Case Report

JHSMR Journal of Health Science and Medical Research

Conjunctival Argyrosis Following Silver Nitrate Injury

Virintorn Prapakornkovit, M.D., Chalisa Jitudomtham, M.D., Weerawat Kiddee, M.D., Orasa Horatanaruang, M.D., Lalitwadee Chotuk, M.D.

Department of Ophthalmology, Faculty of Medicine, Prince of Songkla University, Hat Yai, Songkhla 90110, Thailand.

Received 20 October 2025 ● Revised 22 December 2025 ● Accepted 5 January 2026 ● Published online 23 September 2026

Abstract:

This report describes a rare case of conjunctival argyrosis secondary to a silver nitrate burn, and its clinical presentation, management, and outcome. A 41-year-old female nursing assistant presented with immediate severe irritation in her left eye after accidental exposure to a silver nitrate stick. The injury was classified as a grade II chemical burn, based on Dua's classification. The initial management included copious irrigation with a balanced salt solution, followed by the administration of topical steroids and antibiotics, artificial tears, oral doxycycline, and oral vitamin C. Shortly after treatment, a gray-white hyperpigmented patch developed at the site of the conjunctival burn, consistent with conjunctival argyrosis. The patient experienced no associated vision loss or significant ocular complications during the limited follow-up period. Silver nitrate exposure can lead to conjunctival argyrosis, an uncommon but visually benign sequela. Early recognition and appropriate management are essential for optimizing patient outcomes.

Keywords: argyrosis, chemical injury, conjunctival burn, ocular argyrosis, silver nitrate

Contact: Virintorn Prapakornkovit, M.D.
Department of Ophthalmology, Faculty of Medicine, Prince of Songkla University,
Hat Yai, Songkhla 90110, Thailand.
E-mail: virintorn.p@psu.ac.th

J Health Sci Med Res
doi: 10.31584/jhsmr.20261430
www.jhsmr.org

Introduction

Ocular argyrosis, the most common type of localized argyrosis[1], typically results from the prolonged local use of silver nitrate or colloidal silver compounds. This condition may also develop with the use of certain eyelash and eyebrow dyes[2]. Ocular argyrosis is most often associated with chronic exposure, and acute cases are uncommon.

Silver nitrate, a versatile compound with several medical applications, is typically colorless but turns black when exposed to light or organic materials. Because of its astringent and anti-inflammatory properties, silver nitrate is used to treat superior limbic keratoconjunctivitis (SLK) via chemical cauterization. Notably, a dilute solution of silver nitrate (0.5% or 1%) may be indicated for localized chemical cautery of the superior conjunctiva in refractory cases of SLK[3].

Nevertheless, silver nitrate is also known for its toxic effects. Prolonged contact with or ingestion of silver salts can lead to a condition called generalized argyria in which the skin and mucous membranes develop a gray discoloration, potentially resulting in organ dysfunction.

In this case report, we describe the case of a female patient who developed acute conjunctival argyrosis following direct contact with silver nitrate.

Case report

A 41-year-old female nursing assistant presented to the emergency department after being struck in the left eye with a silver nitrate stick while assisting with burn wound dressing. The patient reported immediate severe irritation and immediately rinsed her eye with 1 L of tap water. The initial evaluation revealed a pH of 7.5 in the affected eye. After further irrigation with 1 L of balanced salt solution, the pH normalized to 7.0. Uncorrected visual acuity (UCVA), which was measured using the Snellen chart, was 20/40–1 in the right eye and 20/80–1 in the left eye. External examination revealed diffuse ciliary injection without involvement of the eyelid skin. Conjunctival examination revealed a gray-white area extending from the lower tarsal conjunctiva to the inferior bulbar conjunctiva in the left eye, sparing the deepest fornix (Figure 1A). No evidence of limbal ischemia was noted. The upper tarsal conjunctiva showed diffuse fine papillae without foreign bodies. Slit-lamp examination revealed diffuse moderate punctate epithelial erosion and a small corneal epithelial defect, measuring 1×1 mm, which stained positively with fluorescein in the inferior paracentral area, but without associated opacity (Figure 1B). The intraocular pressure was within normal limits. Dilated fundus examination revealed no abnormalities in the posterior segment. The patient was diagnosed with chemical injury to the left eye, classified as grade II (limbal involvement of no more than 3 clock hours and no more than 30% conjunctival involvement), based on Dua's classification.

In the outpatient department, an ophthalmologist removed several gray-white patches from the inferior tarsus and fornix using a lidocaine-soaked sterile cotton-tipped applicator (Figure 1C). Fluorescein staining revealed conjunctival burns, with some areas remaining unaffected (Figure 1D). The patient was started on a comprehensive treatment regimen that included the following topical medications: methylprednisolone acetate 1% (every 1 hour [h]), moxifloxacin 0.5% (four times daily), preservative-free artificial tears (every 1 h), and a combination of dexamethasone, neomycin, and polymyxin B ointment applied nightly. In addition, oral doxycycline (100 mg twice daily) and vitamin C (1 g daily) were administered. Considering the severity of the injury, the patient was admitted to our hospital for close monitoring and management.

After one day of treatment, the conjunctival burns appeared more prominently white, with some areas showing hyperpigmentation. Fluorescein staining of the conjunctiva remained stable and the corneal epithelial defect healed,

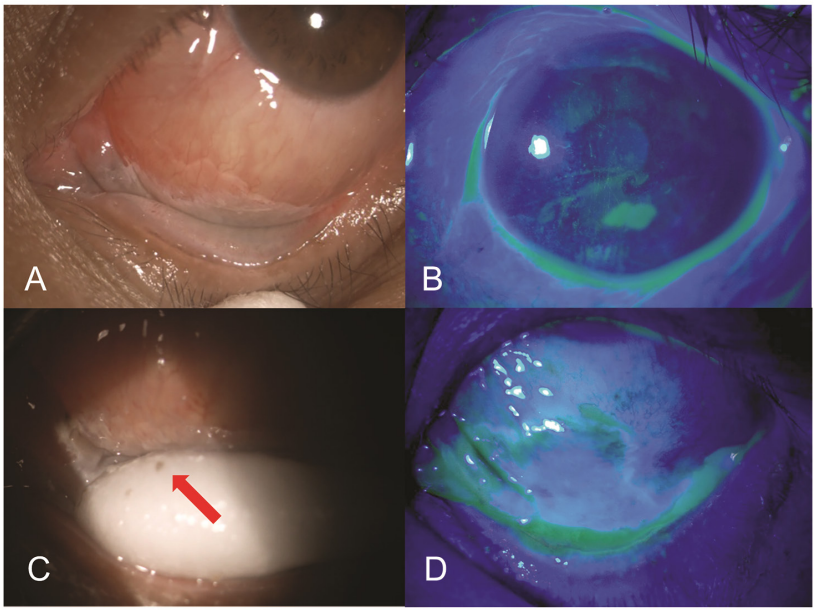

Figure 1 (A) Conjunctival injury photographed immediately on presentation to the emergency department, showing necrotic conjunctival tissue as a gray–white area, with moderate bulbar conjunctival injection and mild chemosis. (B) Fluorescein staining reveals diffuse moderate punctate epithelial erosion (PEE) and a 1×1 mm corneal epithelial defect in the inferior paracentral area. (C) Small gray–white epithelial slough (red arrow). (D) Fluorescein staining of the inferior conjunctiva shows areas of conjunctival burn.

although dense punctate epithelial erosion persisted. The corneal stroma showed slight edema and mild Descemet's membrane folds. The UCVA of the patient was 20/125, whereas her best-corrected visual acuity (BCVA) was 20/80+2. Intraocular pressure remained normal. All medications were administered as prescribed.

By the second day, significant conjunctival hyperpigmentation had developed in the bulbar and tarsal conjunctivae (Figure 2A). The corneal stromal edema resolved, which improved the UCVA to 20/25–2.

On the third day, the conjunctival burn with hyperpigmentation began to shed spontaneously. Small conjunctival hemorrhages were visible but without active bleeding (Figure 2B). The remaining adherent hyperpigmented tissue could not be removed manually with a sterile cotton-tipped applicator.

Topical atropine 1% eye drops were added to the treatment to reduce ciliary spasm, and the intensive steroid drops were tapered to every 2 h. All other medications were maintained.

At the 1-week follow-up appointment, significant improvement was noted: the ciliary injection had decreased, chemosis was absent, the cornea was clear, conjunctival hyperpigmentation had subsided, and the area of conjunctival fluorescein staining was smaller (Figure 2C). After 2 weeks, the conjunctival epithelial defect was further reduced in size, indicating continued healing (Figure 2D). However, the patient was lost to follow-up after this point, and the final outcome could not be determined.

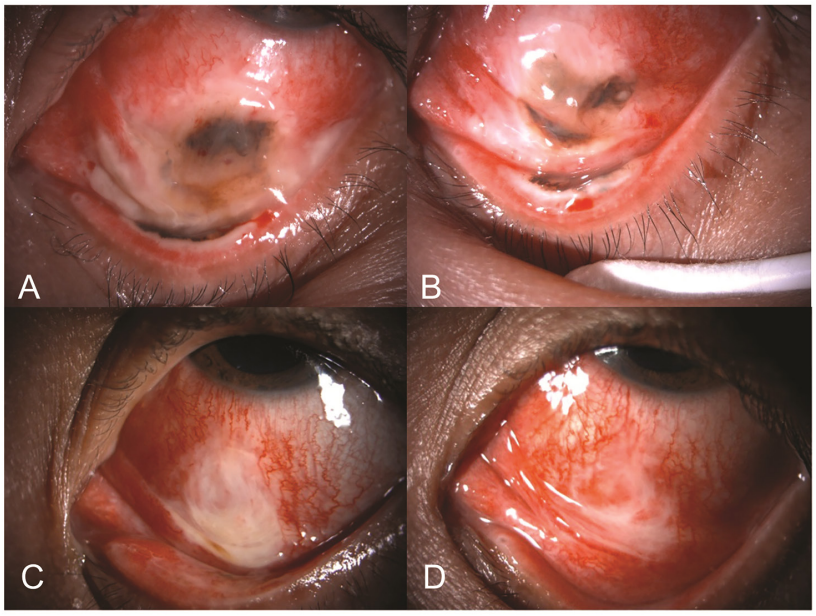

Figure 2 (A) Conjunctival hyperpigmentation in the bulbar and tarsal conjunctivae. (B) Conjunctival burn with hyperpigmentation is beginning to shed. (C) Post-treatment findings on day 7 show significant improvement with a reduced area of conjunctival fluorescein staining. (D) Post-treatment findings on day 14 show the conjunctival epithelial defect is confined to a smaller area.

Discussion

This case report highlights the clinical findings in a patient with conjunctival argyrosis. Although ocular argyrosis has been documented in previous reports[1,2], cases caused by acute direct injury or exposure are considerably rarer than those resulting from chronic exposure. Our report contributes to the understanding of the rapid presentation of conjunctival argyrosis following exposure to silver nitrate and highlights the importance of early recognition and management of this condition.

Argyria is the primary manifestation of chronic inhalation or ingestion of metallic or ionizable silver compounds[4]. Although the exact mechanism underlying dermal argyria is not fully understood, an imbalance between soluble and insoluble silver complexes in the middle or upper dermis may be involved[5].

Argyrosis of the cornea and conjunctiva results from the deposition of inert silver precipitates, and it is often a more sensitive indicator of silver exposure than dermal argyria. Although the exact mechanism of argyrosis has not been studied extensively, a pattern similar to that observed in argyria is expected[6].

Silver nitrate is notably astringent and ionizes rapidly on topical application, particularly when applied in stick form. In the present case, direct exposure of the conjunctiva to a silver nitrate stick led to acute injury to the conjunctival epithelium and the underlying substantia propria. Additionally, this exposure facilitated silver penetration into the tissues.

Despite Lansdown's observation that silver penetration is generally low, local skin discoloration can still occur, as evidenced by the use of sustained silver-release

wound dressings[7]. In our patient, a step-like progression in conjunctival coloration was observed, transitioning from light gray to darker shades. Acute injury to the conjunctival epithelium often results in necrosis, thereby leading to erosion or ulceration[8]. Silver precipitates in conjunctival wound debris are typically lost during the natural repair process, leaving behind a white area marked by conjunctival erosion, edema, and hyperemia. This finding contrasts with that of previous experimental studies[6], which report argyrosis as a permanent condition.

This report has several limitations. The absence of confocal microscopy precluded in vivo assessment of the microstructural depth of silver deposition. It is important to note that, while our patient had a normal fundus examination, retinal and choroidal changes have been reported in previous studies related to chronic exposure[1]. A conjunctival biopsy unfortunately could not be performed in this patient because of the rapid sloughing of the conjunctival hyperpigmentation. This limitation prevented a definitive histological confirmation of the cause of the hyperpigmentation. Additionally, the patient was lost to follow-up, which prevented further evaluation of the long-term outcomes. Nevertheless, we consider it likely that the hyperpigmentation was caused by silver precipitates; it did not affect visual acuity.

Conclusion

Conjunctival argyrosis can result from direct exposure to highly concentrated silver nitrate, thereby causing chemical injury and hyperpigmentation due to silver nitrate deposits, similar to the phenomenon observed with chronic silver exposure. Although ocular argyrosis causes visible conjunctival pigmentation, it typically does not affect long-term visual acuity. Clinicians applying silver nitrate should use appropriate protective equipment, such as safety goggles, to prevent accidental ocular exposure and resultant conjunctival discoloration. Future studies may help clarify optimal preventive measures and explore possible treatments for this rare but distressing complication.

Patient consent

The patient provided written informed consent for the publication of this case report.

Conflict of interest

There are no potential conflicts of interest to declare.

Funding sources

No funding or grant support was received.

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