Bilateral Corneal Endothelial Decompensation Following Airbag-Induced Trauma

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CASE PRESENTATION

A male patient in his late 80s presented with diminished vision in both eyes following a road traffic accident involving airbag deployment (figure 1). A CT scan indicated no facial or skull fractures. Initial visual acuity was measured at 3/60 in the right eye (RE) and perception of light in the left eye (LE). Clinical examination of the RE showed lower lid ectropion, a small corneal abrasion, corneal oedema, fibrin in the anterior chamber, minimal hyphaema and multiple iris stromal tears were noted, with the pupil non-reactive and small. The LE exhibited a large superior corneal abrasion and severe corneal oedema, alongside fibrin and hyphaema, and traumatic mydriasis. B-scan ultrasound confirmed a flat retina, with intraocular pressures recorded at 15mm Hg in the RE and 13mm Hg in the LE. The optic nerve appeared normal, and the macula appeared healthy with no macular oedema. The management plan included preservative-free (PF) cyclopentolate 1% two times per day and PF dexamethasone eye drops six times per day in both eyes for traumatic uveitis, along with PF chloramphenicol eye drops four times per day and chloramphenicol ointment at night for the abrasions. On follow-up the next day, epithelial defects had resolved, and the patient reported slight improvement in vision. Both corneas remained significantly oedematous with Descemet's folds. Further examination revealed iridodialysis and phacodonesis in the RE, with a slightly improved view of the anterior segment.

At 1-week post-accident, there was resolution of fibrin and hyphaema in both eyes, along with slight improvement in corneal oedema in the LE and significant improvement in the RE. Visual acuity improved to 2/60 in the LE and 6/18 in the RE.

At 1-month, visual acuity stabilised at 6/18 in the RE and 3/60 in the LE. Although the LE showed slight improvement in corneal oedema, it remained grossly oedematous despite the resolution of anterior segment inflammation, suggesting permanent endothelial dysfunction. Specular microscopy was performed at this visit and showed RE 2198 cells and LE 619 cells.

We hypothesise that direct impact and subsequent deformation of the cornea may have resulted in endothelial cell loss, which was supported by subsequent specular microscopy showing a significantly reduced cell count. While we do not have preinjury endothelial cell count by way of control, the cell count in the RE measured once visual acuity stabilised to 6/18 was within normal range, which suggests that the preinjury cell count was also likely normal. Aside from the reduction in endothelial cell count, we could not find a cause for the diminution of vision in the left eye.

The patient remains under our care and will be a candidate for corneal endothelial graft if there is no subsequent improvement in the LE. He will need life-long monitoring of his intraocular pressures due to the iridodialysis and angle trauma.

DISCUSSION

Corneal endothelial decompensation following airbag deployment is exceedingly rare but can lead to considerable vision impairment if not managed promptly. A review of the literature via PubMed revealed only two similar cases: one involving a middle-aged woman with localised corneal decompensation in the left eye and another involving a woman in her late twenties who presented with corneal oedema and Descemet's folds in the left eye after a motor vehicle accident with airbag deployment. [1 2] In both cases, specular microscopy confirmed endothelial cell loss. To our knowledge, this is the first reported case of bilateral corneal decompensation following airbag trauma.

During RTAs, airbags deploy at extreme speeds, causing blunt trauma, particularly to individuals seated close to the airbag. While airbags are designed to enhance safety, their rapid inflation generates forces capable of damaging delicate ocular structures. The JAMA network reports that approximately 3% of individuals involved in RTAs with airbag deployment sustain ocular injuries. [3]

Common ocular traumas, such as corneal abrasions, hyphaema and subconjunctival haemorrhage, are often readily identifiable. In contrast, endothelial damage can be subtle, leading to delayed symptoms such as blurred vision and corneal oedema. Clinicians should maintain a high index of suspicion for endothelial damage in patients presenting with post-accident ocular trauma and consider early referral to ophthalmology in such cases.

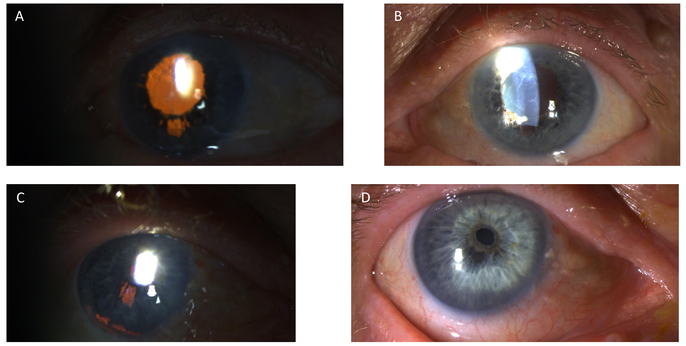


Figure 1 – Anterior Segment Photos
A,B – LE showing traumatic mydriasis, iris transillumination defects, corneal oedema. C,D – RE showing small, non-reactive pupil, iris transillumination defect, ectropion.

Learning points

- ► All patients involved in road traffic accidents with airbag deployment and facial trauma should undergo comprehensive ophthalmological evaluation.
- ► Corneal decompensation can occur in the absence of visible corneal abrasions, necessitating a high degree of clinical suspicion.
- ► Corneal grafting is an option for patients with corneal decompensation that fail to respond to medical management, in this case, the patient is a candidate for endothelial grafting.

SUMMARY

Approximately 3% of individuals in road traffic accidents suffer ocular injuries.

We present a case of a man in his late 80s who presented with bilateral corneal decompensation following airbag deployment during a road traffic accident.

Ocular examination revealed multilevel ocular injury with severe bilateral corneal oedema. This was managed with topical steroids, mydriatics and antibiotic therapy.

At 1-month follow-up, the left eye had persistent corneal oedema. Specular microscopy was performed which confirmed endothelial cell loss making the patient a candidate for a corneal endothelial graft.

We observed blunt trauma from airbag deployment causing endothelial cell loss and subsequent corneal decompensation.

Our findings suggest that patients presenting to the emergency department with facial trauma from airbag injury should be referred for comprehensive ophthalmological assessment and that the reviewing ophthalmologist should have endothelial cell loss in mind during examination.

REFERENCES

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