CLPU is one of the major complications of contact lens wear with unknown etiology, and occurs mostly in extended hydrogel contact lens wearers. It has been described as an acute, sudden onset corneal lesion characterized by circular full-thickness epithelial defect in the periphery or mid-periphery of the cornea, accompanied by moderate to severe pain, photophobia, tearing, and blinking. Photophobia, tearing and minor pain are the main symptoms of CLPU. It most readily occurs upon removal of contact lenses without the use of antibiotics, leaving behind a scar. Both S. aureus and S. epidermidis are frequently isolated from the eye of CLPU patients, although not necessarily from the ulcer itself, and hypersensitivity to staphylococcal antigens has been suggested to be responsible for the formation of CLPU. Bacteriological studies have demonstrated that carriage of Gram positive bacteria, particularly Staphylococci, is associated with an increased risk of having CLPU. A case of CLPU, reported by Jalbert et al., with regular microbiological monitoring of 6 years demonstrated a direct relation of S. aureus with CLPU, as S. aureus, found in large amount in this patient, had never been isolated prior to the CLPU event. A previous study of the bacterial strains isolated from contact lens wearers suggested that S. aureus more frequently produced an array of potentially pathogenic toxins and enzymes than S. epidermidis. These indicate S. aureus is more likely to cause CLPU. The aim of this experiment is to investigate whether antigens from dead cells of S. aureus, or its secretory products, were responsible for CLPU, or whether live bacteria and corneal surface trauma were necessary to cause CLPU.

Materials & Methods

Can immunized rabbit model (S. aureus 031) induce CLPU?

Bacterial antigens: phenol inactivated S. aureus suspension (1 x 10^7 colony-forming units) and S. aureus supernatant

Immunization of rabbits: with the phenol inactivated S. aureus suspension

Contact lens wear: 2 - 4 weeks

Challenged agents: Bacterial antigens and live bacterial cells (see Table 1)

Examinations: slit-lamp

Does extended wear of contact lens induce CLPU?

Duration of contact lens wear: 7 weeks

Bacterium: S. aureus (031)

Examinations: slit-lamp

Is corneal epithelial trauma necessary for CLPU?

Agents (see Table 1): live S. aureus (031), live S. epidermidis (0319) and bacterial antigens (as above)

Contact lens coating: with bacterial cell suspension (OD)280 2.0

Epithelial trauma: 1-mm scratch at corneal periphery

Examinations: slit-lamp examination, histology

Results

Table 1. Experimental designs and observations

<table>
<thead>
<tr>
<th>Treatment</th>
<th>Observation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control</td>
<td>No CLPU-like lesion formed.</td>
</tr>
<tr>
<td>Live S. aureus</td>
<td>CLPU-like lesion formed.</td>
</tr>
<tr>
<td>Immunization</td>
<td>CLPU-like lesion formed.</td>
</tr>
</tbody>
</table>

Discussion

Immunization with S. aureus produced antibodies against the bacterial cell antigens. Pre-immunization with S. aureus, however, was not required for the formation of CLPU. Contact lens induced peripheral ulceration may be induced by the colonization of S. aureus on the ocular surface as well as upon contact lens, but only in the presence of traumatic change in corneal surface. However, only small number of bacteria were recovered from the ulcers (Table 2). The contact lens induced ulceration in this rabbit model presented with mild to moderate inflammatory reactions, and healed quickly upon removal of contact lens without the need of antibiotics.

Conclusions

A corneal epithelial defect and S. aureus are crucial factors in the formation of contact lens induced peripheral ulceration (CLPU).

References


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