First aid for complications of infectious keratitis

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Infectious keratitis is a fairly common entity in India. However while paying attention to the primary entity, the associated events may be overlooked. Enhanced pain usually suggests a worsening of the condition or development of associated problems like secondary glaucoma. However, contrary to logic, a sudden decrease in pain is also liley to suggest a worsening, e.g. perforation of the corneal ulcer. Various such problems with their management are outlined.

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A patient with corneal ulcer will usually be monitored for his primary condition. However, there are certain signs that the clinician should be aware and watch out for. These are findings that would usually denote a change in the status, usually for the worse. The problems that we frequently encounter are:

1. Pain - sudden increase and decrease
2. Corneal thinning/melting
3. Severe anterior chamber reaction
4. Sudden increase in the ocular surface reaction despite medications

Pain

This is a normal protective mechanism associated with any lesion anywhere in the body. The corneal ulcer is no exception. Usually the patients complain of a localized pain in the ocular area of the affected cornea. This is due to the ciliary spasm secondary to the corneal neuronal reflex arc. Ciliary spasm can be effectively relieved by use of cycloplegics such as homatropine eye drops. In addition, the pain is usually well controlled once the topical medication brings the corneal infiltrates under control.

Secondary glaucoma

However, if a patient complains of a sudden or severe increase in pain, one needs to consider secondary glaucoma. This is usually due to the secondary changes in the anterior chamber. They include hypopyon and exudates on the endothelial surfaces causing a block of the trabecular meshwork or secondary trabeculitis in some cases of viral keratitis.

Secondary glaucoma needs aggressive medical management in the form of topical beta blockers and carbonic anhydrase inhibitors (dorzolamide) with or without systemic carbonic anhydrase inhibitors (acetazolamide). We prefer systemic carbonic anhydrase inhibitors due to the potential toxicity of topical inhibitors to the damaged corneal endothelium.[1]

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Perforation

Sudden reduction of pain in a patient with an active uncontrolled keratitis should be looked upon with suspicion rather than relief. Many a times due to the extensive exudation and/or accumulation of the medications (especially antifungal), the underlying wound is hidden. This can lead to a false sense of security and the underlying thinning may be missed. A micro-perforation in the area of the corneal ulcer can lead to natural drainage of the aqueous and relieve the intraocular pressure. This will relieve the pain induced by the secondary glaucoma. However, the collapse of the anterior chamber will lead to the formation of an adherent leukoma as well as closure of the angle of the anterior chamber. This will lead to irreversible secondary angle closure glaucoma with poor chances of visual rehabilitation. Perforations larger than 2 mm also have a significant risk of choroidal detachment.[2]

Corneal Thinning

The most common effect of a microbial infection of the cornea is the necrotic changes of the stromal fibers. This is due to the various actions of the microbe. It can be the direct action of the microbe in breakdown of the stromal fibers. It can be indirect action due to the endotoxins released due to the destruction of the microbial cell wall by the antibiotics or the body defense mechanisms. It can also be due to autolysis induced by various human enzymes like matrix metalloproteinase's (MMPs) that are released due to an uncontrolled activation of the tissue destruction mechanisms.[3,4]

The end result of all these mechanisms is the necrosis of the tissue in the infected area. If this is seen only due to the direct action of the microbes, then the control of the infection will limit the thinning. Thus, a regular watch on the corneal ulcer (we usually recommend a follow-up visit at least 48 h apart in ulcers < 3 mm and every 24 h in larger ulcers) is necessary to prevent such mishaps (V Agrawal, unpublished data).

In infections due to Gram-negative bacteria such as *pseudomonas*, the risk of rapid corneal melt is unusually high. These patients need to be seen as frequently as twice a day till the ulcer begins to heal.

The reason to watch for a corneal melt is that an early melt may respond to treatment and advanced melt is unlikely to respond well (V Agrawal, unpublished data).

In case of early melt, use of topical and systemic tetracycline is believed to help.[5,6] In addition, the use of tissue glue and bandage contact lens can also be useful. If the perforation is larger than 2 mm, then the use of tissue glue is unlikely to be adequate. We have successfully used preserved amniotic membrane as a multilayered application with tissue glue at the wound edges to close such wounds. This is a modification of the method of Hick *et al*.[7] Amniotic membrane also has anti-collagenolytic properties and may help in stabilizing the ocular surface.

Perforations larger than 4 mm and up to 6 mm may need a corneal patch graft to close the fistula. The important point in these cases is to avoid undersizing the graft. The wound edges of a melting area are also likely to be weak. This can lead to cheese wiring of the sutures and wound leaks.

Total corneal melts have been managed with sclerokeratoplasty with guarded results.[8]

In summary, corneal melts are best avoided by rigorous observation and aggressive medical management.

Severe Anterior Chamber Reaction

In severe fungal infection, the descemet's membrane can fail to limit the infection to the corneal stroma. Alternatively, the severe anterior chamber reaction secondary to the toxic effects of the microbe can lead to a complete organized hypopyon formation in the anterior chamber. The use of cycloplegics has been advocated but in our experience it may not be adequate. In such cases, we have used the concept of anterior chamber lavage to debulk the chamber and also reduce the infectious load to help the medications act better. Along with the lavage with Ringer's lactate, there may be a need to remove the organized fibrinoid material from the chamber. The use of a suitable antibiotic or antifungal agent in the lavage fluid is acceptable.[9]

Ocular Surface Condition

Many a times one sees that the corneal ulcer is responding to the medications for first 24-48 h. After this period, the eye becomes significantly redder, there is lid edema and the conjunctival surface too starts to stain with fluorescein. This may be a sign of spreading infection. However, it is more likely to either be an allergic reaction to one of the medications being used or the occurrence due to keratitis medicamentosa. This is differentiated from spreading infection by the fact that the primary lesion size remains constant or shows improvement despite all the adnexal changes.

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