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Atypical severe presentations of the oculocardiac reflex: Two case reports

Physicians who treat patients with facial trauma need to know how to prevent or manage the occurrence of the oculocardiac reflex because it can cause severe hypotensive or bradycardic/asystolic events and cardiac arrest.

ABSTRACT: The oculocardiac reflex is a rare but potential cause of severe hypotensive or bradycardic/asystolic events in patients suffering from facial trauma. Though the most common side effect of the oculocardiac reflex is bradycardia, clinicians should be concerned about a further decline to potentially fatal arrhythmias, asystole, and even cardiac arrest. This article presents two severe cases of the oculocardiac reflex in the setting of facial trauma. The first case involves cardiac arrest at the time of midface fracture reduction. The second case involves severe hypotension requiring vasopressor support secondary to severe intraorbital pressure.

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Vagal reflexes are well known to cause a change in blood pressure or heart rate. This is seen often in medicine and is termed a "vagal response." The most common example of vagal reflex stimulation used clinically involves slowing the heart rate with external carotid massage to correct supraventricular tachycardia. However, other maneuvers, such as rubbing the eyes or temples, can also cause a reduction in blood pressure or heart rate. The oculocardiac reflex is a reflex arc created by the trigeminal and vagus nerves.[1-3] It is defined as a slowing of the heart rate by more than 20% from baseline following globe manipulation or traction of the extraocular muscles.

As with any reflex, there is an afferent and efferent limb.[1] The trigeminal nerve serves as the sensory or afferent limb, while the vagus nerve serves as the motor or efferent limb of the reflex arc. Therefore, the reflex is initiated by activation of stretch receptors in the periorbital and ocular soft tissue, either through direct traction or increased pressure.[4] This leads to stimulation of the vagal motor response, which causes impulses to the sinoatrial node and triggers a slowing of the heart rate.

The most common signs of oculocardiac reflex are bradycardia and hypotension. In severe cases, arrhythmia, asystole, and cardiac arrest can occur.[5,6] This reflex is encountered primarily with pathology causing acutely entrapped muscles, such as orbital floor fractures.[4] There are reported cases of asystole with activation of this reflex due to direct surgical manipulation of the temporalis muscle,[7] but we were not able to find any cases of asystole secondary to indirect manipulation of the orbit or periorbital musculature. We also were not able to find many cases of severe hypotension due to orbital pressure alone.

Case data

Patient 1

A 59-year-old male suffered a fracture to the left zygoma due to a direct punch to the cheek. He was diagnosed with a depressed left zygoma fracture with comminution of the orbital floor. His medical history was positive only for high blood pressure (treated with ramipril and furosemide) and high cholesterol (treated with atorvastatin). His surgery was performed 16 days after the injury to allow time to come off ASA. No preoperative muscle entrapment was present.

Once the patient was successfully under general anesthesia with an oral endotracheal tube, his upper buccal sulcus and lower lid were infiltrated with 0.25% bupivacaine with 1:100 000 epinephrine. An intraoral incision was made, and dissection was taken down to the periosteum, which allowed a retractor to be placed under his zygomatic arch. As soon as the zygoma was reduced, the patient went into asystole.

The ECG tracing demonstrated persistent and unresolving asystole. It was recognized

immediately when the anesthetic machine monitor alarm sounded. The anesthesiologist instructed the surgical team to cease operating immediately and take pressure off the operative site. Further treatment included intravenous injection of 0.6 mg atropine and 15.0 mg ephedrine. As asystole persisted, CPR was initiated. A wide complex agonal rhythm was noted as CPR commenced. Approximately 50 seconds of CPR was performed, during which time narrow QRS complexes became evident on the ECG. When CPR was stopped, a sinus rhythm was present, along with a perfusing blood pressure. The approximate elapsed time from when the event was recognized and spontaneous rhythm returned was between 60 and 90 seconds. Epinephrine was not required because an acceptable blood pressure was detected shortly after resumption of spontaneous sinus rhythm. Due to these events and clinically acceptable reduction of the midface fracture, the incision was closed, and no plate fixation was performed. A forced duction test showed no orbital muscular entrapment.

The patient had an uneventful emergence and extubation. He was transferred to the postanesthetic care unit in stable condition, alert and cooperative. Following hospital discharge, he underwent outpatient cardiology review, including a stress test and 24-hour Holter monitor test. No cardiac disease was detected.

Patient 2

While driving, an 82-year-old male, who was otherwise healthy and living independently, was struck from the side in a motor vehicle collision, which resulted in multiple injuries, including complex midface and depressed skull fractures. The patient was admitted to the ICU and assessed immediately by a plastic surgeon for management of his periorbital fractures. The trauma and ICU team was concerned about ongoing hemorrhage from concomitant pelvic and long bone fractures. The patient was hypotensive and required vasopressor support of 7 mcg/kg/min norepinephrine bitartrate. He was bradycardic at 45 to 55 beats per minute. The Glasgow Coma Scale was 3T owing to sedation needs. Clinical evaluation demonstrated significant proptosis. A diagnosis of orbital compartment syndrome secondary to his displaced orbit and skull base fractures was tested. Intraocular pressures were measured to be 28 mm Hg in the involved globe (OD) and 15 mm in the contralateral globe (OS).

An emergent lateral canthotomy and cantholysis procedure was performed at the bedside under local anesthesia (1% lidocaine with 1:100 000 epinephrine) to reduce the patient's ocular pressures. Immediately after release and with serial evaluations, OD ocular pressures decreased to 9 to 11 mm Hg, which were equal to the contralateral side at that time. Shortly after orbital decompression, the patient's blood pressure and heart rate stabilized, and he no longer required vasopressor support. An ophthalmologist was consulted and did not identify any evidence of intraocular trauma or globe rupture. The patient underwent urgent operative reduction and internal fixation of his facial and depressed skull fractures within 24 hours of decompression.

Discussion

Various stimuli can cause activation of the oculocardiac reflex. Anesthesiologists, ophthalmologists, maxillofacial and plastic surgeons, trauma teams, intensivists, and emergency physicians who deal with patients who have trauma to the structures of the orbit or face need to be aware of this reflex, its potential consequences, and how to manage or prevent its occurrence.

Though the most common side effect of the oculocardiac reflex is bradycardia, clinicians should be concerned about a further decline to potentially fatal arrhythmias, asystole, and even cardiac arrest. Because the oculocardiac reflex is a vagal reflex, it should also be considered in patients with unexplained hypotension. The only definitive treatment is the immediate cessation of the triggering stimulus.

In our first case, it is possible that the oral approach to the zygomatic arch caused mild irritation to the insertion of the temporalis muscle, though Bhattacharjee's report[7] suggested that the muscle's involvement in the reflex arc suggested that it was direct pressure on the muscle that caused the reaction. It is more likely that the delayed nature of the treatment caused the asystole. By 16 days posttrauma, the bones would have started to knit together, and elevation of the zygoma would have caused direct movement of the orbital bones and a sudden change in orbital pressure. Due to the patient being on ASA, the risk of postoperative periorbital hemorrhage necessitated the surgical delay.

In our second case, the displacement of the bony structures into the orbit, along with posttraumatic swelling, caused compression of the globe and orbital musculature. Despite proptosis, the orbit is a closed compartment, and this quickly caused a compartment syndrome to develop. While an awake patient would complain of pain and visual changes, an intubated individual will have no signs or symptoms early in the disease progression. The significant effect of orbital compartment syndrome and the ensuing oculocardiac reflex on the blood pressure and heart rate was seen by the rapid removal of vaso-supportive medications after correction of the orbital pressure.

Summary

The oculocardiac reflex is a rare but potential cause of severe hypotensive or bradycardic/asystolic events in patients suffering from facial trauma and should be considered quickly in the clinical setting. ■

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