

Eplerenone in the Treatment of Polypoidal Choroidal Vasculopathy

Kapil G. Kapoor Alan L. Wagner

Wagner Macula and Retina Center, Eastern Virginia Medical School,
Virginia Beach, Va., USA

Key Words

Eplerenone · Mineralocorticoid antagonist · Polypoidal choroidal vasculopathy

Abstract

Overactivation of mineralocorticoid receptor pathways has been implicated in the pathophysiology of central serous chorioretinopathy (CSCR). Recently, mineralocorticoid receptor antagonists such as eplerenone have demonstrated success in treating subretinal fluid in CSCR. This case demonstrates a patient who was initially presumed to have subretinal fluid secondary to CSCR and was started on a trial of oral eplerenone. It quickly became evident that her subretinal fluid was secondary to a peripapillary polypoidal choroidal vasculopathy network, but she demonstrated a significant improvement with oral eplerenone. To the authors' knowledge, this is the first case of eplerenone use to treat polypoidal choroidal vasculopathy.

Introduction

Overactivation of mineralocorticoid receptor pathways in choroidal vessels have been noted in experimental models of central serous chorioretinopathy (CSCR) [1]. Mineralocorticoid antagonism through oral agents such as eplerenone has subsequently demonstrated success in the treatment of persistent CSCR [2, 3].

Significant subretinal fluid can also be encountered in select cases of neovascular age-related macular degeneration (AMD) and polypoidal choroidal vasculopathy that can present challenges to successful treatment. In this report, successful reduction in subretinal fluid in a case of polypoidal choroidal vasculopathy is demonstrated.

Case Report

A 72-year-old woman presented with a gradual, painless decrease in vision in the left eye. On examination, visual acuity was 20/40 in the right eye and 20/150 in the left eye. Biomicroscopic examination of the left eye revealed significant macular subretinal fluid with the absence of any evident hemorrhage, drusen, retinal pigment epithelial changes, or exudate in either macula. Optical coherence tomography confirmed the presence of subretinal fluid with shaggy photoreceptor outer segments, absence of intraretinal fluid, and some outer segment/inner segment junction changes (fig. 1a). Intravenous fluorescein angiography (IVFA) was deferred due to a history of significant allergic reaction to IVFA in the past. Further history assessment revealed a recent use of oral steroids (2 months previously). The patient was started on eplerenone at 25 mg p.o. b.i.d. based on a presumed diagnosis of CSCR and was followed up for 2 weeks initially to ensure improvement, given other etiologies could not be conclusively ruled out in the absence of IVFA.

At the 2-week follow-up, visual acuity was improved to 20/100, and the subretinal fluid had significantly decreased (fig. 1b). The follow-up was extended to 4 weeks. Up to week 6, visual acuity remained 20/100, and the subretinal fluid had further decreased, but some exudate was evident on examination and OCT (fig. 1c) that suggested an alternative diagnosis may be possible. After review with the patient, the decision was made to premedicate the patient with steroids and Benadryl and to proceed with FA/indocyanine green, which revealed a network of peripapillary polypoidal choroidal vasculopathy with active leakage (fig. 2). Intravitreal Avastin was added to the treatment regimen, and the patient was maintained on oral eplerenone at 25 mg p.o. b.i.d. Four weeks later, nearly all subretinal fluid had resolved and the exudate had regressed (fig. 1d), and vision had improved to 20/50. Up to the 1-year follow-up, the patient has remained stable with absence of intraretinal or subretinal fluid or exudate on eplerenone at 25 mg p.o. b.i.d. and Avastin q8weeks.

Discussion

Polypoidal choroidal vasculopathy is a variant of neovascular AMD that is characterized by branching subretinal aneurysmal polyp-like vessels. Polypoidal choroidal vasculopathy is frequently characterized by subretinal hemorrhage, pigment epithelial detachment, or exudate, in addition to subretinal fluid. The EVEREST study pointed toward the efficacy of photodynamic therapy as being superior to anti-VEGF monotherapy in these lesions, which is further suggestive of its distinction from neovascular AMD [4]. The pathogenesis of polypoidal choroidal vasculopathy remains elusive, but the choroidal vasculature has been implicated, with indocyanine green angiography elucidating these polyp-like vessels best [4].

While the natural history and disease course of polypoidal choroidal vasculopathy and CSCR differ significantly, they share an implication of abnormal dilation of the choroidal vasculature in their presumed pathophysiology. This anatomic overlap begs the question of whether a deeper overlap of polypoidal choroidal vasculopathy and CSCR exists in some patients than has previously been realized. The lack of a diffusely leaky choroid argues against a classic presentation of CSCR in this case, and the identification of polyps in the setting of exudates is more consistent with a typical case of polypoidal choroidal vasculopathy. It is possible that some patients with polypoidal choroidal vasculopathy develop a secondary focal reactive retinal pigment epithelium dysfunction that leads to a CSCR type of response. This secondary accumulation of subretinal fluid with associated shoddy photoreceptor outer segments may independently lend itself to a response with eplerenone treatment [5]. Alter-

natively, it is plausible that mineralocorticoid receptor antagonism may add an adjunctive efficacy in the setting of subretinal fluid from both etiologies, particularly given a potentially shared choroidal pathologic origin.

In this case, oral eplerenone demonstrated efficacy as an adjunct to intravitreal anti-VEGF in a case of polypoidal choroidal vasculopathy. To the authors' knowledge, this is the first report of eplerenone use in the setting of polypoidal choroidal vasculopathy. Further research is needed to better elucidate the precise role of mineralocorticoid antagonists in polypoidal choroidal vasculopathy.

Statement of Ethics

This study complied with the guidelines for human studies, subjects were given informed consent, and the study protocol was approved by the committee for human research.

Disclosure Statement

The authors have no conflicts of interest.

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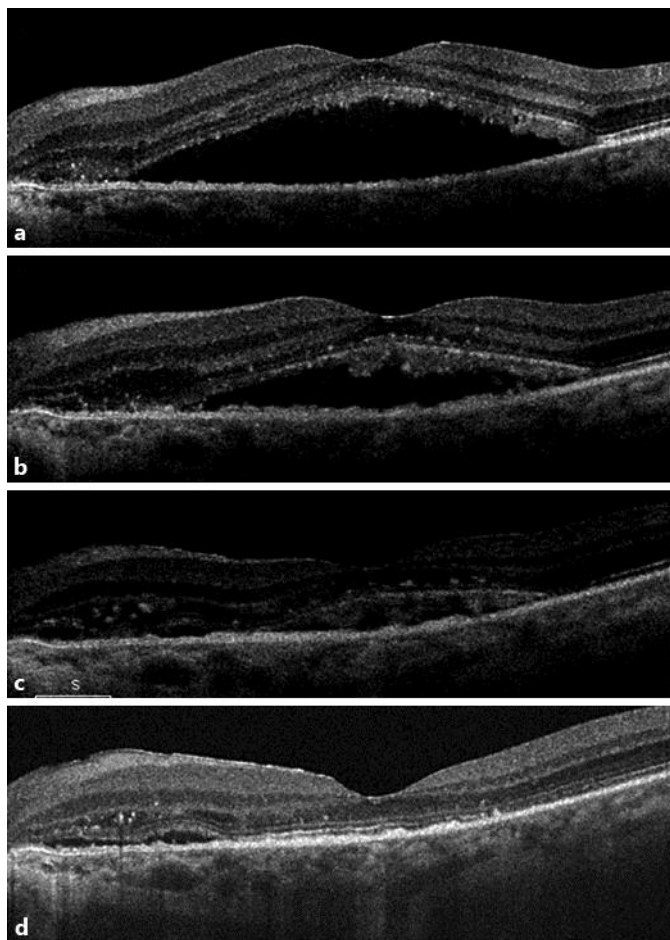


Fig. 1. **a** OCT revealing significant subretinal fluid with shaggy photoreceptor outer layer and no intraretinal fluid. **b** 2-week follow-up. Decreased subretinal fluid after starting eplerenone. **c** 6-week follow-up. Further decrease in subretinal fluid 6 weeks after starting eplerenone, with nasal exudate now definitely evident. **d** 10-week follow-up. Resolution of remaining foveal subretinal fluid with regression of exudate after starting intravitreal Avastin and continuing oral eplerenone at 25 mg p.o. b.i.d.

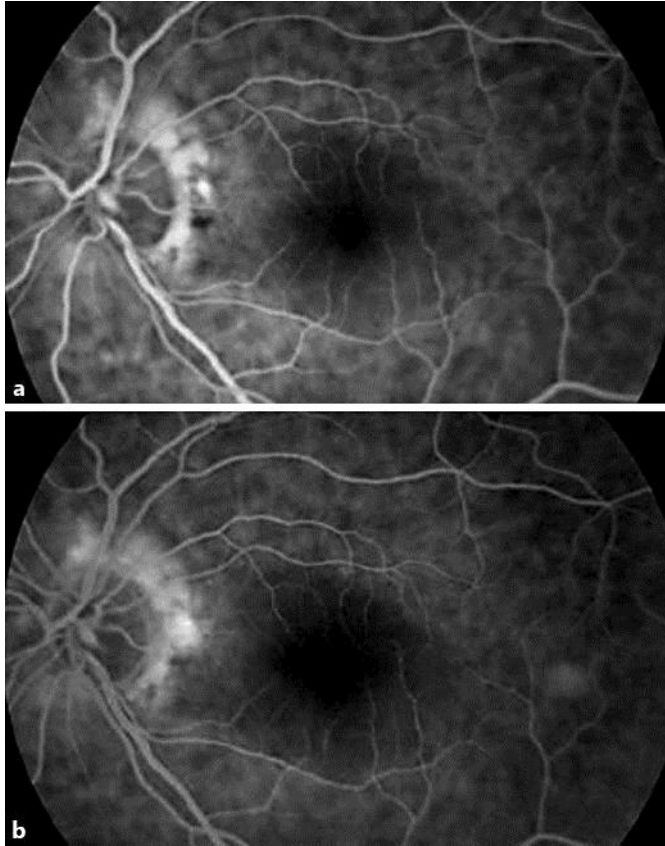


Fig. 2. FA (early; **a**) suggestive of peripapillary polyps that demonstrate leakage on late images (**b**), without evidence of a diffusely leaky choroid.