



# Spontaneous Regression of De Novo Cavernous Malformation in the Spinal Canal after Hemorrhage: A Case Report

Minami Saura<sup>1</sup> Kiyoyuki Yanaka<sup>1</sup> Kuniyuki Onuma<sup>1</sup> Kazuhiro Nakamura<sup>1</sup> Nobuyuki Takahashi<sup>2</sup>  
Aiki Marushima<sup>3</sup> Eiichi Ishikawa<sup>3</sup>

<sup>1</sup> Department of Neurosurgery, Tsukuba Memorial Hospital, Tsukuba, Ibaraki, Japan

<sup>2</sup> Department of Radiology, Tsukuba Memorial Hospital, Tsukuba, Ibaraki, Japan

<sup>3</sup> Department of Neurosurgery, Institute of Medicine, University of Tsukuba, Tsukuba, Ibaraki, Japan

**Address for correspondence** Eiichi Ishikawa, MD, PhD, Department of Neurosurgery, Institute of Medicine, University of Tsukuba, Tsukuba 305-8577, Ibaraki, Japan (e-mail: e-ishikawa@md.tsukuba.ac.jp).

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## Abstract

Spinal cavernous malformations are rare vascular malformations with an unclear natural history, especially in de novo cases without prior radiation exposure or genetic predisposition. Although surgery is often recommended for symptomatic hemorrhagic lesions, spontaneous regression is extremely rare. A 74-year-old man presented with isolated neck pain after a minor head trauma. Computed tomography revealed a hyperdense lesion in the C2 spinal canal, and magnetic resonance imaging (MRI) showed a 16 × 9 × 20 mm intradural, intramedullary mass with protruding extramedullary hemorrhagic features, consistent with a cavernous malformation. A previous MRI 1 year earlier showed no abnormalities, confirming its de novo nature. Surgery was initially planned due to the risk of rebleeding. However, the patient's symptoms resolved spontaneously. A follow-up MRI 9 days later revealed significant regression, with continued involution observed over 5 years. This is the first reported case of spontaneous regression of a de novo spinal cavernous malformation, suggesting that in selected patients, conservative management with serial imaging may be safe in asymptomatic or minimally symptomatic patients showing early regression. Possible mechanisms include hematoma resorption, vascular thrombosis, or cerebrospinal fluid mediated clearance. Further studies are needed to identify predictive factors for regression and refine treatment strategies.

## Keywords

- ▶ cavernous malformation
- ▶ de novo formation
- ▶ hemorrhage
- ▶ regression
- ▶ spine

## Introduction

Cavernous malformations within the spinal canal are rare vascular malformations, significantly less common than their intracranial counterparts. Due to their rarity, the natu-

ral course of spinal cavernous malformations remains poorly understood.<sup>1</sup> While spontaneous regression has been rarely reported in intracranial cavernous malformations, no such cases have been documented in de novo spinal lesions, making their natural history particularly enigmatic.

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Symptoms often arise when the malformation causes bleeding or exerts pressure on surrounding spinal cord tissue, leading to pain, sensory disturbances, or motor deficits.<sup>2</sup> Surgical intervention is generally recommended, when the lesion becomes symptomatic after hemorrhage, primarily to prevent rebleeding and worsening neurological deficits.<sup>3</sup> However, both the optimal timing and surgical approach remain subjects of ongoing debate,<sup>4</sup> especially in anteriorly located spinal lesions, where technical difficulty and the risk of postoperative deficits are considerably higher. In such cases, even more meticulous clinical judgment is required to determine the most appropriate management strategy.

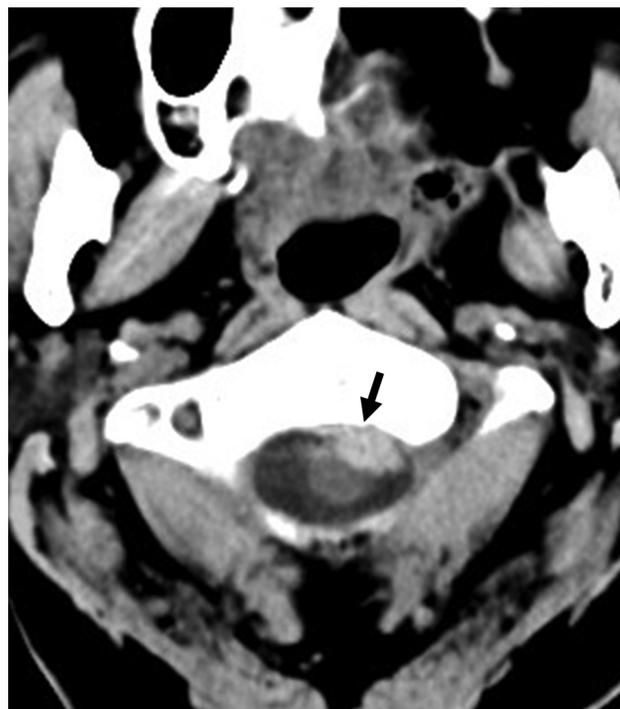
In this report, we present a highly unusual case of a de novo cavernous malformation in the cervical spine that demonstrated spontaneous regression following an initial hemorrhagic event. Prior imaging showed no abnormalities at the lesion site, confirming its classification as de novo formation. Although histopathological confirmation was not available, the imaging findings and clinical presentation strongly suggested a cavernous malformation. This case provides valuable insight into the natural behavior of spinal cavernous malformations and raises important considerations regarding surgical decision-making, particularly in cases where spontaneous regression may be possible.

## Case Description

A 74-year-old male was referred to our hospital with neck pain following an episode of head trauma. Upon examination, he exhibited no neurological deficits. His medical history was negative for prior radiation exposure, genetic predisposition to vascular malformations, or previous spinal lesions. Given the presence of isolated neck pain and no neurological symptoms, an initial computed tomography scan was performed, revealing a hyperdense lesion on the left side of the spinal canal at the C2 level (►Fig. 1). This finding prompted further investigation with magnetic resonance imaging (MRI) to characterize the lesion.

MRI revealed a 16 × 9 × 20 mm intradural, extramedullary mass at the C2 level, along with a smaller adjacent 7-mm intramedullary nodule (►Fig. 2). The lesion exhibited heterogeneous signal intensity on T2-weighted imaging, consistent with hemorrhagic cavernous malformations, and was associated with significant spinal cord edema. On T1-weighted imaging, the lesion appeared mostly isointense, and gadolinium-enhanced imaging showed almost no contrast enhancement. Given that MRI performed 1 year earlier for an unrelated condition showed no abnormalities at this site (►Fig. 3), the lesion was classified as a de novo formation. These imaging findings strongly suggested a cavernous malformation with a recent hemorrhage.

Due to the lesion's size, hemorrhagic features, and the potential risk of rebleeding leading to neurological deterioration, surgical excision was initially planned. However, while awaiting surgery, the patient's neck pain resolved sooner than expected. Follow-up MRI conducted 9 days later during pre-operative evaluation revealed significant regression of the

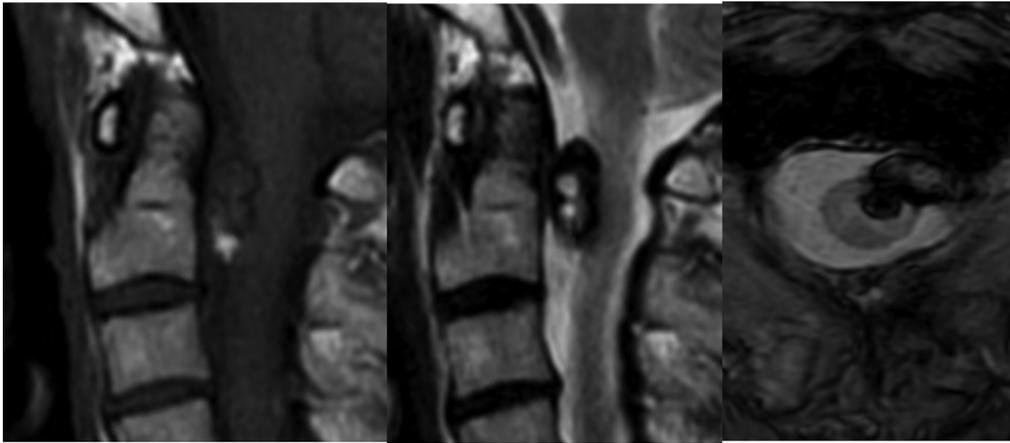


**Fig. 1** Computed tomogram at onset showing a hyperdense lesion (arrow) on the left side of the spinal canal at the C2 level.

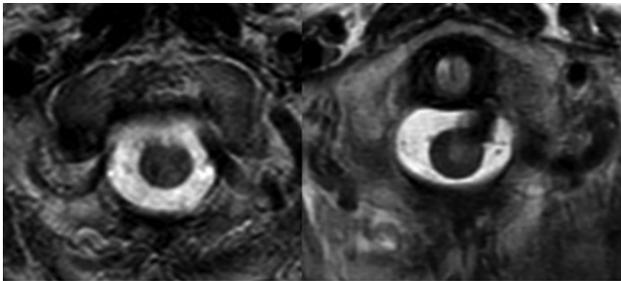
intradural hematoma, reduction of spinal cord edema, and partial shrinkage of the lesion itself (►Fig. 4). Given this unexpected improvement, the surgical plan was reconsidered, and a decision was made to opt for conservative management instead. Over the subsequent 5 years, serial MRIs showed a continuous decrease in the size of the cavernous malformation (►Fig. 4), with complete resolution of the hematoma and no further bleeding events. The patient remained entirely symptom-free throughout this period ►Fig. 5.

## Discussion

The present case provides unique insights into both the de novo formation and the spontaneous regression of cavernous malformations in the spinal canal. To our knowledge, no previous reports have documented the spontaneous regression of de novo spinal cavernous malformation, making this case a unique contribution to literature. While cavernous malformations more commonly develop in the intracranial compartment,<sup>1</sup> their occurrence in the spinal canal remains rare, and their natural history is poorly understood due to the frequent need for surgical intervention.<sup>5</sup> Spontaneous regression of cavernous malformations has been reported in 55% of intracranial cases,<sup>6</sup> which is likely the result of hemorrhage resolution.<sup>6</sup> Similarly, spontaneous regression has been observed in other intracranial vascular malformations, such as capillary hemangiomas and arteriovenous malformations, potentially due to thrombotic occlusion or inflammatory processes.<sup>7,8</sup> In contrast, regression in spinal cavernous malformations, particularly de novo lesions, has not been previously reported, highlighting the uniqueness of this case.



**Fig. 2** Magnetic resonance (MR) images at onset. T1-weighted (left) and T2-weighted (center) MR images showing a  $16 \times 9 \times 20$  mm intradural, extramedullary mass at the C2 level, along with a smaller adjacent 7-mm intramedullary nodule (right). The lesion exhibits nearly isointense signal intensity on T1-weighted image and heterogeneous signal intensity with a hemosiderin rim on T2-weighted image, consistent with a hemorrhagic cavernous malformation. The axial T2-weighted image (left) also showing intramedullary high-intensity suggestive of edema.

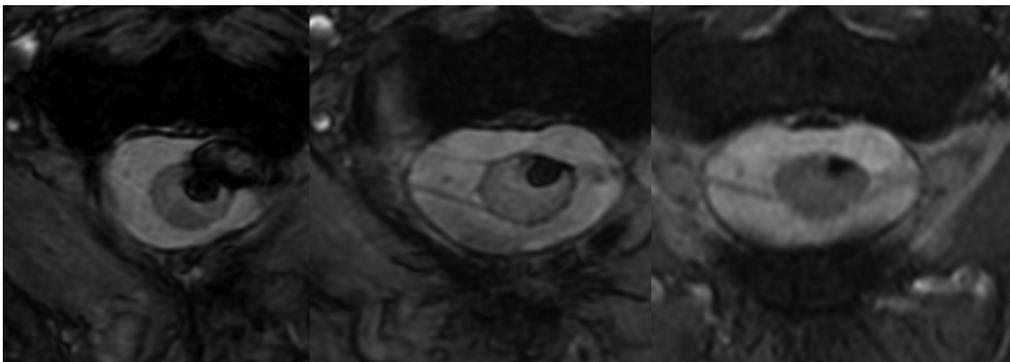


**Fig. 3** A T2-weighted magnetic resonance (MR) image taken approximately 1 year before the onset of the disease (left) and a T2-weighted MR image taken at the time of onset of the disease (right). There is no abnormality in the same area, suggesting de novo formation.

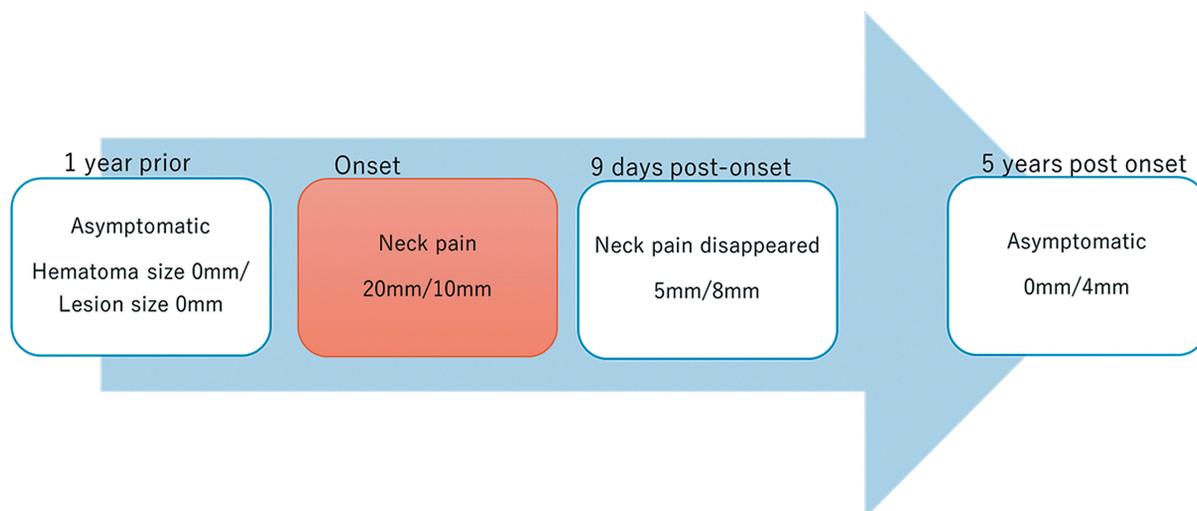
This case, in which a cavernous malformation developed spontaneously without known predisposing factors such as prior radiation therapy<sup>9</sup> or genetic predisposition,<sup>10</sup> and then regressed following hemorrhage, challenges conventional assumptions about the behavior of these vascular lesions. The de novo formation, occurring without radiation exposure or familial predisposition, further suggests poten-

tial roles for localized genetic mutations, dysregulation of angiogenic factors, or microenvironmental changes within the spinal cord as Retta and Glading stated in their study of cerebral cavernous malformation.<sup>11</sup>

The spontaneous regression observed in this case may involve several mechanisms, as seen in intracranial cavernous malformations. Hematoma resorption, well documented in cerebral lesions, likely reduced internal pressure within the malformation, facilitating its collapse.<sup>6</sup> Furthermore, mechanisms such as thrombotic occlusion of vascular lumens,<sup>12</sup> proximity of the lesion to the cerebrospinal fluid (CSF) space,<sup>13</sup> and age-related vascular fragility, may explain the sustained regression observed over 5 years. This theory is supported by the initial MRI findings, which suggested an acute hemorrhagic component, and a follow-up imaging, which showed a significant decrease in hematoma volume, surrounding edema, and lesion size. Regarding thrombotic occlusion within the vascular channels of the malformation, intravascular thrombosis has been documented in cerebral cavernous malformations<sup>12</sup> and could similarly contribute to lesion shrinkage in spinal cases. The formation of thrombi within the vascular spaces may restrict blood flow, leading to



**Fig. 4** T2\*-weighted images showing the regression of hematoma and intramedullary nodule. At onset (left), 9 days after onset (center), and 5 years after onset (right). By day 9, the hematoma and edema have significantly regressed, and the intramedullary nodule reduced in size; at 5 years, the intramedullary lesion remains stable with no recurrence.



**Fig. 5** Timeline showing changes in symptoms and the size of the hematoma and lesion. At symptom onset, both the hematoma and lesion were at their largest. Over time, a gradual reduction in size was observed. Five years after onset, the lesion has remained stable without any signs of enlargement.

the gradual involution of the lesion. Additionally, advanced age may influence this process, as reduced vascular elasticity and compliance could facilitate passive collapse of the cavernous structure over time. Furthermore, the lesion's proximity to the CSF space could have played a role in its regression. The presence of CSF might assist in the transport and absorption of cellular debris from the collapsed cavernous malformation,<sup>13</sup> accelerating lesion reduction. This could represent a unique form of lesion regression more likely to occur in cavernous malformations exposed to the CSF space. The present case expands this limited body of evidence and underscores the need to further investigate factors predisposing to both de novo formation and spontaneous regression, which may ultimately refine decision-making between surgical and conservative approaches.

The decision to perform surgery for spinal cavernous malformations requires an individualized approach, balancing the risks of intervention against the potential for spontaneous regression. Surgery, particularly for anteriorly located lesions, carries a significant risk of neurological deterioration, especially in patients with mild symptoms. While early surgical intervention may prevent rebleeding and further neurological deficits, it also precludes observation of the lesion's natural course. The unexpected regression seen in some cases suggests that close monitoring may be a viable alternative in selected patients. To optimize management, clear criteria are needed to determine when observation should transition to surgery. Regular neurological assessments and imaging follow-up are essential for detecting early signs of progression. If any deterioration occurs, prompt surgical intervention is warranted to prevent permanent deficits. In light of this case, clinicians should consider close observation with serial imaging in asymptomatic or minimally symptomatic patients with spinal cavernous malformations, particularly when early regression is

observed, as this strategy may help avoid unnecessary surgery in select cases.

There are several differential diagnoses for intradural masses, including nerve root hemangioblastoma, which shares certain radiological features with cavernous malformation.<sup>14</sup> However, careful evaluation of imaging findings—such as the presence or absence of cyst formation, enhancement patterns, evidence of hemorrhage, hemosiderin rim, and flow voids—can aid in differentiation.<sup>15,16</sup> Accurate preoperative diagnosis is essential to avoid unnecessary surgical intervention and to guide appropriate management. In our case, although histopathological confirmation was not obtained, and other diagnoses such as hemorrhagic neoplasms cannot be entirely excluded, the diagnosis was strongly supported by characteristic MRI features—including a hemorrhagic intradural extramedullary mass with a hemosiderin rim and lack of flow voids or cystic components—and its stable involution over 5 years. This extended follow-up period, while reinforcing diagnostic confidence, remains a limitation for drawing definitive conclusions about the long-term prognosis. Therefore, while delayed recurrence or complications are rare, extended surveillance should be considered in similar cases to monitor for any late-onset issues. This limitation must be acknowledged when interpreting our findings.

## Conclusion

This case highlights the rare phenomenon of spontaneous regression in de novo spinal cavernous malformation. While these lesions are typically treated surgically due to the risk of neurological decline, our findings suggest that some may regress without intervention. Possible mechanisms include hematoma resorption, vascular collapse, thrombosis, or interaction with CSF. Although based on imaging findings alone

and lacking histopathological confirmation, the diagnosis was supported by characteristic radiological features and stable involution over 5 years. The case also emphasizes that de novo formation and spontaneous regression may share underlying biological pathways related to vascular instability and remodeling. Given the uncertain long-term natural history and the possibility of late recurrence, extended follow-up is warranted. Conservative management with close imaging surveillance may be appropriate in select minimally symptomatic patients, particularly when early regression is documented.

#### Authors' Contributions

M.S. is the main author of the manuscript. K.Y. contributed as the supervisor and assisted with revisions. K.O., K.N., N.T., and A.M. served as advisors, while E.I. provided guidance as an advisor and also supported the revisions.

#### Conflict of Interest

None declared.

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