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CLINICAL INFORMATION

Delayed trigeminocardiac reflex after maxillofacial surgery: case report

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Trigeminocardiac reflex;
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Abstract
Background: Trigeminocardiac reflex is a physiological phenomenon that may occur in head and neck surgery, and is usually benign. However, it may present with exaggerated responses with severe morbidity.
Case report: Male patient, 26 years old, candidate for surgical treatment of zygomatic-orbital complex fracture. The surgery with bilateral nasal packing placed at the end of the procedure was uneventful. After being admitted to the post-anesthesia care unity, the patient complained of shortness of breath and nausea. Pulse oximetry fell below 90% in ambient air, and 100% O_2 was then offered through a Hudson mask. He showed no improvement in oximetry and presented with worsening dyspnea, diffuse wheezing, reduced heart rate, and blood pressure. Atropine was given, which raised the heart rate, but without resolution of hypotension and bronchospasm. Our suspicion was of a trigeminal-cardiac reflex, and then the removal of the nasal packing was done with complete remission of the signs and symptoms.

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Discussion: Florian Kratschmer (1870) was the first to describe the influences of nasal mucosal reflexes on respiration and circulation, which became known as Kratschmer's reflex. It is a reflex arc whose afferent originates in the nerve endings of the trigeminal nerve. The clinical presentation of trigeminocardiac reflex is the occurrence of sudden bradycardia, hypotension, apnea, and gastric hypermotility.
Conclusion: Trigeminocardiac reflex may be a protective neurogenic, oxygen-conserving response with low morbidity, however, exacerbated in certain situations. The interaction between surgeon and anesthesiologist, together with a careful monitoring of blood pressure and heart rate are fundamental for diagnosis and treatment.
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PALAVRAS-CHAVE
Reflexo trigêmino-cardíaco;
Nervo trigêmeo;
Cirurgia ortognática

Reflexo trigêmino-cardíaco tardio após cirurgia buco-maxilo-facial: relato de caso

Resumo
Justificativa: O reflexo trigêmino-cardíaco é um fenômeno fisiológico passível de ocorrer em cirurgias da cabeça e pescoço, e normalmente é benigno. Contudo, pode apresentar respostas exageradas, com grave morbidade.
Relato de caso: Paciente masculino, 26 anos, candidato a tratamento cirúrgico de fratura do complexo zigomático-orbitário. Ato cirúrgico sem intercorrências com tamponamento nasal bilateral no fim. Após admitido na sala de recuperação pós-anestésica, queixou-se de ''falta de ar'' e náusea. A oximetria de pulso caiu abaixo 90% em ar ambiente e foi ofertado então O_2 a 100% sob máscara de Hudson. Não houve melhora da oximetria e apresentou pioria da dispneia, com sibilos difusos, redução da frequência cardíaca e da pressão arterial. Administrada atropina, que elevou a frequência cardíaca, mas sem resolução da hipotensão arterial e broncoespasmo. Aventamos a hipótese de reflexo trigêmino-cardíaco e então foi feita a remoção do tamponamento nasal com remissão completa dos sinais e sintomas.
Discussão: Florian Kratschmer (1870) foi o primeiro a descrever as influências de reflexos da mucosa nasal na respiração e na circulação, o que ficou conhecido como reflexo de Kratschmer. Trata-se de um arco reflexo cuja aferência é originária nas terminações nervosas do nervo trigêmeo. A apresentação clínica do reflexo trigêmino-cardíaco é a ocorrência de súbita bradicardia, hipotensão, apneia e hipermotilidade gástrica.
Conclusão: O reflexo trigêmino-cardíaco pode ser uma resposta neurogênica protetora, oxigênio-conservadora, de baixa morbidade, contudo exacerbada em determinadas situações. A interação entre cirurgião e anestesiologista, aliada à monitoração criteriosa da pressão arterial e do ritmo cardíaco, é fundamental para o diagnóstico e o tratamento.
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Introduction

The trigeminocardiac reflex (TCR) is often a benign physiological phenomenon that may occur in head and neck surgeries.[1] The occurrence of a stimulus at any point along the trigeminal nerve can trigger several undesired responses, such as bradycardia, bradypnea and hypotension, among others; exaggerated responses may result in severe morbidity.

The aim of this study is to report a TCR case and its outcome, as well as to review the literature addressing this phenomenon.

Case report

Male patient, 26 years old, 60 kg, who had an automobile accident and was candidate for zygomatic-orbital complex surgery and nasal fracture reduction. On pre-anesthetic evaluation he denied allergies, other comorbidities, use of medications or addictions, and anesthesia-surgical history without complications. His Previous neurological evaluation, physical examination, and additional tests were normal, then classified as ASA I.

In the operating room, adequate venous access and monitoring were performed. Anesthetic induction was performed with intravenous midazolam (4 mg), fentanyl (180 μg), dextroketamine (15 mg), propofol (120 mg), dexamethasone (9 mg), and cisatracurium (9 mg), and orotracheal intubation was uneventful. Oxygen, medicinal air, and sevoflurane were used for maintenance of anesthesia in a carbon dioxide absorber system and volume-controlled mechanical ventilation. At the end of surgery, intravenous ketoprofen (100 mg) and dipyrone (2 g) were also administered.

The surgical procedure was uneventful and lasted 180 min. Bilateral nasal packing was performed by the

surgical team with dressings lubricated in retinol ointment (5000 IU.g^{-1}), cholecalciferol (900 IU.g^{-1}), and zinc oxide (150 mg.g^{-1}). At the end of the procedure, neuromuscular block was reversed with intravenous neostigmine (2.4 mg) associated with atropine (1 mg), and the patient was extubated uneventfully.

At that time, the patient was responsive to verbal command, 95% pulse oximetry (PO) in ambient air, heart rate (HR) 88 beats per minute (BPM), and blood pressure (BP) of 135 × 90 mmHg, and without complaints. He was then taken to the Post-Anesthesia Care Unit (PACU). About 10 min after being admitted to PACU, the patient initially reported dyspnea and nausea. PO dropped from 96% to a maximum of 92% in ambient air, and 100% humidified O_2 was offered via Hudson mask; however, there was no improvement in dyspnea or PO. Suddenly, his HR decreased from 80–90 to 40 BPM in sinus rhythm, and blood pressure fell from 130 × 90 to 90 × 60 mmHg; the patient was very anxious. Intravenous bolus of atropine (1 mg) was administered, with HR increase to 50 BPM, but without hypotension resolution. Pulmonary auscultation revealed diffuse wheezing in both hemithorax.

We hypothesized that the condition could be a Kratschmer reflex and requested the immediate removal of his nasal packing. From then on, in up to approximately five minutes, there was progressive and complete remission of all signs and symptoms reported. There was no epistaxis, and the blood pressure, HR, and PO values returned to their initial values and pulmonary auscultation was normal.

The patient was discharged from the PACU to the ward with a Modified Aldrete Score of 10 and without complaints. His stay in the ward lasted 120 min, and hospital discharge occurred on the second postoperative day.

Discussion

In 1870, Florian Kratschmer was the first to describe the influence of nasal mucosal reflexes on respiration and circulation, which became known as Kratschmer reflex (KR). In 1969, Blanc confirmed that nasal mucosa stimulation causes bradypnea, bradycardia, and hypotension in dogs under anesthesia. However, since 1908, the oculocardiac reflex (OCR) is the best-known and well-established subtype of TCR.[1]

It is a reflex arc whose afferent originates in the nerve endings of the ophthalmic (V1), maxillary (V2), or mandibular (V3) division of the trigeminal nerve. The afferent stimulus reaches the Gasser ganglion and from there to the sensory nucleus of the V pair at the level of the 4th ventricle floor. The association occurs through the short internucial fibers in the reticular formation, which communicate with the efferent pathway in the motor nucleus of the vagus nerve.[1–3] Hence the trigeminocardiac or trigeminovagal reflex terminologies are more specific and anatomically more descriptive than the other subtypes or variants, such as KR, OCR, oculocutaneous reflex, immersion reflex (the most powerful known autonomic reflex), and maxilo-mandibular-cardiac reflex.[1,4]

In theory, a mechanical (the most reflexogenic), chemical, thermal, or electrical stimulus in the peripheral sensory territory (V1, V2 and V3), in the intracranial part of the trigeminal nerve or in the Gasser's ganglion (GG) can trigger TCR, which allows its classification in peripheral, central, or ganglionar.[4] Ophthalmic procedures or situations that increase intraocular pressure are well known and there are reports of TCR even in blepharoplasties.[1,2] Procedures involving orbital manipulations may present 90% incidence. Craniofacial surgeries, particularly those involving bone mobilization and skull base surgeries, such as those performed at the cerebellopontine angle, cavernous sinus, and pituitary fossa, are potentially reflexogenic. In transsphenoidal hypophysectomy, for example, TCR may occur in 10% of cases.[3] In this case, the nasal packing could have been the triggering mechanical stimulus.

The usual clinical presentation of TCR is the occurrence of sudden bradycardia, hypotension, apnea, and gastric hypermotility. Heart block, short QT interval, sinus arrest, asystole (even without bradycardia prodrome), and ventricular fibrillation have also been described.[1,2] Therefore, the manifestations may vary, depending on the point of reflex stimulation.[4] GG stimulation may trigger sympathetic responses, such as tachycardia and hypertension; this is because GG has sensory fibers (V1, V2 and V3), motor fibers (V3), and sympathetic fibers of the carotid plexus.[4] Other factors may explain the various TCR manifestations, such as polysynaptic connections (many still need clarification) and the role of other areas of the brainstem, other than those placed here;[4] moreover, the peripheral receptors of the V pair are not equally sensitive.[3] There are important anatomical variations in the pathways that form the various reflex arches of TCR. Our patient presented with classic clinical manifestations, in addition to bronchospasm, which should have favored the maintenance of low pulse oximetry even with O_2 supplementation. In the TCR variant described nearly 150 years ago, Kratschmer demonstrated that nasal irritants (cold air, cigarette smoke, ammonia, chloroform and others) also caused laryngospasm and bronchoconstriction.[5] The complaint of nausea in this case may, in theory, be associated to gastric hypermotility.

There are several factors that contribute to the occurrence of TCR, such as hypercapnia, hypoxemia, acidosis, superficial anesthesia, exacerbated basal vagal tone (children), use of opioids, and use of beta-blockers and calcium channel blockers.[1–4] In addition to potent nasal mechanical stimulus due to nasal packing, it is possible that our patient was also exposed to hypercapnia and hypoxemia, although mild and residual, so common in the immediate post-extubation period. Moreover, the nasal packing may have expanded by absorption of blood and secretions and amplified the afferent mechanical stimulus. These factors may have contributed to the onset of TCR. On the other hand, the atropine used for neuromuscular blockade reversal, due to its anticholinergic effect, could have had a protective effect on the triggering of TCR.

Improvement of these predisposing factors, smooth and careful manipulation in the area of trigeminal nerve innervation, and immediate discontinuation of this manipulation may resolve most cases.[4] Local anesthetic infiltration along the afferent pathways of TCR may also be useful.[1] It is well-reported that OCR is fatigable, and since TCR is more generic terminology for the same phenomenon,[4] it may be that subsequent manipulations may attenuate the clinical manifestations. If there is still no improvement, pharmacological strategies are necessary: first, anticholinergics and,

in case of unsatisfactory response, the use of adrenaline is required. In cases of hemodynamic collapse, which are extremely rare, resuscitation maneuvers must be initiated.[1] In our case, the stimulus withdrawal allowed the condition resolution, as the anticholinergic response resulted only in the partial improvement of the clinical picture, which is in agreement with reports of other authors.[5]

Interest in this phenomenon has recently surfaced and its presence seems to be linked to organic homeostasis.[4] Complex neurophysiological responses with endogenous protective effects, such as tolerance to ischemia, hibernation, and diving reflex, are described.[2,3] In response to hypoxic environment, the reflex stimulation of V1 results in bradycardia (parasympathetic response), vasoconstriction, and increased blood pressure (sympathetic response), the reserve of oxygen is preserved during apnea.[4] Thus, TCR has been seen as a protective reflex linked to oxygen conservation, that is, it reduces the consumption of oxygen in the presence of insults that may decrease the oxygen supply.[1]

Recently, several authors have considered that TCR may have late manifestations (hours, days, or even months) after the stimulus.[4] It is possible that our case fit this situation, as the patient was already with a nasal packing at the time of extubation. When triggered, it seems to be possibly associated with late hearing loss, tinnitus,[2,3] and sleep disorders.[4] Researches try to elucidate the neuroprotective role that TCR may eventually play as a preconditioning strategy for ischemia.[2]

Conclusion

It is possible that TCR may be a neurogenic protective and oxygen-conservative response with low morbidity, yet exacerbated in certain situations. The ''vagal'' or ''cardiac'' terminology seems incomplete and inadequate, as the manifestations may also be sympathetic. Finally, the interaction between surgeon and anesthesiologist, together with careful monitoring of blood pressure and heart rate, is fundamental for diagnosis and treatment.

Conflicts of interest

The authors declare no conflicts of interest.

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