

Spinal epidural hematoma as a stroke mimic

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Purpose: Spinal epidural hematoma is a rare but important disease as it can be a stroke mimic. Our aim was to investigate the clinical characteristics of patients with an activated stroke code and spinal epidural hematoma. **Methods:** Patients with an activated stroke code were examined retrospectively. Patients with spinal epidural hematoma were evaluated with further neurological examinations and neuroimaging. **Results:** Of 2866 patients with an activated stroke code, spinal epidural hematoma was detected in 5 (0.2%, 63–79 years, 2 men). In all 5 cases, hematoma was located in the unilateral dorsal region of the spinal canal and spread to 5–9 vertebral segments at the C1–T3 level. None of the patients had a medical history of head or neck injury, coagulopathy, or use of anti-thrombotic agents. All of the patients had occipital, neck, and/or back pain, and their hemiparesis occurred simultaneously or within 1 h after the onset of pain. Hyperalgesia ipsilateral to the hematoma was observed in 1 patient, hypoalgesia contralateral to the hematoma was observed in 1, and quadriparesis and bilateral hypoalgesia were observed in 1. The hematomas spontaneously decreased in size in 4 patients, and cervical laminectomy was performed in the other patient. In the 1860 patients with an activated stroke code and spontaneous eye opening, the sensitivity of pain as a predictor of spinal epidural hematoma was 100%, with a specificity of 88.7%, and positive predictive value of 2.3%. **Conclusion:** Patients with spinal epidural hematoma could present with clinical characteristics mimicking ischemic stroke. Spinal epidural hematoma should be differentiated in patients treated under stroke code activation.

Keywords: Spinal epidural hematoma—Stroke mimics—Stroke code—Myelopathy—Hemiparesis

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Introduction

Spinal epidural hematoma is a rare disease with an annual incidence of 1 per 1,000,000 individuals;¹ however, it is a critical stroke mimic.² As patients with spinal epidural hematoma typically present with acute hemiparesis,^{3,4} such a presentation is not expected to be detected by a computed tomography (CT) scan of the brain, which is the most common neuroimaging choice in patients with an activated stroke code. Indeed, there are several case reports in which patients with spinal epidural hematoma were

misdiagnosed with ischemic stroke and treated with anti-thrombotic therapy, including intravenous thrombolysis,^{5–10} occasionally leading to hematoma expansion.

The necessity of brain magnetic resonance imaging (MRI) for patients with an activated stroke code to exclude stroke mimics remains controversial. Brain MRI is superior to CT for the detection of acute ischemic stroke.¹¹ In most stroke mimics, including spinal epidural hematoma, the final diagnosis might not be confirmed with brain MRI alone. However, if no ischemic lesions responsible for neurological deficits are detected on brain MRI in patients with an activated stroke code, due to a concern for the presence of stroke mimics,² thrombolysis might be withheld, which is sometimes more harmful than it is useless. Conversely, a positive relationship between the early initiation of hyperacute reperfusion therapy and prognosis has been established.^{12–14} Moreover, some studies have suggested that thrombolysis is safe for stroke mimics.^{15–17} Therefore, the indication for thrombolysis may be decided only by a CT scan if CT angiography is not available in the primary stroke center.¹⁸

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Received April 15, 2020; revision received June 3, 2020; accepted June 4, 2020.

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<https://doi.org/10.1016/j.jstrokecerebrovasdis.2020.105030>

In these circumstances, it is useful to investigate how to screen for spinal epidural hematoma using only clinical characteristics for the optimal management of patients with an activated stroke code, including an indication for brain MRI. Especially, in two previous studies, all patients with spinal epidural hematoma had pain, including headache, neck pain, and back pain (13 and 27 patients, respectively).^{1,19} In another study, only 1 of 16 patients with spinal epidural hematoma did not have pain.⁴ Therefore, pain is a potential predictor of spinal epidural hematoma. However, no studies have reported the clinical characteristics of patients with an activated stroke code and spinal epidural hematoma.

Here, we present a study in which patients with an activated stroke code and spinal epidural hematoma were evaluated with further neurological examinations and neuroimaging. We also studied the clinical significance of pain as a predictor of spinal epidural hematoma.

Methods

This was a study of consecutive patients with an activated stroke code on admission to our hospital from August 1, 2009 to July 31, 2019. The study protocol followed the principles outlined in the Declaration of Helsinki, and all patients provided informed written consent for the use of their medical data.

Stroke code criteria

The subjects with an activated stroke code were patients with acute hemiparesis and/or speech disturbance. Patients predominantly presenting with chest and/or back pain or shock indicating aortic dissection or acute coronary diseases, and with very subtle or unrecognized neurological deficits were excluded. According to changes to the indication for thrombolysis and the introduction of endovascular thrombectomy in our hospital, we adjusted the time limit for the standard issuance of the stroke code as follows: Period 1 (August, 2009 to August 2012), estimated arrival time within 3 h after the last time the patient was known to be without a neurological deficit; Period 2 (September 2012 to January 2016), estimated arrival time is within 4.5 h after the last time the patient was known to be without a neurological deficit; and Period 3 (February 2016 to July 2019), estimated arrival time is within 6 h after the identification of a neurological deficit.

Emergent management for patients with an activated stroke code

All patients with an activated stroke code were examined initially by neurologists on admission, and were evaluated with brain MRI/MR angiography; if these were not available, a CT scan was conducted. If there was no

evidence of recent ischemic lesions in the brain, findings of arterial occlusion, including hyperintense vessel signs or gradient echo susceptibility vessel signs responsible for their neurological deficits, nor episodes or findings suggesting status epilepticus, additional examinations including spinal MRI were performed. In all patients with an activated stroke code, specific medical details were recorded including onset time, symptom detection time, arrival time at our hospital, presence of pain including headache, neck pain, or back pain, Glasgow Coma Scale (GCS) score on admission, National Institutes of Health Stroke Scale (NIHSS) score on admission, and final diagnosis in the emergency room.

Statistical analysis

Of the patients with an activated stroke code and a score of 4 for GCS-Eye (spontaneous eye opening) on admission, the sensitivity and specificity of pain as a predictor of spinal epidural hematoma were examined.

Results

A total of 2,866 patients (median age 77 years, interquartile range 66–85 years, men 1,556) with an activated stroke code were admitted to our hospital during the 10-year study period. Table 1 shows the details of their final diagnosis at our emergency room. Consequently, 5 patients (0.2%, 63–79 years, 2 men) were diagnosed with spinal epidural hematoma.

Fig. 1 shows a flow chart for the diagnosis of the patients with an activated stroke code. Of 2,866 patients with an activated stroke code, 1,860 (64.9%) had a score of

Table 1. Final diagnosis of patients with an activated stroke code.

Category	Disease	Number (%)
Brain diseases	Brain infarction and TIA	1,782 (62.2)
	Intracerebral hemorrhage	725 (25.3)
	Subarachnoid hemorrhage	31 (1.1)
	Epilepsy	75 (2.6)
	Head injury	17 (0.6)
	Other	93 (3.2)
	Spinal epidural hematoma	5 (0.2)
Vertebral diseases	Other	8 (0.3)
Systemic diseases	Hypoglycemia	22 (0.8)
	Drug abuse	11 (0.4)
	Other	50 (1.7)
Psychogenic disorders		14 (0.5)
Unknown etiology		33 (1.2)
Total		2,866 (100.0)

TIA: transient ischemic attack

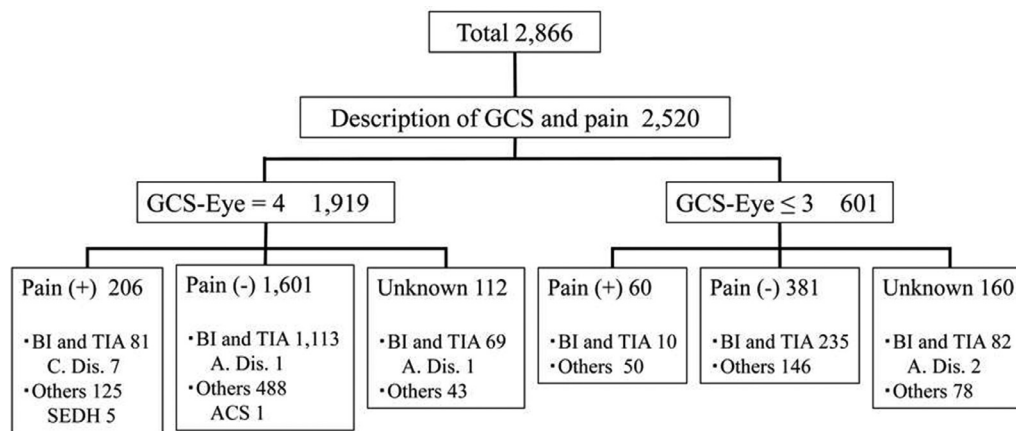


Fig. 1. Pain and final diagnosis in patients with an activated stroke code.

Pain: including headache, neck pain, and back pain

GCS: Glasgow Coma Scale; BI: brain infarction, TIA: transient ischemic attack; C. Dis.: cerebral arterial dissection; SEDH: spinal epidural hematoma; A. Dis.: aortic dissection; ACS: acute coronary syndrome

4 for GCS-Eye. Of these 1,860 patients, 215 (11.3%) had headache or neck pain, of which only 5 (2.4%) were diagnosed with spinal epidural hematoma. the sensitivity of pain as a predictor of spinal epidural hematoma was 100%, with a specificity of 88.7%, positive predictive value of 2.3%, and negative predictive value of 100%. Additionally, cerebral arterial dissection was found in 7 stroke patients with pain. Aortic dissection was diagnosed in 4 patients: 1 without pain, and the other 3 who could not answer if they were in pain because of aphasia or coma. Myocardial infarction was also diagnosed in 1 patient who presented with sudden mutism due to a psychosomatic disorder caused by chest pain.

Case reports (Table 2, Fig. 2)

Patient 1: A 65-year-old woman with chronic hepatitis C and mild thrombocytopenia experienced a sudden right back pain when performing house work. At 1 h after onset, she developed right hemiparesis and was admitted to our hospital. Neurological examinations on admission at 2 h after onset revealed moderate right hemiparesis (manual muscle testing[MMT] score of 3). Her NIHSS score was 4. Using a numerical rating scale (NRS), she rated her persistent right back pain with a score of 3. Cervical MRI after brain MRI revealed a hematoma in the right dorsal region of the spinal canal at the C4–T2 level. She was treated with rest and administration of analgesics; thereafter, her hemiparesis subsided completely, and her back pain decreased until the next morning at 21 h after onset. She was discharged home on Day 3, and her back pain continued to subside until Day 9 with no neurological deficits.

Patient 2: A 78-year-old woman with hypertension experienced a sudden pain on the left side of her neck and left hemiparesis during a work break. Neurological examinations on admission at 1 h after onset revealed

moderate left hemiparesis (MMT score of 3) and moderate hyperalgesia on her left side. Her NIHSS score was 6. She rated her persistent left neck to shoulder pain with an NRS score of 6. Cervical MRI after brain MRI revealed a hematoma in the left dorsal region of the spinal canal at the C3–C7 level. She was treated with rest using a neck collar and administration of analgesics; thereafter, her hemiparesis and sensory disturbance subsided completely on the evening of admission at 9 h after the onset. Her back pain continued to subside until Day 9 when she was discharged with no neurological deficits. On MRI on Day 2, the hematoma was markedly diminished.

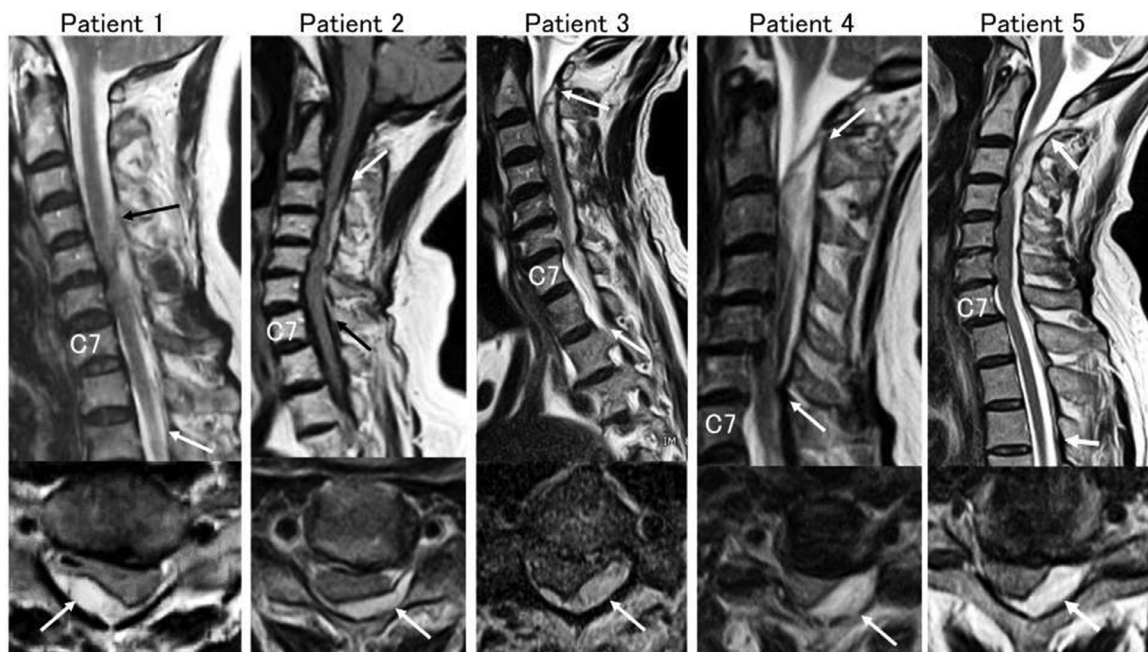
Patient 3: A 79-year-old man with hypertension and dyslipidemia experienced a sudden occipital pain and left hemiparesis soon after defecation. Neurological examinations on admission at 45 min after onset revealed anisocoria (right 3 mm, left 2 mm), mild dysarthria, moderate right hemiparesis (MMT score of 3), and moderate hypoaesthesia on his left side. His NIHSS score was 7. He rated his persistent left neck to shoulder pain with an NRS score of 6. Cervical MRI after brain MRI revealed a hematoma in the left dorsal region of the spinal canal at the C1–T2 level. He was treated with rest using a neck collar and administration of analgesics; thereafter, his neurological deficits, including anisocoria, dysarthria, hemiparesis, and sensory disturbance, subsided completely by the next morning at 6 h after onset. His occipital pain continued to subside until Day 3 and he was discharged on Day 5 with no neurological deficits. On MRI on Day 2, the hematoma was markedly diminished.

Patient 4: A 63-year-old healthy woman experienced a sudden occipital and neck pain during bathing. At 45 min after onset, she developed left hemiparesis and was admitted to our hospital. Neurological examinations on admission at 80 min after the onset revealed mild dysarthria and moderate right hemiparesis (MMT score: arm 2, leg 3). Her NIHSS score was 6. She rated her persistent

Table 2. Clinical characteristics of the present 5 cases.

	Patient 1	Patient 2	Patient 3	Patient 4	Patient 5
Age/sex	65F	78F	79M	63F	64M
Medical history	Chronic hepatitis C, mild thrombocytopenia	Hypertension	Hypertension, dyslipidemia	None	Crohn's disease
Recent traumatic history	None	None	None	None	None
Onset to arrival, min	139	66	46	78	99
Site of pain	Right back	Left neck, shoulder	Occipital	Left neck, shoulder	Neck
NRS of pain	3	6	6	8	9
Pain to palsy, min	60	0	0	45	0
Side of palsy	Right	Left	Left	Left	Bilateral
MMT of palsy, arm/leg	3/3	3/2	3/3	2/3	Left 1/1 Right 4/4
Deficits other than palsy	None	Left hyperalgesia	Left miosis, dysarthria, right hypoalgesia	Dysarthria	Bilateral dysesthesia, dysuria
NIHSS on admission	4	6	7	6	9
Level of hematoma	C4–T2	C3–C7	C1–T2	C1–C6	C2–T3
Platelets, $10^3/\mu\text{L}$	98	231	273	170	137
PT-INR	1.15	1.22	0.91	0.96	1.06
APTT, s	33.9	31.5	36.6	34.3	24.3
Treatment	Rest and analgesics	Rest and analgesics	Rest and analgesics	Rest and analgesics	Laminectomy
Duration of palsy	21 h	9 h	6 h	22 h	Persistent
Duration of pain	9 days	6 days	3 days	22 h	1 month

NRS: numerical rating scale; MMT: manual muscle testing using Medical Research Council Muscle Scale; NIHSS: National Institutes of Health Stroke Scale; PT-INR: prothrombin time-international normalized ratio; APTT: activated partial thromboplastin time.

**Fig. 2.** T2-weighted images of the 5 patients on admission.

Sagittal (upper) and transverse (lower) images showing hematomas (arrows) in the spinal epidural space.

occipital and neck pain with an NRS score of 8. Brain and cervical MRI revealed a hematoma in the left dorsal region of the spinal canal at the C1–T6 level. She was treated with rest and administration of analgesics; thereafter, her hemiparesis subsided completely, and her back pain was also reduced by the next morning at 22 h after onset. She was discharged on Day 11 with no neurological deficits. On MRI on Day 4, the hematoma was markedly diminished.

Patient 5: A 64-year-old man with Crohn's disease woke up at approximately 20 min after falling asleep due to a severe neck pain. He also noticed left hemiparesis and numbness in his bilateral arms and legs and was admitted to our hospital. Neurological examinations on admission at 100 min after onset revealed quadriplegia (MMT score: right 1, left 4) and right dominant dysesthesia in his bilateral arms and legs. His NIHSS score was 9. He rated his persistent left neck to shoulder pain with an NRS score of 9. Brain CT was normal and brain MRI was skipped. Cervical MRI revealed a hematoma in the left dorsal region of the spinal canal at the C2–T3 level. He was transferred to another hospital and treated with emergent cervical laminectomy and evacuation of the hematoma. After surgery, he reported dysuria with urinary urgency. He was treated with rehabilitation; thereafter, he had moderate left hemiparesis and could walk using a walker until 1 month after onset. On MRI on Day 7, the hematoma was markedly diminished.

To summarize the 5 patients, spinal epidural hematoma was located in the unilateral dorsal region of the spinal canal in all cases, and spread to 5–9 (mean 7) vertebral segments at the C1–T3 level. No patient had a history of bleeding tendency. They all had occipital, neck, and/or back pain, and their hemiparesis occurred simultaneously or within 1 h after the onset of pain. Hyperalgesia ipsilateral to the hematoma was observed in 1 patient, hypoalgesia contralateral to the hematoma was observed in 1, and quadriplegia and bilateral hypoalgesia were observed in 1. They were all diagnosed with spinal epidural hematoma on admission, and none of them were administered intravenous thrombolysis for a misdiagnosis of ischemic stroke. The hematomas diminished spontaneously in 4 patients, and cervical laminectomy was performed in 1.

Discussion

In the present study, 5 (0.2%) of 2,866 patients with an activated stroke code were diagnosed with spinal epidural hematoma. To the best of our knowledge, this is the first report in which the frequency and detailed clinical characteristics of patients with an activated stroke code and spinal epidural hematoma are presented. In a previous study, 104 of 411 patients with an activated stroke code had a stroke mimic, although none of them had spinal epidural hematoma.²⁰ In another study, 100 of 5,418

patients treated with intravenous thrombolysis had a stroke mimic, and 1 of them had cervical hematoma.¹⁵

Among the neurological deficits of the 5 patients, the pathophysiological mechanism of hemiparesis ipsilateral to the hematoma was easily explained by its direct compression of the ipsilateral pyramidal tract in the upper cervical lateral column. The hemisensory disturbance contralateral to the hematoma in Patient 3 was explained by its direct compression of the ipsilateral anterior spinothalamic tract in the anterior column. The quadriplegia, bilateral sensory disturbance, and dysuria in Patient 5 may have been caused by broad compression of the spinal cord by the large hematoma. The hyperalgesia ipsilateral to the hematoma in Patient 2 may have been caused by its "contrecoup" compression of the contralateral anterior spinothalamic tract in the anterior column. Patient 3 had miosis ipsilateral to the hematoma, which was diagnosed as a part of Horner syndrome, as reported previously.⁴ Conversely, Patients 3 and 4 had dysarthria. We hypothesized two possible explanations for their dysarthria. First, severe pain caused the psychosomatic symptoms such as slurred speech. In a patient with spinal epidural hematoma reported previously, consciousness disturbance due to a vasovagal reflex caused by pain was observed.⁴ Second, direct compression of the medulla resulted in dysarthria because the hematomas reached the C1 level in both patients.

Next, we discuss whether spinal epidural hematoma can be screened for using only clinical characteristics. First, a medical history indicating a bleeding tendency, including taking antithrombotic agents, coagulopathy, or head injury, was not observed in the present patients, except for mild thrombocytopenia in Patient 1. In previous studies, 30–52% of patients with spinal epidural hematoma did not have any evidence of a hemorrhagic disease.^{3,21} Therefore, it may be difficult to screen for spinal epidural hematoma using medical history.

Second, in the 5 patients, typical symptoms indicating myelopathy or radiculopathy, including quadriplegia and bilateral sensory disturbance, were observed in only 1 patient, and sensory disturbance contralateral to hemiparesis indicating Brown-Séquard syndrome was also present in 1. In a review of the literature, spinal epidural hematoma, paraplegia, and/or bilateral sensory disturbance were observed in 37% of patients, Brown-Séquard syndrome was observed in 7%, and hemiparesis and/or hemisensory disturbance were observed in 36%.³ In Patients 1 and 5, the diagnosis of spinal epidural hematoma may have been relatively easy to make because of their typical clinical characteristics, including back pain, hemiparesis without definitive cortical or cranial nerve involvement and/or distribution of sensory disturbance indicating a spinal lesion. However, it may also be difficult to screen for spinal epidural hematoma using neurological findings.

Finally, all 5 patients complained of occipital, neck, and/or back pain simultaneously or prior to the onset of their neurological deficits, similar to previous studies.^{1,4,19} It is suggested that these were caused by radicular pain or radiating pain. In the present study, pain had a high level of sensitivity and specificity for the detection of spinal epidural hematoma among patients with an activated stroke code; however, its positive predictive value was only 2.3%. Therefore, to identify a patient with spinal epidural hematoma, brain MRI must be performed on approximately 40 times as many patients with any pain, if brain MRI is used as a screening tool.

Conversely, the above-mentioned large-scale studies of thrombectomy indicated that shortening the onset-to-recanalization time by the 15 min necessary to perform MRI or CT angiography increased the number of independent patients at 3 months after onset by only 1–2%.^{12–14} Therefore, we may consider adding brain MRI to the test battery for patients with an activated stroke code if an emergency brain MRI can be completed within 15 min. We appreciate that it is difficult to perform brain MRI on all patients; however, it may be considered for those patients with pain. It is necessary to elucidate clinical factors for stroke mimics with a higher positive predictive value than pain in order to utilize emergency MRI in patients with an activated stroke code.

The present study has some limitations that merit consideration. First, in our hospital, activation of stroke code is determined according to information on a patient provided by the staff of the emergency medical service. Additionally, if a patient has more critical cardiovascular symptoms compared to neurological deficits, including chest and/or back pain or shock, the physicians in our emergency room prioritized the former and do not activate the stroke code. Therefore, some patients with a brain infarction due to myocardial infarction or aortic dissection may not have been included as subjects in the present study. Additionally, patients with paraplegia obviously indicating myelopathy do not have stroke code activation on admission. The exclusion of patients without stroke code activation might have influenced the results. Second, neurological deficits due to spinal epidural hematoma often subside spontaneously, as observed in some of the present cases. Therefore, some patients with spinal epidural hematoma might have been misdiagnosed as transient ischemic attack. To compare the frequency of spinal epidural hematoma in patients with an activated stroke code included in other studies, the criteria for the stroke code activation and neuroimaging for diagnosis should be considered.

Conclusion

In the present study, 5 patients with an activated stroke code were diagnosed with spinal epidural hematoma. In all cases, the hematoma spread within the C1–T3 level.

They had stroke-like symptoms including hemiparesis, and all of them had sudden pain in their head, neck, or back. Spinal epidural hematoma should be differentiated in patients with stroke code activation. The present study indicated that physicians need to keep spinal epidural hematoma in mind when they manage patients with an activated stroke code.

Declaration of Competing Interest

The authors declare that they have no competing interests.

Funding: No targeted funding reported.

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