Case Report	
	

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Title	**Spina Bifida Presenting with a Unique Complication: Uterovaginal Prolapse in a Neonate**
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Abstract

Introduction: Uterovaginal prolapse (UVP) in neonates is a rare condition, with only a few cases reported in the literature. The exact etiology and pathogenesis of neonatal UVP are not fully understood. Nevertheless, it is believed that spina bifida, particularly myelomeningocele, is a common risk factor associated with the development of UVP in neonates. Defective innervation of the pelvic floor in such cases can lead to weakness of the pelvic muscles and ligaments, resulting in congenital uterovaginal prolapse. Consequently, UVP can manifest within the first few days of life or even at birth.

Background: While UVP is relatively common in elderly post-menopausal women, it is uncommon in young girls and exceedingly rare in neonates. In neonates, it is almost always secondary to a weak pelvic floor resulting from spina bifida. However, it remains unclear why UVP is not a consistent feature in all or most cases of spina bifida. Interestingly, most patients in this age group respond well to conservative treatment, and surgical intervention is only rarely required.

We report the case of a newborn female with a large lumbosacral myelomeningocele and cleft lip and palate who presented for routine initial care and work-up. On the day of admission, she developed uterovaginal prolapse, which was managed conservatively with manual reduction and mild sedation in the NICU. Gradually, the frequency of prolapse decreased and, over a period of two weeks, it ceased entirely. At the end of six months of follow-up, she remains asymptomatic with no recurrence of prolapse.

Case Report

A six-hour-old, full-term female newborn with a large lumbosacral myelomeningocele was referred for routine work-up and management. She was born to a non-consanguineous couple via lower-segment cesarean section. Antenatal care was limited, and no antenatal ultrasound was available. The baby cried immediately after birth and had APGAR scores of 6 and 8 at 1 and 5 minutes, respectively. She had passed meconium before arrival at our hospital. On examination, her weight was 2800 grams, and her vital signs were stable. She had a right-sided complete cleft lip and palate, and an approximately 8 × 6 cm, well-covered lumbosacral myelomeningocele (MMC) (Figure 1). The anterior fontanelle was not full. She also had paraplegia. Anal mucosa was visible through the anal opening, and the anocutaneous reflex was absent, suggesting a patulous anus. The rest of the systemic examination was normal.

Her hematological investigations were within normal limits. Renal ultrasound was reported as normal, while cranial ultrasound showed mild hydrocephalus. A 2D echocardiography revealed an ostium secundum atrial septal defect (ASD).

On the day of admission, a few hours later, she developed uterovaginal prolapse (UVP) (Figure 2). The prolapsed tissue was healthy with pink mucosa. Prolapse was digitally reduced under aseptic precautions, but it recurred every few hours with minimal straining by the baby. Each episode was managed with digital reduction. Tube feeding was initiated due to the cleft palate. The baby was nursed in the lateral position to avoid trauma to the MMC. Mild oral sedation was given since she was on tube feeds. Over time, the frequency of prolapse gradually decreased.

A plastic surgery consultation was obtained for the cleft. As the family wished to return home for personal reasons, they were trained in digital reduction of the prolapse and tube feeding. The baby was subsequently discharged. The family was also counseled regarding the management of MMC and hydrocephalus, but they did not consent to MMC repair at that time.

At the two-week follow-up, the parents reported that the prolapse had gradually decreased in frequency and had not recurred. Currently, the child is under close supervision, and at the six-month follow-up, she is thriving well with no recurrence of prolapse. Definitive treatment for MMC is pending, awaiting the family's consent.

Discussion

UVP is a common condition in elderly post-menopausal women but is rarely seen in infants and neonates [1]. In neonates, UVP is mostly associated with spina bifida [2, 3]. Uterine prolapse has been described in ancient medical literature, including the Egyptian Papyri [4]. A 2015 literature review reported only 67 cases of UVP in neonates between 1723 and 2014 [5].

The uterus and vagina are normally supported in their anatomic position by the pelvic diaphragm and endopelvic fascia, which condenses to form the uterosacral, cardinal, and pubocervical ligaments. Congenital UVP may result from weakness of the pelvic floor, usually due to congenital muscular deficiency or defective innervation. The etiology in neonates with spina bifida appears multifactorial—congenital muscular and ligamentous weakness combined with defective innervation contributes to UVP development [2]. Malpas (1955) classified UVP into primary (due

to congenital pelvic support weakness) and secondary (due to increased intra-abdominal pressure during fetal life) [6]. Prolonged breech presentation and birth trauma have also been implicated [7]. Other known risk factors include prematurity, increased intra-abdominal pressure, pelvic skeletal deformities, and cervical anomalies [7].

Management can be conservative and/or surgical. Conservative management is successful in over 90% of cases and typically involves digital reduction of the prolapse, as described by Bayatpour et al. [8]. This technique is simple and can be easily taught to caregivers for use at home. Over time, with the decline of maternal estrogen exposure and resolution of edema, the uterus typically becomes fixed in its normal position and prolapse resolves. Surgical intervention is required when conservative treatment fails. Various surgical techniques are described in the literature, including purse-string suture, sling procedures, cervical sacropexy, uterine ventrosuspension, and abdominal sacrocolpopexy [9]. Some of these may be performed using minimally invasive methods. Radical options like hysterectomy and cervical amputation are rarely required [10].

Learning Points

- UVP is very rare in neonates, with most cases secondary to spina bifida.
- Untreated prolapse may lead to vaginal mucosal injury, increasing the risk of infection. Over time, endometrial metaplasia and urinary retention followed by obstructive uropathy can occur.

- More than 90% of UVP cases respond to conservative treatment, which is simple and can be managed at home.
- Close monitoring and regular follow-up are essential to prevent complications and to intervene if conservative management fails.

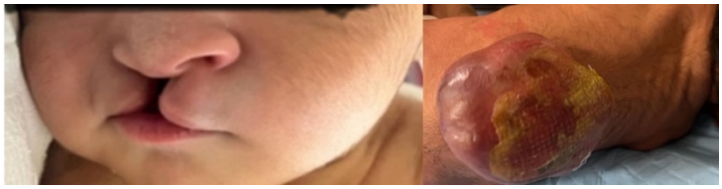

Fig. 1 showing cleft lip and large lumbosacral myelomeningocele.

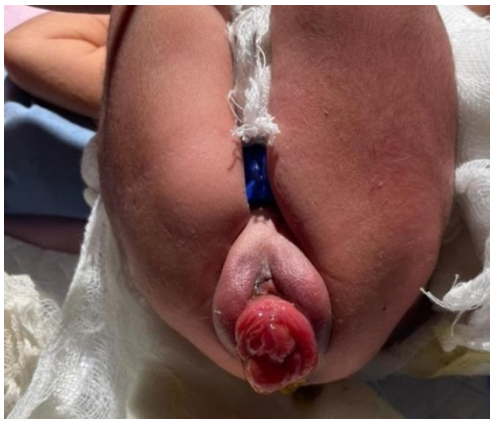

Fig. 2 showing complete uterovaginal prolapse.

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