Trauma Case Reports 25 (2020) 100269

Contents lists available at ScienceDirect

Trauma Case Reports

journal homepage: www.elsevier.com/locate/tcr

Case Report

Spinal infarction caused by hypovolemic shock following massive bleeding from stab wounds to the neck

Tomoyoshi Shibata[a,*], Hidehiko Yoshimatsu[a], Tomoyuki Yano[a], Ryo Karakawa[a], Shinsuke Ishikawa[b], Akira Takeda[b]

[a] Cancer Institute Hospital of Japanese Foundation For Cancer Research, 3-8-31 Ariake, Koto-ku, Tokyo, Japan
[b] Kitasato University Hospital, 1-15-1 kitasato, Minami-ku, Sagamihara-shi, Kanagawa, Japan

ARTICLE INFO

Keywords:
Spinal cord infarction
Massive hemorrhage
Hypovolemic shock
Incise wound

ABSTRACT

A 59-year-old female was brought to our emergency department with hypovolemic shock caused by massive bleeding from neck stab wounds inflicted by herself in a committed suicide. The patient complained of comparatively strong pain on her lower back and there was sensory and motor disturbance of bilateral lower limbs, but there was no trauma on the lumber region, the spine, or the vertebrae. After hemostasis, we performed magnetic resonance imaging, which demonstrated high intensity signal in the spinal and longitudinal area from the Th8 to the conus medullaris, and at center of the frontal horn on the upper thoracic spinal cord (owl's eye appearance) on T2 weighted images. This case was diagnosed as spinal infarction caused by low blood pressure as a result of massive bleeding. The basis of diagnosis were as follows: 1) an acute onset; 2) when the ambulance arrived, she was in hypovolemic shock caused by massive hemorrhage; 3) there was no trauma on the lumber region, the spine, or the vertebrae; 4) with CT taken on admission, aortic disease was not detected; and 5) she was not on any antipsychotic drugs which could cause thrombosis. We treated the patient following management protocol of cerebral infarction, but recovery of sensory and motor disorders was minimal.

Introduction

We experienced a spinal cord infarction (SCI) case caused by massive bleeding from stab wounds in the neck.

Case report

A 59-year-old female was brought to our emergency department with hypovolemic shock caused by massive bleeding from neck stab wounds (Fig. 1). Her husband reported that she had been working in their garden, but after half an hour, he found her lying supine with extensive bleeding.

When the emergency services arrived at 14:17, she was found in a supine position with a 50 × 60 cm bloodstain mark on her shirt. Her pulses were undetectable, heart rate was is 90 times per minute, and the diameters of the pupils were both 2 mm with no light reflexes. Glasgow Coma Scale was E2V1M3. Ambulance team administered oxygen to the patient and she was transferred to our hospital at 15:00. She had a past history of depression and anxiety disorder with the following medications: alprazolam 0.4 mg 3T/Day, clonazepam 1 mg 3T/Day, Lorazepam 0.5 mg 3T/Day, fluvastatin sodium 30 mg 1T/Day, betahistine mesylate 6 mg 3T/Day,

* Corresponding author.
E-mail address: tomoyoshi.shibata@jfcr.or.jp (T. Shibata).

https://doi.org/10.1016/j.tcr.2019.100269
Accepted 30 November 2019
Available online 18 December 2019

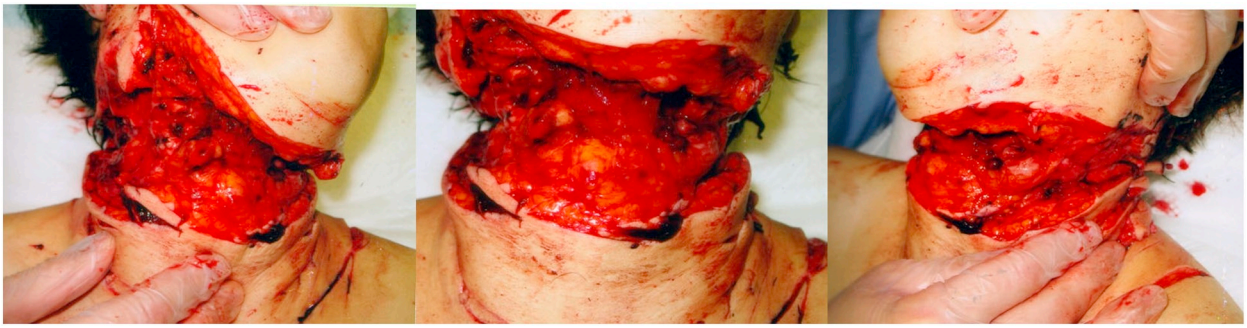

Fig. 1. Many incision wounds were seen on the neck of the patient.

nizatidine 150 mg 2 T/Day. On admission, primary survey was performed. There was nothing noteworthy regarding airway and breathing. Respiratory rate was 26 times per min. Systolic blood pressure was 72 mm Hg, but diastolic blood pressure was undetectable. Heart rate was 90 times per minute. There were many incised wounds in the neck (Fig. 1) and incise wounds on her left wrist. The hypoglossal nerve, the recurrent laryngeal nerve, the phrenic nerve and the vagus nerve appeared intact. The chest X-ray findings were within normal limits. No damage to the aorta, the esophagus, and the thoracic duct was found with computed tomography (CT). Brain CT showed nothing noteworthy. Hemoglobin level decreased from 11.7 g/dl to 8.1 g/dl an hour after admission.

We decided to do operation for hemostasis, but movement disorder of bilateral lower limbs was noted upon removal of the neck collar. And she expressed strong pain sited unclearly on her lower back. We examined the site of pain, which was somewhere around Th10. She had no past problems and at this time she did not experience any trauma to her lower back. In neurological examination, all Manual Muscle Tests (MMTs) were 1 in the right lower limb (quadriceps muscle, hamstrings, tibialis anterior muscle, gastrocnemius muscle, and soleus muscle), and all MMTs were 0 in the left lower limb. There was superficial sensory disturbance, which was more severe on the left side than the right side, in regions below the waist. Patella tendon and Achilles tendon reflex of the bilateral lower limbs were diminished, and there was no anal reflex. There were no abnormal reflexes, and deep sensory and positioning sensory were normal. After we confirmed that the lumbar spine was normal on X-ray, we began operating on the incised wounds under general anesthesia. The following intraoperative findings were observed: incised wounds reached the sternocleidomastoid muscle. The external jugular veins were completely severed on both sides; a 3 mm hole was seen on the right internal jugular vein. The external jugular veins were ligated, and the internal jugular vein was repaired. And ruptured tendons of palmaris longs and flexor carpi radialis of left wrist were repaired. The next morning, there was no change concerning neurological observations. Magnetic resonance imaging (MRI) revealed high intensity signal in spinal and longitudinal area from Th8 to the conus medullaris, and at center of the frontal horn on the upper thoracic spinal cord (owl's eye appearance) on T2 weighted images (Fig. 2). We

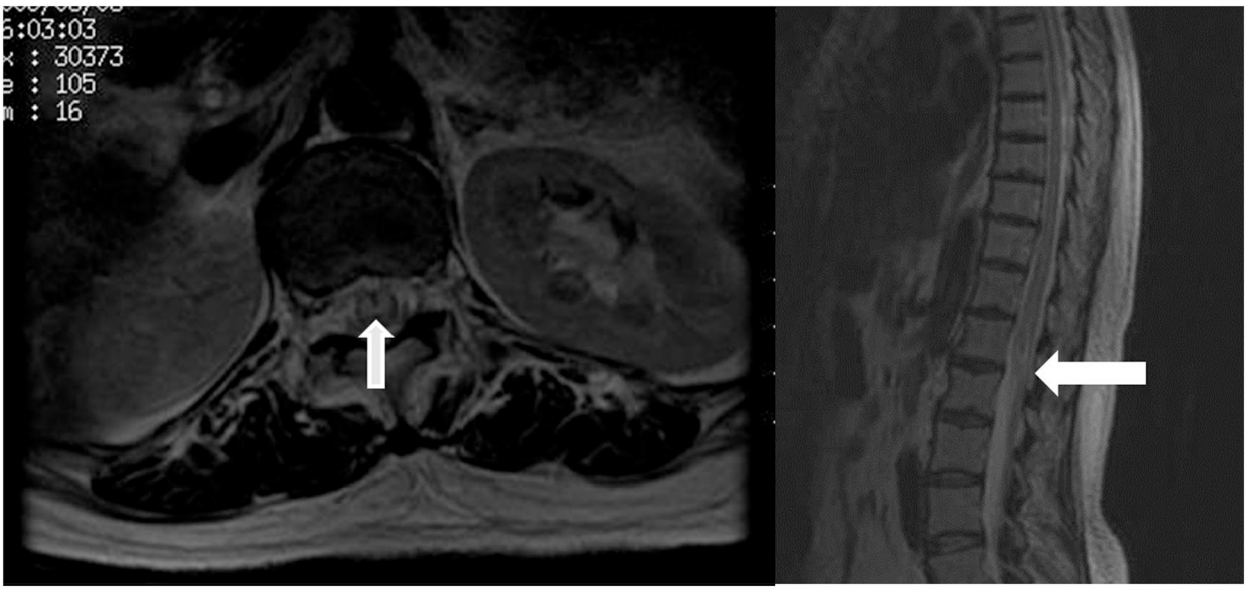

Fig. 2. MRI images taken at the time of admission with a 1.5 T clinical scanner (SIGNA LX; GE Healthcare, Waukesha, WI, USA). (Left) Axial plane T2-weighted fast spin echo imaging. (Right) Sagittal plane T2-weighted fast spin echo imaging. MRI images showed intensified signal in the intraspinal, longitudinal regions from Th 8 to the conus medullaris (white arrows).

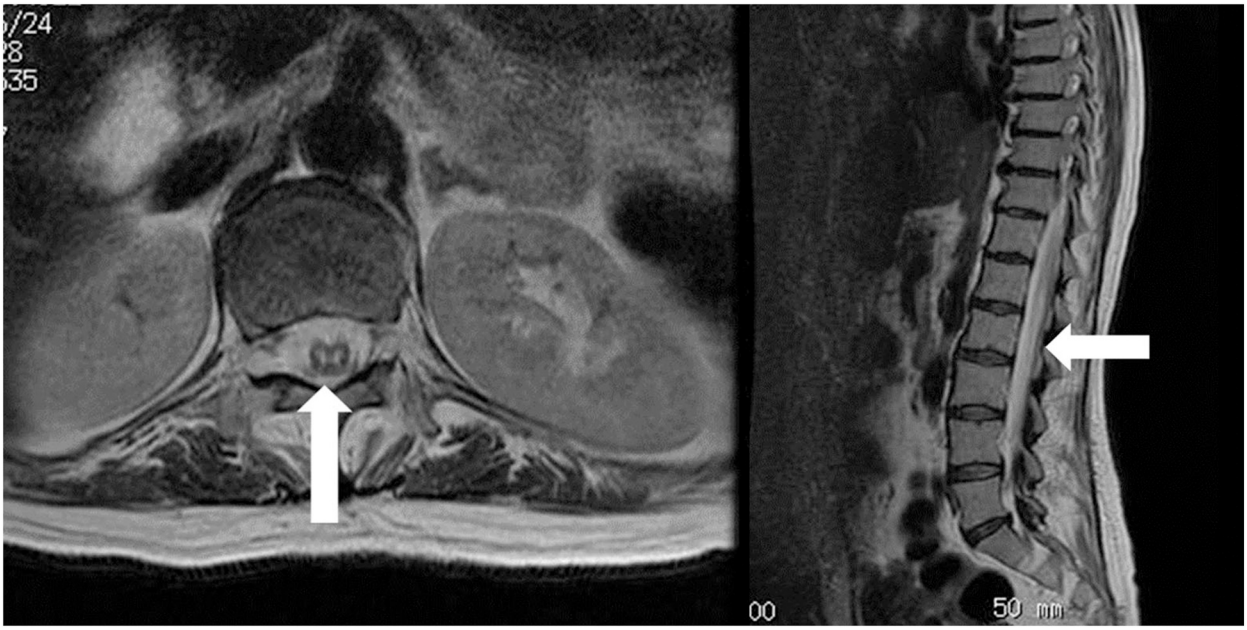

Fig. 3. MRI images taken 14 days after admission under the same protocol as in Fig. 2. No changes were noted.

diagnosed the patient with SCI due to ischemia of the Adam-Kiewicz artery, considering the site of the injury. We started administration of edaravone 30 mg × 2/Day and concentrated glycerin, fructose 500 ml/Day. The general condition stabilized, but paraplegia and dissociated sensory loss remained. Fourteen days after the injury, we performed another MRI, but we could not find any changes (Fig. 3). After 1 year, there was no significant recovery of the superficial sensory systems. Regarding movement disorder, almost all MMT test results of the left lower limb were 0/5 except for the quadriceps muscle, which was 2. All MMT test results of the right lower limb were 2 except for the quadriceps muscle, which was 4. She remained in a sitting position in a wheelchair with trunk support, and she needed assistance in transferring to a wheelchair.

Discussion

SCI is a very rare disease [1]. The causes usually include traumatic aortic injury, dissecting aortic aneurysm, and blockage of blood flow at the time of aortic surgery [2]. SCI occurs most frequently at the cervico-thoracic junction and the lower thoracic region (Adamkeiwicz artery) of the spine. Infarction of the vessel mainly occurs in the frontal spinal artery, which perfuse the frontal 2/3 of the cross-spine.

SCI's diagnosis is made clinically. Usefulness of diagnosis of MRI is relatively low [3]. MRI should be used only to rule out compression spinal disorders [4]; swelling of the spine can be detected, but it is impossible to point out the cause of the swelling.

Identifying the cause of SCI can be very difficult. However, the diagnosis of pathology of spinal vessel disorders is relatively easy considering the follow points [5,6].

1) It has a sudden onset and complaint.
2) The location of pain always reflects the location of spinal cord lesion.
3) Brown-Séquard syndrome is a specific symptom that cannot be seen in lesions other than the spinal cord.
4) Sensory and motor disorder may develop on the same side as a result of mottle damaged spine.

Barrera et al. [7] reported that the severity of early neurological symptoms correlates highly with prognosis of neurological symptoms.

On neurological examination, the lower limbs were flaccid with loss of motor power. Sensory appreciation was impaired to the modalities of touch and pain below the Th8. Posterior column function was preserved. There was no bladder-anal function. MRI T2 weighted images at 24 h after the admission showed high intensity signal in spinal and longitudinal area of from the Th8 to the conus medullaris, and at center of the frontal horn on the upper thoracic spinal cord (owl's eye appearance) [10]. With these findings, the diagnosis of SCI was made. We diagnosed this case as SCI caused by low blood pressure as a result of massive bleeding. The basis of diagnosis was as follows: 1) she was in hypovolemic shock caused by massive hemorrhage; 2) there was no trauma on the lumber region; 3) in CT-examination on admission, aortic disease was not detected; 4) she had not taken any antipsychotic drugs which could cause thrombosis. Monterio L. [8] pointed out that low blood pressure can cause SCI. Lynch et al. [9] reported that a mean blood pressure under 55 mm Hg (equal to the limit of experimental auto-regulation, 50 mm Hg) is a risk of ischemia of the central nervous system.

Because this case was not high-energy trauma and had obvious incised wounds, it took time for us to note paralysis of the lower body. It was found before surgical intervention, which suggested the possibility of SCI. For early diagnosis and treatment, and for avoiding post-diagnostic troubles, a thorough physical examination should always be conducted upon arrival of patients.

Conclusion

We experienced a case where SCI was caused by low blood pressure after massive bleeding.

References

[1] J. Novy, et al., Spinal cord ischemia, Arch. Neurol. 63 (2006) 1113–1120.
[2] S. Toshitaka, H. Kazutoshi, Minoru Akino: spinal cord infarction due to aortic dissection, Spinal and Spinal Cord 21 (2008) 1015–1020.
[3] Makoto Sugiura, Tetsuo Ando, Spinal cord infarction associated with vertebral body infarction: a report of two cases, Spinal and Spinal Cord 21 (10) (2008) 1008–1014.
[4] J. Faig, O. Busse, R. Salbeck, Vertebral body infarction as a confirmatory sign of spinal cord ischemic stroke. Report of three cases and review of the literature, Stroke 29 (1998) 239–243.
[5] I. Kiyoharu, Neurological signs and differential diagnosis, Spinal and Spinal Cord 21 (10) (2008) 982–991.
[6] Yanagi Tsutomu, Tetsuo Ando, Anterior spinal artery syndrome, Spinal and Spinal Cord 6 (1) (1993) 21–28.
[7] S. Salvador de la Barrera, A. Bareca-Buyo, A. Montoto-Marques, et al., Spinal cord infarction: prognosis and recovery in a series of 36 patients, Spinal Cord 39 (2001) 520–525.
[8] L. Monteiro, I. Leite, et al., Spontaneous thoracolumbar spinal cord infarction: report of six cases, Acta Neurol. Scand. 82 (1992) 563–566.
[9] D.R. Lynch, T.M. Dawson, E.C. Raps, et al., Risk factors for the neurologic complications associated with aortic aneurysms, Arch. Neurol. 49 (1992) 284–288.
[10] Shin-ichi Kan, Imaging of infarction of conus medullaris, Neurol. Med. 49 (July 1998) 18–25.