Clinical Imaging 55 (2019) 71–75

Contents lists available at ScienceDirect

Clinical Imaging

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

Neuroradiology

Spinal cord watershed infarction: Novel findings on magnetic resonance imaging

Mindy X. Wang, Garrett Smith*, Mehmet Albayram

Department of Radiology, University of Florida College of Medicine, 1600 SW Archer Road, Gainesville, FL 32611, USA

ARTICLE INFO

Keywords:
Spinal cord
Ischemia
Watershed
MRI
Hypotension

ABSTRACT

Spinal cord watershed ischemia is a rare phenomenon often associated with cardiac arrest, prolonged hypotension, and atherosclerotic disease. It can manifest as central necrosis with peripheral sparing in the transverse axis, and central lesion with rostral and caudal sparing in the longitudinal axis. Few reports provide detailed imaging findings of spinal cord watershed ischemia lesions. We present a patient who experienced watershed infarcts of the brain and spinal cord following prolonged hypotension due to blood loss after an aortic aneurysm repair. The patient experienced loss of neurologic function of the lower extremities and left arm that did not recover following spinal cord ischemia protocol. MRI revealed spinal cord watershed ischemia in both the longitudinal and axial planes with the point of maximal T2 signal hyperintensity in the central cord at T10-T11. Unique findings included zones of central maximal T2 signal hyperintensity with peripheral sparing, and moderate T2 intensity representing partial ischemia between regions of maximal T2 intensity unaffected peripheral regions. Thoracoabdominal computed tomography angiogram revealed extensive intraluminal thrombus and bilateral spinal artery occlusion from T8 to L2 and bilateral severe renal artery stenosis. T7 and L3 spinal arteries were patent. We suspect preexisting atherosclerotic disease played a significant role in the development of widespread watershed lesions following prolonged hypotension and resulted in a clinical and imaging presentation distinct from that seen with isolated anterior spinal artery occlusion. Our unique MRI findings portray a rarely documented pattern of spinal cord watershed ischemia and prompt questions about the role of anatomic idiosyncrasies and preexisting vascular disease in the development of spinal cord watershed ischemia.

1. Introduction

Spinal cord infarction is a rare phenomenon that can occur in the context of atherosclerosis, aortic surgery, dissection, embolic disease, infection, coagulopathies, sickle cell disease, cocaine use and vasculitis [1,2]. Severe hypotension and cardiac arrest may also lead to acute spinal cord ischemia. The clinical presentation depends on the location and extent of infarction and usually occurs abruptly [1]. Symptoms may include bilateral loss of motor function, loss of pain/temperature sensation, flaccid paraplegia, areflexia, and urinary and anal sphincter dysfunction [1]. Magnetic resonance imaging (MRI) is an essential tool for the diagnosis of acute spinal cord ischemia [3,4].

Global ischemia and watershed infarction of the spinal cord are very rare and have received relatively little attention in the medical literature, particularly in the realm of imaging [5–13]. We present a case of spinal cord watershed infarct in the setting of retroperitoneal bleeding, hypotension, myocardial infarction, acute tubular necrosis, and cerebral and cerebellar watershed infarcts following thoracoabdominal aortic aneurysm repair. Novel MRI findings included clear infarcts in the transverse and longitudinal planes that corresponded with prior histopathologic studies on spinal cord watershed ischemia [14].

2. Case report

A 61-year-old female with a history of multiple sclerosis, cerebrovascular accident, multivessel coronary artery disease, hypertension, and hyperlipidemia was admitted for an elective type 4 thoracoabdominal aortic aneurysm repair. Three hours following surgery, she became diaphoretic, bradycardic, and severely hypotensive at 77/60 mm Hg. At this time the patient also reported abdominal pain and experienced a drop in hemoglobin from 10.4 to 7.3 and developed acute tubular necrosis. Echocardiogram at bedside identified dyskinetic left ventricle and a reduced ejection fraction and EKG identified T-wave inversion in V1–4 and new ST elevation. She was diagnosed with ST-elevated myocardial infarction and was urgently sent to the catheterization lab. She had multiple mild-moderate stenotic arterial lesions,

* Corresponding author at: Department of Radiology, University of Florida College of Medicine, P.O. Box 100374, Gainesville, FL 32610-0374, USA.
E-mail addresses: mxwang@ufl.edu (M.X. Wang), garrettrsmith@ufl.edu (G. Smith), albaym@radiology.ufl.edu (M. Albayram).

https://doi.org/10.1016/j.clinimag.2019.01.023
Received 16 November 2018; Received in revised form 27 January 2019; Accepted 30 January 2019
0899-7071/

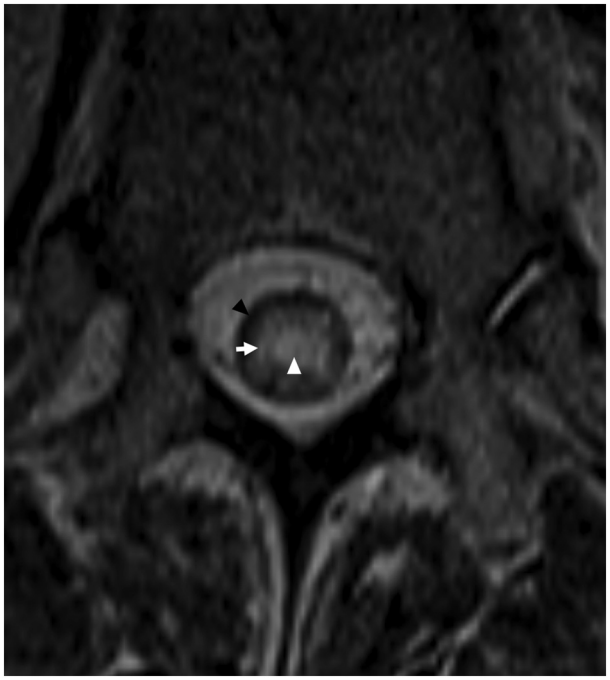

Fig. 1. Axial T2-W MRI of the spinal cord demonstrates central significantly hyperintense area representing severe ischemia and necrosis (white arrowhead), slightly hyperintense region representing partial ischemia in the watershed zone between central gray matter and peripheral white matter (white arrow), and peripheral hypointense rim (black arrowhead) representing non-ischemic preserved peripheral white matter zone.

but none required intervention. The next morning, 18 h post-operatively, the patient was not able to move lower extremities. Spinal cord ischemia was suspected and she was started on the spinal cord ischemia protocol comprising placement of a spinal drain and maintenance of mean arterial pressure over 100 mm Hg.

MRI on T2-weighted (T2-W) sequences obtained the next day, 19–20 h following initial presentation of leg weakness, revealed spinal cord ischemia in both the longitudinal and axial planes with the point of maximal T2 signal hyper intensity in the central cord at T10-T11 (Figs. 1, 2). Axial images revealed central T2 signal hyperintensity, peripheral white matter sparing, and partial ischemia between the central gray matter and peripheral white matter. Diffusion weighted MR imaging (DWI) was also performed for thoracic and lumbar regions; however, interpretation of these images was inconclusive due to artefact and low 1.5 T field strength. Additionally, she had watershed infarcts in the cerebrum and cerebellum (Fig. 3) and a left-sided retroperitoneal hematoma. Retrospective review of her preoperative thoracoabdominal computed tomography angiogram (CTA) revealed extensive intraluminal thrombus and bilateral occlusion of her spinal arteries from T8 to L2, including possible involvement of her Adamkiewicz artery, and bilateral severe renal artery stenosis (Fig. 4).

She was continued on a spinal cord ischemia protocol but did not recover any lower extremity function during her 11-day hospitalization. She was discharged to a long-term acute care facility for aggressive physical and occupational therapy. Information regarding the patient's long-term outcomes was unavailable.

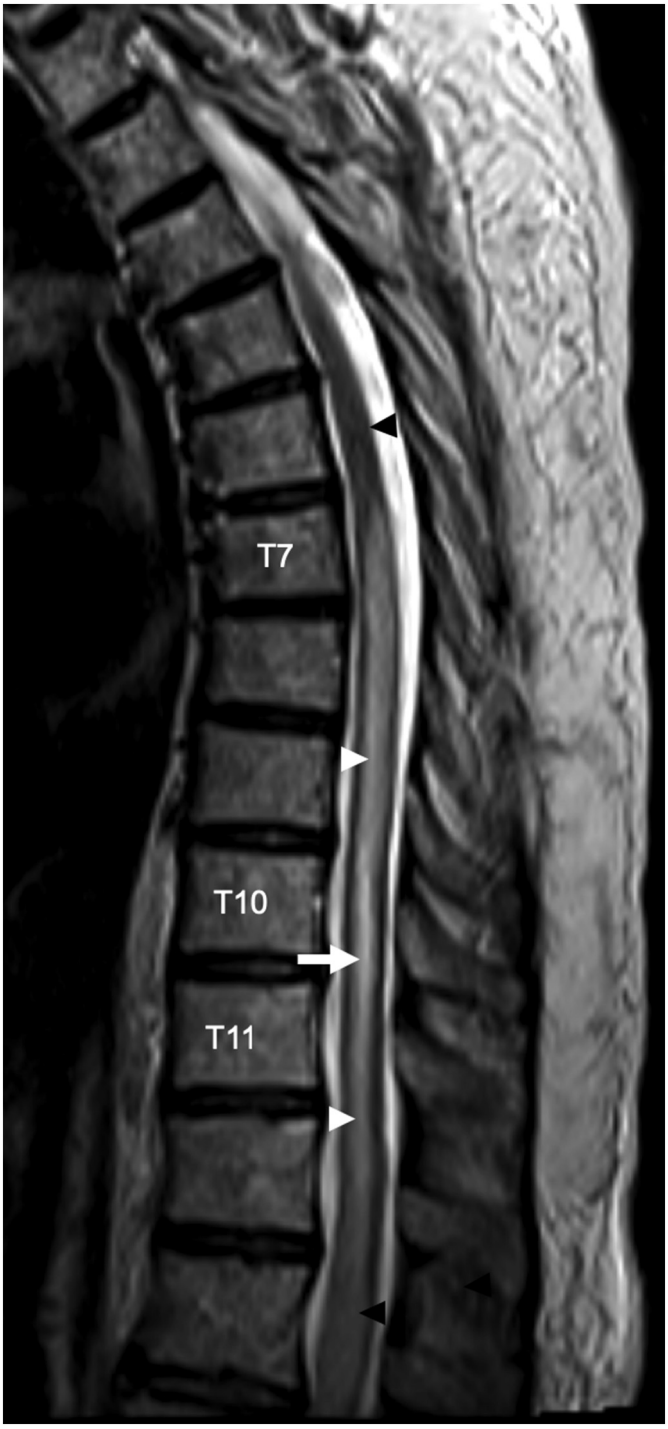

Fig. 2. Sagittal T2-W MRI of the spinal cord reveals slight cord expansion with increased T2 signal extending from T7 to L1 suggestive of acute spinal cord infarction. Severe ischemia and central necrotic area (curved arrow) are located at the level of T10-T11 (white arrow), the center of the longitudinal watershed zone. A gradient from central necrosis to peripheral sparing can also be appreciated in the sagittal view at this level. Partially ischemic lesions are seen rostral and caudal to the necrotic region (white arrowheads) until the levels of T7 and T12 where tissue is unaffected (black arrowheads).

3. Discussion

Watershed infarctions in the brain have been documented in the setting of severe prolonged hypotension and cardiac arrest [11,15]. Though less common, spinal cord watershed ischemia can also occur in the same setting [5,6,8–10,13,14,16–21]. Spinal cord infarction generally occurs due to two mechanisms: 1) direct arterial injury from triggering factors, such as aortic pathologies, surgery, dissection, embolus, vasculitis, and 2) infarct/ischemia following severe prolonged hypotension or cardiac arrest [10]. Clinically, spinal cord watershed ischemia presents similarly to infarction of the anterior spinal artery territory with bilateral loss of motor function and pain/temperature

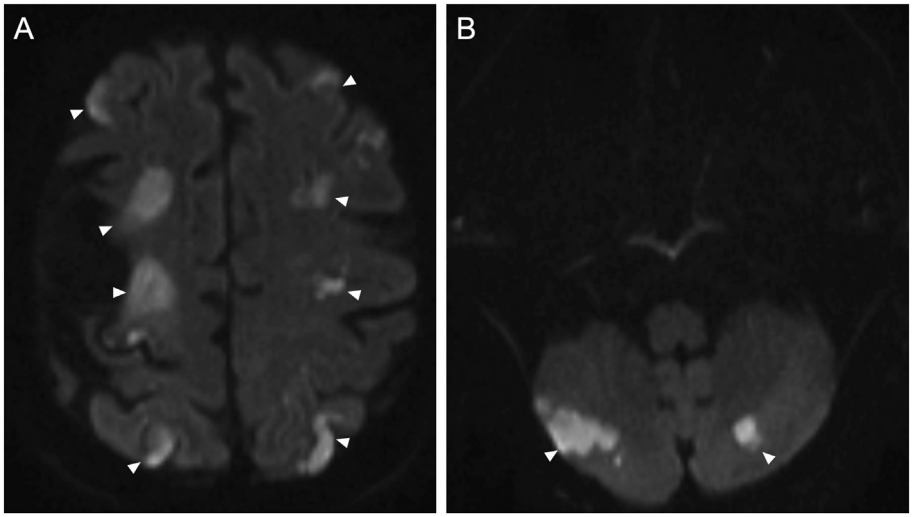

Fig. 3. Axial diffusion weighted MR images of the cerebrum and cerebellum. Hyperintense ischemic lesions (white arrowheads) are seen in the watershed territory of the supratentorial (A) and infratentorial (B) compartments.

sensation [1].

The watershed zone of ischemic vulnerability within the spinal cord has classically been positioned at the midthoracic level (T4–T6), a classification based largely on case reports and anatomic studies noting a relative hypovascularity of the cord at these levels [12,20,22]. However, the longitudinal watershed can also occur at T8-T9 and lumbosacral nerve levels and varies among individuals [19,20]. In our case, the longitudinal watershed centered at level T10-T11. Thoracoabdominal CTA revealed extensive intraluminal thrombus and bilateral occlusion of the patient's spinal arteries from T8 to L2, which supplies the T8-L2 territories (Fig. 4A). T7 and L3 spinal arteries were patent (Fig. 4). Level T10-T11 is located midway between T8 and L2 and had the least arterial flow. Therefore, this was the most disrupted region in our case. The location, extent, and severity of a longitudinal watershed lesion depends on several factors, such as intraluminal thrombosis, presence of spinal artery occlusion or high-grade stenosis, level of abnormal spinal arteries, and severity of hypotension or blood loss. Extensive spinal arterial abnormalities, such as chronic occlusion or dissection, may precipitate spinal cord watershed ischemia. Due the extensive natural collateral arterial network of spinal cord and proximity of the spinal cord to the heart and systemic circulation, it may be difficult to elicit watershed ischemia without multiple spinal arterial occlusions or stenoses. Our patient may therefore have been predisposed to watershed ischemia due to her severe atherosclerotic disease and occlusions affecting multiple spinal arteries and likely her Adamkiewicz artery, prior to surgery. Larger retrospective studies also implicate atherosclerotic disease as a primary risk factor in spinal cord infarction [3].

Spinal MRI is an essential tool for diagnosing acute ischemia in the spinal cord [4]. Cord ischemia is usually seen with high T2 signal and cord enlargement [1]. However, these findings are nonspecific and can be seen in transverse myelitis or other inflammatory pathologies. With the aid of DWI, significant cytotoxic edema and diffusion restriction in the setting of acute spinal cord pathology can help differentiate acute spinal cord ischemia from other cord abnormalities such as transverse myelitis [10]. DWI is a key diagnostic tool for brain ischemia, however, evaluation of spinal cord ischemia with this approach is challenging, and neither DWI nor T2-W image findings can differentiate watershed ischemia from acute anterior spinal artery ischemia secondary to local lesions, such as aortic abnormalities and dissection. DWI is difficult to apply to the spinal cord due to size of the spinal cord, CSF flow artefact, and susceptibility artefact of the bony spine. DWI of the spine also demands strong gradients and advanced hardware. Generally, diffusion weighted images at the spinal level are preferred in sagittal planes, as this view allows for larger coverage, shorter acquisition time, and less artefact [1,23].

Despite its promise in differential diagnosis of spinal cord ischemia, there are just two studies describing sensitivity, specificity and false negativity of DWI in spinal cord ischemia. Kumral et al. [24] found pathological DWI lesion in 12 out of 22 patients (55%) and pathological T2 lesion in 26 out of 36 (72%). There is no information about timing of MR study in this study. This study was done from 1998 and 2008 using a 1.5 T MRI machine. Artemis et al. [25] found abnormal DWI findings in all 7 out of 7 patients during first 24 h after onset of acute myelopathy symptoms. T2 signal abnormalities were also detected in all 7 out of 7 patients during first 24 h. Newer technology make account for the more consistent presence of positive imaging findings in this study compared to the study by Kumral et al. [24]. Additionally, in an animal model of spinal cord infarction involving bilateral embolization of T9-T11 intercostal arteries of dogs, ADC values were found to diminish continuously after embolization, falling to their lowest point at 24 h [23]. In our case, we could not see any DWI abnormality in imaging performed 18 h after symptom onset. Our MR examination was done with 1.5 Tesla MR machine installed almost 10 years ago. We believe the absence of positive findings on DWI in our study may be due to use of older imaging technology and imaging artefacts. Though less likely, temporal evolution of the lesion may also have also obscured indicators of ischemia. It is also possible that our generic DWI image acquisition parameters used for brain imaging were not suitable for evaluation of spinal cord cytotoxic edema with specific features.

Ishizawa et al. demonstrated the arterial subdivision of the central artery and peripheral arteries that account for the transverse watershed pattern [14]. The central artery is a branch of the anterior spinal artery and supplies most of the gray matter and adjacent white matter. The peripheral arteries are anastomosing pial arterial branches supplied by anterior and posterior spinal arteries; they encircle the cord and supply the posterior horn and white matter. The watershed lies in between the central and peripheral compartments [14]. In Ishizawa et al.'s histopathologic studies, spinal cord watershed ischemia demonstrated central gray matter coagulation and/or liquefaction necrosis and unchanged peripheral white matter. The transverse watershed zone, including peripheral gray matter and adjacent central white matter, showed significant ischemia [14]. The authors concluded that central gray matter necrosis is likely associated with selective vulnerability of the gray matter to ischemia, while the peripheral gray matter and adjacent central white matter are likely associated with intrinsic

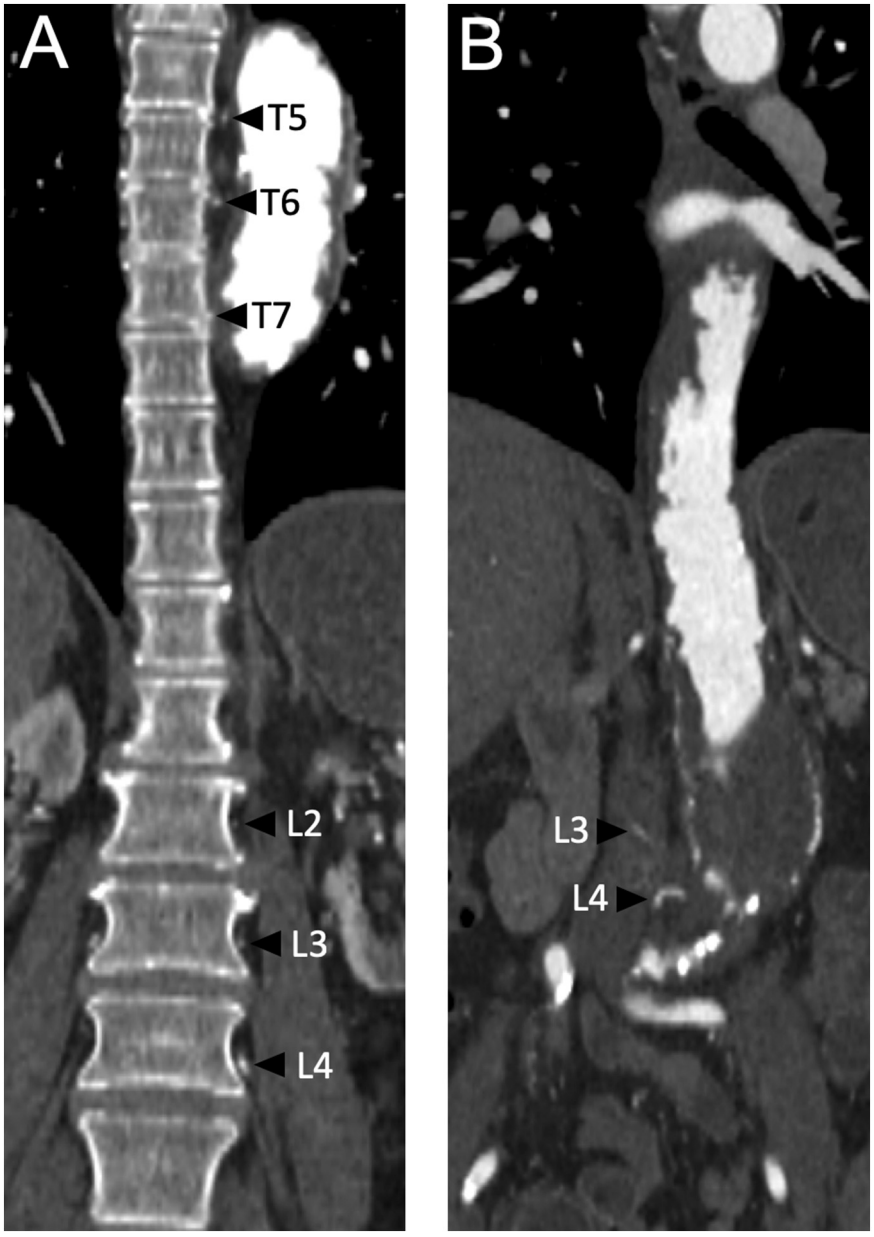

Fig. 4. Coronal CTA images revealed patent right sided spinal arteries at the level of T5, T6, T7 on the right (A) and also L3 and L4 in the lower abdomen (A, B). However, there is no evidence of spinal arteries seen between T7 and L2 level (A). Diffuse atherosclerosis of the abdominal aorta is also seen.

vulnerability of the watershed to systemic hypotension or low blood-flow states [14].

Our MR findings provide MRI correlates of the histopathology findings of spinal watershed infarction documented by Ishizawa et al. Very high T2 signal was present in the central cord in our patient at the level of T10-T11, possibly representing severe necrosis in the central gray matter; this level was the most affected region due to paucity of collateral arterial flow and selective vulnerability of gray matter to ischemia. There was circumferential mild-moderate increase T2 signal in the peripheral gray matter, and associated central white matter representing ischemic tissue in the transverse watershed. This is in agreement with studies postulating increased vulnerability of spinal gray matter to ischemia due to higher metabolic demands of this tissue relative to white matter [20]. Lastly, the peripheral white matter showed no abnormal T2 signal changes and remained unaffected. This signal pattern, consistent with watershed ischemia in the spinal cord parenchyma, has not previously described on MRI studies, and no other study to our knowledge has reported similar combined spinal, cerebral and cerebellar watershed lesion MRI findings following prolonged hypotension within the same patient. Only one case report has shown similar watershed pattern of ischemia in sagittal T2 weighted imaging. We found that the sagittal T2 image from this case study revealed longitudinal central hyperintensities, peripheral sparing, and moderate hyperintensity representing partial ischemia between these two zones. Like our case, this case also occurred following prolonged hypotension and large intraabdominal hematoma [13]. The authors of this study, however, did not address a three-phasic pattern of watershed ischemia reflecting that described in histopathologic studies. We believe our case to be the clearest reflection of the histopathological studies of watershed infarction, such as described previously described [14].

Spinal cord ischemia is generally related to emboli or plaques related to the aorta, therefore evaluation of arterial anatomy is also important in the setting of spinal cord ischemia [4]. Modern multi-slice CT machines provide fine details in evaluation of the aortic spinal arteries. MR angiogram is another choice for evaluation of spinal vasculature. In our case CT angiogram revealed extensive intraluminal thrombus and

bilateral occlusion of her spinal arteries from T8 to L2, including possible involvement of her Adamkiewicz artery.

In summary, spinal cord watershed infarction can occur as a devastating complication in the setting of atherosclerotic disease and hypotension. This article highlights unique MRI findings of watershed infarction in the spinal cord and the importance of its prompt detection and characterization for proper patient management. Future studies are needed understanding the interaction of factors, such as atherosclerotic disease and systemic hypotension, that may contribute to different patterns of spinal cord watershed infarction, as well as the imaging modalities best tailored to detect watershed infraction at different stages of natural history of the disease.

Funding

This research did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit sectors.

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