

Case Report

Roller Coasters and Retinal Detachment: Case Series and Review of Acceleration-Deceleration Retinal Injury

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Keywords

Retinal detachment · Acceleration · Traumatic · Roller coaster · Case report

Abstract

Introduction: Anecdotal reports and limited reports suggest a possible link between activities involving rapid acceleration and retinal detachment. We present two novel such cases and review existing literature to investigate the plausibility of this association and delineate in what populations such an association may be more likely. **Case Presentation:** We report 2 cases of retinal detachment following roller coaster riding. The first, a 24-year-old woman with a family history of retinal detachment, presented with floaters after consecutive rides and was found to have an inferior temporal macula-sparing retinal detachment with associated retinal breaks. The second case, a 25-year-old female with a history of high myopia, presented with visual field defect and was found to have a macula-on retinal detachment with an accompanying tear at the edge of an area of lattice degeneration. Both were successfully treated with pneumatic retinopexy followed by laser retinopexy. **Conclusion:** Rapid acceleration/deceleration forces, such as those experienced on roller coasters, could potentially lead to retinal detachment. Structural predisposition is likely necessary for acceleration/deceleration injury to lead to retinal detachment, with all known cases having risk factors, including high myopia and positive family history. These same forces in eyes without structural predisposition have resulted in hemorrhage, but not detachment.

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Introduction

A retinal surgeon goes to the amusement park with his family. Standing at the base of a roller coaster, he remembers a colleague's claim that the rapid twists and turns can cause a retinal detachment. He pauses. Should the retinal surgeon get on the roller coaster?

To date, limited research has addressed the relationship between amusement rides or extreme sports and the risk of retinal detachment. Anecdotes and case reports document instances of retinal detachment occurring in close temporal association with riding roller coasters and bungee jumping. This raises the question of whether activities involving rapid changes in velocity carry the risk of retinal damage – and if so, by what mechanism.

The most common type of retinal detachment is rhegmatogenous retinal detachment (RRD), where a retinal break allows vitreous fluid to influx into the subretinal space [1]. Almost all traumatic retinal detachments are RRD [2]. Known risk factors for RRD include myopia, cataract surgery, personal history of retinal detachment in the fellow eye, family history, and trauma to the head or orbit [1].

Apart from direct blunt trauma to the head or orbit, whether indirect trauma inflicted by rapid changes in velocity may also increase the risk of RRD is poorly understood. We present two novel cases of retinal detachment occurring in close temporal association with roller coaster riding. Existing evidence for the risk of retinal detachment with rapid acceleration/deceleration forces, and the mechanistic plausibility of this association, are examined.

Case Presentation

Case 1

A 24-year-old healthy female presented to the emergency department because of floaters in her left eye. She presented approximately 2 h after riding a roller coaster three times earlier that day. The patient reported experiencing blurred vision after the third ride and sought immediate medical attention after riding a roller coaster four consecutive times. Family history was significant for retinal detachment in her mother. She was mildly myopic. BCVA was 20/20 OU at the presentation.

A dilated fundus exam revealed a bilateral tilted disc but was otherwise normal in the right eye. In the left eye, examination revealed an inferior temporal retinal detachment extending from 4:00 to 6:00. The detachment spared the macula and there was no posterior vitreous detachment (shown in Fig. 1). This was caused by two paravascular irregular retinal breaks/holes with adjacent retinal hemorrhage located at 4:00 near the equator (shown in Fig. 2). No other breaks or tears were found during the scleral depressed exam. There was no posterior vitreous detachment in either eye. The patient was treated with pneumatic retinopexy with C3F8 gas followed by a laser barricade (shown in Fig. 3). Two years after treatment, there was no progression of the retinal detachment (shown in Fig. 4). The patient was counseled to avoid acceleration/deceleration activities.

Case 2

A 25-year-old female presented by referral from her optometrist to the emergency room with a 5-day history of a visual field defect in her left eye. She was previously healthy and on no medications. She was a –7 diopter myope and had a family history of retinal detachment in her father.

She described intense pressure on her head with head bobbing after going on a roller coaster 5 days prior. Examination revealed a left macula-on retinal detachment with a retinal tear at the edge of lattice degeneration in the superotemporal periphery. The detachment was

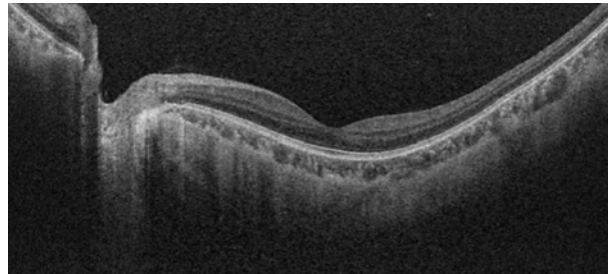


Fig. 1. Optical coherence tomography at presentation showing no involvement of the macula and absence of posterior vitreous detachment.

successfully treated with pneumatic retinopexy with SF₆ followed by laser retinopexy. The CARE Checklist has been completed by the authors for these cases and is attached as online supplementary material (for all online suppl. material, see <https://doi.org/10.1159/000540878>).

Discussion

Traumatic RRD accounts for a small proportion of all retinal detachment, with an even smaller proportion of traumatic RRD occurring in association with recreational activities [3], most commonly in contact sports [4]. These cases all involve direct trauma to the orbit or head. Experimental blunt orbital trauma demonstrates the mechanism of injury in direct trauma due to the deformation of the globe and shockwave propagation through the vitreous [5]. On the other hand, injury related to the rapid acceleration/deceleration forces experienced on roller coasters is indirect, occurring because of unconstrained movements of the head. This mechanism of injury has been characterized in the context of traumatic brain and ocular injury as contrecoup type 1, in which injury occurs because of a sudden change in the velocity of the head [6]. Previous evidence for acceleration/deceleration-associated ocular injury comes from a small number of case reports in the literature. To our knowledge, 2 previous cases of RRD occurring shortly after a roller coaster ride have been reported [7]. The first, a 54-year-old female, experienced vision loss within hours of riding a roller coaster, while the second, a 35-year-old female, noticed only floaters. Both were significantly myopic with refractive error of more than 6 diopters, and in both cases, the detachments occurred in the superior retina through a region of lattice degeneration. Only the first case had mild vitreous hemorrhage.

Vitreous hemorrhage [8, 9] or intraretinal hemorrhage [10] alone without retinal detachment has also been reported following roller coaster riding in young individuals without known risk factors. Similarly, the ride “Spaceball,” in which the rider is rapidly spun, resulted in multiple instances of ocular hemorrhage and one instance of retinal tear without detachment [11]. In a tragic roller coaster accident that resulted in the death of a 20-year-old healthy woman from severe whiplash, post-mortem examination of the eyes revealed optic nerve sheath and intraretinal hemorrhages and extensive RPE detachment but only focal small areas of retinal detachment [12]. Notably, the affected individuals in each of these cases were not known to have myopia or other RRD risk factors.

Like roller coasters, bungee jumping involves rapid acceleration/deceleration that would translate to similar forces on the retina. Two instances of bungee jumping-associated retinal detachment have been reported. The first was a healthy 28-year-old male with known myopia (−3D bilaterally), who noticed monocular blurred vision and flashes within minutes of bungee jumping and was found to have an inferior retinal tear and detachment [13]. The second, a 23-year-old male with a history of myopia (−2D) and lattice

Fig. 2. Ultra-wide field fundus photo showing 2 paravascular irregular tears located at 4:00 near the equator of the eye. No other breaks, tears, or lattice degeneration were found during a 360 scleral depressed exam.

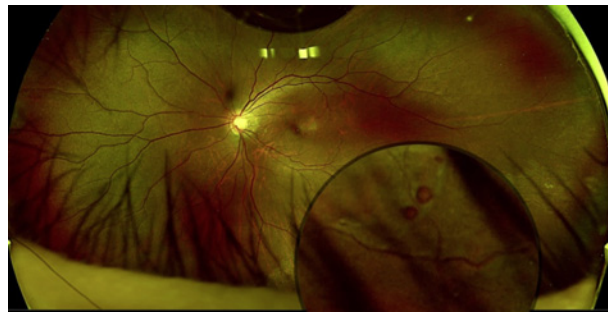


Fig. 3. Ultra-wide field fundus photo showing early laser retinopexy reaction 10 days post laser barricade treatment. Residual C3F8 gas from failed pneumatic retinopexy can be seen in the vitreous cavity.

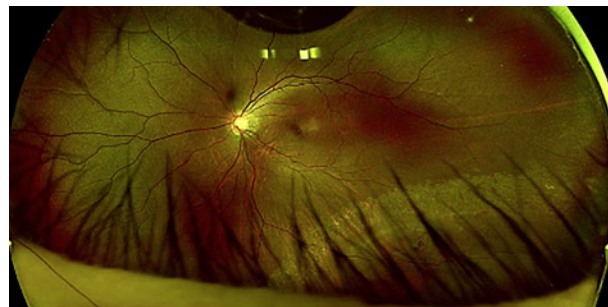
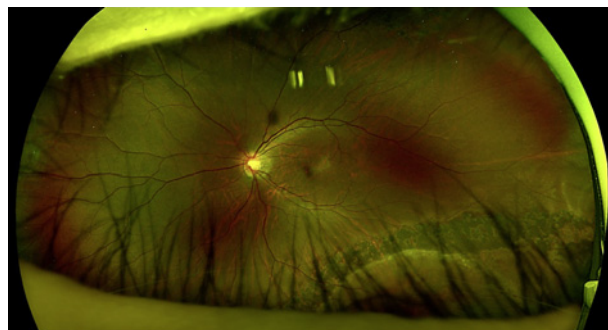


Fig. 4. Ultra-wide color photo showing no progression of the retinal detachment beyond the laser barricade 2 years after treatment. Patient remained asymptomatic.



degeneration, was found to have bilateral retinal holes, right RRD and intraretinal hemorrhage [14]. The occurrence of RRD depends on the interaction between vitreoretinal traction and predisposing structural factors, including liquefaction of the vitreous, anomalous vitreoretinal adhesions, and areas of degeneration or retinal thinning. Activities involving rapid changes in the velocity of the head, and therefore of the orbit, may alter the dynamics of vitreoretinal traction. In theory, the forces produced by rapid head acceleration/deceleration on amusement rides could overcome vitreoretinal adhesive forces [15, 16]. However, these forces alone are probably not sufficient to cause retinal breaks without underlying structural changes.

There is a clear pattern of predisposing risk factors among the cases of patients with RRD following rapid acceleration/deceleration injury: all had myopia, and most had lattice degeneration (Table 1). Indeed, most cases of RRD in young individuals are associated with high myopia [17, 18]. The lifetime risk of RRD is 3-fold higher with myopia compared to emmetropic eyes, and 39-fold higher in high myopia because of retinal thinning, early vitreous syneresis, and abnormal vitreoretinal adhesions surrounding lattice degeneration [19]. As an extreme counterexample, the “Fastest Man on Earth,” John Stapp, experienced an equivalent

Table 1. Summary of retinal detachment associated with rapid acceleration/deceleration

Case	Activity	Risk factors	Presentation (time post-activity)
24 F	Roller coaster	Myopia, family history of RD	Floaters (2 h)
25 F	Roller coaster	High myopia (−7D), lattice degeneration	Visual field defect (NA)
54 F [7]	Roller coaster	High myopia (−6D), lattice degeneration	Visual field defect (within hours)
35 F [7]	Roller coaster	High myopia (−6D), lattice degeneration	Floaters (within hours)
23 M [14]	Bungee jumping	Myopia (−2D), lattice degeneration	Blurriness, floaters (NA)
28 M [13]	Bungee jumping	Myopia (−3D)	Blurriness, flashes (immediate)

force of 46.2 G when coming to a stop on a rocket-powered sled in 1954 [20], and while intravitreal hemorrhage rendered him temporarily blind, he did not experience retinal detachment. Likewise, violent head-shaking injuries in young adults resulted in retinal hemorrhages in areas of maximal vitreoretinal adhesion but did not cause retinal detachment [21, 22].

It is possible that roller coasters cause occult tears in some riders. Detachments resulting from blunt trauma often are not clinically apparent until several months or even years after the tear-causing injury [2, 5]. Moreover, in the young healthy eye, well-formed vitreous acts as a tamponade, such that even a full-thickness retinal tear does not result in detachment [23]. However, based on the autopsy results of extreme acceleration/deceleration injury to healthy young eyes [12], occult retinal tears from roller coasters are less likely. In younger riders in whom vitreoretinal adhesion is more uniform and the vitreous remains highly viscous, acceleration-deceleration injury appears more likely to cause intraocular hemorrhage without retinal detachment [8–10].

It could also be considered that roller coasters cause the progression of existing tears. From autopsy reports, as much as 10% of the adult population has asymptomatic retinal tears [1, 24]. Left untreated, these rarely progress to retinal detachment [25–27]. The vitreous currents and sudden changes in the vitreoretinal traction produced on roller coasters may allow fluid into the subretinal space, causing the progression of asymptomatic retinal tears to RRD shortly after riding. Whether there were pre-existing tears in any of the reported cases is not known.

Conclusion

Activities involving rapid acceleration/deceleration of the head, such as riding a roller coaster or bungee jumping, can potentially transmit forces to the vitreoretinal interface sufficient to provoke retinal detachment, as evidenced by a small number of cases in individuals with predisposing risk factors. These factors include myopia or high myopia with associated lattice degeneration and family history. While individuals with known risk factors for RRD may consider precautions at the amusement park such as sensible ride choices and ensuring proper head supports, current evidence based on limited case reports is not sufficient to make broad recommendations to patients. For the ophthalmologist, the index of suspicion for RRD in young patients presenting with

symptoms of RRD may be heightened if there is a history of rapid acceleration/deceleration, even without direct trauma to the head or orbit. Finally, should the retinal surgeon get on the roller coaster for the third time? Depending on his risk factors, maybe after a dilated fundus exam.

Statement of Ethics

Written informed consent was obtained from the patient for publication of the details of their medical case and accompanying images. Research Ethics Approval was not required for this paper as per the University of Toronto Research Ethics Board. The study complies with the guidelines for human studies and was conducted in accordance with the World Health Organization Declaration of Helsinki.

Conflict of Interest Statement

The authors have no conflicts of interest to declare.

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Author Contributions

L.P. contributed to acquisition of data, drafting the manuscript, and final approval. M.C.P. and E.M. contributed to acquisition of data, critical revision, and final approval. S.N. and R.G.D. contributed to critical revision and final approval. P.Y. contributed to conception and design, acquisition of data, critical revision, and final approval.

Data Availability Statement

All data generated or analyzed during this study are included in this article and its online supplementary material. Further inquiries can be directed to the corresponding author.

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