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Unexpected difficult airway management due to the use of ACE inhibitors: case report

Vía aérea difícil no prevista secundaria a consumo de inhibidores de la enzima convertidora de angiotensina. Reporte de caso

Keywords: Airway Management, Angioedema, Captopril, Anaphylaxis, Case Reports

Palabras clave: Manejo de la Vía Aérea, Angioedema, Captopril, Anafilaxia, Informes de Caso

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Abstract

Angioedema induced by the use of angiotensin converting enzyme (ACE) inhibitors is an uncommon but life-threatening complication, especially when the airway is affected, creating unexpected difficult airway management.

A prompt differential diagnosis with anaphylactic shock is critical, given that adrenaline treatment does not improve angioedema.

We report a case of angioedema induced by ACE inhibitor following in-hospital administration of captopril, with almost impossible intubation, and secondary aspiration during airway management. Angioedema was erroneously treated, because it was mistakenly considered to be an anaphylactic reaction, and it could have ended in death.

Resumen

El angioedema es una complicación poco frecuente relacionada con el uso de inhibidores de la enzima convertidora de angiotensina, pero potencialmente mortal, especialmente en el caso de afectar a la vía aérea, generando vías aéreas difíciles no previstas.

Es de vital importancia realizar un rápido diagnóstico diferencial del cuadro con el shock anafiláctico, dado que el tratamiento con adrenalina, no mejora el angioedema.

Presentamos un caso de angioedema tras administración intrahospitalaria de Captopril a un paciente sano, sin vía aérea difícil prevista, generando una intubación casi imposible y broncoaspiración secundaria durante el manejo de la vía aérea. El cuadro clínico se desencadenó por la confusión del angioedema, con una reacción anafiláctica, realizándose un tratamiento inapropiado. Las consecuencias del mismo pudieron ser mortales.

Case description

This is the case of a 45-year-old male patient with no known allergies, a history of untreated hypertension and depression, and no relevant family history.

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The patient was admitted to the hospital for 3 days due to renal failure and electrolyte and fluid imbalance (metabolic alkalosis, hypokalemia, and symptomatic hypomagnesemia) that were resolved. The condition developed after 4 days of refusal to eat and drink during the summer in an attempt at inflicting self-harm as a result of a divorce process. At the time of the usual verification of vital signs by the nursing staff on the day of discharge, the patient was found to have high blood pressure (BP) of 188/100 and was given captopril 25 mg, ordered by the attending physician.

Following the administration of the drug, the patient began to report discomfort and showed evidence of lingual pyrexia, oropharingeal itching, and swelling of the labial mucosa. An allergic reaction was diagnosed based on the clinical findings and the patient was given dexclorpheniramine 5-mg intravenous (IV) plus hydrocortisone 100-mg IV. Six hours into the onset of the condition, the patient reported difficulty swallowing and breathlessness, with 91% oxygen saturation, triggering activation of resuscitation on the basis of a clinical diagnosis of anaphylactic shock.

Upon arrival to the resuscitation service, the patient was found to have significant facial and cervical edema, a sensation of dyspnea, 92% oxygen saturation (despite 50% oxygen supplementation through face mask), sinus tachycardia of 130 bpm, and BP of 145/86. No pathological signs were found on auscultation.

The patient was treated with intravenous corticosteroid, inhaled bronchodilator, and reservoir oxygen therapy which improved the dyspnea sensation and oxygen saturation to 95%. Given the vital signs (tachycardia and hypertension), adrenaline was not administered and it was decided to monitor and observe the patient.

After 4 hours, the patient developed severe respiratory distress with tachypnea and 83% oxygen saturation despite oxygen therapy, increased cervical diameter and lip swelling. The decision was made to proceed to intubation and rapid sequence induction.

Following induction, the patient desaturated down to 50% O_2 saturation on pulse oximetry. On direct laryngoscopy, the patient was found to be Cormack III/IV, making it impossible to intubate using the traditional laryngoscope blade or the Frova introducer.

It was then considered to follow the Difficult Airway Management Algorithm of the American Society of Anaesthesiology 2013.[1]

Face mask ventilation was found to be very difficult, with an inspiratory volume lower than required by weight. Despite low-volume ventilation, the patient presented vomiting, probably due to the pathophysiology of the angioedema itself, and also perhaps because the patient had been fasting for more than 8 hours and ventilation had been unsuccessful. Vomiting worsened saturation, to the point that it was not sensed by the pulse oximeter.

Based on the description contained in the guidelines and given the fact that the patient could not be intubated or ventilated, a supraglottic device was considered but then discarded due to the presence of vomiting, leading to the consideration of the next step in the guidelines, consisting of an emergency cricothyrotomy. However, given neck anatomy, the resucitation staff was skeptical about the outcome.

The team decided to change strategy and intubate with a fiberoptic bronchoscope, considering that no other option was feasible given the nature of the case, the unavailability of otolaryngology specialists in the hospital, and the fact that the attending physician was experienced in the use of the device. Finally, intubation with the fiberoptic bronchoscope was successful despite the fact that airway visualization was not good, after 10 minutes of saturations below 90%.

The clinical team had not anticipated a difficult intubation before the emergency because the patient was stable on arrival to the resuscitation service, and no consideration was given to a diagnosis of angioedema induced by the use of angiotensin-converting enzyme (ACE) inhibitors.

The patient remained intubated for 30 hours until the medical staff was certain that lip, lingual, and cervical edema had resolved. The patient was diagnosed with ACE inhibitor-induced angioedema and received treatment with 2 bags of fresh frozen plasma, leading to full remission of the facial and cervical edema. The patient received prophylaxis with amoxicillin/clavulanic acid for aspiration. Blood gases were within normal ranges from the moment the patient was intubated, and the only remarkable laboratory findings were leukocytosis of 17,000 and ACE activity of 11. The patient was extubated without further incidents.

On discharge 6 days later, there were no clinical signs of aspiration pneumonia, although radiological imaging showed right lower pulmonary lobe condensation.

The patient reported that following captopril administration, he noticed an itching sensation in the oropharynx and difficulty breathing, but remembered nothing else after anesthetic induction for intubation. Consent from the medical institution was obtained for the publication of this case for teaching purposes.

Discussion

Figures of 2001 estimated that 40 million people were treated with ACE inhibitors in the world.[2] Angioedema was estimated to occur in 0.1% to 2.2% of all patients treated with ACE inhibitors, although it appears that this figure is an underestimation, considering that many cases of angioedema are mistaken for allergic reactions to medications, as was the case in the patient presented in this paper.

It is crucial to identify the cases and to know how to respond to angioedema associated with the use ACE inhibitors because progression of this condition may be fatal as time goes by. It is also of the greatest importance not to think that drug effects on the airway are usually allergic reactions because, in the case of an ACE inhibitor, the condition is more often related to angioedema.[3]

Although the pathophysiology of angioedema is not totally clear, ACE inhibitors are known to favor bradykinin accumulation by inhibiting its breakdown. This kinin is a powerful vasodilator, capable of inducing edema. Angioedema has been attributed to this effect on bradykinin metabolism. In the case of our patient, in whom a reduction by almost 1/2 in the activity of the ACE was documented, bradykinin accumulation could have been even greater, leading to more severe clinical manifestations of vasodilation.[4,5]

The most common symptoms found in ACE inhibitor-induced angioedema are swelling of the lips and tongue, and dyspnea. ACE inhibitor-induced angioedema usually affects head and neck, making airway management difficult. For this reason, a difficult airway must always be kept in mind in patients with respiratory distress following the administration of an ACE inhibitor. Swelling usually resolves within 24 to 48 hours.

Angioedema is frequently associated with abdominal symptoms such as pain, nausea, and vomiting. For this reason, these patients must be considered high-risk when it comes to orotracheal intubation because airway swelling is compounded by a high probability of vomiting during intubation.[6]

Clinical guidelines on the management of angioedema have been recently published in the United States,[7] addressed to emergency physicians, to avoid fatal cases, usually due to difficult airway management. Those guidelines underscore the need to give priority to airway management in patients with facial swelling, especially the lips, always using the algorithm for a difficult airway and avoiding supraglottic devices. This point is especially important because failure to recognize a difficult airway due to angioedema, and its differences with an unexpected airway in terms of vomiting results in a tendency to use guidelines which are not practical in this emergent situation, usually based on the use of a supraglottic device, which is actually contraindicated in these cases.

Conventional treatments such as H1 and H2 antagonists, oral corticosteroids, and adrenaline have been shown not to be effective in angioedema progression,[8] and should be avoided. In recent years, the use of fresh frozen plasma for the treatment of this condition has been growing, although its usefulness is not clear either.[9] It is worth noting that when a patient shows facial signs of edema, the airway needs to be secured because there is a high probability of progression, resulting in an unexpected difficult airway as in the case we describe.

There is ongoing research using bradykinin receptor antagonist icatibant, showing effectiveness of this agent in cases of angioedema.[10] However, large-scale clinical trials are required.

Conclusion

Prompt diagnosis of ACE inhibitor-induced angioedema is of vital importance to avoid fatal cases due to difficult airway management. It is critical to remember that angioedema is not a form of allergic reaction and, consequently, is not treated the same way.

The use of ACE inhibitors needs to be included as part of the signs of alert of urgent difficult airway considering the wide use of these medications and their potential effect on the airway. There is also a need to underscore the importance of the presence of vomiting when it comes to documenting a difficult airway and following the current intubation guidelines.

Ethical responsibilities

Human and animal protection. The authors declare that no human or animal experiments were performed for this research.

Data confidentiality. The authors declare that they followed the protocols of their institutions regarding patient data disclosure.

Privacy and informed consent. No patient data appear in this article.

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References

1. Practice Guidelines for Management of the Difficult AirwayAn updated report by the American Society of Anesthesiologists. Task Force on Management of the Difficult Airway. American Society of Anesthesiologists, Inc. Lippincott Williams & Wilkins. Anesthesiology 2013;118:251–270.
2. Sondhi D, Lippmann M, Murali G. Airway compromise due to angiotensin-converting enzyme inhibitor-induced angioedema: Clinical experience at a large community teaching hospital. Chest 2004;126:400–404.
3. Toh S, Reichman ME, Houstoun M, et al. Comparative risk for angioedema associated with the use of drugs that target the renin–angiotensin–aldosterone system. Arch Intern Med 2012;172:1582–1589.
4. Holm JP, Ovesen T. Increasing rate of angiotensin-converting enzyme inhibitor-related upper airway angio-oedema. Dan Med J 2012;59:A4449.
5. Francisco Tocornal J, Tatiana Espinoza B, Jan Karisruher S, et al. Angioedema by users of Inhibitors of the Angiotensin Converting

Enzyme in Otorhynolaryngology. Rev Otorrinolaringol Cir Cabeza Cuello 2006;66:179–184.
6. Chiu AG, Newkirk KA, Davidson BJ, et al. Angiotensin-converting enzyme inhibitor-induced angioedema: a multicenter review and an algorithm for airway management. Ann Otol Rhinol Laryngol 2001;110:834–840.
7. Bernstein JA, Cremonesi P, Hoffmann TK, et al. Angioedema in the emergency department: a practical guide to differential diagnosis and management. Int J Emerg Med 2017;10:15.
8. Al-Khudari S, Loochtan MJ, Peterson E, et al. Management of angiotensin-converting enzyme inhibitor-induced angioedema. Laryngoscope 2011;121:2327–2334.
9. Hassen GW, Kalantari H, Parraga M, et al. Fresh frozen plasma for progressive and refractory angiotensin-converting enzyme inhibitor-induced angioedema. J Emerg Med 2013;44:764–772.
10. Illing EJ, Kelly S, Hobson JC, et al. Icatibant and ACE inhibitor angioedema. BMJ Case Rep 2012;30: doi: 10.1136/bcr-2012-006646.