TWO CASES OF ACETIC ACID BURNS OF THE CORNEA*

by

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Glacial acetic acid, though extensively used in manufacturing processes, is not commonly seen as a cause of chemical burns of the eye. Two cases which have been seen recently are thought to be of sufficient interest to warrant description, there being little reference to such injuries in the literature.

In both cases glacial acetic acid was put by mistake into the eye by the patient, instead of drops that were prescribed for treatment of an eye condition. In both cases the chemical was washed out of the eye immediately with tap-water and the reaction has been identical. They are, therefore, considered to give some indication of the course likely to be taken by an injury caused by a concentrated solution of this substance.

Case Reports

Case 1.—A man, aged 47 years, was admitted to hospital March 11, 1949, having mistaken acetic acid for adrenaline drops which had been prescribed for a partially blocked naso-lacrimal duct.

He reported to hospital on the same day as the injury, having immediately washed his eye out with tap-water, followed by an irrigation with sodium bicarbonate at a first-aid station. The report on admission was as follows:

- Cornea: Fine honeycomb opacity. There was extensive loss of epithelium and the whole of the corneal surface was staining.
- Conjunctiva: Very oedematous, with some irregular staining especially in the lower fornix.

At this stage it was difficult to see the condition of the structures deep to the cornea. The eye did not look very seriously damaged and a favourable prognosis was given. An amniotic membrane graft was inserted into the lower fornix on admission and gutt. atropine 1 per cent. instilled.

March 14, 1949. The eye looked quieter, and although the cornea was still stained extensively the patient was considered fit for discharge and out-patient treatment.

March 16, 1949. The corneal condition had improved and it was now possible to get an indistinct view of the iris, which was intensely inflamed. The patient was re-admitted to hospital.

March 19, 1949. A paracentesis was performed because the tension in the eye was raised.

April 2, 1949. The patient was discharged from hospital still showing considerable corneal staining and a fine honeycomb opacity, through which it was difficult to ascertain the state of the iris. In spite of every endeavour the pupil had remained firmly contracted.

The patient reported at regular intervals to the out-patient department, some-

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times with more corneal staining and sometimes with less, until May 26, 1949, when it was realised that he had a completely anaesthetic cornea. It was, therefore, decided that he would benefit from a tarsorraphy, which was accordingly performed. He has since remained comparatively comfortable, and the corneal staining has become progressively less, but there has been little or no improvement in the corneal opacity or in the corneal anaesthesia.

It is stressed that the original appearance of the eye was not sufficiently serious to suggest that either iritis or corneal anaesthesia was likely to develop.

Case 2.—An old lady, aged 66 years, had an Elliot’s trephine performed on the left eye for chronic glaucoma on April 30, 1949. She was discharged on May 11, 1949, with the trephine draining well. On May 13, 1949, she reported to the hospital stating that she had by mistake put glacial acetic acid in her eye instead of the pilocarpine drops that she had been given on leaving hospital. The condition on admission was reported as follows:

**Cornea**

Hazy and oedematous appearance of the lower 3/4 of the cornea with loss of epithelium and deep staining over this area.

**Conjunctiva**

Very oedematous, with some staining in the lower fornix.

One per cent. atropine drops were instilled and an amniotic membrane graft was inserted into the lower fornix. Again the eye did not appear seriously damaged and a favourable prognosis was given.

A further amniotic membrane graft was inserted three days later and more atropine instilled. By the time this graft had absorbed it was realized that the patient had a fairly intense iritis, with the formation of posterior synechiae which could only partially be broken down with atropine and mydricaine.

May 26, 1949. Our attention having now been drawn to the anaesthetic cornea in Case 1, we discovered that there was complete corneal anaesthesia over the affected area in this case also, and that the sensation was markedly impaired over the unaffected part. As there was still some corneal sensation a tarsorraphy was not thought to be necessary.

This case progressed more favourably, but nevertheless very slowly, the tarsorraphy not being completely healed until June 30, 1949. A permanent opacity remained.

**Discussion**

Many writers have drawn attention to the fact that acid burns of the eye are less harmful than alkali burns, and there is sufficient experimental work to suggest that this is true. This is only of theoretical value. Chemical burns of the eye are always accidental, and a concentrated acid solution is not necessarily less damaging than a weak alkali.

Valuable information on the effect of acetic acid on the tissues

**Table**

*Penetration Rates of Fixatives*

<table>
<thead>
<tr>
<th>Fixative</th>
<th>Mean Distance (mm.)</th>
<th>Time (minutes)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Acetic acid</td>
<td>42</td>
<td>1</td>
</tr>
<tr>
<td>Mercuric chloride</td>
<td>106</td>
<td>15</td>
</tr>
<tr>
<td>Absolute alcohol</td>
<td>23</td>
<td>15</td>
</tr>
<tr>
<td>Osmium tetroxide</td>
<td>15</td>
<td>15</td>
</tr>
</tbody>
</table>
Acetic Acid Burns of the Cornea

is available from experience in histological laboratories. Acetic acid is used as a fixative for histological preparations on account of its extremely rapid rate of penetration of the tissues. As shown in the Table (Underhill, 1932), acetic acid penetrates approximately half the distance in one minute that mercuric chloride penetrates in 15 minutes.

Guillery (1910) describes a primary and secondary opacity of the cornea occurring in acid burns of the cornea. He suggests that the cornea clears up soon after the primary opacities have occurred, and may become transparent, so that the full extent of the corneal damage does not become apparent for some days. He suggests that the corneal tissues become fixed in the same way as histological preparations and that the tissue is dead and without reaction, but that this necrotic state does not immediately become apparent.

Two points in these cases are worthy of consideration:

(1) The presence of corneal anaesthesia.
(2) The development of an intense iritis.

The presence of corneal anaesthesia is a sign of prognostic significance. In trivial injuries the cornea is hypersensitive, in moderate injuries sensitivity is diminished, and in severe cases it is absent. This is a valuable indication of the degree of damage immediately after the injury when its true extent cannot readily be estimated. If sensitivity remains permanently absent then it is an indication for protection by a tarsorraphy.

The early recognition of iritis is also of importance. This may be difficult through a cornea that has already been rendered opaque by injury. In such cases its presence should be assumed and early and vigorous treatment instituted. Delay in the treatment of an iritis may well lead to the formation of dense posterior synechiae so that the pupil will not readily dilate with atropine. If an iritis is anticipated and an amniotic membrane graft is indicated, the pupil should be dilated with mydricaine, and 1 per cent. or 2 per cent. atropine ointment placed in the conjunctival sacs before inserting the graft. The graft should be left in situ only for 24 hours, after which the eye should be examined with special reference to the state of the iris. If necessary a further graft can then be inserted.

The mechanism by which the iritis is produced does not seem clear. The rapid rate of penetration of the tissues by acetic acid would suggest that this substance may penetrate the cornea and set up irritation of the iris; on the other hand, it may be due to the absorption of necrotic corneal tissue into the anterior chamber. However the iritis is produced, there is no doubt that
some substances produce it more readily than others. Ammonia is well known to set up an iritis with a comparatively slight amount of corneal damage, whereas lime produces more corneal and conjunctival damage with less tendency to an iritis except in very severely damaged eyes.

It is therefore suggested that chemicals should be considered not as acids or alkalis, but rather as those substances which readily set up iritis and those which do not. In the first group, the first essential is the treatment of the iritis, in the second, amniotic membrane can safely be inserted and left for 48 hours without the fear of irretrievable damage occurring to the eye before it is looked at again.

**Summary**

Two cases of acetic acid burns of the cornea are described. Attention is drawn to the risk of iritis developing in these injuries, and the importance of early treatment of this condition is stressed. The value of the corneal sensitivity as a prognostic sign is indicated.

I wish to thank Professor Sorsby for permission to publish these two cases.

**REFERENCES**


Two Cases of Acetic Acid Burns of the Cornea

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