Management of Airway Compromise following Thyroid Cyst Hemorrhage after Thrombolytic Therapy

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ABSTRACT

The risk of hemorrhage after therapeutic administration of tissue plasminogen activator (tPA) is well known. Cases of post-administration hemorrhage have been reported within many organ systems, including several reports of hemorrhage in the head and neck leading to airway obstruction. We present a case of a sixty-two year old female with undiagnosed thyroid goiter who received tPA for acute ischemic stroke and developed acute airway compromise. The surgical airway response team was called due to inability to ventilate or intubate. An incision into the mass allowed release of colloid and blood, decompressing the airway and facilitating ventilation and intubation. Hemithyroidectomy for mass removal was delayed for three days to allow normalization of post-tPA coagulopathy. A review of the literature indicates that this is the first report of acute airway obstruction due to intrathyroid hemorrhage after tPA administration. Discussion of related cases, treatment, and clinical challenges will follow.

INTRODUCTION

Intrathyroid hemorrhage causing mass effect sufficient to cause acute airway compromise is rare, and is usually precipitated by blunt trauma or iatrogenic insult to the neck as in fine needle aspiration.1-3 Spontaneous hemorrhage into a thyroid mass has also been described; possible precipitants for these events include unidentified physical exertion or the use of oral anticoagulants such as aspirin or warfarin.1-3 Tissue plasminogen activator (tPA) is a thrombolytic agent accepted as therapy for selected cases of acute ischemic cerebrovascular insults, pulmonary embolism, myocardial infarction, deep venous thrombosis, and portal vein thrombosis. A rare side effect of tPA’s thrombolytic activity is spontaneous hemorrhagic complications in other organ systems. There have been several reports of acute airway obstruction that developed after administration of tPA in acute ischemic stroke. Some cases identify a trauma that precipitates hemorrhage, such as lingual hematoma after tongue biting, sublingual and submandibular hematoma attributed to jaw thrust, upper airway hemorrhage due to tongue biting during external chest compressions, and uvular hematoma from laceration by Magill’s forceps.3 There are also reports of massive aerodigestive tract hemorrhage, prevertebral hematoma, and lingual hematoma in which no trauma could be identified.2-5 We report a case in which acute airway compromise developed after administration of tPA for acute ischemic stroke due to hematoma development within a cystic thyroid goiter with no clear traumatic etiology.

CASE REPORT

The patient is a 64-year-old woman who had no known past medical history before presentation. She was found at work to have confusion, aphasia, and right-sided hemiparesis. She was transported via ambulance to a local hospital. Head CT findings were consistent with acute infarct in the left frontal lobe. Her complete blood count was within normal limits. She received intravenous saline, midazolam, and two doses of intravenous tPA. 0.09 mg/kg of tPA was given over 2 minutes, followed by 0.81 mg/kg over 60 minutes.

The patient was transported to the neurovascular intervention suite for angiography and intervention. During preparation, the anesthesiologist noted progressive shortness of breath. Gross swelling of the patient’s neck, with blunting of the normal cartilaginous landmarks, was observed. Bag-mask ventilation was unsuccessful. The otolaryngology service was paged. On arrival, the sternal notch was the only palpable landmark in the patient’s neck. 3 cc of 1% lidocaine with epinephrine was injected subcutaneously 1 cm above the sternal notch, and a 3 cm vertical incision in the midline ending 1 cm superior to the sternal notch was made with a No. 15 surgical blade. After the incision was deepened, a high-pressure stream of blood and clear fluid flowed from the neck. With the relief, bag-mask ventilation was successful. The patient was intubated with difficulty due to distorted laryngotracheal anatomy; the trachea appeared to course posteriorly and to the patient’s right.

The patient was then transported to the operating room for neck exploration and hemostasis. A large thyroid mass was noted, but excision was delayed due to the recent administration of tPA. A post-operative CT with contrast of the head and neck on hospital day 1 revealed a large left neck hematoma displacing the left common carotid artery and overlying a large soft tissue mass with subcutaneous emphysema (see Figure 1).

During the next few days, the patient remained stable on a ventilator. She received fresh frozen plasma with the goal of speeding reversal of her profibrinolytic state. Her neck remained grossly distended (see Figure 2). On hospital day 3, the left thyroid lobe was excised and the neck was explored for control of any further bleeding. A small tear of the left pyriform sinus was also discovered and repaired; it was thought to have been a result of traumatic intubation in the interventional suite. A formalized tracheostomy was also performed (see Figure 3).

Pathology reported a 13.7 x 10.2 x 3.3 cm nodular goiter with central hemorrhage and marked cystic degeneration. On HD13, TSH was 1.99 (reference range 0.358 – 3.740).

CONCLUSIONS

This case suggests that acute intrathyroid hemorrhage causing upper airway obstruction may develop after therapeutic administration of tPA. Thorough examination and careful monitoring of vital signs and clinical condition in patients who receive tPA is necessary. This is especially important when transfer of patient care occurs after tPA administration and when the patient is unable to communicate, as occurred in this case. Emergent surgical intervention may be warranted if the development of airway compression is not detected early; in this case, emergent incision over the mass was needed for decompression and relief of airway obstruction. Recent administration of tPA may complicate surgical planning.

REFERENCES

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