 doi: 10.2176/jns-nmc.2023-0275

NMC Case Report Journal 11, 61-67, 2024

Subarachnoid Hemorrhage Confirmed by Magnetic Resonance Imaging in a Patient with Brain Death owing to Hypoxic Encephalopathy Following Suicide by Hanging

Sotaro OSHIDA,[1,2] Tomoki YOKOSAWA,[3] Shizuka ARAYA,[2] Shinpei SATO,[2] Taro SUZUKI,[2] Yosuke AKAMATSU,[1] and Kuniaki OGASAWARA[1]

[1]*Department of Neurosurgery, Iwate Medical University, Yahaba, Japan*
[2]*Department of Neurosurgery, Iwate Prefectural Ofunato Hospital, Ofunato, Japan*
[3]*Emergency and Critical Care Center, Iwate Prefectural Ofunato Hospital, Ofunato, Japan*

Abstract

Although true subarachnoid hemorrhage (SAH) is an atypical complication owing to suicide by hanging, pseudo-SAH can often develop because of hypoxic encephalopathy. Therefore, differentiating pseudo-SAH from true SAH using brain computed tomography (CT) is often challenging. In Japan, an individual's cause of brain death must be determined to be eligible for organ donation, regardless of whether true SAH is involved or not. Herein, we report a case of SAH confirmed by magnetic resonance imaging (MRI) in a patient with brain death owing to hypoxic encephalopathy following suicide by hanging. A 48-year-old man attempted suicide by hanging. Upon arrival at the hospital, he developed pulseless electrical activity with apnea. Although spontaneous circulation returned within a few minutes of his arrival, spontaneous breathing did not recover. The patient was in deep comatose state without response to pain stimulation, brainstem reflexes, or electrical activities on an electroencephalogram. Consequently, the patient met diagnostic criteria for clinical brain death based on the Japanese organ transplantation law. Brain CT revealed global hypoxic injury and high density in the basal cisterns and subarachnoid space. Brain MR T2*-weighted imaging revealed low intensity at the left Sylvian fissure underlying the hematoma. These findings indicated brain death owing to hypoxic encephalopathy following hanging, and incidental true SAH was confirmed by MRI. Donor surgery and organ transplantation were performed. Spontaneous SAH can often develop secondary to hanging, and brain MRI can effectively determine whether the cause of brain death involves true SAH.

Keywords: suicide by hanging, pseudo-subarachnoid hemorrhage, subarachnoid hemorrhage, magnetic resonance image, Japan

Introduction

Suicide by hanging has a higher lethality than other methods of suicide, such as self-poisoning and -mutilation.[1,2] One study reported that of 4,024 consenting organ donors in Australia and New Zealand, 226 died by suicide by hanging (5.6%).[3] Although the mechanism of death may differ depending on the method used, death by hanging usually results from cerebral ischemia caused by airway obstruction or vascular occlusion.[4] Hemorrhagic symptoms, such as subarachnoid hemorrhage (SAH), are rare in hanging, and a few such cases have been reported in patients following incomplete hanging.[5,6] Increased attenuation of the basal cisterns and subarachnoid spaces on computed tomography (CT) scans characterizes true SAH. However, pseudo-SAH is also a reported phenomenon in patients with hypoxic encephalopathy, in which increased attenuation in cortical sulci and within the major cisterns mimics the appearance of true SAH.[7-9] Pseudo-SAH is thought to be caused by difference in contrast because of decrease in signal due to brain hypoattenuation and increase in signal due to engorgement of the venous

Received November 16, 2023; Accepted December 22, 2023

structures. However, pseudo-SAH or true SAH may be challenging to diagnose using brain CT in brain death owing to suicide by hanging. In general clinical practice, whether the pathological condition of suicide by hanging is pseudo-SAH or true SAH is not a concern. However, an individual's cause of death must be determined to be eligible for organ donation, regardless of whether true SAH is involved or not.[10] Although brain magnetic resonance imaging (MRI) is effective for detecting bleeding lesions after a subacute period,[11] use of MRI to diagnose SAH in cases of brain death owing to suicide by hanging has not yet been reported. Herein, we report a case of SAH confirmed by gradient recalled echo (GRE) T2*-weighted imaging (T2*-WI) in a patient with brain death owing to hypoxic encephalopathy following suicide by hanging.

Case Report

A 48-year-old man with depression attempted suicide by hanging. His wife found him suspended from a beam with a rope in his garage. According to the statement made by his wife, he hung for approximately 5-8 minutes. He was limp and cyanotic without spontaneous movement. His wife immediately initiated cardiopulmonary resuscitation (CPR) after the man was taken down. He was transported to our hospital in an ambulance while undergoing CPR. Upon arrival at our hospital (approximately 45 minutes after his discovery), he had pulseless electrical activity with apnea. Although spontaneous circulation was recovered following intravenous injection of first dose (using the 1: 1000 strength ampoule) of adrenaline (1 mg/mL) a few minutes after his arrival, spontaneous breathing did not recover owing to severe brain damage. His consciousness remained at a Glasgow Coma Score of 3 even after the heartbeat resumed. Responses to deep pain stimulation were absent. His bilateral pupils were fixed and dilated to 7.5 mm. Pupillary light reflexes were absent. Moreover, brainstem reflexes, such as eyelash, cough, and oculocephalic reflexes, were absent. Electrical activities on electroencephalograms were absent. The CT scans were not obtained during the acute phase owing to his extremely unstable general condition. His general condition stabilized after about 2 weeks, but his brainstem reflexes and electroencephalograms remained absent. The patient met the criteria and was diagnosed as clinically brain dead based on the Japanese organ transplantation law. Organ transplantation was explained to and discussed with the donor's family. The donor's family requested that his organs be donated and provided informed consent for the same. All procedures were reviewed and approved by the ethics committee of Iwate Prefectural Ofunato Hospital and were performed in accordance with the ethical standards of the Declaration of Helsinki. In Japan, the cause of clinical brain death must be strictly determined based on radiological findings; therefore, he underwent a brain CT scan,[10] which was obtained on day 24, revealing findings of global hypoxic encephalopathy, such as diffuse cerebral edema, loss of the gray-white matter boundary, effacement of the sulci, ventricles, and basal cisterns, and high density of CT value in the basal cisterns and subarachnoid spaces (Fig. 1). As determining whether the CT findings reflected true SAH or pseudo-SAH was challenging, brain MRI was performed on day 26. The abnormality findings in the left Sylvian fissure displayed low intensity on T1-weighted imaging and GRE T2*-WI (Fig. 2A and D), suggesting that SAH is only in the left Sylvian fissure. Fluid-attenuated inversion recovery (FLAIR) image showed hyperintensity in the left Sylvian fissure and basal cistern and sulci (Figs. 2C and 3C). T1-, T2-, and T2*-WI revealed no abnormal findings in the basal cistern (Figs. 2B and 3A, B, and D), indicating the presence of pseudo-SAH except for the left Sylvian fissure. Maximum-intensity projection reconstruction of time-of-flight (TOF) MR angiography (MRA) showed loss of the supraclinoid intracranial arterial flow signal (Fig. 4A). In addition, intracranial TOF MRA demonstrated external carotid artery branches and loss of vascular flow in the internal carotid arteries (Fig. 4B). These CT and MRI findings indicated brain death owing to hypoxic encephalopathy following suicide by hanging, and incidental true SAH was confirmed by MR GRE T2*-WI. Owing to the absence of all brainstem reflexes, an isoelectric electroencephalogram, and a negative apnea test, brain death was diagnosed according to the protocols of the Japan Organ Transplant Network in Japan.[12] Donor surgery and organ transplantation were performed 2 days after the diagnosis of clinical brain death.

Discussion

Suicide by hanging is a form of ligature strangulation in which the force applied to the neck is derived from the gravitational force on the body or parts of the body. Independent and synergistic mechanisms exist through which hanging can cause death. These include (1) occlusion of the carotid or vertebral arteries, (2) venous occlusion, (3) airway obstruction resulting from pushing the base of the tongue against the roof of the pharynx or crushing the larynx or trachea, (4) stretching of the carotid sinus causing reflex cardiac arrest, and (5) spinal cord-brainstem disruption.[6] These mechanisms result in respiratory and cardiac arrest, leading to hypoxic-ischemic injury. The most common abnormality is diffuse cerebral edema owing to cerebral ischemia, and SAH is an atypical complication that occurs after suicide by hanging. Ikeda et al. (1989) demonstrated the course of respiration and circulation during the agonal period of death owing to suicide by hanging in dogs.[13] They reported that blood pressure rapidly increased after ligature to a maximum of 1.5 times the original level, which was maintained for approximately 1-1.5 minutes and then gradually decreased almost simultaneously with

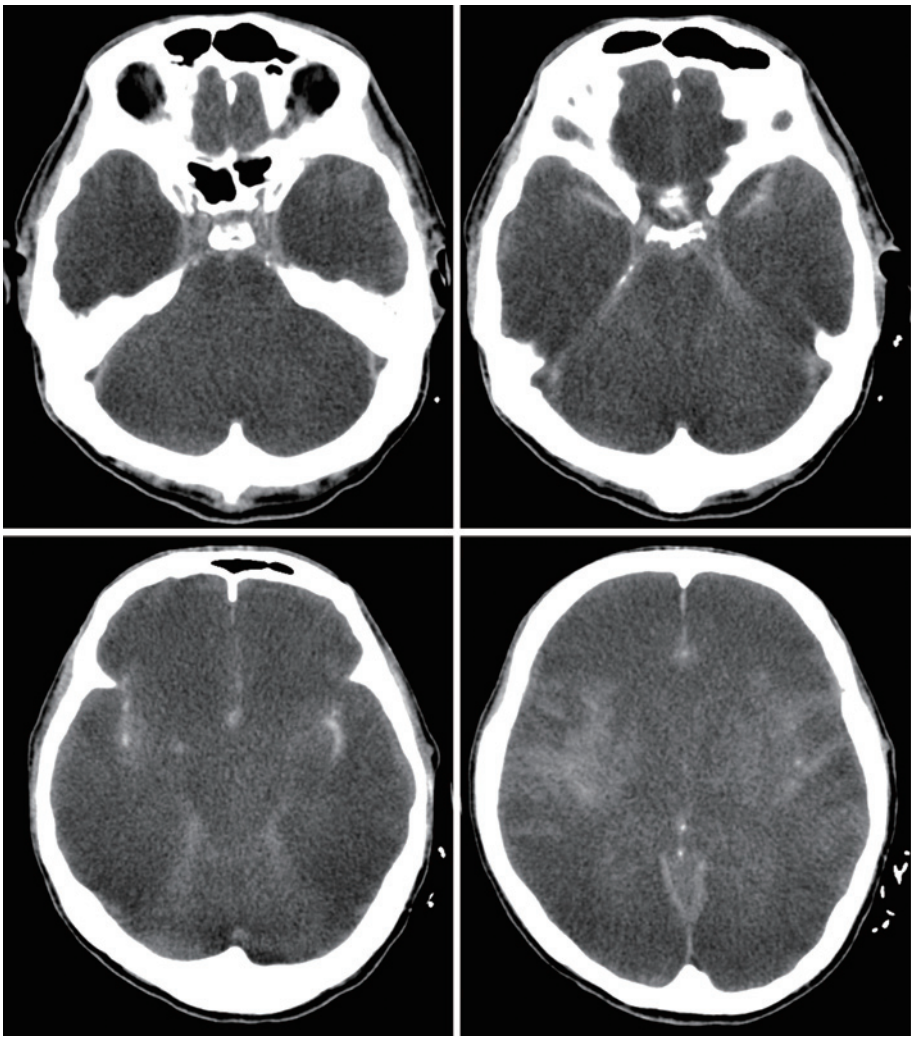

Fig. 1 Noncontrast brain computed tomography reveals diffuse brain edema, loss of gray-white matter contrast, and abnormally highly dense subarachnoid spaces of the basal and Sylvian cisterns and sulci.

respiratory arrest over 2-3 minutes. Schroder et al. (1983) reported the histological findings of brain autopsies of suicidal hanging victims.[14] They identified hyperemia in 78% of the cases owing to an abrupt blood supply to the venous and capillary parts. Additionally, minimal bleeding into the cerebral perivascular space was observed in 50% of cases. These reports suggest that hanging SAHs may reflect the spontaneous rupture of vessels within a high-pressure venous system.

Early recognition of brain death is important to expedite organ transplantation and prevent futile medical interventions. However, concept, criteria, practice, and documentation inconsistencies exist internationally and within countries.[15] For example, electroencephalogram testing necessary for legally determining brain death in Japan is only required in 22 of 70 countries. Although the essential requirements may change in the future based on international consensus, the current criteria for determining brain death in Japan are as follows: (1) deep coma and loss of spontaneous respiration owing to brain damage; (2) dilated and fixed pupils; (3) loss of all brain stem reflexes; (4) flat brain waves by electroencephalogram; (5) definitive diagnosis of a disease resulting in brain death; (5) no possibility of recovery with all appropriate and current treatments; and (6) two or more doctors with requisite expertise and experience confirming no changes after a second test conducted 6 or more hours later.[10,12] With these criteria, the cause of brain death must be determined by radiological imaging of CT or MRI in Japan; however, only a few reports exist on brain CT and MRI in suicide by hanging victims.[5,16-19] Although brain CT is useful for diagnosing hypoxic encephalopathy in suicide by hanging, detecting SAH is difficult because of the possibility of pseudo-SAH. Meanwhile, certain ancillary brain blood flow-based methods (angiography, transcranial Doppler, and scintigraphy) have been validated by the American Academy of Neurology to be used as confirmation in situations of uncertainty. In these brain blood flow-based methods, the absence of cere-

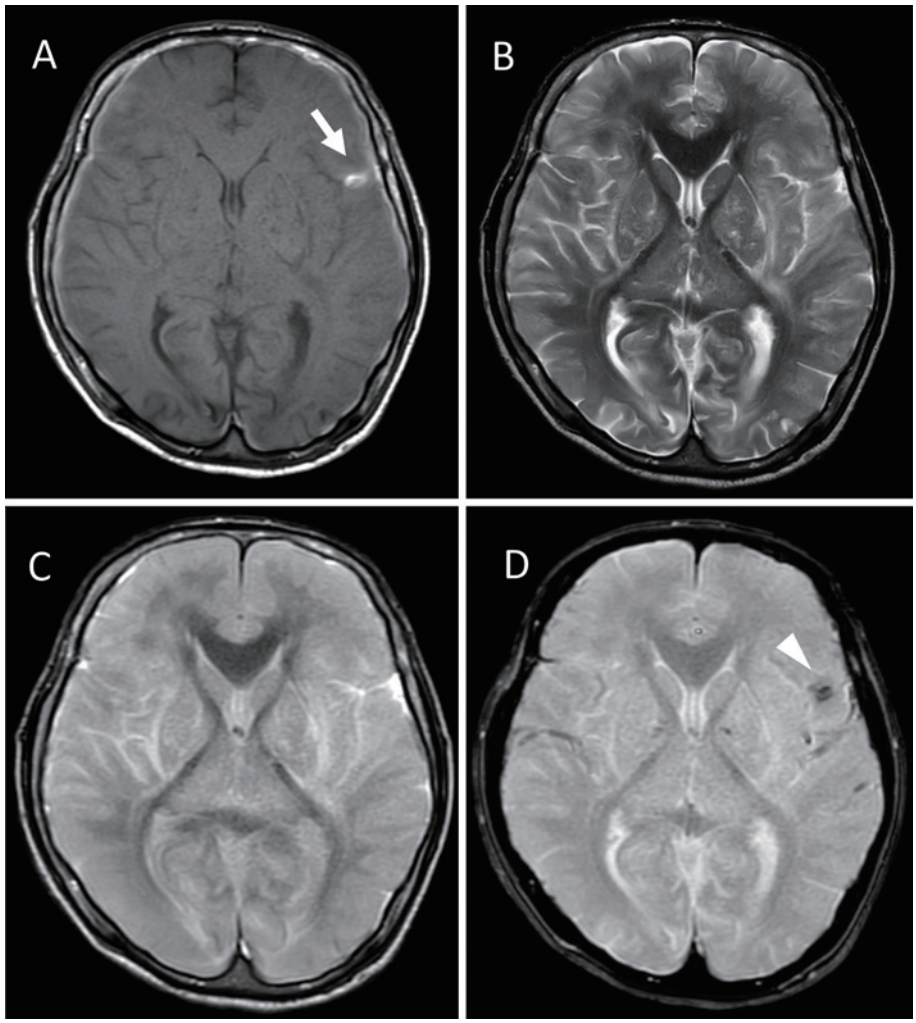

Fig. 2 Magnetic resonance imaging obtained 26 days after the onset. Axial T1-, T2-, Fluid-attenuated inversion recovery (FLAIR), and T2*-weighted imaging (T2*-WI) at the anterior horn of the lateral ventricle showing diffuse cerebellar and cerebral edema with effacement of the sulcal spaces (A-D). T1-weighted imaging reveals a small high-intensity region in the left Sylvian fissure (arrow) (A), and axial (T2*-WI) imaging shows a hypointense region in the same region (arrowhead) (D). These findings reveal a small subarachnoid hemorrhage in the left Sylvian fissure. FLAIR image showing hyperintensity in the left Sylvian fissure and the basal cistern and sulcus (C).

bral vessels above the supraclinoid internal carotid artery diagnoses the brain death. Alternative methods for evaluating cerebral blood flow include CT angiography and TOF MRA. However, similar to CT angiography, insufficient diagnostic confidence exists with TOF MRA to be used routinely as a confirmatory test for brain death. Similar to the other methods for assessing cerebral blood flow, the radiological diagnosis of TOF MRA in brain death is based on the absence of visualizing the intracranial internal carotid artery, although the reliability of these tests remain controversial. In our case, the blood flow in the intracranial internal carotid artery was not visualized.

Pseudo-SAH is a rare radiographic sign in marked hypoxic-ischemic injury and spontaneous intracranial hypotension.[7-9] The mechanism underlying the development of pseudo-SAH on brain CT is not fully understood.[7] Some authors have suggested that severe brain edema compresses the dural sinuses and compromises venous drainage from the brain. This results in engorgement of the superficial veins, which stand out against the edematous slow-attenuated brain parenchyma and mimic SAH.[8,9] Yuzawa et al. (2008) reported that the incidence of pseudo-SAH in patients with traumatic cardiac arrest was 20%.[20] Lee et al. (2017) reported that the prevalence of pseudo-SAH was about 8% in unwitnessed asystole arrest.[21] Given et al. (2003) reviewed seven cases of generalized cerebral edema accompanied by increased basal cisternal attenuation; however, none of those patients had true SAH on lumbar puncture or autopsy.[22] Although this method has high diagnostic accuracy, it can be highly invasive. Honton et al. (2020) diagnosed sulcal hyperdensity on CT scans as pseudo-SAH based on the lack of hyperintensity on the

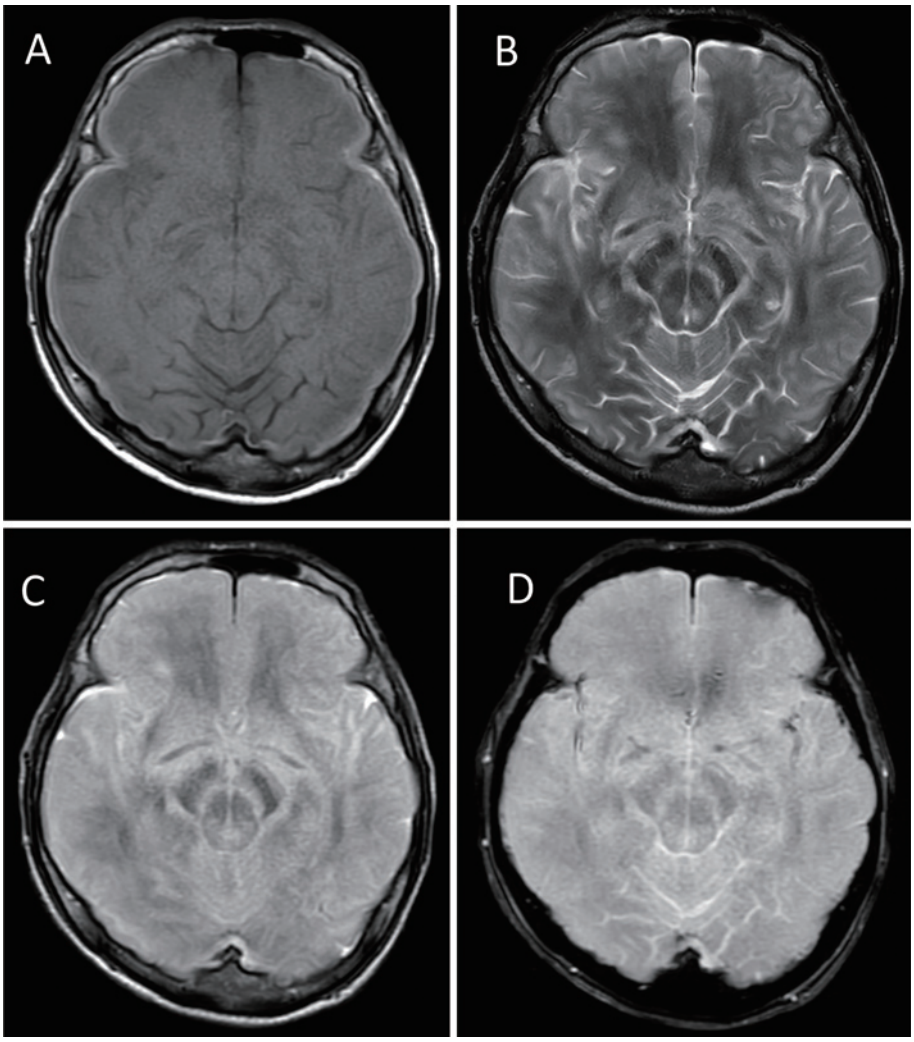

Fig. 3 Fluid-attenuated inversion recovery image reveals hyperintensity in the basal cistern and sulcus (C), but T1-, T2-, and T2*-WI reveals no abnormal findings in the basal cistern (A, B, D).

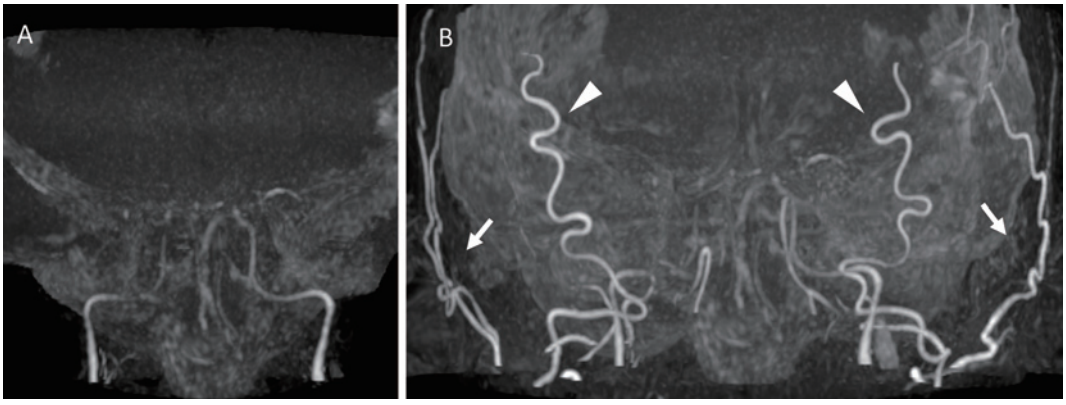

Fig. 4 Maximum-intensity projection reconstruction of time-of-flight (TOF) magnetic resonance angiography (MRA) shows loss of vascular flow within the supraclinoid internal carotid arteries but reveals opacification of the intracranial vertebral and basilar arteries (A). Intracranial TOF MRA demonstrates external carotid artery branches and loss of vascular flow in the internal carotid arteries (B). The superficial temporal arteries (arrows) and occipital arteries (arrowheads) are visualized.

FLAIR image of MRI.[23] However, even a high signal on FLAIR may not indicate bleeding because hyperintensity on FLAIR images has been reported in some pathological states, such as those with meningitis, meningeal carcinomatosis, and a small amount of protein leakage in the subarachnoid space.[24] We previously reported sulcal hyperintensity on FLAIR in epileptic chronic subdural hemorrhage without abnormal findings in GRE T2*-WI.[25] The FLAIR images demonstrated diffuse hyperintensity in the cerebral sulci and basal cistern. Conversely, GRE T2*-WI has a higher detection rate of cerebral bleeding than conventional MRI sequences and CT because GRE T2*-WI is sensitive to paramagnetic materials; it can easily identify and reveal details of the lesions that contain paramagnetic matter.[26-28] Furthermore, the scope of iron deposition corresponding to the scope of the revealed lesions on the GRE T2*-WI was pathologically confirmed.[29] Therefore, brain MR GRE T2*-WI is useful for diagnosing pseudo-SAH and true SAH in patients with hypoxic encephalopathy.[11,18] Although hemorrhagic symptoms, such as SAH, are rare in suicidal hanging,[5,6] MR GRE T2*-WI may reveal detailed findings of true SAH in suicidal hanging. The MR GRE T2*-WI may be effective in diagnosing pseudo-SAH/true-SAH in suicidal hanging, especially in determining the cause of brain death for organ transplantation.

Conclusions

Spontaneous SAH can often develop following suicidal hanging. Although SAH may be challenging to confirm on brain CT in cases of brain death by suicide by hanging, MR GRE T2*-WI is effective for differentiating true SAH or pseudo-SAH.

Conflicts of Interest Disclosure

We have completed and submitted to the Japan Neurosurgical Society our COI self-report for the past 3 years. All authors have no conflict of interest.

References

1) Farmer R, Rohde J: Effect of availability and acceptability of lethal instruments on suicide mortality: an analysis of some international data. *Acta Psychiatr Scand* 62: 436-446, 1980
2) Meel BL: A study on the incidence of suicide by hanging in the sub-region of Transkei, South Africa. *J Clin Forensic Med* 10: 153-157, 2003
3) Fayed M, Pusapati R, Widdicombe N, et al.: Characteristics of organ donors who died from suicide by hanging in Australia and New Zealand: a retrospective study. *Cureus* 13: e19243, 2021
4) Iserson KV: Strangulation: a review of ligature, manual, and postural neck compression injuries. *Ann Emerg Med* 13: 179-185, 1984
5) Kim TH, Lee SH, Kim DH, et al.: Non-aneurysmal and non-traumatic subarachnoid hemorrhage after attempted suicide by incomplete hanging. *Clin Exp Emerg Med* 4: 56-59, 2017
6) Schwab N, Díaz L, Galtés I: Intracerebral and subarachnoid hemorrhage after suicidal "near-hanging". *Int J Legal Med* 136: 1359-1362, 2022
7) You JS, Park S, Park YS, Chung SP: Pseudo-subarachnoid hemorrhage. *Am J Emerg Med* 26: 521.e1-521.e2, 2008
8) Schievink WI, Maya MM, Tourje J, Moser FG: Pseudo-subarachnoid hemorrhage: a CT-finding in spontaneous intracranial hypotension. *Neurology* 65: 135-137, 2005
9) Westwood AJ, Burns JD, Green DM: Teaching NeuroImages: pseudo-subarachnoid hemorrhage. *Neurology* 78: e54, 2012
10) Health and Labour Sciences Research Grant Special Research Project: Research Teams on the Protocol for Brain Death: Manual on the Legal Determination of Brain Death (1999 Report). Tokyo, Japan Medical Journal, 1999 (in Japanese)
11) Fazekas F, Kleinert R, Roob G, et al.: Histopathologic analysis of foci of signal loss on gradient-echo T2*-weighted MR images in patients with spontaneous intracerebral hemorrhage: evidence of microangiopathy-related microbleeds. *Am J Neuroradiol* 20: 637-642, 1999
12) Japan Organ Transplant Network Organ Donor Facility Committee (ed): Protocols for Organ Donor Facilities. Tokyo, Japan Organ Transplant Network, 2010, pp 6-7 (in Japanese)
13) Ikeda N, Takahashi H, Umetsu K, Suzuki T: The course of respiration and circulation in death by carbon dioxide poisoning. *Forensic Sci Int* 41: 93-99, 1989
14) Schröder R, Saternus KS: Congestion in the area of the head and changes in the brain caused by suicidal hanging death. *Z Rechtsmed* 89: 247-265, 1983(in German)
15) Greer DM, Shemie SD, Lewis A, et al.: Determination of brain death/death by neurologic criteria: the world brain death project. *JAMA* 324: 1078-1097, 2020
16) Brancatelli G, Sparacia G, Midiri M, D'Antonio V, Sarno C, Lagalla R: Brain damage in hanging: a new CT finding. *Neuroradiology* 42: 209-210, 2000
17) Nakajo M, Onohara S, Shinmura K, et al.: Computed tomography and magnetic resonance imaging findings of brain damage by hanging. *J Comput Assist Tomogr* 27: 896-900, 2003
18) Lee BK, Jeung KW, Lee HY, Lim JH: Outcomes of therapeutic hypothermia in unconscious patients after near-hanging. *Emerg Med J* 29: 748-752, 2012
19) Solhi H, Pazoki S, Mehrpour O, Alfred S: Epidemiology and prognostic factors in cases of near hanging presenting to a referral hospital in Arak, Iran. *J Emerg Med* 43: 599-604, 2012
20) Yuzawa H, Higano S, Mugikura S, et al.: Pseudo-subarachnoid hemorrhage found in patients with postresuscitation encephalopathy: characteristics of CT findings and clinical importance. *AJNR Am J Neuroradiol* 29: 1544-1549, 2008
21) Lee BK, Kim YJ, Ryoo SM, et al.: "Pseudosubarachnoid hemorrhage sign" on early brain computed tomography in out-of-hospital cardiac arrest survivors receiving targeted temperature management. *J Crit Care* 40: 36-40, 2017
22) Given CA II, Burdette JH, Elster AD, Williams DW III: Pseudo-subarachnoid hemorrhage: a potential imaging pitfall associated with diffuse cerebral edema. *AJNR Am J Neuroradiol* 24: 254-256, 2003
23) Honton B, Sauguet A, Farah B, Gellee S, Rivière LD: Extended pseudo-subarachnoid hemorrhage post-percutaneous coronary intervention. *JACC Case Rep* 2: 2394-2396, 2020
24) Taoka T, Yuh WT, White ML, Quets JP, Maley JE, Ueda T: Sulcal hyperintensity on fluid-attenuated inversion recovery MR images in patients without apparent cerebrospinal fluid abnormality. *AJR*

Am J Roentgenol 176: 519-524, 2001
25) Oshida S, Akamatsu Y, Matsumoto Y, et al.: A case of chronic subdural hematoma demonstrating the epileptic focus at the area with sulcal hyperintensity on fluid-attenuated inversion recovery image. *Radiol Case Rep* 14: 1109-1112, 2019
26) Kidwell CS, Saver JL, Villablanca JP, et al.: Magnetic resonance imaging detection of microbleeds before thrombolysis: an emerging application. *Stroke* 33: 95-98, 2002
27) Schellinger PD, Jansen O, Fiebach JB, Hacke W, Sartor K: A standardized MRI stroke protocol: comparison with CT in hyperacute intracerebral hemorrhage. *Stroke* 30: 765-768, 1999
28) Fiebach JB, Schellinger PD, Gass A, et al.: Stroke magnetic resonance imaging is accurate in hyperacute intracerebral hemorrhage: a multicenter study on the validity of stroke imaging. *Stroke* 35: 502-506, 2004
29) Patel MR, Edelman RR, Warach S: Detection of hyperacute primary intraparenchymal hemorrhage by magnetic resonance imaging. *Stroke* 27: 2321-2324, 1996

Corresponding author: Sotaro Oshida, MD, PhD
Department of Neurosurgery, Iwate Medical University, 2-1-1 Idaidori, Yahaba, Iwate 028-3695, Japan.
e-mail: soshida@iwate-med.ac.jp