Polypoidal choroidal vasculopathy (PCV) is a disease entity initially described by Yannuzzi et al. in 1990. The prevalence of PCV among patients with age-related macular degeneration (AMD) is estimated to be 30% to 50% in Asian people. PCV consists of an abnormal vascular network with polypoidal lesions at its termini under the retinal pigment epithelium (RPE).

### Classification of PCV

PCV can be classified into three types. The first type is the most common and involves choroidal vasculature abnormalities in the narrow sense. Vessels of the network show focal dilatation, constriction, and tortuosity, and the number of vessels is small. The second type is polypoidal choroidal neovascularization (CNV). Many vessels...
Radiating from a feeder vessel can be seen to have grown centrifugally, and polypoidal lesions appear at the vessel termini. Radiation-associated choroidal neovasculopathy is the third type. The pathogenesis of PCV remains controversial. PCV in the narrow sense has been described as being inner choroidal vessel abnormalities. A CNV variant, however, was also reported. These clinical and pathologic findings indicate the phenotypes of clinically observed PCV to represent different diseases. Therefore, treatment of PCV should be considered based on pathogenesis. PCV is, however, treated in a manner similar to that employed for CNV of AMD.

**TREATMENT OPTIONS: PDT**

Photodynamic therapy (PDT) is reportedly useful for improving visual acuity, although only for a short period, because polypoidal lesions, which cause bleeding and exudation, readily disappear with PDT, while network vessels persist. In our study, pre-PDT factors predicting better visual acuity 1 year after PDT were better initial visual acuity (ie, within 0.6 logMAR or less), smaller diameter of network vessels plus polypoidal lesions, and the absence of a polypoidal lesion under the fovea. However, the number of recurrent polypoidal lesions at the termini of extending or persisting network vessels increases or subretinal CNV occasionally appears with longer follow-up (Figures 1 and 2). Therefore, visual acuity increases at 1 year but has decreased to a level similar to that before PDT by 2 years following PDT.

**TREATMENT OPTIONS: ANTI-VEGF AGENTS**

Anti-vascular endothelial growth factor (VEGF) agents are another possibility for treating PCV. Although intravitreal bevacizumab (Avastin, Genentech, Inc.) monother-
apy was reported to be effective for transiently reducing exudative retinal detachment, polypoidal lesions persisted or enlarged (Figure 3). Bevacizumab does not apparently contribute to reducing network vessels. These findings may indicate either that its high molecular weight of 150 kD makes bevacizumab too large to penetrate the retina or that neither polypoidal lesions nor network vessels have VEGF receptors.

If the pathogenesis of PCV in the narrow sense was choroidal vasculature abnormalities, anti-VEGF drugs would not be useful for treating PCV. If the high molecular weight of bevacizumab was the problem, ranibizumab (Lucentis, Genentech, Inc.), which has a molecular weight of only 48 kD, would be anticipated to penetrate the retina quickly and bind to VEGF.

**ONGOING STUDY FOR PCV**

A prospective randomized clinical trial, comparing the efficacy of intravitreal ranibizumab monotherapy, PDT monotherapy, and combination therapy with ranibizumab and PDT, is currently ongoing in Asia.

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