MACULAR HOLE CLOSURE WITH TOPICAL STEROIDS

Sofia Prenner, Howard F. Fine, MD, MHSc
FINANCIAL DISCLOSURE

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

• 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)
    • $p = 0.14$
RESULTS

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

• 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
RESULTS

• 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
RESULTS

• 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.
DISCUSSION

• 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.
DISCUSSION

• 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.
DISCUSSION

• 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.