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A Case of Type 54 Human Mastadenovirus Keratoconjunctivitis Causing Severe Broad Epithelial Defect Ten Years after LASIK Surgery

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Epidemic keratoconjunctivitis (EKC) is a keratitis caused by the highly infectious human mastadenovirus (HAdV) (1,2). Here, we report a case of EKC caused by HAdV type 54 (1–3) and resulting in extensive corneal epithelial defect. The case occurred 10 years after laser-assisted in situ keratomileusis (LASIK) surgery.

A 36-year-old man presented with pain in his right eye 10 years following uncomplicated LASIK procedures for myopia in both eyes at another hospital. All family members had contracted EKC from the man’s 2-year-old son, who was diagnosed by a local ophthalmologist using a rapid diagnostic test in early August 2015. On August 11 (day 1), the man noticed pain in his right eye. He visited a local ophthalmology clinic and was prescribed 0.5% levofloxacin and 0.1% fluorometholone eye drops. The pain gradually worsened, and an ocular infection was evident in his left eye on day 6; the man presented at Fukuoka University Hospital on day 7.

On examination, the man had difficulty opening the right eye due to edema of the eyelid. The right eye had a severe ocular infection, and a conjunctival pseudomembrane was observed (Fig. 1A). The right cornea had a circular epithelial defect coinciding with the LASIK flap edge (Fig. 1B, C). The left eye also had an ocular infection, but no epithelial defect or subepithelial opacification was observed. Visual acuity was 20/20 (uncorrectable) in the right and 20/25 (corrected) in the left eye. Conjunctival scrapings of the right and left eyes were positive and negative, respectively, on immunochromatography testing (Capilia Adeno Eye, Tauns Laboratories Inc., Izunokuni, Japan). HAdV-54 was detected in the conjunctival scrapings of both eyes, using the method reported previously that is based on polymerase chain reaction amplification, sequencing, and phylogenetic analysis (4). Based on the clinical course, slit lamp examination findings, and laboratory test results, we diagnosed the man’s condition as EKC. The conjunctival pseudomembrane was removed, and the patient was treated with 0.1% betamethasone and 0.3% gatifloxacin eye drops.

On day 8, the right eye exhibited extensive desquamation of the corneal epithelium that appeared to coincide with the LASIK flap edge (Fig. 1D). Visual acuity was 20/40 (uncorrectable) in the right and 20/20 (corrected) in the left eye. Conjunctival scrapings of both eyes were examined on the method reported previously that is based on polymerase chain reaction amplification, sequencing, and phylogenetic analysis (4). Based on the clinical course, slit lamp examination findings, and laboratory test results, we diagnosed the man’s condition as EKC. A circular-shaped corneal epithelial defect with an edge coinciding with the LASIK flap (arrows) in the right eye on his first visit is visible. C) Right eye on first visit. Arrows indicate the edge of the LASIK flap. D) Right eye on day 8. The right eye showed extensive desquamation of the corneal epithelium that appear to coincide with the flap edge (arrows).

Fig. 1. Photograph of patient’s right upper eyelid on his first visit. A) Conjunctival dot hemorrhages (arrowheads) and pseudomembranes (area enclosed in line) are shown. B) A circular-shaped corneal epithelial defect with an edge coinciding with the LASIK flap (arrows) in the right eye on his first visit is visible. C) Right eye on first visit. Arrows indicate the edge of the LASIK flap. D) Right eye on day 8. The right eye showed extensive desquamation of the corneal epithelium that appear to coincide with the flap edge (arrows).

On day 9, the corneal epithelium started to regenerate beyond the LASIK flap. On optical coherence tomography of the anterior segment of the right eye, the central corneal thickness was 471 μm, and the LASIK flap edge was observed in the corneal stroma. There was no stromal damage that resembled LASIK flap desquamation (Fig. 2). In contrast, the central thick-
ness of the left eye was 516 μm. The man’s right cornea continued to gradually reepithelialize, and it had almost completely reepithelialized by day 16. On day 32, his right eye had recovered completely, except for slight opacification. Visual acuity was 20/25 (uncorrectable) in the right and 20/16 (corrected) in the left eye.

Ethical approval of this study was given by the Ethics Committee of Fukuoka University (16-2-24), and informed consent was obtained from the patient.

In 2015, we had an epidemic of EKC with HAdV-54 in Japan. The clinical and epidemiological characteristics of this epidemic were reported, including cases of corneal complications and intra-family infection (5); however, these clinical characteristics had also been reported in the past with several HAdV types. Thus, it cannot be directly concluded that HAdV-54 has serious virulence against the ocular surface from this single case. The specific point in the present case is that HAdV-54 was detected and a broad epithelial defect that coincided with the LASIK flap edge was also observed.

To our knowledge, although a few EKC cases after LASIK have previously been reported (6), HAdV was not detected in laboratory tests in those cases. However, HAdV-54 was detected in our case, and it was associated with severe ocular disorders. A case in which dislocation of the LASIK flap happened 5 years after LASIK due to blunt trauma has been previously reported (7). Thus, we initially assumed that the broad corneal epithelial defect occurred due to dislocation of the LASIK flap. However, in a previous report that measured the thickness of the cornea under the flap, its thickness was between 395 μm and 244 μm (8), which was much thinner than that in our case (471 μm) (Fig. 2). The small reduction in corneal thickness compared with that of the contralateral eye (45 μm) also leads to the presumption that entire flap loss did not occur in our case. As there are few reports on cases similar to the present case, it is difficult to explain the reason for LASIK flap loss in EKC in our case; however, it might be possible that incomplete trigeminal nerve innervation induced weakness of the epithelial construction after LASIK surgery. It has been reported that it takes an average of one year after surgery to recover corneal sensation after LASIK, and the trigeminal nerve is involved in maintaining corneal epithelial homeostasis (9). In our case, even though 10 years had passed after LASIK was performed, the corneal epithelium might have been defective at a specific layer due to incomplete corneal sensation recovery.

In summary, we encountered a case of EKC with HAdV-54 that caused an extensive corneal epithelial defect after LASIK. When a patient who had undergone LASIK suffers from EKC, the patient should be examined for possible corneal complications, and careful observation of the clinical course is necessary, as the condition can worsen. These precautions will be of significant benefit for public health.

Conflict of interest None to declare.

REFERENCES