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Diffuse Oral Facial Cavernous Hemangioma Causing Severe Airway Obstruction after Intramuscular Ketamine

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SUCCESSFUL prevention of airway-related catastrophes begins with recognizing the potential problem.[1] In this paper, we describe a patient with diffuse oral facial cavernous hemangioma in whom severe airway obstruction occurred unexpectedly, soon after intramuscular ketamine injection.

Case Report

A 3-yr-old boy weighing 12 kg was scheduled for digital subtraction angiography and embolization of diffuse oral facial cavernous hemangioma. He had no previous exposure to anesthesia or surgery. Coughing, crying, vomiting, or straining was often associated with minimal generalized swelling of the hemangioma, but swallowing and breathing were never affected, and spontaneous resolution generally took no more than a few minutes. He had no history of obstructive sleep apnea, hemoptysis, or bleeding in the oral cavity.

General examination was unremarkable. Laboratory investigations were within normal limits. Local examination revealed: (1) an everted lower lip with a rough surface and bluish discoloration; (2) normal facial contours with slightly boggy cheeks; (3) patchy bluish discoloration of skin over the cheeks and lower jaw; (4) minimal macroglossia with discolored margins; (5) sparse and patchy bluish discoloration of entire oropharyngeal mucosa; (6) class I airway[2] and a normal voice; and (7) normal head and neck mobility. To the examiner, signs and symptoms did not appear to indicate any potential airway difficulty.

After oral triclofos, 50 mg intramuscular ketamine with 0.3 mg atropine was given. About 5 min later, when the child appeared to be drowsy, peripheral vein cannulation was attempted, and the stimulation caused transient breath holding. Soon thereafter, the hemangioma suddenly enlarged, leading to airway obstruction and distorted facial contours (fig. 1A and B).

At this stage, the patient's tongue, lips, and cheeks were greatly swollen to a thickness of an inch or more, and the oral cavity, including the hypopharynx, was reduced to a potential space after rapid inward expansion of all the structures in and around the oral cavity. Airway patency could not be restored by inserting either an oropharyngeal or a nasopharyngeal airway. The heart rate increased to 140–150 beats/min, and cyanosis appeared. Orotracheal intubation was performed immediately under direct laryngoscopy, and low-flow oxygen was given *via* "T" connector.

The tracheal intubation was atraumatic except for slight bleeding from the glossoepiglottic mucosal fold. The hemangioma was found to extend into the tonsillar bed, soft palate, and posterior pharyngeal wall; however, the vocal cords, epiglottis, and aryepiglottic folds were not involved.

Subsequently, the orotracheal tube was replaced by a nasotracheal tube. Vital signs gradually returned to within normal limits. The pa-

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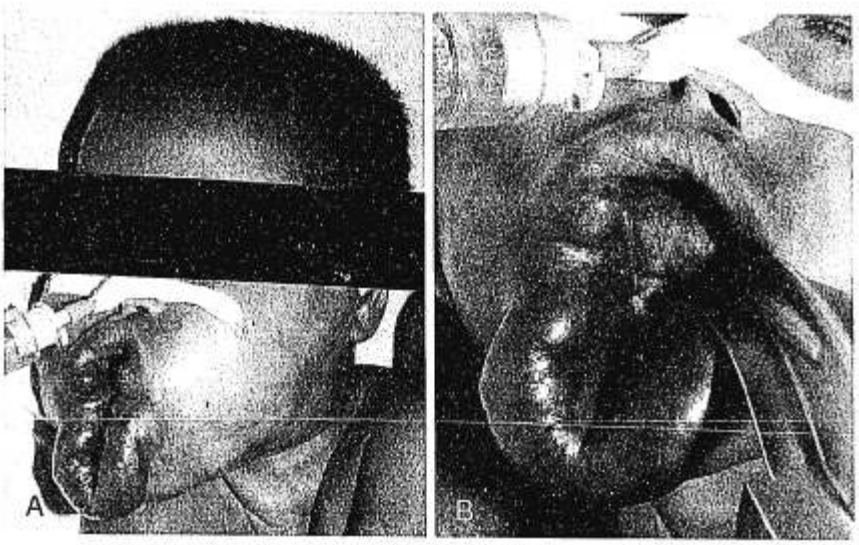

Fig. 1. (*A* and *B*) Facial swelling 2 hours after its onset.

tient regained full consciousness in about 5 h. Generalized swelling continued to regress, and, after 6 h, the oral cavity partially resumed its preanesthetic appearance, and the trachea was extubated. Twenty-four hours later, facial and intraoral structures were back to their preanesthetic state. The patient had no difficulty in breathing or swallowing (fig. 2). He was sent home the next day.

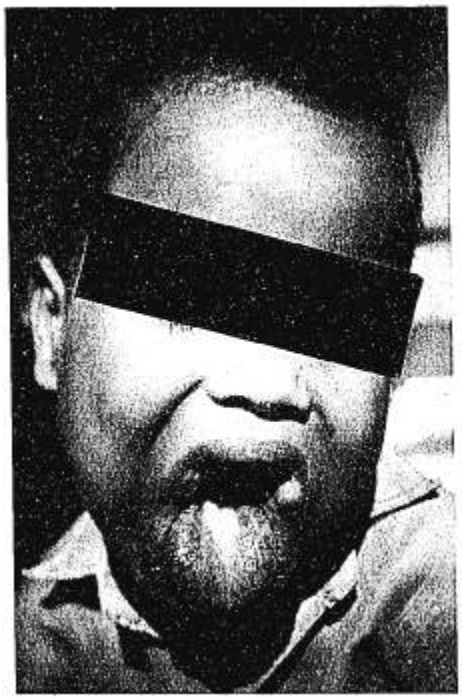

Fig. 2. Near-total disappearance of the facial swelling, 24 hours after its onset.

Three months later, radiologic intervention was again attempted under general anesthesia. No premedication was given, and an intravenous infusion was started before anesthetic induction. After preoxygenation, anesthesia was induced with 120 mg intravenous thiopental (13 kg body weight) while the patient was in the reverse Trendelenburg position. Tracheal intubation was facilitated by 20 mg intravenous succinylcholine. Anesthesia was maintained by 50% nitrous oxide and 0.5–1.5% halothane in oxygen while the patient breathed spontaneously. The hemangioma swelling increased marginally for 10–15 min after tracheal extubation. Unfortunately, radiologic intervention was aborted because of technical problems.

Discussion

Unlike solid nonvascular tumors and anatomic structural abnormalities causing fixed airway defects, diffuse oral facial cavernous hemangiomas may jeopardize airway patency with unpredictable severity during general anesthesia. The cavernous hemangiomas are known to sequester large amounts of blood.[3] However, sudden and severe airway obstruction has not been previously described as a complication of anesthetic management in children with oral facial hemangiomas.

Because the respiratory problem commenced soon after an episode of breath holding, one may conclude that breath holding was the precipitating event. However, before anesthesia, even vigorous coughing did not cause any upper airway obstruction. Thus, in our patient, ketamine and atropine may have played a significant role, with breath holding as a contributing factor. Both ketamine and atropine are known to increase cardiac output and systemic arterial blood pressure.

Atropine is also known to cause dilatation of the "normal" vessels of the face,[4] and ketamine also causes smooth muscle relaxation, particularly in those vascular beds that are depleted or devoid of a normal autonomic control. It has been demonstrated that ketamine directly causes vascular smooth muscle dilatation by an active process while causing sympathetically mediated vasoconstriction.[5,6] Thus, the net effect of ketamine on the resistance of different vascular beds will depend on the state of sympathetic innervation of those beds.[5,6]

Vascular malformations, including cavernous hemangiomas, are known to be deficient of normal autonomic control. Cavernous hemangiomas, although predominantly venous lesions, may contain true arteriovenous malformations or abnormal microarteriovenous fistulous malformations.[7] The vascular bed of the hemangioma, not being under normal autonomic control, will effectively respond passively to autonomic or drug-induced changes. The hemangioma will also respond with the changes in vascular tone (resistance and compliance) caused by pharmacologic agents having a direct effect on smooth muscle, or *via* the drug affecting the endothelial cell, which then subsequently affects smooth muscle tone.

If we presume that ketamine caused increased cardiac output, increased right heart filling pressures, increased systemic vascular resistance, and decreased venous compliance of the normal innervated vascular bed, and caused dilatation of hemangioma vessels by its direct action and, because it was not under autonomic control, the dilatation remained unopposed by its sympathetically mediated vascular effects, then ketamine may have caused the following effects on the hemangiomatous malformation.

First, the increased cardiac output and systemic vascular resistance would increase blood pressure such that the vascular bed of the hemangioma, which did not constrict, would now receive more blood *via* its arterial connections and microarteriovenous fistulae.

Second, if we assume that the abnormal venous network within the hemangioma had very high compliance and, hence, little elastic recoil to rapidly empty this increased blood flow, the hemangioma would increase in size until the pressure inside it rose sufficiently to empty the blood as fast as it was coming in, *i.e.*, a new steady state.

Third, the second component of increased vascular tone, produced by diffuse sympathetic tone on the normally innervated vascular beds, would be a decrease in venous compliance. This would facilitate the shifting of blood from normal venous capacitance beds to the hemangioma.

Fourth, dilatation of hemangioma vessels (a direct effect of ketamine unopposed by its sympathetic effects) would cause decreased resistance and increased compliance, further facilitating the sequestration of blood in the hemangioma; and fifth, increased right heart filling pressures may also decrease the emptying of the hemangioma, particularly if the hemangioma exhibits a highly alinear pressure–compliance relationship.

Retrospectively, ketamine and atropine appear to have been an inappropriate choice of drugs for this case. The relative superiority of thiopental and halothane could be demonstrated in this patient. However, the behavior of large diffuse hemangiomas with different anesthetic agents requires further evaluation.

A possible catastrophe was averted by timely tracheal intubation. Although fraught with the danger of trauma, tracheal intubation was atraumatic and not very difficult. Other options would have had significant problems. Laryngeal mask airway (LMA) could have been tried, but there was very little space in the hypopharynx to accommodate even the smallest LMA, and it could also have traumatized the friable and swollen structures. Tracheostomy was avoided, because it would have resulted in severe bleeding from the site, which was full of engorged cutaneous vessels.

In patients with vascular malformations involving airways (in contrast to patients with fixed airway defects), the nature and severity of airway obstruction resulting from anesthetic maneuvers may not become apparent during preanaesthetic airway assessment. We feel that airways with vascular malformations must always be recognized as potentially difficult airways. Although thiopental and halothane were successfully used in this patient, an anesthetic technique that decreases vascular tone *via* blockade of sympathetic output to normally innervated beds (as opposed to direct actions reducing vascular tone, which may affect both normal innervated and abnormally innervated beds) would be a rational approach in the management of patients having diffuse and large vascular malformations involving the airways.

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Aneurysmal Compression of the Trachea and Right Mainstem Bronchus Complicating Thoracoabdominal Aneurysm Repair

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PATIENTS presenting for repair of thoracoabdominal aneurysms pose many challenges for the anesthesiologist. The procedure requires a complicated anesthetic technique that must be adapted to the needs of specific clinical situations. A common practice is the use of double-lumen endobronchial tubes (DLT) and one-lung ventilation (OLV) to facilitate surgical exposure of the aneurysm.[1] This technique may be contraindicated in situations in which the aneurysm may cause compression of the trachea and left mainstem bronchus.[1,2] We report a case in which compression of the right mainstem bronchus precluded the use of a DLT and OLV in the repair of a large thoracoabdominal aneurysm.

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Key words: Anesthesia: thoracic. Anesthetic techniques: one-lung ventilation. Surgery: thoracic aneurysmectomy.

Case Report

A 77-year-old man was admitted to the hospital for elective repair of an extensive dissecting thoracoabdominal aneurysm (DeBakey type IIIB). During the 3–4 weeks before surgery, the patient had been experiencing progressive dyspnea on exertion with increasingly limited activity. The patient had also complained of progressive dysphagia and experienced a 20-pound weight loss in the 2 months before operation.

Preoperative evaluation revealed a thin, ill-appearing man in no apparent distress. There were no gross abnormalities of the head and neck, and the trachea was midline. Examination of the lungs with the patient in the upright position was unremarkable. Laboratory studies were significant only for a hematocrit of 31%. The chest x-ray revealed a widened mediastinum with normal-appearing lung fields. There was no report of tracheal or bronchial compression. A CT scan of the chest showed a dissecting aneurysm of the descending thoracic and abdominal aorta that was 8 cm at its widest margin.

In the operating room, radial artery, central venous, and pulmonary artery catheters were placed. A catheter was placed in the lumbar subarachnoid space for drainage of cerebrospinal fluid. Anesthesia was induced uneventfully with thiamylal, fentanyl, and pancuronium. Because of the patient's symptoms of dyspnea, we elected to intubate the trachea with a 8.5-mm single-lumen endotracheal tube, and to perform bronchoscopy before placement of a left-sided DLT. After tracheal intubation, it was noted that chest movement was asymmetrical and that breath sounds were diminished on the right side. Bronchoscopy revealed that the lower one-third of the trachea was approximately 50% narrowed, and that the right mainstem bronchus was almost completely occluded by external compression (figs. 1 and 2).

Two concerns arose at this point. First, we were reluctant to pass a DLT beyond the tracheal and bronchial obstruction, because it was unclear to what extent the aneurysm may have eroded the airway structures. Secondly, had we placed a DLT in the left mainstem bronchus, ventilation of the right lung may not have been possible because