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Cervical Cord Compression Due to Osteochondroma in Hereditary Multiple Exostoses: A Case Report and Review of Literature

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Abstract

Hereditary multiple exostoses (HME) is an autosomal dominant skeletal disorder; it is characterized by multiple osteochondromas that usually arise from the metaphyses of long bones. We report the case of a 12-year-old boy with familial HME who presented with vertigo, imbalance, and gait ataxia. Examination revealed vertical nystagmus, a positive Romberg's sign, and hyperalgesia below the C2 level with preserved motor power. Magnetic resonance imaging and computed tomography showed a bony outgrowth arising from the right C2 lamina and projecting into the spinal canal, causing posterior extradural compression of the spinal cord without myelomalacia, together with non-union of the anterior and posterior arches of the atlas. He underwent C2 laminectomy with complete excision of the lesion, which arose from the inner surface of the right C2 lamina and extended cranially beneath the right hemiarch of C1. Histopathology confirmed osteochondroma. The postoperative course was uneventful, with no new neurological deficits. This case shows that an intracanal cervical osteochondroma in a child with HME may present with subtle symptoms, such as vertigo and imbalance, rather than overt weakness. Clinicians caring for children with HME should have a low threshold for cross-sectional spinal imaging when new neurological symptoms develop.

Categories: Genetics, Pediatrics, Neurosurgery
Keywords: c2 laminectomy, cervical osteochondroma, compressive myelopathy, hereditary multiple exostoses (hme), pediatric spine surgery

Introduction

Hereditary multiple exostoses (HME), also known as multiple osteochondromas or diaphyseal aclasis, is an autosomal dominant skeletal disorder characterized by multiple cartilage-capped bony outgrowths that arise most often from the juxta-epiphyseal regions of the long bones [1,2]. Its incidence is estimated at 1 in 50,000 to 1 in 100,000 [2]. The disorder results mainly from heterozygous loss-of-function variants in EXT1 or EXT2, which encode glycosyltransferases required for heparan sulfate biosynthesis [1]. Recognized complications include skeletal deformity, neurological compression, and malignant transformation to chondrosarcoma, which occurs in approximately 3% to 5% of patients with HME [3].

Spinal involvement in HME is more frequent than was previously recognized. In a magnetic resonance imaging (MRI) screening study of 44 patients with HME, 68% had exostoses arising from the spinal column, and 27% had lesions encroaching on the spinal canal, most of them clinically silent [4]. Symptomatic spinal cord compression is less common and has been reported in approximately 1% to 9% of patients [5]. The cervical spine is the most frequent spinal location [6], and a recent meta-analysis of cervical osteochondromas compared lesions associated with HME with solitary lesions [7]. Cord compression is also increasingly recognized in children; a 2022 review identified 27 pediatric cases of spinal osteochondroma with cord compression [8]. The upper cervical spine deserves particular attention. The atlas (C1) is a ring without a vertebral body that supports the skull, and the axis (C2) carries the odontoid process on which the atlas rotates; together they form the craniovertebral junction, where the spinal canal is normally capacious but where the cord lies close to the lamina of C2 posteriorly. A lesion arising from the inner surface of the C2 lamina therefore compresses the cord from behind. The mechanism by which a cervical lesion produces vertigo and imbalance is not immediately obvious. The dorsal columns and spinocerebellar pathways, which convey proprioceptive information from the limbs, lie dorsally and laterally in the cord and are therefore affected first by a dorsal extradural lesion, so that unsteadiness may precede weakness. Proprioceptive afferents from the upper cervical segments also converge with vestibular and visual inputs in the vestibular nuclei, and a mismatch between these inputs can itself produce dizziness [9]. We report the case of a 12-year-old boy with familial HME who presented with vertigo and imbalance caused by an osteochondroma of the C2 lamina compressing the cervical spinal cord, in association with non-union of the anterior and posterior arches of the atlas.

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Case Presentation

A 12-year-old boy, born to non-consanguineous parents and with normal developmental milestones, presented with a seven-day history of giddiness, a spinning sensation, imbalance, and progressive difficulty in standing and walking. He had a known history of multiple bony swellings involving the long bones, consistent with HME. He had previously undergone excision of a tibial swelling, with histopathology confirming osteochondroma. His father and younger brother also had similar swellings, supporting a diagnosis of familial HME. Genetic testing for EXT1 or EXT2 variants was not performed; the diagnosis was based on the clinical and radiological features together with the autosomal dominant family history.

On physical examination, multiple hard, non-compressible swellings were noted over the tibia, femur, and fibula. Neurological examination revealed a positive Romberg's sign and vertical nystagmus. Generalized hyperalgesia was noted below the C2 level, though motor power was preserved.

Magnetic resonance imaging (MRI) of the cervical spine demonstrated a bony outgrowth measuring approximately 9 × 12 mm in cross-section and 18 mm in length arising from the right lamina of the C2 vertebra, projecting cranially and anteromedially into the spinal canal with canal narrowing (bony canal anteroposterior diameter approximately 5.5 mm; dural sac 4.7 × 13 mm at the C1-C2 level). The canal anteroposterior diameter measured 10.5 mm at C2-C3 and C3-C4, 10.2 mm at C4-C5, 9.5 mm at C5-C6, and 10.0 mm at C6-C7 and C7-T1. The bony outgrowth showed medullary continuity with the C2 lamina, and no obvious cartilage cap was noted over it. There was extradural compression of the spinal cord, which was displaced anteriorly and to the left, with no abnormal contrast enhancement. There were no signs of myelomalacia (Figures *1*-*3*). Whole-spine screening showed no other lesions.



FIGURE 1: Sagittal T2-weighted MRI of the cervical spine showing the bony outgrowth arising from the C2 lamina (arrow), projecting into the spinal canal at the level of the C2 vertebra and compressing the spinal cord posteriorly, without signs of myelomalacia; the dens and C3 vertebral body are seen anteriorly.

MRI: magnetic resonance imaging

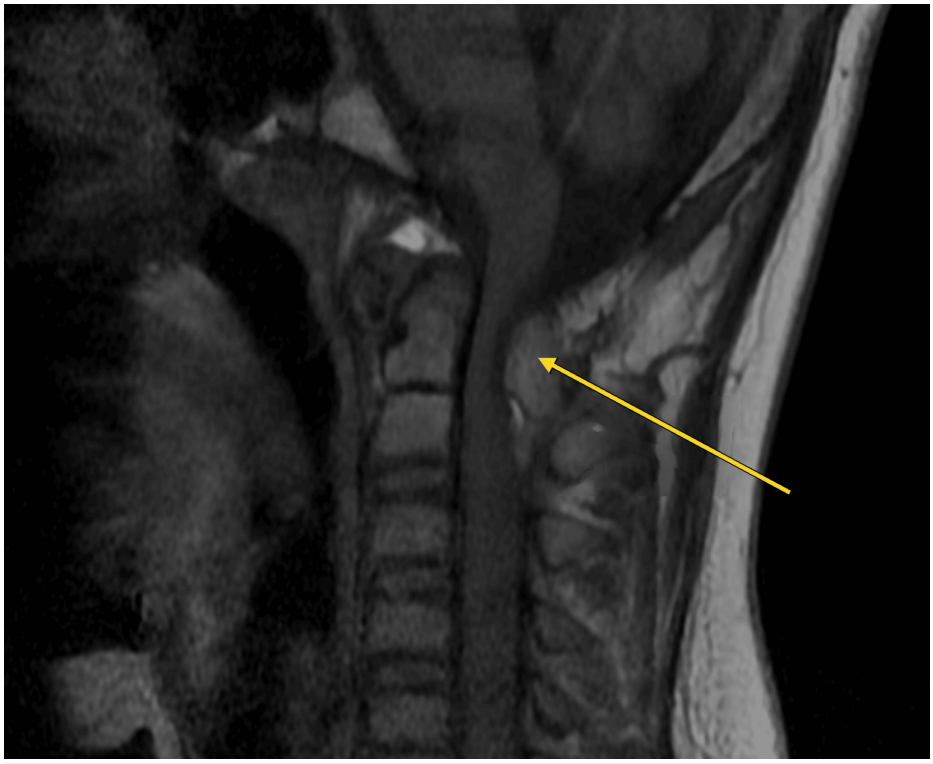


FIGURE 2: Sagittal T1-weighted fast spin-echo MRI showing the lesion (arrow) within the spinal canal posterior to the spinal cord at the level of the C2 vertebra; the dens and C3 vertebral body are seen anteriorly.

MRI: magnetic resonance imaging

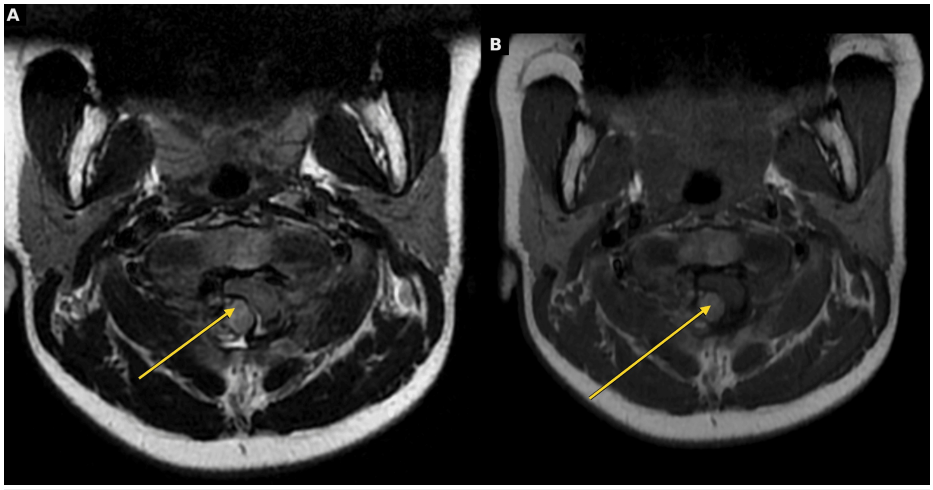


FIGURE 3: Axial T2-weighted (A) and T1-weighted (B) MRI at the level of the lesion, showing the bony outgrowth (arrows) within the posterior spinal canal on the right, with the spinal cord displaced anteriorly and to the left.

MRI: magnetic resonance imaging

Computed tomography (CT) revealed canal narrowing (minimum midsagittal bony canal diameter approximately 4 mm) and non-union of the anterior and posterior arches of the atlas (Figure *4*). The atlantoaxial joint was normal, and the remaining vertebral bodies and posterior elements showed no abnormality.

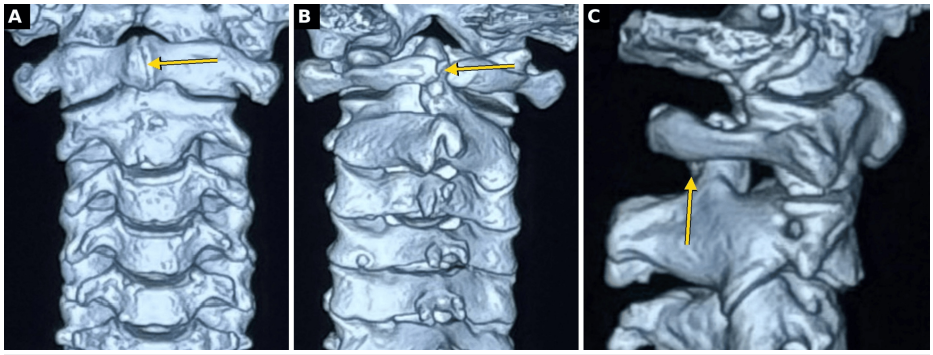


FIGURE 4: Three-dimensional volume-rendered CT reconstruction of the cervical spine. Anterior (A) and posterior (B) views showing non-union of the anterior and posterior arches of the atlas (arrows), respectively. Lateral view (C; posterior to the left) showing the lesion (arrow) at the C2 lamina beneath the posterior arch of C1.

CT: computed tomography

The differential diagnosis included a solitary osteochondroma, osteoid osteoma, osteoblastoma, chondrosarcoma arising from malignant transformation of HME, and an aneurysmal bone cyst.

The patient underwent a C2 laminectomy with excision of the extradural lesion under general anesthesia. Intraoperatively, the lesion was found to arise from the inner surface of the right C2 lamina and extended cranially beneath the right hemiarch of C1. The posterior tubercle of the C1 arch was underdeveloped. The ligamentum flavum and atlanto-axial membrane were thickened and adherent to the dura. Complete excision of the lesion was achieved (Figure *5*).

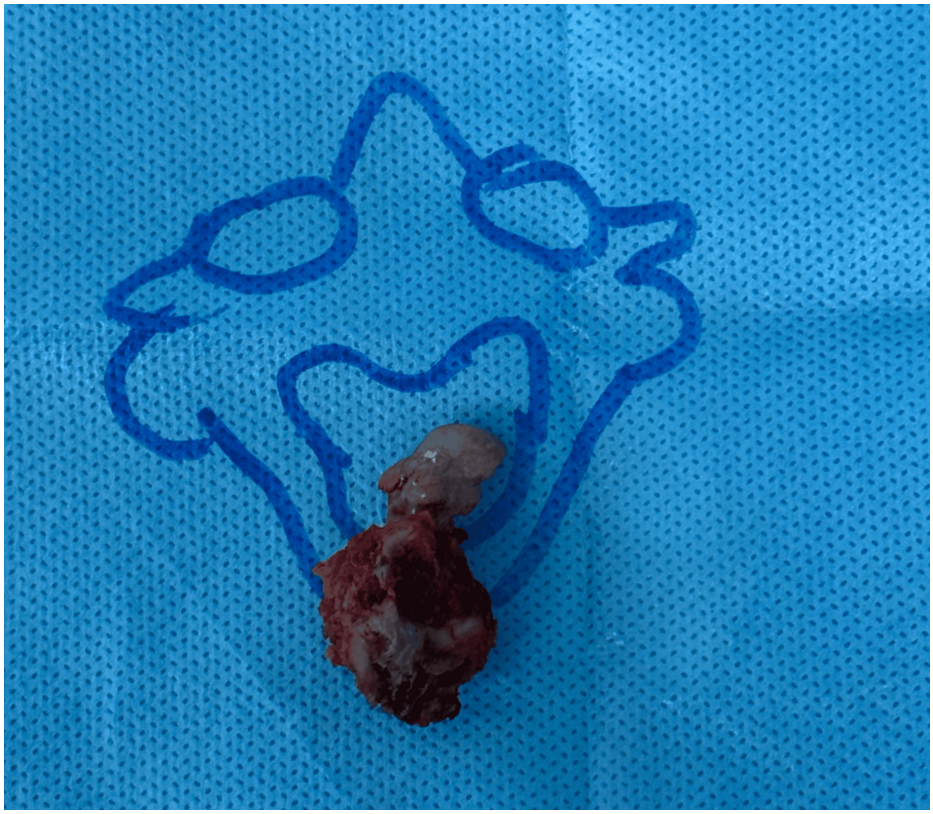


FIGURE 5: Gross photograph of the excised specimen on the surgical drape, alongside a hand-drawn outline of the vertebra marking the site of origin of the lesion. The specimen was removed en bloc from the inner surface of the right C2 lamina and consists of a bony base with an overlying pale, glistening cartilage-covered surface.

Histopathology confirmed the diagnosis of osteochondroma, showing a cartilaginous cap with benign chondrocytes undergoing endochondral ossification, merging with trabecular bone and marrow spaces (Figures *6*-*7*).

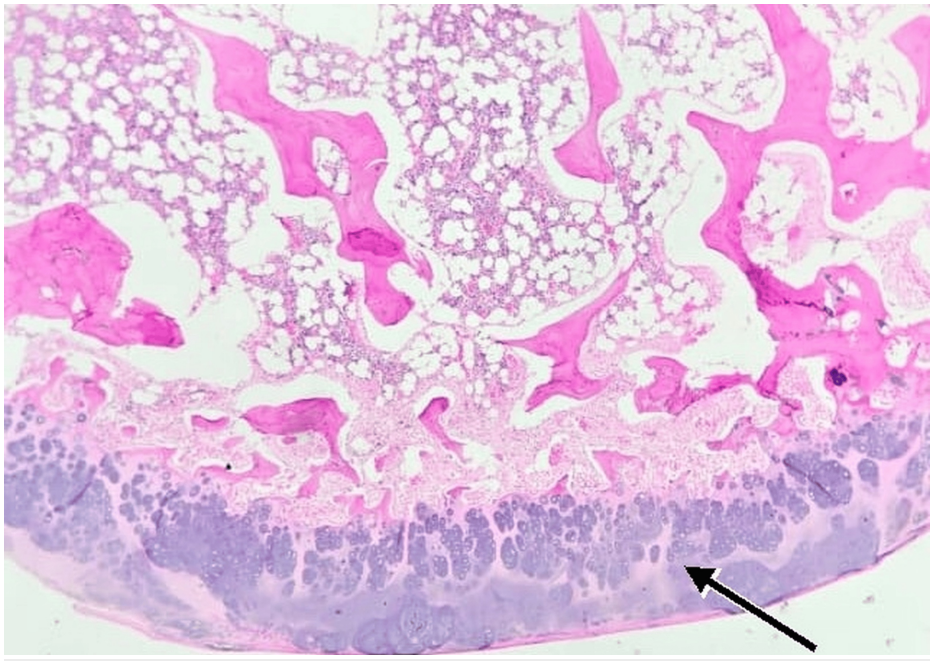


FIGURE 6: Histopathology of the excised lesion (hematoxylin and eosin stain) showing the cartilaginous cap (arrow) overlying trabecular bone with intervening marrow spaces.

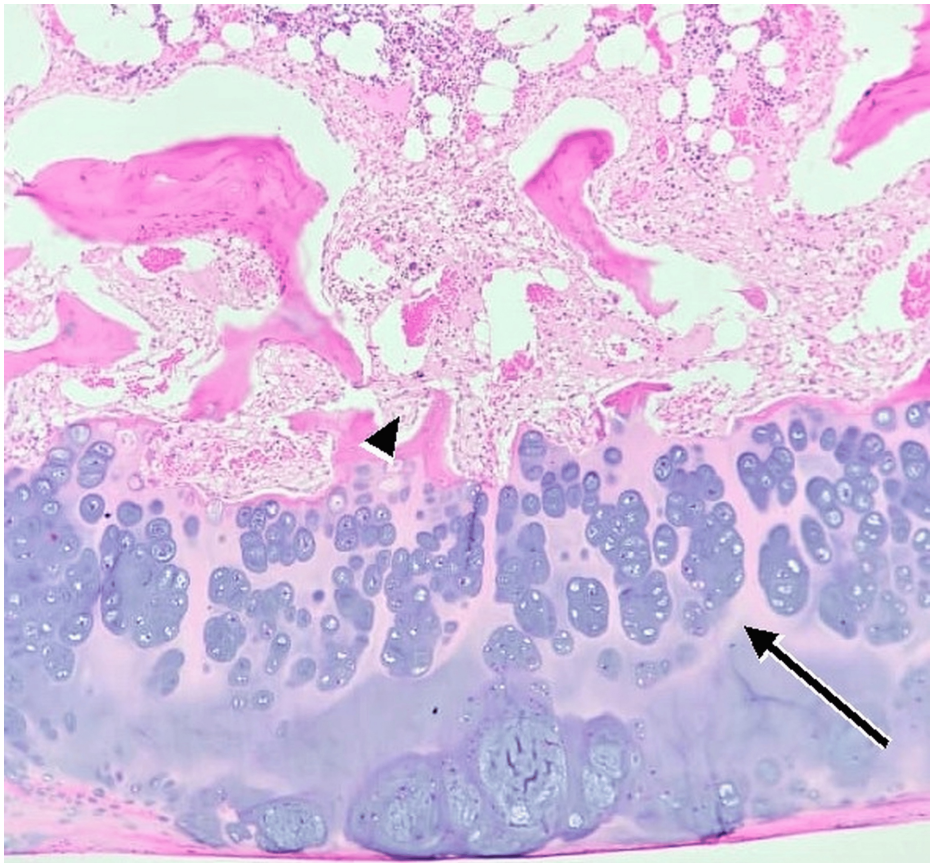


FIGURE 7: Higher-magnification view (hematoxylin and eosin stain) showing benign chondrocytes within the cartilaginous cap (arrow) undergoing endochondral ossification at the junction with trabecular bone (arrowhead).

The postoperative course was uneventful, and the patient recovered without any new neurological deficits. He was followed regularly over six months, during which his vertigo and imbalance resolved completely. On examination, Romberg's sign was negative, and the nystagmus and hyperalgesia had resolved, with preserved motor function. Long-term surveillance is planned to monitor for recurrence or new spinal lesions.

Discussion

Spinal osteochondromas in HME are often asymptomatic and easily missed. In the screening series reported by Roach et al., patients with intracanal lesions were typically neurologically normal, and plain radiographs identified the lesions in only a minority of patients [4]. In a pediatric series, Ashraf et al. found intracanal osteochondromas causing cervical spinal stenosis in two of nine children with HME who underwent axial imaging for pain, weakness, or dizziness [10].

Reported pediatric cases of cervical cord compression in HME have usually presented with overt myelopathic features, including acute cord compression from a C7-T1 laminar exostosis in a four-year-old boy [11] and Brown-Séquard syndrome from a C7 laminar osteochondroma in a seven-year-old boy [12]. The axis may also be involved in children, as in a boy with an exostosis compressing the axis [13] and a child with a giant osteochondroma of the axis [14]. Our patient was unusual in that his presenting complaints were giddiness, a spinning sensation, and imbalance, and his examination showed vertical nystagmus and a positive Romberg's sign with preserved motor power. His symptoms also developed over only one week, in contrast to the indolent course usually attributed to these slowly growing lesions, suggesting that a long-standing lesion may become symptomatic abruptly once the cord is critically compressed. Such subtle findings may not immediately suggest spinal cord compression, and they highlight the need to consider cervical pathology whenever a child with HME develops a new neurological symptom.

Several mechanisms may account for vestibular and balance symptoms in this setting. A dorsal extradural lesion compresses the posterior and posterolateral cord first, where the dorsal columns and spinocerebellar tracts carry proprioceptive information, while the corticospinal tracts lie more ventrolaterally; this

distribution explains the combination of a positive Romberg's sign and gait ataxia with preserved motor power in our patient. Proprioceptive afferents from the upper cervical segments project to the vestibular nuclei and cerebellum, where they are integrated with vestibular and visual input, and disturbance of this input can produce dizziness through sensory mismatch [9]. Vertical nystagmus, in particular downbeat nystagmus, is a recognized localizing sign of lesions at the cervicomedullary junction [15], and its presence in our patient supported localization to the craniovertebral junction rather than to a peripheral vestibular cause.

Selected reported pediatric cases of cervical or cervicothoracic cord compression due to osteochondroma in HME are summarized in Table *1*, together with the present case. In contrast with these reports, which describe motor or long-tract deficits, our patient presented with vestibular and balance symptoms, and canal and dural sac dimensions were documented on both CT and MRI.

Reference	Age/sex	Level	Presentation	Treatment	Outcome
[11]	4/M	C7-T1	Cord compression	Not stated	Not stated
[12]	7/M	C7	Brown-Sequard syndrome	Excision	Improved
[13]	Child/M	C2 (axis)	Cord compression	Excision	Not stated
[14]	Child	C2 (axis)	Cord compression	Excision	Not stated
Present case	12/M	C2 lamina	Vertigo, imbalance, gait ataxia	C2 laminectomy and excision	Improved

TABLE 1: Selected reported pediatric cases of cervical cord compression due to osteochondroma in hereditary multiple exostoses.

Details are as reported in the cited publications; entries marked "Not stated" were not available.

M: male

Cross-sectional imaging is essential in these patients. Plain radiographs are unreliable for detecting spinal lesions in HME [4], whereas CT and MRI together allow accurate preoperative localization of an intraspinal osteochondroma and its relationship to the spinal cord [16]. The diagnosis of osteochondroma depends on continuity of the lesion with the cortex and medullary cavity of the parent bone [3]. After skeletal maturity, continued growth or a cartilage cap thicker than 1.5 cm suggests malignant transformation [3]; this threshold does not apply directly to a 12-year-old, and histopathology in our patient showed a benign cartilaginous cap. Normative values for the bony cervical canal in children show a sagittal diameter that increases with age and remains relatively constant below C3 [17]; in our patient, the canal was reduced to approximately 4 mm on CT at the level of the lesion, compared with 9.5 to 10.5 mm at every level from C2-C3 to C7-T1 on MRI, confirming severe focal stenosis. The two modalities were complementary: CT defined the osseous origin of the lesion from the right C2 lamina, its continuity with the medullary cavity, and the degree of bony canal narrowing, whereas MRI demonstrated the dural sac, the direction of cord displacement, the absence of cord signal change, and the absence of a cartilage cap, and the MRI findings were correlated with CT at the time of reporting.

Our patient also had non-union of the anterior and posterior arches of the atlas, with an underdeveloped posterior tubercle of C1. Congenital defects of the posterior arch of the atlas are usually discovered incidentally, although symptoms may follow trauma to the head or neck [18]. To our knowledge, no association between such atlas defects and HME has been established. We therefore regard this finding as a likely incidental congenital variant rather than a cause of our patient's symptoms: his vertigo, imbalance, and sensory disturbance were attributable to cord compression by the C2 lesion, the atlantoaxial joint was normal on CT, and there was no evidence of instability. Its relevance was anatomical rather than symptomatic: the deficient posterior arch was noted in preoperative planning at the craniovertebral junction, although in this patient it did not alter the surgical approach or the extent of decompression.

Posterior decompression with excision of the lesion is the usual treatment for a symptomatic intracanal osteochondroma, and neurological recovery after excision has been reported in both children and adults [8,12,16]. Resection of posterior elements in the cervical spine may raise concern about instability, and anterior stabilization and fusion have been described to prevent instability after excision of a cervical laminar exostosis in HME [19]. In our patient, the excision was limited to a C2 laminectomy, and the postoperative course was uneventful.

Because intracanal lesions are often clinically silent, screening spinal MRI has been advocated for patients

with HME [4,10], and a prospective spine-at-risk program recommended MRI at preoperative evaluation because unrecognized intraspinal exostoses can cause neurological injury during sedated procedures [20]. However, a national survey of HME experts found wide variation in screening practice and no consensus guideline [6]. In the absence of a consensus guideline, we suggest that any new neurological symptom in a child with HME, including vertigo or imbalance, should prompt cross-sectional imaging of the cervical spine, and that imaging be considered before procedures requiring sedation or positioning of the neck. Long-term clinical and radiological surveillance is planned for our patient to detect recurrence or new spinal lesions. This report has several limitations. It describes a single patient with six months of follow-up, so the clinical observations and outcome cannot be generalized to all children with HME. Postoperative imaging was not obtained during this period because the family declined further imaging in a child who had made a complete clinical recovery, and genetic confirmation of an EXT1 or EXT2 variant was not available. Longer follow-up with imaging is required to assess recurrence and the durability of neurological recovery.

Conclusions

Cervical spinal cord compression is a rare but critical complication of HME in children. Early symptoms may be subtle, such as vertigo and gait imbalance, and require a high index of suspicion in patients with known HME. CT and MRI are complementary and essential for defining the bony anatomy and the degree of cord compression. In reported cases, timely surgical decompression with complete excision of the lesion has been associated with favorable neurological outcomes, particularly in the absence of myelomalacia; the present report describes a single patient followed for six months and therefore does not permit conclusions about long-term outcome or recurrence, and continued clinical and radiological surveillance is planned. In children with HME, vestibular and balance symptoms may dominate the presentation of an intracanal cervical osteochondroma, and preserved motor power should not be taken as reassurance. Any new neurological symptom in such a child, however nonspecific, warrants cross-sectional imaging of the cervical spine, including the craniovertebral junction, so that decompression can be performed before irreversible cord injury occurs.

Additional Information

Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

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Critical review of the manuscript for important intellectual content: Suprajaa Desingu, Venkata Shashank Davuluri, Visvanathan Krishnaswamy, Vasugi Gramani Arumugam, Balasubramanian Kannan

Supervision: Suprajaa Desingu, Venkata Shashank Davuluri, Visvanathan Krishnaswamy, Vasugi Gramani Arumugam

Disclosures

Human subjects: Informed consent for treatment and open access publication was obtained or waived by all participants in this study. issued approval SRIHER2025/PGMC779/MED. **Conflicts of interest:** In compliance with the ICMJE uniform disclosure form, all authors declare the following: **Payment/services info:** All authors have declared that no financial support was received from any organization for the submitted work. **Financial relationships:** All authors have declared that they have no financial relationships at present or within the previous three years with any organizations that might have an interest in the submitted work. **Other relationships:** All authors have declared that there are no other relationships or activities that could appear to have influenced the submitted work.

References

1. Bukowska-Olech E, Trzebiatowska W, Czech W, et al.: Hereditary multiple exostoses-a review of the molecular background, diagnostics, and potential therapeutic strategies. Front Genet. 2021, 12:759129. 10.3389/fgene.2021.759129
2. Rueda-de-Eusebio A, Gomez-Pena S, Moreno-Casado MJ, Marquina G, Arrazola J, Crespo-Rodríguez AM: Hereditary multiple exostoses: an educational review. Insights Imaging. 2025, 16:46. 10.1186/s13244-025-01899-6
3. Murphey MD, Choi JJ, Kransdorf MJ, Flemming DJ, Gannon FH: Imaging of osteochondroma: variants and

complications with radiologic-pathologic correlation. Radiographics. 2000, 20:1407-1434. 10.1148/radiographics.20.5.g00se171407

4. Roach JW, Klatt JWB, Faulkner ND: Involvement of the spine in patients with multiple hereditary exostoses . J Bone Joint Surg Am. 2009, 91:1942-1948. 10.2106/JBJS.H.00762
5. Burki V, So A, Aubry-Rozier B: Cervical myelopathy in hereditary multiple exostoses . Joint Bone Spine. 2011, 78:412-414. 10.1016/j.jbspin.2011.02.021
6. Montgomery BK, Cahan EM, Frick S: Spinal screening MRI trends in patients with multiple hereditary exostoses: national survey. Cureus. 2019, 11:e6452. 10.7759/cureus.6452
7. Fowler J, Takayanagi A, Fiani B, et al.: Diagnosis, management, and treatment options: a cervical spine osteochondroma meta-analysis. World Neurosurg. 2021, 149:215-225.e6. 10.1016/j.wneu.2021.01.148
8. Mamdouhi T, Unadkat P, Edelman MC, Johnson AA, Fein Levy C, Mittler MA: Solitary pediatric osteochondroma of the spine with cord compression. Cureus. 2022, 14:e23342. 10.7759/cureus.23342
9. Li Y, Yang L, Dai C, Peng B: Proprioceptive cervicogenic dizziness: a narrative review of pathogenesis, diagnosis, and treatment. J Clin Med. 2022, 11:6293. 10.3390/jcm11216293
10. Ashraf A, Larson AN, Ferski G, Mielke CH, Wetjen NM, Guidera KJ: Spinal stenosis frequent in children with multiple hereditary exostoses. J Child Orthop. 2013, 7:183-194. 10.1007/s11832-013-0484-9
11. Ezra N, Tetteh B, Diament M, Jonas AJ, Dickson P: Hereditary multiple exostoses with spine involvement in a 4-year-old boy. Am J Med Genet A. 2010, 152A:1264-1267. 10.1002/ajmg.a.33345
12. Han IH, Kuh SU: Cervical osteochondroma presenting as brown-sequard syndrome in a child with hereditary multiple exostosis. J Korean Neurosurg Soc. 2009, 45:309-311. 10.3340/jkns.2009.45.5.309
13. Korinth MC, Ramaekers VT, Rohde V: Cervical cord exostosis compressing the axis in a boy with hereditary multiple exostoses. Case illustration. J Neurosurg. 2004, 100:223. 10.3171/ped.2004.100.2.0223
14. Tahasildar N, Sudesh P, Goni V, Tripathy SK: Giant osteochondroma of axis in a child with multiple hereditary exostoses: case report and review of literature. J Pediatr Orthop B. 2012, 21:280-285. 10.1097/BPB.0b013e32834c3186
15. Wagner JN, Glaser M, Brandt T, Strupp M: Downbeat nystagmus: aetiology and comorbidity in 117 patients . J Neurol Neurosurg Psychiatry. 2008, 79:672-677. 10.1136/jnnp.2007.126284
16. Aithala JP: C2 intraspinal osteochondroma causing spinal cord compression in a patient with multiple hereditary exostoses. Indian Spine J. 2022, 5:137-141. 10.4103/isj.isj_55_20
17. Markuske H: Sagittal diameter measurements of the bony cervical spinal canal in children . Pediatr Radiol. 1977, 6:129-131. 10.1007/BF00972103
18. Currarino G, Rollins N, Diehl JT: Congenital defects of the posterior arch of the atlas: a report of seven cases including an affected mother and son. AJNR Am J Neuroradiol. 1994, 15:249-254.
19. Ergün R, Okten AI, Beşkonakli E, Akdemir G, Taşkin Y: Cervical laminar exostosis in multiple hereditary osteochondromatosis: anterior stabilization and fusion technique for preventing instability. Eur Spine J. 1997, 6:267-269. 10.1007/BF01322449
20. Vu CL, Lindberg AW, Bompadre V, White KK, Bauer JM: Prospective spine at risk program for prevalence of intracanal spine lesions in pediatric hereditary multiple osteochondromas. Spine Deform. 2020, 8:1069-1074. 10.1007/s43390-020-00130-4