

## Lumbosacral lipomyelomeningocele with anomalous osseous limb in a 3-month-old female

Sean L. Wilkes, MS, ALM; Jay J. Choi, MD, MSc; and Veronica J. Rooks, MD

A patient with lipomyelomeningocele (known in utero) presented for MRI characterization prior to surgical procedure at three months of age. Cross-sectional imaging revealed a spinal dysraphism of the lower lumbar spine, with a posterior spinal defect spanning L4 to S2 subcutaneous fat intrusion, and distal spinal cord extrusion. An osseous excrescence was also appreciated, articulating with the left iliac bone. This case demonstrates the youngest known lipomyelomeningocele with accessory limb and the abnormal growth of multiple tissue types at the site of spinal dysraphism—a potential consequence of dedifferentiated cell proliferation originating from a secondary neural tube defect or rachipagus parasitic twinning.

### Introduction

Spinal dysraphisms are developmental congenital anomalies resulting from dorsal embryo malformations. These malformations are classified as open (as found in spina bifida cystica) or closed (as seen in spina bifida occulta), and are typically discovered in utero, at birth, or during infancy. Lipomyelomeningocele is a closed neural-tube defect (NTD) formed mainly due to a defect in primary neurulation in which mesenchymal tissue enters into neural placode and forms lipomatous tissue (1-3). The spinal cord becomes tethered to the fat and may then be pulled downward in the course of development as well as extruded from the vertebral canal into adjacent tissues. Various clinical impairments may develop.

### Case report

A 3-month-old female presented with lipomyelomeningocele for further characterization prior to surgical resection. The patient was born at 39 weeks via spontaneous vaginal delivery with no other known complications. A 3-cm lumbosacral lipomyelomeningocele was discovered via MRI at 28 weeks of gestation. Fetal MR imaging was performed at 28 weeks of gestation using a 12-channel body coil with the same 1.5 T Signa MRI unit. The following sequences were obtained: axial T2 single shot FSE (SSFE, with and without fat suppression); sagittal T2 SSFE; and coronal T2 SSFE. The field of view was 41 x 41 mm, and the matrix size was 384 x 224 with a NEX of 0.57. The slice thickness was 6 mm, with spacing of 1 mm.

Neonatal head ultrasound was performed at birth to evaluate for ventriculomegaly, which was normal. The patient demonstrated normal motor and sensory activity and was subsequently discharged with plans for followup in an outpatient setting and referral to a pediatric neurosurgeon. At three-month followup, the patient continued to demonstrate absence of any sensorimotor or developmental pathology. MR imaging was performed to prepare for neurosurgical intervention. 3D MR imaging of the lumbar spine was performed using a body spine array coil with a 1.5TGE Healthcare. The following sequences were obtained: axial proton density FSE, T1 FSE (with and without fat suppression), and T2 FSE (with and without fat suppression); sagittal T1 FSE non-fat-suppressed and T2 FSE; and gadolinium-enhanced axial and sagittal T1 FSE (with fat suppression). The field of view was 28 x 28 mm,

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**Citation:** Wilkes SL, Choi JJ, Rooks VJ. Lumbosacral lipomyelomeningocele with anomalous osseous limb in a 3-month-old female. *Radiology Case Reports*. (Online) 2015;10(1):1051.

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Dr. Wilkes is affiliated with the Uniformed Services University of the Health Sciences, Bethesda MD, and Drs. Choi and Rooks are both in the Department of Radiology, Tripler Army Medical Center, Honolulu HI. Contact Dr. Wilkes at [sean.wilkes@usuhs.edu](mailto:sean.wilkes@usuhs.edu).

**Competing Interests:** The authors have declared that no competing interests exist.

**DOI:** 10.2484/rcr.v10i1.1051

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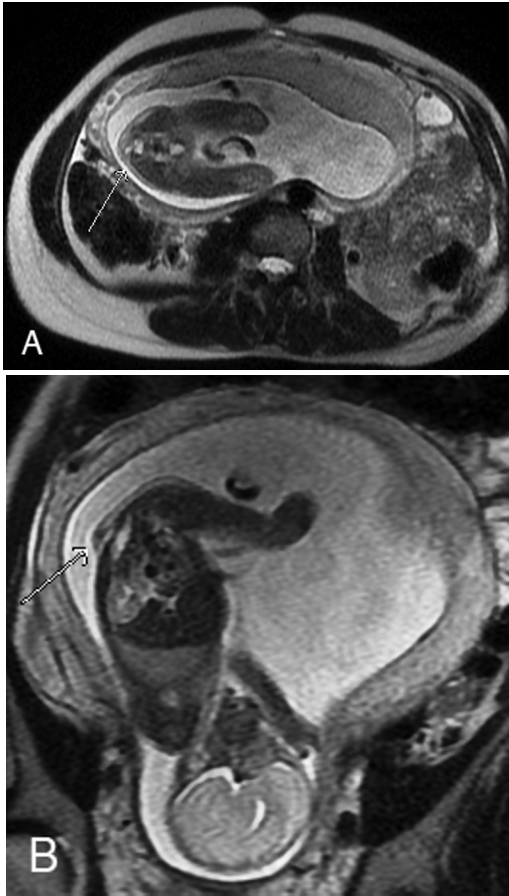


Fig. 1. Axial T2 SSFSE non-fat-suppressed image. The arrow demonstrates protrusion of CSF and neural elements through a covered posterior fusion defect (A). Sagittal T2 SSFSE non-fat-suppressed image demonstrates protrusion of CSF and neural elements through a covered posterior fusion defect (B).

and the matrix size was 256 x 128 with a NEX of 2.0. The slice thickness was 5 mm, with spacing of 2 mm.

The MR images (Figs 1-3) demonstrated a defect of the posterior elements spanning L4-S2 covered by subcutaneous tissue. Subcutaneous fat was noted within the spinal column at the level of the defect on the left, causing rightward shift of the distal spinal cord and surrounding CSF. The distal spinal cord was low-lying, exiting the defect at the L4-5 level with the CSF-filled meningocele and extending cranially to the L3 level.

A thin curvilinear osseous limb, 4 cm in length with cortex and medullary bone, articulating with the left sacrum, was seen extending posteriorly through the subcutaneous fat, consistent with an osseous limb.

### Discussion

Lipomyelomeningoceles are a category of spinal dysraphisms due to a defect in primary neurulation from non-disjunction in which mesenchymal tissue enters into the

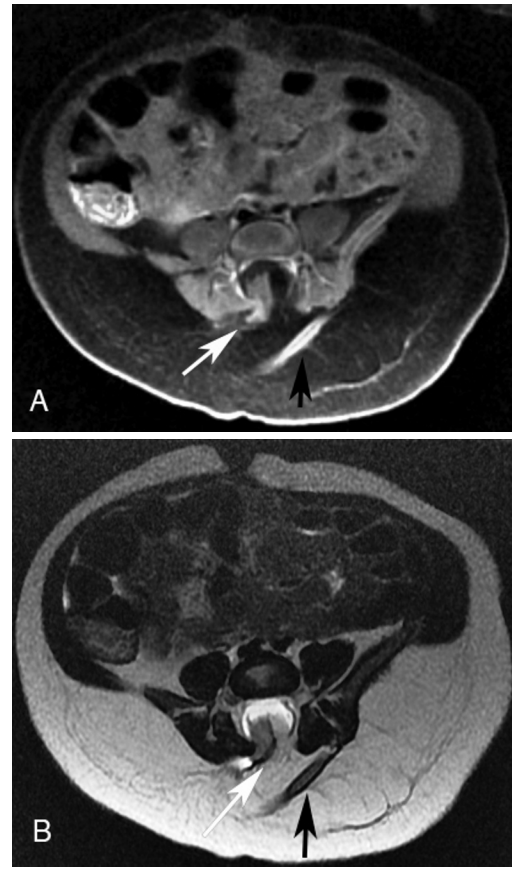


Fig. 2. Axial T1 postcontrast, fat sat demonstrates sacral defect (white arrow) and the sacral accessory limb (black arrow) (A). Axial T2 FRFSE non-fat-suppressed image demonstrates the same (B).



Fig. 3. Sagittal T2 FRFSE image demonstrates a lipomyelomeningocele (arrow), not in communication with an adjacent simple cyst.

neural placode and forms lipomatous tissue (Fig. 4). The spinal cord then becomes tethered to the lipomatous mass. Our patient, with documented intrauterine meningocele, presented at birth with a lipomyelomeningocele at the lumbar-sacral level associated with an osseous appendage. Similar anomalous findings have been reported previously by Lee et al. and Wasnik et al. (4, 5). Wasnik et al. de-

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