Clinical Case Reports

ISSN: 2675-3626

Chronic Cervicothoracic Extradural Hematoma Presenting With Progressive Neck Flexion Deformity in an Adolescent: A Rare Case Report

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Background: Spinal epidural hematomas (SEHs) are rare but potentially a fatal cause of spinal cord compression. This report describes a case of chronic cervicothoracic SEH presenting with progressive neck flexion deformity.

Case presentation: We present a 16-year-old girl with a 5-month history of progressive neck pain, flexion deformity, and features of myeloradiculopathy. Neurologic examination revealed hyperreflexia (3+), bilateral extensor plantar responses, diminished right grip strength, right-sided limb weakness (4/5 power) and spastic gait. Magnetic resonance imaging (MRI) showed a C6-D1 epidural collection with T1 hypointensity and T2 hyperintensity causing spinal cord compression. She underwent C5–D1 laminectomy with complete hematoma evacuation. Following complete evacuation of the hematoma and excision of this sac-like epidural layer, a second true dural mater was visualized. Histopathology confirmed chronic hematoma with sac like epidural layer with neovascularization and fibrous membrane formation. Postoperatively, the patient achieved complete neurological recovery (5/5 power and normal bilateral grip strength) with no recurrence at six months follow up.

Conclusion: Chronic cervicothoracic SEH should be considered in adolescents presenting with progressive neck pain, deformity, and myeloradiculopathy, even without trauma. Identification of a hematoma membrane resembling dual dura suggests chronic SEH features and is important for achieving complete evacuation. Prompt MRI evaluation, and timely surgical decompression can achieve complete neurological recovery.

Keywords: Spinal epidural hematoma, Cervical spine, Adolescent, Spontaneous hematoma, Spinal cord compression

INTRODUCTION

Spinal epidural hematomas (SEHs), bleeding into the spinal canal's epidural space, are rare but potentially fatal causes of spinal cord compression and neurological deficits[1, 2]. The estimated incidence of SEHs is 0.1 per 100,000 individuals per year[3]. SEHs typically occur in the fourth or fifth decades of life with slight male predominance[4, 5]. Most cases present suddenly with severe back or neck pain followed by progressive neurological deficits[2]. Typical risk factors include trauma, underlying coagulopathy, anticoagulant therapy, vertebral hemangiomas, hypertension and iatrogenic[2]. Compared with the thoracolumbar regions, cervical spine involvement is rare because of the narrow spinal canal and its proximity to brainstem, which increase the risk of neurological deficits[6]. Magnetic resonance imaging (MRI) is the preferred diagnostic modality as it allows direct visualization of the spinal cord and extent of compression[2]. Patients with minimal symptoms may be managed with conservative treatment, while those with moderate to severe or progressive deficits require urgent surgical decompression and hematoma evacuation[2]. We report a rare case of chronic cervicothoracic SEH in a 16-year-old girl presenting with progressive neck flexion deformity and myeloradiculopathy.

CASE REPORT

A 16-year-old female with no significant medical history or trauma presented to the emergency department in the month of June, with a 5-month history of progressive neck pain. The pain was insidious in onset, which gradually progressed and then worsened despite rest and analgesic medication. About two months after symptoms onset, she developed neck deformity that gradually progressed to a fixed flexion deformity over the subsequent 2 months. Any attempt at extension caused excruciating pain which radiated bilaterally to the upper limbs (right > left). At the onset of illness, she reported episodic vomiting (2–3 times daily for 10 days) that resolved with symptomatic treatment. One week before admission, vomiting recurred at the same frequency (2–3 episodes per day) and progressively increased to five episodes on the day of presentation. She

Submitted: 11 December 2025
Accepted: 09 January 2026
Published: 19 January 2026

reported no history of trauma, fever, cough, headache, neck rigidity, or any other constitutional symptoms.

Clinical examination

The patient was moderately built and nourished with stable vital signs (blood pressure 110/70 mmHg, pulse 90/min, oxygen saturation 99% on room air). Systemic examinations were unremarkable. Routine blood investigations were within normal limits. Neurological examination revealed the patient was conscious, coherent, and oriented (Glasgow Coma Scale of E4V5M6; Mini-Mental Status Examination score was 30/30). Cranial nerve examination was normal with pupils equal and reactive to light. Motor examination demonstrated spasticity in all four extremities. Grip was markedly diminished on the right with pain radiating to the right upper extremity. The patient had bilateral extensor plantar responses (positive Babinski sign), hyperreflexia (3+) in all limbs, and right-sided upper and lower limb weakness (4/5 power). The patient's cognitive function remained normal, and a neurological test showed no indications of meningitis (negative Kernig's and Brudzinski's signs). The patient was ambulatory but with spastic gait limited by fixed neck flexion deformity.

Neuroimaging findings

MRI of the cervical spine with whole-spine screening and contrast studies revealed epidural collection extending from C6 to D1, causing significant compression of spinal cord and thecal sac. The lesion appeared hypointense on T1-weighted image and hyperintense on T2-weighted images, with peripheral contrast enhancement, features suggestive of chronic epidural hematoma (Figure 1 and 2).

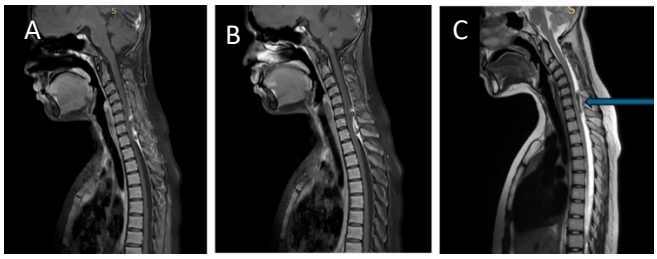

Figure 1. Preoperative MRI of the cervicothoracic spine. (A, B) Post-Contrast T1Weighted Sagittal view (C) T2 Weighted Sagittal view

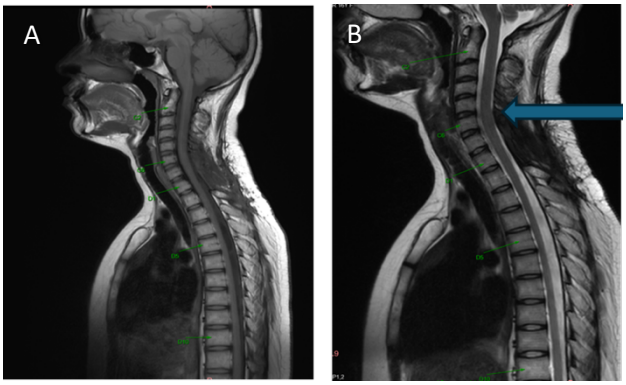

Figure 2. Postoperative MRI at 6-month follow-up. (A) T1-Weighted and (B) T2-Weighted sagittal view showing complete epidural evacuation with restoration of normal spinal cord morphology.

Surgical management

The patient was taken for emergency cervicothoracic spinal exploration and underwent C5–D1 laminectomy under general anesthesia. Intraoperatively, a thick-walled, sac-like structure resembling dura was identified overlying a chronic epidural hematoma extending from C6 to D1.(Figure 3) After complete evacuation of the hematoma and excision of this sac-like epidural layer, a second true dural mater was visualized. (Figure 4) The dura was intact with no evidence of tears or cerebrospinal fluid leak. No calcification was identified within the evacuated hematoma.

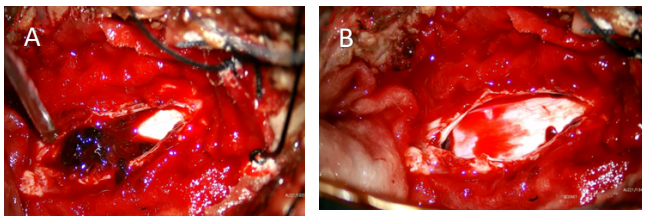

Figure 3. Intra-operative images suggestive of thick epidural layer under which there lies epidural hematoma. A) epidural hematoma after exposure B) evacuation of hematoma

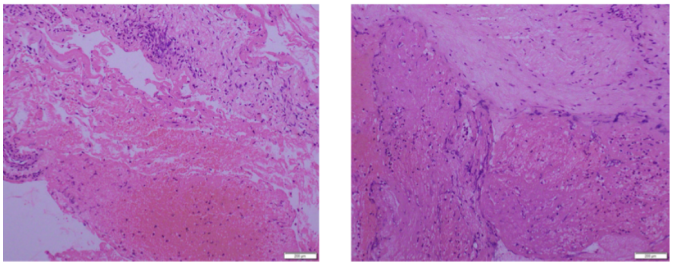

Figure 4. Histopathological features of chronic epidural hematoma (H&E, ×100) A thick epidural layer that was opened to evacuate the epidural hematoma

DOI: 10.46900/apn.v8i1.328

Postoperative course

Postoperatively, the patient recovered uneventfully. By postoperative day 5, she was ambulatory and able to perform daily activities independently, with significant improvement in neck posture, motor strength, and gait. Systemic examinations were unremarkable. Neurological examination showed: conscious, coherent, and oriented (Glasgow Coma Scale E4V5M6); normal cranial nerve function, motor power MRC grade 5/5, normal bilateral grip strength, normal deep tendon reflexes, flexor plantar responses bilaterally, and normal sensory examination. There were no signs of meningeal irritation. The patient was discharged on the same day of mobilization and she came for follow up at 1, 3, and 6 months. Both at 1- and 3-month follow-up visits, the patient was clinically stable. A post-operative MRI cervical spine with whole spine screening scan was performed at 6 months showed no sign of recurrence or residual hematoma from C5 to D1.

Histopathological analysis

Specimen analysis revealed thickened dura over the chronic epidural hematoma and prominent neovascularization, confirming a chronic organized hematoma under a thick epidural layer (Figure 5).

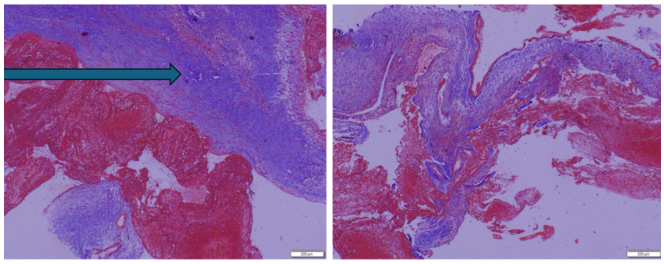

Figure 5. Masson's trichrome staining, showing thickened dura (green arrow) with neovascularization and subdural hemorrhagic collection.

DISCUSSION

Spontaneous cervicothoracic SEH is a rare cause of spinal cord compression in adolescents, typically presenting with subacute neck pain with radiation to bilateral upper limbs and progressing to neurologic deficits[1]. Atypical features such as flexion contractures and radiating upper-limb pain rarely appear in adolescents[7-9]. We report a 16-year-old girl with a 5-month history of chronic progressive neck pain, fixed flexion deformity, myeloradiculopathy, reduced right hand grip with reduced power in the right upper and lower limbs.

Our patient's presentation (neck pain with radiation, motor weakness, and a flexed neck deformity) demonstrates how SEH can manifest subtly in adolescents with vague complaints[2, 10]. Furthermore, the absence of trauma, infection, or systemic symptoms lowers clinical suspicion, leading to delayed diagnosis. Therefore, clinicians must maintain a high index of suspicion for progressive neurological deficits in pediatric patients for prompt diagnosis and intervention[9, 11].

The pathogenesis of spontaneous SEH is unclear; most cases are attributed to the rupture of the epidural venous plexus, while arterial lesions are less documented[5, 12]. The cervicothoracic region is particularly susceptible due to its unique anatomy and vascular supply[12, 13]. In our patient, the absence of underlying vascular malformation or coagulopathy truly suggests spontaneous hemorrhage. The chronicity of the condition likely resulted from the gradual accumulation of blood within the epidural space and delayed symptom progression.

MRI Spine with contrast is highly sensitive and specific in identifying extent of hematoma and exact degree of spinal cord compression[13, 14]. In our case, MRI revealed a C6-D1 epidural lesion with typical imaging features for mass effect and epidural blood products. The lesion appeared hypointense on T1-weighted images, hyperintense on T2-weighted images, with peripheral contrast enhancement, consistent with a chronic hematoma[5]. Also, peripheral contrast enhancement seen on MRI correlated with the thick, vascularized fibrous membrane identified intraoperatively and confirmed by histopathological examination.

Surgical decompression is the standard treatment option for spontaneous SEH, especially with progressive neurological deficits or cord compression[8, 10, 15]. Conservative management may be appropriate in patients with no deficits or rapid spontaneous improvement[14]. Our patient underwent C5–D1 laminectomy with complete evacuation of hematoma from C6-D1. Following excision of a sac-like layer, a second, true dural layer was visualized. This represents a chronic spontaneous SEH case with progressive deformity and intraoperative dural-like membrane overlying the intact dura, initially resembling dual dural layers. It was reported that inflammatory processes within chronic hematomas lead to granulation tissue formation, neovascularization, and development of fibrous membrane[16, 17].

DOI: 10.46900/apn.v8i1.328

Our patient achieved complete neurological recovery at 6-month follow-up without recurrence. Factors that contributed to better recovery included younger age, milder preoperative motor impairment (MRC grade 4/5), timely surgical intervention, and limited cranio-caudal extent. Early diagnosis and prompt decompression decrease the risk of neurological sequelae, with most pediatric patients undergoing surgery within 12 hours of neurological deterioration achieving better outcomes[15, 18]. In contrast, patients with complete deficits and delayed treatment have significantly poorer outcomes[12, 15]. Despite our patient's 5-month history of symptoms, complete recovery was achieved with timely surgical decompression.

CONCLUSION

Children and adolescents may exhibit more diverse and subtle first appearances than adults. Therefore, this case underscores that chronic SEH should be considered in adolescents presenting with progressive neck pain, deformity, and myeloradiculopathy, even in the absence of trauma. Flexion deformity, chronic progression, and the absence of trauma should not deter clinicians from considering SEH in the differential diagnosis for unexplained progressive neurological symptoms. Identification of a hematoma membrane resembling dual dura suggests chronic SEH features and is important for achieving complete evacuation. Despite prolonged history of symptoms, Prompt MRI evaluation and timely surgical decompression resulted in complete neurological recovery.

ACKNOWLEDGMENTS

DISCLOSURES

Ethical approval

This study was performed in line with the principles of the Declaration of Helsinki. Not required approval for this single anonymized case report, as per institutional policy. Written informed consent for publication was obtained from the patient's legal guardian

Consent to participate

The patients gave consent to use their information and images for research purposes. *Consent for publication*

Conflict of interest

The authors report no conflict of interest concerning the materials or methods used in this study or the findings specified in this paper

Funding

This research received no specific grant from any funding agency in the public, commercial or not-for-profit sectors

Artificial intelligence

The authors affirm that no artificial intelligence tools were used in the writing, editing, or content generation of this manuscript. All work was conducted manually, based on thorough research and academic expertise.

NO ARTIFICIAL INTELLIGENCE ASSISTANCE WERE EMPLOYED IN THE PREPARATION OF THIS MANUSCRIPT

CONTRIBUTIONS

-**Dr. Prakash Mahantshetti**: Supervision, Validation, Writing – review & editing
-**Dr. Nikhita Kalyanshetti**: Supervision, Validation, Writing – review & editing
-**Dr. Chandan Miriyala**: Conceptualization, Data curation, Formal Analysis, Investigation, Methodology, Resources, Writing – original draft, Writing – review & editing

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DOI: 10.46900/apn.v8i1.328