Chemical Injuries to the Cornea: Case Report, Review and Treatment

By:

Previous candidate. Actual case report submission

Abstract

Purpose. To present a case study and discussion on the medical and surgical management of chemical injuries to the cornea.

Case Report. A 58-year-old male presented to the office after one day duration of being splashed with NaOH (Lye) in his left eye under high pressure. Visual acuity was 20/100 with erythema and edema to the lids and lashes. His conjunctiva had 2-3+ injection with an inferior defect and approximately 2½ clock hours of limbal involvement. The cornea had a 95% epithelial defect with sloughed tissue along the edges and the anterior chamber view was hazy but had appreciable cell. The patient was diagnosed with a Grade II alkali burn. Vigamox (moxifloxacin) was prescribed every two hours and Pred Forte (prednisolone acetate) initiated every hour, as well as Atropine two times a day, Ciloxan (ciprofloxacin) ointment at night and non-preserved artificial tears every 1-2 hours. Topical citrate and ascorbate were prescribed four times a day through a compounding pharmacy. After three months of intensive therapy the cornea had residual areas of punctate keratopathy and stromal scarring, but the patient was comfortable on non-preserved tears and best corrected to 20/20.

Conclusions. Chemical burns are one of the most urgent ocular emergencies and prompt medical management by the primary eye care provider with high dose corticosteroids, and other anti-inflammatories, can help prevent ocular morbidity.

Key Words: Acid, Amniotic Membrane, Alkali, Chemical Injuries, Corticosteroids, Limbal Stem Cells

Introduction

Chemical burns are one of the most urgent ocular emergencies. They pose a significant threat to vision, and represent a potentially blinding injury that has substantial ocular morbidity. The prognosis of a burned eye depends not only on the severity of the burn, but also the insulting agent, the rate at which therapy is initiated, and how therapy is prescribed. Chemical burns may be caused by either acids or alkaline agents. Acids tend to cause less damage than alkalis as many corneal proteins bind the acid and act as a chemical buffer. Alkaline agents are particularly damaging as they have both hydrophilic and lipophilic properties. This allows them to rapidly penetrate cell membranes and quickly enter the anterior chamber.[1] Recovery from ocular burns is dependent on the extent of damage to corneal, limbal and conjunctival tissues at the time of injury. Damage to intraocular structures further influences the final visual outcome. Classification schemes help provide guidelines based on the corneal appearance and extent of limbal and conjunctival involvement, and allow the clinician to determine the severity of the injury and the prognosis for the injured eye.

Case Report

Incident – 4/9/2008

Dennis L, a 58-year-old male was splashed with NaOH (Lye) in his left eye under high pressure on April 9, 2008. He was working as a laundry consultant several hundred miles away from home in Iowa, and was fixing a clogged drainage pipe at the time of the injury. He typically wears myopic prescription eyewear, but removed his glasses to allow him to see better down the clogged pipe. Unfortunately, he was not wearing safety glasses at the time. His eyes were immediately flushed on site and then driven directly to an urgent care facility, where they repeated irrigation and neutralized the ocular pH. He was prescribed Vigamox (moxifloxacin) four times a day, and told to follow up in 24 hours in his home town.

Initial Visit – 4/10/2008

When presenting to the office on April 10, 2008, DL complained that his vision was blurry and that the eye was uncomfortable. His pH was immediately tested and was found to be 7 in both the right and left eye. His ocular and medical histories were unremarkable, and he was not taking any medications, and denied any allergies to known medications. Pupils, extraocular muscles and confrontation fields were intact OU. Visual acuity with correction (OD +0.75 -1.25 x 157, OS -0.50 -0.75 x 090, Add +2.00 OU) in the right eye was 20/20, and 20/100 (pinhole 20/40) in his left affected eye.

Slit lamp examination of his right eye was unremarkable. Evaluation of his left eye showed 2-3+ erythema and edema to the lids and lashes. The conjunctiva had 2-3+ injection with an inferior defect of approximately 2½ clock hours of limbal involvement (ie, ischemia, staining and blanching over the limbal area between the clock hours of 4:30-7). The cornea had a 95% epithelial defect with sloughed tissue along the edges and the anterior chamber view was hazy but had trace-to-1+ cell. The iris and lens was clear. Tonometry by applanation was 14mmHg OU using Fluress. He was dilated with 1% tropicamide and the cup to disc ratio was .35 round OU. The macula and peripheral retina was intact in both eyes.

The differential diagnosis in this case consisted of the type of chemical that got splashed into the eye, and the extent of damage it created, from Grade I-IV. He was initially diagnosed with a Grade II alkali burn from NaOH. However, since the extent of limbal involvement can progress the first few days following a chemical injury, his severity level was reassessed at each visit, monitoring for progression into a Grade III or IV injury.

To help control inflammation, his cornea was scraped to remove the sloughed, necrotic tissue. Leaving it behind would have created a nidus for further inflammation.

His Vigamox was continued, but increased to every two hours. And since there is a very short window to control inflammation following an alkali burn, Pred Forte (prednisolone acetate) was initiated at every hour. He was instructed to keep at that high dose for approximately one week. In addition, Atropine was added two times a day and Ciloxan (ciprofloxacin) ointment at night, with non-preserved artificial tears every 1-2 hours. Topical citrate and ascorbate was prescribed four times a day through a compounding pharmacy, also as an adjunct to control inflammation and promote stromal keratocyte activation and collagen development. A bandage contact lens was not used due to the fact that his limbal area was too vulnerable and it would create further irritation.

A long discussion was had with DL on the nature of the chemical he encountered (NaOH), and its potential to do real harm long term. Lye is one of the most potent alkali agents, and has the potential to enter into the anterior chamber in seconds, and although the cornea was intact, the next couple of weeks would be critical in determining his long term prognosis. Despite the fact the chemical was neutralized, further damage to his limbal stem cells, surrounding ocular adnexa, ciliary body and lens may appear in time, and his condition will have to be monitored closely.

DL was instructed to return to the office in one day.

1 day follow up - 4/11/2008

DL reported stating he believed his vision was somewhat better, but that the eye was a little swollen and somewhat uncomfortable. He attempted to be compliant with his medications, but stated he was using Vigamox and Pred Forte both every hour OS because he couldn't remember which one was which (and he lost the written instructions he was given). He was using the Atropine BID, Ciloxan ung qhs, and non-preserved artificial tears q1-2h OS. He attempted to get the topical citrate and ascorbate through the compounding pharmacy as recommended, but did not since it was not covered by his insurance and the cost was too high.

His corrected vision through his current eyewear was 20/20 OD and 20/50 OS PH 20/25. The right pupil had normal direct and consensual response, but the left pupil showed no movement due to Atropine. There was no APD detected by reverse testing. Extraocular muscles and confrontation fields were intact OU. On slit lamp examination his right eye continued to show no effects from the chemical and remained unremarkable.

Evaluation of his left eye showed 2-3+ erythema and edema to the lids and lashes. The conjunctiva had 2-3+ injection. His left cornea continued to have a large epithelial defect, which was graded somewhat larger than the previous day at almost 100%. This was expected, since he was debrided the previous day, and any damaged and necrotic epithelium will typically denude itself. There was also 2-3+ descemet fold and striae in the cornea along with haze developing in the stroma due to edema. The limbus however still showed about 2½ clock hours of limbal ischemia and blanching with overlying stain between the clock hours of 4:30-7, and did not appear to expand from the previous day. His anterior chamber view was a little more difficult to view due to the stromal haze, but 1+ cell was noted OS. The iris was dilated but clear as was the lens. Tonometry by applanation was 14mmHg OS using Fluress. His pupil was already dilated from the Atropine and the cup to disc ratio was .35 round OS. The macula and peripheral retina was intact OS.

DL confirmed that Lye was indeed the chemical in the laundry pipe and brought in the MSDS sheet from his employer confirming NaOH as the cause of the damage which had a pH of 12. The severity of his damage was reassessed, and his diagnosis of Grade II alkali burn from NaOH remained since he had less than 4 clock hours of involvement (i.e., Grade II = < 1/3 limbal involvement). Nonetheless, his severity was reassessed over the next several visits.

He was instructed to continue the Vigamox and Pred Forte both at every hour. Doing both every hour helped him be more compliant, and since he is at increased risk for a secondary bacterial infection following a chemical injury, the additional antibiotic may help him suppress that response. His Atropine was continued at two times a day, as well as his Ciloxan (ciprofloxacin) ointment at night, with non-preserved artificial tears every 1-2 hours.

It was discussed with DL that the results from today's exam were stable, and his condition remained guardedly optimistic. There is still a long way to go, but there was no further involvement of his limbal area. Nonetheless, the goal is still the same. Suppress the inflammation, and get his epithelial defect to close in.

DL was instructed to return to the office in one day.

2 day follow up – 4/12/2008

DL reported stating his eye was starting to experience a little discomfort and pain. He was using the medications as prescribed. Vigamox and Pred Forte both every hour OS, and Atropine BID and Ciloxan ung qhs, along with non-preserved tears OS as often as he could get them in.

His corrected vision through his current eyewear was 20/20 OD and 20/70 OS PH 20/40. The right pupil had normal direct and consensual response, but the left pupil showed no movement due to Atropine. There was no APD detected by reverse testing. Extraocular muscles and confrontation fields were intact OU. On slit lamp examination the right eye continued to show no effects from the chemical and remained unremarkable.

Evaluation of his left eye showed 1-2+ erythema and edema to the lids and lashes. The conjunctiva had 2-3+ injection. The left cornea continued to have a large epithelial defect, but was starting to close in on his superior nasal and temporal edges. The defect was graded at 90%. There continued to be 2-3+ descemet fold and striae noted in the cornea, along with haze in the stroma due to edema. His limbus still showed only 2½ clock hours of limbal ischemia and blanching, with overlying stain between the clock hours of 4:30-7, and did not appear to expand from the previous day. The anterior chamber view had 1+ cell OS. The iris was dilated but clear as was the lens. Tonometry by applanation was 16mmHg OS using Fluress. He was already dilated from the Atropine and the cup to disc ratio was .35 round OS. The macula and peripheral retina was intact OS.

The diagnosis of Grade II alkali burn from NaOH remained and was confidently discussed with DL since he started to get a little re-epithelialization at this visit. It may not progress to a Grade III, but he will continually be reassessed.

He was instructed to continue the Vigamox and Pred Forte both at every hour. And continue Atropine two times a day, and Ciloxan (ciprofloxacin) ointment at night, with non-preserved artificial tears every 1-2 hours.

DL was instructed to return to the office in two days.

4 day follow up – 4/14/2008

DL reported stating his eye was feeling a little better but didn't think there was any change in his vision. He was using the medications as prescribed. Vigamox and Pred Forte both every hour OS, and Atropine BID and Ciloxan ung qhs, along with non-preserved tears OS as often as he could get them in.

His corrected vision through his current eyewear was 20/20 OD and 20/80 OS PH 20/40. The right pupil had normal direct and consensual response, but the left pupil showed no movement due to Atropine. There was no APD detected by reverse testing. Extraocular muscles and confrontation fields were intact OU. On slit lamp examination the right eye continued to show no effects from the chemical and remained unremarkable.

Evaluation of his left eye showed 1-2+ erythema, and 1+ edema to the lids and lashes. The conjunctiva had 2-3+ injection. The left epithelial defect on the cornea had closed in a bit more over the last two days on both the nasal and temporal edges, and there was even some movement inferiorly. The defect was graded at 75%. There continued to be 2-3+ descemet fold and striae noted in the cornea, along with haze in the stroma due to edema. His limbus still showed only about 2clock hours of limbal ischemia and blanching but the conjunctiva overtop was beginning to fill in and took less stain. The anterior chamber view had 1+ cell OS. The iris was dilated but clear as was the lens. Tonometry by applanation was 16mmHg OS using Fluress. He was already dilated from the Atropine and the cup to disc ratio was .35 round OS. The macula was intact OS.

His condition remained stable as a Grade II alkali burn from NaOH and he was instructed to continue the Vigamox and Pred Forte both at every hour. He was also to continue Atropine two times a day, and Ciloxan (ciprofloxacin) ointment at night, with non-preserved artificial tears every 1-2 hours.

It was discussed with DL that his condition was improving and remained guardedly optimistic.

DL was instructed to return to the office in three days.

1 week follow up – 4/17/2008

DL reported stating his eye had been a little sore over the last day or so, and felt it was more uncomfortable in the mornings, compared to the afternoon. He was using the medications as prescribed. Vigamox and Pred Forte both every hour OS, and Atropine BID and Ciloxan ung qhs, along with non-preserved tears OS as often as he could get them in.

His corrected vision through his current eyewear was 20/20 OD and 20/50 OS PH 20/40. The right pupil had normal direct and consensual response, but the left pupil showed no movement due to Atropine. There was no APD detected by reverse testing. Extraocular muscles and confrontation fields were intact OU. On slit lamp examination the right eye continued to show no effects from the chemical and remained unremarkable.

Evaluation of his left eye showed 1+ erythema and edema to the lids and lashes. The conjunctiva had 1-2+ injection. The left epithelial defect was finally starting to close in on all sides, including inferiorly. There still remained a central epithelial defect that was graded at 50%. There continued to be 2+ descemet fold and striae noted in the cornea, along with haze in the stroma due to edema. The limbus showed 2 clock hours of limbal ischemia and blanching but the conjunctival defect overtop healed in. The anterior chamber had Trace to 1+ cell OS. The iris was dilated but clear as was the lens. Tonometry by applanation was 9mmHg OS using Fluress. He was already dilated from the Atropine and the cup to disc ratio was .35 round OS. The macula was intact OS.

His condition remained stable as a Grade II alkali burn from NaOH and he was instructed to decrease the Vigamox to QID and Pred Forte to BID. He was also to cut back on the Atropine to QD. To help support lubrication and anti-infective therapy, his Ciloxan (ciprofloxacin) ointment was increased to TID, and non-preserved artificial tears every 1 hour.

It was discussed with DL that his condition was improving and remained guardedly optimistic. At this point his Pred Forte needed to be drastically reduced.

He was educated that while Pred Forte is of considerable value in reducing inflammation and tissue destruction following this type of chemical injury. It also interferes with proper healing, and must be used with caution after the end of the first week following a severe chemical injury, or it could actually create the progressive thinning we are trying to prevent. A Progestational steroid, which doesn't impede healing as much as a corticosteroid, was recommended and prescribed through a compounding pharmacy, but since it was not covered under his insurance plan, he refused.

DL was instructed to return to the office in three days, but reported he was going out of town for a week. Although it was discussed that this was a crucial time in his healing, and he needed to stay close to the office, he reported he need to travel for work.

DL returned to the office in 8 days.

2 week follow up – 4/25/2008

DL reported stating he was unable to come in sooner and needed to go out of town. He felt his vision has been improving but was running out of medications. He was using the medications as prescribed. Vigamox QID, Pred Forte BID, Atropine QD and Ciloxan ung TID, along with non-preserved tears q1h OS.

His corrected vision through his current eyewear was 20/20 OD and 20/80 OS PH 20/50. The right pupil had normal direct and consensual response, but the left pupil showed no movement due to Atropine. There was no APD detected by reverse testing. Extraocular muscles and confrontation fields were intact OU. On slit lamp examination the right eye continued to show no effects from the chemical and remained unremarkable.

Evaluation of his left eye showed trace erythema and edema to the lids and lashes. The conjunctiva had 1+ injection. The left epithelial defect had almost completely healed in. There still remained a tiny central area that stained with fluorescein and was graded at 1%. There continued to be 2+ descemet fold and striae noted in the cornea, along with haze in the stroma due to edema. The limbus showed 2 clock hours of limbal ischemia and blanching but no conjunctival defect. The anterior chamber view was still hazy but appeared to be free of cell. The iris was dilated but clear as was the lens. Tonometry by applanation was 20 mmHg OS using Fluress. He was already dilated from the Atropine and the cup to disc ratio was .35 round OS. The macula was intact OS.

It was discussed with DL that his condition was improving and that he should feel more optimistic about his recovery. He had made it past the critical two week period without a stromal melt, and had almost completely re-epithelized.

His condition was upgraded to improving Grade II alkali burn from NaOH and he was instructed to continue the Vigamox at QID, Pred Forte BID and Atropine QD. As well as the Ciloxan ointment TID and non-preserved artificial tears every 1 hour.

DL was instructed to return to the office in one week. He was again going out of town and would do his best to be back in a week.

DL returned to the office in 10 days.

3 ½ week follow up – 5/5/2008

DL reported stating his vision has been improving but feeling dry. He also reported some light sensitivity but felt this was improving as well. He was using the medications as prescribed. Vigamox QID, Pred Forte BID, Atropine QD and Ciloxan ung TID, along with non-preserved tears q1h OS.

His corrected vision through his current eyewear was 20/20 OD and 20/60 OS PH 20/25. The right pupil had normal direct and consensual response, but the left pupil showed no movement due to Atropine. There was no APD detected by reverse testing. Extraocular muscles and confrontation fields were intact OU. On slit lamp examination the right eye continued to show no effects from the chemical and remained unremarkable.

Evaluation of his left eye showed trace erythema and resolved edema to the lids and lashes. The conjunctiva had Tr-1+ injection. The left epithelial defect had finally completely healed in. There was some superficial punctate keratitis but no defect. There was 1+ descemet fold, but he still had haze in the stroma. The limbus was starting to show signs of vascularization over the previously blanched areas and there was no conjunctival defect. The anterior chamber remained free of cell. The iris was dilated but clear as was the lens. Tonometry by applanation was 20 mmHg OS using Fluress. He was already dilated from the Atropine and the cup to disc ratio was .35 round OS. The macula was intact OS.

It was discussed with DL that his condition was continuing to improve and looked very optimistic. He was continuing to heal as hoped, and has not shown any signs of a corneal melt or IOP spike, and he showed no signs of ocular adnexa sequelae.

His condition was stable at an improving Grade II alkali burn from NaOH, and he was instructed to discontinue the Vigamox and Atropine, but continue the Ciloxan ointment TID and non-preserved artificial tears every 1 hour. Since his epithelial defect had finally healed in, and his acute risk of a stromal melt had passed, the Pred Forte was increased to QID to help deter scarring of his stroma. The non-preserved artificial tears were continued every 1-2 hours to help with his dryness, but he was educated that this could be a long term side effect, and that he may need to consider additional treatment options for it in the future (ie, punctual plugs, Restasis ophthalmic emulsion, etc).

DL was instructed to return to the office in 10 days.

5 ½ week follow up – 5/15/2008

DL reported stating his eye was starting to feel much better but still has a little dryness, especially in the morning. He was using the medications as prescribed. Pred Forte QID, Ciloxan ung TID, along with non-preserved tears q2h OS.

His corrected vision through his current eyewear was 20/20 OD and 20/40 OS PH 20/25. The pupils were equal round and reactive to light and showed no afferent pupillary defect. Extraocular muscles and confrontation fields were intact OU. On slit lamp examination the right eye continued to show no effects from the chemical and remained unremarkable.

Evaluation of his left eye showed trace erythema to the lids and lashes. The conjunctiva had trace injection. The epithelium continued to remain intact but there was 2+ superficial punctate keratitis. The descemet fold was less at trace-to-1+, but there was still haze in the stroma. The limbus continued to show signs of vascularization over the previously blanched areas and there was no conjunctival

defect. The anterior chamber remained free of cell. The iris was clear as was the lens. Tonometry by applanation was 20 mmHg OS using Fluress.

It was discussed with DL that his condition was continuing to improve and looked very optimistic. His more serious issues are behind him, but his dryness may be persistent or a byproduct of a month and a half of copious topical medications, etc.

His Grade II alkali burn from NaOH was resolving. He was instructed to discontinue the Ciloxan ointment and replace it with Refresh PM ointment TID. His Pred Forte was cut to BID for two weeks then QD for two additional weeks and then discontinued. The non-preserved artificial tears were continued every 1-2 hours.

DL was instructed to return to the office in 4 weeks.

9 week follow up – 6/9/2008

DL reported stating he felt his vision had improved a lot over the last month. He was using the medications as prescribed and had tapered off the Pred Forte. He was currently taking Refresh PM ointment TID along with non-preserved tears q2-4h OS.

His corrected vision through his current eyewear was 20/20 OD and 20/25 OS PH 20/20. The pupils were equal round and reactive to light and showed no afferent pupillary defect. Extraocular muscles and confrontation fields were intact OU. On slit lamp examination the right eye continued to show no effects from the chemical and remained unremarkable. Manifest refraction OS -1.25 -0.75 x 098 20/20 (almost a diopter shift in myopia).

Evaluation of his left eye showed almost normal appearing lids, lashes and conjunctiva. The epithelium continued to remain intact and there was trace-to-1+ superficial punctate keratitis. The descemet fold was gone, but there was still haze in the inferior nasal stroma. The limbus continued to show signs of vascularization over the previously blanched areas and there was no conjunctival defect. The anterior chamber remained free of cell. The iris was clear as was the lens. Tonometry by applanation was 19 mmHg OS using Fluress.

It was discussed with DL that his condition was continuing to improve, and he was pleased with the outcome. He was educated that he does appear to have some residual scarring of his cornea, but it was not affecting his vision and may continue to improve over time. His spectacle Rx has also changed, but he would wait to update it, to be sure the increased myopia was permanent.

His Grade II alkali burn from NaOH was resolving nicely. He continued the Refresh PM ointment TID, and non-preserved artificial tears every 2-4 hours.

DL was instructed to return to the office in 6 weeks.

3 month follow up – 7/14/2008

DL reported stating he felt things have been going well with his eyes. The left one still felt dry on occasion, but he is using the Refresh PM ointment only at night and non-preserved tears 3-4x a day OS.

His corrected vision through his current eyewear was 20/20 OD and 20/25+ OS PH 20/20. The pupils were equal round and reactive to light and showed no afferent pupillary defect. Extraocular muscles and confrontation fields were intact OU. Manifest refraction OD +1.00 -0.75 x 168 20/20, OS -0.50 -1.25 x 094 20/20 +2.25 add OU (closer to his original rx OS). Atlas topography showed K's OD 43.87 @ 180, 44.50 @ 090; OS 44.25 @094, 45.37 @004 along with a central flattening inferiorly over scarred area OS.

On slit lamp examination the right eye continued to show no effects from the chemical and remained unremarkable. His left cornea still showed areas of punctate keratopathy, but less stromal scarring OS. The limbus continued to show signs of vascularization over the previously blanched areas and there was no conjunctival defect. The anterior chamber remained free of cell. The iris was clear as was the lens. Tonometry by applanation was 16mmHg OD, 14 mmHg OS using Fluress. The cup to disc ratio was .35 round OU. The macula was intact OS.

It was discussed with DL that he was stable, and both he and his doctor were pleased with the outcome. He was educated that the residual scarring of the cornea was improving, and the change in his spectacle Rx was less, and that he was actually seeing quite well out of his current pair of glasses.

His long term prognosis was also discussed, and although it appears favorable, he is still at risk of developing glaucoma, and he will most likely continue to have persistent dry eye issues due to loss of the limbal stem cells, and may potentially have other ocular sequelae that cannot be predicted. He should follow up in 3 months (6 months post injury) and then yearly.

Long Term Outcome 2013

The patient has been seen yearly since the trauma occurred in 2008 and continues to do well. He does have persistent dry eye complaints, but this is normal and he understands the outcome could have been much worse. He was lucky in that he received attention immediately following the trauma, and irrigated appropriately on site, and at the urgent care clinic. We also did our best to suppress the more intense second wave of inflammation by treating him aggressively for the first week, and I feel we did our part to help prevent a stromal melt. He has also been fortunate long term because he has not developed an IOP spike, or had any other ocular sequelae.

Although this patient was seen in 2008, if I were to see them today, there is a couple of things I would do differently. First, I would have placed a sutureless amniotic membrane, most likely a Prokera Classic, on the eye within the first 24 hours. Their ease of use, and ability to utilize chair side, make them an excellent adjunct for the chemical injury patient. I may have also used Durezol instead of Pred Forte every hour to help get an added anti-inflammatory effect from both the medication and membrane (but insurance coverage may have dictated otherwise with this patient). Another option we may utilize now would also be autologous vs. umbilical cord serum. Both have merit for the chemical injury patient, and if we can make work with the logistics around obtaining it in our area, we typically do.

Follow up visits OS	**4/10** (initial visit)	**4/11** (1 day F/U)	**4/12** (2 day F/U)	**4/14** (4 day F/U)	**4/17** (1 week F/U)	**4/25** (2 week F/U)	**5/5** (3 ½ week F/U)	**5/15** (5 ½ week F/U)	**6/9** (9 week F/U)	**7/14** (3 month F/U)
Visual Acuity	20/100 PH 20/40	20/50 PH 20/25	20/70 PH 20/40	20/80 PH 20/40	20/50 PH 20/40	20/80 PH 20/50	20/60 PH 20/25	20/40 PH 20/25	20/25 PH 20/20	MR 20/20
Lids / Lashes	2-3+ Erythema and edema	2-3+ Erythema and edema	1-2+ Erythema and edema	1-2+ Erythema and 1+ edema	1+ Erythema and edema	Tr Erythema and edema	Tr Erythema and no edema	Tr Erythema	Normal	Normal
Conj	2-3+ injection	2-3+ injection	2-3+ injection	2-3+ injection	1-2+ injection	1+ injection	Tr-1+ injection	Tr injection	Normal	Normal
Cornea	95% epi defect, Haze	100% epi defect Haze, 2-3+ folds	90% epi defect Haze, 2-3+ folds	75% epi defect, Haze, 2-3+ folds	50% epi defect, Haze, 2+ folds	1% epi defect, Haze, 2+ folds	0% epi defect, Haze, 1+ folds, SPK	Haze, Tr-1+ folds, SPK	Haze/ scar, No folds, SPK	Haze/ scar, SPK
Limbal Ischemia	2 ½ clock hours	2 ½ clock hours	2 ½ clock hours	2 clock hours	2 clock hours	2 clock hours	2 clock hours	2 clock hours	2 clock hours	2 clock hours
Ant Ch	1+ cell	1+ cell	1+ cell	1+ cell	Tr-1+ cell	D/Q	D/Q	D/Q	D/Q	D/Q
Iris / Lens	clear	clear	clear	clear	clear	clear	clear	clear	clear	Clear

Follow up visits OS	**4/10** (initial visit)	**4/11** (1 day F/U)	**4/12** (2 day F/U)	**4/14** (4 day F/U)	**4/17** (1 week F/U)	**4/25** (2 week F/U)	**5/5** (3 ½ week F/U)	**5/15** (5 ½ week F/U)	**6/9** (9 week F/U)	**7/14** (3 month F/U)
Plan	Vigamox Q2h Pred Forte Q1h Atropine BID Ciloxan Ung QHS Non-preserv Tears Q1h Topical Citrate and Ascorbate QID (pt never got)	Vigamox Qh1h Pred Forte Q1h Atropine BID Ciloxan Ung QHS Non-preserv Tears Q1h	Vigamox Qh1h Pred Forte Q1h Atropine BID Ciloxan Ung QHS Non-preserv Tears Q1h	Vigamox Qh1h Pred Forte Q1h Atropine BID Ciloxan Ung QHS Non-preserv Tears Q1h	Vigamox QID Pred Forte BID Atropine QD Ciloxan Ung TID Non-preserv Tears Q1h	Vigamox QID Pred Forte BID Atropine QD Ciloxan Ung TID Non-preserv Tears Q1h	D/ C Vigamox Pred Forte QID D/C Atropine Ciloxan Ung TID Non-preserv Tears Q1-2h	Taper Pred Forte BID x 2wks, then QD for 2wks and D/C D/C Ciloxan Ung Non-preserv Tears Q1-2h Add - Refresh PM ung TID	Non-preserv Tears Q1-2h Refresh PM ung TID	Non-preserv Tears Q4-6h Refresh PM ung QHS

Etiology of Chemical Burns

The incidence of chemical burns is 30 per 10,000 employees in the workforce, with the significant majority (82-90%) occurring in men.[1,2,3] The visual impact of chemical injuries is magnified in this population as they generally occur in patients in the prime of their life (16-45 years old). Alkali agents are twice as likely to be the causative agent of chemical burns compared to acids. Ninety percent of chemical burns are accidental, and 10% are intentional and are usually a result of assault. Two-thirds of chemical injuries occur in the workplace, while one-third occur in the home and a very small percentage are school based. Most workplace injuries occur at construction sites, chemical plants, and machine factories. Eye injuries account for 4-7% of all workplace injuries, but 84% of all eye injuries in the workplace are chemical burns. While safety goggles are helpful at home, they do very little to prevent the high impact and pressure of a chemical splashed in the workplace.[2] Fortunately, most chemical injuries are mild and have a good prognosis.

Anatomy

The corneal epithelium plays an important role in maintaining the smoothness of the optical surface, helping to prevent micro-organisms from infecting deeper tissue, keeping the stroma dehydrated, and regulating the metabolic activity of the stromal keratocytes. It is made up of 5-6 layers of non-keratinized stratified squamous cells which are free of goblet cells. It rapidly renews itself and loses its surface cells into the tear film every 4-6 days.[1] Normal corneal epithelium is maintained by continuous circumferential and centripetal movement of peripheral corneal epithelium toward the visual axis. This balances the apoptosis and cellular loss resulting from anterior movement of basal epithelial cells to the surface and into the tear film.

At the limbus, the epithelium will thicken to approximately 10 cell layers thick, and in the perilimbal conjunctiva, the ridges that are found perpendicular to the cornea are known as the *limbal palisades of Vogt*. These ridges make up the basal cell layer and lay protected deep in a thickened sheet of epithelial cells that contain a rich blood supply. This is where the limbal stem cells reside.[1] The limbal stem cells mediate corneal repair and renewal, are self-renewing and can proliferate indefinitely to survive the duration of the host's life. They also act as a barrier to conjunctival epithelial cells and prevent them from migrating on to the corneal surface.[1] Human limbal stem cells can be identified both in vivo and in vitro, through their expression of the p63 transcription factor.[3-4] When ocular burns destroy the limbus and create a limbal stem-cell deficiency, the cornea then has to acquire its epithelium by way of the conjunctiva. This invasion of conjunctival cells leads to neovascularization, chronic inflammation and stromal scarring, with opacification and loss of vision in a process known as conjunctivalization. Regardless of the source of regenerating epithelium, the rate of migration after a chemical injury is delayed due to persistent inflammation, epithelial basement membrane damage, and degradation of basement membrane fibronectin by plasminogen activator.[2] Epithelial migration may be enhanced by adequate ocular lubrication, reduction of inflammation, epidermal growth factor and fibronectin. It has been well documented that a sterile ulceration is never seen in the presence of an intact epithelium.[1]

An intact epithelium will effectively prevent the development of a microbial and /or sterile ulceration (non-microbial).

The stroma of the cornea is made up of 80% collagen and numerous keratocytes. Keratocytes are pluripotent cells of neuroectodermal origin and have the potential to function in varying capacities.[1] Their main function is the maintenance and regeneration of the corneal stroma. During development and repair they are capable of a wide variety of fibroblastic activity, including phagocytosis of collagen fibrils, secretion of collagen, production of glycosaminoglycan ground substance, collagenase and collagenase inhibitors. The metabolic function of the keratocytes is believed to be regulated by cytokines from the epithelium, inflammatory cells, and from other keratocytes. Keratocytes are typically static and show relatively low metabolic activity, with no appreciable synthesis of collagen or collagenase. Following injury however, keratocytes become active and are mobilized from adjacent regions to repopulate the area of injury, with migration beginning on the endothelial side. In moderately severe chemical injuries, complete repopulation may be delayed, taking as long as 14 days and may not occur at all in advanced injuries.

The conjunctival epithelium consists of two or more layers of stratified columnar epithelium with numerous goblet cells. Its role is to facilitate smooth movement of the eyelid over the cornea, help maintain normal lid-globe apposition, produce mucus essential for tear film stability, and provide vascular support to the limbus. The conjunctiva also has stem cells which are located deep in the fornix and migrate in a centripetal fashion out from the fornix to cover the bulbar and tarsal conjunctiva. They too can be compromised in an ocular burn though it is less likely.

Acids and Alkalis

The logarithmic measure of hydrogen ion concentration is known as pH. Substances with a pH less than 7 are acidic and those with a pH greater than 7 are basic or alkaline. Irritants are substances that have a neutral pH, and typically cause more of a foreign body sensation than true damage. Detergents are surfactants that are neither acidic or alkali. They are categorized by the charge they carry: cationic (positive), anionic (negative), non-ionic (none), and zwitterionic (dual charge).[1]

Acids have a pH below 7, and irreversible damage becomes more likely the closer the offending agent's pH gets to 2.5. The corneal epithelium offers protection against weak or diluted acids by binding to corneal proteins. This creates protein coagulation that acts as a barrier against further penetration. Acid burns are often non-progressive and superficial, except when hydrofluoric acid (HF) is involved. Due to its low molecular weight and small size, the fluoride ion can readily penetrate through the stroma. It acts more like an alkali than an acid, and can cause significant anterior segment destruction.[1] It also has the potential to cause fatal metabolic imbalances (hypercalcemia) when there is sufficient dermal penetration, typically with as little as 2.5% body burn.[7] Therefore, concurrent management with the patient's primary care physician is required in cases of hydrofluoric acid burns. The most common acids to cause ocular burns are sulfuric (H_2SO_4), sulfurous (H_2SO_3), hydrofluoric (HF), acetic (CH_3CO_2H), chromic (H2CrO4), and hydrochloric (HCl) acids.

Chemicals that carry a pH greater than 7 are basic or alkaline and are correlated with a much higher likelihood of ocular morbidity. As the pH rises, barriers to penetration are destroyed, allowing deeper penetration to anterior segment structures. Alkali burns penetrate the ocular structures more rapidly, are more destructive to collagen fibers, and can cause alterations to the trabecular meshwork in seconds. The most common causes of alkali injury are ammonia (NH_3), lye (NaOH), potassium hydroxide (KOH), magnesium hydroxide ($Mg[OH]_2$), and lime ($Ca[OH]_2$). Calcium hydroxide or lime, which is found in plaster and cement, is the most common cause of alkali burns in the workplace. Ammonium hydroxide is one of the most common causes of home based injuries, and unfortunately is also the most destructive alkali and can penetrate into the anterior chamber within seconds.

One factor that will determine the success of the treatment is the penetrability of the chemical (Table 1). With acids, the agent with the slowest rate of penetration is sulfuric acid, followed by hydrocholoric acid, and sulfurous acid. Hydrofluoric acid exhibits the fastest rate of penetration. The rate of alkali penetration from slowest to fastest is calcium hydroxide, followed by potassium hydroxide, sodium hydroxide, and ammonium hydroxide.[1]

Irrigation

The severity of chemical burn is a function of both toxicity and duration of exposure, which makes prompt irrigation the most critical step in the treatment plan. The duration of contact is directly correlated with the severity of the damage. Therefore, it is imperative to initiate flushing immediately after the eye comes in contact with a harmful substance. In addition to corneal tissue damage, some substances can change the pH of the aqueous within seconds, ultimately altering trabecular meshwork structures, leading to permanent anatomical changes.

When a patient calls to ask what they should do after exposure to an acid or alkali, they should be instructed to immediately irrigate their eyes before they do anything else. They should be directed to rinse both eyes under low pressure with the most readily available water source for a minimum of 15 minutes. Recommendations such as a sink faucet, or hose without a spray nozzle could be used under low to medium pressure. The patient could also be instructed to use the shower fully clothed to flush both eyes at the same time if needed (Table 2). If possible, another person should help hold the eyelids open while the eye is irrigated. Whatever the source of irrigation, try to keep the water tepid or at room temperature. Finally, do not delay irrigation for contact lens removal. Contacts can be removed after irrigation, and may often come out during the flushing process.

Exam

Each component to the examination has a specific purpose and should begin only after irrigation has occurred and the pH has been neutralized between 7.0 and 7.2. When evaluating a chemical burn patient it is critical to inspect the fornices for residual debris. Certain chemicals such as calcium hydroxide precipitate and collect in the fornix creating a nidus for persistent damage. These crystallized precipitates keep the pH from returning to normal and have the potential to create further damage. Therefore, the pH levels should be checked every 15-20 minutes during the irrigation process.[1]

When evaluating the cornea and conjunctiva, first determine the area of involvement by assessing the extent of fluorescein staining. Be careful not to underestimate as a large chemical burn can completely denude the epithelium and prevent fluorescein uptake. Also estimate the depth of corneal penetration and loss of stromal clarity by visualizing other anterior segment structures. Assess the depth of limbal involvement and conjunctival penetration by determining the presence or absence of limbal ischemia. Blanching of vessels at the limbus is a sign of ischemia and cell death and should be measured in clock hours of involvement.

A measurement of intraocular pressure, preferably with a tonopen, must be acquired. Chemical injuries have the potential to create an acute pressure spike due to trabecular meshwork alterations and needs to be monitored throughout treatment.

Finally, if the patient is aware of the offending agent, both the Material Safety Data Sheet (www.msdsonline.com) and poison control center (www.aapcc.org) offer a wealth of information about the toxicity of the substance. Here you can find the chemical's pH, and other factors that may be beneficial while developing a treatment regimen.

Classification

Several classification systems exist to categorize the extent of chemical injuries. The most widely used is based on the Roper-Hall system which divides injuries into four grades (Table 3, Figure 1-4), each with increasing severity based upon corneal appearance and extent of limbal ischemia.[4-6]

Grade I - Limbal stem cells are not affected and ischemia is not present. There will be a large epithelial defect but the underlying cornea will remain clear. The prognosis for full recovery of normal corneal appearance and function is excellent.

Grade II - Partial limbal ischemia is present affecting less than ⅓ of the limbus. The cornea may be hazy but anterior segment structures are typically visible. The prognosis is favorable but persistent epithelial dysfunction can be present along with conjunctivalization.

Grade III - Extensive limbal ischemia is present involving ⅓ to ½ of the limbus. Most of the limbal stem cells are damaged, and stromal haze will limit the visualization of iris and lens. The prognosis in Grade III is guarded, and surgery may be required for visual rehabilitation and recovery.

Grade IV - Greater than ½ of the limbus is ischemic along with complete loss of the corneal epithelium. There is also loss of proximal conjunctival epithelium. The cornea will appear porcelainized, and its opaqueness will not allow for views of other anterior segment structures. The prognosis is extremely poor and the extent of tissue damage, more specifically limbal stem cell death, increases the likelihood of significant ocular morbidity.

Phases of recovery

McCulley et al divided the clinical course into several distinct phases.[1,2,7] In each phase, multiple events occur as the cornea attempts to repair itself. These include epithelial regrowth and migration, collagen synthesis and degradation, and activation and migration of keratocytes.[1,2,7]

Acute phase (Day 0-7) - Re-epithelialization begins if there are sufficient limbal stem cells. Treatment efforts are directed at encouraging epithelial migration, controlling inflammation, and avoiding topical medications and bandage contact lenses that may be damaging to the fragile epithelium. Keratocyte activation also starts during this phase, allowing the initiation of collagen synthesis with little or no collagen breakdown.

Intermediate phase (Day 7-21) - Epithelial migration continues. Grade I injuries are typically re-epithelialized, but Grade II will most likely have persistent epithelial defects. Grades III-IV will have minimal to no re-epithelialization. Keratocytes will continue to function during this phase to repair the damage to the stroma, but activity from collagenases peak and concurrently start to break down collagen. Treatment during this phase attempts to maximize collagen synthesis and minimize collagenase activity.

Late repair phase (After day 21) – Grade I will typically have a normal corneal surface re-established. Grade II will have focal limbal stem cell loss and focal conjunctivalization. Grade III shows delayed re-epithelialization and the cornea may become repopulated with conjunctival epithelium. Grade IV will continue

Inflammation Control

Chemical injuries have two waves of inflammation that occur following the trauma.[1,2] The first wave occurs within 12-24 hours and little can be done to prevent it. It is brought about by blood elements from the injured conjunctiva and uvea, necrotic tissue, and chemotaxis. The second and more destructive wave of inflammation typically begins at day 7 and peaks when corneal repair and degradation are maximal, usually between days 14-21. Aggressive treatment of the first wave and preventative treatment of the second wave with high dose corticosteroids and other anti-inflammatories will help minimize inflammation and hopefully prevent a corneal melt. While corticosteroids are of considerable value in reducing inflammation and tissue destruction following a chemical injury, they interfere with keratocyte migration and collagen synthesis and must be used with caution after the end of the first week following a severe chemical injury. The key is to successfully maximize the corticosteroids' anti-inflammatory effects during the first 7-10 days while the risk-benefit ratio is more favorable and then dramatically cut back.[1,2] Progestational steroids can be used throughout because they only have a minimal effect on keratocyte function and can be used with relative safety in the management of an acute chemical injury. Keratocyte synthesis of collagen may also be compromised by deficient levels of ascorbate in the aqueous following a severe chemical injury. Deficiency of anterior chamber ascorbate following experimental chemical injuries result in morphological abnormalities in collagen-synthesizing fibroblasts and corneal ulceration and perforation.[1,2]

Medical Management

During the acute phase, the goal is to encourage epithelial healing while controlling inflammation and prevent infection. Grade I injuries should be placed on topical antibiotics four times daily. Cycloplegics should be given along with non-preserved artificial tears. Even mild injuries have significant inflammation, therefore prednisolone acetate 1% four times a day is recommended.[2] Patients with mild injuries will often complain of discomfort for months following the trauma. Treating their dry eye and meibomian gland dysfunction may dramatically help re-establish their ocular surface and comfort.[2]

The intensive use of topical corticosteroids in the acute phase cannot be overstressed in patients with Grade II-IV injuries.[1,2,8] Corneal melting from activation of stromal collagenases typically does not occur until 10 to 14 days after the injury.[1] Therefore, the first week of treatment represents a window in which corticosteroids can be used to help suppress the intense inflammation and give the ocular surface a chance to heal. Prednisolone acetate 1% hourly while awake during the first week, followed by a dramatic taper after seven days to twice daily is required for proper inflammation control.[1,2]

Autologous or umbilical cord serum should be considered during the acute phase to help promote re-epithelialization. Autologous serum utilizes a patient's own blood, but it does not contain red blood cells or clotting factors. It does replace individualized antibodies, giving it anti-inflammatory and anti-scarring properties. Umbilical cord serum is similar, but has additional growth factors including Epidermal Growth Factor, Transforming Growth Factor-b, and neurotrophic factors, such as Substance P, insulin-like growth factor-1, and nerve growth factor. The concentrations of these growth factors are noted in higher concentrations in umbilical cord serum compared than peripheral blood serum.[9,10]

Topical sodium ascorbate (10%) and citrate (10%) promote collagen synthesis and should be used during the acute phase to help rebuild stromal tissue. The levels of these substances will be diminished due to anterior segment and ciliary body involvement. Oral Vitamin C will not be converted to ascorbate in the anterior chamber and is of limited value.[2,8]

Sutureless amniotic membranes have a tremendous benefit during the acute phase and should be placed on the eye within the first week following the trauma.[11] Their ease of access and powerful anti-inflammatory properties make them a necessary adjunct when treating chemical injuries. Amniotic membranes are the innermost of the 3 membranes that form the fetal membrane. They have been shown to reduce pain, stimulate epithelialization and stem cell proliferation. They also prevent further efflux of immune cells, thereby reducing inflammation. [9,11-14]

Topical fluoroquinolones should be used at least 4 times a day and will help reduce the risk of a secondary bacterial superinfection[2], as well as long acting cycloplegics such as Atropine or homatropine. Oral narcotics for pain control are helpful and oral tetracycline derivatives can be used to inhibit matrix metalloproteinases and reduce further inflammation.[1,2] When glaucoma agents are needed, and they often are, oral medications should be used instead of topical agents.[15]

Topical NSAIDs are not indicated due to their potential issues with epithelial healing and their lack of appropriate anti-inflammatory effects to control the patient's inflammation.[2] In the acute phase, the

eye is typically too inflamed to tolerate a bandage contact lens and should be avoided [1,2], and phenylephrine and other vasoconstrictors should not be used because they may increase ischemia.[15]

Surgical Management

Surgical management is needed when healing has slowed, or when medical management is no longer effective. If epithelial healing falters or is absent, or if progressive corneal melting occurs despite intensive medical therapy, surgical intervention may be required. Fortunately, there are many surgical maneuvers which may decrease the chances of significant ocular and visual morbidity. Surgical treatments range from simple debridement of necrotic tissue, to limbal stem cell transplantation and artificial corneas.

Surgical management is needed when certain clinical findings are present, or when medical management is no longer effective. If epithelial healing falters or is absent, or if progressive corneal melting occurs, surgical intervention may be required. Fortunately, there are many surgical maneuvers which may decrease the chances of significant ocular and visual morbidity.

In addition to sutureless amniotic membranes, amniotic membrane transplants have been widely used in many cases. They can be sutured into place following injury, and may help reduce intolerable pain, while acting as a temporizing measure until a limbal stem cell transplant can take place. A study by Tandon et al showed the rate of epithelial healing was significantly better than compared to the control group treated with standard medical therapy alone.[17]

Limbal stem cells play a vital role in healing and recovery following a chemical injury. Limbal stem cell transplantation (LSCT) is another surgical technique that involves the harvesting of two crescents of peripheral corneal limbal epithelium with a corresponding section of conjunctiva from the limbus. If this section is taken from the uninjured eye, the transplant is considered an autograft; if taken from a close relative, it is an allograft. The section of tissue is then expanded in culture in vitro. This procedure can be performed as early as 3 weeks following the injury, and the success is dependent on medical control of inflammation prior. A LSCT is the only technique available to re-establish a normal corneal phenotype. An intact epithelium is also effective at minimizing corneal ulceration.

Another surgical procedure involves the implantation of a keratoprosthesis, an artificial cornea with approval for use in the United States. They were once thought to be the most successful method of visual rehabilitation in chemically burned eyes.[18], and have been shown to help restore vision in severely damaged corneas. The 3 most common devices are AlphaCor®, Boston Keratoprosthesis (Boston KPro), and Oculaid Keratoprosthesis[1].

Other surgical techniques that may be used are penetrating keratoplasty, tenoplasty, as well as the use of Avastin to limit corneal neovascularization.

Ocular Sequelae

In addition to the expected changes to the cornea and conjunctiva following a chemical burn, it is important to realize that less common, but equally damaging, findings can affect other ocular structures.

The eyelid may see permanent changes to its anatomical structure in the wake of a chemical splash. It is common to see cicatricial entropion months after the injury and subsequent trichiasis, as well as ectropion, lagophthalmos, symblepharon and ankyloblepharon. [19] Consultation with oculoplastics will likely be needed.

It is also possible to develop a chronic recurrent uveitis, and it is not unlikely to see cataracts develop.[19] Persistent dry eye is also very common due to xerophthalmia and loss of limbal stem cells.[19] And one of the most frequently overlooked side effects following a chemical burn is glaucoma. It can be a result of several factors, but necessary treatments of topical steroids can cause or worsen the disease. Glaucoma is likely the single most important element limiting the visual outcome.[16] Unfortunately, damage to the cornea makes tonometry difficult. It is critical to remember that glaucomatous changes are likely after a chemical burns, and the prudent practitioner should diagnose and treat accordingly. Of note, many agree that glaucoma following a chemical burn is likely the most preventable complication.[1,2]

Conclusion

Chemical burns are one of the most urgent ocular emergencies and prompt medical management with high dose corticosteroids and other anti-inflammatories can help prevent ocular morbidity. The goals of treatment in a patient with a chemical injury are restoration of the normal ocular surface anatomy, controlling inflammation, and improving corneal clarity. With today's understanding of chemical injuries and newer treatment modalities available, it is possible to maintain and restore vision in all but the most severely burned eyes.

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Table 1

Rates of Penetration (Slowest to Fastest)

- Sulfuric Acid ⟶ Hydrochloric acid ⟶ Sulfurous acid ⟶ Hydrofluoric acid
- Calcium hydroxide ⟶ Potassium hydroxide ⟶ Sodium hydroxide ⟶ Ammonium hydroxide

Table 2

Techniques for Irrigation

Eye Irrigation Method #1 -- Flushing:

Slowly pour lukewarm water into the eye from a pitcher or glass.

Or, place your head under a gently running faucet or shower. Hold the eyelid open during this process.

Eye Irrigation Method #2 -- Immersion:

Immerse the entire face into a sink or basin filled with lukewarm tap water.

With the face under water, open and close the eyelids. You may need to use your fingers. Look from side to side.

Table 3 Classification of Chemical Burns

	CORNEA	LIMBAL ISCHEMIA	PROGNOSIS
GRADE I	EPITHELIAL DEFECT	NONE	EXCELLENT
GRADE II	HAZY	< ⅓ of LIMBUS	FAVORABLE
GRADE III	STROMAL HAZE	⅓ to ½ of LIMBUS	GUARDED
GRADE IV	PORCELAINIZED	> ½ of LIMBUS	POOR

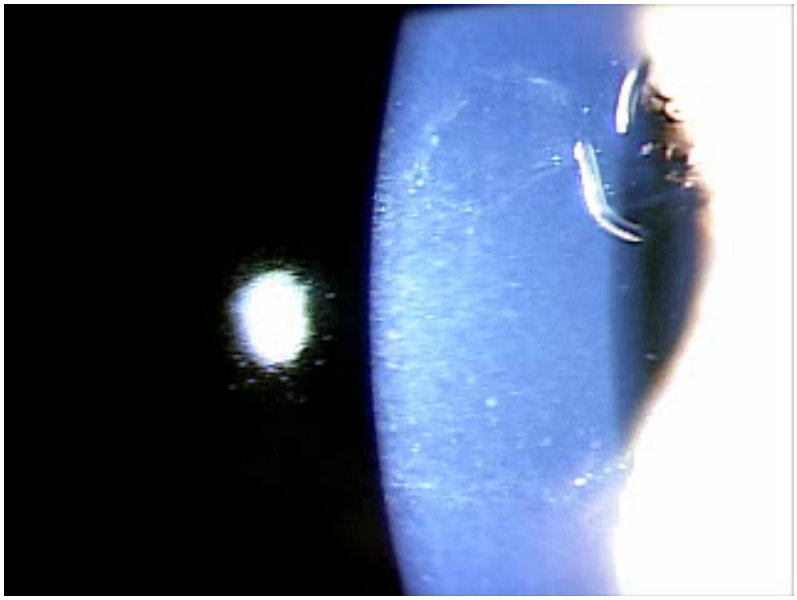

Figure 1 Acute grade I chemical burn with epithelial defect, and no limbal ischemia. My personal photo (but not the patient in case report).

Pictures from reference #2 (*Colby K. Chemical Injuries of the Cornea*). Photos can be found in that article on page 3. I do not have official permission to use these photos, they are merely for demonstration purposes for AAO Diplomate case report submission.

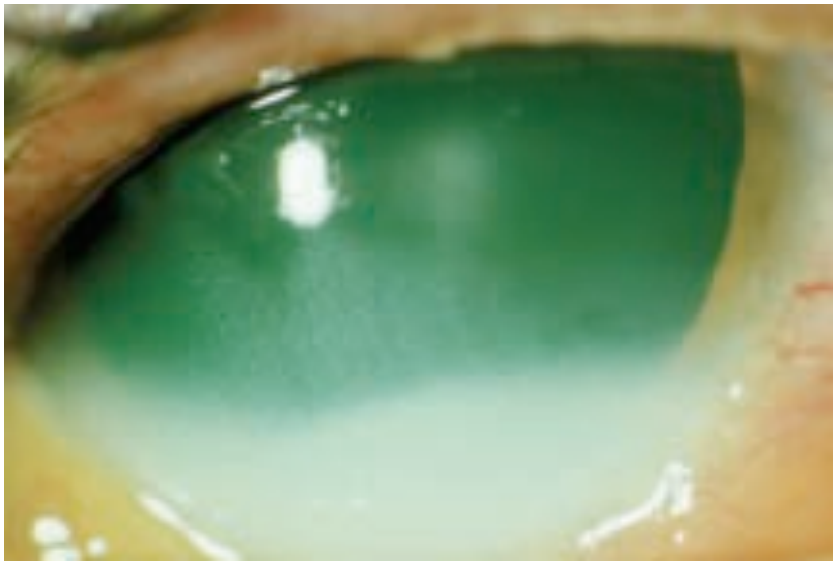

Figure 2 Acute grade II burn with focal (approximately 3 clock hours) of limbal stem cell loss.

Figure 3 Acute grade III burn showing extensive corneal haze impeding visualization of the iris and limbal ischemia of approximately 6 clock hours.

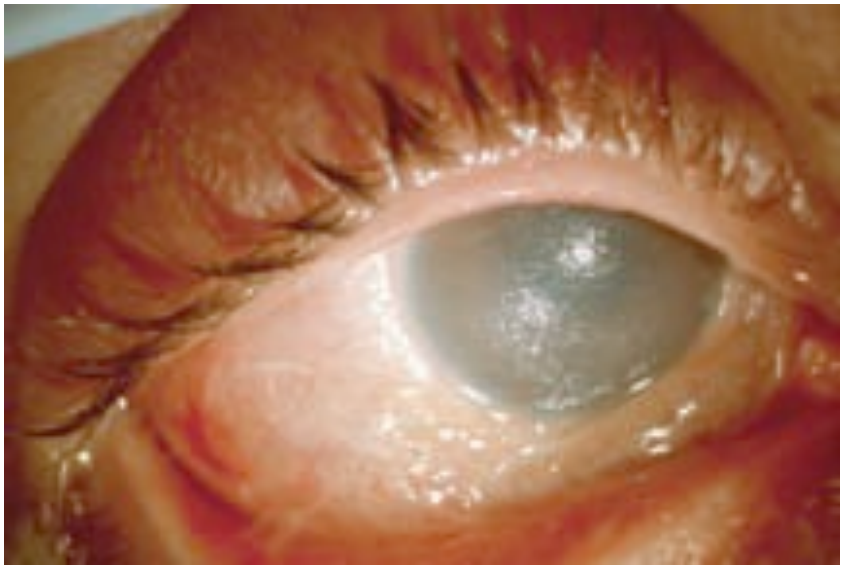

Figure 4 Acute grade IV burn with porcelainization of the cornea and bulbar conjunctiva.