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Case Report

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Severe Headache and Hypertension due to Cerebral Venous Sinus Thrombosis in a Patient With Cervical Spinal Cord Injury: A Case Report

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Conflict of Interest
The authors have no financial conflicts of interest.

ABSTRACT

Autonomic dysreflexia is suspected when patients with spinal cord injury report headaches and hypertension. A 52-year-old man was diagnosed with C5-C6-C7 cervical spinal cord injury, traumatic subdural hemorrhage, intracerebral hemorrhage, and skull fracture. The patient underwent surgery at another hospital. The patient was hospitalized for comprehensive rehabilitation after 7 months. The assessment revealed an American Spinal Cord Injury Association Impairment Scale grade A at the C7 level due to complete spinal cord damage. Evaluation of muscle weakness that occurred after experiencing severe headache and hypertension revealed an intracerebral hemorrhage caused by cerebral venous sinus thrombosis. The patient showed improvement in muscle strength over time and was monitored for warfarin administration. Furthermore, cerebral venous sinus thrombosis should be considered as a differential diagnosis when patients with spinal cord injury who have experienced polytrauma complain of headache and hypertension, as they share clinical symptoms with autonomic dysreflexia. Additional evaluations, such as imaging examinations, should be conducted, as necessary.

Keywords: Autonomic dysreflexia; Spinal cord injury; Thrombosis; Intracerebral hemorrhage

INTRODUCTION

Autonomic dysreflexia is considered a common cause of headaches and high blood pressure in patients with cervical spinal cord injury.[3,12] However, since cerebral vein thrombosis also presents with headache and hypertension as clinical symptoms, these should be considered in the differential diagnosis.[1,2]

Cerebral vein thrombosis is a rare cerebrovascular disease, and its common risk factors include contraceptive pill use, infections, and head injuries. Its symptoms and clinical outcomes vary depending on the affected area. Therefore, the duration from onset of symptoms to diagnosis commonly involves several days.[1,2]

Cases of cerebral vein thrombosis, not autonomic dysreflexia, in patients with multiple injuries, especially those with head injury, are uncommon. Herein, we report a case of cerebral vein

thrombosis in a patient with cervical spinal cord injury who experienced high blood pressure and severe headache. We suggest that cervical spinal cord injury should be included in the differential diagnosis of autonomic dysreflexia, as they share the same clinical symptoms.

CASE REPORT

A 52-year-old man who was living independently and was able to walk unassisted was brought to the emergency room of another hospital on April 11, 2020, after falling from 6 m of height. He had no other medical underlying medical conditions, except for high blood pressure. He was diagnosed with C5-C6-C7 cervical spinal cord injury (**FIGURE 1**), T4 burst fracture, traumatic subdural hemorrhage (**FIGURE 2**), intracerebral hemorrhage, skull fracture, and left tibia fracture. He underwent open reduction internal fixation for left tibia fracture and posterior interbody fusion, T2-3-5-6 for T4 burst fracture. For brain hemorrhage, intracranial pressure (ICP) control and conservative treatment were performed.

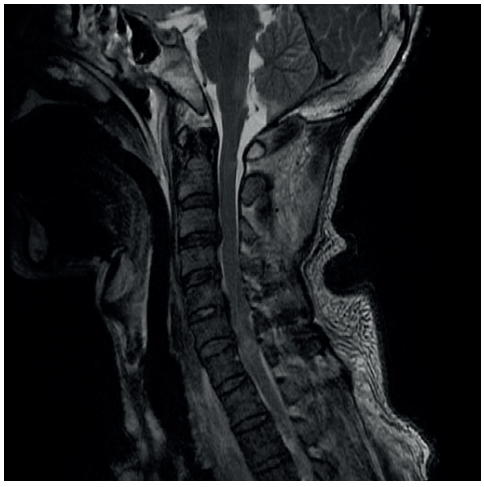

FIGURE 1. Cervical spine magnetic resonance imaging findings on the day of the injury.

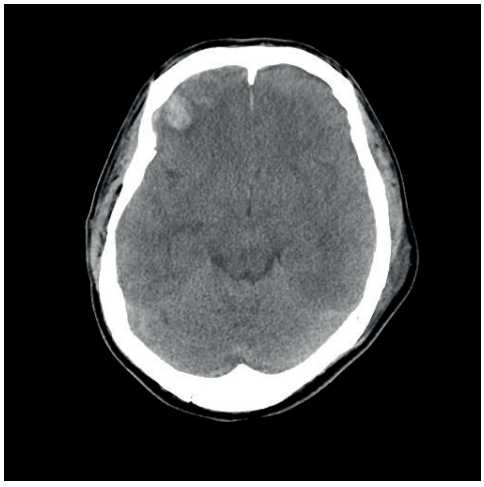

FIGURE 2. Brain computed tomography findings on the day of the injury.

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He was hospitalized for comprehensive rehabilitation on November 24, 2020, 7 months after the injury. At the time of hospitalization, assessment revealed American Spinal Cord Injury Association Impairment Scale grade A at the C7 level due to complete damage of the spinal cord. Additionally, he had an indwelling urethral catheter. He consistently complained of headaches, including those involving the posterior neck, since he was hospitalized, with a visual analog scale score of 7 points. He reported having persistent headache after the trauma in April and was undergoing conservative care for the pain after being diagnosed with cerebral hemorrhage at another hospital. Additionally, he took rivaroxaban 20 mg per day to prevent deep vein thrombosis.

The patient had autonomic dysreflexia, a complication of cervical spinal cord injury that causes a rapid increase in blood pressure. Consequently, he was placed under observation, with a focus on rapid increases in blood pressure and patency of the indwelling catheter.

At approximately 3 AM on the fifth day of hospitalization, he complained of a severe headache and eye pain, "as if his eyeballs were coming out", with a visual analog scale score of 9 points. His blood pressure was 200/100 mmHg. The indwelling urethral catheter was intact and patent. Blood pressure was controlled with antihypertensive medication (5 mg of amlodipine) administered within 1 hour of the increase in blood pressure, which subsequently decreased to 110/70 mmHg after 2 hours. There was no notable muscle weakness in the manual muscle test conducted at 7 AM. A progress observation was made since his headache had improved. At that time, the patient's laboratory findings were normal.

Two days later, on the seventh day of hospitalization, the manual muscle test confirmed that the arm extensors and wrist flexor and extensor muscles had decreased from level 5 to 2. Brain computed tomography (CT) revealed an intracerebral hemorrhage of 4.8 cm in the right parietal lobe. After consulting the neurosurgery department, mannitol (20% mannitol, 150 mL, 4 times per day) was added to the prescription. The treatment was completed with emphasis on blood pressure control, autonomic dysreflexia, urination, and defecation.

The examination conducted in consultation with a neurosurgeon and neurologist showed that the cerebral hemorrhage (**FIGURE 3**) was different from the hemorrhagic transformation caused by general cerebral infarction. Considering the possibility of hemorrhagic changes caused by cerebral vein thrombosis, brain and venae cerebri magnetic resonance imaging was performed on the 10th day of hospitalization (**FIGURE 4**). Treatment with low-molecular-weight heparin was initiated as the patient was diagnosed with dural sinus thrombosis. His condition improved, as indicated by the reduced bleeding on brain CT. Subsequently, trace brain CT confirmed the reduction in bleeding. Treatment was continued by changing the drug treatment to warfarin, with a starting dose of 4 mg per day.

In the manual muscle test of the left upper limb conducted on the 30th day of hospitalization, the arm extensor and wrist flexor and extensor muscles improved from level 2 to 4. Additionally, his headaches improved and blood pressure remained normal.

In cerebral magnetic resonance imaging performed 2 and 3 months later (**FIGURES 5** & **6**), venous blood flow was not observed in the left venous sinus, jugular vein, and internal jugular vein; therefore, low-molecular-weight heparin was administered for an extended period.

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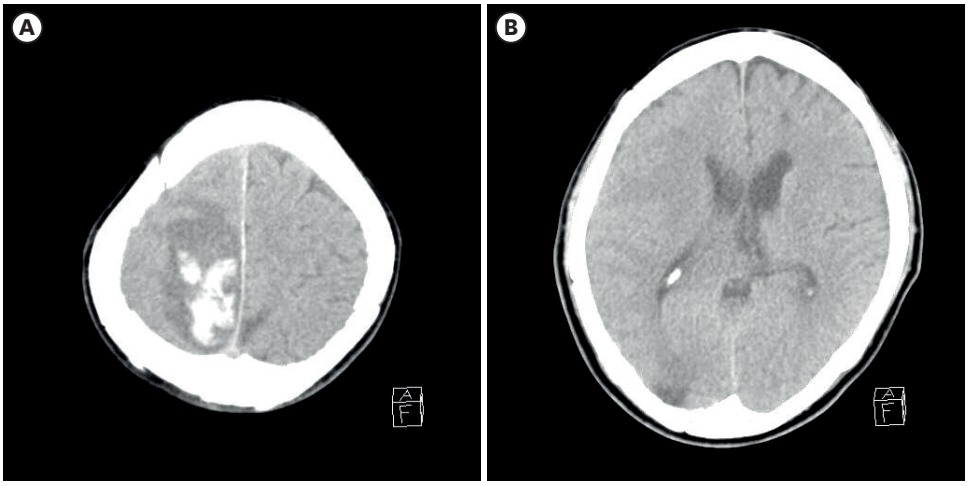

FIGURE 3. After symptoms develop, initial brain computed tomography findings of the patient.
(A) New appearance of ICH (about 4.8cm in size) with surrounding edema in the right parietal lobe. (B) Transverse sinus view.

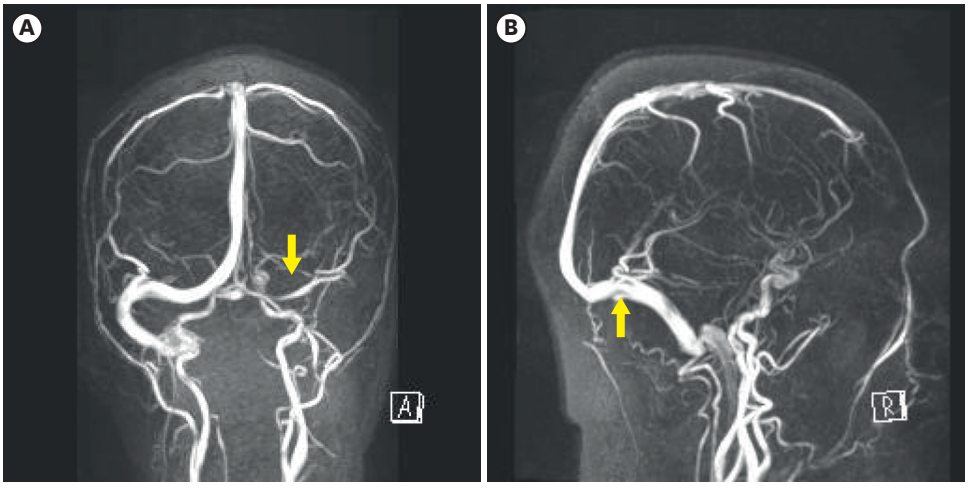

FIGURE 4. Magnetic resonance venogram of the patient.
(A) Transverse image of MR venogram of the patient. Left transverse sinus stenosis, non-visualization, left sigmoid sinus, jugular bulb, internal jugular vein, and dural sinus thrombosis. (B) Sagittal image showing a left transverse sinus stenosis.

DISCUSSION

Upon observing symptoms such as paroxysmal high blood pressure and severe headache in patients with spinal cord injury at the T6 or higher level in the thoracic vertebrae, autonomic dysreflexia should be suspected. In healthy individuals, maximal parasympathetic vasodilation occurs when the sympathetic nervous system is exaggerated, correcting the increased sympathetic tone. However, in patients with spinal cord injury, the parasympathetic corrective response is unable to move below the level of spinal cord injury, resulting in sustained vasoconstriction and eventually in hypertension. Autonomic dysreflexia is characterized by a rapid increase in blood pressure caused by sudden asthenia in the sympathetic nervous system due to irritation in the bladder or intestines.[10,14-16] It is commonly reported in 48–90% of patients with spinal cord injury and paraplegia with a high

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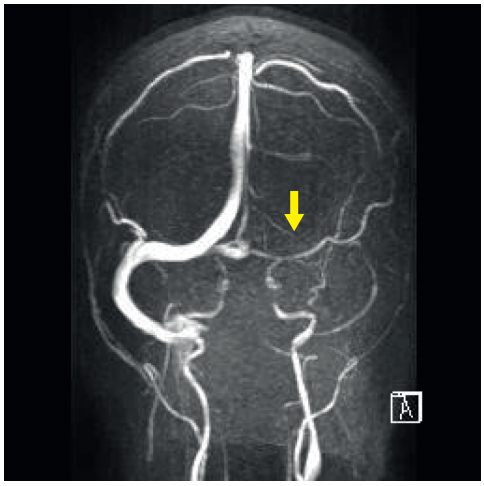

FIGURE 5. Follow-up magnetic resonance venogram after 2 months. Venous flow was absent in the left sigmoid sinus, jugular bulb, and internal jugular vein.

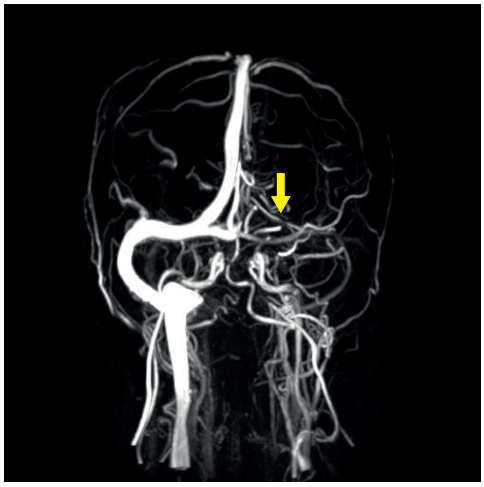

FIGURE 6. Follow-up magnetic resonance venogram after 3 months. No significant change is noted compared with the previous magnetic resonance venogram.

level of neurological damage. Our patient was also suspected of having autonomic dysreflexia due to high blood pressure. Consequently, cerebral hemorrhage was confirmed due to cerebral vein thrombosis.

Cerebral vein thrombosis is nonspecific because of blood clots occurring in the cerebral venous sinus and deep cerebral veins, and it manifests as varied symptoms, wherein clinical progress is difficult to predict. Headache is one of the most common symptoms of cerebral vein thrombosis, observed in nearly 31–91% of patients. Cerebral venous sinus thrombosis is related to genetic factors, blood clotting, contraceptive pill use, infections, or head injuries.[1,15] It can also cause papilledema and various neurological symptoms due to increased cerebral pressure, leading to hemorrhagic changes and cerebral edema. Patients with a cerebral infarction that does not fit in the branch area of the cerebral artery should be suspected of having cerebral vein thrombosis. Appropriate tests, such as magnetic resonance imaging combined with magnetic resonance venography,[7] must be conducted immediately since nonspecific symptoms such as headaches may not be diagnosed immediately.

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For diagnosis, imaging should be done along with the patient's clinical symptoms. The manifestations are variable but include severe headache, blurred vision, fainting or loss of consciousness, loss of control over movement in part of the body, seizures, and coma. No laboratory test can rule out cerebral venous thrombosis (CVT).

Imaging diagnosis can also be made by cerebral angiography or magnetic resonance venography to distinguish it from common cerebral infarction. In brain CT, if multiple sites of bleeding are observed, if the bleeding is in the subcortical area, or if the area where intracerebral hemorrhage does not fit the artery area and the boundaries are unclear, venous infarction caused by cerebral vein thrombosis should be suspected. Although it is true that CT plays an important role in neurologic emergency, magnetic resonance venogram is thought to be the best imaging technique for diagnosis of CVT.

Treatment includes anticoagulation, urokinase, and recombinant tissue plasminogen activator because the venous circulation must be opened by removing blood clots that block the vein. Patients can be treated with an anticoagulation method using low-molecular-weight heparin, which has been used for years to treat cerebral vein thrombosis. After acute anticoagulation treatment, long-term anticoagulation treatment is required for 3–6 months. In some cases, longer administration may be required. Endovascular therapy aims to reduce the burden of blood clots by administering fibrinolytic agents topically or by removing them mechanically. Increased ICP requires a neurologically important unit, and medical therapies for elevated ICP include osmotic therapy, hyperventilation, and bed head elevation. In the case of seizures, antiepileptic drugs are given to those with clinical evidence of seizures.

In this report, the patient was thought to have developed cerebral vein thrombosis due to a hypercoagulable state in the body because of extensive brain damage at the time of trauma. [4-6,8,9]

Cerebral vein thrombosis treatment requires the use of anticoagulants even in the case of intracerebral hemorrhage.[11,16] Thus, the present patient was administered nadroparin 2,850 unit/0.3 mL twice a day for 10 days, which was then changed to warfarin with a target prothrombin time of 2–3 levels. According to a recent study, direct oral anticoagulant (DOAC) agent such as apixaban, dabigatran, rivaroxaban, and edoxaban have been added as options for prevention and treatment of CVT. In patients with spinal cord injury, we consider that the administration of DOACs to patients with high-risk factors for CVT as one of the options.[13]

CONCLUSION

Herein, we report a rare case of hemorrhagic changes caused by cerebral vein thrombosis in a patient with cervical spinal cord injury, with a history of multiple injuries, headache, and elevated blood pressure. This case highlights the importance of performing additional evaluations such as imaging examination, considering that cerebral vein thrombosis as well as autonomic dysreflexia occur when a patient with multiple injuries, especially head injury, has a headache and elevated blood pressure.

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REFERENCES

1. Capecchi M, Abbattista M, Martinelli I. Cerebral venous sinus thrombosis. **J Thromb Haemost 16**:1918-1931, 2018
PUBMED | CROSSREF
2. Caplan LR. Cerebral venous thrombosis in Caplan LR (ed): Caplan's stroke, ed 4. Philadelphia, PA: Saunders Elsevier, pp554–577, 2009
3. Curt A, Nitsche B, Rodic B, Schurch B, Dietz V. Assessment of autonomic dysreflexia in patients with spinal cord injury. **J Neurol Neurosurg Psychiatry 62**:473-477, 1997
PUBMED | CROSSREF
4. Delgado Almandoz JE, Kelly HR, Schaefer PW, Lev MH, Gonzalez RG, Romero JM. Prevalence of traumatic dural venous sinus thrombosis in high-risk acute blunt head trauma patients evaluated with multidetector CT venography. **Radiology 255**:570-577, 2010
PUBMED | CROSSREF
5. Dobbs TD, Barber ZE, Squier WL, Green AL. Cerebral venous sinus thrombosis complicating traumatic head injury. **J Clin Neurosci 19**:1058-1059, 2012
PUBMED | CROSSREF
6. Ferrera PC, Pauze DR, Chan L. Sagittal sinus thrombosis after closed head injury. **Am J Emerg Med 16**:382-385, 1998
PUBMED | CROSSREF
7. Ferro JM, Canhão P. Cerebral venous sinus thrombosis: update on diagnosis and management. **Curr Cardiol Rep 16**:523, 2014
PUBMED | CROSSREF
8. Gosk-Bierska I, Wysokinski W, Brown RD Jr, Karnicki K, Grill D, Wiste H, et al. Cerebral venous sinus thrombosis: incidence of venous thrombosis recurrence and survival. **Neurology 67**:814-819, 2006
PUBMED | CROSSREF
9. Hsu PJ, Lee CW, Tang SC, Jeng JS. Pearls & Oy-sters: Delayed traumatic intracerebral hemorrhage caused by cerebral venous sinus thrombosis. **Neurology 83**:e135-e137, 2014
PUBMED | CROSSREF
10. Karlsson AK. Autonomic dysreflexia. **Spinal Cord 37**:383-391, 1999
PUBMED | CROSSREF
11. Krassioukov A. Autonomic function following cervical spinal cord injury. **Respir Physiol Neurobiol 169**:157-164, 2009
PUBMED | CROSSREF
12. Krassioukov A, Warburton DE, Teasell R, Eng JJ; Spinal Cord Injury Rehabilitation Evidence Research Team. A systematic review of the management of autonomic dysreflexia after spinal cord injury. **Arch Phys Med Rehabil 90**:682-695, 2009
PUBMED | CROSSREF
13. Lurkin A, Derex L, Fambrini A, Bertoletti L, Epinat M, Mismetti P, et al. Direct oral anticoagulants for the treatment of cerebral venous thrombosis. **Cerebrovasc Dis 48**:32-37, 2019
PUBMED | CROSSREF
14. Mathias CJ, Frankel HL. Cardiovascular control in spinal man. **Annu Rev Physiol 50**:577-592, 1988
PUBMED | CROSSREF
15. Sébire G, Tabarki B, Saunders DE, Leroy I, Liesner R, Saint-Martin C, et al. Cerebral venous sinus thrombosis in children: risk factors, presentation, diagnosis and outcome. **Brain 128**:477-489, 2005
PUBMED | CROSSREF
16. Wan D, Krassioukov AV. Life-threatening outcomes associated with autonomic dysreflexia: a clinical review. **J Spinal Cord Med 37**:2-10, 2014
PUBMED | CROSSREF

https://doi.org/10.13004/kjnt.2022.18.e33