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SEVERE OCULAR INJURY AFTER BUTTON BATTERY EXPOSURE

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□ **Abstract—Background: Button batteries can cause local tissue necrosis within 2 h of exposure due to hydrolysis of tissue fluid and generation of hydroxide ions. Tissue damage resulting from battery exposure has been associated with acute and chronic complications via several routes, however, previous experience with ocular battery exposures is predominantly limited to batteries that have exploded or penetrated the eye. Objectives: A case is presented of an intact battery causing significant damage after ocular exposure without penetrating the eye. Case Report: An 18-year-old woman presented to the Emergency Department after a toy balloon propelled a button battery into the patient's eye. The battery did not penetrate the orbit and was intact upon removal from the inferior fornix in the operating room 4 h later. The patient had severe conjunctival ulceration, subconjunctival hemorrhage, vitreous opacification, and a partially dilated pupil, with the greatest area of injury adjacent to the negative pole of the battery. The eye was extensively irrigated and the patient was treated with topical antibiotics, steroids, and a daily rodding procedure to prevent conjunctival adhesions. The eye ultimately healed over the subsequent 6 months, with normal visual acuity on follow-up. Conclusion: Prolonged ocular exposure to an intact battery can cause significant tissue necrosis, which may threaten sight. Early removal is critical to prevent significant ocular damage and visual compromise.** © 2013 Elsevier Inc.

□ **Keywords—button battery; ocular exposure**

INTRODUCTION

Button batteries lodged in the esophagus may cause localized necrosis within 2 h, with subsequent but often delayed esophageal perforation, esophageal strictures, tracheoesophageal or aortoesophageal fistulas, vocal cord paralysis, and spondylodiscitis (1). The necrosis is thought to be due to local generation of current and subsequent formation of hydroxide ions (1). Nasal exposure to button batteries has manifested as bleeding due to erosion into adjacent blood vessels, septal perforation, meatal stenosis, and necrosis of the nasal turbinates (2–5). Otic battery exposures have been associated with tympanic membrane perforation, stenosis of the ear canal, hearing impairment, facial nerve paralysis, and chondritis (3,4). A battery placed in the vagina caused severe mucosal burns (6). Although there are prior case reports of ocular injury caused by exploding batteries, splashing contents into the eye and causing penetrating trauma, ocular damage after exposure to an intact battery is unusual.

CASE REPORT

A previously well 18-year-old woman presented to an Emergency Department (ED) approximately 90 min after a toy balloon containing a light-emitting diode burst, propelling its button battery into the patient's right eye. The button battery, identified from a similar balloon, was a 7.9-mm-diameter (3.6 mm high) manganese dioxide (alkaline) cell with imprint code LR41. The patient was in severe distress, and a complete ocular examination was not possible at the time of presentation. An X-ray study demonstrated the battery in the anterior orbit in spatial relationship to the external eye, with no evidence of

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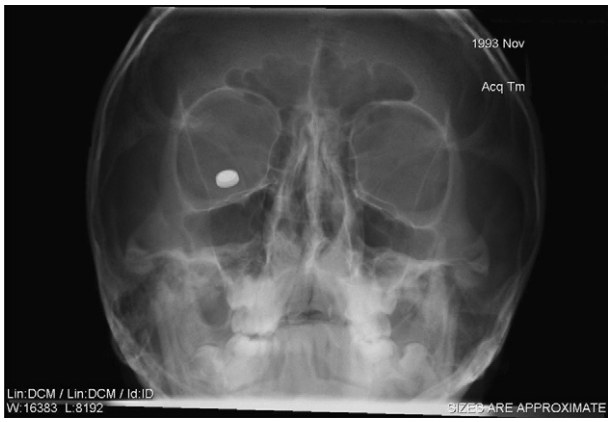

Figure 1. Skull X-ray study demonstrating battery in anterior orbit.

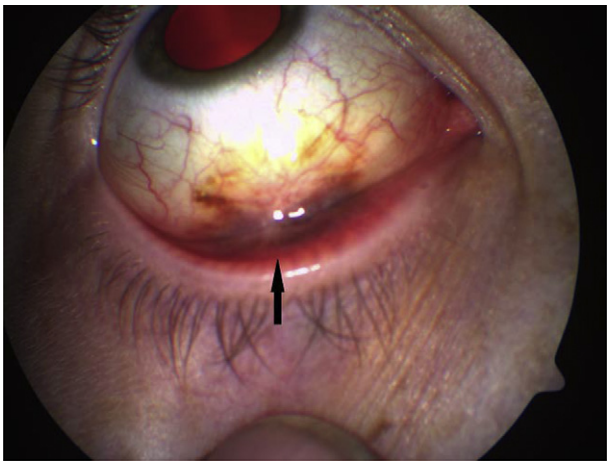

Figure 2. Curved peripheral conjunctival scar suggestive of the shape of a button battery present at 3 months post-injury.

penetration (Figure 1). Because the patient did not tolerate attempts at removal in the ED, she was taken to the operating room.

The battery was removed from the inferior conjunctival fornix under general anesthesia approximately 4 h after the injury occurred. The negative battery pole was identified as being adjacent to the bulbar conjunctiva. Further inspection confirmed that the battery did not penetrate the orbit, but was deeply embedded in the conjunctiva, which was severely ulcerated beneath the battery, possibly down to the sclera. An element of blunt trauma was suggested by irregular pupil enlargement and anterior chamber cellular activity. Vitreous condensation was noted inferiorly, with vitreous gel opacification inside the eye adjacent to the injured ocular wall. This did not affect the patient's vision due to its peripheral location. Significant subconjunctival hemorrhage was also present. The ocular surface was extensively irrigated. Despite significant tissue injury, the patient had normal visual acuity post-operatively.

In the first few weeks after the injury, the patient underwent a daily rodding procedure, which used a glass rod to separate the bulbar and tarsal conjunctiva and prevent adhesion of the tarsal conjunctival scar to the developing scar in the inferior bulbar conjunctiva. Topical steroids and antibiotic ointment were administered during the immediate post-operative period. The steroids were slowly tapered over the following weeks.

Three weeks post-injury, the patient had equal and normal visual acuity. There was moderate inflammation of the bulbar conjunctiva with associated black-brown discoloration. Subsequent follow-up over the next 3 months demonstrated stable visual acuity, with normal intraocular pressure. The only abnormality was a very peripheral conjunctival scar with a curved posterior edge suggestive of the shape of the button battery (Figure 2). The conjunctival defect and diffuse subconjunctival hemorrhage continued to heal slowly, with associated scarring. Initially, the lower half of the iris was not functioning properly, leaving the pupil partially dilated. At last follow-up, 6 months post-injury, pupil size, shape, and reactivity had returned to normal, suggesting that the initial blunt component of the injury was fairly minor. The vitreous thickening had completely resolved by the 6-month stage.

DISCUSSION

Most of our insight into the pathophysiology of button battery exposures is informed by case series and experimental studies involving battery ingestions. There are several case reports regarding ocular exposures, but these predominately occurred in the setting of battery explosion or splashed battery acid (7–9). This case is unusual in that the battery was intact and did not penetrate the orbit, yet still caused significant tissue injury.

Exposures to button batteries cause local tissue damage through three potential mechanisms (10–16). The most significant of these is the generation of an external electrical current, which hydrolyzes surrounding tissue fluid and produces hydroxide ions (alkali) that cause tissue necrosis. Perforating injuries of primary sites and adjacent structures may result. The negative pole of the battery, which is the narrow side without a "+" sign, is the site of hydroxide accumulation, thus tissue adjacent to this negative pole generally suffers more injury (1). The finding that the negative pole was in contact with the area of greatest tissue damage on the ocular surface in this patient suggests alkali generation as the major mechanism of injury. In addition, there are two secondary mechanisms by which tissue injury can occur, including direct leakage of alkaline battery contents and local physical pressure or mechanical trauma (10–16). The effects of button battery exposures are from local corrosive effects; metal-related toxicity is not expected. Experience

with battery ingestions suggests that tissue necrosis may occur as early as 2 h after ingestion, making timely removal of the battery imperative (1).

Experience with battery exposures to the eye is limited, and the vast majority of data are related to car batteries or batteries that have exploded (7–9). In these case reports, injury occurs via different mechanisms. Car batteries often contain acid that causes chemical burns and coagulation necrosis, whereas concussive injuries can result in hyphema, angle closure, and tissue edema. Vitreous hemorrhage, traumatic cataracts, optic nerve injury, and retinal detachment have also been reported (7,8). There may also be penetrating injury from battery fragments, as similar injuries have been reported with alkaline watch batteries that have exploded (7–9).

Care for such injuries is generally supportive, with copious irrigation and careful inspection. Surgical intervention may be required in more severe cases. Topical steroids and antibiotics are used as adjunctive therapy. Despite these interventions, long-term and sight-threatening complications may occur, thus early consultation with an ophthalmologist is mandatory (17).

CONCLUSION

It has been established that button batteries may cause tissue necrosis in the esophagus due to local effects within 2 h of ingestion. The case described is unusual in that it involves ocular injury from an intact battery, suggesting that there is the potential for significant injury via this route of exposure. Button batteries in the eye should be removed as soon as possible to minimize ocular damage and visual compromise.

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