# Myopic Choroidal Neovascularization

## *A 10-year Follow-up*

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***Purpose:*** To clarify the long-term visual outcome of choroidal neovascularization (CNV) in eyes with high myopia in Asian patients.

***Design:*** We reviewed the medical records of 25 consecutive patients (27 eyes) with myopic CNV who were followed up for at least 10 years after the onset of CNV. Visual acuity was examined 10 years after CNV onset.

***Intervention:*** Demographic and clinical data were obtained from the patients' medical records.

***Main Outcome Measures:*** Visual acuity readings during the 10 years after CNV onset.

***Results:*** At the onset of CNV, 19 eyes (70.4%) had a visual acuity better than 20/200, and six eyes (22.2%) had a visual acuity better than 20/40. Three years after the onset of CNV, 15 eyes (55.5%) retained a visual acuity of better than 20/200. At 5 and 10 years after the onset, however, visual acuity dropped to 20/200 or less in 24 eyes (88.9%) and in 26 eyes (96.3%), respectively. The logarithm of the minimum angle of resolution (logMAR) visual acuity was significantly worse at 5 and 10 years after onset as compared with that at CNV onset. Chorioretinal atrophy developed around the regressed CNV in 26 eyes (96.3%) at 5 and 10 years after the onset of CNV.

***Conclusions:*** Long-term visual outcome of myopic CNV is extremely poor. The visual acuity of almost all of the patients dropped to 20/200 or less within 5 to 10 years after the onset of CNV, secondary to the development of chorioretinal atrophy around the regressed CNV. These findings indicate that active treatments should be recommended to prevent long-term visual impairment in Asian patients with myopic CNV. *Ophthalmology 2003; 110:1297–1305 © 2003 by the American Academy of Ophthalmology.*

High myopia is a major cause of legal blindness in many developed countries.[1–3] It affects 27% to 33% of all myopic eyes, corresponding to a prevalence of 1.7% to 2% in the general population of the United States.[4] High myopia is especially common in Asia and the Middle East.[4] In Japan, the number of cases of myopia is unknown, but pathologic or high myopia affects 6% to 18% of the myopic population and 1% of the general population.[5]

High myopia is associated with progressive and excessive elongation of the eyeball, which results in various funduscopic changes within the posterior staphyloma.[6–9] These changes include areas of atrophy of the retinal pigment epithelium and choroid, lacquer cracks in Bruch's membrane, subretinal hemorrhage, and choroidal neovascularization (CNV; so-called Fuchs' spot).[10–13] Among the various myopic fundus lesions, macular CNV is the most common vision-threatening complication of high myopia.[14–16]

Active treatments such as photodynamic therapy recently have been applied for myopic CNV.[17,18] Knowledge of the natural history of myopic CNV is important for defining the indications of new treatments. Previous studies describing the natural prognosis of myopic CNV, however, are somewhat conflicting.[11,12,14–16,19] Some studies describe a favorable course of myopic CNV,[14,15] and others report a poor prognosis.[11,12,16] In most of the previous studies, however, the follow-up period was relatively short in that patients were followed up for no more than a few years.[11,12,14,19]

The present study indicates that vision in eyes with myopic CNV progressively declines over the long term. To our knowledge, however, there are no reports of a long-term follow-up study in a large population of eyes with myopic CNV. We followed up patients with myopic CNV for more than 10 years to clarify the long-term visual prognosis of myopic CNV.

## Patients and Methods

Twenty-five consecutive patients (27 eyes) with high myopia and submacular (subfoveal or juxtafoveal) CNV were identified using

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clinical records who were treated from 1988 through 2002 at the high myopia clinic at the Tokyo Medical and Dental University and were enrolled in the present study. Informed consent was obtained from all patients. The study was approved by the Ethics Committee of the University. Inclusion criteria for this study were: (1) refractive error of −8 diopters (D) or more; (2) fundus changes typical of pathologic myopia: chorioretinal atrophy, lacquer cracks, atrophic patches; (3) presentation within 6 months of onset of symptoms, that is, loss of visual acuity, metamorphopsia, or both; (4) fluorescein angiographic documentation of macular CNV; and (5) minimum follow-up of 10 years. Patients who underwent laser photocoagulation or surgical treatment of CNV and those with less than 10 years follow-up were excluded from the study. Additional exclusion criteria included a history of visual loss resulting from myopic chorioretinal atrophy, history of retinal detachment surgery, history of cataract surgery, diabetic retinopathy, or other retinal vascular diseases; age-related maculopathy; dense cataract; glaucoma; and ocular injuries.

The initial evaluation included refraction, axial length measurements, best-corrected visual acuity, detailed fundus drawings using indirect stereoscopic ophthalmoscopy, fluorescein angiography, and color photographs. These procedures were repeated as necessary during the follow-up visits. Visual acuity was assessed monocularly using the Snellen visual acuity chart. The measurement of Snellen visual acuity was always conducted by one of the same three orthoptists under consistent lighting conditions and was confirmed by one of the authors (KOM or TT). The early phase of the fluorescein angiogram was independently reviewed by three of the authors (TY, KOM, and SF), and the size of the CNV on fluorescein angiogram was measured. The case was considered subfoveal if any portion of a new vessel system was under the center of the fovea. Other cases were considered juxtafoveal.

To quantify the enlargement of the area of chorioretinal atrophy around the CNV, color fundus photographs or fluorescein angiograms were scanned into a computer using a Scanjet IICX/T (Hewlett Packard, Palo Alto, CA). These images were first cropped on a personal computer using PhotoShop (version 5.0). The file was then exported to NIH Image software (version 1.62), and the outlines of the CNV and chorioretinal atrophy were plotted on a computer screen. The CNV outlines were decided based on the early phase of the fluorescein angiogram. Thereafter, the total number of pixels within the CNV or chorioretinal atrophy was calculated. The area of chorioretinal atrophy in each patient was defined by the following equation: (total number of pixels of chorioretinal atrophy − total number of pixels of CNV at initial examination)/number of pixels of the optic disc area (DA).

For the purpose of analysis, Snellen visual acuity data were transformed into equivalent logarithm of the minimum angle of resolution (logMAR) values. Changes in visual acuity during the follow-up period were analyzed using paired *t* tests. A *P* value of less than 0.05 was considered to be statistically significant.

# Results

The characteristics of the patients are summarized in Table 1.

## Initial Examination

Mean age at the initial visit was 46.9 years (range, 26–61 years). Mean refraction was −15.4 D (range, −9.0 to −22.5 D), and mean axial length was 29.3 mm (range, 26.8–33.1 mm). The initial Snellen visual acuity was 20/40 or more in six eyes (22.2%), 20/40 to 20/200 in 13 eyes (48.1%), and 20/200 or less in eight eyes (29.6%). The baseline mean logMAR at the initial examination was 0.75 ± 0.48. Mean CNV size was 0.92 disc diameter (DD; range, 0.2–1.6 DD). The CNV location was subfoveal in 22 eyes (81.5%) and juxtafoveal in five eyes (18.5%).

Table 1. Patient and Study Eye Characteristics at the Initial Examination

| | |
|---|---|
| Gender, no. persons (eyes) | |
| Men | 5 (5) |
| Women | 20 (22) |
| Age (yrs), mean (SD) | 46.9 (12.7) |
| Refractive error (D), mean (SD) | −15.4 (4.3) |
| Axial length (mm), mean (SD) | 29.3 (2.3) |
| Initial Snellen visual acuity | |
| >20/40 | 6/27 (22.2%) |
| 20/40–20/200 | 13/27 (48.1%) |
| <20/200 | 8/27 (29.6%) |
| Baseline logMAR, mean (SD) | 0.75 (0.48) |
| Size of CNV (DD), mean (SD) | 0.92 (0.6) |
| Location of CNV | |
| Subfoveal | 22/27 (81.5%) |
| Juxtafoveal | 5/27 (18.5%) |

CNV = choroidal neovascularization; D = diopters; DD = disc diameter; logMAR = logarithm of the minimum angle of resolution; SD = standard deviation.

## Follow-up Examination

The follow-up period ranged from 120 to 159 months (average, 132 months).

## Changes in Snellen Visual Acuity

Figure 1 shows the distribution of initial and final visual acuities. Most of the patients had decreased visual acuity after more than 10 years of follow-up. Figure 2 and Table 2 show the distribution of Snellen visual acuity during the follow-up period. At the initial examination, 70.4% of the eyes had a visual acuity better than

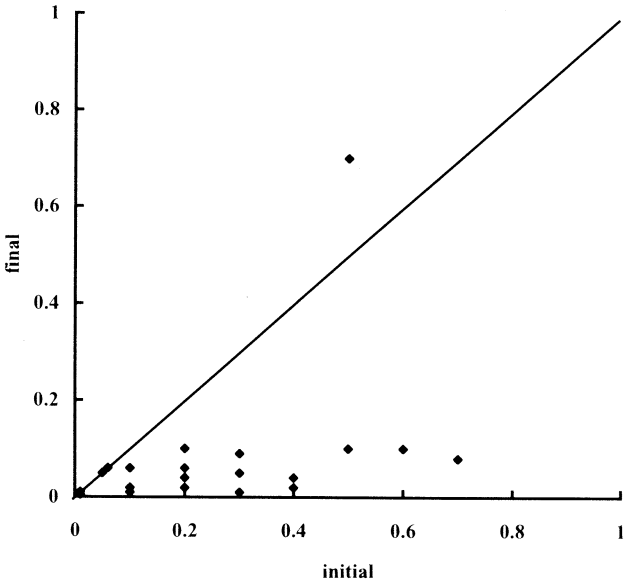



**Figure 1.** Distribution of initial and final visual acuities. Dots on the line indicate unchanged visual acuity, dots above the line indicate improvement, and dots below the line indicate worse visual acuity.

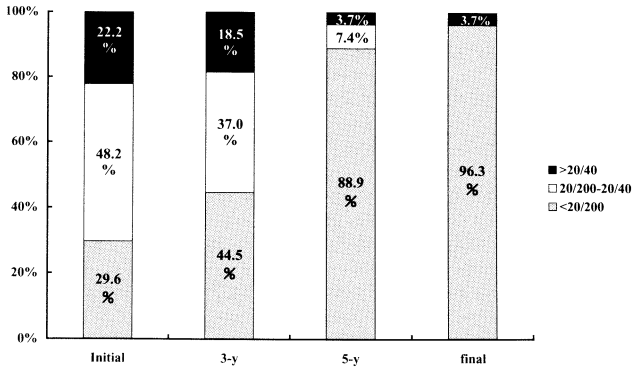



**Figure 2.** The shift in the distribution of Snellen visual acuity during the follow-up.

20/200, and 22.2% of the eyes had a visual acuity of 20/40 or greater. Only 29.6% of the eyes had a visual acuity of 20/200 or less at the initial examination (Table 2). Three years after the initial examination, the number of eyes that had a visual acuity of 20/200 or less increased to 44.5%. A visual acuity of 20/200 or better was retained by 55.5% of the eyes at 3 years after CNV onset. Five years after the onset of CNV, the visual acuity distribution decreased dramatically. In 88.9% of the eyes, vision dropped to 20/200 or less. At the final examination, 96.3% of the eyes had a visual acuity of 20/200 or less. Only one eye retained a visual acuity of better than 20/40 during the follow-up of more than 10 years (patient 3).

## Changes in Logarithm of the Minimum Angle of Resolution Visual Acuity

Table 3 shows the change in logMAR visual acuity during the follow-up period. The initial mean logMAR visual acuity was 0.75 ± 0.48. Three years after the initial examination, the mean logMAR visual acuity was 0.95 ± 0.55, which was not statistically different from the initial value ($P > 0.05$, paired *t* tests). Five years after the initial visit, however, the mean logMAR visual acuity increased to 1.25 ± 0.41, and at the final examination, the logMAR visual acuity was 1.32 ± 0.43. The mean logMAR visual acuity both at 5 years and at the final examination was significantly increased as compared with the mean logMAR visual acuity at the initial examination ($P = 0.03$ and 0.02, respectively, paired *t* test).

## Absorption of Bleeding and Rebleeding from Choroidal Neovascularization

At the initial visit, 25 of 27 eyes had subretinal bleeding around the CNV. The bleeding from the CNV in these 25 eyes disappeared in an average of 7.6 months (range, 1–15 months) from the initial visit (Table 4). Rebleeding occurred in 6 of 27 eyes (22.2%) during the follow-up period.

Table 3. Change in Logarithm of the Minimum Angle of Resolution Visual Acuity during the Follow-up

| Logarithm of the Minimum Angle of Resolution Visual Acuity | | | |
|---|---|---|---|
| Initial (SD) | 0.75 (0.48) | n.s. | $P = 0.03$† |
| 3-year (SD) | 0.95 (0.55) | | |
| 5-year (SD) | 1.25 (0.41) | | $P = 0.02$* |
| Final (SD) | 1.32 (0.43) | | |

n.s. = ; SD = standard deviation.
*Paired *t* test.

## Development and Enlargement of Chorioretinal Atrophy around Choroidal Neovascularization

Chorioretinal atrophy around the regressed CNV developed in 20 of 27 eyes (74.1%) 3 years after the initial examination (Table 2). At 5 years after the onset and at the final examination, however, chorioretinal atrophy developed in 26 of 27 eyes (96.3%). Chorioretinal atrophy had developed in all but one eye after the 5-year follow-up (patient 3).

The mean size of the area of chorioretinal atrophy around the regressed CNV 3 years after the onset of CNV was 1.21 ± 0.98 DD. Five years after the onset and at the final examination, the mean area of chorioretinal atrophy increased to 2.87 ± 2.18 DD and 5.64 ± 4.70 DD, respectively (Table 2).

## Case Reports

**Patient 1: Poor Visual Outcome.** A 34-year-old woman sought treatment on May 11, 1990, with decreased visual acuity in her left eye. At the initial examination, the patient's best-corrected visual acuity was 20/20 in the right eye and 20/40 in the left eye. The refractive error was −14.5 D in the right eye and −13.5 D in the left eye, and the axial length measurements were 27.4 mm in the right eye and 27.5 mm in the left eye. The left fundus at the initial examination had the characteristic grayish fibrovascular membrane (Fig 3A, arrow) with slight bleeding. Fluorescein angiogram at the initial examination showed slight hyperfluorescence at the site of CNV (Fig 3B, arrow). Three years after the initial visit (August 25, 1993), the patient's visual acuity in the left eye was 20/40. The left fundus had a whitish fibrovascular membrane (Fig 3C, arrow). Small subretinal bleeding was observed temporal to the CNV (Fig 3C, arrowhead). Chorioretinal atrophy around the CNV was not evident. Five years after the initial examination (July 1, 1995), the

Table 2. Change in Visual Acuity and Incidence of Chorioretinal Atrophy during the Follow-up Period

| | Initial | 3-year | 5-year | Final |
|---|---|---|---|---|
| Visual acuity | | | | |
| >20/40 | 6/27 (22.2%) | 5/27 (18.5%) | 1/27 (3.7%) | 1/27 (3.7%) |
| 20/200–20/40 | 13/27 (48.1%) | 10/27 (37.0%) | 2/27 (7.4%) | 0/27 (0%) |
| <20/200 | 8/26 (29.6%) | 12/27 (44.4%) | 24/27 (88.9%) | 26/27 (96.3%) |
| Chorioretinal atrophy around myopic CNV | 0/27 (0%) | 20/27 (74.1%) | 26/27 (96.3%) | 26/27 (96.3%) |
| Size of chorioretinal atrophy (DD), mean (SD) | | 1.21 (0.98) | 2.87 (2.18) | 5.64 (4.70) |

CNV = choroidal neovascularization; DD; disc diameter; SD = standard deviation.

Table 4. Patient and Study Eye Characteristics during the Follow-up Period

| Characteristics | |
|---|---|
| Follow-up period (mos), mean (SD) | 132 (10) |
| Period until absorption of bleeding from CNV (month), mean (SD) | 7.6 (5.3) |
| Rebleeding from CNV (eyes) | 6/27 (22%) |

CNV = choroidal neovascularization; SD = standard deviation.

CNV completely regressed (Fig 3D, arrow). The CNV became flat and pigmented, and no bleeding was observed around the CNV. A large area of chorioretinal atrophy developed around the CNV (Fig 3D, arrowhead). The patient's visual acuity in the left eye at that time decreased to 20/200. Ten years after the initial visit (July 14, 2000), the area of chorioretinal atrophy around the regressed CNV was slightly enlarged (Fig 3E, arrowhead). Fluorescein angiogram at that time showed choroidal filling defect at the site of chorioretinal atrophy (Fig 3F, arrowhead) around the regressed CNV (Fig 3F, arrow). The patient's visual acuity in the left eye at that time was 20/200.

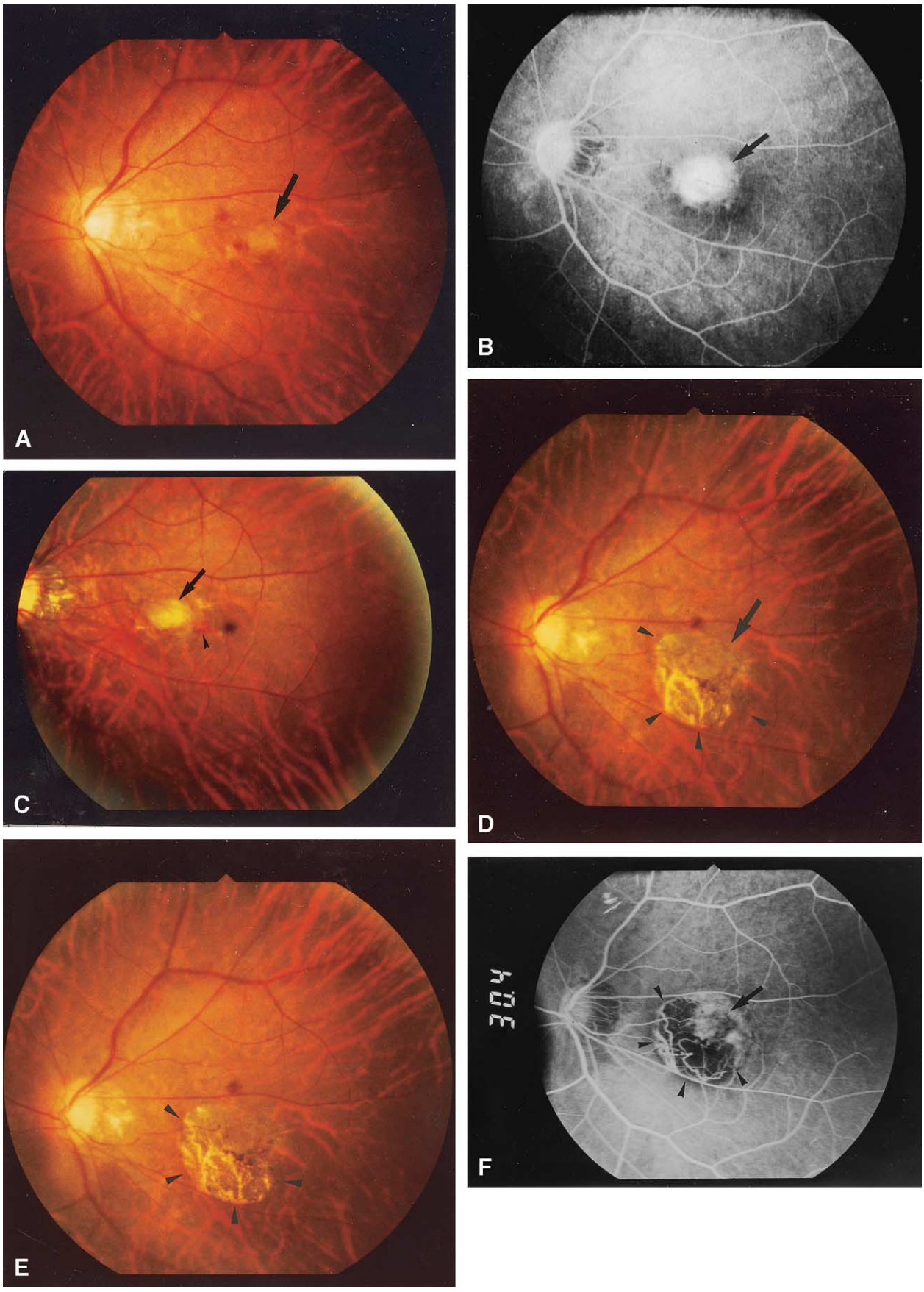
**Patient 2: Poor Visual Outcome.** A 38-year-old man sought treatment on March 3, 1989, with decreased visual acuity in his right eye. At the initial examination, the patient's best-corrected visual acuity was 20/50 in the right eye and 20/20 in the left. The refractive error was −11.0 D in the right eye and −9.0 D in the left, and the axial length measurements were 26.9 mm in the right eye and 26.2 mm in the left. The right fundus at the initial examination had the characteristic grayish fibrovascular membrane (Fig 4A, arrow) surrounded by retinal bleeding (Fig 4A, arrowhead) at the nasal end of the lacquer crack. Three years after the initial examination (February 10, 1992), the bleeding was absorbed. A small area of atrophy formed on the nasal side of the regressed CNV (Fig 4B, arrowhead). Fluorescein fundus angiogram at that time showed a window defect at the site of chorioretinal atrophy (Fig 4C, arrowhead) around the CNV (Fig 4C, arrow). The patient's visual acuity in the right eye at that time decreased to 20/100. Five years after the initial visit (February 21, 1994), the CNV regressed into fibrovascular scar tissue (Fig 4D, arrow) and became flat. Chorioretinal atrophy around the regressed CNV enlarged (Fig 4D, arrowhead). The patient's visual acuity in the right eye at that time decreased to 20/200. Ten years after the initial visit (January 9, 1999), the area of chorioretinal atrophy enlarged further (Fig 4E, arrowhead). The CNV became unrecognizable within a large area of chorioretinal atrophy. Fluorescein fundus angiogram at that time showed a choroidal filling defect at the site of chorioretinal atrophy (Fig 4F, arrowhead) around the regressed CNV (Fig 4F, arrow). The patient's visual acuity in the right eye at that time was 20/200.

**Patient 3: Good Visual Outcome.** A 26-year-old woman sought treatment on January 4, 1992, with decreased vision in her left eye. At the initial examination, the patient's best-corrected visual acuity was 20/20 in the right eye and 20/40 in the left. The refractive error was −9.5 D in the right eye and −10.5 D in the left, and the axial length measurements were 26.4 mm in the right eye and 26.9 mm in the left. The left fundus at the initial examination had the grayish fibrovascular membrane of CNV (Fig 5A, arrow) surrounded by slight retinal edema. A fluorescein fundus angiogram at the initial examination revealed hyperfluorescence corresponding to CNV (Fig 5B, arrow). Three years after the initial examination (July 19, 1995), the CNV regressed and became pigmented (Fig 5C, arrow). The patient's visual acuity in the left eye recovered to 20/30 in 3 years. More than 10 years after the initial examination (March 5, 2002), the left fundus showed a regressed CNV (Fig 5D, arrow). There was no chorioretinal atrophy around the regressed CNV throughout the follow-up period. The patient's visual acuity in the left eye at the final examination remained 20/30.

## Discussion

Choroidal neovascularization is a sight-threatening complication that occurs in 5% to 10% of patients with high myopia.[4,20] Much attention recently has focused on treatments for myopic CNV, including photodynamic therapy,[17,18] laser photocoagulation,[10,21–23] surgical extraction of CNV,[24–27] and macular translocation.[28–30] For this reason, it is important to understand the natural course of myopic CNV to evaluate these treatments. Choroidal neovascularization in high myopia is generally considered to have a relatively self-limited course,[14] in contrast to CNV secondary to age-related macular degeneration. Previously reported studies of visual acuity outcomes of myopic CNV, however, are somewhat conflicting.[11,12,14–16] There are some reports of a favorable course of myopic CNV,[14,15] and others describe a poor visual prognosis for myopic CNV.[11,12,16] In general, the follow-up period of the visual acuity outcome in these previously reported studies of myopic CNV, however, was short. Hotchkiss and Fine[12] reported that 50% of 33 patients had a visual decrease of more than two Snellen lines over a period of 24 months. This was not a pure natural history study, however, because 22% of the eyes had undergone laser photocoagulation of the CNV. Fried et al[15] followed up 55 eyes with myopic CNV and noted improved or stable visual acuity in 63% of eyes after a mean follow-up of 60 months. Also, CNV was not detected in 23% of the eyes on fluorescein angiography in their study. Avila et al[14] reported on a series of 70 eyes of 58 patients with untreated CNV associated with degenerative myopia. They reported that the visual acuity remained stable or improved in 54% of the eyes after a mean follow-up period of 41 months. Recently, Tabandeh et al[19] followed up 22 eyes in 22 patients with myopic CNV and reported that the final visual acuity was 20/200 or less in 73% of their eyes after a mean follow-up period of 49

**Figure 3.** Patient 1, a 34-year-old woman. **A,** The left fundus at the initial examination (May 1990) showed a choroidal neovascular membrane (CNV) with bleeding in the macula (arrow). Visual acuity was 20/40, and the refractive error was −13.5 diopters. **B,** Fluorescein angiogram at the initial examination. Five minutes after the dye injection, the CNV was slightly hyperfluorescent (arrow). **C,** Three years later (August 1993), the left fundus had a whitish fibrovascular membrane (arrow). Small subretinal bleeding was observed temporal to the CNV (arrowhead). Visual acuity was 20/40. **D,** Five years later (July 1995), the CNV became flat and pigmented (arrow) with no bleeding observed around the CNV. A large area of chorioretinal atrophy developed around the CNV (arrowhead). Visual acuity decreased to 20/200. **E,** Ten years later (July 2000), the area of chorioretinal atrophy around the regressed CNV enlarged (arrowhead). Visual acuity at that time was 20/200. **F,** A fluorescein fundus angiogram obtained 10 years after the onset of CNV showed choroidal filling defect at the site of chorioretinal atrophy (arrowhead) around the regressed CNV (arrow).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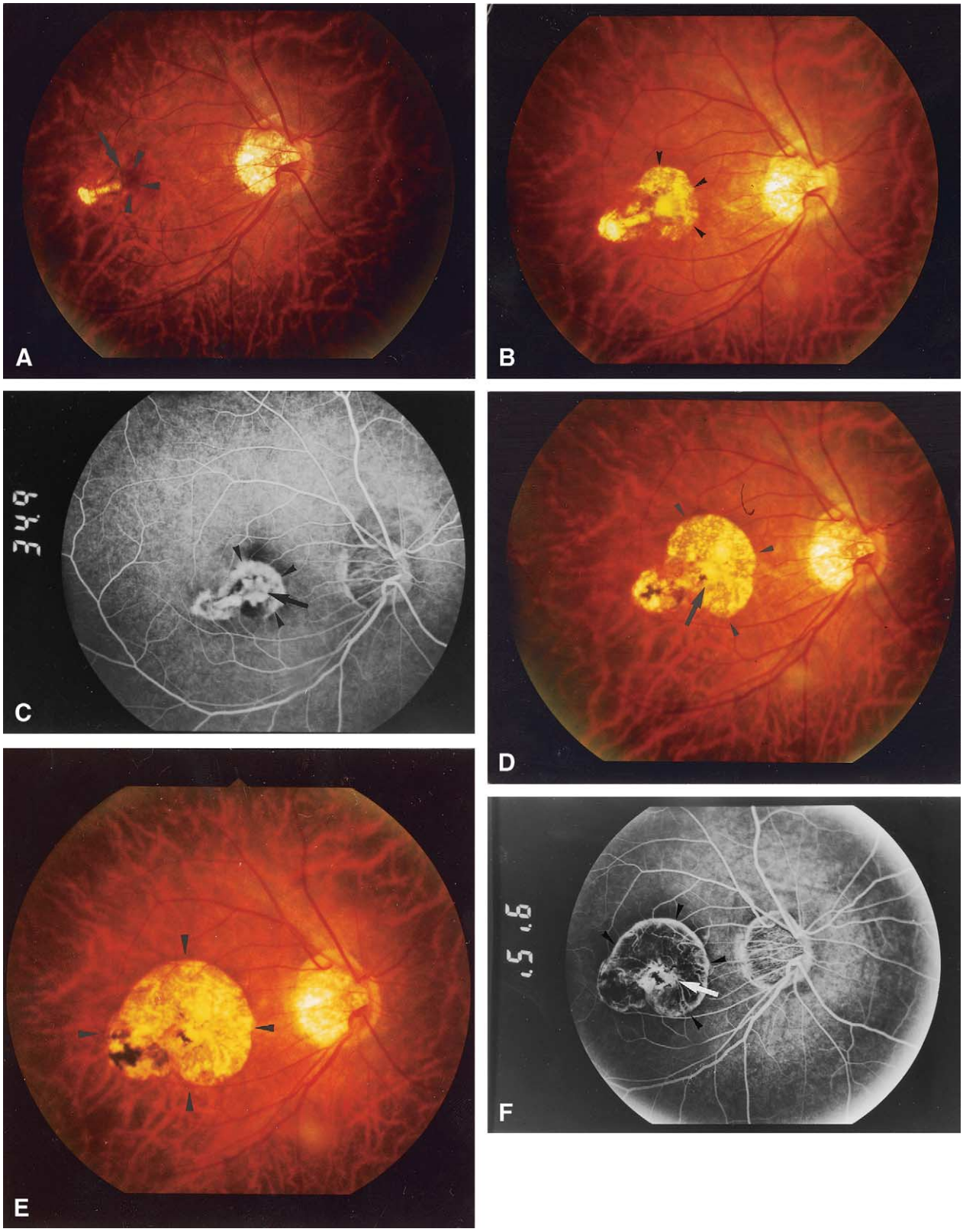


**Figure 4.** Patient 2, a 38-year-old man. **A,** The right fundus at the initial examination (March 1989) showed a choroidal neovascular membrane (CNV; arrow) with subretinal bleeding (arrowhead). Visual acuity was 20/50, and the refractive error was −11.0 diopters. **B,** Three years later (February 1992), the bleeding was absorbed. A small area of chorioretinal atrophy formed on the nasal side of the CNV (arrowhead). Visual acuity decreased to 20/100. **C,** Fluorescein fundus angiogram 3 years after the onset of CNV showed window defect at the site of chorioretinal atrophy (arrowhead) around the CNV (arrow). **D,** Five years later (February 1994), the CNV regressed into a fibrovascular scar tissue (arrow) and became flat. The area of chorioretinal atrophy around the regressed CNV enlarged (arrowhead). Visual acuity decreased to 20/200. **E,** Ten years later (January 1999), chorioretinal atrophy enlarged further (arrowhead). The CNV became unrecognizable within a large area of chorioretinal atrophy. Visual acuity was 20/200. **F,** A fluorescein fundus angiogram obtained 10 years after the onset of CNV showed choroidal filling defect at the site of chorioretinal atrophy (arrowhead) around the regressed CNV (arrow).

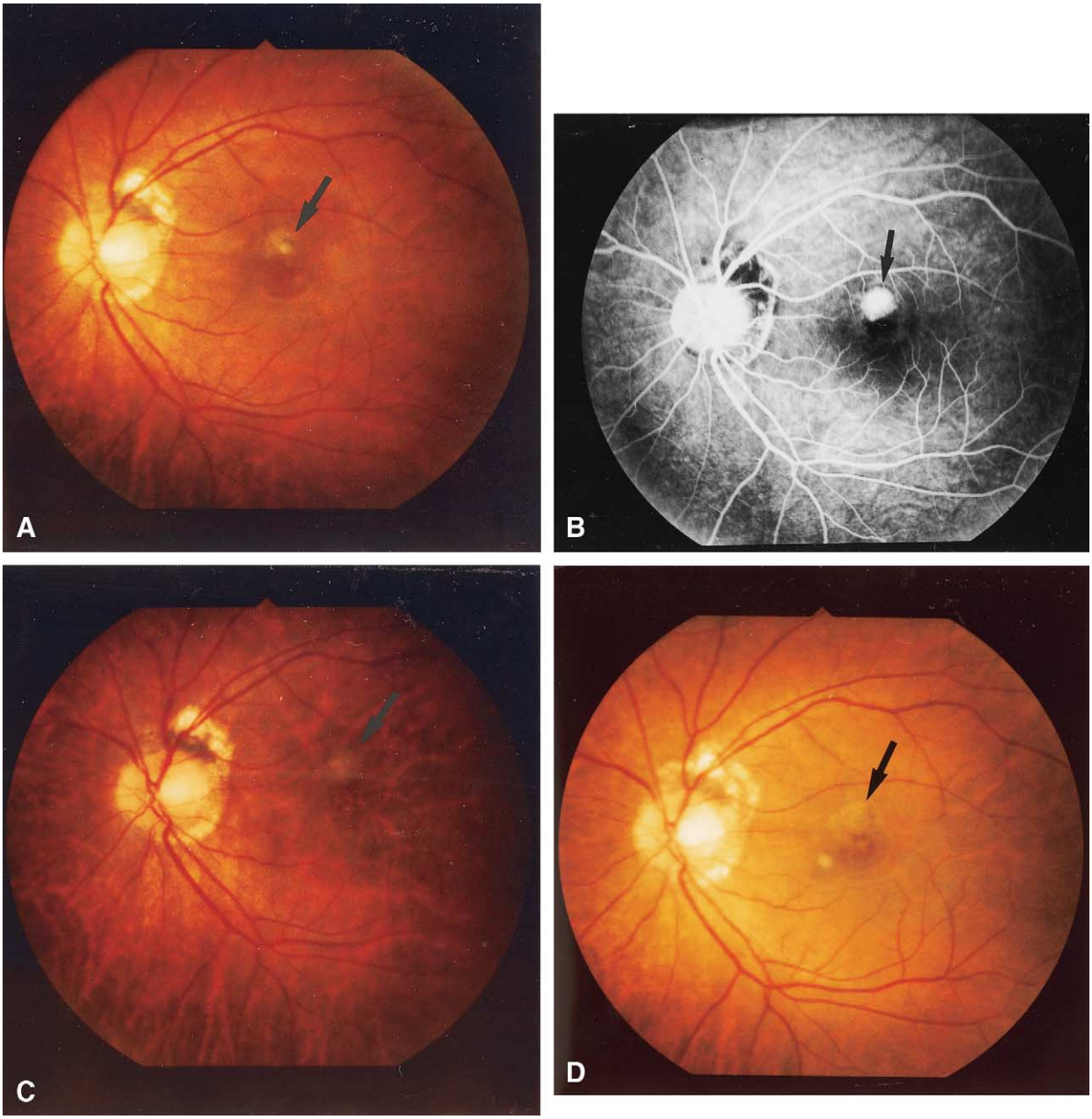


**Figure 5.** Patient 3, a 26-year-old woman. **A,** The left fundus at the initial examination (January 1992) showed a choroidal neovascular membrane (CNV; arrow) with slight retinal edema. Visual acuity was 20/40, and the refractive error was −9.5 diopters. **B,** A fluorescein fundus angiogram at the initial examination revealed an area of hyperfluorescence corresponding to the CNV (arrow). **C,** Three years later (July 1995), the CNV regressed (arrow). No subretinal bleeding was observed. Visual acuity recovered to 20/30. **D,** Ten years later (March 2002), the CNV became flat and slightly pigmented (arrow). No chorioretinal atrophy formed around the regressed CNV throughout the follow-up period. Visual acuity remained 20/30.

months; only older patients (50 years of age or older) were included.

The present study indicates that the long-term visual outcome of myopic CNV is extremely poor. There was a characteristic course of visual decline during the 10-year follow-up period. Until 3 years after the onset of CNV, the logMAR visual acuity did not indicate a significant visual decrease. The logMAR visual acuity was significantly worse, however, 5 years after the onset of CNV. The distribution of the Snellen visual acuity had a similar pattern of visual decrease during the 10-year follow-up. At the initial examination, approximately 70% of the patients had a visual acuity of better than 20/200, and 22% of the patients had a visual acuity of 20/40 or better. Even 3 years after the initial visit, almost half of the patients retained a visual acuity of better than 20/200. Five years after the onset, however, the distribution pattern of Snellen visual acuity dramatically worsened. Approximately 90% of the patients

had a visual acuity of 20/200 or less. Ten years after the onset, almost all the patients (96.3%) had a visual acuity of 20/200 or less. These results suggest that a progressive and steady visual decrease over the long term (after 5 years) may be a characteristic feature of myopic CNV. Therefore, long-term follow-up is necessary to determine the exact visual prognosis of myopic CNV, and the findings of the present study strongly indicate that treatment to prevent the progression of chorioretinal atrophy around myopic CNV to maintain long-term vision is important.

What causes the progressive and gradual visual decrease in eyes with myopic CNV long after the onset? Avila et al[14] reported that myopic CNV has low activity and a self-limiting course. Supporting their study,[14] the present study revealed that the bleeding from CNV was absorbed quickly (in approximately 7 months), and the recurrence of CNV was not common (rebleeding was observed in only 22% of the eyes). Five and 10 years after the onset, the CNV completely regressed in all cases and became flat and sometimes unrecognizable, as shown in the case reports. Therefore, CNV activity itself may not be a major factor influencing the long-term visual decrease of myopic CNV.

The development of chorioretinal atrophy around the regressed CNV is a later complication of myopic CNV.[4,31] Chorioretinal atrophy develops long after the onset of CNV and gradually enlarges. In the present study, the frequency of the development of chorioretinal atrophy increased as time passed. Three years after CNV onset, chorioretinal atrophy formed in 74.1% of the eyes; however, 5 and 10 years after the onset, chorioretinal atrophy developed in almost all the eyes examined (96%). Furthermore, the area of chorioretinal atrophy gradually enlarged during the follow-up (Table 2). Chorioretinal atrophy progressed to involve the fovea centralis, causing eccentric fixation and resulting in a decrease in Snellen visual acuity.

Based on the findings of the present study, the long-term effect of treatments to improve the visual outcome of myopic CNV must be considered. In contrast to other ocular disorders accompanying CNV (e.g., age-related macular degeneration), a temporary therapeutic effect such as absorption of subretinal bleeding or disappearance of fluorescein dye leakage may not be a useful marker for determining the effectiveness against myopic CNV. The prevention of later development of chorioretinal atrophy may be another goal to avoid the further visual decrease over the long-term.

In summary, we followed up 27 eyes with myopic CNV for more than 10 years. The results indicate that the long-term visual prognosis of myopic CNV is extremely poor. In almost all eyes with myopic CNV, visual acuity dropped to 20/200 or less within 10 years after the onset. Based on this evidence, appropriate treatment should be developed to avoid progressive visual impairment over the long term in eyes with myopic CNV.

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