ratory pressure may have superimposed sufficient work of breathing in this patient with decreased pulmonary reserve to cause symptoms. At successful extubation, this patient was on CPAP, which may have stented his airway sufficiently to relieve partial collapse. Residual neuromuscular blockade, although not apparent in this patient, might still have been detected had more sophisticated evaluation been available, *e.g.,* double-burst stimulation. Finally, the patient's ventricular function was known to be decreased and probably was unable to adapt to changing preload and afterload stressors.

Although the diagnosis of NPE in this patient was primarily by exclusion, its management was straightforward and consisted of ventilation and diuresis. However, the patient's ability to maintain adequate ventilation after extubation was uncertain because the proximal extent of tracheal collapse was unknown. Consequently, extubation was performed in the operating room with the availability of a rigid bronchoscope and tracheostomy equipment.

In summary, this patient with chronic obstructive pulmonary disease (COPD) and saber-sheath trachea suffered pulmonary edema associated with emergence from general endotracheal anesthesia. Saber-sheath trachea in this patient, as is typical, resulted in significant reduction in tracheal caliber caused by reduction in strength of the tracheal cartilage. Pulmonary edema in this patient was most likely low pressure, compatible with the diagnosis of NPE. Awareness of the association of saber-sheath trachea with COPD[7] in similar patients is probably warranted.

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Anaphylactic Shock to Neuromuscular Blocking Agent: A Familial History

Philippe Duvaldestin, M.D., Claire Wigdorowicz, M.D.,† Inanna Gabriel, M.D.†*

THE incidence of anaphylactic reactions during anesthesia is estimated in France to be approximately 1/3,500 general anesthesia.[1–3] Muscle relaxants are the most common cause of anaphylactic reactions during anesthesia.[3,4] We report two cases of anaphylactic shock in relation to the administration of nondepolarizing neuromuscular blocking agents that occurred in the members of a family, which suggests that there may be a familial predisposing factor to anaphylactic reactions induced by muscle relaxants.

* Professor and Chairman.

† Staff Anesthesiologist.

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Address reprint requests to: Dr. Duvaldestin: Department of Anesthesia, Henri Mondor Hospital, 51 avenue du Maréchal de Lattre de Tassigny, 94010 Creteil, France. Address electronic mail to: philippe.duvaldestin@hmn.ap-hop-paris.fr

Key words: Anaphylaxis; muscle relaxants; heredity.

Case Report

A 62-yr-old woman weighing 50 kg without preexisting disease was undergoing thyroidectomy during general anesthesia for euthyroid goiter. Anesthesia was induced with 150 μg fentanyl and 350 mg

thiopental associated with 40 mg rocuronium to facilitate tracheal intubation. Immediately after induction of anesthesia, a generalized cutaneous rash developed that was associated with severe hypotension (systolic arterial pressure was 35 mmHg at 2 min after induction) and tachycardia at 115 beats/min. No bronchospasm was present. Immediate treatment consisted of 1 mg intravenous adrenaline and the infusion of 500 ml hydroxyethylstarch. Blood sampling for serum tryptase levels was immediately performed. Return to normal hemodynamic status was obtained after 18 min, and surgery proceeded uneventfully, with anesthesia being maintained with nitrous oxide and isoflurane. Diagnosis of anaphylactic shock was confirmed from the increased serum tryptase level, which was 30 U/l, with control value being less than 2 U/l. Results of intradermal skin tests performed 6 months later were strongly positive for rocuronium at the dilution of 1/1,000. The skin test results were also positive at the dilution of 1/100 for pancuronium, atracurium, and mivacurium, but were negative for vecuronium, succinylcholine, thiopental, fentanyl, and latex.

During the postoperative visit on the day after surgery, the patient informed us that her brother experienced a similar incident 8 yr previously during anesthesia. The review of anesthetic and medical record of the brother revealed that he was 52 yr old and weighed 78 kg when he underwent general anesthesia for surgery of varices of the lower limbs. He was known to have allergic asthma. Induction of anesthesia consisted of 10 mg diazepam and 100 μg fentanyl, followed by 400 mg thiopental and 15 mg alcuronium. A generalized cutaneous rash, followed by cardiovascular collapse and bronchospasm were observed immediately after induction of anesthesia. Successful resuscitation was achieved after the administration of 1 mg intravenous adrenaline. Surgery was cancelled, and the patient was operated on a few months later during epidural anesthesia. In 1997, the two patients were referred to our allergy clinic for skin tests. Results of intradermal skin tests performed on the brother were positive for suxamethonium (1/100 dilution) and atracurium, mivacurium, vecuronium (1/10 dilution) and were negative for pancuronium, fentanyl, and latex. Alcuronium was not tested.

Discussion

This is the first clinical report of a family history of anesthetic anaphylactic shock. In both cases, anaphylactic shock can be attributed to allergy to muscle relaxants. In the sister, who received rocuronium, cutaneous test results were positive for rocuronium. In her brother, alcuronium, which was the muscle relaxant administered, could not be tested, but positive test results for other muscle relaxants were observed. Cross-sensitivity is frequent in patients who have reacted to muscle relaxants.[2,4,5] We cannot rule out the possibility that there is a common genetic basis for these two reactions. However, we also realize that because of the 2.5 million patients undergoing anesthesia with muscle relaxants in France yearly, according to a recent national survey (not yet published), it is entirely possible that these coincident reactions occurred solely because of chance. Alternatively, because there may be cross-sensitization between relaxants and other compounds, the two patients may have shared a common environmental exposure. Hereditary susceptibility to anaphylactic reactions during anesthesia has been discussed; however, cutaneous tests revealed no duplicated allergy among members, including identical twins of families of patients reacting to muscle relaxants.[4] Allergic reaction to muscle relaxants are usually immunoglobulin E mediated, although nonallergic reactions caused by histamine release may occur.[6] Linkage between immunoglobulin E responses underlying allergic reactions and chromosomes 5q and 11q has been claimed.[7,8] However, a recent study of genetic variants among patients with asthma and atopy could not demonstrate any true linkage.[9] The man of the current report had allergic asthma, and it is known that those patients may experience a higher incidence of severe anesthetic allergic reaction than the rest of the population.[4] However, preoperative allergic tests in those patients are considered to be unnecessary because of their low predictive value.[10]

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