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PRACTICAL PEARL

Thyroid Hemorrhage Causing Airway Obstruction After Intravenous Thrombolysis for Acute Ischemic Stroke

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Abstract
Background There are several life-threatening complications associated with intravenous thrombolysis after acute ischemic stroke such as symptomatic intracerebral hemorrhage, orolingual angioedema, or less frequent, bleedings of the mucosa or ecchymosis. Aside from these known critical incidents, rare and unfamiliar complications may be even more challenging, as they are unexpected and may mimic events that appear more frequently. We report a rare and unusual acute complication of intravenous thrombolysis with recombinant tissue plasminogen activator (rt-PA) (0.9 mg/kg) administered for acute ischemic stroke.
Methods Medical records, radiologic imaging, and pathologic specimens were reviewed.
Results A 86-year-old woman developed acute respiratory failure 20 h after thrombolysis with suspected angioedema triggered by intravenous rt-PA. The inspiratory stridor and dyspnea were unresponsive to bronchodilators, corticosteroids, and inhaled adrenaline. After endotracheal intubation, laryngoscopy showed no significant supraglottic narrowing. Thyroidal sonography and cervical computed tomography revealed a thyroidal mass causing a tracheal and vascular compression compatible with thyroidal hemorrhage. Sonography showed a nodular goiter of the right thyroid gland. A total thyroidectomy was performed and histologic analysis confirmed a hemorrhage of the right thyroidal lobe.
Conclusions Acute airway obstruction with respiratory failure due to thyroidal hemorrhage after intravenous thrombolysis is an important life-threatening complication, mimicking an anaphylactic reaction or a more frequent orolingual angioedema.

Keywords Acute ischemic stroke · Intravenous thrombolysis · Complication · Intubation · Airway obstruction · Neurocritical care

Introduction

There are several challenging and life-threatening complications associated with intravenous thrombolysis after acute ischemic stroke that may worsen outcome, such as symptomatic intracerebral hemorrhage, orolingual angioedema, or less frequent mucosa bleedings or ecchymosis, as well as acute myocardial rupture when intravenous recombinant tissue plasminogen activator (rt-PA) is given in the setting of subacute myocardial infarction [1]. However, there are other rare complications which may be even more challenging, as they remain unexpected and may mimic events that appear more frequently.

We report an unusual complication of intravenous thrombolysis with rt-PA (0.9 mg/kg) administered for acute ischemic stroke.

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Case

An 86-year-old woman presented with a sudden onset of a left-sided weakness and dysarthria. Her past medical history was relevant for arterial hypertension and mitral regurgitation. Initial head computed tomography (CT) showed a hyperdense middle cerebral artery sign of the right M1-segment, suspicious for a thromboembolic occlusion, and excluded an intracerebral hemorrhage. Neurologic examination revealed a sensomotoric, faciobrachial left hemiparesis with an ipsilateral hemineglect and dysarthria. The National Institutes of Health Stroke Scale (NIHSS) was 11. Intravenous thrombolysis was administered with weight-adapted rt-PA 60 min after admission, and 260 min after symptom onset, with an immediate neurofunctional improvement (NIHSS 4) except for a persisting proximal weakness of the left arm and an ipsilateral incomplete facial paresis. Follow-up head CT revealed no hemorrhagic transformation or intracerebral bleeding. Newly diagnosed atrial fibrillation corroborated the etiological hypothesis of a thromboembolic cause of the stroke.

Twenty hours after intravenous rt-PA was given, the patient developed acute respiratory failure with an inspiratory stridor and dyspnea. These symptoms were unresponsive to bronchodilators, corticosteroids, and inhaled adrenaline. After endotracheal intubation, laryngoscopy showed no supraglottic narrowing, ruling out an orolingual angioedema. Thyroidal sonography detected an enlarged hyperechogenic thyroid gland and cervical CT revealed a thyroidal homogenous hypodense mass (Fig. 1, white arrows) including smaller hyperdensities (Fig. 2a, white arrows) causing a tracheal and vascular compression (Fig. 2a, black arrows) compatible with thyroidal hemorrhage. In addition, sonography showed a nodular goiter of the right thyroid gland. Therefore, a total thyroidectomy was performed. Histologic examination revealed multiple nodules and a hemorrhage of the right thyroid (Fig. 3a–d). The left thyroid gland appeared unremarkable and there was no evidence of malignancy on both sides. One day after the thyroidectomy, the patient was extubated and had no signs of airway obstruction but a deterioration of the weakness with a left-sided hemiplegia. A follow-up head CT revealed a slight expansion and hemorrhagic transformation of the infarct into the *centrum semiovale*. During the following 2 weeks, the patient's dysarthria and weakness gradually decreased with a remaining, ataxic, faciobrachial paresis, and she was discharged to a

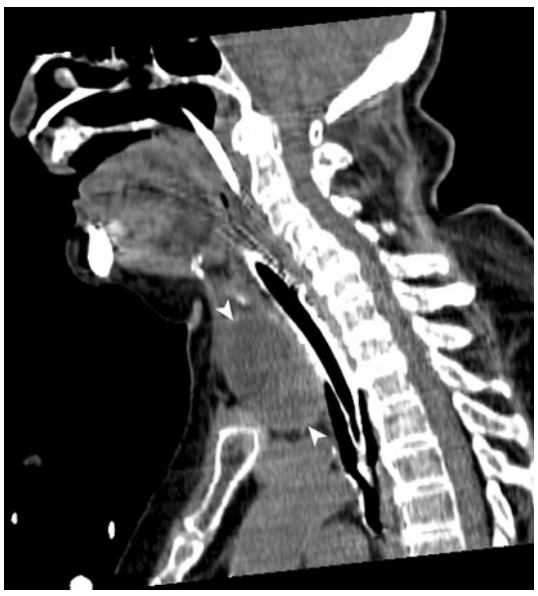

Fig. 1 Sagittal cervical computed tomography showing a thyroidal homogenous hypodense mass with focal tracheal and vascular compression (*white arrows*)

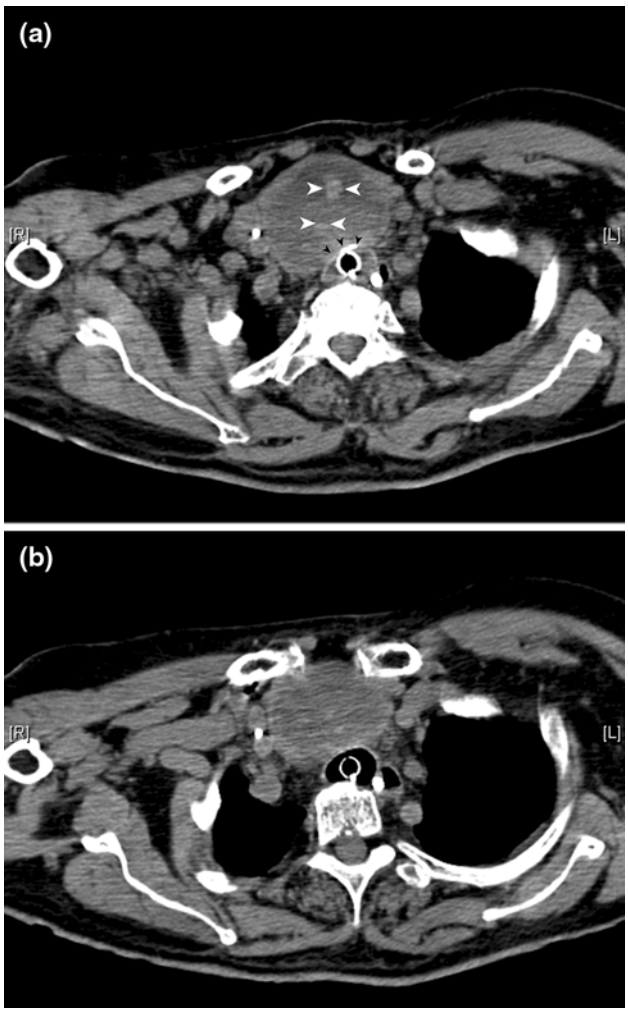

Fig. 2 Transversal cervical computed tomography. **a** Thyroidal homogenous hypodense mass including smaller hyperdensities (*white arrows*) with tracheal and vascular compression (*black arrows*). **b** Intratracheal tube below the area of compression

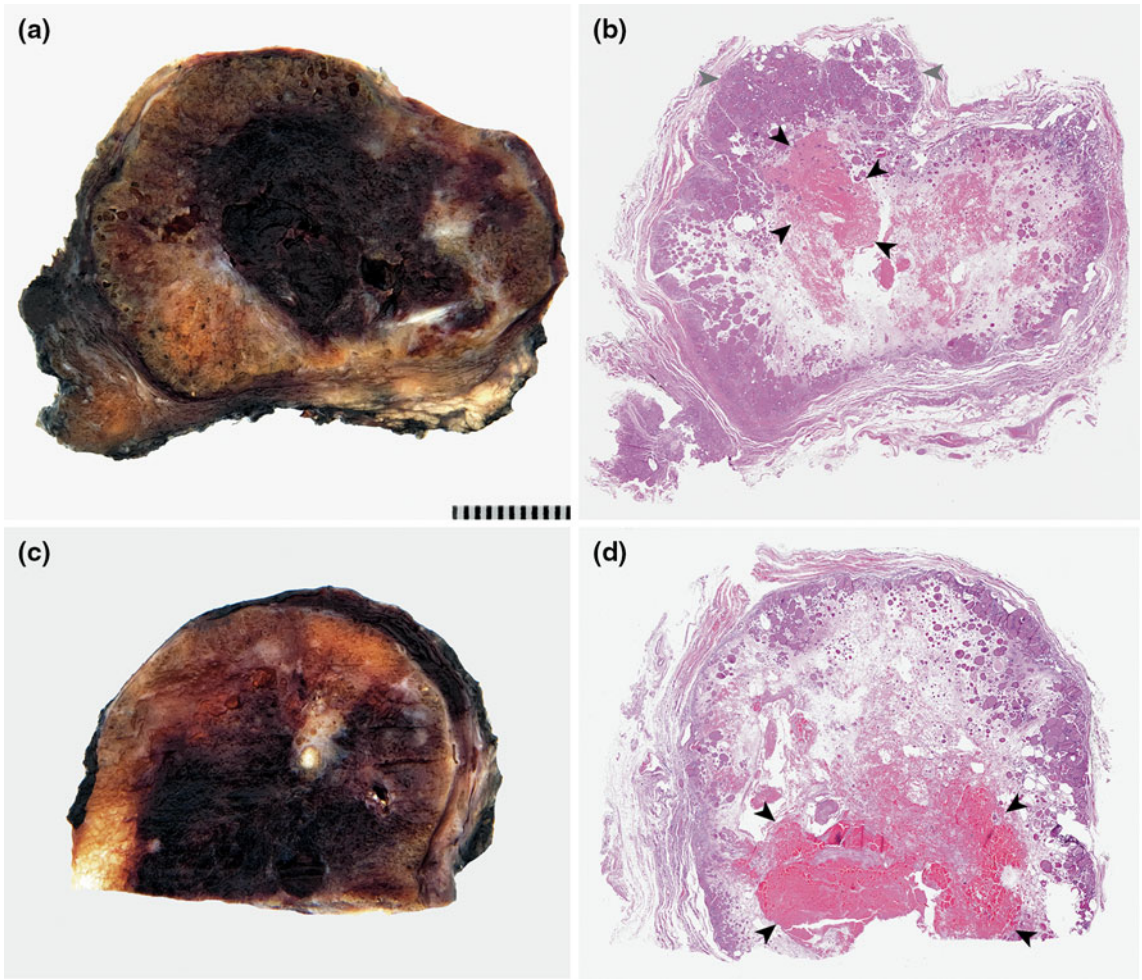

Fig. 3 Thyroidectomy specimen. **a**, **c** Macroscopic images of cut surfaces of the thyroidectomy specimen with a large central hemorrhage. **b**, **d** Whole mount histologic section revealing nodular hyperplasia (*gray arrows*) with a fresh central hemorrhage (*black arrows*) (hematoxylin–eosin)

rehabilitation center with a Medical Research Council scale of 4/5 in her left arm. After 1 month of stationary rehabilitation, the patient recovered completely with the exception of a persistent, subtle, ataxic gait. Two years after the stroke the patient has fully recovered and living independently on her own.

Discussion

This is the first case report of an isolated thyroidal hemorrhage complicating intravenous thrombolysis following ischemic stroke. The most deleterious and life-threatening complication after thrombolysis in this setting is a symptomatic intracerebral hemorrhage, which occurs in 6–15 % of patients [2, 3] and is associated with unfavorable outcome [4–6]. Hemorrhagic transformation of strokes, on the other hand, is reported to be far more frequent (8–35 %) and mostly remains clinically asymptomatic [7, 8]. Orolingual angioedema is reported in 1.5–5 % of stroke patients treated with intravenous rt-PA [9, 10]. Less frequent complications are bleedings of the mucosa or ecchymosis.

Thyroidal hemorrhage with relevant compression of surrounding structures is rare and mostly iatrogenic or caused by trauma. Some reports describe thyroidal hemorrhage related to oral anticoagulation or coexisting thyroidal toxicosis [11, 12]. To date, there are few reports of spontaneous hemorrhage in thyroid goiter and only one case report of thyroidal hemorrhage following thrombolysis after myocardial infarction [13], reflecting that thyroid hemorrhage is a rare complication of intravenous thrombolysis. However, physicians should be aware of this life-threatening complication, as early recognition and rapid intervention can lead to good outcome. Despite this complication, our patient had a good neurofunctional recovery and long-term outcome after intravenous thrombolysis and rehabilitation.

Conclusions

Acute respiratory failure due to thyroidal hemorrhage after intravenous thrombolysis is an important life-threatening complication, mimicking an anaphylactic reaction or more frequent orolingual angioedema.

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Conflict of interest Dr. Raoul Sutter, Dr. Gianmarco M. Balestra, Dr. Elisabeth Bruder, and Dr. Mandy Weissenburg have no conflicts of interest to disclose.

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