Bilateral corneal contusion and angle recession caused by an airbag

EDITOR,—Airbags have been installed as standard equipment on most new cars in order to enhance automobile safety. Several reports of airbag associated injuries have recently appeared.[1-7]

We describe herein a case of severe ocular trauma caused by an airbag to a front seat passenger.

CASE REPORT

A 20-year-old woman was the belted front seat passenger involved in a car accident in a 1994 model car. The driver was killed in the accident. Our patient suffered mild contusion of the chest, Colles' fracture of the right arm, and multiple abrasions and blunt contusion marks on the face. An ophthalmic examination revealed visual acuity limited to hand movement in the right eye, and to only light perception in the left eye. The eyelids were swollen, with marked chemosis and subconjunctival haemorrhages. Opaque corneas and hyphaema were noted in each eye. No details of the fundus could be detected in either eye. A diagnosis of airbag injury was made and her eyes were irrigated with water.

On examination 2 days later, visual acuity was 20/50 in the right eye and remained unchanged in the left eye. The cornea was thick with Descemet's folds and haemorrhages were present over the irides. Sphincterotomy in the left eye gave the left pupil an oval shape. The fundus was indiscernible, and the ultrasound examination demonstrated attached retina and clear vitreous.

Two weeks after the accident, visual acuity improved to 20/50 in both eyes. Intraocular pressure was 12 mm Hg in both eyes. Gonioscopy showed an angle recession of one nasal quadrant in the right eye, and an angle recession of the whole circumference in the left eye. The rest of the anterior segment examination was unremarkable in both eyes. The right fundus was normal; however, retinal haemorrhages and oedema were present in the left fundus.

One month later, the uncorrected visual acuity was 20/20 and intraocular pressure was 16 mm Hg, again in both eyes. The slit-lamp and fundus examinations were within normal limits in both eyes.

COMMENT

Airbags are designed primarily to protect the driver and passengers from smashing against the steering wheel, dashboard, or windshield during frontal collisions. They inflate in about 10 ms in response to sudden longitudinal deceleration of approximately 20 kph and deflate within seconds. Gaseous and particulate components (sodium hydroxide, carbon monoxide) are emitted in the vehicle interior at airbag deployment.[1]

Although airbags are designed to be a safety device, they have recently been reported to be associated with facial and ocular injuries.[1-7] Skin abrasions and eyelid ecchymoses, the most common facial injuries, are usually short lived. Ocular injuries include orbital fractures, corneal oedema, abrasions, hyphaema, angle recession, lens subluxation, commotio retinae, choroidal rupture, retinal and vitreous haemorrhage, and retinal tears and detachment.[2-6] The airbag striking the face at high velocity and with great force is probably responsible for such injuries. Keratitis and corneal oedema have also been reported, and were attributed to the fine alkali aerosol released from the bag.[1]

Our patient's injuries were most probably inflicted by the airbag hitting the face, and the corneal oedema requiring irrigation with water was almost certainly caused by the aerosol released from the airbag.

Airbag injuries to a front seat passenger are rare[7] because airbags were initially installed on the driver's side, causing only the driver to suffer these kinds of injuries.

The medical community should be alert to the potential ocular injuries induced whenever an airbag is activated: immediate irrigation of the eyes with water is recommended followed by a prompt referral to an ophthalmologist.

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Airbag injury during low impact collision

EDITOR,—The fact that motor vehicle trauma continues to be a leading cause of morbidity and mortality in America, and the overwhelming evidence that airbags reduce fatalities in frontal crashes has led to airbags being standard equipment on many new and domestic cars.[1] However, reports of airbag associated ocular injuries are increasing with the more widespread use of these devices.[2-9] Because of the nature of the motor vehicle accident in the previously reported cases of airbag associated injuries the amount of trauma the patients may have sustained, without the airbag, may have been significant.

We report a case of a driver who had significant ocular and upper extremity trauma, due to airbag deployment while parking her car. The passenger, who was not subjected to an airbag, was uninjured. Because of the nature of the accident, and the uninjured passenger, we speculate that our patient may have sustained minimal injury if her airbag had not deployed.

CASE REPORT

A 49-year-old woman, wearing sunglasses, with a three point lap-shoulder seatbelt was parking a 1992 Toyota Camry when she hit a pole head on. She was travelling forward, at about 16 km/h (10 mph) in a parking lot. The passenger in the car, who was reading, thought the driver stopped abruptly, and was surprised to see that the car had hit a light pole and the driver's airbag had deployed. There was no passenger-side airbag, and the passenger was not injured.

The driver of the car was treated for head and brow lacerations. On ophthalmic examination, her visual acuity was 20/25 right eye and 20/400 left eye. The left cornea had a large, interpalpebral epithelial defect, with prominent Descemet's folds centrally. The tear pH was 7·0 in both eyes. Eversion of the left eyelid and sweeping of the conjunctival fornices yielded several pieces of glass from the patient's shattered sunglasses, which were found broken on the car floor. Each eye was irrigated with balanced salt solution, followed by normal saline. Fundus examination showed a vitreous haemorrhage, without view of the retina. Contact ultrasonography revealed that the retina was attached. She also had a radial and ulnar fracture that required orthopaedic surgery. Three days after the accident, the patient's vision improved to 20/70, with resolution of the vitreous haemorrhage. Fundus examination showed an attached retina without retinal tears.

COMMENT

The airbag in the car that this patient was driving is designed to deploy in response to a collision force greater than that created by a crash into a fixed, non-deforming barrier at approximately 19–25 km/h (12–16 mph). However, a sharp impact like a rock striking the undercarriage of the vehicle may trigger airbag deployment. Whether the airbag malfunctioned and activated without sufficient forward deceleration force, or something under the vehicle triggered the airbag, is not known. It may also be that the airbag sensor was accurate and fully operational, since our patient was travelling at about 16 km/h.

There is abundant evidence that airbags reduce fatalities when deployed for high speed frontal crashes.[1] The airbag is actuated in response to sudden longitudinal decelerations which, in turn, activate the ignition of a sodium azide propellant cartridge. The liberation of nitrogen gas from the combustion of sodium azide results in instant inflation of the airbag.[2] During inflation, the airbag is propelled out of its storage compartment at speeds typically more than 160 km/h (100 mph).[6] Airbag associated ocular injuries occur from the blunt trauma or the liberation of gas when the airbag inflates. Ocular injuries from airbag associated injuries include hyphaemas, alkali keratitis, and vitreous and retinal haemorrhage.[2-9] It has been suggested that eyeglass wear presents an additional risk factor for ocular injury during airbag inflation.[9] Polycarbonate lenses have an increased resistance to impact shatter over glass or other types of plastic. However, shattering of the frames appears to be a greater problem. Further study in this area is warranted.

No one knows for sure what injuries the driver avoided by the airbag inflation. But, considering the impact of the collision and the extent of this patient's injuries, it seems reasonable to assume the injuries the driver sustained because of the airbag are more significant than she would have sustained without it.

Our case is interesting because of the extensive injuries, from a low impact

collision, sustained by the driver while the passenger, without an airbag, had no injuries. While there is ample literature to show that the use of airbags decreases driver fatalities,[1] there are no data showing the optimum threshold impact for their deployment. Optimum threshold deployment pressure should be studied in order to maximise the benefits of fatality reduction and minimise the risks of airbag induced morbidity.

Also, in light of recent literature concerning airbag associated injuries, it would seem reasonable that airbag impact sensors should be checked regularly.

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Unilateral *Malassezia furfur* blepharitis after perforating keratoplasty

EDITOR,—*Malassezia furfur* (*Pityrosporum* genus), a yeast considered part of the normal skin microflora, is also known to cause pityriasis versicolor, folliculitis, and seborrhoeic dermatitis. We report two cases of unilateral blepharitis where *Malassezia* was retrieved from eyelid scrapings. These occurred after an uncomplicated perforating keratoplasty for keratoconus in two young (central European) patients, and resolved rapidly after local antifungal therapy.

CASE REPORTS

Case 1

A woman aged 22 underwent perforating keratoplasty. The postoperative period was uneventful. Prednisolone acetate eyedrops were applied postoperatively five times daily, and later at reduced frequency, without other therapy. At follow up 6 months later, she exhibited a slightly pigmented scaling lesion of the upper eyelid on the side of surgery, without itching or discharge. The eyelid margin was unaffected, the skin was not inflamed or ulcerated (Fig 1). There was no evidence of conjunctival infection, and the transplanted cornea was clear. No skin lesions were seen elsewhere.

Fungal infection was suspected and an ink-potassium hydroxide preparation of an eyelid scraping disclosed a spherical yeast (Fig 2) characteristic of *M furfur*. No culture was performed. Local antifungal ointment (clotrimazole) was prescribed. She missed the follow up visit, but 3 weeks later reported that she was asymptomatic. When seen 6 months later the condition had resolved.

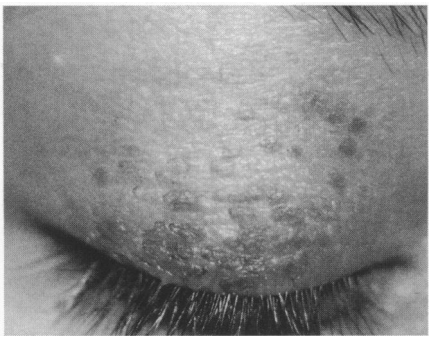

Figure 1 Pigmented scaling lesion of upper eyelid (case 1).

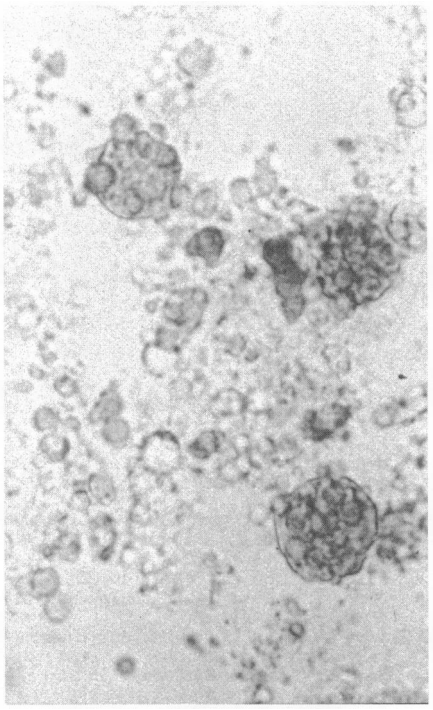

Figure 2 Potassium hydroxide staining of eyelid scraping showing spherical yeast M furfur.

Case 2

A 19-year-old man presented at follow up with a brownish scaling lesion of the upper eyelid on the side of surgery, 1 month after the operation. Postoperative treatment was identical to case 1. The eyelid margin was seborrhoeic with slightly mucopurulent discharge (Fig 3). The corneal transplant was clear, but there was mild conjunctival injection, photophobia, and tearing. The latter were probably related to the surgery, not to the eyelid lesion. A dermatologist prescribed an antibiotic-fluocinolone ointment. However, 9 months later the condition was unchanged.

This case was strikingly similar to case 1; the same organism was found. Local antifungal treatment was followed by complete resolution within 2 weeks.

COMMENT

Blepharitis is a common ophthalmic disease, involving various infection agents.[1–3] *M furfur*, a dimorphic lipophilic yeast, is part of the normal cutaneous microflora. It has been linked to seborrhoeic blepharitis, but without convincing proof.[4] The dimorphic forms have long been considered different organisms.[5] While the mycelial form is associated with

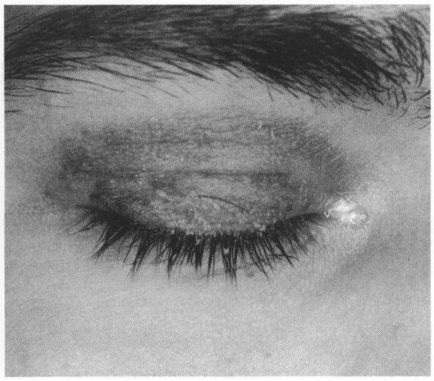

Figure 3 Brown pigmented lesion of upper eyelid (case 2), with seborrhoeic scales on eyelid margin and mucopurulent discharge.

pityriasis versicolor, the yeast form causes seborrhoeic dermatitis. In pityriasis versicolor the skin becomes scaly and skin pigmentation is altered, while seborrhoeic dermatitis gives excessive scaling and chronic inflammation.

Clinically, case 1 better fits the pityriasis versicolor category; however, the organism was a round yeast and mycelial elements were not seen. Case 2 exhibited features of pityriasis versicolor and seborrhoeic blepharitis simultaneously, with microscopic picture similar to case 1.

Information about factors predisposing to diseases caused by *M furfur* is limited. Being an opportunistic pathogen, a defect in the host defence mechanism is presumed. The strikingly similar clinical presentation and history of our patients may have a common pathogenesis. Neither patient had evidence of atopic disease, which might link keratoconus to fungal susceptibility. The lesion being unilateral, without other skin lesions, argues for a local disorder. Possibly prolonged corticosteroid use had modulated local lymphocyte transformation depressing cellular immune response. Other factors, however, cannot be excluded – for example, both patients reported that they had not washed the periorbicular region since the operation. Possibly the patients' age could be significant; we have never seen this condition in older patients after perforating keratoplasty.

It is important to consider the possibility of fungal infection in longstanding blepharitis. Eyelid scrapings are easy to perform and are valuable in establishing therapeutic guidelines.

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