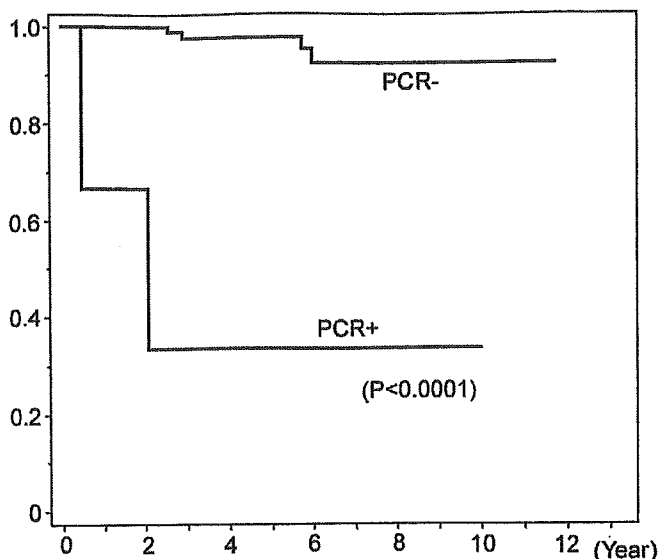

Figure 2. Incidence of postoperative retinal detachment with or without posterior capsule rupture (PCR) in the intraocular lens (IOL) group. The presence of PCR significantly increases the incidence of postoperative retinal detachment. PCR− = IOL group without PCR; PCR+ = IOL group with PCR.

Complications

The vision in 1 eye in the IOL group decreased to no light perception because of a central retinal vein occlusion and neovascular glaucoma, and another eye in the same group had vision reduced to light perception, even after successful retinal reattachment by vitreous surgery for proliferative vitreoretinopathy associated with giant breaks at the ora serrata. One case required hard contact lens correction in both eyes after IOL, because of the development of keratoconus.

A retinal detachment was the cause of deteriorated postoperative vision in the remaining cases in both groups. None of the patients in the IOL group developed an IOL luxation or subluxation. Croypexy for retinal breaks was performed intraoperatively in 7 eyes of the IOL group and 1 eye of the aphakia group. No eyes developed postoperative retinal detachment except 1 eye of the IOL group with PCR.

Influence of Posterior Capsule Rupture on Postoperative Retinal Detachment

Posterior capsule rupture occurred in 3 eyes of the IOL group (2.3%) and in 6 eyes (16.2%) of the aphakia group ($P = 0.375$, chi-square test; Table 2). The incidence of postoperative retinal detachment in the IOL group without a PCR (3/129 eyes, 2.3%) was significantly lower than in the aphakia group without a PCR (8/31 eyes, 25.8%; Fig 1; $P<0.0001$, Mantel–Cox test by Kaplan–Meier estimate).

The period when postoperative retinal detachment occurred after uncomplicated cataract surgery was 51.8 ± 21.7 months in the IOL group and 34.0 ± 23.1 months in the aphakia group ($P = 0.231$, unpaired t test). Because the aphakia group had longer follow-up allowing more time for postoperative retinal detachments to occur and to be detected, the incidence of retinal detachment was evaluated in part by reporting the rate with time. In the first year, the postoperative detachment occurred in 0/130 eyes (0%) in the IOL group and 2/33 eyes (6.1%) in the aphakia group
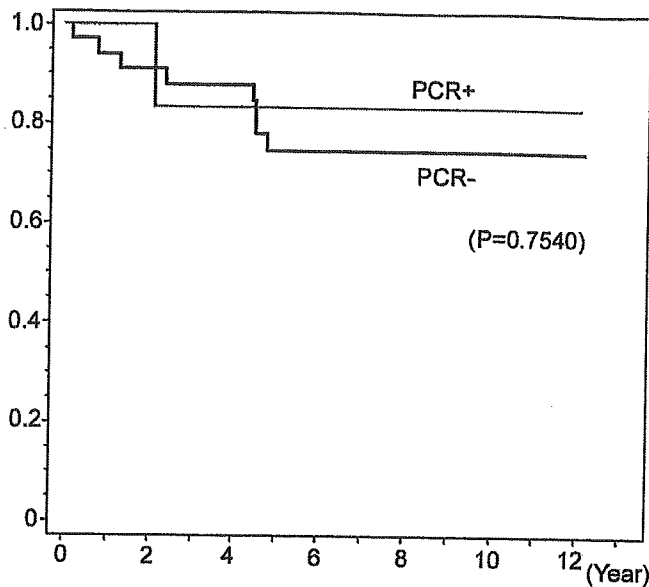
Figure 3. Incidence of postoperative retinal detachment with or without posterior capsule rupture (PCR) in the aphakia group. The presence of PCR does not increase the incidence of postoperative retinal detachment. PCR− = aphakia group without PCR; PCR+ = aphakia group with PCR.

$(P = 0.0077$, Fisher exact test), and in the second year, 0/103 eyes (0%) and 3/33 eyes $(9.1\%, P = 0.0133$, Fisher direct method), 2/79 eyes (2.5%) and 4/33 eyes (12.1%) in the third year $(P = 0.0610$, chi-square test), 2/63 eyes (3.2%) and 4/33 eyes (12.1%) in the fourth year $(P = 0.177$, chi-square test), 2/52 eyes (3.8%) and 8/30 eyes (26.7%) in the fifth year $(P = 0.0040$, chi-square test), and 3/37 eyes (8.1%) and 8/28 eyes (28.6%) in the sixth year $(P = 0.0445$, chi-square test), respectively.

Postoperative retinal detachment did not develop in highly myopic eyes of the IOL group, but in 2 eyes of the aphakia group after uncomplicated cataract surgery retinal detachments were seen. However, causative retinal breaks located at the pars plana or the ora serrata with features of retinal detachments associated with atopic dermatitis.

Although the numbers are small, the incidence of retinal detachment was higher in the IOL group with a PCR (2/3 eyes, 66.7%) than in the aphakia group with a PCR (1/6 eyes, 16.7%; $P = 0.114$, Mantel–Cox test by the Kaplan–Meier estimate). The incidence of retinal detachment in the IOL group without a PCR (3/129 eyes, 2.3%) was significantly lower than in the IOL group with a PCR (2/3 eyes, 66.7%; Fig 2; $P<0.0001$, Mantel–Cox test by the Kaplan–Meier estimate), but the incidence was not significantly different from the aphakia group with a PCR (1/6 eyes, 16.7%) and without a PCR (8/31 eyes, 25.8%; Fig 3; $P = 0.754$, Mantel–Cox test by the Kaplan–Meier estimate).

Influence of Posterior Capsulotomy on Postoperative Retinal Detachment

Posterior capsulotomy was performed by yttrium–aluminum–garnet laser in 51 eyes (39.5%) of the IOL group and in 12 eyes (38.7%) of the aphakia group $(P = 0.933$, chi-square test). The incidence of retinal detachment after posterior capsulotomy was significantly higher in the aphakia group (3/12 eyes, 25.0%) than in the IOL group (1/51 eyes, 2.0%; $P = 0.0152$, Mantel–Cox test by the Kaplan–Meier estimate), and the incidence of retinal detachment in cases without capsulotomy was also significantly higher in the aphakia group (5/19 eyes, 26.3%), than in the IOL group (2/78 eyes, 2.6%; $P = 0.0108$, Mantel–Cox test by the Kaplan–Meier estimate). In the IOL group, the incidence of retinal detachment was similar in patients with and without posterior capsulotomy $(P = 0.390$, Mantel–Cox test by the Kaplan–Meier estimate). Also, the incidence of postoperative retinal detachment was not significantly different in the patients with and without posterior capsulotomy in the aphakia group $(P = 0.621$, Mantel–Cox test by Kaplan–Meier estimate; Table 2).

Location of Breaks Causing Postoperative Retinal Detachment

The retinal breaks associated with the retinal detachment were located at the ora serrata (from the ora serata to the posterior side of the vitreous base) in 2 eyes and at the pars plana in 1 eye in the IOL group with an intact posterior capsule (Table 3). Two eyes developed a retinal detachment caused by giant breaks extending

Table 3. Postoperative Retinal Detachment after Cataract Extraction

Group	Age	Gender	Eye	Preoperative VA	PCR	YAG	Duration (mos)
IOL	21	M	L	20/100	None	None	72
IOL	21	M	R	20/25	None	None	69
IOL	22	M	R	20/2000	None	YAG	35
IOL	15	M	L	HM	PCR	None	25
IOL	41	M	R	20/40	PCR	None	6
Aphakia	16	M	L	20/500	None	None	57
Aphakia	21	F	L	HM	None	None	53
Aphakia	25	M	L	20/2000	None	YAG	9
Aphakia	14	F	L	20/16	None	YAG	54
Aphakia	14	F	R	20/60	None	YAG	54
Aphakia	29	M	R	HM	None	None	15
Aphakia	28	M	R	HM	None	None	28
Aphakia	24	M	R	20/500	None	None	2
Aphakia	17	M	R	FC	PCR	None	25

duration = time duration between retinal detachment and cataract surgery; ERM = epiretinal membrane; F = female; FC = finger count; following = giant break at the ora serrata; HM = hand movements; L = left; LP = light perception; M = male; PCR = posterior capsule rupture; R = right; RD buckling; VA = visual acuity; vit = vitrectomy; YAG = yttrium–aluminum–garnet laser posterior capsulotomy.

more than a quadrant at the ora serrata in the IOL group with intraoperative PCR.

In the aphakia group with an intact posterior capsule, the breaks were located at the ora serrata in 5 eyes (including 1 eye with a giant break) and at the pars plana in 3 eyes. A giant break was found in 1 eye at the ora serrata in the aphakia group with intraoperative PCR. Thus, all 3 eyes with PCRs developed retinal detachments with giant breaks at the ora serrata. No posterior vitreous detachment was present in any eye with a postoperative retinal detachment.

Treatment for Retinal Detachments

All eyes that developed a retinal detachment were treated by a single procedure of scleral buckling and/or vitreous surgery, with the exception of 2 eyes that required additional vitreous surgery because of a postoperative macular pucker and scleral buckling surgery, including gas tamponade for recurrent retinal detachment. These eyes recovered vision to 20/16 postoperatively.

Discussion

The results of this study demonstrated that although the mean visual acuity was not significantly different in the 2 groups, the incidence of retinal detachment was significantly lower after cataract extraction with IOL implantation than without IOL implantation in patients with atopic dermatitis. The number of pseudophakic retinal detachments increases with increasing duration of follow-up.[9,10] Although the aphakia group had longer follow-ups allowing more of a chance to have postoperative retinal detachments, a significant difference ($P < 0.05$) in the incidence of postoperative retinal detachment in the IOL group was also seen after longer follow-up periods of the fifth and sixth years. In addition, intraoperative PCR appeared to be a risk factor for retinal detachment in the IOL group, but not in the aphakia group, although the numbers were very small.

The pathogenesis of cataract and retinal detachment as-sociated with atopic dermatitis has not been determined. One possible cause of the retinal detachment is the repetitive mild trauma that patients who have atopic dermatitis receive, because they often relieve itching by slapping their faces instead of scratching. The similarity in the locations of the retinal breaks that cause the retinal detachment with those of traumatic retinal detachment supports this possibility.[11]

The theory that atopic shock organ is from an ectodermal origin has also been suggested to be involved in the pathogenesis of cataracts and retinal detachments in atopic patients.[12] An increase in the aqueous level of a tissue-destroying major basic protein derived from eosinophils in patients with atopic cataracts and/ or retinal detachments has been reported to explain damage of the lens capsule, retina, and ciliary epithelium in these patients.[13,14] The presence of a major basic protein[14] and an increase in the numbers of apoptotic cells[15] in the anterior lens capsule excised from patients with atopic cataracts, detected by immunohistochemical studies, support this hypothesis, especially to explain the pathogenesis of atopic cataracts.

Fibrosis and contraction of the residual lens capsule can cause traction on the ciliary body by transmitting traction along the zonules of Zinn.[8] Tears at the pars plicata may be created by this traction if there is a pre-existing fragility of the ciliary epithelium. Although retinal detachment caused by pars plicata tears can also be seen in phakic patients,[16] contraction of the residual lens capsule may increase the risk of retinal detachment in aphakic patients. The zonules of Zinn are partially connected to the pars plana through the peripheral vitreous[17]; therefore, not only the pars plicata breaks, but also the pars plana breaks, and even retinal breaks near the ora serrata, can be created by a similar mechanism.

The feasibility of preventing postoperative retinal detachment by implanting an IOL with in-the-bag fixation to

in the Intraocular Lens and Aphakia Groups

Retinal Breaks	RD Operation	Postoperative VA	Following Operation	Final VA	Retinal Attachment	Follow-up (mos)	Complications
Ora serrata	SB	20/16	Vit	20/16	Yes	113	ERM
Ora serrata	SB	20/16		20/16	Yes	109	
Pars plana	SB, vit	20/20		20/40	Yes	106	
Giant	SB, vit	20/20		LP	Yes	52	
Giant	SB, vit	20/20		20/60	Yes	25	
Giant	SB	20/20		20/2000	Yes	134	
Pars plana	SB	20/20		20/22	Yes	110	
Ora serrata	SB	20/20	SB removal	20/100	Yes	122	Glaucoma
Ora serrata	SB	20/16		20/16	Yes	165	
Ora serrata	SB	20/16		20/16	Yes	165	
Ora serrata	SB	20/20		20/22	Yes	130	
Pars plana	SB	20/20		20/40	Yes	134	
Pars plana	SB	20/20		20/25	Yes	81	
Giant	SB	20/16	SB, gas	20/16	Yes	54	

operation = following operation after retinal detachment surgery; follow-up = follow-up period after cataract surgery; gas = intravitreal gas injection; giant operation = operation for retinal detachment after cataract surgery; retinal attachment = presence of retinal attachment after surgery; SB = scleral