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Case Report

Myocardial ischemia due to oculocardiac reflex in patient with myocardial hypertrophy undergoing upper blepharoplasty

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Abstract

Oculocardiac reflex (OCR) is caused by pressure or traction of intraorbital structures and results in bradycardia. Patients with myocardial hypertrophy can suffer myocardial ischemia due to reduced coronary vascular reserve. In this case report we describe patient who developed myocardial ischemia as a result of OCR induced by traction of intraorbital soft tissue structures during upper blepharoplasty.

Introduction

The oculocardiac reflex (OCR) is a reflex afferent limb of which is trigeminal nerve and efferent limb is vagus nerve. OCR is usually caused by pressure on the oculus, traction of ocular muscles, or traction of the conjunctiva resulting in bradycardia and hemodynamic compromise in severe cases [1].

The incidence of OCR in eyelid surgery ranges between 20 and 25% [2, 3].

Patients with myocardial hypertrophy are prone to myocardial ischemia due to mismatch of myocardial mass and its vascularization or reduced coronary vascular reserve [4]. The incidence of myocardial ischemia in patients undergoing cosmetic surgery is unknown.

Treatment of OCR includes immediate cessation of the stimuli and atropine administration [1].

Case report

A 52-year-old female patient with no history of angina pectoris, normal physical exercise tolerance (>6 MET), normal ECG had upper blepharoplasty under local anesthesia. From her history was known that she had apical hypertrophic cardiomyopathy with no clinical manifestation.

Ten minutes after beginning of surgery during traction of medial hernia sac (Figure 1) patient felt bad and complained of severe retrosternal burning pain radiating to left hand. Standard monitoring (2-lead ECG, NIBP, SpO2) was set up and showed severe sinus bradycardia and hypotension (HR 32 bpm, BP 65/28 mmHg) and ST segment depression >2 mm in V5 lead. Surgery was stopped. Treatment included epinephrine to restore normal HR and BP (4 boluses 10 mcg each) and then phenylephrine to restore diastolic BP and hence coronary perfusion pressure (3 boluses 25 mcg each). Acetylsalicylic acid 300 mg was administered to chew by patient. Twelve-lead ECG showed ST segment depression in leads V3-V5. Twelve minutes later normal HR and BP were achieved (MAP > 80 mm Hg) and ischemic ECG changes and patient's complaints disappeared. Fentanyl 50 mcg was administered after stabilization. Additional local anesthesia of intraorbital soft tissue structures was advised and fulfilled by the surgeon. Then surgery was finished uneventfully. Troponin I (CITO Test) two hours after surgery was negative. Twelve-lead ECG two hours after surgery and next day were normal. The postoperative course was uneventful and the patient was discharged on the next day.

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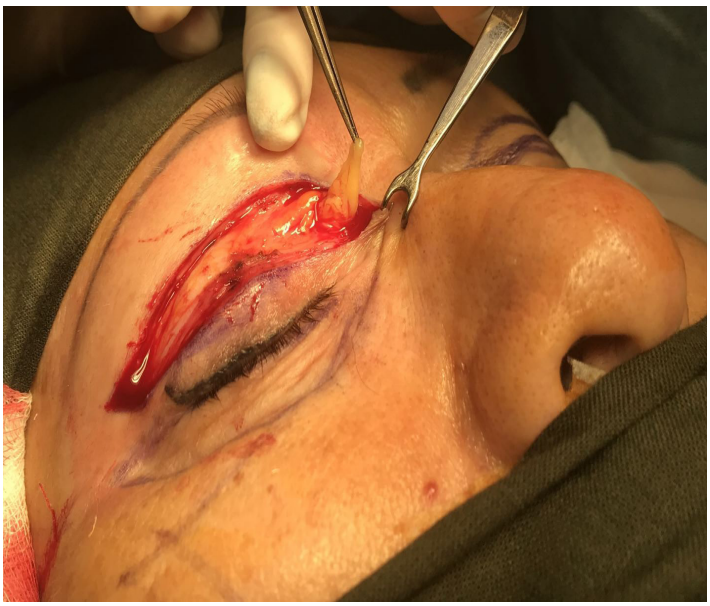

Figure 1: *Traction of medial hernia sac during upper blepharoplasty. Image of another patient with the same surgery.*

Discussion

Treatment of OCR involves immediate cessation of the stimuli and atropine administration. In cases of hemodynamic depression epinephrine administration is needed [1].

In cases of myocardial ischemia and hypotension coronary perfusion pressure must be restored to break the vicious circle when hypotension leads to coronary hypoperfusion, which leads to myocardial ischemia and depression, which leads to hypotension. Besides, myocardial depression leads to increased left ventricle diastolic pressure, which decreases subendocardial circulation and worsens myocardial ischemia. So, in this case atropine was not considered as it does not act as a pressor and promptly enough to restore coronary perfusion pressure and normal circulation. We used epinephrine as the initial agent as it acts as a pressor and chronotropic agent. When HR established > 60/min, boluses of phenylephrine were used to achieve MAP > 80 mm Hg and not to provoke tachycardia.

Optimal intraoperative BP for noncardiac surgical patients remains controversial. But MAP 65 mm Hg is considered as a threshold for intervention [5]. Taking into consideration the fact that patients with reduced coronary vascular reserve can require higher coronary perfusion pressures we chose MAP 80 mm Hg as a hemodynamic target.

ECG changes showed apical (but not another) myocardial ischemia, which corresponded with zone of myocardial hypertrophy. This suggests that perfusion of hypertrophic myocardium is highly dependent on coronary perfusion pressure.

OCR prophylaxis includes premedication with atropine and retrobulbar block [1]. In this case surgery under local anesthesia was planned and no preoperative anesthesiologist's consultation was obtained. Premedication by surgeon is impractical in our clinic. The practice and incidence of premedication by surgeon is not described in literature. Besides, premedication with atropine can lead to tachycardia and myocardial ischemia in cardiac patients [1], thus it should be used as a case-based approach. Retrobulbar block is not safe enough and is not used in this type of surgery [6].

Conclusion

Oculocardiac reflex can occur due to traction of intraorbital soft tissue structures. Patients with myocardial hypertrophy can develop myocardial ischemia due to oculocardiac reflex. Circulation in hypertrophic myocardium is highly dependent on coronary perfusion pressure.

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