

torts the thoracic aorta is not much different in *Figure 4A* than it appears in *Figure 2A*.

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This case report by Citow et al. details the very unusual incidence of primary extranodal Hodgkin's disease of the spine presenting as spinal cord compression. These authors describe a case of initial misdiagnosis in today's medicolegal climate. The initial radiological and clinical

presentations were certainly consistent with tuberculosis of the spine and warranted presumptive treatment. The authors were very astute in pursuing the correct diagnosis after clinical progression despite appropriate medical treatment.

My initial surgical procedure would have been an anterior transthoracic approach. The posterior parascapular technique is certainly an option but results in a longer fixation segment (nine levels in this case) and probably would be associated with a higher risk of neurological complications in this patient. In general

terms, I believe that an anterior spinal lesion resulting in pathological focal kyphosis and anterior spinal cord compression should be corrected surgically via an anterior approach. This approach allows more effective, biomechanically sound correction of the sagittal plane deformity, in addition to being a more direct and probably safer approach, than the approach the authors used to treat the spinal cord compression.

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Conus Perimedullary Arteriovenous Fistula with Intracranial Drainage: Case Report

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OBJECTIVE AND IMPORTANCE: Perimedullary arteriovenous fistulae (AVFs) do not commonly present with subarachnoid hemorrhage or intracranial venous drainage causing neurological symptoms. We present a case with both of these features. The patient was inadvertently treated for an unruptured intracranial aneurysm before his true problem was recognized.

CLINICAL PRESENTATION: A 65-year-old man presented with sudden-onset lower-extremity weakness, diplopia, nausea, and dysarthria on the day of admission. A lumbar puncture documented subarachnoid hemorrhage, and imaging studies revealed a left middle cerebral artery aneurysm. It was noted during surgery that this aneurysm was unruptured, and the patient did not exhibit improvement after surgery.

INTERVENTION: Spinal angiography demonstrated a spinal perimedullary AVF feeding from the left T12 radicular artery; venous drainage extended rostrally into the posterior fossa venous system. The AVF was surgically occluded via a posterior laminectomy at the level of the AVF. After surgery, the patient's symptoms began to abate.

CONCLUSION: Conus perimedullary AVFs can have venous drainage that extends as far as intracranial veins, which can lead to confusing clinical findings because the symptoms may suggest an intracranial process, although the lesion is in the spine. Surgeons must be aware of this confusing presentation. (*Neurosurgery* 49:457-462, 2001)

Key words: Perimedullary arteriovenous fistula, Petrosal sinus, Subarachnoid hemorrhage, Veins

Intradural spinal perimedullary arteriovenous fistulae (AVFs) were first described by Djindjian et al. (6) and, by most accounts, are the second most common type of spinal arteriovenous malformation (AVM) (5, 7, 23). As with most other kinds of spinal AVMs, perimedullary AVFs can cause symptoms through steal, venous congestion, or, less commonly, hemorrhage or mass effect and usually present with either chronic, insidious, progressive myelopathy or sudden, catastrophic, subarachnoid or intramedullary hemorrhage (1, 4, 5, 10, 18, 21, 23). It is unusual, however, for patients to present with symptoms from both categories. It is even more unusual for a conus perimedullary AVF to drain intracranially and cause symptoms of brainstem dysfunction attributable to venous congestion. We report the first case in the literature of a patient with a conus perimedullary AVF with intracranial venous drainage that caused subarachnoid hemorrhage and bulbar signs; the patient also harbored an incidental intracranial aneurysm, and he was inadvertently treated for the aneurysm before the true cause of his symptoms (his perimedullary AVF) was discovered and obliterated.

CASE REPORT

A 65-year-old right-handed man presented to another hospital with sudden-onset lower-extremity weakness, diplopia, nausea, and dysarthria on the day of admission. The patient had complained of intermittent headaches for 2

weeks before admission; the initial evaluation indicated paranasal sinus disease, and the patient was treated by his primary care physician with nasal decongestants and azithromycin. On the day of admission, the patient had been moving about in his house when he suddenly felt his legs become weak and he fell to the ground. His sudden weakness was not associated with headache, back pain, loss of consciousness, or seizure, but the patient did lose control of his bladder and bowels. He was brought to the emergency room, and the evaluation there revealed marked lower-extremity weakness and hyporeflexia, without associated sensory deficits. The patient also had right abducens palsy, mild right facial weakness, and dysarthria, without tongue or palatal asymmetry. With further questioning, the patient reported 2 months of increased difficulty in walking. He had previously been able to easily walk 6 to 8 blocks; however, his endurance had decreased in the previous 2 months and he could then walk only 1 to 2 blocks before needing rest. This change was not associated with back pain, numbness, paresthesia, or changes in bowel or bladder function. The past history of the patient was significant for hypertension and excision of a malignant melanoma 3 years before admission, without any evidence of metastatic disease.

At admission, the patient underwent head computed tomography, which demonstrated no intracranial hemorrhage or brain abnormalities; paranasal sinus opacification was evident, consistent with his previous history of sinus disease. Cervical and lumbar magnetic resonance imaging (MRI) scans revealed degenerative joint disease and mild/moderate cervical and lumbar canal stenosis, to a degree insufficient to explain the profound deficits of the patient. There were prominent flow voids, suggesting engorged perimedullary vessels, but these did not attract attention at the time the MRI scans were obtained (Fig. 1). Brain MRI scans and magnetic resonance angiograms were obtained at the same time as the spinal MRI scans, and these demonstrated a left middle cerebral artery (MCA) aneurysm (not shown) and T2-weighted hyperintensity in the pons and medulla (Fig. 1). A lum-

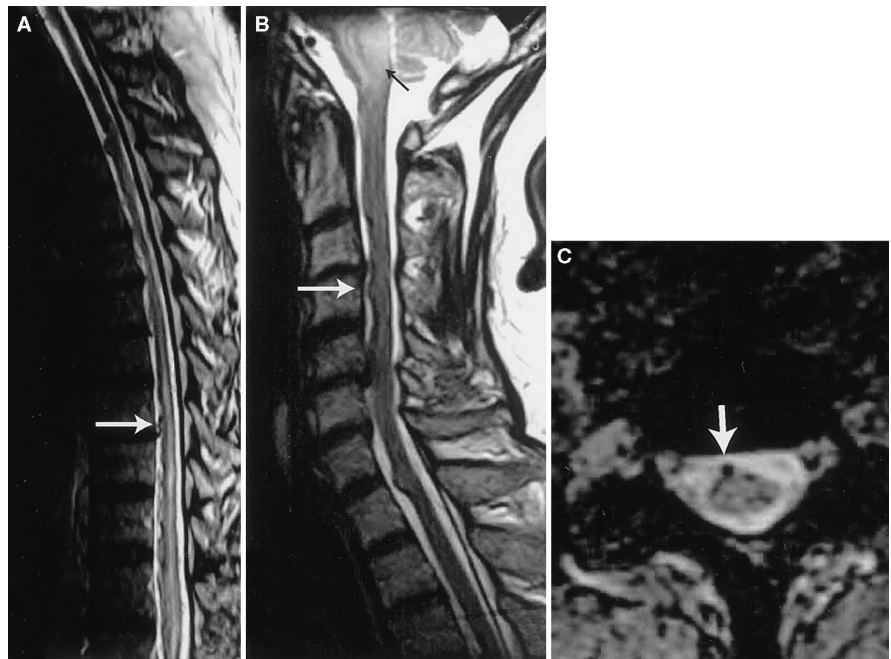


FIGURE 1. T2-weighted MRI scans of the thoracic (A) and cervical (B and C) spine, showing tortuous extramedullary flow voids (white arrows) consistent with engorged perimedullary veins extending the entire length of the spinal canal. B, sagittal MRI scan also demonstrating increased T2-weighted signal intensity within the pons and medulla (black arrow). C, axial T2-weighted scan of the caudal cervical spine, showing an enlarged extramedullary flow void on the anterior aspect of the spinal cord, consistent with an engorged perimedullary vein.

bar puncture was performed, and evaluation of the cerebrospinal fluid revealed 540,000 red blood cells, 1,200 white blood cells, and normal protein and glucose levels. Another lumbar puncture performed 2 days later demonstrated similar cell counts, and neither procedure was considered traumatic. Four-vessel cerebral angiography confirmed the presence of the left MCA aneurysm; no vasospasm or other cerebrovascular abnormality was noted. Although the findings were not classic indications of intracranial aneurysm rupture, the physicians caring for the patient at that time recommended surgical clipping of his aneurysm. The patient was taken to the operating room on his third hospital day, where the surgeon performed a left pterional craniotomy and clipped the left MCA aneurysm without difficulty. The surgeon noted that the aneurysm did not seem to have ruptured previously. After surgery, the patient developed no complications but he exhibited no improvement in his lower-extremity weakness

and he lost joint position sense in his right leg. Further review of the spinal MRI scans indicated a spinal vascular abnormality, and the patient was referred to our cerebrovascular service for spinal angiography and treatment.

At the time of arrival at our facility, the patient demonstrated persistent left facial weakness, dysarthria, and lower-extremity weakness. Cerebral and spinal angiograms were obtained. These demonstrated successful clipping of the left MCA aneurysm, with no residual lesion and no vasospasm. However, the spinal angiogram demonstrated a perimedullary AVF centered at L2, with supply at T12 from the left radicular artery contribution to the anterior spinal artery and additional posterior spinal artery supply at L1 and T10 from the right and left radicular arteries, respectively (Fig. 2). Interestingly, the venous drainage coursed cephalad, through perimedullary veins, throughout the entire length of the spinal canal to the cervicomedullary junction and finally emptied, through intracranial perimed-

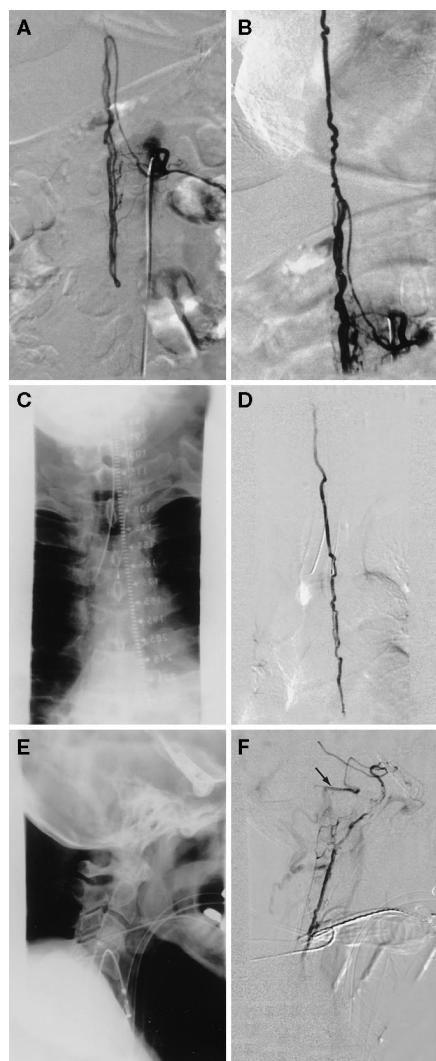


FIGURE 2. *A*, digitally subtracted, anteroposterior, spinal angiogram, showing injection of contrast material through the left T12 radicular artery contribution to the anterior spinal artery, as well as abnormally early emergence of contrast material into a perimedullary draining vein. *B*, slightly later phase of the same injection, at higher magnification, showing an abnormally early flow of contrast material from the anterior spinal artery into a perimedullary vein, ascending rostrally within the draining vein. *C*, anteroposterior fluoroscopic view of the thoracic region, to provide bony landmarks for orientation in the digitally subtracted image in *D*; this shows how contrast material injected through the left T12 radicular artery, as in *A*, leads to early abnormal emergence of contrast material in an ascending perimedullary vein that drains throughout the extent of the thoracic spine. *E*, lateral fluoroscopic view of the craniocervical region, to provide bony landmarks for orientation in the digitally subtracted image in *F*; this shows how contrast material injected through the left T12 radicular artery leads to emergence of contrast material in the perimedullary and perimesencephalic veins in the posterior fossa. The *black arrow* in *F* indicates the superior petrosal sinus, through which contrast material empties into the transverse-sigmoid sinus system on the left (anteroposterior view not shown).

ullary and perimesencephalic veins, into the petrosal sinus (Fig. 2).

Because of the progressive nature of the deficits of the patient and the clear evidence that this perimedullary AVF had both ruptured and caused venous congestion affecting his pons and mesencephalon, we recommended surgical obliteration of the fistula. The patient agreed, and he was taken to the operating room, where wide laminectomies at L1 and L2 were performed. After opening of the dura, the nerve roots of the conus were carefully separated, and the conus medullaris and filum terminale were identified. Using the large draining vein of the fistula as a map, we identified the fistula site on the anterior surface of the conus (Fig. 3). From this location, we were also able to identify and isolate the small posterolateral spi-

nal artery supply, which was coagulated and divided. The conus was rotated slightly, to yield a good view of the anterior spinal artery as it fed into the fistula, and the more proximal anterior spinal artery was identified and carefully dissected from the conus. The isolated segment of the anterior spinal artery feeding into the fistula was then occluded with a 5-mm AVM clip, and the fistula was noted to darken considerably. As an extra precaution, the draining vein was occluded close to the fistula with another 5-mm AVM clip. This completely isolated the fistula and occluded both its inflow and outflow. We then coagulated the fistula with bipolar cautery, to shrink it and prevent additional refilling.

The patient tolerated the procedure well, and postoperative angiograms

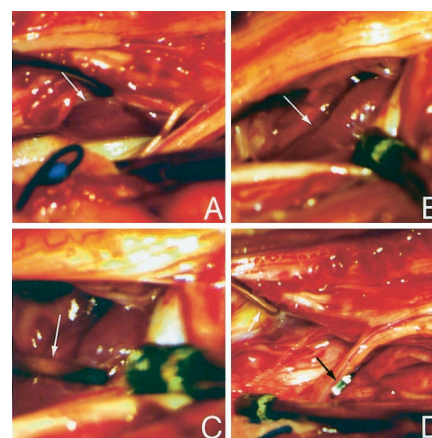


FIGURE 3. *A*, intraoperative view of the surgical field, showing the engorged draining vein of the perimedullary AVF, with arterialized blood (*white arrow*). *B*, higher-magnification view, showing the arteriovenous connection (*white arrow*). *C*, same view as in *B*, showing the aneurysm clip applied to the fistulous connection (*white arrow*). *D*, lower-magnification view of the surgical field, showing the same aneurysm clip as in *C*, applied to the arteriovenous connection, as well as a smaller clip applied to the draining vein more caudally (*black arrow*).



FIGURE 4. Digitally subtracted spinal angiogram with injection of contrast material through the left T12 radicular artery, showing that, after surgery, the fistulous connection was obliterated and no abnormal venous drainage persisted.

confirmed that the AVF had been obliterated (Fig. 4). By the time of discharge, the patient demonstrated improved strength and sensation in both legs and was able to walk cautiously with a walker. His dysarthria and facial weakness both resolved by the end of his hospitalization. He was transferred to a rehabilitation center 7 days after surgery and, at the follow-up examination 6 weeks after surgery, was walking with the assistance of only a cane.

DISCUSSION

This case is unusual because the patient's complaints and the diagnostic test results did not agree with the classic description of either intracranial subarachnoid hemorrhage attributable to an aneurysm or a spinal cord perimedullary AVF. In retrospect, this disagreement was attributable to two factors, i.e., the aneurysm was not the cause of this patient's symptoms and the perimedullary AVF had a very unusual drainage pattern, which made localization of the symptoms very difficult.

Spinal cord AVMs are a heterogeneous group of vascular anomalies, including both "true" AVMs and AVFs. In the past 15 years, recognition of differences in pathogenesis, anatomic features, pathophysiological characteristics, radiographic appearance, clinical manifestations, and optimal treatment for these various disorders has led to the construction of a classification system, as follows (4, 7, 8, 11, 23). Type I spinal AVMs are spinal dural AVFs with a direct connection between the dural branch of the spinal ramus of a radicular artery and an intradural medullary vein. Type II and III spinal AVMs are true glomus AVMs; type II (glomus) spinal AVMs are completely contained within the substance of the spinal cord, whereas type III (juvenile) spinal AVMs extend to involve surrounding paraspinous tissues. Type IV spinal AVMs are intradural leptomeningeal fistulous connections between an extramedullary artery and a dilated perimedullary vein and are commonly called perimedullary spinal cord AVFs (12). Subsequent work has attempted to subclassify perimedullary spinal AVFs on the basis of architecture and flow (2, 16–18), but that is not the focus of this report. The cause of perimedullary AVFs remains a mystery; most are considered congenital lesions (5), but cases resulting from traumatic injury have been reported (4, 9). These lesions become clinically manifest in the third to sixth decades of life and demonstrate no sex preference (4, 9, 18, 23). They are most commonly observed in the conus medullaris or cauda equina (4, 5, 9, 18, 23), although cervical and thoracic perimedullary AVFs have also been reported (13, 15, 18, 19).

As noted by Wong and Awad (23), spinal vascular malformations can mimic a diverse group of diseases affecting the central and peripheral nervous systems, and the syndromes associated with spinal vascular malformations are rarely stereotypical. This was particularly true in this case. The patient's cranial nerve deficits and the brain MRI results suggesting ischemia in the pons and medulla indicated an intracranial disorder, but the patient's leg weakness and hyporeflexia indicated a spinal cord lesion. In retrospect, spinal MRI was a correct first step in diagnosis, but the evidence for a spinal vascular anomaly was overlooked, perhaps because an intracranial lesion (the left MCA aneurysm) was discovered simultaneously. Nevertheless, the confusing nature of this patient's presentation is now clear; he had a spinal vascular lesion that, because of the extremely unusual pattern of venous outflow, was causing symptoms in two widely separated regions of the central nervous system. Ricolfi et al. (21) recently reviewed their experience with giant spinal perimedullary AVFs and did present the case of a 5-year-old boy with subarachnoid hemorrhage and progressive paraplegia secondary to a T12 perimedullary AVF of the conus with intracranial venous drainage. As we can best tell from the scant primary data provided in their report, however, their patient did not present with the same constellation of bulbar signs and spinal cord dysfunction. In addition, their case involved a giant spinal perimedullary AVF, and our case demonstrates that dramatic symptoms involving the entire neuraxis can occur even with a smaller lesion.

Accurate diagnoses and anatomic localization are needed for the appropriate treatment of spinal perimedullary AVFs. Myelography was historically used to identify and localize spinal perimedullary AVFs, but MRI and spinal angiography are now most commonly used to identify these lesions (4, 5, 7, 11). MRI can indicate spinal perimedullary AVFs by documenting serpentine flow voids in the subarachnoid extramedullary space; in addition, MRI can demonstrate subarachnoid hemorrhage or changes in the brain and spinal cord that suggest hemorrhage or venous congestion, as in this case. However, MRI does not adequately delineate the ana-

tomic features of the arterial feeders and venous drainage of these lesions. Spinal angiography is required to define the arterial and venous anatomic features of the fistulae sufficiently to guide therapy.

Spinal perimedullary AVFs are associated with grim prognoses if left untreated. Patients experience either progressive symptoms of spinal cord dysfunction attributable to presumed venous congestion or steal or a stuttering course punctuated by episodes of sudden neurological impairment attributable to rupture (1, 2, 5, 7, 8, 11, 12, 18, 23). Spinal perimedullary AVFs can be successfully treated by using either microsurgical occlusion or endovascular obliteration, and the relative merits of these two approaches are better described elsewhere (2–5, 10, 11, 13, 14, 17, 18, 20, 22, 23). We chose surgical occlusion because the major supply originated from the anterior spinal artery. Occlusion of this supply via the endovascular route can be treacherous, because it is difficult to control the deposit of embolic material; this puts the dominant arterial supply to the spinal cord at risk. In addition, the fistula was located in an area that is easily accessed via a standard surgical approach, with little risk of surgery-related morbidity.

CONCLUSIONS

Spinal perimedullary AVFs may have venous drainage that extends as far as intracranial veins; this can lead to confusing clinical findings because the symptoms may suggest an intracranial process, although the lesion is in the spine. This case emphasizes the need for awareness of this potentially confusing presentation. With the knowledge gathered from this case, surgeons should be better prepared to diagnose spinal perimedullary AVFs and to plan treatments for patients with these lesions.

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COMMENTS

This is an interesting case report that emphasizes referred neurological symptoms attributable to spinal cord arteriovenous malformations (AVMs). Although these classic data are well known (1, 2), they point to the importance of the venous drainage of the AVM and the absence of correlation between the location of the shunt (whatever its type or size) and the level of symptoms observed.

In this case report, the clinical history was typical of neither subarachnoid

hemorrhage nor a supratentorial lesion. Sudden weakness of both legs, with diplopia, nausea, and dysarthria, should have indicated a lesion in the pons and not a left middle cerebral artery aneurysm, especially in the absence of sudden intense headache. With modern radiological tools, more incidental lesions are now being discovered. It may be difficult to avoid acting on imaging findings that have no link to the patient history. After intraoperatively observing that the aneurysm was unruptured, the authors searched elsewhere for the cause of the subarachnoid bleeding diagnosed during the lumbar puncture, and they discovered a spinal perimedullary arteriovenous fistula (AVF). As stated above, this finding is classic, although rare. Spinal AVFs can exhibit venous drainage extending as far as intracranial veins. This case report should remind us to pay close attention to clinical signs and anamnesis.

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This is a report of a rare and very interesting case of a spinal perimedullary fistula in the low thoracic region that drained upward and secondarily arterialized pial veins in the posterior fossa, causing both hemorrhage and brainstem dysfunction. This case reminds me of one I reported with my colleagues in 1986 (1). Our patient almost underwent proton-beam irradiation to treat what was initially interpreted as a posterior fossa AVM. As we studied the films in more detail, we were struck by the fact that we observed only arterialized veins in the posterior fossa, without any real arterial input. Consideration of this finding in conjunction with the fact that the patient had exhibited slowly progressing paraparesis, which had been erroneously attributed to his "posterior fossa AVM," led us to speculate that perhaps there was a vascular lesion in the spinal cord that was draining upward and secondarily

arterializing the veins of the posterior fossa. In fact, with spinal angiography, the patient was proven to have a huge, upward-draining, perimedullary fistula in the low thoracic region, arterializing the entire venous system of the posterior fossa. Because the fistula was located anteriorly and was fed by the anterior spinal artery, I chose to treat it via a thoracotomy and a two-level corpectomy, with intradural direct closure of the fistula. The case presented by the authors is more complicated, because the patient had an incidental aneurysm and, in addition, was simultaneously exhibiting symptoms resulting from subarachnoid hemorrhage, from brainstem dysfunction, and apparently also from spinal cord involvement.

The reverse of this situation is also known to occur, and we have had experience with two cases in which the patient presented with myelopathy resulting from an intracranial dural fistula in the posterior fossa that drained downward, secondarily arterializing the perimedullary veins and causing progressive spinal

cord myelopathy resulting from venous congestion. The lesson from these cases is that, when intracranial arterialized veins are observed without an obvious intracranial AVM or dural fistula, one must look for the vascular abnormality in the spinal cord; conversely, when a patient presents with myelopathy and arterialized veins in the spinal cord without an obvious spinal AVM or spinal dural fistula, one must look for an intracranial dural fistula that drains downward.

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1. Heros RC, Debrun GM, Ojemann RG, Lasjaunias PL, Naessens PJ: Direct spinal arteriovenous fistula: A new type of spinal AVM—Case report. *J Neurosurg* 64:134–139, 1986.

The authors present the interesting case of a patient with a conus perimedullary AVF with intracranial drainage and bulbar signs. They describe a lesion in the region of the conus medullaris, with com-

ponents of both an AVF and an AVM. These lesions can have extensive venous drainage, and it is not surprising that they can extend as far as the intracranial venous system. We are in the process of describing a modified classification system for spinal cord vascular malformations and have considered it necessary to create a new subcategory, i.e., the conus AVM, given the unique nature of lesions in this region. Conus AVMs are location-specific lesions and, as mentioned above, exhibit features of both AVFs and AVMs of the spinal cord. In our experience, they can be both perimedullary and intramedullary. Additionally, they manifest with progressive myeloradiculopathy and subarachnoid hemorrhage. As in the case described by the authors, we have observed very complex patterns of venous drainage in association with these lesions.

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