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Catastrophic Neurological Collapse Following Push-Up Exercise: A Rare Case of Spontaneous Hematomyelia Causing Acute Paraplegia in a Healthy Young Adult

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ABSTRACT

Spontaneous hematomyelia, or non-traumatic intramedullary spinal cord hemorrhage, is a rare and potentially devastating neurological condition. Unlike hematomyelia resulting from overt trauma, spontaneous cases occur in the absence of significant injury and present substantial diagnostic and therapeutic challenges. Etiologies are diverse and include spinal cord neoplasms, coagulopathies, anticoagulant use, spinal arteriovenous malformations (AVMs), radiation-induced vasculopathy, minor mechanical insults, and systemic inflammatory conditions such as COVID-19. Given its rarity and the high risk of permanent neurological impairment, timely recognition is essential. We report the case of a previously healthy 19-year-old male who developed sudden-onset lower limb weakness and back pain following a session of push-up exercises. Within 3 h, he progressed to complete paraplegia. Initial treatment at a local primary hospital was unsuccessful, and he was referred to Hawassa University Comprehensive Specialized Hospital on the fourth day of illness. Magnetic resonance imaging (MRI) of the spine revealed an intramedullary spinal cord hemorrhage consistent with spontaneous hematomyelia. Based on imaging features and clinical context, a presumptive diagnosis of juvenile spinal AVM was made; however, digital subtraction angiography (DSA) was not feasible due to financial and infrastructural constraints. The patient received conservative management, including supportive care and physiotherapy. Unfortunately, no neurological recovery was observed at nine-month follow-up. This case underscores the need to consider spontaneous hematomyelia in the differential diagnosis of acute flaccid paraplegia, especially in young, previously healthy individuals without trauma or anticoagulant exposure. It also raises awareness of physical exertion, such as upper-body resistance exercise, as a potential trigger for spinal AVM rupture. Moreover, the case highlights the significant impact of limited diagnostic and therapeutic resources in low-income settings, which may hinder timely intervention and worsen neurological outcomes.

1 | Introduction

The term hematomyelia refers to an intramedullary hemorrhage, defined as bleeding confined within the parenchyma of the spinal cord. This rare entity is a neurological emergency due to its potential for rapid and irreversible neurological deterioration [1, 2]. Hematomyelia can be categorized into traumatic and non-traumatic (spontaneous) subtypes, with traumatic causes being

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Summary

- Spontaneous hematomyelia, though rare, should be considered in the differential diagnosis of acute paraplegia, particularly in young, healthy individuals without trauma or anticoagulant use.
- This case highlights a catastrophic spinal arteriovenous malformation (AVM) rupture likely triggered by physical exertion (push-up exercise), resulting in irreversible paraplegia.
- Limited access to advanced diagnostic tools such as spinal angiography in low-resource settings significantly hampers timely intervention and worsens neurological outcomes.
- Heightened clinical awareness and early imaging are essential to improve patient prognosis.

the most prevalent, typically resulting from spinal cord injury, procedures such as lumbar puncture, or surgical interventions [3].

Spontaneous hematomyelia, though rare, presents a significant diagnostic and therapeutic challenge due to its diverse etiology and nonspecific clinical presentation [4]. Known causes include coagulation disorders (e.g., hemophilia, von Willebrand disease, thrombocytopenia, disseminated intravascular coagulation) [5], vascular malformations (such as arteriovenous malformations, cavernomas, and dural arteriovenous fistulas) [6–8], and neoplastic conditions, including ependymomas, astrocytomas, and metastases [9].

Furthermore, inflammatory and infectious conditions such as myelitis, syringomyelia, arachnoiditis, meningomyelitis, and syphilis have been implicated [10, 11]. Cases of spontaneous hematomyelia have also been documented in association with aortic aneurysms, autoimmune vasculitides, and anticoagulation therapy, particularly with warfarin or direct oral anticoagulants (DOACs) [12, 13].

More recently, COVID-19 has emerged as a potential contributor, likely through its prothrombotic and inflammatory effects on the vasculature, with rare cases of spontaneous spinal cord hemorrhage reported during or following SARS-CoV-2 infection [14, 15]. The clinical presentation is often atypical, and patients may exhibit sudden-onset back pain, rapidly progressing motor and sensory deficits, autonomic dysfunction, and paraplegia, mimicking conditions like transverse myelitis or Guillain-Barré syndrome [16].

Among all causes, spinal vascular malformations, particularly arteriovenous malformations (AVMs), play a prominent role in younger patients. These congenital lesions may remain asymptomatic until a sudden rupture occurs, leading to acute hematomyelia with catastrophic outcomes [17, 18]. Typically, spinal AVMs present with gradually progressive myelopathy, but rupture into the spinal cord parenchyma results in acute and severe neurologic compromise [19].

Our case is unique in that it highlights a potential mechanical trigger: a push-up exercise. Although most reported cases of spontaneous hematomyelia occur in the absence of exertion and while the patient is at rest, this case suggests that sudden upper-body exertion may precipitate bleeding in patients with latent vascular lesions. This rare association underscores the importance of considering physical activity-induced AVM rupture in the differential diagnosis of acute myelopathies [20–22].

2 | Case History and Examination

A 19-year-old male arrived at a local primary hospital with a two-day history of bilateral lower extremity weakness. The weakness was symmetric, ascending, and had a sudden onset, reaching complete paralysis of the lower limbs within approximately 1 h. The onset occurred within 3 h of performing push-up exercises. He also experienced associated symptoms including sweating, upper and lower back pain, and difficulty with urination and defecation. He had no previous medical history and no family history of a similar incident.

After spending one day at the primary hospital, he was referred to our university hospital with a working diagnosis of paraplegia secondary to transverse myelitis. Upon arrival, his vital signs were within normal limits. On neurologic evaluation, muscle power was 0/5 in the lower extremities and 5/5 in the upper extremities. Muscle tone was hypotonic in the lower extremities and normotonic in the upper extremities. Bilateral deep tendon reflexes were reduced to 1/4 at the knees and ankles, while reflexes in the biceps, triceps, and brachioradialis were 2/4. Sensory examination revealed impairment of all sensory modalities below the nipple line, approximately at the T4 level. The remainder of the general physical examination revealed no abnormalities.

3 | Differential Diagnosis, Investigation, and Management

3.1 | Differential Diagnosis

1. Transverse Myelitis—This is a common initial consideration for acute non-compressive myelopathy. It typically presents with bilateral motor, sensory, and sphincter deficits over hours to days. However, transverse myelitis often follows a viral illness or autoimmune trigger and may show T2-hyperintense cord lesions without evidence of hemorrhage on MRI [11, 13].
2. Spinal Cord Tumors (e.g., ependymomas, astrocytomas) – These may cause progressive or acute symptoms due to hemorrhage into the tumor or surrounding cord. Hemorrhagic transformation in intramedullary neoplasms can mimic spontaneous hematomyelia [9].
3. Spinal Arteriovenous Malformations (AVMs) – These congenital vascular lesions can rupture spontaneously or after minor exertion, leading to intramedullary hemorrhage. Juvenile AVMs are particularly implicated in young individuals and are a leading cause of spontaneous hematomyelia [6–8, 17–19].
4. Spinal Cavernomas—These vascular malformations may present with chronic symptoms or acute neurological deterioration when bleeding occurs. MRI may show a

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"popcorn-like" appearance with mixed signal intensities [6, 7].

5. Hemorrhagic Myelitis—Rarely, inflammatory causes such as neuromyelitis optica spectrum disorders (NMOSD) or viral infections (e.g., herpes simplex, cytomegalovirus) can result in hemorrhagic necrosis of the spinal cord [10, 11].
6. Anticoagulant-induced Hematomyelia—Warfarin and direct oral anticoagulants can cause spontaneous bleeding into the spinal cord, especially in patients with underlying vascular lesions [12, 13].
7. Spinal Epidural Hematoma—Presents with sudden severe back pain and neurologic deficits, but typically shows an epidural collection compressing the cord rather than an intramedullary lesion [3].
8. Vascular Events (e.g., Spinal Cord Infarction) – Anterior spinal artery syndrome may present acutely with paraplegia, pain, and sphincter disturbance, but MRI findings and lack of hemorrhage help differentiate it [3, 23].
9. Guillain-Barré Syndrome (GBS) – Though often considered in cases of acute flaccid paralysis, GBS typically spares sensory level and bowel/bladder involvement, and MRI of the spine is usually normal or shows enhancement of nerve roots [16].

3.2 | Investigations

Following initial laboratory evaluation, the patient's serum electrolytes, erythrocyte sedimentation rate (ESR), renal function tests, liver function tests, and total blood count were all within normal limits. The coagulation profile showed a prothrombin time (PT) of 14 s, an international normalized ratio (INR) of 0.99, and a partial thromboplastin time (PTT) of 26 s, which were all within the standard reference range for our institution. Screening for infectious markers, including HBsAg, retroviral infection (RVI), and anti-HCV, yielded negative results.

An MRI of the entire spine was obtained and the findings are presented in Figures 1 and 2. Based on the MRI results and the clinical presentation, digital subtraction angiography (DSA) of the spinal cord was planned to further evaluate for underlying vascular malformation. However, due to financial constraints, the patient was unable to undergo the procedure. In our country,

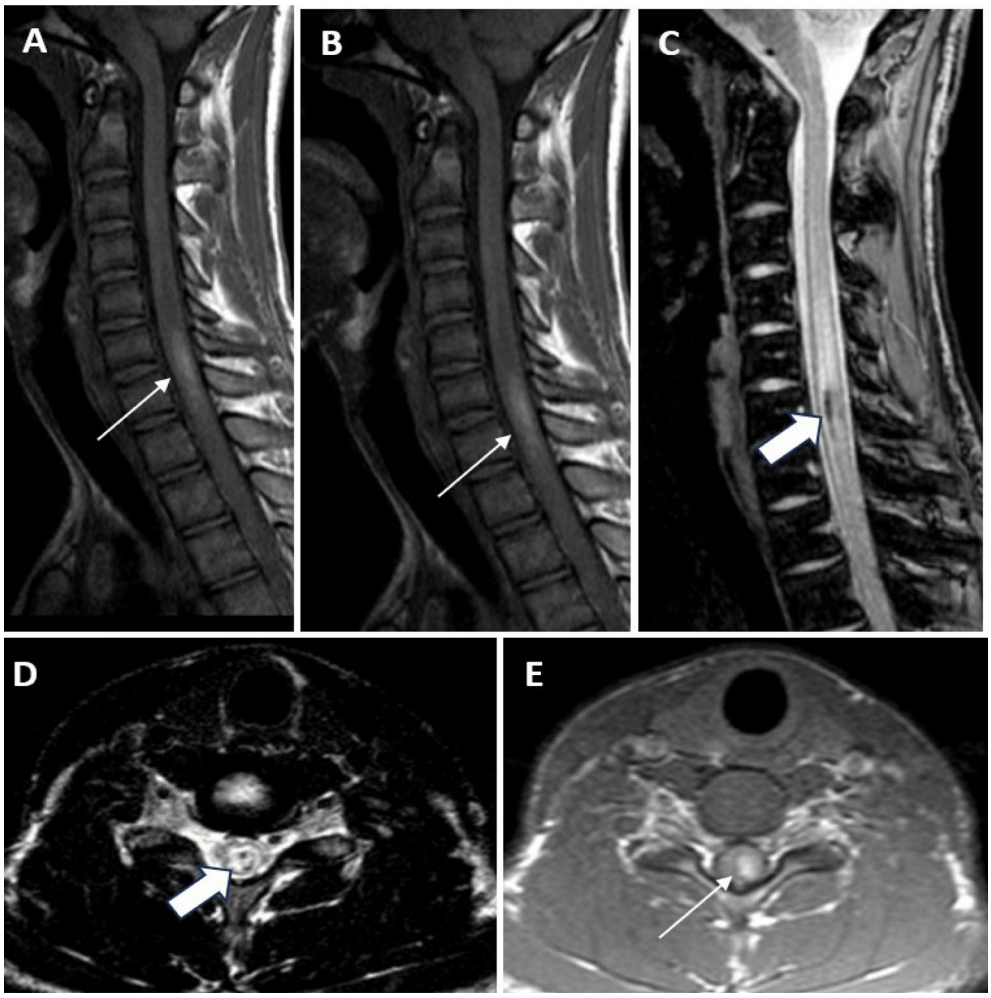


FIGURE 1 | Sagittal and axial MRI images of cervical spine of a patient with intramedullary hemorrhage showing short segment intramedullary lesion in the cervical spinal cord from C6 to C7-Th1 level with intrinsic T1 hyper intensity (thin arrow in A) and dark T2 signal intensity (thick arrow in D). No contrast enhancement (B). Internal blooming artifact is present in the sagittal gradient echo sequence (thick arrow in C). Axial T1-weighted image at the C7 level demonstrating intrinsic T1 hyper intensity (thin arrow) within the spinal cord, consistent with intramedullary hemorrhage (E).

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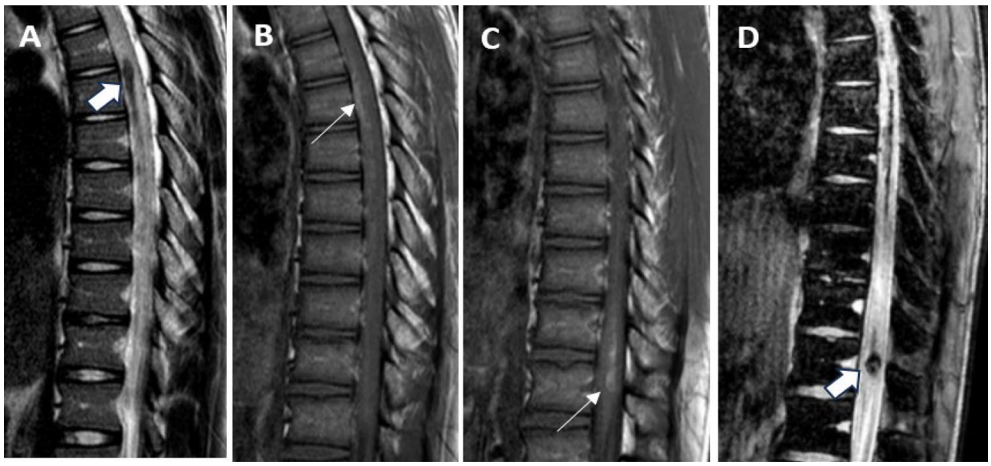


FIGURE 2 | Sagittal MRI images of thoracic spine of a patient with intramedullary hemorrhage showing long segment intramedullary lesion in the thoracic spinal cord from Th5-Th6 to conus medullaris level having intrinsic T1 hyper intensity (thin arrow in B and C) and dark T2 signal intensity (thick arrow in A). It has susceptibility artifact on gradient echo sequence (thick arrow in D).

DSA is only available at a single private center located in the capital city, which limited access. Given the clinical and radiologic findings, the diagnosis of spontaneous hematomyelia most likely secondary to juvenile arteriovenous malformation (AVM) was made.

4 | Treatment

The patient was initiated on deep vein thrombosis (DVT) prophylaxis with subcutaneous heparin at a dose of 5000 IU twice daily. In addition, bedside physiotherapy was commenced to support early rehabilitation. The patient remained hospitalized for 13 days, during which time his condition remained stable. He was subsequently discharged with instructions to continue physiotherapy at home and was scheduled for follow-up at our neurology clinic. Over a nine-month follow-up period, there was no significant improvement in his neurological status, and he continued with home-based physiotherapy.

5 | Conclusion and Results (Outcome and Follow-Up)

Spontaneous hematomyelia, though exceedingly rare, is a diagnostically critical entity in patients presenting with acute-onset paraplegia. Clinicians must maintain a high index of suspicion, particularly in young patients with rapid progression and no history of trauma.

This case emphasizes the potential role of physical exertion as a triggering factor in individuals harboring asymptomatic spinal vascular malformations. Equally, it underscores the systemic inequities in healthcare infrastructure, especially regarding access to advanced neuroimaging and interventional radiology in low-resource countries.

The patient remained hospitalized for 13 days and was discharged with instructions for home-based physiotherapy and follow-up in the neurology clinic. During 9 months of follow-up, there was no significant neurological improvement.

Timely referral, imaging, and appropriate management—when accessible—are essential to preventing permanent neurological sequelae. Further studies are needed to explore modifiable risk factors, improve resource allocation, and optimize early recognition and diagnostic pathways in similar healthcare settings.

6 | Discussion

This case illustrates the devastating impact of spinal AVM rupture, culminating in spontaneous hematomyelia and irreversible paraplegia. Several case series have similarly reported poor neurological outcomes in patients with spinal AVMs, particularly in the absence of prompt diagnosis and surgical or endovascular intervention [6, 17, 24].

The pathophysiological mechanism involves the rupture of a fragile vascular nidus within the spinal cord parenchyma, leading to acute hemorrhage and mass effect, which compromise the spinal cord's vascular supply and lead to ischemia, necrosis, and rapid neurologic decline [23]. The typical clinical course involves abrupt, intense spinal pain, followed by motor, sensory, and autonomic dysfunction within hours [3, 12]. This temporal profile closely mirrors that of our patient.

One of the major challenges in managing such cases, particularly in resource-limited settings, is the unavailability of advanced imaging modalities. In high-income countries, the diagnostic approach includes contrast-enhanced MRI, MR angiography, and digital subtraction angiography (DSA), the latter being the gold standard for characterizing spinal AVMs and guiding intervention [7, 19, 25]. However, in low-income nations like ours, even MRI access may require travel over hundreds of kilometers, and DSA is often unaffordable or unavailable in public hospitals.

Moreover, due to overlapping clinical features, these cases are frequently misdiagnosed as transverse myelitis, infectious myelopathy, or compressive myelopathy, leading to delays in definitive treatment [11, 13, 26]. Our patient was initially labeled

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with transverse myelitis, which underscores the importance of including vascular causes in the differential diagnosis of acute flaccid paraplegia, especially in young adults with rapid progression and back pain.

Timing of intervention remains a major prognostic determinant. Literature strongly supports that early surgical decompression or embolization, when indicated, significantly improves neurological outcomes and prevents permanent disability [3, 6, 17]. In our case, although surgery was not feasible, the fact that imaging was obtained relatively promptly highlights the potential for improving diagnostic efficiency even in under-resourced systems.

This case also calls attention to the role of physical exertion in precipitating hemorrhage in individuals with unrecognized AVMs. Activities that increase intrathoracic pressure, such as Valsalva maneuvers, straining, or intense upper-body workouts, may transiently raise venous pressure in spinal vessels, potentially triggering rupture in preexisting lesions [20, 21, 27]. Given the absence of similar prior reports in our region, this association merits further exploration through prospective registries and clinical reporting networks.

Author Contributions

Yegzeru Belete: conceptualization, data curation, validation, writing – original draft, writing – review and editing. **Abera Kuma:** conceptualization, supervision, writing – review and editing. **Amanuel Anegagregn:** conceptualization, data curation, writing – original draft, writing – review and editing. **Abdulkerim Girma:** visualization, writing – review and editing.

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We are appreciative that our patient gave us permission to compile this case report.

Ethics Statement

This case report was exempt from institutional ethical approval. It was prepared in accordance with the CARE guidelines. The study protocol was reviewed by the Institutional Review Board of Hawassa University College of Medicine and Health Sciences, which determined that ethical approval was not required.

Consent

The patient provided a written informed consent that permitted the publication of the case and case-related images.

Conflicts of Interest

The authors declare no conflicts of interest.

Data Availability Statement

The data used to comprise this case report is shared in the case presentation. Further data sharing is not applicable. Enquiries regarding the case report can be directed to the corresponding author.

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