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Thrombolytic Associated Pontine Hemorrhage in a Stroke Patient

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Abstract Stroke is a leading cause of disability and death, and the use of tissue plasminogen activator (tPA) has been shown to improve outcomes in ischemic stroke. However, despite the favorable outcome from the use of tPA, it has been associated with complications, including intracranial hemorrhage, major systemic hemorrhage, and angioedema. Though there are few reports of intracerebral hemorrhage complicating tPA use, pontine hemorrhage, a sub-set of intracerebral hemorrhage, is rarely reported. We report a case of pontine hemorrhage in an ischemic stroke patient after tPA and discussed the management of this rare complication of tPA.

Keywords: *stroke, CVA, tissue plasminogen activator, pontine hemorrhage, intracerebral hemorrhage*

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1. Introduction

There are about 795,000 stroke cases in the United States yearly, and stroke is a leading cause of severe long-term disability and death [1]. Intravenous recombinant tissue plasminogen activator (tPA) is currently the only pharmacotherapy approved by the US Food and Drug Administration for ischemic stroke. In addition, the use of tPA early in ischemic stroke has been shown to improve outcomes [2,3]. However, only a tiny percentage of those with acute ischemic stroke, about 2% to 5%, receive tPA [4]. Despite the favorable outcome from the use of tPA, it has been associated with complications, including symptomatic intracranial hemorrhage, major systemic hemorrhage, and angioedema [4]. The incidence of symptomatic intracranial hemorrhage in several major studies ranged from 1% to 11.2. Risk factors for symptomatic hemorrhage include age, black race, male gender, obesity, increased stroke severity, diabetes, hyperglycemia, uncontrolled hypertension, combined antiplatelet use, large areas of early ischemic change, atrial fibrillation, and congestive heart failure [3-5]. Though there have been few reports of intracerebral hemorrhage complicating tPA use, pontine hemorrhage, a sub-set of intracerebral hemorrhage, is rarely reported. We report the case of pontine hemorrhage in an ischemic stroke patient after tPA.

2. Case

A 60-year-old female with underlying paranoid schizophrenia, hepatitis C, hypertension, and migraines presents to the emergency department for left-sided weakness, onset 40 minutes before presentation. Initial numbness started over her left hand and radiated up to her left arm and face, with the patient subsequently developing left lower extremity weakness. Examination showed an alert and oriented patient with decreased sensation on her left face, arm, and hand and decreased strength on her left lower extremity.

In the emergency department, code stroke was initiated, and the patient was placed on continuous oxygen and cardiac monitoring. Systolic blood pressure was 212/115 at presentation with a heart rate of 95, and the patient was given IV labetalol 20 mg push. She was then started on labetalol drip, with blood pressure going down to the 130s over the 70s. Laboratory tests were grossly unremarkable.

Initial computed tomography (CT) brain showed no acute pathology (Figure 1, Figure 2). Tele-neurology was consulted, who recommended starting tPA. The patient was administered tPA with an improvement of symptoms. After tPA administration patient was able to lift both the left arm and left leg with no facial asymmetry noted on smiling. The patient was also able to answer questions appropriately.

Two hours after tPA, the patient complained of a headache associated with one episode of vomiting. The patient was sent for a repeat CT brain and returned from CT agitated with recurrent vomiting. The patient became unresponsive with decerebrate posturing and intermittent seizure-like activity and was subsequently intubated in the emergency department. CT brain post-tPA demonstrated acute parenchymal hemorrhage in the inferior midline pons 1.4 x 1.3 x 1 cm size with minimal edema (Figure 3, Figure 4). A neurologist was consulted and recommended the patient be taken off sedation for a neurology exam.

After being 45 mins off sedation, the patient was examined, and she had bilateral pinpoint pupils and a Glasgow Coma Scale score of 3. The patient received two units of platelets and one pooled unit of cryoprecipitates and was transferred to a tertiary hospital for neuro-intensive care unit monitoring.

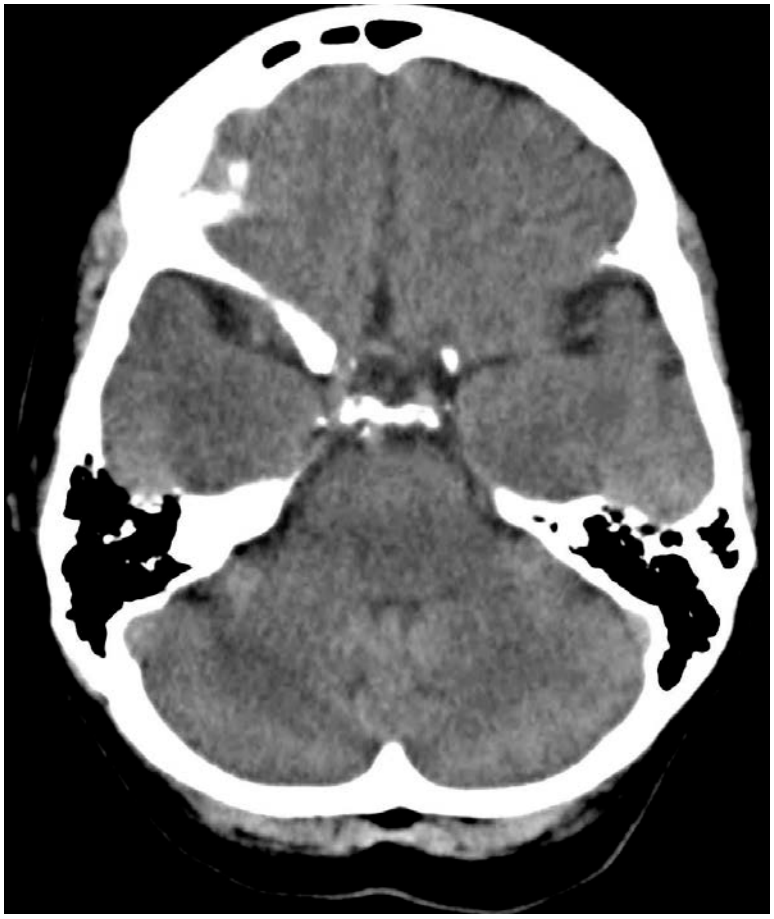

Figure 1. Stroke protocol axial brain computer tomography with no evidence of pontine hemorrhage

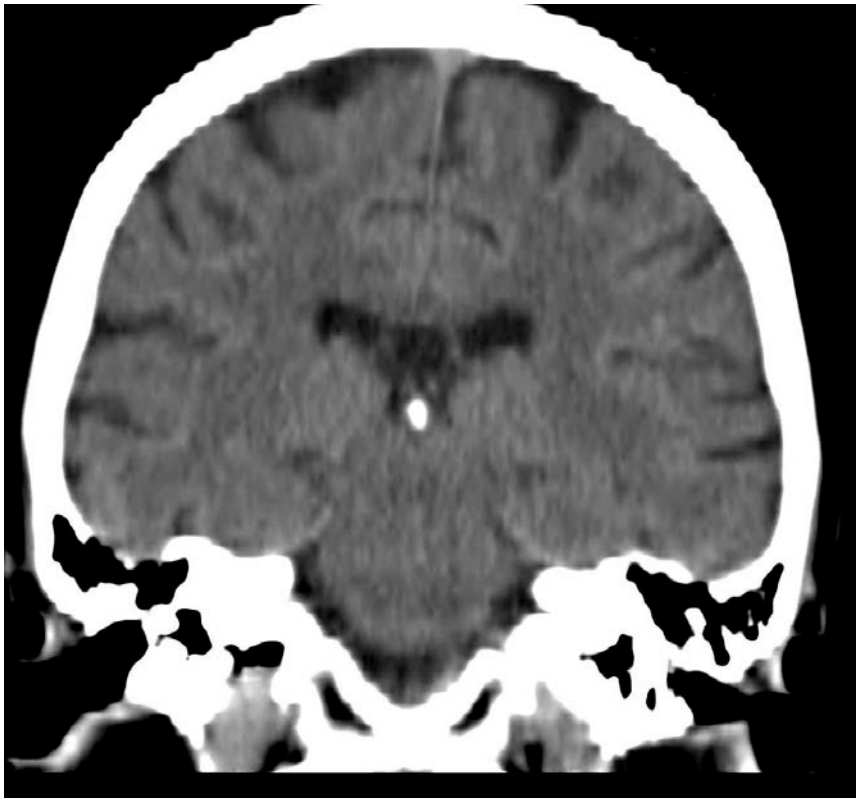

Figure 2. Stroke protocol coronal brain computer tomography with no evidence of pontine hemorrhage

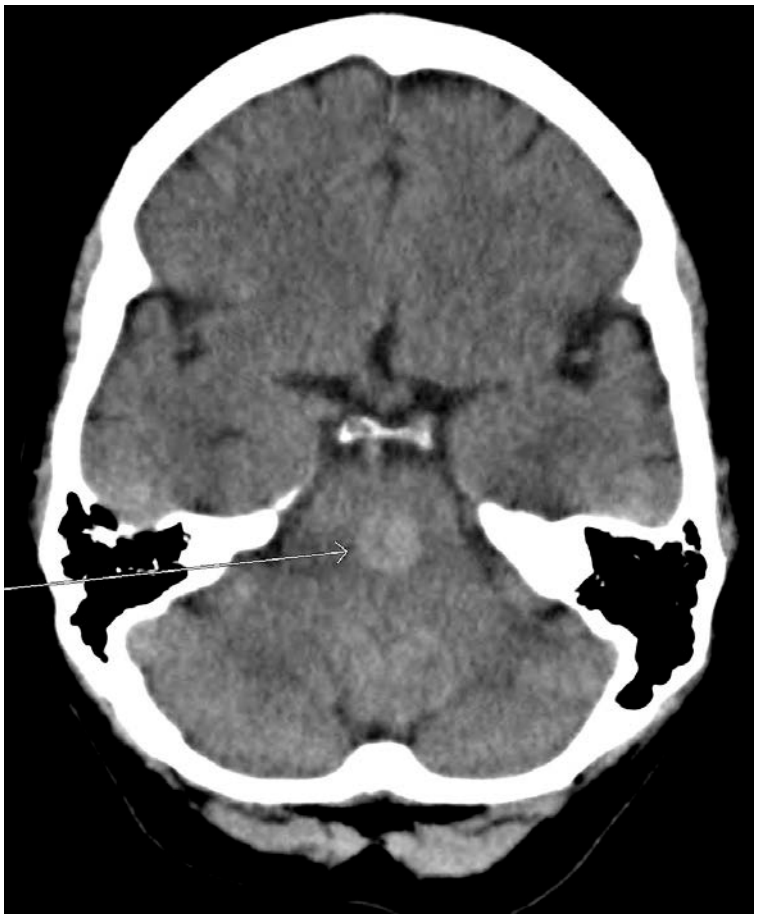

Figure 3. Axial brain computer tomography through the level of the pons 2 hours after tissue plasminogen activator (tPA) administration demonstrating pontine hemorrhage

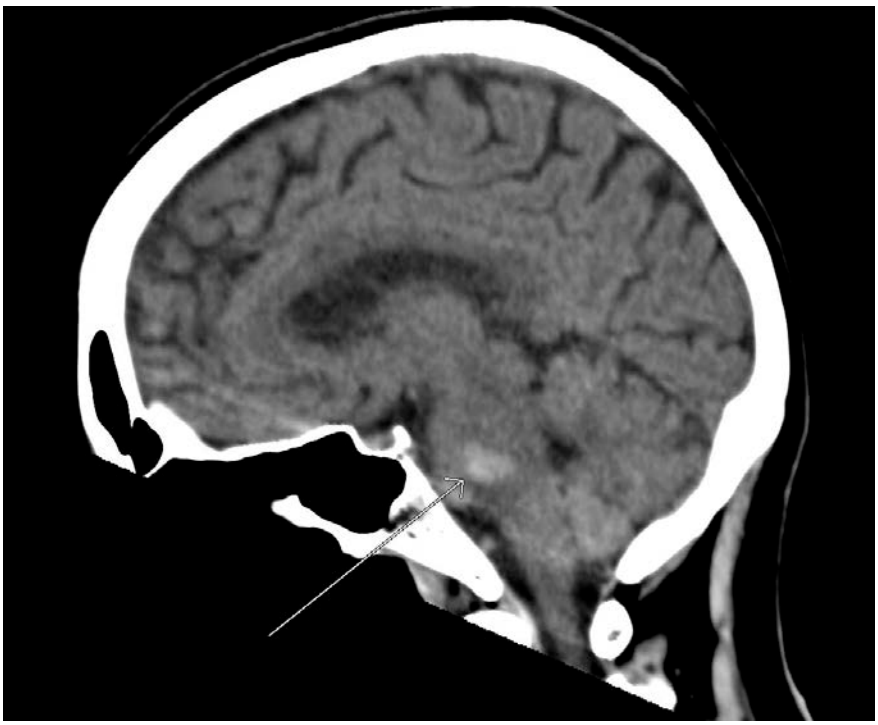

Figure 4. Sagittal brain computer tomography through the level of the pons 2 hours after tissue plasminogen activator (tPA) administration demonstrating pontine hemorrhage

3. Discussion

Symptomatic intracerebral hemorrhage (sICH) is the most dreaded complication of tPA [6]. Acute onset of headache, vomiting or impaired consciousness level in a patient who received tPA could indicate an intracerebral bleed [7]. However, a definite diagnosis of intracerebral bleed can only be made through imaging. Computed

tomography (CT) and magnetic resonance imaging (MRI) have equal ability to identify acute intracerebral hemorrhage, its size and location, and hematoma enlargement [7]. While MRI is superior at delineating the extent of perihematomal edema, herniation, and any underlying structural lesions, CT may be superior at demonstrating a ventricular extension of intracerebral bleeds [7].

Management of patients with thrombolytic associated symptomatic intracerebral hemorrhage is challenging. Currently, there are no evidence-based guidelines for managing thrombolytic associated symptomatic intracerebral hemorrhage. The American Heart Association/American Stroke Association has suggested empirically replacing clotting factors and platelets in patients with symptomatic intracerebral hemorrhage. However, it acknowledges the lack of strong evidence to support any specific therapy [7,8]. Analysis of procoagulant therapies used in a large single urban tertiary care hospital, including administering fresh frozen plasma, cryoprecipitate, vitamin K, platelets, and aminocaproic acid, did not show a statistically significant association with any patient outcome [8]. However, the discovery of continued bleeding (>33% increase in intracerebral hemorrhage volume) in some patients with a thrombolytic associated symptomatic intracerebral hemorrhage who did not receive procoagulant therapy suggests that therapy may be beneficial [8]. Our patient received platelet and cryoprecipitate and was transferred to a neuro-intensive care unit.

Pontine hemorrhage is associated with high morbidity and mortality. In a study reviewing predictive factors of poor outcome in pontine hemorrhage, 55% of the patients died shortly after hospital admission, 24% were severely or moderately disabled, and 21% had a good recovery [9]. Another study found a 60% mortality in patients with thrombolysis-related intracranial hemorrhage [10]. A history of hypertension, coma on admission, the need for mechanical ventilation, absent motor response, absent corneal reflex or oculocephalic responses, hyperthermia (core temperature >39 °C), tachycardia (>110 beats/min), the volume of intracerebral hemorrhage, extrapontine extension of bleed, intraventricular extension, and acute hydrocephalus on admission were associated with mortality in patients with pontine hemorrhage [9,11,12]. Conversely, being alert on admission and having small unilateral pontine hemorrhage were associated with good recovery [9].

4. Conclusion

In conclusion, we present a case of thrombolysis-related pontine hemorrhage, a very rare and severe complication of tPA. Despite the favorable outcome from the use of tPA, this case highlights a severe complication associated with tPA and the need to carefully screen stroke patients to ensure that only patients without contraindications to tPA receive it.

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