TIBBIYOT
TA'LIMI & INNOVATSIYALARI

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- 14.00.02 - Morfologiya;
- 14.00.03 - Endokrinologiya;
- 14.00.04 - Otorinolaringologiya;
- 14.00.05 - Ichki kasalliklar;
- 14.00.06 - Kardiologiya;
- 14.00.07 - Gigiena;
- 14.00.08 - Oftal'mologiya;
- 14.00.09 - Pediatriya;
- 14.00.10 - Yuqumli kasalliklar;
- 14.00.11 - Dermatologiya va venerologiya;
- 14.00.12 - Tibbiy reabilitologiya;
- 14.00.13 - Nevrologiya;
- 14.00.14 - Onkologiya;
- 14.00.15 - Patologik anatomiya;
- 14.00.16 - Normal va patologik fiziologiya;
- 14.00.17 - Farmakologiya va klinik farmakologiya;
- 14.00.18 - Psixiatriya va narkologiya;
- 14.00.19 - Klinik radiologiya;
- 14.00.20 - Tibbiy genetika;
- 14.00.21 - Stomatologiya;
- 14.00.22 - Travmatologiya va ortopediya;
- 14.00.23 - Hamshiralik ishini tashkil etish;
- 14.00.24 - Sud tibbiyoti;
- 14.00.27 - Xirurgiya;
- 14.00.28 - Neyroxirurgiya;
- 14.00.41 - Xalq tabobati;
- 14.00.35 - Bolalar xirurgiyasi;
- 14.00.34 - Yurak-qon tomir xirurgiyasi

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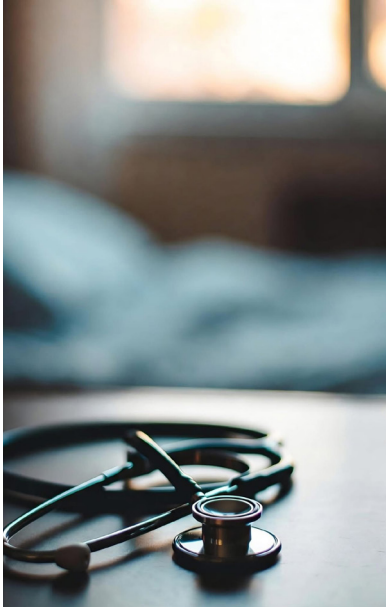
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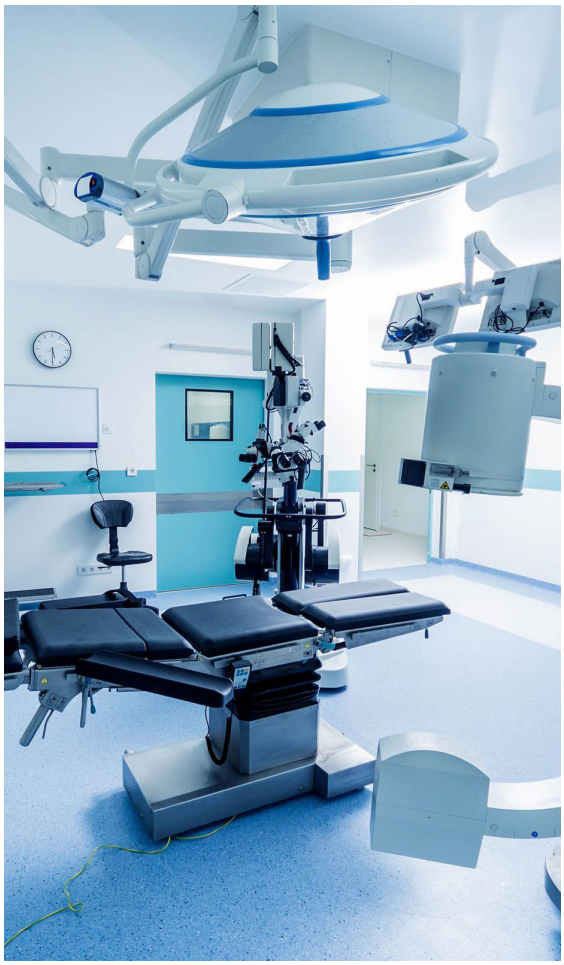
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MUNDARIJA

ТОШКЕНТ ХАЛҚАРО МОЛИЯ МАРКАЗИ ВА СОҒЛИҚНИ САҚЛАШ ТИЗИМИНИ РИВОЖЛАНТИРИШ: ЯНГИ ҚОНУННИНГ АҲАМИЯТИ 12
Шуҳрат Боймуротов

IMPROVEMENT OF THE SURGICAL APPROACH TO THE TREATMENT OF CRANIOVERTEBRAL JUNCTION ANOMALIES ASSOCIATED WITH SPINAL MALFORMATIONS (CLINICAL EXPERIENCE) 17
Arzikulov Sh.T., Ismailova R.O., Jabborov N.I.

ARTERIAL GIPERTENZIYA VA 2 TIP QANDLI DIABET: PATOGENETIK BOG‘LIQLIKLAR VA GENETIK PREDISPOZITSIYA 25
Ellamonov Suxrobjon No‘mon o‘g‘li

БОЛАЛАРДА ПСОРИАЗНИНГ КЛИНИК-БИОКИМЁВИЙ, ИММУНОПАТОГЕНЕТИК ХУСУСИЯТЛАРИНИ ҲИСОБГА ОЛГАН ҲОЛДА КОМПЛЕКС ДИАГНОСТИКА ВА ТЕРАПИЯСИНИ ТАКОМИЛЛАШТИРИШ 32
Хаитов Кахрамон Нажмитдинович, Абидов Хасанходжа Алишерович, Махкамова Мухлиса Фархадовна

GLUTEN BILAN BOG‘LIQ SALOMATLIK XAVFLARI VA OZIQ-OVQAT NAZORATINING ZAMONAVIY YONDASHUVLARI: TANQIDIY ADABIYOTLAR SHARHI 48
Abdullaeva Dilafruz G‘ayratovna, Razikov Farrux Ibragim o‘g‘li, Abdullaeva Dilafruz G‘ayratovna

SLEEP AND CIRCADIAN RHYTHMS IN RELATION TO ANDROGEN STATUS IN MIDDLE-AGED MEN: CURRENT EVIDENCE AND CLINICAL IMPLICATIONS 58
Feruza Lyutpillayevna Azizova, Abdukhalim Kholiddin ugli Abdurakhimov

BOLALARDAGI NEYROGEN DEFORMATSIYALARNING UMUMIY KLINIK TAVSIFI VA ULARNI KELTIRIB CHIQARUVCHI OMILLAR TAHLILI 66
Xalimov Ravshan Jurabayevich

KESARCHA KESISHDAN KEYINGI UZOQ MUDDATLI ASORATLAR VA REABILITATSIYA STRATEGIYALARI (ADABIYOTLAR SHARHI) 70
Atayeva Farzona Nuriddinovna

KATTALARDA MODIFIKATSIYALANGAN INTRAMEDULLYAR VINTLAR YORDAMIDA KAFT SUYAKLARI SINIQLARINI KAM INVAZIV OSTEOSINTEZ QILISH 75
Xudoyorov Farhod Rustamovich

УСОВЕРШЕНСТВОВАННАЯ МЕТОДИКА ДИНАМИЧЕСКОЙ АНГИОНЕФРОСЦИНТИГРАФИИ ДЛЯ РАСШИРЕННОЙ КОЛИЧЕСТВЕННОЙ ОЦЕНКИ ФУНКЦИИ ПОЧЕК 85
Хикматов Акром Азимджанович

IMPROVEMENT OF THE SURGICAL APPROACH TO THE TREATMENT OF CRANIOVERTEBRAL JUNCTION ANOMALIES ASSOCIATED WITH SPINAL MALFORMATIONS (CLINICAL EXPERIENCE)

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Abstract: Craniovertebral junction (CVJ) anomalies are complex and rare disorders that require precise diagnostic evaluation and individualized treatment strategies. We report a rare case of Arnold–Chiari II malformation with basilar invagination, secondary hydrocephalus, and closed spinal dysraphism in a pediatric patient. A 12-year-old child presented with severe headaches, motor deficits, and signs of increased intracranial pressure. Imaging revealed Chiari II malformation, basilar invagination, and compensated tethered cord syndrome. Neurophysiological studies indicated compensated spinal dysfunction. The patient underwent posterior C1–C2 fixation with odontoid distraction, resulting in clinical and radiological improvement. This case highlights the importance of tailored neurosurgical approaches in the management of complex CVJ anomalies. Ventral decompression achieved through posterior distraction may obviate the need for additional lumbar intervention, emphasizing the importance of accurate neuroimaging and functional assessment in surgical planning.

Key words: Chiari II malformation; basilar invagination; craniovertebral junction; tethered cord syndrome; pediatric neurosurgery; posterior fixation.

Annotatsiya: Kraniovertebral birikma (KVB) anomaliyalari murakkab va kam uchraydigan patologik holatlar bo‘lib, aniq diagnostika va individual davolash taktikasini talab qiladi. Ushbu maqolada bolalar yoshidagi bemorda Arnold–Chiari II malformatsiyasining bazilyar invaginatsiya, ikkilamchi gidrosefaliya va yopiq spinal dizrafiya bilan birgalikda uchragan noyob klinik holati taqdim etilgan. 12 yoshli bola kuchli bosh og‘rig‘i, harakat buzilishlari va intrakranial bosim oshishi belgilari bilan murojaat qilgan. Neyrovizualizatsiya ma’lumotlarida Chiari II malformatsiyasi, bazilyar invaginatsiya va kompensatsiyalangan fiksatsiyalangan orqa miya sindromi aniqlangan. Neyrofiziologik tekshiruvlarda orqa miya faoliyatining kompensatsiyalangan buzilishi qayd etilgan. Bemorga C1–C2 darajasida orqa fiksatsiya va tishsimon o‘simtani distraksiyalash amaliyoti bajarilgan, natijada klinik va radiologik jihatdan ijobiy dinamika kuzatilgan.

Ushbu klinik holat kraniovertebral birikmaning murakkab anomaliyalarida individual yondashuvga asoslangan neyroxirurgik taktikaning muhimligini ko‘rsatadi. Orqa distraksiya orqali amalga oshirilgan ventral dekompressiya qo‘shimcha bel sohasidagi jarrohlik aralashuviga ehtiyojni bartaraf etishi mumkin. Bu esa jarrohlik davolashni rejalashtirishda aniq neyrovizualizatsiya va funksional baholashning muhimligini ta’kidlaydi.

Kalit so‘zlar: Chiari II malformatsiyasi; bazilyar invaginatsiya; kraniovertebral birikma; fiksatsiyalangan orqa miya sindromi; bolalar neyroxirurgiyasi; orqa fiksatsiya.

Аннотация: Аномалии краниовертебрального перехода (КВП) представляют собой сложные и редкие патологические состояния, требующие точной диагностики и индивидуализированной тактики лечения. Представлен редкий клинический случай мальформации Арнольда–Киари II типа, сочетавшейся с базилярной инвагинацией, вторичной гидроцефалией и закрытой спинальной дизрафией у пациента детского возраста.

Описание клинического случая: Ребёнок в возрасте 12 лет предъявлял жалобы на выраженные головные боли; при обследовании выявлены двигательные нарушения и признаки повышения внутричерепного давления. По данным нейровизуализации диагностированы мальформация Киари II типа, базилярная инвагинация и компенсированный синдром фиксированного спинного мозга. Нейрофизиологические исследования выявили компенсированную дисфункцию спинного мозга. Пациенту выполнена задняя фиксация на уровне C1–C2 с дистракцией зубовидного отростка, в результате чего отмечена клиническая и рентгенологическая положительная динамика.

Представленный клинический случай подчёркивает важность индивидуализированного нейрохирургического подхода при сложных аномалиях краниовертебрального перехода. Вентральная декомпрессия, достигнутая посредством задней дистракции, может позволить избежать дополнительного вмешательства на поясничном уровне, что подчёркивает важность точной нейровизуализации и функциональной оценки при планировании хирургического лечения.

Ключевые слова: мальформация Киари II типа; базилярная инвагинация; краниовертебральный переход; синдром фиксированного спинного мозга; детская нейрохирургия; задняя фиксация.

INTRODUCTION

The craniovertebral junction (CVJ) represents one of the most complex anatomical regions of the human body, where the skull base transitions into the cervical spine and houses critical neurovascular structures. Congenital and acquired CVJ anomalies are characterized by abnormal relationships between the occipital bone, atlas (C1), and axis (C2), often leading to brainstem or upper cervical spinal cord compression. Over the past decades, advances in computed tomography (CT), magnetic resonance imaging (MRI), and neuronavigation have enabled more accurate assessment of these deformities and their biomechanical implications. Nevertheless, the clinical presentation of CVJ anomalies remains variable, ranging from subtle brainstem symptoms to progressive myelopathy and respiratory dysfunction. Despite the growing understanding of CVJ pathophysiology, major controversies persist regarding the optimal surgical strategy. Determining the indications for decompression, the necessity of anterior versus posterior approaches, and the threshold for stabilization remains a matter of debate.

The philosophy of “fixation as decompression,” pioneered by Goel A. [1,2,11], has transformed the management of atlantoaxial instability, emphasizing reduction and fixation over extensive bone removal. However, discrepancies between neurosurgical schools continue to shape practice patterns. Western authors such as Brockmeyer et al. [3] highlight early reconstruction and pediatric stabilization, while others [4] advocate for neurophysiological monitoring and combined ventral and dorsal approaches in rigid or irreducible deformities. The lack of uniform treatment algorithms underscores the need for individualized and evidence-based surgical decision-making. In this context, the present study focuses on [insert your focus, e.g. “the evaluation of complex CVJ anomalies combining basilar invagination, Chiari malformation, and associated instability”]. We aim to analyze diagnostic criteria, define rational indications for surgical intervention, and assess the outcomes of posterior fixation as a key element of decompression. By integrating clinical, radiological, and intraoperative data, this work seeks to clarify the principles of surgical planning in rare and complex CVJ deformities and to contribute to the development of a more standardized treatment approach. This clinical case is presented due to its exceptional rarity, diagnostic complexity, and the necessity for a differentiated surgical approach. The case provides a platform for discussion of unresolved issues and potential directions for improving the management of rare craniovertebral junction (CVJ) anomalies.

RESEARCH METHODOLOGY

This study was conducted as a clinical observational study aimed at evaluating and improving surgical approaches for the treatment of craniovertebral junction anomalies associated with spinal malformations. The study included patients diagnosed with structural abnormalities of the craniovertebral junction and associated spinal deformities based on clinical, neurological, and radiological findings. Preoperative assessment included neurological examination and imaging studies, particularly computed tomography and magnetic resonance imaging, to determine the anatomical characteristics of the malformation, spinal cord compression, and instability. The choice of surgical approach was individualized according to the anatomical configuration of the craniovertebral junction, the type and severity of spinal malformation, neurological status, and the presence of instability or compression. Surgical outcomes were assessed using postoperative neurological status, correction of the pathological anatomy, stabilization of the craniovertebral junction, and the incidence of perioperative complications. Comparative evaluation of preoperative and postoperative clinical and radiological findings was used to determine the effectiveness and safety of the improved surgical approach.

ANALYSIS AND RESULTS

A 12-year-old child was admitted to the Republican Specialized Neurosurgery Center (Tashkent) with complaints of severe pressure-like headaches exacerbated in the upright position, recurrent vomiting without preceding nausea, progressive lower-limb weakness and numbness worsening toward the evening, impaired gait, reduced exercise tolerance, unsteadiness while walking, and episodes of dizziness. At the age of 2 years, the patient underwent herniotomy at the L2–L5 level for a diagnosed meningocele. Postoperatively, mild, non-progressive neurological symptoms were observed for several years. Over the past six months, however, the child's condition progressively deteriorated.

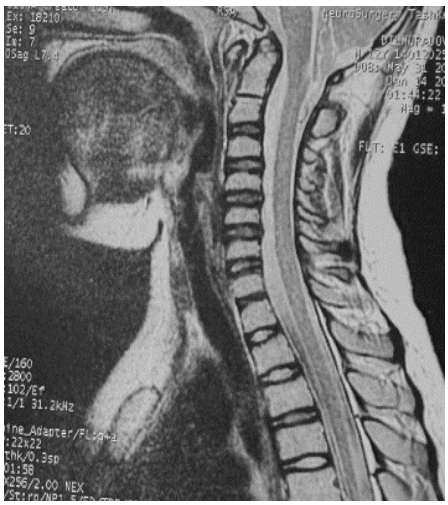


Figure 1. MRI of the craniovertebral junction showing 16.1 mm of cerebellar tonsillar descent and displacement of the fourth ventricle.

Neuroimaging Findings. MRI of the brain and craniovertebral junction revealed cerebellar tonsillar descent measuring 16.1 mm below the foramen magnum, with caudal displacement of the medulla oblongata and fourth ventricle (Figure 1). Ventriculomegaly of the lateral and third ventricles indicated secondary obstructive (tentorial) hydrocephalus. The posterior fossa was markedly small, with ventral compression of the brainstem, consistent with Arnold–Chiari malformation type II. Lumbosacral MRI demonstrated: Spina bifida from L2–S3 (Figure 2); Meningomyeloradiculocele at L4–L5; Bony septum (diastematomyelia) at L3–L4; Post-herniotomy changes at L2–L5; Barrel-shaped deformity of the L3 vertebral body.

Neurophysiological Evaluation. Electroneuromyography (ENMG) revealed bilateral radiculopathy at L3–S1, more pronounced on the right, with slowed motor conduction velocity but no acute denervation—findings consistent with compensated tethered cord syndrome (TCS).

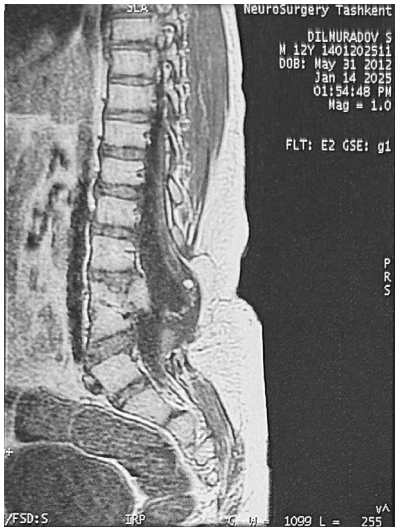


Figure 2. Lumbosacral MRI showing spina bifida from L2–S3.

Brainstem auditory evoked potentials (BAEP) showed mild irritation at the level of the superior olives and lower brainstem, as well as asymmetric efferent dysfunction of the accessory nerve, more pronounced on the right.

Craniometry and Instability Assessment. Key radiographic measurements were: Atlantodental interval (ADI): 5.7 mm (indicating atlantoaxial instability); Harrison's line: 13.2 mm (consistent with basilar invagination); The odontoid process was located above Wackenheim's line, confirming ventral brainstem compression (Figure 3).

Surgical Intervention. The decision to perform C1–C2 fixation with posterior distraction was based on preoperative craniometric analysis demonstrating that ventral brainstem compression was the primary driver of symptoms. Posterior fossa decompression was deemed unnecessary because cerebellar tonsillar descent was not the main cause of CSF flow obstruction, and postoperative MRI confirmed restoration of CSF dynamics without additional decompression. Furthermore, hydrocephalus was compensated and did not require shunting.

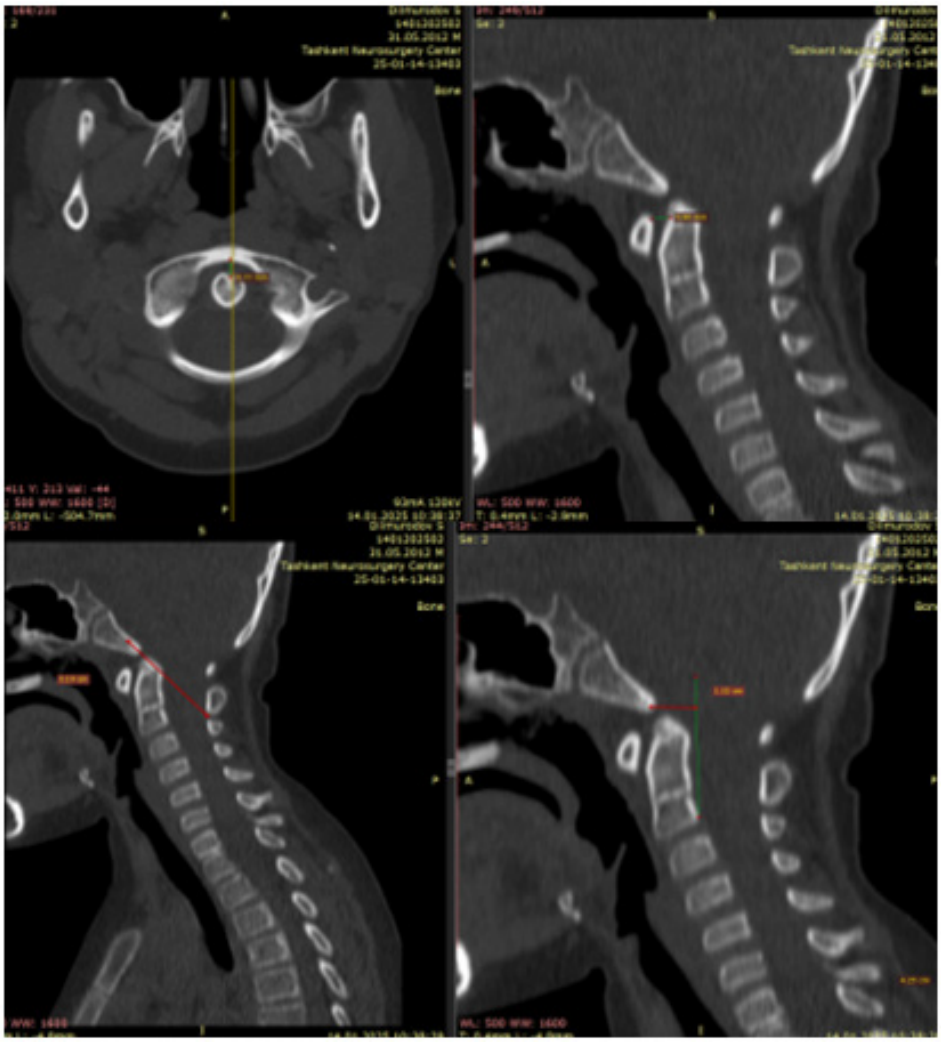


Figure 3. CT craniometry showing an abnormal atlantodental interval (5.7 mm), Wackenheim's line, and Harrison's line (13.2 mm), suggestive of basilar invagination.

Posterior stabilization at C1–C2 was performed under general anesthesia. Midline dissection extended from the occiput to C3. The laminae of C1–C3 and the occipital bone were exposed. Intraoperatively, pathological mobility of the atlantoaxial joint was confirmed. Bilateral C1–C2 transpedicular screws were inserted using the Harms and Magerl techniques under fluoroscopic control. Distraction of the screws achieved ventral decompression through caudal traction of the odontoid process. Connecting rods were applied, completing posterior fixation and anterior decompression (Figure 4).

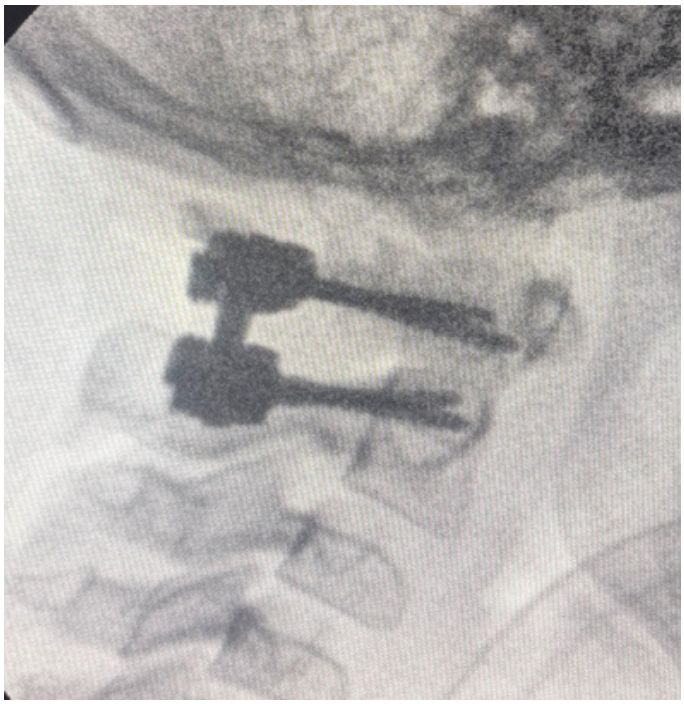


Figure 4. Intraoperative image showing placement of bilateral C1–C2 transpedicular screws and posterior distraction of the odontoid process.

Rationale for Surgical Strategy. Serial imaging revealed that the cerebellar tonsillar descent was long-standing and stable, while the atlantoaxial instability and cranial migration of the odontoid had progressed compared with prior scans. This progression, together with the patient's clinical deterioration, indicated that the primary pathological mechanism was ventral compression secondary to instability rather than worsening of the congenital posterior fossa malformation. Based on these findings, C1–C2 fixation with distraction and realignment was selected as the first-line surgical approach. The goal was to correct basilar invagination, restore the vertical alignment of the craniovertebral junction, and achieve indirect decompression of the brainstem and cervicomedullary junction. This approach aligns with the modern concept proposed by Goel et al., according to which basilar invagination and Chiari malformation represent secondary manifestations of chronic atlantoaxial instability, and stabilization alone may reverse neural compression and normalize cerebrospinal fluid (CSF) flow.

Simultaneous posterior fossa decompression was deliberately avoided because, in the presence of marked instability and a small posterior fossa, removal of posterior bony structures could exacerbate ventral deformation, increase craniovertebral mobility, and potentially lead to further brainstem compression. Additionally, considering the patient's compensated tethered cord syndrome and secondary hydrocephalus, gradual realignment and stabilization of the craniovertebral junction were deemed safer to prevent abrupt changes in CSF pressure that might accompany direct decompression. The postoperative course demonstrated significant improvement in gait and limb strength, with resolution of headache and vomiting, accompanied by radiological evidence of brainstem realignment and improved CSF flow. If neurological or radiological improvement had not been achieved after fixation, a second-stage posterior fossa decompression was planned as a contingency option. However, the observed clinical and imaging outcomes confirmed that fixation alone was sufficient to address the underlying pathology in this patient. The patient was monitored in the ICU for 24 hours. On postoperative day 1, headache significantly decreased, motor function in the upper limbs improved, and vomiting ceased. By postoperative day 5, the patient was able to walk independently.

At 10 days postoperatively, follow-up MRI and CT craniometry demonstrated significant anatomical and functional improvement: Effective ventral decompression was achieved through posterior C1–C2 distraction. Regression of medullary and cerebellar edema was observed on sagittal and axial MRI sequences. Restoration of cerebrospinal fluid (CSF) pathways was confirmed at the level of the fourth ventricular outlets and along the ventral surface of the brainstem. Craniometric parameters showed marked improvement: Atlantodental interval (ADI): reduced from 5.7 mm preoperatively to 4.3 mm postoperatively, indicating improved atlantoaxial alignment. Wackenheim's clivus line: the odontoid process was located below the line, confirming the absence of odontoid retroflexion and normalization of the clivus–dens relationship. Harrison's line: decreased from 13.2

mm to 9.3 mm, reflecting significant restoration of the craniovertebral junction anatomy and improved ventral decompression.

Postoperative Course. Clinically, the patient showed substantial improvement by postoperative day 5, including complete resolution of headache, cessation of vomiting, restored independent ambulation, and no residual neurological deficits. At 10 days postoperatively, follow-up MRI and CT craniometry demonstrated significant anatomical and functional improvement (Figure 5). The patient was discharged in satisfactory condition after 14 days and returned to school after 2 months with no neurological deficits. No postoperative complications were noted. Long-term follow-up at the RSSPMCN was recommended.

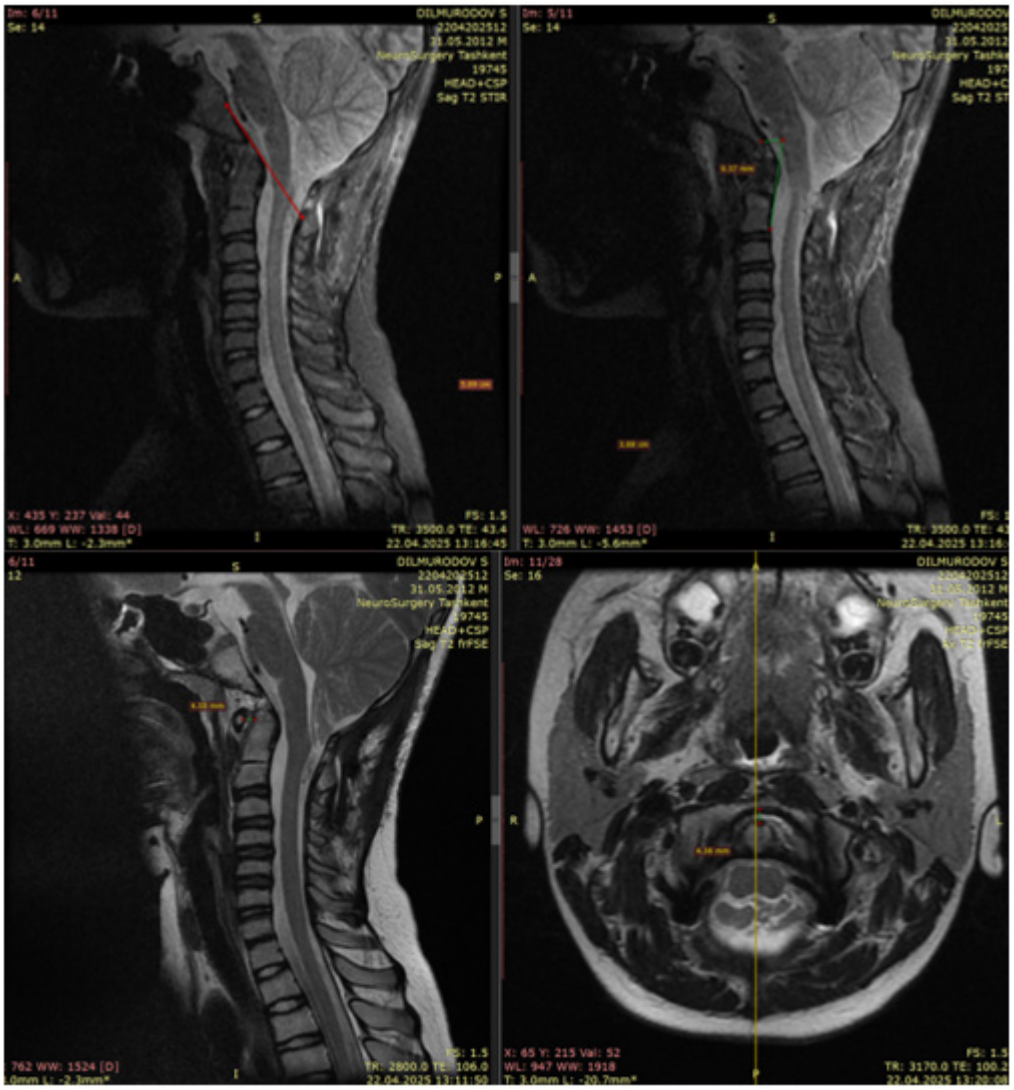


Figure 5. Postoperative MRI at day 10 demonstrating effective ventral decompression and restoration of CSF circulation.

The presented case illustrates a rare and complex constellation of craniospinal anomalies, including Arnold–Chiari malformation type II (CM-II), basilar invagination (BI), secondary hydrocephalus, and sequelae of closed spinal dysraphism, such as prior L2–L5 herniotomy, meningo(myelo)radiculocele, and compensated tethered cord syndrome (TCS). This combination represents a significant diagnostic and therapeutic challenge requiring a multidisciplinary and highly individualized approach.

Pathophysiological Considerations. CM-II is typically associated with open neural tube defects, particularly myelomeningocele, and is predominantly encountered in pediatric populations. Unlike CM-I, CM-II is characterized by more pronounced hindbrain herniation, brainstem compression, and complex anomalies involving the fourth ventricle and posterior fossa. Hydrocephalus is frequently present due to obstruction at the level of the foramina of Magendie and Luschka [5].

Basilar invagination is uncommonly described in the context of CM-II, especially in adults. However, when present, BI may exacerbate the neuro-orthopedic sequelae by contributing to cervicomedullary compression and worsening cerebrospinal fluid (CSF) flow obstruction. In our patient, BI likely played a critical role by compounding pre-existing CSF circulation disturbances and increasing axial traction forces at the craniovertebral junction (CVJ). The patient's history of childhood L2–L5 herniotomy and retrospectively diagnosed TCS introduced a relevant clinical dilemma: whether the progressive neurological deficits were primarily attributable to upper cervical (CVJ) compression or to caudal spinal cord tethering. The literature suggests that, in patients with CM-II and spinal dysraphism, both mechanisms may coexist and interact synergistically [8]. However, serial neurophysiological studies, including electromyography (EMG) and motor evoked potentials (MEP), can help identify the predominant pathophysiological mechanism. In our case, EMG revealed no signs of active denervation, supporting the presence of compensated tethered cord syndrome and shifting the focus of pathogenesis toward CVJ compression and disturbed CSF flow.

Rationale for Choosing C1–C2 Fixation with Distraction. Management of CVJ anomalies associated with CM-II and BI traditionally involves multiple surgical strategies, including posterior fossa decompression (PFD), ventriculoperitoneal (VP) shunting, endoscopic third ventriculostomy (ETV), untethering procedures, and posterior fixation. In this case, however, posterior C1–C2 fixation with distraction was chosen as the sole surgical strategy for the following reasons [6].

Dominant mechanism — ventral compression. Preoperative craniometry revealed a markedly increased atlantodental interval (ADI: 5.7 mm), suggesting atlantoaxial instability. Wackenheim's line and Harrison's line measurements (13.2 mm) confirmed significant basilar invagination and severe ventral brainstem compression.

Tonsillar descent was secondary. Although the cerebellar tonsils descended 16.1 mm, there was no significant posterior fossa crowding requiring PFD [12].

Hydrocephalus was compensated. Ventricular dilatation was mild, and there was no evidence of acute intracranial hypertension; thus, CSF diversion (VP shunting or ETV) was not indicated.

Indirect ventral decompression via distraction. Posterior distraction restored the ventral CSF pathways by realigning the odontoid process and decompressing the brainstem, thereby eliminating the need for anterior or combined approaches. This individualized surgical strategy minimized overall invasiveness, directly targeted the primary pathophysiological mechanism, and avoided unnecessary multiple interventions.

Mechanism of Symptom Development. A key point raised by the reviewer concerned whether the patient's symptoms were primarily caused by mechanical compression or by disturbed CSF flow. In this patient, the predominant mechanism was ventral brainstem compression due to BI, while secondary CSF flow obstruction contributed to compensated hydrocephalus. Preoperative MRI demonstrated marked deformation of the ventral medulla and upper cervical spinal cord. Clinically, progressive gait disturbance, lower-limb weakness, and occipital headaches correlated with compression of the ventral pyramidal tracts and brainstem nuclei. Restoration of CSF circulation and regression of medullary edema after distraction further support the conclusion that mechanical compression was the primary driver, whereas CSF obstruction played a secondary role.

Anatomical Location of CSF Flow Restoration. Preoperatively, CSF flow was markedly reduced in the ventral subarachnoid space around the medulla and at the fourth ventricular outlets. Postoperative MRI demonstrated restored CSF circulation, particularly at: 1. the lateral apertures of Luschka; 2. along the ventral surface of the medulla and pons; and 3. within the basal cisterns. These findings confirm that posterior C1–C2 distraction successfully addressed CSF obstruction by realigning the odontoid process and decompressing the ventral brainstem.

Posterior Distraction as an Alternative to PFD and Shunting [7]. Traditionally, patients with CM-II, BI, and secondary hydrocephalus undergo combined surgical approaches, including PFD and CSF diversion. However, in this case: PFD was avoided because tonsillar descent was not the primary source of CSF obstruction. VP shunting or ETV was not required due to compensated hydrocephalus. Posterior distraction alone restored CSF dynamics and relieved brainstem compression, thus eliminating the need for additional interventions. These findings align with a growing body of evidence suggesting that posterior fixation with distraction can serve both stabilizing and decompressive functions in selected CVJ anomalies, reducing the necessity for multistage procedures [10].

This case highlights the exceptional complexity of managing multi-component craniospinal anomalies, particularly when upper and lower spinal pathologies coexist and potentially interact. A tailored, pathophysiology-driven approach is essential, prioritizing the anatomical region that currently drives the clinical symptoms. In our patient, ventral brainstem compression and disturbed CSF dynamics at the CVJ were the dominant contributors to the neurological deficits and were successfully addressed by posterior C1–C2 fixation with distraction [11]. The lower spinal lesions, including compensated TCS, remained clinically stable and did not require intervention

at this stage. Close longitudinal monitoring with serial MRI and neurophysiological testing remains critical to detect any future evolution toward instability, renewed tethering, or worsening hydrocephalus.

CONCLUSION AND RECOMMENDATIONS

Thus, an individualized surgical approach utilizing posterior distraction and fixation may offer a safe and effective alternative to anterior decompression in pediatric craniovertebral junction (CVJ) anomalies. In the present case, the key step was the elimination of ventral compression via posterior odontoid distraction, which allowed for effective decompression without the need for anterior surgical access or cerebrospinal fluid (CSF) diversion procedures. Comprehensive preoperative diagnostics—including craniometric analysis, neurophysiological assessment, and careful consideration of the patient's surgical history (notably, prior repair of spinal dysraphism)—enabled the formulation of a tailored surgical strategy with minimal risk. These results reinforce the necessity of a multidisciplinary approach and emphasize the importance of pathophysiological stratification in each individual case. Nevertheless, long-term follow-up and comparative clinical studies are essential to definitively assess the efficacy of posterior distraction as a treatment modality for CVJ anomalies.

In similar cases, particularly those involving complex CVJ anomalies in pediatric patients, intraoperative neurophysiological monitoring is strongly recommended to ensure the functional safety of surgical procedures, especially when operating near the brainstem and spinal cord. Postoperative follow-up should extend for at least 12 months to enable objective evaluation of neurological outcomes, recurrence rates, and compensatory changes. Comparative studies of posterior distraction versus anterior decompression in patients with similar anatomical profiles are warranted to identify the most effective surgical strategy and refine the selection criteria for the surgical approach. Patients with CVJ anomalies should be enrolled in prospective multicenter trials aimed at developing unified protocols and evidence-based standards for the treatment of pediatric craniovertebral junction anomalies.

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