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Congenital Neonatal Synchronous Triple and Dual Neural Tube Defects with Thoracolumbar, Lumbar and Sacral Myelomeningoceles: Two Case Reports

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Abstract

This manuscript describes two exceptionally rare neonatal presentations of synchronous multiple neural tube defects (NTDs), involving separate thoracolumbar, lumbar, and sacral myelomeningoceles. We report two neonates presenting with synchronous dual and triple, open and closed spinal dysraphisms—distributed across thoracolumbar, lumbar, and sacral regions—without associated cranial defects. Both infants underwent early surgical repair. Case 1 demonstrated favorable early neurological outcomes, while Case 2 exhibited persistent lower-limb weakness and neurogenic bladder.

Keywords: Dual Neural Tube Defects; Synchronous Spinal Dysraphism; Thoraco-Lumbar Myelomeningocele; Lumbar Myelomeningocele; Sacral Myelomeningocele; Congenital Spinal Anomalies; Neonatal Neurosurgery; Open Spinal Dysraphism; Multiple Neural Tube Defects; Embryological Neurulation Failure

Introduction

Dual or multiple neural tube defects (NTDs) are exceptionally rare congenital anomalies, representing <2% of all NTDs [1]. Their synchronous occurrence in the thoracolumbar, lumbar, and sacral regions is even more uncommon and presents significant diagnostic, surgical, and prognostic challenges [2]. NTDs arise from incomplete closure of the neural tube between days 18–28 of embryogenesis. While single-site defects such as myelomeningocele are well recognised, multiple synchronous NTDs are exceedingly rare, with only isolated reports in the literature.

We present two neonates with synchronous triple and dual myelomeningoceles, contributing to the limited global literature on this rare phenomenon..

Case Reports

Case 1

A term female neonate was delivered via spontaneous vaginal delivery to a 27-year-old primigravida with no antenatal folate supplementation. Pregnancy was uneventful, and anomaly scans reportedly normal.

At birth, three distinct dorsal midline defects were noted:

1. Upper lumbar lesion (L1–L2):3 × 3 cm myelomeningocele,skin covered sac with visible umbilication at the centre neural placode, no CSF leak.
2. Lower lumbar lesion (L3–L5): 5 × 5 cm myelomeningocele, fully skin covered lesion.
3. Sacro-coccygeal sinus with pigmented surrounding skin and spina bifida occulta.

Neurological examination revealedno muscle or joint weakness, preserved knee extension, normal anal tone, normal upper limb function.

Diagnostic Assessment: Ultrasound scan of brain and spine confirmed two separate open

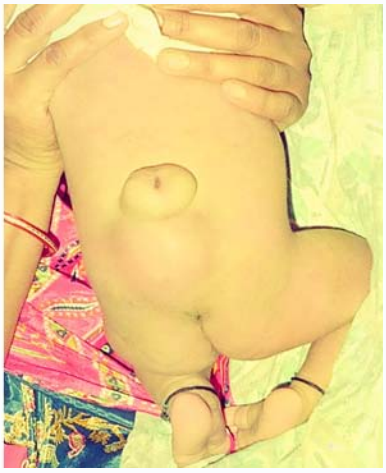

Figure 1: Clinical appearance of tripleupper lumbar, lower lumbar and sacral myelomeningoceles in Case 1.
Three distinct dorsal midline defects at birth:
Upper lumbar lesion (L1–L2): 3 × 3 cm myelomeningocele, skin covered sac with visible umbilication at the centre neural placode, no CSF leak, Lower lumbar lesion (L3–L5): 5 × 5 cm myelomeningocele, fully skin covered lesion and the Sacral sinus with pigmented surrounding skin and spina bifida occulta.

dysraphic defects in the lumbar region and spina bifida occulta in the sacrococcygeal area, no intervening segmental fusion, low-lying conus at L4, no Chiari II malformation and normal ventricles.

Therapeutic Intervention: Surgical repair was performed with excision of non-functional neural tissue, reconstruction of neural placode, dural closure with autologous fascia, layered paraspinous muscle and skin closure.

Outcome and Follow-up: Postoperative recovery was uneventful. At 6-month follow-up, good lower-limb motor function. With normal bowel, bladder functions and no hydrocephalus.

Case 2

A preterm male neonate (36+4 weeks) was born to a 32-year-old multiparous mother with poorly controlled gestational diabetes. No teratogenic exposures were reported.

Clinical Findings: Two separate thoraco-lumbar and sacral defects were identified. Thoracolumbar lesion (T122–L3) measuring 4 × 3.5 cm myelomeningocele with very thin and open coverings dorsally and CSF leak present with a dimple around the neural placode.

Sacral lesion (S2–S3) measuring 1.5 × 2 cm myelomeningocele with central umbilication and flattened neural placode.

Neurological examination showed absent ankle dorsiflexion, weak knee flexion, absent anal reflex and normal upper limb and cranial nerve examination.

Diagnostic Assessment: Ultrasound brain and spine revealed dual open spinal dysraphisms, low-lying conus at L5, mild ventriculomegaly, no Chiari II malformation.

Therapeutic Intervention: Urgent surgical repair was undertaken within 12 hours due to CSF leakage with debridement of non-viable neural tissue, dural reconstruction, muscle and skin closure using bilateral advancement flaps.

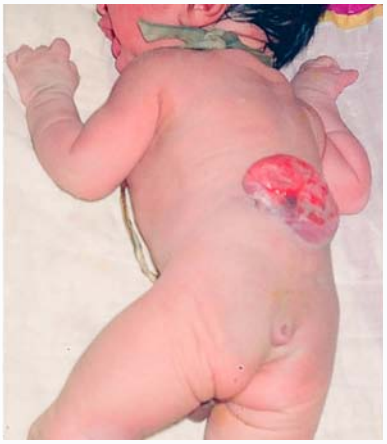

Figure 2: Clinical appearance of dual myelomeningoceles in Case 2. Larger lumbar lesion (4 × 3.5 cm) at L2–L3 with active CSF leakage and a smaller sacral lesion (1.5 × 2 cm) at S2–S3. The sacral placode appears flattened with epithelialised margins.

Feature	Case 1 (Triple NTDs)	Case 2 (Dual NTDs)
Gestation	Term	Preterm (36+4 weeks)
Maternal risk factors	No folate supplementation	Poorly controlled gestational diabetes
Lesion levels	L1–L2, L3–L5, sacrococcygeal	T12–L3, S2–S3
Number of defects	Three	Two
CSF leak	None	Present (thoracolumbar lesion)
Conus level	L4	L5
Chiari II malformation	Absent	Absent
Ventriculomegaly	None	Mild, progressive
Lower-limb motor function	Normal	Weakness (ankle, knee)
Bladder function	Normal	Neurogenic bladder
VP shunt	Not required	Required at 20 weeks
Early outcome	Excellent	Moderate impairment

Table 1: Comparison of two cases.

Outcome and Follow-up: At 3-month follow-up the patient had persistent lower-limb weakness, neurogenic bladder requiring catheterisation, progressive ventriculomegaly requiring VP shunt at 20 weeks.

Discussion

Neural tube defects remain among the most challenging congenital anomalies encountered in neonatal practice, yet the vast majority present as single, isolated lesions [3]. In contrast, the two neonates described in our report were born with synchronous dual and triple open spinal dysraphisms—a phenomenon so rare that only a small handful of cases have been documented worldwide [4]. Their presentation immediately raised questions that extend far beyond routine clinical management: neurulation fail at two and three distinct sites, associated embryological mechanisms which could allow such a pattern and how should clinicians approach diagnosis and surgical

Theory	Description	Relevance to Cases
Multisite primary neurulation failure	Simultaneous closure defects at multiple embryonic sites	Explains triple lesions in Case 1
Multisite "zippering" failure	Disruption of bidirectional closure along the neural tube	Consistent with discontinuous defects
Secondary rupture theory	Previously closed tube reopens due to mechanical/vascular insult	Possible in thin-walled thoracolumbar lesion in Case 2
Environmental/ metabolic factors	Folate deficiency, maternal diabetes, hyperthermia	Present in Case 2 (diabetes)
Genetic susceptibility	Multifactorial inheritance	May contribute to multifocal patterns

Table 2: Embryological mechanisms.

planning when the anatomy itself defies expectation.

These cases offered a unique window into the complexity of early embryonic development, while simultaneously challenging our assumptions about the clinical spectrum of open spinal dysraphism [5]. Both infants required urgent, carefully coordinated neurosurgical intervention, yet their early outcomes diverged in ways that underscore the delicate interplay between lesion level, CSF dynamics, and neurological vulnerability. By presenting these two cases side by side, we highlight not only the rarity of dual NTDs but also the clinical nuances that influence prognosis, even when the anatomical pattern appears superficially similar.

The rarity of triple and dual myelomeningoceles—representing far less than 2% of all neural tube defects—makes each case a valuable lens through which to examine competing embryological theories. Dual or multiple NTDs challenge traditional embryological models [6]. These cases invite renewed consideration of:

- Multisite primary neurulation failure, suggesting simultaneous defects at separate closure points.
- The multisite "zippering" model, in which closure progresses bidirectionally and may falter at more than one location.
- Secondary reopening or rupture, implying that previously closed segments may be vulnerable to mechanical or vascular disruption.
- Environmental and metabolic influences, including maternal folate status and glycemic control, which may modulate closure integrity.

Diagnosis typically involves prenatal ultrasound and MRI, followed by comprehensive imaging (MRI, CT scans) after birth to assess the full extent of both lesions and any associated conditions. Differential diagnosis include single myelomeningocele with satellite meningocele, split cord malformation with dual external sacs, dermal sinus tract with secondary cystic expansion, amniotic band disruption sequence [7].

Dual myelomeningoceles pose unique challenges: Atypical cutaneous findings may obscure the presence of a second lesion. Neurological deficits may not correlate with the most obvious lesion. Surgical planning must consider tethering, CSF dynamics, and skin coverage [8].

Clinical implications

The co-occurrence of these conditions presents significant clinical challenges and potentially more severe outcomes than either condition alone:

- **Neurological Deficits:** The patient would likely experience motor and sensory impairment below the level of the *more severe* lesion (likely the MMC), ranging from muscle weakness to paralysis, as the neural tissue in both defects is damaged or tethered.
- **Associated Conditions:** The patient is at high risk for associated anomalies common with MMCs, such as:

1. **Hydrocephalus:** The buildup of fluid in the brain, often requiring a shunt.
2. **Chiari II malformation:** Downward displacement of brain tissue into the spinal canal, which can cause life-threatening breathing and swallowing problems.
3. **Tethered Cord Syndrome:** Scarring and stretching of the spinal cord, which can worsen neurological function as the child grows.

- **Organ Dysfunction:** The patient may experience significant bladder and bowel dysfunction due to nerve damage.
- **Surgical Complexity:** Managing two distinct, complex lesions in different regions (even if both are lumbar) requires highly specialized surgical planning to ensure stable coverage of neural tissue while addressing associated complications [9].

Comparison of the two cases (Table 1)

These cases provide valuable insights intothe embryological basis of multifocal neurulation failure, the diagnostic importance of full-length spinal clinical examination and imaging, early prenatal and perinatal detection, timely surgical repair, early multidisciplinary management, surgical considerations when managing multiple open dysraphic lesions, early neurological and urological outcomes following repair.

To our knowledge, very few reports have documented three and two distinct open spinal dysraphisms in the same neonate, and even fewer have compared early outcomes across two such cases [10]. We

Surgical Step	Key Considerations	Application in Cases
Lesion prioritisation	Address leaking or higher-risk lesion first	Case 2: thoracolumbar lesion repaired urgently
Placode reconstruction	Avoid neural compression; recreate tubular structure	Both cases
Dural closure	Autologous fascia preferred; watertight seal essential	Both cases
Muscle coverage	Paraspinous advancement flaps to reduce tension	Case 2 required bilateral flaps
Skin closure	Tension-free closure to prevent breakdown	Achieved in both cases
Postoperative monitoring	Hydrocephalus, infection, tethering	Case 2 developed hydrocephalus

Table 3: Surgical considerations.

believe this report will be of interest and will resonate with clinicians across to neonatology, paediatric neurosurgery, radiology, biology and developmental embryology.More broadly, it contributes to the ongoing conversation about how—and why—neural tube closure can fail in such an unusual multifocal pattern.

Neural tube defects offer a rare opportunity to observe the consequences of disrupted embryological events that occur within an extraordinarily narrow developmental window. The two neonates described in our report presented with synchronous triple and dual open spinal dysraphisms, a pattern that challenges traditional models of primary neurulation. Their anatomy raises fundamental questions about how the neural tube closes, how closure sites interact, and how multifocal failure can occur in an otherwise normally developing embryo [11].

By presenting two neonates with distinct but synchronous thoracolumbar, lumbar and sacral defects, we highlight how clinical observations can illuminate embryological processes that remain incompletely understood. The contrasting postoperative outcomes of these infants further underscore how the highest-level lesion, rather than the number of defects, determines neurological prognosis—an insight that aligns with developmental principles of segmental innervation.

These two cases presented an unusual and demanding surgical scenario: synchronous triple and dual open spinal dysraphisms in neonates, each requiring urgent yet carefully prioritised operative intervention. While single-site myelomeningocele repair is well established, the presence of two anatomically distinct lesions raises critical questions about operative sequencing, tissue handling, CSF dynamics, and postoperative risk mitigation.

Management involves a multidisciplinary approach, including neurosurgeons, urologists, orthopedic surgeons, and physiotherapists, to manage lifelong complications.

Immediate Surgery: The open MMC defect requires prompt surgical closure within 48 to 72 hours of birth to prevent infection and further damage.

Staged or Simultaneous Repair: The HLMMC may be repaired simultaneously or in a staged manner, depending on the specifics of the case and patient stability.

Long-term Follow-up: The patient will require ongoing monitoring and potential further surgeries (e.g., shunt placement for hydrocephalus, spinal cord untethering) throughout their life.

Our report highlights the real-time decision-making that shaped each surgical plan:

- Determining the order of repair when two defects compete for immediate attention.
- Assessing skin availability and flap options to ensure tension-free closure across multiple sites.
- Balancing urgency against stability, particularly in the presence of CSF leakage.
- Evaluating the risk of tethering when reconstructing two separate neural placodes.
- Anticipating postoperative hydrocephalus, especially in the context of altered CSF flow after dual repair.

Although both neonates underwent early surgical correction, their postoperative trajectories diverged significantly. These differences underscore how lesion level, CSF leak status, and intraoperative findings influence neurological and urological outcomes[12]. By presenting these cases side by side, we offer a rare comparative perspective on how surgical strategy—not just anatomy—shapes prognosis.

Conclusion

These cases highlight the importance of meticulous clinical and radiological evaluation in neonates with atypical or multiple dorsal midline lesions. Early multidisciplinary intervention is essential to optimise outcomes.

Key Take Home Learning Points

- Triple and dual neural tube defects are extremely rare and require high clinical suspicion.
- Full-length spinal USS/MRI is essential to identify synchronous lesions.
- Early surgical repair reduces infection risk and prevents further neurological deterioration.
- Prognosis depends on the highest-level lesion and associated anomalies rather than the number of defects.
- Multidisciplinary follow-up is crucial for managing bladder, bowel, and motor outcomes.

Compliance Checklist

Patient and Public Involvement

- The patient's caregivers were involved in providing consent and clinical history.
- No direct patient/public involvement in study design, analysis, or manuscript preparation.
- The case is reported to raise awareness of adverse drug effects in infants.

Ethics Approval and Consent to Participate

- Ethics approval was not required for a single case report under institutional policy.
- Written informed consent was obtained from the infant's parents for publication.

Consent for Publication

- Parents provided written consent for publication of clinical details and anonymized images (if any).
- Identifying information has been removed to ensure confidentiality.

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Competing Interests

- The authors declare no competing interests.
- No financial or personal relationships influenced the preparation of this manuscript.

Data Availability Statement

- All data relevant to the case are included in the article.
- No additional datasets were generated or analyzed.

Provenance and Peer Review

- Not commissioned; externally peer reviewed.

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