Case report of the year 2010

Statin-associated weakness in myasthenia gravis: a case report
Michael J Keogh, John M Findlay, Simon Leach and John Bowen

Most accessed article of the year

Atrial fibrillation in healthy adolescents after highly caffeinated beverage consumption: two case reports
Jennifer R Di Rocco, Adelaide During, Peter J Morelli, Marybeth Heyden and Thomas A Biancariello

Visual recovery in a patient with total hyphema, neovascular glaucoma, long-standing retinal detachment and no light perception vision: a case report
Olusola Olawoye, Christopher C Teng, Uri Shabto, Jeffrey M Liebmann, Francis A L'Esperance and Robert Ritch

Car sunshade-induced craniofacial injury: a case report
Mahdi Sharif-Alhoseini, Hadi Khatibi, Mojtaba Chardoli and Vafa Rahimi-Movaghar

Using an Ishikawa diagram as a tool to assist memory and retrieval of relevant medical cases from the medical literature
Kam Cheong Wong
Journal of Medical Case Reports is an open access, peer-reviewed online journal that will consider any original case report that expands the field of general medical knowledge.

Reports should show one of the following:

1. Unreported or unusual side effects or adverse interactions involving medications
2. Unexpected or unusual presentations of a disease
3. New associations or variations in disease processes
4. Presentations, diagnoses and/or management of new and emerging diseases
5. An unexpected association between diseases or symptoms
6. An unexpected event in the course of observing or treating a patient
7. Findings that shed new light on the possible pathogenesis of a disease or an adverse effect

Journal of Medical Case Reports will also consider research articles related to case reporting. Suitable research articles include but are not limited to: N of 1 trials, meta-analyses of published case reports, research addressing the use of case reports and the prevalence or importance of case reporting in the medical literature and retrospective studies that include case-specific information (age, sex and ethnicity) for all patients.

Editorial Board

Editor-in-Chief
Professor Michael Kidd AM
Flinders University, Australia

“*In the era of evidence-based practice, we need practice-based evidence. The basis of this evidence is the detailed information from the case reports of individual people which informs both our clinical research and our daily clinical care. Each case report published in this journal adds valuable new information to our medical knowledge.*”

Professor Michael Kidd AM is the Executive Dean of the Faculty of Health Sciences at Flinders University in Australia, past president of the Royal Australian College of General Practitioners and president-elect of the World Organization of Family Doctors (WONCA). He is the founder and Editor-in-Chief of the *Journal of Medical Case Reports*, the world’s first journal devoted to case reports from all medical disciplines.

Deputy Editors
Dr Elie Dib
Sanford School of Medicine, USA

Professor Christian Koch
University of Mississippi Medical Center, USA

Dr Richard Rison
Neurology Consultants Medical Group, USA

Dr Jean Karl Soler
The Family Practice, Malta

Dr Geoff Wong
University College London, UK
Statin-associated weakness in myasthenia gravis: a case report

Michael J Keogh*, John M Findlay, Simon Leach, John Bowen

Abstract

Introduction: Myasthenia gravis is a commonly undiagnosed condition in the elderly. Statin medications can cause weakness and are linked to the development and deterioration of several autoimmune conditions, including myasthenia gravis.

Case presentation: We report the case of a 60-year-old Caucasian man who presented with acute onset of dysarthria and dysphagia initially attributed to a brain stem stroke. Oculobulbar and limb weakness progressed until myasthenia gravis was diagnosed and treated, and until statin therapy was finally withdrawn.

Conclusion: Myasthenia gravis may be underappreciated as a cause of acute bulbar weakness among the elderly. Statin therapy appeared to have contributed to the weakness in our patient who was diagnosed with myasthenia gravis.

Introduction

Myasthenia gravis (MG) is characterised by fatigable muscle weakness and has an incidence of only 1 in 5 to 10,000 people [1]. Autoimmune myasthenia gravis, often in association with thymus hyperplasia or thymoma, can affect young adults. However, it is now recognized that myasthenia gravis is actually more prevalent in middle-aged and older groups than younger age groups [2]. In elderly patients, bulbar presentation is common [3] and often mislabeled as a stroke [4] leading to poorer rates of survival [5].

Statins (inhibitors of 3-hydroxy-3-methyl-glutaryl-CoA reductase) lower the incidence of cerebrovascular disease and coronary heart disease. Statin use has increased dramatically over the last decade, with a four-fold increase from 1996 to 1998 [6].

Although generally well-tolerated, statins may have primary care discontinuation rates of up to 30% [7] due to their side effects such as headache, myalgia, paraesthesia, and abdominal discomfort.

Here, we report a case of acute myasthenia gravis presenting in a 60-year-old Caucasian man whose condition deteriorated until immunosuppressive therapy was commenced and statin therapy was withdrawn.

Case presentation

A 60-year-old Caucasian man of British origin was admitted to our hospital in September 2007 following acute onset of dysarthria and dysphagia. He was diagnosed with diabetes mellitus and hyperlipidaemia three months prior to presentation.

He had no visual disturbance or sensorimotor symptoms in his limbs or torso on presentation. He was commenced on gliclazide, ramipril and aspirin when he was diagnosed with diabetes and hyperlipidaemia 3 months earlier. He was also started on simvastatin at that time, but this was stopped following the development of proximal muscle weakness, myalgia, and an elevated creatine kinase (CK) of 2599 (normal: <200), which all resolved upon the termination of this medication. Gliclazide, ramipril and aspirin, however, were continued.

Aside from the finding of mild dysarthria, examination revealed that our patient had no remarkable conditions. Results of routine haematology, biochemistry, thyroid function tests, and creatine kinase were also unremarkable. His serum cholesterol was 6.1 mmol/L and his random blood glucose was 11.2 mmol/L.

An initial diagnosis of a brain stem stroke was considered, so dipyridamole and atorvastatin were added to his medication four days after his admission to our hospital. Meanwhile, a computed tomography (CT) brain scan showed that he had no obvious infarct.
Our patient remained stable over the next few days with a mild dysarthria and dysphagia (tolerating soft food), but no other symptoms or signs were noted.

One week after his admission to our hospital, his dysarthria and dysphagia worsened. Bilateral fatigable ptosis, diplopia, fatigable weakness of his neck flexion, and shoulder abduction were noted for the first time. A previously planned cranial magnetic resonance brain scan was thus cancelled.

Edrophonium testing demonstrated a dramatic transient improvement in his dysarthria, and a diagnosis of myasthenia gravis with high titre anti-acetylcholine receptor antibodies was confirmed. A serum immunoglobulin assay revealed an IgA level of <0.05 g/L. He was noted to have normal IgG and IgM, and no para protein band.

Our patient was then commenced on treatment with pyridostigmine. He was also started on incrementally increasing prednisolone every other day. Regular monitoring of his respiratory function was also initiated.

His respiratory function worsened over the next 3 days. His spirometry also deteriorated. He developed a new fatigable diplopia and an inability to stand from a low squat position, together with increasing neck and proximal limb weakness.

In view of his deteriorating state, intravenous immunoglobulin therapy (IVIg) was commenced. Following immunological advice regarding his low IgA titre, it was decided to use Vigam Immunoglobulin (2 g/kg over the next 4 days), which did not result in any adverse effect.

No objective gains were noted over the subsequent week, and a repeat CK yielded a result of 842 mmol/L. His atorvastatin medication was then stopped two weeks after it had been introduced. Following this, our patient showed significant improvement in ptosis, a resolution of diplopia, and improved neck, shoulder, and elbow power. His ability to stand from a low squat position returned, and significant spirometric improvements were also seen.

His CK readings fell over this period and returned to normal levels one week after the cessation of his statin medication (Figure 1).

Our patient remained stable until two weeks later when, just prior to a planned discharge, a further deterioration and unresponsiveness to a second course of IVIg necessitated respiratory and nutritional support, intensive care, and plasma exchange.

Following prolonged treatment, his muscle strength improved and he returned to independent living at home four months after his admission to our hospital. His gastrostomy feeding tube and tracheostomy were removed 10 months after he was discharged from our hospital.

**Discussion**

Myasthenia gravis has an incidence of only 1 in every 5 to 10,000 people and is potentially fatal. A recent study suggests that 2.2% of patients admitted with myasthenia gravis overall died during admission [8], and that the risk could be reduced by 69% if the patient is under the care of a neurologist. It is thus important not to readily dismiss the condition and that appropriate referrals are made.

The actual incidence of statin-exacerbated myasthenia is unknown, and only a handful of reports of statin-associated myasthenia gravis have ever been described [9-11].

Out of 6 published case reports, only 5 patients were noted to have some degree of recovery and only one patient had a complete recovery upon termination of statin therapy [11].

How statins could appear to exacerbate MG is unclear. It is possible that the mechanism actually reflects a “double hit” phenomenon of defective neuromuscular transmission secondary to antibody-mediated post-synaptic acetylcholine receptor dysfunction in combination with a statin-induced myopathy.

The clear development of a statin myopathy with simvastatin treatment prior to the onset of myasthenia in our patient is consistent with the possibility of a second (atorvastatin- induced) myopathy coalescing with the onset of myasthenia gravis. The symptomatic improvement that followed his withdrawal from atorvastatin treatment resulted from the resolution of this statin myopathy.

We also considered other potential causes of deterioration such as sepsis, steroid-induced worsening of MG, steroid myopathy, and cholinergic crisis, but we considered their development less likely based on clinical grounds.

We cannot rule out completely the possibility that the worsening of our patient’s MG simply reflected a progression of his MG. However, the clinical course of his condition, as well as the statin-induced proximal limb pain and weakness (without bulbar features) he experienced prior to his presentation, raises at the very least the possibility that a component of his initial deterioration was statin-related.

Similarly, we note that his improvement could have reflected the immunosuppressive effects of therapy for his MG rather than the withdrawal of his atorvastatin treatment. It seems probable, however, that both factors played a significant role in the improvement of his clinical state.

The development of other autoimmune disorders such as dermatomyositis [12], polymyalgia rheumatica, vasculitis [13], and Lupus-like syndrome [14] upon initiation
of statin therapy [13] raises the possibility that in predisposed individuals, statins may precipitate an immunological trigger that is analogous to penicillamine-induced MG [15] although clearly different in temporal respect. However, given the paucity of reports and the widespread use of statins, the possibility of chance association cannot be excluded still.

Conclusion
Myasthenia gravis is a potentially fatal condition that should be considered in elderly patients with bulbar symptoms. Statin medication should be introduced cautiously and considered as a potential cause or precipitant of worsening muscle strength in patients with myasthenia gravis.

Consent
Written informed consent was obtained from the patient for publication of this case report and any accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of this journal.

Abbreviations
CK: creatinine kinase; CT: computed tomography; IVIg: intravenous immunoglobulins; MG: myasthenia gravis.

Acknowledgements
None

Authors’ contributions
MJK reviewed the patient’s clinical data, performed the literature search, and wrote the initial draft of the manuscript. JWF, SL and JB reviewed the initial draft and finalized the manuscript. All authors read and approved the final manuscript.

Competing interests
The authors declare that they have no competing interests.

Received: 29 January 2008
Accepted: 20 February 2010 Published: 20 February 2010

References

Figure 1 This image of a graph shows creatinine kinase readings during admission, and a correlation to clinical progress.

Cite this article as: Keogh et al: Statin-associated weakness in myasthenia gravis: a case report. Journal of Medical Case Reports 2010 4:61.
Atrial fibrillation in healthy adolescents after highly caffeinated beverage consumption: two case reports

Jennifer R Di Rocco¹, Adelaide During², Peter J Morelli³*, Marybeth Heyden³, Thomas A Biancaniello³

Abstract

Introduction: Energy drinks and highly caffeinated drinks comprise some of the fastest growing products of the beverage industry, often targeting teenagers and young adults. Cardiac arrhythmias in children related to high caffeine consumption have not been well described in the literature. This case series describes the possible association between the consumption of highly caffeinated drinks and the subsequent development of atrial fibrillation in the adolescent population.

Case presentations: We report the cases of two Caucasian adolescent boys of 14 and 16 years of age at the time of presentation, each without a significant cardiac history, who presented with palpitations or vague chest discomfort or both after a recent history of excessive caffeine consumption. Both were found to have atrial fibrillation on electrocardiogram; one patient required digoxin to restore a normal sinus rhythm, and the other self-converted after intravenous fluid administration.

Conclusion: With the increasing popularity of energy drinks in the pediatric and adolescent population, physicians should be aware of the arrhythmogenic potential associated with highly caffeinated beverage consumption. It is important for pediatricians to understand the lack of regulation in the caffeine content and other ingredients of these high-energy beverages and their complications so that parents and children can be educated about the risk of cardiac arrhythmias with excessive energy drink consumption.

Introduction

Atrial fibrillation is extremely rare in the pediatric population, almost always occurring in association with structural heart disease, such as rheumatic mitral valve disease, congenital heart disease with dilated atria, and rarely, as a complication of intra-atrial surgery [1]. Patients may present with palpitations, dyspnea, fatigue, light-headedness, or syncope. The electrocardiogram is characterized by disorganized atrial activity without discrete P waves. The ventricular response is often irregularly irregular. Without a prior cardiac or family history, other inciting causes such as thyrotoxicosis, infectious pericarditis, and pulmonary emboli should be considered in the previously healthy child presenting with new-onset atrial fibrillation [2].

Exogenous causes of atrial fibrillation through a substrate such as caffeine have not been widely reported in the literature, especially in the pediatric population. A large-scale Danish study evaluating adult human caffeine consumption and arrhythmias did not find a higher risk of atrial fibrillation or flutter with variable oral consumption of caffeine from everyday sources [3]. A controlled trial of escalating doses of caffeine in dogs surprisingly found that serum caffeine actually decreased the propensity for atrial fibrillation [4]; another canine trial demonstrated an increase in cardiac arrhythmias with high doses of caffeine administered [5]. A recent case report outlined a correlation between prolonged inhaled salbutamol and concurrent chocolate abuse, leading to an atrial arrhythmia in an adult, postulating that the caffeine in the chocolate coupled with the short-acting beta agonist triggered the arrhythmia [6]. Another case report described a 58-year-old man with atrial fibrillation and a dilated cardiomyopathy, which...
resolved when he discontinued his excessive caffeine consumption [7].

Caffeine is a natural stimulant found in tea leaves, coffee beans, and cacao, and is one of the most popular psychoactive substances used today. Caffeine causes central and peripheral nervous system stimulation through antagonism of adenosine receptors and also has dopaminergic properties, which lend to its addictive potential [8]. The half-life of caffeine in a normal healthy adult is estimated to be from 2.5 to 10 hours, depending on the individual. Long-term consumption of caffeine or consumption of large amounts of caffeine will prolong its half-life [9,10]. The US Food and Drug Administration deems, “caffeine is generally recognized as safe when used in cola-type beverages up to a level of 0.02 percent” [11]. The population as a whole has variable sensitivity to the stimulant effects of caffeine; one’s tolerance and dependence on caffeine seem to be somewhat heritable and may be linked to genetic polymorphisms [8]. The physiologic and psychological effects of caffeine have been studied in adults but have not been systematically analyzed in children [8].

Energy drinks and highly caffeinated drinks comprise some of the fastest-growing products of the beverage industry, often targeting teenagers and young adults [8,12]. This case series describes the possible association between the consumption of highly caffeinated drinks and the development of cardiac arrhythmias, specifically atrial fibrillation, in the adolescent population. We report two cases of atrial fibrillation in healthy adolescent boys after the consumption of energy drinks.

**Case presentation**

**Case 1**

A 14-year-old Caucasian boy with no significant past medical history presented with persistent “heart fluttering” two hours after a running race. He denied recent illness and denied drug ingestion, but reported drinking an unknown quantity of a highly caffeinated drink the day before. He also reported drinking a Red Bull™ energy drink five days before admission and feeling the same fluttering sensation. His physical examination revealed an irregularly irregular heart rate at approximately 130 beats per minute with a 1/6 vibratory systolic ejection murmur at the left lower sternal border. Thyroid-function tests and serum calcium were normal. His electrocardiogram (ECG) showed narrow-complex tachycardia with atrial fibrillation and occasional atrial flutter (Figure 1). Cardiac ECG revealed a structurally normal heart without thrombus. He was treated with one dose of digoxin as a partial load at 7.5 μg/kg and quickly converted to normal sinus rhythm with a heart rate of 70 to 80 beats per minute (Figure 2). On follow-up examination in cardiology clinic one month later, the patient had a normal cardiac examination, a normal ECG, and no further symptoms of arrhythmia.

---

![Figure 1 Electrocardiogram showing narrow-complex tachycardia with atrial fibrillation and occasional atrial flutter with irregularly irregular ventricular response](Image)

Heart rate, 122 beats per minute; QRS, 88 ms; QTc, 433 ms.
Case 2
A 16-year-old Caucasian boy with a history of attention-deficit hyperactivity disorder, asthma, and allergies presented to the emergency department with intoxication and vomiting after falling and sustaining minor head trauma. He had ingested an unknown quantity of Red Bull™ mixed with vodka at a party. He denied chest pain, syncope, palpitations, shortness of breath, and fever. His home medications included amphetamine and dextroamphetamine (Adderall XL), 30 mg daily; montelucast (Singulair), 10 mg daily; loratadine (Claritin), 10 mg daily; and doxycycline, 100 mg daily for acne. Physical examination revealed an irregularly irregular heartbeat at 160 beats per minute with no murmurs. ECG showed chaotic atrial tachycardia/atrial fibrillation with rapid ventricular response (Figure 3). Blood ethanol level was 155 mg/dl. Cardiac enzymes were unremarkable, and serum electrolytes, thyroid-function tests, and a lipid profile were normal. A cardiac ECG revealed a structurally normal heart without thrombus. Computed tomography of his brain was normal. The patient was given a bolus of 2 L of normal saline, and his heart rate responded by decreasing from 160 beats per minute to 90 to 110 beats per minute. He remained hemodynamically stable and was placed on a cardiac monitor overnight with continued intravenous fluid support. Approximately 12 hours after presentation, he spontaneously reverted to a normal sinus rhythm (Figure 4). He remained asymptomatic with a normal sinus rhythm during subsequent cardiology follow-up the next week.

Of note, the Division of Pediatric Cardiology at the Stony Brook University Medical Center has cared for two other cases of atrial fibrillation in healthy adolescents after excessive caffeine consumption in the past five years. These cases were not included in this series, as the patients were unable to be located to provide their consent.

Discussion
Soft drinks containing caffeine are the major source of caffeine intake in children and adolescents, and their caffeine consumption has risen exponentially in the last 30 years [8]. The fastest-growing trend is toward highly caffeinated beverages known as “energy drinks,” which differ from “sports drinks” such as Gatorade™. The general public is unlikely to be educated about the amount of caffeine in energy drinks and the possible ill effects that these drinks may cause in children and adolescents who consume them [13]. Energy drinks contain three to four times the caffeine as a typical soda and promise to boost performance and to enhance metabolism. Energy drinks like Full Throttle™, Red Bull™, SoBe No Fear™, and Monster™ typically contain a combination of caffeine, carbohydrates, B vitamins, amino acids, and other ingredients. One 8.2-ounce can of Red Bull™ contains
80 mg of caffeine (0.03%), twice as much as a 12-ounce soda; and one 16-ounce can of SoBe No Fear™ contains 141 mg of caffeine, four times as much as a soda. Mountain Dew™, which is marketed along with other sodas, contains more caffeine than other typical sodas at 55 mg per 12 ounces (Figure 5) [14,15]. Little regulation occurs with the production and marketing of energy drinks in the United States of America, with caffeine content between energy drinks ranging from 50 mg to 505 mg per bottle [16]. With the lack of regulation and strong marketing campaign toward young male athletes, energy drinks are becoming a serious threat to adolescents and are postulated to have caused grave consequences in an Australian athlete [17].
In our case series, both patients had essentially normal ECGs, ruling out endogenous cardiac causes for their arrhythmia, and both had admitted to consuming highly caffeinated drinks before atrial fibrillation developed. The patient in Case 1 admitted to consumption of a highly caffeinated beverage the day before his presentation, which was likely metabolized by the time he received medical care; one could argue that he may have had a prolonged caffeine half-life because of his chronic caffeine use. He did complain of the same palpitations after energy-drink consumption earlier that week; perhaps his high caffeine intake led to intermittent atrial fibrillation, which was exacerbated by his vigorous athletic activity on the day of presentation.

The patient in Case 2 had concurrent ingestion of alcohol with energy drinks and was also taking a baseline stimulant medication at the time of his presentation in atrial fibrillation. It is unclear how these other factors contributed to his arrhythmia; he certainly could have induced atrial fibrillation by alcohol intoxication, especially as his arrhythmia resolved with fluid resuscitation alone. As he did receive medical care within the expected half-life of caffeine, the timeline would fit for atrial fibrillation influenced by caffeine intoxication; the combination of alcohol and caffeine intoxication could have certainly led to his arrhythmia.

**Conclusion**

With the increasing popularity of energy drinks in the pediatric and adolescent population, physicians should be aware of the arrhythmogenic potential associated with their consumption. It is important for pediatricians to understand the lack of regulation in the caffeine content and other ingredients of these high-energy beverages and their complications, so that parents and children can be educated at well visits and sports physicals. We must inform the public on the potential health hazards related to excessive intake of caffeine-containing beverages by children and adolescents; the caffeine content of energy drinks should be better regulated and reported on food labels; and the purchase of energy drinks by the young consumer should be more closely monitored.

Given the possibility of cardiac arrhythmias and other untoward effects developing from caffeine use and abuse, further clinical trials reviewing the physiological effects and addictive potential for children and adolescents should be pursued, given the paucity of caffeine literature in this age group. Perhaps future studies could evaluate serum caffeine levels in pediatric patients who present with arrhythmias and concurrent caffeine consumption; this may be a useful measure to quantify into a risk model, should this correlation continue to be observed.

**Patient’s Perspective**

“One night...I felt dizzy and lightheaded. I realized that my heart was beating abnormally fast. My chest felt alien to me because my heart was beating with no set rhythm and was shifting around inside my rib cage. I thought I had somehow knocked my heart loose from its rightful place and now it was swinging about inside my body, beating erratically. I was admitted to the intensive care unit...I was amazed at the professional intensity with which the doctors and nurses performed their duties. The cardiologist told us it could be a fluke occurrence maybe caused by sugar or caffeine intake. I was put on a drug through my IV. The next morning... (my heart rate) had dropped to normal...I was instantly in a more affable mood...Some may say this was the spark that ignited a fire inside me to pursue a career in the medical field.”

**Consent**

Written informed consent has been obtained from the patient and parent of a patient who was a minor at the time for publication of this case report and accompanying images. Copies of the written consents are available for review by the Editor-in-Chief of this journal.

**Author details**

1 The Medical College of Wisconsin, Pediatric Hospital Medicine, Suite C560, CCC, P.O. Box 1997, Milwaukee, WI, 53201-1997, USA. 2 Beth Israel Medical Center, 1st Ave at 16th Street, New York, NY, 1003, USA. 3 Stony Brook University Department of Pediatric Cardiology, HSC T-11, 040, Stony Brook, NY, 11794-8111, USA.

**Authors’ contributions**

JRDR compiled, edited, and wrote the cases and also wrote the abstract, introduction, and discussion, and performed an updated review of the literature. AD initially summarized two cases, performed a literature review,
and contributed to the introduction and discussion. PM was heavily involved in caring for the patients, selecting them for the case report and editing the manuscript; MBH and TAB were both directly involved in the care of the patients, helped select them for this case report series, and remained supportive of the manuscript and its editing. All authors read and approved the final manuscript.

Competing interests

The authors declare that they have no competing interests.

Received: 17 November 2009 Accepted: 19 January 2011
Published: 19 January 2011

References

11. FDA Basics. “Why isn’t the amount of caffeine a product contains required on a food label?”. U.S. Food and Drug and Administration; [http://www.fda.gov].
15. The Caffeine Database. [http://www.energyfiend.com/the-caffeine-database].

Cite this article as: Di Rocco et al.: Atrial fibrillation in healthy adolescents after highly caffeinated beverage consumption: two case reports. Journal of Medical Case Reports 2011 5:18.

Submit your next manuscript to BioMed Central and take full advantage of:

• Convenient online submission
• Thorough peer review
• No space constraints or color figure charges
• Immediate publication on acceptance
• Inclusion in PubMed, CAS, Scopus and Google Scholar
• Research which is freely available for redistribution

Submit your manuscript at www.biomedcentral.com/submit
Visual recovery in a patient with total hyphema, neovascular glaucoma, long-standing retinal detachment and no light perception vision: a case report

Olusola Olawoye1, Christopher C Teng1,2*, Uri Shabtai1, Jeffrey M Liebmann1,3, Francis A L’Esperance4 and Robert Ritch1,2

Abstract
Introduction: We report the case of a patient with total hyphema, neovascular glaucoma, long-standing retinal detachment and no light perception vision, who regained counting fingers vision with complete regression of neovascularization following anterior chamber washout, intravitreal bevacizumab, pars plana vitrectomy, and silicone oil placement. This represents a rare case in which a patient with no light perception vision was able to regain functional vision.

Case presentation: A 63-year-old Caucasian man with a 55-year history of long-standing retinal detachment after trauma presented to our facility with pain and redness, a total hyphema, no light perception vision and an intraocular pressure of 60 mmHg (right eye). He had a history of diabetes mellitus and coronary artery disease. Following anterior chamber washout, he was found to have neovascular glaucoma, for which intravitreal bevacizumab was administered. After washout and intraocular pressure control, his visual acuity improved to light perception. He subsequently underwent vitrectomy, membrane peeling, endolaser and silicone oil placement to reattach his retina, and then a second retinal reattachment procedure. Following these procedures, he had visual recovery to counting fingers vision in his right eye at five metres, complete regression of neovascularization, and intraocular pressure of 10 to 12 mmHg on one antiglaucoma medication.

Conclusion: Functional vision can be regained despite long-standing retinal detachment.

Introduction
Long-standing retinal detachments (over one year) with poor visual acuity are typically associated with cystic degeneration of the macula and retina, loss of pigment from the underlying retinal pigment epithelium, proliferative vitreoretinopathy, and poor visual outcome after retinal reattachment surgery [1].

Chronic retinal detachment is a cause of rubeosis iridis and neovascularization of the anterior chamber angle with subsequent neovascular glaucoma (NVG). NVG represents one of the most severe forms of secondary glaucoma, caused by a number of ocular and systemic conditions. Retinal ischemia and hypoxia initiate the release of angiogenesis factors, with consequent development of new vessels.

We report the case of a patient with total hyphema, NVG, long-standing retinal detachment and no light perception (NLP) vision, who regained counting fingers (CF) vision with complete regression of the neovascularization following anterior chamber (AC) washout, intravitreal bevacizumab, and two retinal reattachment surgeries.

Case presentation
A 63-year-old Caucasian man presented to our facility with a four-week history of pain and redness in his right eye. He had had a traumatic retinal detachment of the right eye (55 years ago) after being struck in the eye...
with a stone. He subsequently developed a cataract in his right eye, for which he underwent cosmetic lensectomy at age 25. His best corrected visual acuity post lensectomy was light perception (LP), with a persistent retinal detachment. He was left aphakic in his right eye. At age 39, he had laser retinopexy in his left eye for lattice degeneration. He had a history of diabetes mellitus, quadruple cardiac bypass surgery, and defibrillator implantation.

On examination, his visual acuity was NLP (right eye) and 20/20 (left eye). External examination showed ptosis and exotropia in his right eye. Slit lamp examination revealed right eye nasal and temporal band keratopathy, mild corneal edema, total hyphema and no posterior view given the hyphema (Figure 1). He had an unremarkable examination of his left eye, with early nuclear sclerosis. Intraocular pressure (IOP) by Goldmann applanation tonometry was 60 mmHg (right eye) and 10 mmHg (left eye). Dilated fundus examination of his left eye revealed two areas of laser retinopexy surrounding lattice degeneration at 1:00 and 3:00 o’clock. Ultrasound of the right eye revealed low-lying retinal detachment with vitreous hemorrhage (Figure 2).

Immediate AC paracentesis to relieve pain and pressure reduced the IOP to 38 mmHg. Over the following two weeks, his IOP fluctuated between 36 to 50 mmHg (right eye), with no resolution of the hyphema or pain. AC washout was performed, and during surgery he was noted to have NVG with 360° rubeosis iridis, and vitreous hemorrhage with ghost cells. Over the next three weeks, he had two doses of 1.25 mg/0.05 ml intravitreal bevacizumab two weeks apart to treat his neovascularization.

Over eight weeks, his IOP gradually decreased to 15 mmHg (right eye) on four antiglaucoma medications, and his visual acuity improved from NLP to LP. His retina was noted to be normal in color and not necrotic or cystic. Given the good appearance of the retina and because he had recovered LP vision, we decided to see if vision would improve further by repairing the detachment. At two months after AC washout and three months after presentation, pars plana vitrectomy, membrane peel, retinotomy with aspiration of subretinal blood, endolaser retinopexy, inferior iridotomy, air/fluid exchange and retinal reattachment with silicone oil were performed.

Following surgery, his vision improved to counting fingers vision in the right eye at five metres, with IOP of 12 to 17 mmHg (right eye) on two antiglaucoma medications. There was complete regression of the rubeosis. His IOP remained stable over the next year on the same medication regimen. Fundus photography performed during a follow-up visit revealed a flat retina in both eyes, though there was residual fibrosis in the right eye (Figure 3). However, one year after retinal reattachment, he was noted to have an inferior tractional retinal detachment in the right eye with areas of subretinal fibrosis.

He subsequently had a second membrane peeling, removal of subretinal membranes, drainage of subretinal fluid, controlled retinectomy, and endolaser retinopexy. Postoperatively, his best corrected visual acuity remained
CF in the right eye at five metres and his IOP remained stable at 10 to 12 mmHg on timolol 0.25% once daily. The optic nerve and macula had retinal pigment epithelial hypertrophy and subretinal fibrosis (Figure 3). Our patient is currently being monitored with visual acuity of CF at five metres in the right eye and IOP 19 mmHg on timolol 0.25% once daily at last follow-up.

Discussion

With modern surgical techniques, a greater than 90% primary anatomic success rate can be expected following retinal detachment repair [1]. Despite this high level of anatomic success, visual results may remain compromised because of permanent functional damage due to macular detachment. The most important predictor of visual recovery after retinal detachment repair is preoperative visual acuity, which is directly related to the height of macular detachment [2]. A shorter duration of detachment and younger age are also important in visual recovery. Visual recovery following macula-off retinal detachment declines in an exponential fashion in relation to increasing duration of the detachment [3].

Chronic retinal detachments can also lead to complications such as proliferative vitreoretinopathy and rubeosis iridis. Iris neovascularization (INV) and NVG are highly correlated with retinal ischemia, which stimulates production of vascular endothelial growth factor (VEGF), a key molecule mediating neovascularization [4]. Intravitreal injection of VEGF has been shown to produce INV and NVG in non-human primates, and inhibition of endogenous VEGF is effective for suppressing the retinal ischemia induced INV [5].

Bevacizumab (Genentech, San Francisco, CA, USA) is a full-length humanized monoclonal antibody that binds all isoforms of VEGF. Recent reports using intravitreal bevacizumab injections have reported rapid and marked regression of neovascular vessels in INV and NVG [6]. Complete resolution of iris and angle neovascularization has also occurred after intravitreal bevacizumab.

Our patient presented with a total hyphema, NVG, elevated IOP, and long-standing traumatic retinal detachment with NLP vision. Trauma accounts for approximately one in 10 retinal detachments; the visual prognosis for eyes with NLP vision after trauma is dismal [7]. Of 52 eyes with a presenting vision of NLP, two improved to hand motion and two improved to LP vision following surgery [7]. Eyes with an initial acuity of hand motions or better correlated with significantly better visual outcome, but when the initial vision was LP or NLP, poor visual outcomes (57% to 100%) were more likely.

Brinton et al. [8] reported a series of 106 eyes with trauma involving the posterior segment; 55 eyes (52%) achieved final visual acuity of 20/100 or better following surgery. The eyes that underwent vitrectomy within 14 days of the injury had a better final visual outcome than those that underwent later vitrectomy. In 1982, Burton [3] reported that of patients with macula-off retinal detachments, 53% of patients who underwent surgery by nine days achieved visual acuity 20/20 to 20/50, with poor outcomes for long-standing detachments.

Despite our patient’s 55-year duration of long-standing retinal detachment, following AC hyphema washout, the retina had good color. Given this finding, the decision was made to repair the detachment and he was able to regain CF vision after two retinal surgeries. Suzuki and Hirose [9] reported a case of visual recovery from NLP in total retinal detachment of three months duration.
Their patient was able to regain CF vision after two surgeries and postulated that some retinal receptors were able to escape deterioration.

We believe that our patient was able to regain vision because of the low height of the long-standing retinal detachment. Previous studies have shown a positive relationship between the extent of the macular elevation and final visual acuity [3]. In experimental detachments in owl monkeys, Machemer [10] found that photoreceptor cell degeneration increased as the distance between the pigment epithelial layer and the photoreceptors increased. Our patient likely had areas of neurosensory retina intact, which allowed him to have some visual recovery after the retinal procedures.

Additionally, IOP control likely contributed to the improvement of vision. Wittstrom et al. [11] reported that a significant lowering of IOP seemed to improve the function of the central retina, as demonstrated by increased amplitudes and reduced implicit times assessed with multi-focal electrotetoretinography.

To the best of our knowledge, there has been no previous similar report of visual recovery in a patient with long-standing traumatic retinal detachment. We hope that with future advances, stem cells and retinal progenitor cells may be transplanted into diseased retinas to integrate and develop synaptic connections with host cells, and further improve visual function.

Conclusion
Functional visual recovery is possible despite long-standing retinal detachment with NLP vision.

Consent
Written informed consent was obtained from the patient for publication of this case report and any accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of this journal.

Acknowledgements
This study was supported by the Arthur and Phyllis Bargonetti fund of the New York Glaucoma Research Institute, New York, NY. OO was an International Council of Ophthalmology Fellow.

Author details
1Enrhorn Clinical Research Center, The New York Eye and Ear Infirmary, New York, NY, USA. 2Departments of Ophthalmology, New York Medical College, Valhalla, NY, USA. 3New York University Medical Center, New York, NY, USA. 4Columbia University College of Physicians and Surgeons, New York, NY, USA.

Authors’ contributions
OO and CCT were involved in acquiring data, conception, design and writing the manuscript; US and FAL were involved in patient care and manuscript preparation; RR and JML were involved in patient care, conception, design, drafting and revising the manuscript. All authors have read and approved the final manuscript.

Competing interests
The authors declare that they have no competing interests.

Received: 29 November 2009 Accepted: 17 June 2011 Published: 17 June 2011

References

Cite this article as: Olawoye et al.: Visual recovery in a patient with total hyphema, neovascular glaucoma, long-standing retinal detachment and no light perception vision: a case report. Journal of Medical Case Reports 2011, 5:221.

Submit your next manuscript to BioMed Central and take full advantage of:
• Convenient online submission
• Thorough peer review
• No space constraints or color figure charges
• Immediate publication on acceptance
• Inclusion in PubMed, CAS, Scopus and Google Scholar
• Research which is freely available for redistribution

Submit your manuscript at www.biomedcentral.com/submit
Abstract

Introduction: We report the case of a man who sustained a craniofacial injury after spontaneous lateral airbag deployment resulting in his face being struck by a car sunshade. This highlights the potential damage that can be caused by any object placed between a lateral airbag and a car occupant.

Case presentation: We report the case of a 33-year-old Caucasian man who was the driver in a frontal collision. He had opened the car sunshade and turned it 90° towards the left. As he was driving, he struck a bus, causing the driver’s lateral airbag to spontaneously deploy. The airbag pushed the sunshade against his face and injured him.

Conclusions: Car sunshades can cause significant craniofacial injury. We suggest that sunshade design must be improved to reduce the risk of potential injuries to car occupants. We recommend a new, safer sunshade design.

Introduction

Although there are reports of injuries caused by airbags [1-5], we are unaware of any literature describing injuries from car sunshades. We report a case of severe craniofacial injury after spontaneous lateral airbag deployment that caused the sunshade to strike the driver’s head. We also discuss the mechanism of sunshade induced injuries.

Case presentation

A 33-year-old Caucasian man was referred to Shahid-Rasi Hospital in Shahindezh (West Azerbaijan province, Northwestern Iran) after a motor vehicle crash. He was driving on a two-way mountain road in a south-north direction before sunset. Because of sunlight, he opened the car sunshade and turned it 90° towards the left. As he was driving at 60 km/hour on a sharp curve in the road, his car suddenly hit a bus coming from the opposite direction. The driver’s lateral and steering wheel airbags spontaneously deployed. The lateral airbag pushed the sunshade against his face so hard that the sunshade was completely deformed and caused injury to the left side of the face (Figure 1). He suffered abrasions on the left side of the face, retinal damage, and fractures of the skull base and nose. He also suffered superficial right forearm burns due to the rupture of the steering wheel airbag. His left hand was caught in the steering wheel, resulting in left distal radial and ulnar fractures. He underwent operative fixation of his nose and wrist fractures and was referred to an ophthalmologist for evaluation of his retinal injury.

Discussion

Airbag-associated injury occurs in 43% of airbag deployments [6]. Typically, airbag-related injuries are minor, but severe or fatal injuries are also reported [7]. Minor injuries such as abrasions, contusions and lacerations are usually detected on the face, neck, chest, and upper extremities [8,9]. Airbag deployment also releases high-temperature gases, including nitrogen and carbon dioxide, and produces sodium hydroxide, a very irritating alkaline material, which can cause superficial and even full thickness burns [10,11]. As demonstrated by this case, an opened and turned sunshade can also be a potentially dangerous object between a lateral airbag and a driver or passenger.

Conclusions

When the lateral airbag deploys, it pushes the sunshade onto the occupant’s face and head. Consequently, it seems that vehicles with side airbags should not have moveable sunshades.
sunshades that can be placed in the lateral position. We
suggest the design and use of sunshades that do not pro-
ject into the vehicle or the use of sunglasses.

Consent
Written informed consent was obtained from the patient
for publication of this case report and any accompany-
ing images. A copy of the written consent is available
for review by the Editor-in-Chief of this journal.

Author details
1 Sina Trauma and Surgery Research Center, Sina Hospital, Tehran University
of Medical Sciences, Tehran, Iran. 2 Donya-e-Khodro Weekly, Tehran, Iran.
3 Emergency Department of Hazrat-e-Rasool Hospital, Tehran University
of Medical Sciences, Tehran, Iran. 4 Research Centre for Neural Repair,
University of Tehran, Tehran, Iran.

Authors’ contributions
MS wrote the case report and performed the literature search. HK wrote
the Farsi version of the draft and organized the photographs. MC and VRM
designed the methodology, discussed, and edited the draft. All authors read
and approved the final manuscript.

Competing interests
The authors declare that they have no competing interests.

Received: 7 June 2010 Accepted: 10 May 2011 Published: 10 May 2011

References
1. Nguyen CS, Chase DM, Wing DA: Severe fetal skull fracture and death
subsequent to a motor vehicle crash with frontal airbag deployment.
due to airbag deployment and three-point restraint. J Trauma 2009, 67:
E98-E101.
3. Huber CD, Lee JB, Yang R, King AI: Head injuries in airbag-equipped
motor vehicles with special emphasis on AIS 1 and 2 facial and loss of
with side airbag deployments - a descriptive study. Accid Anal Prev 2007,
39:22-27.
5. Monihsoue SJ, Kelly MD: Airbag-related chest wall burn as a marker of
related to airbag deployment: a case report and review of literature.
9. Sato Y, Ohshyna T, Kondo T: Air bag injuries - a literature review in
consideration of demands in forensic autopsies. Forensic Sci Int 2002,
10. Heimbach D: Full-thickness burn to the hand from an automobile airbag.

Cite this article as: Sharif-Alhoseini et al.: Car sunshade-induced
craniofacial injury: a case report. Journal of Medical Case Reports 2011
5:175.

Submit your next manuscript to BioMed Central
and take full advantage of:

• Convenient online submission
• Thorough peer review
• No space constraints or color figure charges
• Immediate publication on acceptance
• Inclusion in PubMed, CAS, Scopus and Google Scholar
• Research which is freely available for redistribution

Submit your manuscript at
www.biomedcentral.com/submit
Using an Ishikawa diagram as a tool to assist memory and retrieval of relevant medical cases from the medical literature

Kam Cheong Wong

Abstract

Studying medical cases is an effective way to enhance clinical reasoning skills and reinforce clinical knowledge. An Ishikawa diagram, also known as a cause-and-effect diagram or fishbone diagram, is often used in quality management in manufacturing industries.

In this report, an Ishikawa diagram is used to demonstrate how to relate potential causes of a major presenting problem in a clinical setting. This tool can be used by teams in problem-based learning or in self-directed learning settings.

An Ishikawa diagram annotated with references to relevant medical cases and literature can be continually updated and can assist memory and retrieval of relevant medical cases and literature. It could also be used to cultivate a lifelong learning habit in medical professionals.

Introduction

Doctors are accustomed to learning from their more experienced peers as well as from their own experiences in treating their patients [1]. Because of this, it is important that they develop learning techniques that are proactive and encourage a lifelong learning orientation. Case reports can provide valuable sources of information for others to learn from. Studying medical cases is an effective way to enhance clinical reasoning skills and reinforce clinical knowledge [2]. A case report provides important and detailed information about a patient that is often lost in larger studies [3]. Reading case reports is also intellectually stimulating. When clinicians or medical students analyze a clinical problem, they usually start with potential common causes. For example, if a patient presents with secondary amenorrhea, a clinician will consider common causes such as pregnancy and use of contraceptive medications before exploring other less common but critical causes such as hyperprolactinemia, ovarian cancer and so on.

When clinicians are faced with a puzzling clinical problem, they may search journals that publish clinical cases for information about the condition [4]. There are various sources for medical cases such as the Journal of Medical Case Reports, BMJ Case Reports and the New England Journal of Medicine. However, because of the diversity of the case reports, it may be difficult to recall and organize the located material in a systematic manner in order to explain a clinical problem. Ishikawa diagrams are an efficient way of organizing case reports in a clinical setting.

Methods

The Ishikawa diagram was invented by Kaoru Ishikawa, who pioneered quality management techniques in Japan in the 1960s. The diagram is considered one of the seven basic tools of quality control [5]. It is also known as a fishbone diagram because of its shape. The ‘fish head’ represents the main problem. The potential causes of the problem, usually derived from brainstorming sessions or research, are indicated in the ‘fish bones’ of the diagram.

As an example for illustration, ‘secondary amenorrhea/oligomenorrhea’ has been chosen as the main presenting problem. ‘Secondary amenorrhea/oligomenorrhea’ is indicated in the head of the Ishikawa diagram (Figure 1). When searching for the potential causes of the main presenting problem, one can either work in a team with others...
Clinicians would conduct brainstorming sessions and search the relevant journals to find potential causes for secondary amenorrhea/oligomenorrhea, listing them on a whiteboard or flipchart. The list would then be reviewed to extract relevant causes in the context of the main presenting problem. These causes would then be organized in the 'fishbones' of an Ishikawa diagram (Figure 1). There is no limit to the number of 'fishbones' in the diagram. Each 'fishbone' can be subdivided into smaller 'bones' if necessary to show the relationship of all potential causes to the presenting problem. For example, 'chemotherapy and radiotherapy' are indicated in the branch of the 'fishbone' that shows the cause of ovarian failure, a potential cause for secondary amenorrhea/oligomenorrhea (Figure 1). The cited references for the relevant case reports and literatures are also indicated in the Ishikawa diagram so that readers can retrieve the case reports and relevant literatures easily.

The potential causes for secondary amenorrhea/oligomenorrhea have been identified and categorized in four groups related to 'women's reproductive systems', 'other systems in the body', 'psychosocial', and 'miscellaneous, for example drugs'. The causes include pregnancy [6], polycystic ovarian syndrome [6], amenorrhea after oral contraceptive/depot medroxyprogesterone treatment [7], eating disorder (e.g., anorexia nervosa) [7], premature ovarian failure [8], excessive physical exercise [9, 10], excessive stress [11], prolonged use of drugs, for example anti-psychotics, and after oral contraceptive/depot medroxyprogesterone treatment.

Conclusions

Rare but critical cases should be studied and included in an Ishikawa diagram to remind clinicians of relevant information during their clinical reasoning processes. For example, the Journal of Medical Case Reports has published the case of a 22-year-old lactating woman who presented with four months of amenorrhea associated with signs of virilization. The patient was diagnosed as having an androgen secreting steroid cell...
tumor of the ovary [15]. In addition, BMJ Case Reports has published a case demonstrating the relationship between hypothyroidism and secondary amenorrhea.

Important learning points are highlighted: serum thyroid stimulating hormone (TSH) should be measured in every adolescent with menstrual irregularities, multicystic ovaries as a presenting manifestation of juvenile hypothyroidism is a rare occurrence and represents advanced disease, and appropriate diagnosis and levothyroxine replacement therapy is effective and it can prevent inadvertent surgery [13].

Furthermore, the reader should appraise the published case to assess the credibility of the information and should look for updated information in the future. For example, if the readers are not fully convinced of the explanation for the pathophysiology of ‘specificity spill over’ phenomenon that may contribute to multicystic ovaries [13,17], he or she should search for more information about it and look out for future publications on this topic. Information gathered from other sources can be included in the diagram as well, such as the paper published in the British Journal of Obstetrics and Gynaecology, which has substantiated information about ovarian cancers and amenorrhea [8]. In this way, continually organizing and updating information on an Ishikawa diagram can cultivate lifelong learning habits in medical professionals.

Medical educators can also apply Ishikawa diagrams to facilitate problem-based learning when teaching medical students and junior doctors. Starting with a clinical vignette, facilitators can help medical students and junior doctors to identify the main presenting problem of a patient, conduct brainstorming sessions and search in the literature to find the potential causes, then categorize these causes in an Ishikawa diagram. The Ishikawa diagram can then be kept by individual learners for continual updating when they acquire new or relevant information. In short, an Ishikawa diagram can assist memory and the retrieval of relevant medical case reports and literatures.

Acknowledgements
I would like to thank the Journal of Medical Case Reports and BMJ Case Reports for providing access to the case reports, and the peer reviewers and Dr Myra Dunn (Education Officer at Beyond Medical Education, Australia) for their comments and suggestions. The author’s Academic Registrar position was funded by General Practice Education & Training (GPET), Canberra, Australia.

Author details
1University of Sydney, Sydney Medical School, NSW, Australia. 2University of Western Sydney, School of Medicine, NSW, Australia. 3Beyond Medical Education, NSW/WIC, Australia. 4George Street Medical Practice, Bathurst, NSW, Australia.

Competing interests
The author declares that he has no competing interests.

Received: 16 November 2010 Accepted: 29 March 2011

References

Cite this article as: Wong: Using an Ishikawa diagram as a tool to assist memory and retrieval of relevant medical cases from the medical literature. Journal of Medical Case Reports 2011 5:120.

Submit your next manuscript to BioMed Central and take full advantage of:
• Convenient online submission
• Thorough peer review
• No space constraints or color figure charges
• Immediate publication on acceptance
• Inclusion in PubMed, CAS, Scopus and Google Scholar
• Research which is freely available for redistribution

Submit your manuscript at www.biomedcentral.com/submit