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Cardiorespiratory arrest due to grade IV anaphylactic shock with succinylcholine, report of a case and review of the literature

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ABSTRACT

Anaphylactic shock caused by anesthetic agents is a serious complication that can lead to the death of the patient despite well-conducted resuscitation. Succinylcholine is the primary source of anaphylactic shock in anesthesia. Used for over 60 years as the product of choice in emergency situations and during difficult intubation; its use is wide in all countries of the globe. We report the observation of a 77-year-old patient who underwent ophthalmological surgery under general anesthesia. After induction with propofol and placement of a laryngeal mask, a decision to orotracheal intubation was made given the ventilation difficulties. An anesthetic protocol combining a hypnotic to deepen the narcosis with propofol and curarization using suxamethonium was chosen. A few minutes after intubation, ventilator capnia is undetectable and the pulse is unobtainable while an ECG tracing persists. CPR is performed by external cardiac massage, bolus injection of adrenaline (4 mg in cumulative doses) associated with manual ventilation with FIO2 at 100%, cessation of any current product, filling with crystalloids and 250ml of bicarbonate 4, 2%. An effective pulse and blood pressure are found after no-flow for one (1) minute and low-flow for 10 minutes. Samples were taken for additional explorations. With the success of cardiopulmonary resuscitation and non-urgent surgery, it was decided not to begin the intervention and to transfer the patient to intensive care for continued treatment in the face of suspicion of serious anaphylactic shock. After twenty-four (24) hours of monitoring, he was extubated and his release from intensive care was authorized after the seventy-second hours. The allergological consultation carried out after the event concluded that there was allergic hypersensitivity to suxamethonium, a molecule contraindicated for life in this patient.

Key words: anaphylactic shock, curares, cardiac arrest, suxamethonium, Abbeville, France

Introduction

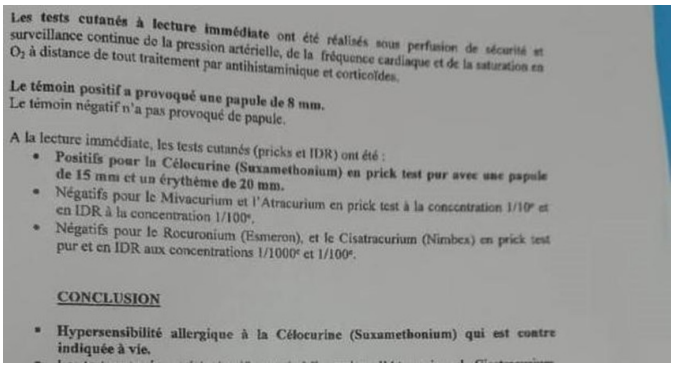
Anaphylactic shock caused by anesthetics and particularly muscle relaxants very often occurs in unpredictable situations, sometimes with a severe prognosis. This allergic reaction can occur without any other previous history . It is a serious complication that can lead to the death of the patient despite well-conducted resuscitation. **[1]**

The establishment of the observatory of the Group for the Study of Allergic Reactions per Anesthetics (GERAP) made it possible to clearly identify muscle blockers as the cause of half of allergic reactions in the intraoperative period **[1].** Suxamethonium (or succinylcholine), the only depolarizing curare, long used as the curare of choice in emergency situations, particularly in difficult intubations, is the primary source of anaphylactic shock in anesthesia **[2].** This very uncomfortable and formidable situation constitutes a challenge for the resuscitator anesthetist when it occurs.

Given the diagnostic, therapeutic and evolutionary interest of this rare manifestation in current practice, we report here a clinical observation of grade IV anaphylactic shock with intraoperative celocurin at the Abbeville hospital center (France).

Clinical case: We report the clinical file of a 77-year-old patient, with a pacemaker implanted in 2018, device well in place and functioning well according to the last consultation. His history is marked by: Severe obesity with a BMI of 36, well-controlled arterial hypertension, complete arrhythmia due to atrial fibrillation, bilateral venous insufficiency, smoking at 10 PA stopped 22 years ago. This patient was on: Eliquis 5mg; Amiodarone 200mg; Bisoprolol ; Hydrochlorothiazide 12.5mg. He was scheduled for eye surgery (cataract) under indicated general anesthesia in addition to topical anesthesia due to claustrophobia. The pre-anesthetic consultation allowed him to be classified as ASA III because apart from his history, the clinical and paraclinical assessment at the time of the consultation was unremarkable. On the day of the operation after reception, installation and monitoring with the boc, a propofol induction was carried out to allow the insertion of a laryngeal mask. Faced with the difficulties of ventilation under a laryngeal mask, it was decided to perform orotracheal intubation, thus requiring deepening of the narcosis with propofol and curarization using suxamethonium. A few minutes after intubation, ventilator capnia is undetectable and the pulse is unobtainable while an ECG tracing persists. Clinically, urticaria and skin redness predominating on the face, neck and thorax were observed. Faced with this picture, cardiopulmonary resuscitation was immediately initiated by external cardiac massage, bolus injection of adrenaline (4 mg in cumulative doses) associated with manual ventilation with FIO2 at 100%. Any new reinjection of any product and/or in progress has been suspended. Filling with crystalloids and 250ml of 4.2% bicarbonate was carried out. An effective pulse and blood pressure are found after no-flow for 1 minute and low-flow for 10 minutes. Samples were taken for additional explorations.

With successful cardiopulmonary resuscitation and elective surgery ; it was decided to postpone the intervention and transfer the patient to intensive care for continued treatment due to suspicion of serious anaphylactic shock. After twenty-four (24) hours of monitoring, he was extubated and his release from intensive care was authorized after the seventy-second hours. The allergological consultation carried out after the event concluded that there was allergic hypersensitivity to suxamethonium, a molecule contraindicated for life in this patient.



Les tests cutanés à lecture immédiate ont été réalisés sous perfusion de sécurité et surveillance continue de la pression artérielle, de la fréquence cardiaque et de la saturation en O_2 à distance de tout traitement par antihistaminique et corticoïdes.

Le témoin positif a provoqué une papule de 8 mm.
Le témoin négatif n'a pas provoqué de papule.

A la lecture immédiate, les tests cutanés (pricks et IDR) ont été :

- Positifs pour la Célocurine (Suxamethonium) en prick test pur avec une papule de 15 mm et un érythème de 20 mm.
- Négatifs pour le Mivacurium et l'Atracurium en prick test à la concentration 1/10e et en IDR à la concentration 1/100e.
- Négatifs pour le Rocuronium (Esmeron), et le Cisatracurium (Nimbex) en prick test pur et en IDR aux concentrations 1/1000e et 1/100e.

CONCLUSION

- Hypersensibilité allergique à la Célocurine (Suxamethonium) qui est contre indiquée à vie.

Image 1: report of the allergological consultation

Discussion: Our clinical case highlights the sudden, unpredictable and serious nature of anaphylactic shock to succinylcholine occurring during anesthetic induction. Despite the precautions surrounding surgical interventions, immediate peranesthetic allergic hypersensitivity reactions represent approximately 780 cases per year in France, or an incidence of 100.6 cases per million interventions, or one in 10,000 **[3].** The severity of these reactions varies, but despite rapid treatment, the mortality rate is **3 to 8%. [4,5].** The seventh multicenter survey concerning the epidemiology of peranesthetic anaphylactic and anaphylactoid reactions in France (January 2001 to December 2002) confirmed that the drugs most frequently responsible for anaphylaxis are curares (55%), with among them a preponderant place of succinylcholine (37.6%) and rocuronium **[6].** Our case presented a clinical history and symptoms compatible with anaphylactic shock to succinylcholine and it was anaphylactic shock grade IV from the outset with cardiovascular manifestations such as cardiac arrest. Cardiovascular manifestations make prognostic severity. As peranesthetic anaphylactic reactions are often sudden and unexpected, opportunities to collect hemodynamic data are rare and few data are available in the literature **[7]** . The only cases reported are isolated and correspond to incidental monitoring, initially implemented for the intervention as in the article by **Beaupré et al.** , the latter having visualized on transesophageal ultrasound a drop in preload following anaphylactic shock with antibiotics. **[8],** The physiopathological mechanisms also come from animal experiments, particularly dogs **[9] . Mitsuhata et al.** measured the variations in systolic and diastolic pressures of the left ventricle of seven (7) dogs during anaphylactic shock. They note that the function of the left ventricle is preserved and that the reduction in venous return plays a key role in reducing blood pressure. In 4 cases, the initial presentation was cardiac arrest with 2 cases of asystole and 2 cases of ventricular fibrillation. In cases of anaphylaxis with immediate cardiac arrest, myocardial dysfunction has been suggested, probably secondary to myocardial infiltration by mast cells **[10].** Our data also match those published by **C. Dauthier et al** . In a series of 18 patients who presented with peranesthetic anaphylactic shock with celocurin, all cases presented compatible clinical symptoms and were all severe anaphylactic shock of grade III and IV. **[7],** Skin signs were present in our case and their prevalence reported in the literature was 15 to 70% **[3].** Bronchospasm was part of the clinical presentation in our clinical case and was also 61% of cases in the study by **C. Dauthier et al.** cases, while its prevalence reported in the literature is 18 to 48.40% **[3, 7].** It should also be noted that the absence of these suggestive clinical signs can contribute to diagnostic and therapeutic delay. An isolated collapse was the clinical presentation in 22% of cases; the diagnosis is particularly difficult in this situation of collapse at induction. The clinical presentation can therefore be relatively nonspecific and the diagnosis of anaphylaxis thus delayed. **[3],** This calls for increasing promotion of courses, internships and simulator training, throughout the career of a resuscitator anesthesiologist. Our observation complements the abundant literature concerning the complications

linked to the administration of succinylcholine, the use of which must be limited to strict indications.

Conclusion: An allergic reaction to a curare remains a rare but serious event. Whose prognosis is conditioned by the speed of diagnosis and recognition of the severity grade. Stabilization of vital functions must be followed by prolonged continuous monitoring and medium-term follow-up. Succinylcholine is the primary source of anaphylactic shock in anesthesia. Most often the patient who experiences anaphylactic shock with curare receives it for the first time. This suggests cross-sensitization with other drugs. Currently, there is therefore no recognized allergy risk factor and the indications for an allergy assessment are the exploration of a reaction occurring during anesthesia and the search for cross-allergy between curares.

REFERENCES:

1. Malinovsky JM, C tacquard. AM Chiriac et al. News on curare anaphylaxis. Rev Fr Allergology. 2017. 547-550.
2. Gronert GA . Cardiac arrest after succinylcholine: mortality greater with rhabdomyolysis than receptor upregulation. Anesthesiology. March 2001;94(3):523 -529.
3. Mertes PM, Alla F, Tréchot P, Auroy Y, Jougla E, Study group of peranesthetic anaphylactoid reactions. Anaphylaxis during anesthesia in France: an 8-year national survey. J Allergy Clin Immunol 2011; 128:366–73.
4. M. Fourel a, N. Diot b, R. Dubost b et al . Demonstration of sensitization to curares during the diagnosis of an allergy to pholcodine. Revue française d'allergologie 52 (2012) 97–100.
5. Mertes PM, Tajima K, Regnier-Kimmoun MA, Lambert M, Iohom G et al. Perioperative anaphylaxis. Med Clin North Am 2010; 94:761–89.
6. Dong SW, Mertes PM, Petitpain N, Hasdenteufel F et al. Hypersensitivity reactions during anesthesia. Results from the ninth French survey (2005–2007). Minerva Anestesiol 2012; 78:868-78.
7. C. Dauthier, M. Le Dorze, C. Le Coq et al : Choc anaphylactique à la succinylcholine : revue de la littérature, analyse d'une augmentation locale d'incidence, à propos de 18 cas. AFAR.2014.98-99.
8. Beaupre PN, Roizen MF, Cahalan MK, et al. Hemodynamic and twodimensional transesophageal echocardiographic analysis of an anaphylactic reaction in a human. Anesthesiology. 1984;60(5):482-484.
9. Mitsuhata H, Hasome N, Saitoh J et al. Evaluation of left ventricular diastolic function in IgE-mediated anaphylaxis in dogs. Acta Anaesthesiol Scand. août 1995;39(6):791-796.
10. Mertes PM, Pinaud M. [What are the physiopathological mechanisms? How can severe anaphylactoid reactions be explained]. Ann Fr Anesth Reanim. mai 2002;21 Suppl 1:55s-72s.
11. Smith HM, Jacob AK, Segura LG, Dilger JA, Torsher LC. Simulation education in anesthesia training: a case report of successful resuscitation of bupivacaine-induced cardiac arrest linked to recent simulation training. Anesth Analg. mai 2008;106(5):1581-1584, table of contents.
12. Laroche D, Debaene B. et al. How to relate the observed event to anaphylaxis? Practice of diagnostic investigations. Ann Fr Anesth Reanim. mars 2011;30(3):280-293.