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Acute Ocular Keratopathy Secondary to Calotropis Latex Exposure: A Case Report in the United Arab Emirates

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Thuslim Sheik [1], Marriam Tasneem [1], Gargi Sharma [1], Simanta Khadka [2]

1. Department of Ophthalmology, Gulf Medical University, Ajman, ARE 2. Department of Ophthalmology, Thumbay University Hospital, Ajman, ARE

Corresponding author: Thuslim Sheik, thuslimsheik@gmail.com

Abstract

Ocular exposure to *Calotropis procera* latex is an uncommon but clinically significant cause of toxic keratopathy, particularly in regions where the plant is endemic. The latex contains bioactive compounds capable of inducing corneal endothelial dysfunction and stromal edema, often with minimal epithelial involvement. We report a case of a 27-year-old male who presented with acute-onset redness, pain, and blurred vision in the right eye following accidental exposure to *Calotropis procera* latex. Initial examination revealed reduced visual acuity (6/24), conjunctival congestion, superficial punctate keratitis, and a quiet anterior chamber. Imaging with anterior segment optical coherence tomography and specular microscopy supported corneal involvement. After medical management, rapid clinical improvement was observed, with visual acuity improving to 6/9 within 24 hours and complete recovery to 6/6 within one week. Resolution of corneal edema, Descemet's folds, and conjunctival congestion was noted, with complete recovery at three weeks follow-up and no residual sequelae. *Calotropis procera* latex exposure can result in reversible toxic keratopathy, which should be treated with prompt irrigation and early initiation of topical corticosteroid therapy. Increased awareness is essential in regions where the plant is prevalent, including the Middle East.

Categories: Ophthalmology
Keywords: calotropis procera, corneal edema, descemet's membrane folds, endothelial toxicity, plant latex injury, toxic keratopathy, united arab emirates

Introduction

Calotropis procera, commonly known as Sodom apple or Madar, is a xerophytic shrub belonging to the family Apocynaceae, widely distributed across tropical regions and desert climates throughout the world, especially in West and South Asia, Africa, and Indochina [1,2]. It produces a thick, milky latex that is released when the leaves or stems are broken. Although the plant has long been used in traditional medicine for its anti-inflammatory and analgesic properties, the raw latex is highly toxic when applied locally, particularly to the ocular surface [2,3].

Ocular exposure is a rare phenomenon that most commonly occurs accidentally during improper handling of the plant, plucking flowers, agricultural work, or eye rubbing after physical contact with its latex. The latex contains several alkaloids, such as calotropin, catotoxin, calcilin, and gigantin, along with other bioactive compounds that trigger an intense inflammatory reaction [4-7]. Clinically, toxicity has been described as occurring in two phases: an initial acute chemical injury characterized by burning, lacrimation, and epithelial disruption; followed by a toxic phase marked by painless diminution of vision due to the anesthetic properties of the latex, along with corneal stromal edema and Descemet's membrane folds [2,6,7].

Several case reports and small case series have identified corneal edema with striate keratopathy characterized by folds in Descemet's membrane due to corneal swelling as a prominent clinical feature [2,3,6]. Endothelial involvement has been shown in selected cases using specular microscopy, showing reduced endothelial cell counts and morphological alterations, suggesting direct endothelial toxicity [2]. While most patients usually recover with timely topical steroid therapy, delayed presentation may increase the risk of complications [2,4,5].

While the majority of published cases originate from India and other parts of South Asia, *Calotropis* is also found in the Middle East, including the United Arab Emirates (UAE). Due to increasing urban landscaping, desert activities, and agricultural farming in the UAE, it is important to create awareness regarding the effects of this accidental ocular exposure. Hence, we present a case of *Calotropis procera*-induced ocular toxicity in a patient from the UAE, highlighting the clinical features, diagnostic evaluation, and treatment of this condition.

Case Presentation

A 27-year-old male of Asian nationality presented to the Ophthalmology department with complaints of

redness, pain, and blurred vision in the right eye. He reported a history of accidental entry of plant latex into his right eye while trying to pluck a leaf on the previous evening. While sitting behind a *Calotropis* plant and talking to a family member over the phone, he accidentally plucked a leaf (Figure *1*), resulting in a splash of milky latex into his right eye. This led to immediate, excruciating pain and sudden visual blurriness. He rushed to wash the eye with running tap water and applied over-the-counter lubricating eye drops from a local pharmacy. He ultimately presented to the hospital due to worsening ocular pain and persistent blurriness. He was initially managed with copious irrigation of the affected eye before proceeding with further ophthalmic examination.



FIGURE 1: Calotropis procera plant.

The image displays *Calotropis procera*, an endemic plant. Image reproduced from Wikimedia Commons under the Creative Commons Attribution-Share Alike 4.0 International (CC BY-SA 4.0) license [8].

Examination findings at presentation showed uncorrected visual acuity (UCVA) of 6/24 in the right eye (OD) and 6/6 in the left eye (OS). Gross torchlight examination showed right periorbital and eyelid ecchymosis with a few urticarial lesions, consistent with an associated cutaneous reaction after latex exposure. Slit-lamp examination of the right eye showed diffuse conjunctival congestion, a few areas of superficial punctate keratitis (SPK), corneal striae, Descemet's membrane folds, and central corneal haze (Figure *2*). The anterior chamber was quiet and normal in depth. The findings of the anterior and posterior segments were within normal limits in the fellow eye. Intraocular pressure was 16 mm Hg in both eyes. Ocular investigations were carried out, imaging the cornea with a specular microscope and anterior segment optical coherence tomography (Figure *3*). Baseline anterior segment optical coherence tomography (AS-OCT) measured a central corneal thickness of 636 µm. Specular microscopy reported a central corneal thickness of 621 µm, endothelial cell density of 2536 cells/mm^2, and coefficient of variation of 35%; hexagonality was unavailable (Figure *3*).



FIGURE 2: Examination findings of the right eye at presentation.

A: Ecchymosis and a few urticarial rashes present over periorbital skin and eyelid. Diffuse conjunctival congestion.

B: Magnified slit view of the cornea showing presence of central haziness due to localized edema, Descemet's folds, and corneal striae.

C: Superficial punctate keratitis after fluorescein dye staining.

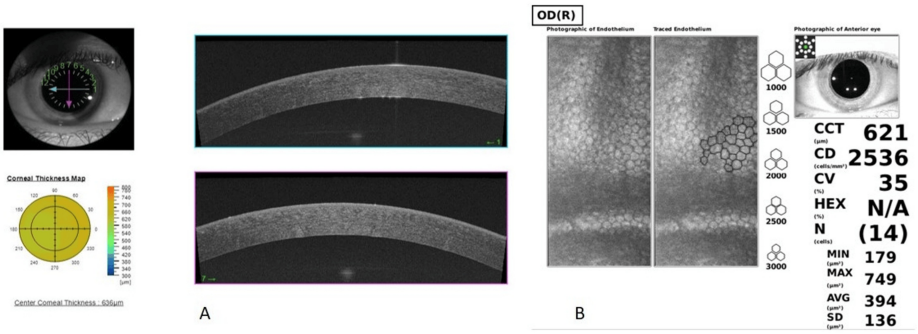


FIGURE 3: Corneal imaging using Anterior Segment Optical Coherence Tomography and Specular Microscopy.

A: Anterior segment Optical Coherence Tomography revealing a central corneal thickness of approximately 636 µm.

B: Corresponding Specular Microscopic analysis of the corneal endothelium showing central corneal thickness of 621 µm, endothelial cell density of 2536 cells/mm², coefficient of variation of 35%, unavailable hexagonality, and a sample of 14 analyzed cells. The small baseline sample limits the reliability and serial comparability of the endothelial parameters.

The patient was managed with oral levocetirizine 5 mg once daily at bedtime for seven days, topical prednisolone acetate 1% suspension four times daily for five days, followed by a gradual taper to three times daily, twice daily, and once daily at five-day intervals. Topical moxifloxacin was administered four times daily for three weeks, along with lubricating eye drops, one drop three to four times daily. A combination topical antibiotic-steroid ointment was also applied at bedtime for 10 days. Moxifloxacin eye drops were used during the day to provide topical antibacterial coverage, while the combination antibiotic-steroid ointment was applied at bedtime to provide additional overnight coverage. The topical antibiotics were used as prophylactic coverage following the chemical injury to the corneal surface.

The patient was reviewed the next day, when he reported feeling symptomatically better and having a reduction of visual symptoms. Examination findings demonstrated improved UCVA to 6/9 in the right eye. The findings also revealed decreased ecchymosis of the periorbital skin, decreased congestion of the conjunctiva, resolved corneal SPKs, and a few corneal striae and Descemet's membrane folds, with a relatively clear cornea in the center. Intraocular pressure was 17 mmHg OD and 16 mmHg OS. The patient was advised to continue the same treatment plan and was scheduled for review after a week. The patient was compliant with the medications and was symptomatically relieved at his one-week follow-up. The findings suggested a UCVA of 6/6 and the presence of mild conjunctival congestion and a clear cornea. Specular microscopy at this visit reported a central corneal thickness of 562 µm, endothelial cell density of 2473 cells/mm², coefficient of variation of 28%, hexagonality of 50%, and 113 analyzed cells (Figure *4*). The prednisolone acetate taper was continued. His subsequent follow-up three weeks after the initial presentation was uneventful, with full recovery, and the findings were within normal limits (Figure *4*).

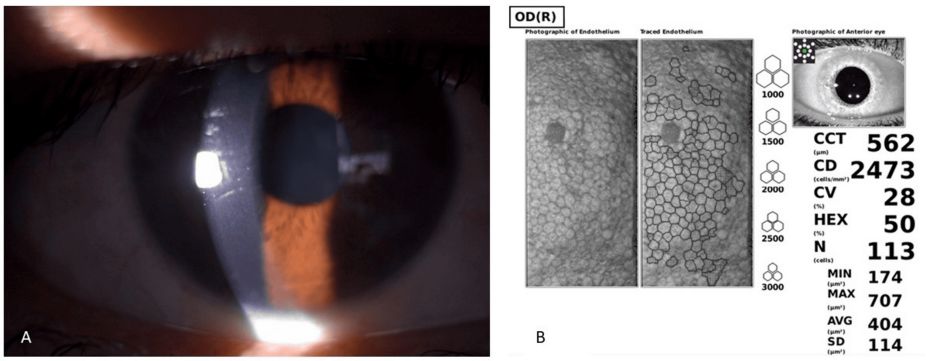


FIGURE 4: Examination and investigation findings during the follow-up.

A: Slit lamp parallel-piped picture of the cornea after one week, showing resolution of conjunctival congestion with reduced corneal striae and Descemet's folds.

B: Specular microscopy of the right eye showing central corneal thickness of 562 µm, endothelial cell density of 2473 cells/mm², coefficient of variation of 28%, hexagonality of 50%, and a sample of 113 analyzed cells.

Discussion

Ocular exposure to the latex of *Calotropis procera* produces a distinct clinical presentation that requires careful evaluation [1]. Diagnosis mainly relies on obtaining a clear history of accidental contact with the plant's milky latex. Patients usually report a sudden decrease in vision, often starting with a brief burning sensation that soon becomes painless, along with photophobia and blurred vision. This absence of persistent pain is considered to be an important clinical feature and is believed to be due to the anaesthetic properties of the latex itself [6].

The most common presentation includes conjunctival redness and corneal edema. Imaging often reveals folds in the Descemet's membrane, causing visible striate keratopathy [2]. Pachymetry is useful during the acute phase to monitor increased corneal thickness [2].

Even when the exposure history is clear, other conditions should still be considered in the differential diagnosis, as the latex of *Calotropis procera* has an acidic pH of approximately 4.2, due to which its initial presentation can closely resemble an acidic chemical injury to the eye [6]. However, unlike typical chemical burns that primarily affect the corneal epithelium, *Calotropis* toxicity appears to cause greater damage to the corneal endothelium while relatively sparing the epithelium [6]. The clinical appearance of stromal edema and membrane folds can also mimic viral disciform keratitis [7]. In more severe cases, there has been significant toxin penetration causing toxic iridocyclitis presenting with cells and flare in the anterior chamber resembling uveitis [2]. If this persistent inflammation leads to increased intraocular pressure, differentiating the condition from inflammatory glaucoma becomes necessary [7].

Published studies have reported a male predominance among patients with ocular *Calotropis* exposure [7,9]. This may partly reflect differences in occupational and environmental exposure, although accidental exposure can occur in any individual handling the plant.

There is some variability in clinical findings in studies across different populations, mainly regarding the severity of conjunctival congestion and corneal folds [7,9]. Prompt and thorough ocular irrigation is an important component of initial management to minimize continued exposure of the ocular surface to the latex [2,7].

In the majority of published literature, most patients achieve favorable visual outcomes when treatment is initiated at an early stage of presentation [2,6,7,9]. In this case, the use of topical steroids, antibiotics, and supportive care led to rapid improvement. Visual acuity returned to 6/6 within one week, the cornea became clinically clear, and the central corneal thickness measured by specular microscopy decreased from 621 µm at presentation to 562 µm at one week. The clinical course is consistent with reports describing favorable recovery.

This result aligns with existing literature showing that most patients recover well with standard medical management [2,7,10-12]. The uncomplicated recovery in this patient supports early ophthalmic evaluation following *Calotropis* latex exposure [6,9,12].

Although clinical and visual recovery is commonly reported, subclinical endothelial abnormalities may persist [6,7,10]. In this patient, endothelial cell density measured 2536 cells/mm² at presentation and 2473

cells/mm² at one week. However, the baseline analysis included only 14 cells compared with 113 cells at follow-up, and hexagonality was unavailable at baseline. These measurements therefore do not establish significant endothelial cell loss or complete endothelial recovery. The short follow-up period and absence of repeat imaging at three weeks are additional limitations, and longer-term follow-up would be required to assess residual endothelial compromise.

As *Calotropis procera* is common in the UAE for landscaping and traditional uses, clinicians should remain aware of this uncommon cause of toxic keratopathy, and emphasize immediate irrigation and treatment following accidental exposure. Preventive strategies may include public education regarding the potential ocular toxicity of the latex, appropriate warning signage in areas where the plant is prevalent, and occupational safety education with suitable eye protection for workers who regularly handle the plant.

Conclusions

Calotropis procera latex exposure can produce a characteristic but potentially misleading form of toxic keratopathy, making a clear history of exposure essential for diagnosis. This case demonstrates that early presentation, prompt irrigation, and timely use of topical corticosteroids are key to achieving rapid and complete visual recovery. In regions such as the UAE, where the plant is prevalent, greater awareness may help prevent delays in diagnosis and management.

Additional Information

Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

Concept and design: Thuslim Sheik, Gargi Sharma, Simanta Khadka, Marriam Tasneem

Acquisition, analysis, or interpretation of data: Thuslim Sheik, Gargi Sharma, Simanta Khadka, Marriam Tasneem

Drafting of the manuscript: Thuslim Sheik, Gargi Sharma, Marriam Tasneem

Critical review of the manuscript for important intellectual content: Thuslim Sheik, Gargi Sharma, Simanta Khadka, Marriam Tasneem

Supervision: Thuslim Sheik, Simanta Khadka

Disclosures

Human subjects: Informed consent for treatment and open access publication was obtained or waived by all participants in this study. **Conflicts of interest:** In compliance with the ICMJE uniform disclosure form, all authors declare the following: **Payment/services info:** All authors have declared that no financial support was received from any organization for the submitted work. **Financial relationships:** All authors have declared that they have no financial relationships at present or within the previous three years with any organizations that might have an interest in the submitted work. **Other relationships:** All authors have declared that there are no other relationships or activities that could appear to have influenced the submitted work.

References

1. Bhardwaj GS, Jain A, Jangid T, Jangir RN, Sharma A: Calotropis procera: a comprehensive review of its phytochemistry, ethnomedicinal uses, and pharmacological potential. S Afr J Bot. 2025, 185:235-76. 10.1016/j.sajb.2025.08.001
2. Waikar S, Srivastava VK: Calotropis induced ocular toxicity. Med J Armed Forces India. 2015, 71:92-4. 10.1016/j.mjafi.2012.08.017
3. Pandey N, Chandrakar AK, Garg ML, Patel SS: Calotropis procera -induced keratitis. Indian J Ophthalmol. 2009, 57:58-60. 10.4103/0301-4738.44492
4. Kaur R, Baghla P, Dahiya P, Rai S: Calotropis procera-induced keratitis. Delhi J Ophthalmol. 2024, 34:154-6. 10.4103/DLJO.DLJO_9_24
5. Mittal R, Chauhan A: A study of Calotropis induced ocular toxicity in Western Rajasthan . Indian J Appl Res. 2017, 7:42-3.
6. Bahkir FA: Spectrum of Calotropis procera latex-induced ocular toxicity . J Clin Ophthalmol Res. 2020, 8:125-7. 10.4103/jcor.jcor_11_20
7. Basak SK, Bhaumik A, Mohanta A, Singhal P: Ocular toxicity by latex of Calotropis procera (Sodom apple) . Indian J Ophthalmol. 2009, 57:232-4. 10.4103/0301-4738.49402
8. Calotropis procera. (2025). Accessed: August 19, 2026: https://commons.wikimedia.org/wiki/File:Calotropis_procera_kz09.jpg.
9. Rathi VM, Das AV, Mahajan M, Khanna RC: Demographics and clinical profile of patients with ocular

Calotropis poisoning in India. Indian J Ophthalmol. 2021, 69:2417-20. 10.4103/ijo.IJO_3434_20
10. Al-Mezaine HS, Al-Amry MA, Al-Assiri A, Fadel TS, Tabbara KF, Al-Rajhi AA: Corneal endothelial cytotoxicity of the Calotropis procera (ushaar) plant. Cornea. 2008, 27:504-6. 10.1097/ICO.0b013e3181611c34
11. Kumar J, Singh S, Garg A: Ocular toxicity caused by Calotropis . IOSR J Dent Med Sci. 2020, 19:29-34.
12. Goyal A, Kumar D: Ocular toxicity by latex of Calotropis procera in Rajasthan . J Evol Med Dent Sci. 2014, 3:12808-12. 10.14260/jemds/2014/3699