Intravitreal Bevacizumab for Choroidal Neovascularization Secondary to Angioid Streaks

Angioid streaks (AS) are irregular ruptures of the Bruch membrane that typically radiate from the optic disc. Through these cracks, new blood vessels may proliferate, generating choroidal neovascularization (CNV), which represents the main cause of visual loss in these patients. Laser photocoagulation has been widely used both to stop CNV and to stabilize visual acuity (VA) in patients with AS. The high rate of recurrences and functional problems related to the expansion of CNV or laser-induced scar toward the fovea have encouraged the evaluation of different treatment options. Photodynamic therapy (PDT) with verteporfin has been used to limit or delay visual damage caused by this aggressive disease, but its efficacy on macular function seems to be limited to a short period.

Vascular endothelial growth factor (VEGF) has been implicated in several diseases of the eye in which neovascularization and increased vascular permeability occur; thus, drugs inhibiting the VEGF bioactivity may provide a novel therapeutic option. Off-label use of intravitreal bevacizumab (IVB) has been introduced in the treatment of neovascular age-related macular degeneration, cystoid macular edema, neovascular glaucoma, pathologic myopia, and CNV due to AS. While the long-term safety and efficacy of IVB use have yet to be ascertained, these short-term results suggest that IVB use may represent an advantageous approach in the management of these pathologic conditions.

Herein we report on the clinical course of 5 patients with subfoveal CNV secondary to AS treated with IVB and followed up for 3 to 9 months.

Report of Cases. Our study population consisted of 5 patients with AS (1 woman, 4 men) with a mean (SD) age of 53.8 (7.52) years, all with skin biopsy–proven pseudoxanthoma elasticum. Two patients (2 and 5) had been previously treated with PDT; patient 2 was treated twice. Intravitreal bevacizumab therapy was proposed because of episodes of relapse. The other 3 patients (1, 3, and 4) had not received any treatment prior to IVB therapy (Table 1).

All patients received a complete ophthalmologic evaluation. No signs suggestive of age-related macular degeneration were found. Fluorescein (FA) and indocyanine green angiography criteria included evidence of leakage caused by CNV secondary to AS. Intravitreal bevacizumab treatment was recommended for (1) symptomatic lesions (recent decrease in VA and/or metamorphopsia); (2) presence of leakage on FA and indocyanine green angiography; and (3) presence of intraretinal or subretinal fluid documented by optical coherence tomography. All patients gave their written informed consent to the treatment (bevacizumab, 1.25 mg in 0.05 mL). Follow-up visits were carried out 1 week after the treatment and then monthly for 9 months. Two patients (4 and 5) received 2 injections at 6-week intervals (Table 1).

Mean central retinal thickness (CRT) at baseline was 325.6 µm (range, 255-394 µm). At the last check, mean CRT reduction was 41.6 µm (range, 54-20 µm), with a mean thickness of 299.6 µm (range, 218-340 µm) (Table 2). Best-corrected visual acuity (BCVA) was 10/16 at baseline and 10/10 at the last check. Mean CRT reduction was 41.6 µm (range, 54-20 µm), with a mean thickness of 299.6 µm (range, 218-340 µm) (Table 2).

Abbreviations: IVBs, intravitreal bevacizumab injections; PDTs, photodynamic therapies with verteporfin.
be worsened by the VEGF stimula-
tivity present in CNV, which could
due to the high angiogenic ac-
current available laser
Comment.
Lastly, neither systemic nor local ad-
reached a value of 10/16 3 months
At the beginning of the study,
ICB injection, their BCVA increased to 10/
and at the last follow-up check, his
The BCVA of patient 4 quickly im-
result was stable up to the end of fol-
One week after the injec-
the present study has some
limitations, including the limited
number of patients and the short pe-
follow-up period. Nevertheless,
considering the relative rarity of the dis-
ease, it would be difficult to conduct
randomized controlled trials, which
require a higher number of pa-
tients with CNV secondary to AS.
Michele Rinaldi, MD
Roberto dell’Omo, MD
Mario R. Romano, MD
Flavia Chiosi, MD
Ugo Cipollone, MD
Ciro Costagliola, MD
Correspondence: Dr Costagliola, Di-
partimento di Scienze per la Salute,
Università degli Studi del Molise, Via
Francesco De Sanctis s.n.c., 86100
Campobasso, Italy (ciro.costagliola
@unimol.it).
Financial Disclosure: None re-
ported.
1. Gass JDM. Angioid Streaks: Stereoscopic Atlas of
Macular Diseases. St Louis, MO: Mosby; 1997:
118-123.
come of choroidal neovascularization in angi-
oid streaks after photodynamic therapy. Retina.
3. Rich RM, Rosenfeld PJ, Puliafito CA, et al. Short-
term safety and efficacy of intravitreal bevac-
uzumab (Avastin) for neovascular age-related
macular degeneration. Retina. 2006;26(5):
495-511.
4. Teixeira A, Moreas N, Farah ME, Bonomo PP. Choroidal
neovascularization treated with in-
travitreal bevacizumab (Avastin) in angioid
835-836.
bevacizumab for idiopathic choroidal neovas-
cularization after previous injection with pos-
terior subtenon triamcinolone. Am J Ophthal-

**Table 3. CNV Lesion Size and Last FA Results**

<table>
<thead>
<tr>
<th>Patient</th>
<th>Total Area of CNV, mm²</th>
<th>Last FA</th>
<th>Last</th>
<th>Baseline</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>0.99</td>
<td></td>
<td></td>
<td>0.99</td>
</tr>
<tr>
<td>2</td>
<td>0.26</td>
<td></td>
<td></td>
<td>0.21</td>
</tr>
<tr>
<td>3</td>
<td>0.82</td>
<td></td>
<td></td>
<td>0.68</td>
</tr>
<tr>
<td>4</td>
<td>0.19</td>
<td></td>
<td></td>
<td>0.16</td>
</tr>
<tr>
<td>5</td>
<td>0.88</td>
<td></td>
<td></td>
<td>0.87</td>
</tr>
</tbody>
</table>

Abbreviations: CNV, choroidal neovascularization; FA, fluorescein angiography; Min, leakage size less than 50% of the area found at baseline; No, no leakage.

Comment. Current available laser treatments for CNV secondary to AS have a poor outcome. This might be because of the high angiogenic activity present in CNV, which could be worsened by the VEGF stimulation and up-regulation induced by the treatment itself. These data support IVB use in the management of CNV due to AS. In our experience, IVB injection did not modify the CNV size, whereas an increase in final lesion size in CNV due to AS has been reported after PDT. In our small series of patients, FA leakage diminished in patients 1, 3, and 5 and was completely absent in patients 2 and 4. A reduction of CRT was recorded in all subjects. This change of retinal morphology is likely to be the result of a combined antiexudative effect due to the decrease of vessel permeability and the antiproliferative effect due to the inhibition of further CNV growth following the VEGF blockage. The smallest reduction was recorded in the 2 patients who had previously undergone PDT. These anatomical improvements were associated with concomitant increases in VA (a mean of 3-4 lines). The mechanism of this outcome remains uncertain.

A previous case report of CNV due to AS treated with IVB by Teixeira and coworkers shows an improvement of the patient’s BCVA. Posttreatment optical coherence tomoscopy and FA imaging showed no presence of subsensory fluid or leakage, respectively. Our data seem to confirm the efficacy and safety of anti-VEGF therapy in eyes with CNV secondary to AS, although we realize that the present study has some limitations, including the limited number of patients and the short period of follow-up. Nevertheless, considering the relative rarity of the disease, it would be difficult to conduct randomized controlled trials, which require a higher number of patients with CNV secondary to AS.