In general, alkali injuries are worse than acid injuries. Alkali causes saponification of cell membrane fatty acids, which leads to cell membrane disruption. In contrast, acids denature proteins, causing protein precipitation, which actually acts to block deeper penetration by the acid.
In general, alkali injuries are worse than acid.
In general, alkali injuries are worse than acid. Alkali causes [mechanism...], which leads to [effect on cells].
In general, alkali injuries are worse than acid. Alkali causes saponification of cell membrane fatty acids, which leads to cell membrane disruption.
In general, alkali injuries are worse than acid. Alkali causes saponification of cell membrane fatty acids, which leads to cell membrane disruption. In contrast, acids [mechanism of injury], causing [effect], which actually acts to [protective effect].
In general, alkali injuries are worse than acid. Alkali causes saponification of cell membrane fatty acids, which leads to cell membrane disruption. In contrast, acids denature proteins, causing protein precipitation, which actually acts to block deeper penetration by the acid.
In general, alkali injuries are worse than acid. Alkali causes saponification of cell membrane fatty acids, which leads to cell membrane disruption. In contrast, acids denature proteins, causing protein precipitation, which actually acts to block deeper penetration by the acid.

Management of chemical injury can be divided into the [first] phase and the [post-first] phase.
In general, alkali injuries are worse than acid. Alkali causes saponification of cell membrane fatty acids, which leads to cell membrane disruption. In contrast, acids denature proteins, causing protein precipitation, which actually acts to block deeper penetration by the acid.

Management of chemical injury can be divided into the **acute** phase and the **post-acute** phase.

---

**Re acid-base injury of the ocular surface: Fill in the blanks**

In general, alkali injuries are worse than acid. Alkali causes saponification of cell membrane fatty acids, which leads to cell membrane disruption. In contrast, acids denature proteins, causing protein precipitation, which actually acts to block deeper penetration by the acid.

Management of chemical injury can be divided into the **acute** phase and the **post-acute** phase.
In general, alkali injuries are worse than acid. Alkali causes saponification of cell membrane fatty acids, which leads to cell membrane disruption. In contrast, acids denature proteins, causing protein precipitation, which actually acts to block deeper penetration by the acid.

Management of chemical injury can be divided into the **acute** phase and the **post-acute** phase. The treatment goal in the acute phase is **[gooooo...]**. **[...oooooal]**
In general, alkali injuries are worse than acid. Alkali causes saponification of cell membrane fatty acids, which leads to cell membrane disruption. In contrast, acids denature proteins, causing protein precipitation, which actually acts to block deeper penetration by the acid.

Management of chemical injury can be divided into the **acute** phase and the **post-acute** phase. The treatment goal in the **acute** phase is **removing the chemical**.

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Re **acid-base injury** of the ocular surface: Fill in the blanks

A
In general, alkali injuries are worse than acid. Alkali causes saponification of cell membrane fatty acids, which leads to cell membrane disruption. In contrast, acids denature proteins, causing protein precipitation, which actually acts to block deeper penetration by the acid.

Management of chemical injury can be divided into the acute phase and the post-acute phase. The treatment goal in the acute phase is removing the chemical. This is accomplished by [maneuver 1] and [maneuver 2].
In general, alkali injuries are worse than acid. Alkali causes saponification of cell membrane fatty acids, which leads to cell membrane disruption. In contrast, acids denature proteins, causing protein precipitation, which actually acts to block deeper penetration by the acid.

Management of chemical injury can be divided into the acute phase and the post-acute phase. The treatment goal in the acute phase is removing the chemical. This is accomplished by irrigation and sweeping the fornices.

Re acid-base injury of the ocular surface: Fill in the blanks
In general, alkali injuries are worse than acid. Alkali causes saponification of cell membrane fatty acids, which leads to cell membrane disruption. In contrast, acids denature proteins, causing protein precipitation, which actually acts to block deeper penetration by the acid.

Management of chemical injury can be divided into the acute phase and the post-acute phase. The treatment goal in the acute phase is removing the chemical. This is accomplished by irrigation and sweeping the fornices.

The post-acute phase has 4 goals:

1) 

2) 

3) 

4)
In general, alkali injuries are worse than acid. Alkali causes saponification of cell membrane fatty acids, which leads to cell membrane disruption. In contrast, acids denature proteins, causing protein precipitation, which actually acts to block deeper penetration by the acid.

Management of chemical injury can be divided into the acute phase and the post-acute phase. The treatment goal in the acute phase is removing the chemical. This is accomplished by irrigation and sweeping the fornices.

The post-acute phase has 4 goals:

1) Decrease [one word]

2) Control [abb.]

3) Promote [two words]

4) Promote [two words]
In general, alkali injuries are worse than acid. Alkali causes saponification of cell membrane fatty acids, which leads to cell membrane disruption. In contrast, acids denature proteins, causing protein precipitation, which actually acts to block deeper penetration by the acid.

Management of chemical injury can be divided into the acute phase and the post-acute phase. The treatment goal in the acute phase is removing the chemical. This is accomplished by irrigation and sweeping the fornices.

The post-acute phase has 4 goals:

1) Decrease inflammation

2) Control IOP

3) Promote wound healing

4) Promote epi healing
In general, alkali injuries are worse than acid. Alkali causes saponification of cell membrane fatty acids, which leads to cell membrane disruption. In contrast, acids denature proteins, causing protein precipitation, which actually acts to block deeper penetration by the acid.

Management of chemical injury can be divided into the acute phase and the post-acute phase. The treatment goal in the acute phase is removing the chemical. This is accomplished by irrigation and sweeping the fornices.

The post-acute phase has 4 goals:

1) *Decrease inflammation*. This is accomplished with intense [route of admin] [drug] X [amount of time]

2) *Control IOP*

3) *Promote wound healing*

4) *Promote epi healing*
In general, alkali injuries are worse than acid. Alkali causes saponification of cell membrane fatty acids, which leads to cell membrane disruption. In contrast, acids denature proteins, causing protein precipitation, which actually acts to block deeper penetration by the acid.

Management of chemical injury can be divided into the acute phase and the post-acute phase. The treatment goal in the acute phase is removing the chemical. This is accomplished by irrigation and sweeping the fornices.

The post-acute phase has 4 goals:
1) Decrease *inflammation*. This is accomplished with intense topical steroids x 1-2 weeks

2) Control IOP

3) Promote wound healing

4) Promote epi healing
In general, alkali injuries are worse than acid. Alkali causes saponification of cell membrane fatty acids, which leads to cell membrane disruption. In contrast, acids denature proteins, causing protein precipitation, which actually acts to block deeper penetration by the acid.

Management of chemical injury can be divided into the acute phase and the post-acute phase. The treatment goal in the acute phase is removing the chemical. This is accomplished by irrigation and sweeping the fornices.

The post-acute phase has 4 goals:

1) Decrease *inflammation*. This is accomplished with intense topical steroids x 1-2 weeks, at which time it must be tapered off so as not to [undesirable side effect]

2) Control IOP

3) Promote wound healing

4) Promote epi healing
In general, alkali injuries are worse than acid. Alkali causes saponification of cell membrane fatty acids, which leads to cell membrane disruption. In contrast, acids denature proteins, causing protein precipitation, which actually acts to block deeper penetration by the acid.

Management of chemical injury can be divided into the acute phase and the post-acute phase. The treatment goal in the acute phase is removing the chemical. This is accomplished by irrigation and sweeping the fornices. The post-acute phase has 4 goals:

1) *Decrease inflammation*. This is accomplished with intense topical steroids x 1-2 weeks, at which time it must be tapered off so as not to inhibit wound healing.

2) *Control IOP*

3) *Promote wound healing*

4) *Promote epi healing*
In general, alkali injuries are worse than acid. Alkali causes saponification of cell membrane fatty acids, which leads to cell membrane disruption. In contrast, acids denature proteins, causing protein precipitation, which actually acts to block deeper penetration by the acid.

Management of chemical injury can be divided into the acute phase and the post-acute phase. The treatment goal in the acute phase is removing the chemical. This is accomplished by irrigation and sweeping the fornices.

The post-acute phase has 4 goals:

1) *Decrease inflammation*. This is accomplished with intense *topical steroids* x 1-2 weeks, at which time it must be tapered off so as not to inhibit wound healing. Two useful adjuvant therapies are [drug] and [dietary… supplement].

2) *Control IOP*

3) *Promote wound healing*

4) *Promote epi healing*
In general, alkali injuries are worse than acid. Alkali causes saponification of cell membrane fatty acids, which leads to cell membrane disruption. In contrast, acids denature proteins, causing protein precipitation, which actually acts to block deeper penetration by the acid.

Management of chemical injury can be divided into the acute phase and the post-acute phase. The treatment goal in the acute phase is removing the chemical. This is accomplished by irrigation and sweeping the fornices.

The post-acute phase has 4 goals:

1) *Decrease inflammation*. This is accomplished with intense topical steroids x 1-2 weeks, at which time it must be tapered off so as not to inhibit wound healing. Two useful adjuvant therapies are doxy and citric acid.

2) *Control IOP*

3) *Promote wound healing*

4) *Promote epi healing*
In general, alkali injuries are worse than acid. Alkali causes saponification of cell membrane fatty acids, which leads to cell membrane disruption. In contrast, acids denature proteins, causing protein precipitation, which actually acts to block deeper penetration by the acid.

Management of chemical injury can be divided into the acute phase and the post-acute phase. The treatment goal in the acute phase is removing the chemical. This is accomplished by irrigation and sweeping the fornices. The post-acute phase has 4 goals:

1) *Decrease inflammation*. This is accomplished with intense topical steroids x 1-2 weeks, at which time it must be tapered off so as not to inhibit wound healing. Two useful adjuvant therapies are doxy and citric acid. Both are potent chelators of extracellular \([\text{ion}]\), which is needed for degranulation.

2) **Control IOP**

3) **Promote wound healing**

4) **Promote epi healing**
In general, alkali injuries are worse than acid. Alkali causes saponification of cell membrane fatty acids, which leads to cell membrane disruption. In contrast, acids denature proteins, causing protein precipitation, which actually acts to block deeper penetration by the acid.

Management of chemical injury can be divided into the acute phase and the post-acute phase. The treatment goal in the acute phase is removing the chemical. This is accomplished by irrigation and sweeping the fornices.

The post-acute phase has 4 goals:

1) Decrease inflammation. This is accomplished with intense topical steroids x 1-2 weeks, at which time it must be tapered off so as not to inhibit wound healing. Two useful adjuvant therapies are doxy and citric acid. Both are potent chelators of extracellular Ca\(^{2+}\), which is needed for PMN degranulation. PMN = Polymorphonuclear leukocytes; ie, neutrophils

2) Control IOP

3) Promote wound healing

4) Promote epi healing

Re acid-base injury of the ocular surface: Fill in the blanks
In general, alkali injuries are worse than acid. Alkali causes saponification of cell membrane fatty acids, which leads to cell membrane disruption. In contrast, acids denature proteins, causing protein precipitation, which actually acts to block deeper penetration by the acid.

Management of chemical injury can be divided into the acute phase and the post-acute phase. The treatment goal in the acute phase is removing the chemical. This is accomplished by irrigation and sweeping the fornices.

The post-acute phase has 4 goals:

1) Decrease inflammation. This is accomplished with intense topical steroids x 1-2 weeks, at which time it must be tapered off so as not to inhibit wound healing. Two useful adjuvant therapies are doxy and citric acid. Both are potent chelators of extracellular Ca$^{2+}$, which is needed for PMN degranulation. Of course, a [drug class] is used as well.

2) Control IOP

3) Promote wound healing

4) Promote epi healing
In general, alkali injuries are worse than acid. Alkali causes saponification of cell membrane fatty acids, which leads to cell membrane disruption. In contrast, acids denature proteins, causing protein precipitation, which actually acts to block deeper penetration by the acid.

Management of chemical injury can be divided into the acute phase and the post-acute phase. The treatment goal in the acute phase is removing the chemical. This is accomplished by irrigation and sweeping the fornices.

The post-acute phase has 4 goals:

1) Decrease inflammation. This is accomplished with intense topical steroids x 1-2 weeks, at which time it must be tapered off so as not to inhibit wound healing. Two useful adjuvant therapies are doxy and citric acid. Both are potent chelators of extracellular Ca$^{2+}$, which is needed for PMN degranulation. Of course, a cycloplegic agent is used as well.

2) Control IOP

3) Promote wound healing

4) Promote epi healing
In general, alkali injuries are worse than acid. Alkali causes saponification of cell membrane fatty acids, which leads to cell membrane disruption. In contrast, acids denature proteins, causing protein precipitation, which actually acts to block deeper penetration by the acid.

Management of chemical injury can be divided into the acute phase and the post-acute phase. The treatment goal in the acute phase is removing the chemical. This is accomplished by irrigation and sweeping the fornices.

The post-acute phase has 4 goals:

1) *Decrease inflammation*. This is accomplished with intense topical steroids x 1-2 weeks, at which time it must be tapered off so as not to inhibit wound healing. Two useful adjuvant therapies are doxy and citric acid. Both are potent chelators of extracellular $\text{Ca}^{2+}$, which is needed for PMN degranulation. Of course, a cycloplegic agent is used as well.

2) *Control IOP*. This is best done with [drug and route] to avoid [undesirable side effect of different route].

3) *Promote wound healing*

4) *Promote epi healing*
Re *acid-base injury* of the ocular surface: Fill in the blanks

In general, alkali injuries are worse than acid. Alkali causes saponification of cell membrane fatty acids, which leads to cell membrane disruption. In contrast, acids denature proteins, causing protein precipitation, which actually acts to block deeper penetration by the acid.

Management of chemical injury can be divided into the acute phase and the post-acute phase. The treatment goal in the acute phase is removing the chemical. This is accomplished by irrigation and sweeping the fornices.

The post-acute phase has 4 goals:

1) *Decrease inflammation*. This is accomplished with intense topical steroids *x 1-2 weeks*, at which time it must be tapered off so as not to inhibit wound healing. Two useful adjuvant therapies are *doxy* and *citric acid*. Both are potent chelators of extracellular Ca^{2+}, which is needed for PMN degranulation. Of course, a *cycloplegic agent* is used as well.

2) *Control IOP*. This is best done with PO Diamox to avoid epithelial toxicity from topical hypotensives.

3) *Promote wound healing*

4) *Promote epi healing*
In general, alkali injuries are worse than acid. Alkali causes saponification of cell membrane fatty acids, which leads to cell membrane disruption. In contrast, acids denature proteins, causing protein precipitation, which actually acts to block deeper penetration by the acid.

Management of chemical injury can be divided into the acute phase and the post-acute phase. The treatment goal in the acute phase is removing the chemical. This is accomplished by irrigation and sweeping the fornices.

The post-acute phase has 4 goals:

1) *Decrease inflammation*. This is accomplished with intense topical steroids x 1-2 weeks, at which time it must be tapered off so as not to inhibit wound healing. Two useful adjuvant therapies are doxy and citric acid. Both are potent chelators of extracellular Ca^{2+}, which is needed for PMN degranulation. Of course, a cycloplegic agent is used as well.

2) *Control IOP*. This is best done with PO Diamox to avoid epithelial toxicity from topical hypotensives.

3) *Promote wound healing*. This is done with [supplement], which increases AC [supplement] levels and promotes [structural protein] synthesis.

4) *Promote epi healing*
In general, alkali injuries are worse than acid. Alkali causes saponification of cell membrane fatty acids, which leads to cell membrane disruption. In contrast, acids denature proteins, causing protein precipitation, which actually acts to block deeper penetration by the acid.

Management of chemical injury can be divided into the acute phase and the post-acute phase. The treatment goal in the acute phase is removing the chemical. This is accomplished by irrigation and sweeping the fornices. The post-acute phase has 4 goals:

1) **Decrease inflammation.** This is accomplished with intense topical steroids x 1-2 weeks, at which time it must be tapered off so as not to inhibit wound healing. Two useful adjuvant therapies are doxy and citric acid. Both are potent chelators of extracellular Ca^{2+}, which is needed for PMN degranulation. Of course, a cycloplegic agent is used as well.

2) **Control IOP.** This is best done with PO Diamox to avoid epithelial toxicity from topical hypotensives.

3) **Promote wound healing.** This is done with ascorbic acid, which increases AC ascorbate levels and promotes collagen synthesis.

4) **Promote epi healing**
In general, alkali injuries are worse than acid. Alkali causes saponification of cell membrane fatty acids, which leads to cell membrane disruption. In contrast, acids denature proteins, causing protein precipitation, which actually acts to block deeper penetration by the acid.

Management of chemical injury can be divided into the acute phase and the post-acute phase. The treatment goal in the acute phase is removing the chemical. This is accomplished by irrigation and sweeping the fornices.

The post-acute phase has 4 goals:

1) *Decrease inflammation*. This is accomplished with intense topical steroids x 1-2 weeks, at which time it must be tapered off so as not to inhibit wound healing. Two useful adjuvant therapies are doxy and citric acid. Both are potent chelators of extracellular Ca^{2+}, which is needed for PMN degranulation. Of course, a cycloplegic agent is used as well.

2) *Control IOP*. This is best done with PO Diamox to avoid epithelial toxicity from topical hypotensives.

3) *Promote wound healing*. This is done with ascorbic acid, which increases AC ascorbate levels and promotes collagen synthesis. (Careful! Ascorbic acid is [organ-damage].)

4) *Promote epi healing*
In general, alkali injuries are worse than acid. Alkali causes saponification of cell membrane fatty acids, which leads to cell membrane disruption. In contrast, acids denature proteins, causing protein precipitation, which actually acts to block deeper penetration by the acid.

Management of chemical injury can be divided into the acute phase and the post-acute phase. The treatment goal in the acute phase is removing the chemical. This is accomplished by irrigation and sweeping the fornices.

The post-acute phase has 4 goals:

1) Decrease inflammation. This is accomplished with intense topical steroids x 1-2 weeks, at which time it must be tapered off so as not to inhibit wound healing. Two useful adjuvant therapies are doxy and citric acid. Both are potent chelators of extracellular Ca$^{2+}$, which is needed for PMN degranulation. Of course, a cycloplegic agent is used as well.

2) Control IOP. This is best done with PO Diamox to avoid epithelial toxicity from topical hypotensives.

3) Promote wound healing. This is done with ascorbic acid, which increases AC ascorbate levels and promotes collagen synthesis. (Careful! Ascorbic acid is nephrotoxic.)

4) Promote epi healing.

Re acid-base injury of the ocular surface: Fill in the blanks
In general, alkali injuries are worse than acid. Alkali causes saponification of cell membrane fatty acids, which leads to cell membrane disruption. In contrast, acids denature proteins, causing protein precipitation, which actually acts to block deeper penetration by the acid.

Management of chemical injury can be divided into the acute phase and the post-acute phase. The treatment goal in the acute phase is removing the chemical. This is accomplished by irrigation and sweeping the fornices.

The post-acute phase has 4 goals:

1) *Decrease inflammation*. This is accomplished with intense topical steroids x 1-2 weeks, at which time it must be tapered off so as not to inhibit wound healing. Two useful adjuvant therapies are *doxy* and *citric acid*. Both are potent chelators of extracellular Ca\(^{2+}\), which is needed for PMN degranulation. Of course, a *cycloplegic agent* is used as well.

2) *Control IOP*. This is best done with PO Diamox to avoid epithelial toxicity from topical hypotensives.

3) *Promote wound healing*. This is done with *ascorbic acid*, which increases AC ascorbate levels and promotes *collagen* synthesis. (Careful! Ascorbic acid is *nephrotoxic*.)

4) *Promote epi healing* with 3 maneuvers: [1 drug; 1 device; 1 surgery]
In general, alkali injuries are worse than acid. Alkali causes saponification of cell membrane fatty acids, which leads to cell membrane disruption. In contrast, acids denature proteins, causing protein precipitation, which actually acts to block deeper penetration by the acid.

Management of chemical injury can be divided into the acute phase and the post-acute phase. The treatment goal in the acute phase is removing the chemical. This is accomplished by irrigation and sweeping the fornices.

The post-acute phase has 4 goals:

1) *Decrease inflammation.* This is accomplished with intense topical steroids x 1-2 weeks, at which time it must be tapered off so as not to inhibit wound healing. Two useful adjuvant therapies are doxy and citric acid. Both are potent chelators of extracellular Ca$^{2+}$, which is needed for PMN degranulation. Of course, a cycloplegic agent is used as well.

2) *Control IOP.* This is best done with PO Diamox to avoid epithelial toxicity from topical hypotensives.

3) *Promote wound healing.* This is done with ascorbic acid, which increases AC ascorbate levels and promotes collagen synthesis. (Careful! Ascorbic acid is nephrotoxic.)

4) *Promote epi healing* with 3 maneuvers: PF ATs, BCL, tarsorrhaphy.