

Case Report

Spontaneous Spinal Subarachnoid Hematoma after Recombinant Tissue Plasminogen Activator (rt-PA) Treatment for Acute Ischemic Stroke: A Case Report

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An 83-year old man with acute ischemic stroke was treated by intravenous recombinant tissue plasminogen activator (rt-PA). Five hours later, he developed progressive paraplegia with sensory impairment below C6 dermatome (Frankel class A). Cervical spinal MRI showed intradural extramedullary hematoma posterior to the C5 vertebral body with multiple levels of cervical stenosis and kyphotic deformity. We performed C5 corpectomy with removal of subarachnoid hematoma, C6-7 discectomy and fusion. At 6 month postoperative, the patient could walk for a short distant and still continued rehabilitation program. Spontaneous spinal subarachnoid hematoma is an extremely rare complication following rt-PA administration. Physicians must realize this condition when patients have spinal pain or progressive neurological deficit after receiving intravenous rt-PA.

Keywords: Spontaneous spinal subarachnoid hematoma, Recombinant tissue plasminogen activator, rt-PA, Progressive paraparesis

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Intravenous administration of recombinant tissue plasminogen activator (rt-PA) is the gold standard treatment for acute ischemic stroke within 4.5 hour after symptom onset⁽¹⁾. Intracerebral hemorrhage is a common hemorrhagic complication of thrombolysis therapy with rt-PA⁽²⁾, whereas spontaneous spinal hemorrhage or hematoma is very rare complication; most patients have spontaneous spinal epidural hematoma⁽³⁻⁶⁾. We report a case of spontaneous spinal subarachnoid hematoma that caused spinal cord compression in a patient with multilevel cervical stenosis and kyphosis of cervical spine who received rt-PA for the treatment of acute ischemic stroke.

Case Report

An 83-year-old man presented to the emergency department with left hemiparesis for 2 hours. He had coexisting coronary artery disease,

hypertension, gout and hyperlipidemia for many years. Neurological examination revealed left upper motor neuron type facial palsy, grade 4/5 motor power strength in the left upper extremity and grade 3/5 in the left lower extremity. Decreased pinprick sensation on left side of the body was noted. National Institutes of Health Stroke Scale (NIHSS) was 5. Electrocardiography showed atrial flutter which may be the cause of cardio-embolic stroke. Emergency non-contrast enhanced computerized tomography (CT) of the brain showed hyperacute infarction at the right basal ganglia with mild edema of the lesion. The patient had no contraindication for rt-PA treatment, then 90 mg of rt-PA was injected intravenously. Five hours following initiation of rt-PA treatment, he developed painless progressive quadriparesis. Physical examination showed grade 0/5 motor power strength with flaccid paralysis, loss of light touch, pinprick, temperature and vibratory sensations below C6 dermatomes. Diminished deep tendon reflex and loss of anal sphincter tone were mentioned. Non-contrast enhanced CT of the brain showed minimal subarachnoid hemorrhage and intraventricular hemorrhage, no detection of new infarction lesion. These findings could not explain

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the development of progressive quadriparesis. Consequently, emergency magnetic resonance imaging (MRI) of the cervical spine was performed and revealed a focal mass at C5 spinal level with isointensity on T1-weighted image (T1WI) and hyperintensity on T2-weighted image (T2WI). The characteristic of lesion was compatible with hematoma and a vascular-like structure at the posterior border of hematoma. We were aware of a vascular lesion which can cause bleeding. Subsequent spinal angiography showed no abnormal vascular lesion.

Because the patient developed rapidly progressive neurological deficit to complete spinal cord paralysis (Frankel class A) and cervical spinal MRI showed multiple level of spinal stenosis extending from C4 to C7 with kyphosis as show in Fig. 1, the patient

underwent C5 anterior corpectomy with hematoma evacuation and C6-7 discectomy to correction of cervical kyphosis. After C5 corpectomy, the epidural space appeared normal then we performed dural opening and hematoma was seen beneath the arachnoid membrane. The arachnoid membrane was opened and hematoma was removed totally. Some bleeding from small vessels attached to the posterior surface of hematoma was seen and stopped with bipolar electrocautery. The dura mater was closed in water-tight fashion. Fibular allograft was placed between C4 and C6 vertebral bodies, and then anterior cervical plate and screws was applied from C4 to C7 spinal levels. Pathological report revealed no abnormal vascular lesion or tumor cell in the hematoma.

At 6 month after surgery, the patient still had spastic quadriparesis with grade 4/5 motor strength on the left side and grade 3/5 on the right side. He felt some dysesthesia on both lower legs. He was able to walk in a short distance and still continued rehabilitation program.

Discussion

Intravenous administration of rt-PA is a gold standard treatment for acute ischemic stroke and about 6.4% of patients who receive this thrombolytic agent develop intracerebral hemorrhage within 36 hours⁽⁷⁾. Generally, spontaneous spinal hematomas usually occur in the patients with coagulopathy or hematologic abnormalities⁽⁶⁾. Spinal hematoma following thrombolysis treatment is very rare and is usually caused by the treatment of myocardial infarction⁽⁸⁾ because the dosage of rt-PA in this condition is higher than that in acute ischemic stroke and intravenous heparin is frequently used. The combination of rt-PA and heparin may increase the risk of spinal hematoma⁽⁹⁾. Some reports showed occurrence of spinal hematoma following thrombolytic therapy for acute ischemic stroke. All of them were spinal epidural or subdural hematomas^(3,4,10). To the best of our knowledge, this case may be the first report of spontaneous spinal subarachnoid hematoma after intravenous rt-PA treatment for acute ischemic stroke.

Common presenting symptoms of spinal hematoma are pain and progressive paraparesis or quadriparesis⁽³⁻⁶⁾. Pain can be located in spinal areas or in remote areas, such as the shoulder. Some patients with minor trauma after stroke may increase risk of spinal hematoma⁽³⁾.

Management of any form of spinal hematoma depends on patient's neurological status. In cases with

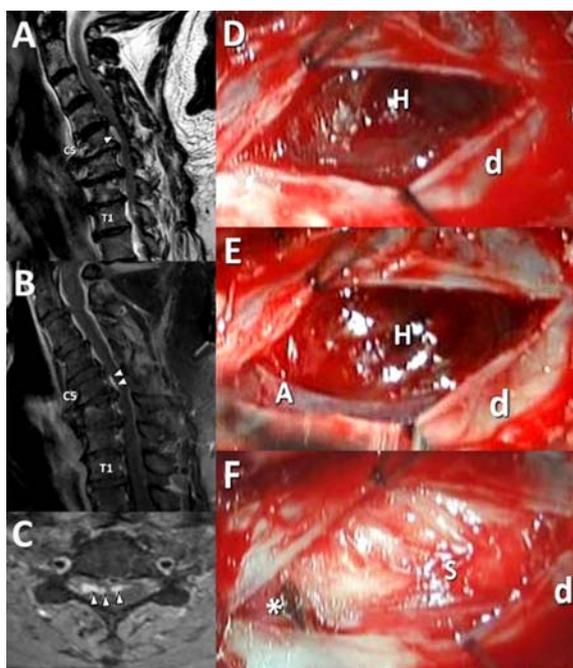


Fig.1 A) Sagittal MRI T2W showing hypersignal intensity lesion at the C5 vertebral level with multiple levels of spinal stenosis and cervical kyphosis. B, C) MRI after gadolinium injection showing vascular-like enhancement at the posterior border of lesion (arrowheads). D) Microsurgical view after opening the dura mater (d) showing hematoma (H) under thin layer of arachnoid membrane (A). E) After opening arachnoid membrane (A), hematoma (H) was clearly seen. F) After complete removal of hematoma, spinal cord (S) and small vessel incorporated in hematoma (asterisk) are found.

minor or no neurological deficit, conservative treatment is the treatment of choice. In contrast, if patients develop severe neurological deficit caused by spinal cord compression, surgical decompression must be done as soon as possible. Kreppel et al reported that patients who were treated within 12 hours of the onset had the best recovery rate⁽⁶⁾.

Regarding surgical management in the present patient, we preferred anterior corpectomy with hematoma evacuation because he had coexisting cervical kyphosis. If we performed surgery by posterior approach, it may worsen kyphotic curve of the cervical spine and we can correct cervical kyphosis by anterior approach. Another reason is that the hematoma may have some attachment with blood vessels visualized on pre-operative spinal MRI and bleeding can be stopped easily by anterior approach, but it is very difficult to stop bleeding by posterior approach.

Our patient had high risk for development of spinal hematoma because he had received both intravenous rt-PA and heparin to treat his cardiac condition. However, the definite cause of hematoma is still cryptic because spinal angiography and pathological examination of hematoma showed no unidentifiable pathology. One possible cause which can elucidate the occurrence of spinal subarachnoid hematoma is thrombolytic therapy for acute ischemic stroke by using rt-PA.

Conclusion

We reported a case of spontaneous spinal subarachnoid hematoma as an unusual complication of intravenous rt-PA treatment for acute ischemic stroke. Physicians must realize this condition when patients have spinal pain or progressive neurological deficit after receiving intravenous rt-PA.

What is already known on this topic?

Spontaneous spinal hematoma after intravenous administration of rt-PA is a rare complication. The most common presenting symptom is neck pain or back pain and progressive weakness. Surgery is the treatment of choice in patients with weakness and must be done as soon as possible.

What this study adds?

This case may probably be the first case report

of spontaneous spinal subarachnoid hematoma after intravenous administration of rt-PA for acute ischemic stroke. Favorable outcome can be achieved by surgical decompression.

Potential conflicts of interest

None.

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ภาวะเลือดออกใต้เยื่อหุ้มไขสันหลังชั้นอะแรคนอยด์ในผู้ป่วยโรคหลอดเลือดสมองอุดตันที่ได้รับการฉีดยาละลายลิ่มเลือดทางหลอดเลือดดำ: รายงานผู้ป่วย 1 ราย

หลักชัย พลวิจิตร, อาศิระ อิวาตะ, ทาดาฮาดะ มาซาฮิโกะ

ผู้ป่วยชายไทย อายุ 83 ปี มีอาการของหลอดเลือดสมองอุดตันเฉียบพลันและอ่อนแรงของแขนขาซ้าย ผู้ป่วยได้รับการรักษาโดยการให้ยาละลายลิ่มเลือด 5 ชั่วโมงต่อมาผู้ป่วยมีอาการขาอ่อนแรงทั้งสองข้างและระบบประสาทรับความรู้สึกเสียตั้งแต่ระดับ C6 dermatome ลงไป ภาพการตรวจแม่เหล็กไฟฟ้าของกระดูกสันหลังระดับคอ พบเลือดออกใต้เยื่อหุ้มไขสันหลังชั้นอะแรคนอยด์ตรงกับกระดูกสันหลังส่วนคอระดับที่ 5 ผู้ป่วยได้รับการรักษาโดยการผ่าตัดกระดูกสันหลังระดับคอและเอาลิ่มเลือดออก หลังการผ่าตัดรักษา 6 เดือน ผู้ป่วยมีการฟื้นตัวได้ดีสามารถเดินได้ในระยะทางสั้นๆ ภาวะเลือดออกใต้เยื่อหุ้มไขสันหลังชั้นอะแรคนอยด์เป็นภาวะแทรกซ้อนที่พบได้น้อยมากหลังการรักษาโรคหลอดเลือดสมองอุดตันเฉียบพลันด้วยการให้ยาละลายลิ่มเลือดทางหลอดเลือดดำ แพทย์ผู้รักษาควรคิดถึงภาวะนี้ในผู้ป่วยโรคหลอดเลือดสมองอุดตันที่ได้รับยาละลายลิ่มเลือดทางหลอดเลือดดำและมีอาการปวดกระดูกสันหลังหรือมีอาการอ่อนแรงของแขนขาภายหลังได้รับยา
