

Persistent loss of marginal corneal arcades after chemical injury

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Changes in the limbal microvasculature following a chemical eye injury are essential for prognosis and management. At the slit lamp, it can be difficult to assess, here using fluorescein and indocyanine green angiography we show that anterior segment angiography may be informative to assess objectively the limbal microvascular changes over the follow-up period.

Key words: Anterior segment OCT, anterior segment angiography, limbal ischemia, marginal corneal arcades, ocular chemical burn

Chemical injury to the ocular surface is an ophthalmic emergency that may result in damage to the marginal corneal arcades (MCA), limbal microvasculature, and limbal epithelial stem cell niche.^[1-3] Accurate evaluation and monitoring of this microvasculature is essential for prognosis and management, if assessed at the slit lamp it can be challenging.^[1,2] Fluorescein and indocyanine green angiography (FA and ICGA), and to a lesser extent optical coherence tomography angiography (OCTA), have been shown to be informative in the diagnosis of these vascular changes.^[4-6]

There is little information, however, on long term longitudinal changes to the MCA and limbal vasculature following a chemical injury, which may be of importance in planning clinical and surgical interventions that depend on a healthy supportive peri-limbal microvasculature. We report the vascular changes that occur in a patient over a 6-year time period following a chemical injury.

Case Report

A 57-year-old lady presented with a stage II (Roper-Hall classification^[7]) alkaline chemical injury to her right eye. At

presentation, her best-corrected visual acuity (BCVA) was 20/30 Snellen. She had an inferonasal epithelial defect crossing the limbus with corneal stromal haze and a clinical appearance of limbal ischemia over four clock hours [Fig. 1a]. She was treated with abundant irrigation, followed by topical steroids, antimicrobials, ascorbic acid, and systemic tetracycline. The patient was followed up with regular slit-lamp biomicroscopy, ICGA, and FA using a scanning laser ophthalmoscope (HRA2; Heidelberg Engineering, Heidelberg, Germany) to quantify the area of limbal ischemia and vascular changes. At days 1 and 7 after presentation, there was evident inferonasal limbal ischemia with extensive loss of the MCA, accompanied by both early and late fluorescein and indocyanine green leakage [Fig. 2].

Three months after the injury, although there was a partial recovery of the peri-limbal vasculature, areas of loss of vascularity, especially the MCA remained, evident on ICGA [Fig. 2]. Recovery of the peri-limbal vasculature and MCA continued, however, even after six years, there were still areas of capillary loss and earlier fluorescein leakage in comparison to the unaffected fellow eye [Fig. 2]. BCVA remained at 20/20 and clinically there was residual corneal scarring with lipid deposition, the ocular surface remained stable with no epithelial breakdown [Fig. 1b].

Discussion

Clinical assessment of limbal ischemia after chemical injury is challenging and studies have demonstrated low clinical agreement and inter-observer consistency.^[1] FA and ICGA are useful tools in identifying, quantifying, and monitoring the MCA and limbal vasculature.^[2,4,5] Our case of chemical injury highlights the limits of clinical color photography in detecting these vascular abnormalities. It is of note that the vascular abnormalities remained over a 6-year period and were not apparent on slit-lamp biomicroscopy or color photography. Although OCTA is an alternative and less invasive procedure than FA and ICGA, it provides limited information about vessel maturity. Early fluorescein leakage is a maker of vessel immaturity and or vessel damage. ICG does not leak from healthy vessels and the leakage present in this case indicates the extent of the vascular damage. This indicates that even in those areas where the vasculature was present, the vessels were damaged and unlikely to provide adequate support to the limbal tissue. This is important as Huang *et al.* reported that limbal epithelial stem cells cannot maintain their stemness or proliferation without the support of the limbal microvasculature net microenvironment.^[8]

Conclusion

Monitoring the changes in the MCA and limbal vasculature following a chemical injury using FA and ICGA is important to assess the vascular injury and the extent of its recovery.

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Figure 1: Color anterior segment photographs of the patient's right eye, showing (a) six clock hours of nasal limbal ischemia with five clock hours of associated corneal opacity, (b) resolved limbal ischemia and clear cornea 6 years post-injury

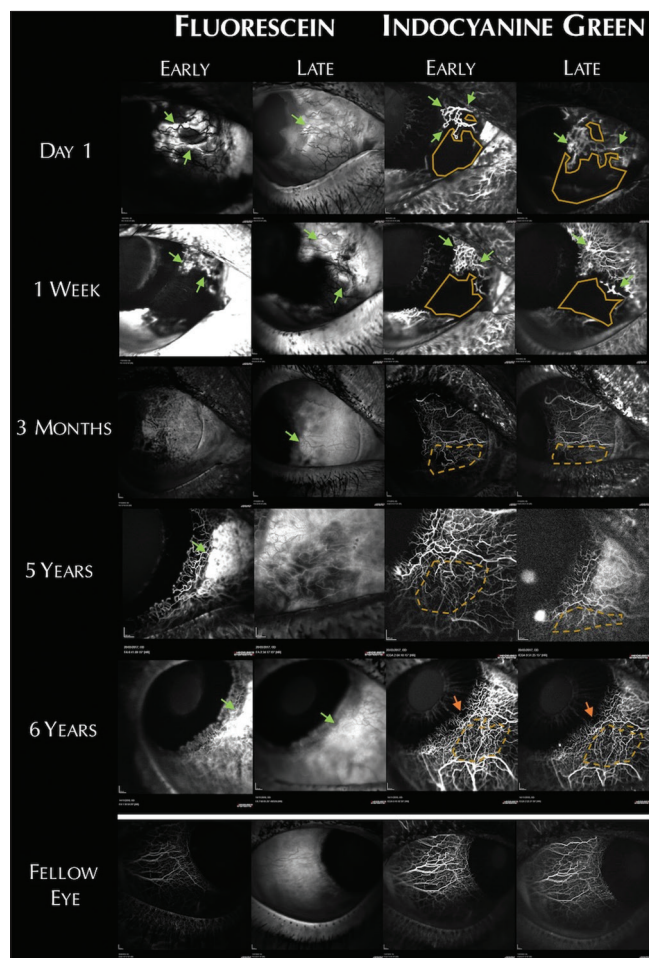


Figure 2: Serial fluorescein and indocyanine green angiographs early and late phase at different time points. Images of the fellow eye to compare the pattern and the restored peri-limbal vasculature post-injury. Green arrows show leakage. Orange arrows show loss of marginal corneal arcade. Yellow lines show the ischemic area

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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Conflicts of interest

There are no conflicts of interest.

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