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Polypoidal choroidal vasculopathy: Angiographic characterization of the network vascular elements and a new treatment paradigm ☆

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Abstract

Macular exudative manifestations secondary to choroidal neovascular lesions remain the leading cause of definitive visual impairment and legal blindness in the elderly. During the past decade, advances in ophthalmic imaging systems have enabled the recognition of presumed new distinct choroidal neovascular lesions that share some unique clinical and angiographic peculiarities as well as better comprehension of the pathophysiologic mechanisms related to such entities.

Amongst presumed newer exudative maculopathies, polypoidal choroidal vasculopathy, which has been described as a distinct choroidal abnormality characterized by inner choroidal vascular network of vessels ending in polyp-like structures only identified on indocyanine green angiography and mostly affecting African–American and Asian descendents, has gained special interest from the ophthalmic community particularly because of its growing recognition among patients with clinical appearance of neovascular age-related macular degeneration. Thus far, however, the exact nature of the vascular structure of the polypoidal choroidal vasculopathy lesion remains unclear and data from recent studies have conflicted with the initial concept of a benign exudative maculopathy with long-term preservation of good vision. All together, such factors make difficult the establishment of an appropriate treatment, if any, for the entity.

Herein, by using a modified technique of conventional indocyanine green angiography, we demonstrate new information about the morphologic characteristics, and to some extent the blood flow dynamics perfusion, of the polypoidal choroidal vasculopathy lesion. Our results suggest that the PCV lesion should be considered a variety of choroidal neovascularization rather than a distinct clinical entity, characterized by one single large neovascular complex presenting well-defined arterial neovascular vessels arising from one major ''ingrowth site'' and draining vessels that present aneurysm-like dilations corresponding to the polyp-like structures typically described for the entity. Finally, the visual acuity and angiographic findings observed after selective ingrowth site photothrombosis corroborate the existence of one major ''ingrowth site'' for the PCV neovascular complex and point toward a new treatment paradigm for this variety of choroidal neovascularization.

Abbreviations: BCVA, best-corrected visual acuity; CNV, choroidal neovascularization; ICG, indocyanine green; IMP, indocyanine green-mediated photothrombosis; ISiMP, ingrowth site indocyanine green-mediated photothrombosis; LogMAR, logarithm of the minimum angle of resolution; NV, neovascular vessel; PCV, polypoidal choroidal vasculopathy.

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1. Introduction

1.1. Terminology

In 1985, Stern et al. first reported a new clinical entity characterized by recurrent subretinal or subretinal pigment epithelium (RPE) hemorrhage and exudation in the macula associated with reddish-orange subretinal lesions in black women (Stern et al., 1985). This potentially blinding condition originally described as "multiple recurrent serosanguineous retinal pigment epithelial detachment in black women" (Stern et al., 1985; Perkovich et al., 1990), or as "posterior uveal bleeding syndrome" (Kleiner et al., 1990), was subsequently renamed by Yannuzzi et al., who give it its present name: "polypoidal choroidal vasculopathy" (PCV) (Yannuzzi et al., 1990). Since these initial reports more than 40 studies about the entity have been published enabling a better comprehension of the possible pathophysiologic mechanisms related to PCV.

1.2. Clinical features

PCV has no longer been considered an exclusivity of pigmented individuals as it has been described in Brazilian (Andrade et al., 2002), French (Mohand-Said et al., 2002), German (Schneider et al., 2001), Italian (Scasellati-Sforzolini et al., 2001), and Irish (Lip et al., 2000) patients and may be present in about 8–13% of white patients with clinical appearance of exudative age-related macular degeneration (Ciardella et al., 2004). To date, no particular clinical condition has been proved to correlate directly with PCV. Ross et al. have postulated that PCV may result from hypertensive insults to the choroidal vascular beds (Ross et al., 1996); however, it has also been suggested that hypertension, particularly in patients receiving antihypertensive treatment, is associated with neovascular complications in age-related macular degeneration (Hyman et al., 2000). Extended and, to some extent, conflicting information exists nowadays about the age of onset, gender preponderance, and natural course of PCV. Although most commonly diagnosed in patients between the ages of 50 and 65 years (Ciardella et al., 2004), descriptions of the entity can be found in a patient aged 20 (Yannuzzi et al., 1997) up to patients aged 88 and 90 years old (Shiraga et al., 1999; Ahuja et al., 2000; Lafaut et al., 2000a). In respect to gender preponderance, one can find reports demonstrating PCV involving predominantly women over men by a ratio of approximately 4.7:1 (Yannuzzi et al., 1999) as well as affecting equally men