MACULAR HOLE CLOSURE WITH TOPICAL STEROIDS

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FINANCIAL DISCLOSURE

No financial disclosures

SUMMARY

• Topical difluprednate monotherapy led to macular hole closure in a cohort of four patients.

INTRODUCTION

- OCT and ultrasonography findings illustrate the evolution of vitreoretinal traction in the development of macular holes.
- Macular holes have been observed in patients after vitrectomy without obvious traction.

INTRODUCTION

- The hydration theory of macular hole development suggests a nontractional mechanism.
- Approaches for nonsurgical management of macular holes are evolving.
- There may be a role for topical steroids in the closure of full-thickness macular holes.

METHODS

- Retrospective chart review
 - 4 patients with full-thickness macular holes were included.
 - All patients were treated with topical difluprednate 0.05% monotherapy QID.
- Mean treatment duration was 15 weeks (range, 12–20 weeks).

METHODS

- Baseline data was collected
 - Visual acuity (VA)
 - Macular hole structure (OCT)
 - IOP

METHODS

- Additional data was collected:
 - Time to hole closure
 - Final macular anatomy
 - Final VA
 - Complications of treatment
 - Need for additional intervention

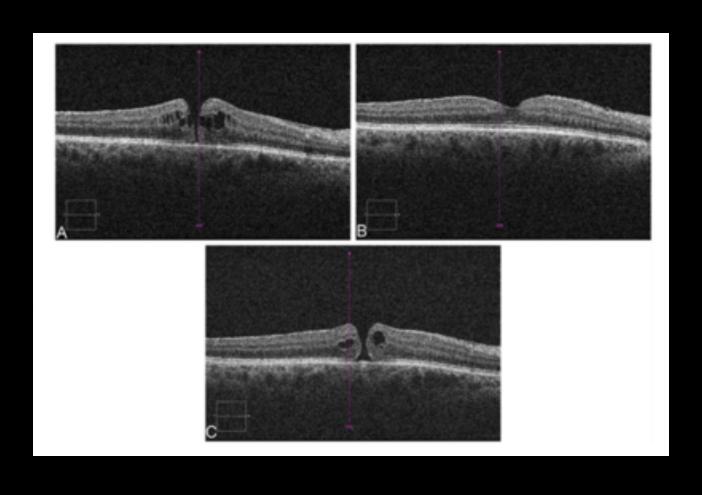
- Four patients (2 male and 2 female)
 - Mean age of 67 years (range, 59–78)
- Three patients had prior vitrectomy for:
 - Retinal detachment (2)
 - Retained lens fragments (1)
- One patient presented with a de novo idiopathic macular hole.
- One patient had a known history of steroid-induced ocular hypertension.

- Mean logMAR VA before and after hole closure were compared using a paired two-tailed t-test.
 - Acuities were then converted to Snellen VA.
 - Baseline VA = 20/42 (range 20/25 20/60)
 - Post-closure vision = 20/26 (range 20/25 20/30)
 - p = 0.14

- OCT imaging was used to assess macular hole status.
- Mean time to macular hole closure was 5 weeks (range, 2–12 weeks).

- All macular holes initially closed.
 - Two patients had hole closure without recurrence.
 - Two patients developed steroid-induced ocular hypertension.
 - Holes recurred when steroids were discontinued.

FIGURE 1



- This patient demonstrated an "on-and-off" phenomenon on two separate occasions
 - OCT images demonstrated a marked reduction in macular edema that coincided with macular hole closure
- The other patient demonstrated a similar "on-and-off" phenomenon

- These two patients developed steroid induced ocular hypertension
 - Steroids were permanently discontinued
 - Both holes were closed successfully with vitrectomy, ILM peeling, and SF6 gas tamponade without positioning

- Treatment with topical steroid drops was complicated by increased IOP
 - Patient 1 (Max: 40)
 - Patient 2 (Max: 26)
- IOP normalized after stopping difluprednate drops and:
 - Adding brimonidine/timolol 0.2%/0.5% for Patient 1.
 - Adding travoprost 0.004% for Patient 2.

- The hydration theory offers a complementary model to help further explain macular hole development.
- Macular edema may lead to hole persistence by precluding edge approximation.
- Steroids may be useful in reducing this fluid and lead to hole closure.

- These holes may have closed spontaneously and not in response to drug exposure.
- The "on- and-off" phenomenon of hole closure and re-opening with steroid exposure and removal, supports the notion of steroid efficacy.

- There may be a role for topical steroids in the treatment of macular holes, particularly after vitrectomy.
- Steroid-induced ocular hypertension should be considered when utilizing topical drops in this population.

- This study is limited by:
 - Small sample size
 - Retrospective design
 - Lack of control group
- Further studies should investigate the utility of topical steroids in the treatment of this disease process in a prospective, randomized, and controlled design.