Myelomeningocele (MMC) is a common congenital defect affecting 0.5–1 in 1,000 live births. Management involves surgical repair of the neural tissues and soft-tissue cover of the defect with the immediate goals of protecting the spinal cord, preserving function and, preventing cerebrospinal fluid (CSF) leakage and infection. Often, small defects are managed by direct closure while complex reconstruction is usually required for larger ones. Techniques utilized include skin grafting, and local rotational, advancement and transposition flaps, such as V-Y, double Z-plasty, Limberg and rhomboid flaps. More advanced multi-layered soft-tissue reconstruction that includes periosteal and musculocutaneous flaps such as latissimus dorsi or gluteus maximus flaps have also been advocated.

Wound complications are the most common complications encountered in the early period following MMC repair and cause significant morbidity. Furthermore, these wounds are typically compounded by infection, ischemia, and CSF leakage resulting in increased morbidity.

We report the case of a neonate who developed extensive wound necrosis with dehiscence following primary repair of myelomeningocele. The large defect was reconstructed using transposition fasciocutaneous flaps and negative pressure wound therapy applied over the flap donor sites resulting in wound closure, alleviating the need for further surgery. We report this case to highlight the importance of local flap design in reconstruction of the complex wounds seen following meningomyelocele repair wound necrosis. Additionally, we report the unique utilization of negative pressure wound therapy in the management of myelomeningocele.

CASE PRESENTATION

A female neonate with a prenatal diagnosis of myelomeningocele and hydrocephalus was delivered at term by cesarean section weighing 3206g with a head circumference 35cm. Local examination revealed a midline thoracolumbar 8 × 5 cm mass with CSF leakage. Postnatal contrast computed tomography scan and MRI confirmed a posterior arc spinal defect with Arnold Chiari II malformation and urological anomalies.

Primary closure of the defect was undertaken within the first 24 hours postnatal by neurosurgeons. The dura was repaired with artificial membrane and fibrin sealant applied to prevent CSF leakage. The skin edges were undermined considerably and direct midline closure of the wound performed.

Two weeks later, necrosis of the wound skin edges developed leading to progressive wound dehiscence and exposure of the underlying artificial membrane covering the dura. This was accompanied by superficial surgical-site

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The child was managed with dressings and optimized in preparation for secondary repair. Two months later, the patient was returned to theater for reconstruction. Intraoperatively, the artificial membrane was removed, and spinal cord was found covered by spontaneous tissue bridging under the artificial membrane. After debridement, the resulting skin defect measured 9 × 4 cm (Fig. 1). We reconstructed this defect using 2 transposition fasciocutaneous flaps measuring 11 × 5.5 cm and 9.5 × 5 cm from the right posterior chest wall and left gluteal area respectively, avoiding areas initially undermined during the primary repair operation. The flap donor areas were covered with Terudermis artificial dermis (Olympus Terumo Biomaterials Corp, Tokyo, Japan) (Fig. 2).

Three weeks later, the wounds were noted to be clean with no CSF leakage. A small additional 1 × 1 cm superficial wound developed following dermal necrosis of the superior flap without exposure of the dura. NPWT was then applied over the flap donor sites and the site of tip necrosis with a view to prepare the wound bed for secondary closure or skin grafting (Fig. 3). V.A.C VeraFlo Therapy (Acelity, San Antonio, Tex.) was used at -75 mm Hg with dressings changed twice a week. However, over a period of 2 weeks, wound closure was achieved alleviating the need for further surgery (Fig. 4).

DISCUSSION
Wound necrosis with dehiscence following repair of MMC is a serious impediment to recovery that can result in CSF leakage, damage of exposed neural tissues and life-threatening infection. The key etiologic factors in the pathophysiology of these wounds are hematoma formation, surgical-site infection and local tissue ischemia often associated with high wound tension. Wound complications occur postoperatively in 24% of MMC patients, half of which are superficial wound dehiscence, while major flap necrosis requiring surgical reconstruction complicates 1–8% of repairs.

With the limited literature on the secondary repair of MMC available, our approach aimed at covering the defect with well-vascularized local flaps by keeping the flap base away from the area of tissue originally undermined during the initial failed repair. Additionally, the flap donor areas were covered with artificial dermis as opposed to primary closure to allow for a tension-free closure of the wound and avert recurrence of flap necrosis. We opted for artificial dermis as opposed to split-thickness skin grafts (STSG) as a precaution considering local wound ischemia.
may have contributed to wound necrosis and failure of the primary repair. We planned to reassess the wounds after 1–2 weeks for areas of flap necrosis and perform an STSG to cover these and the initial flap donor areas at once instead of performing multiple STSG, which may have been unduly distressing for both the child and the parents. NPWT was then used for wound bed preparation and to deter infection but proceeded to attain complete wound closure, avoiding the need for STSG.

NPWT has been successfully used in the treatment of various wounds in pediatric patients. Despite this, its use in the management of MMC has been limited due to concerns regarding CSF leakage. Correspondingly, literature is scarce. Katano H et al. reported a case of secondary MMC repair that applied NPWT directly to the 9.42 cm² (3 × 4 cm) defect before surgical reconstruction following wound necrosis of the primary repair. In contrast, our patient suffered a defect 3 times larger, 28.27 cm² (4 × 9 cm) with wide exposure of the artificial dura membrane that necessitated the different approach as direct application of NPWT may have caused injury to the exposed neural tissues and CSF drainage. We instead applied flap coverage for the exposed area and NPWT for wound bed preparation of the flap donor areas. Although further studies are needed, we believe our approach may be better suited for reconstruction of large wounds after major wound necrosis or in the primary repair of large MMC than the former.

**SUMMARY**

We report this case to highlight the importance of local flap design in preserving flap vascularity and the combined utilization of NPWT for closure of flap donor sites in the secondary reconstruction of wounds following extensive necrosis with dehiscence of MMC repair. NPWT combined with flap transfer in the management of MMC may reduce the need for further invasive reconstruction.

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