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Original Article

Histopathological presence of dermal elements in resected margins of neural structures obtained from initial repair surgery for myelomeningocele

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ABSTRACT

Background: Development of dermoid or epidermoid cysts in myelomeningocele (MMC) sites is generally thought to occur in a delayed fashion due to implantation of dermal elements during initial repair surgery. Another theory is that dermal and dermoid elements may already be present within dysplastic neural structures at birth.

Methods: We experienced histopathological presence of dermal elements in resected tissues at initial repair surgery in four out of 18 cases with MMC who required resection of parts or margins of the neural structures to perform cord untethering. Since one of these cases has already been reported, we describe the clinicopathological findings for the remaining three cases.

Results: In Case1, cryptic dermoid elements were discovered in the terminal filum-like structure (FT-LS) caudal to the open neural placode (NP). The FT-LS had histopathological characteristics similar to the retained medullary cord. In Case 2, dermoid elements were discovered in the caudal margin of the dysplastic conus medullaris. In Case 3, a thin squamous epithelial layer overlapped the rostral margin of the NP where the NP was located near the skin. Case 1 developed an epidermoid cyst at 1 year and 2 months of age, which was totally resected.

Conclusion: Prenatally existing cryptic dermoid elements in the caudal portion of neural structures and remnants of dermal elements overlapping the rostral margin of the NP are associated with delayed occurrence of dermoid/epidermoid cysts. Postoperative histopathological investigation of the resected specimens is recommended. Once dermal elements are revealed, repeated imaging examination and additional surgery should be considered.

Keywords: Dermoid/epidermoid cyst, Inclusion tumor, Keratin, Retained medullar cord, Squamous epithelium

INTRODUCTION

Dermoid or epidermoid cysts and related spinal cord tethering are one of the serious complications that may develop in patients who have undergone previous repair surgery for myelomeningocele (MMC) during the neonatal period.[21,24] The origin of these tumors is generally explained by incomplete resection and implantation of dermal elements surrounding

a dysplastic neural structure such as open neural placode (NP) at the initial repair surgery, which consequently become inclusion tumor.[5] There have been a few reports on dermoid elements already present within a dysplastic neural structure in the MMC site. These dermoid elements were incidentally found at the initial repair surgery, in the terminal filum-like structures (TF-LS) distal to the NP,[2] at the dorsolateral surface of the NP,[19] and at the dorsal surface of the conus.[11] Storrs concluded that dermal hamartomas are frequent findings from pathological examination of excised NP in patients with MMC.[23] We previously reported a neonate with MMC in whom a dermoid cyst was revealed in the dysplastic conus medullaris (CM) in MMC sac at the initial surgery, which had possibly ruptured during the prenatal period.[10] In the present report, we describe the histopathological presence of dermal elements in the resected tissues which made up parts or margins of the neural structures at the time of initial repair surgery in three additional cases with MMC. We also discuss the surgical strategy for repair of MMC based on these findings to prevent dermoid/epidermoid cysts occurring later.

MATERIALS AND METHODS

From January 2018 to September 2022, 24 patients with MMC underwent initial repair surgery at Fukuoka Children's Hospital. At surgery, an open (dysplastic) NP, that is myeloschisis, or a dysplastic CM was resected at the intermediate zone which consists of pia-arachnoid membrane between the neural structure and the surrounding skin[9,20] under a microscope. The resection line was placed with a space between the skins to avoid remnants of dermal elements, paying attention to minimize the damage of neural structures. After untethering the cord, open NP was approximated with pia-arachnoidal sutures using unabsorbable monofilament. Subsequently, multiple-layer closure, including the dura mater and muscle layer, subcutaneous tissue, and skin, with skin flap when required, was performed as described previously.[12,16] For intraoperative neurophysiological monitoring (IONM), we recorded evoked compound muscle action potentials from the external anal sphincter, hamstrings, and gastrocnemius muscles by direct electrical stimulation of the NP, CM, and nerve roots as described previously.[12] Pre- and postnatal magnetic resonance imaging (MRI), including 3D-heavily T2-weighted image (3D-hT2WI), was performed as described in the previous reports.[7,8]

Of the 24 MMC patients, 18 patients required resection of parts or margins of the neural structures to perform complete untethering of the cord. The resected tissues were placed in formalin. Routine histopathological sections were performed with hematoxylin and eosin (H&E) staining and immunostaining such as glial fibrillary acidic protein (GFAP) for neuroglial tissues and CK 5/6 and CK14 for cytokeratin as part of standard diagnostic analysis.

RESULTS

In the resected specimens of 4 (22%) out of 18 patients, histopathological examination revealed dermal or dermoid elements. Since we have already reported one of the four cases in detail,[10] the clinical and histopathological findings for the remaining three cases have been described below.

CASE REPORTS

Case 1

Case 1 was a female infant born prematurely at 26 weeks and 2 days and weighing 784 g. She exhibited an open NP in the open spinal canal at the lumbosacral level. She had slight impairment in plantar flexion and in anal function. Repair surgery for MMC 1 day after birth revealed a TF-LS caudal to the NP [Figure 1a]. Nerve roots emanated from the ventral aspect of the NP, but not from the TF-LS. The border between the NP and the TF-LS was macroscopically apparent, where the width changed steeply, although no confirmation by IONM was performed. The NP was separated from the surrounding skin. The TF-LS was resected from the NP to untether the cord [Figures 1a and b]. No dermoid elements were macroscopically observed. The open NP was approximated with pia-arachnoidal sutures using unabsorbable monofilament to perform myeloplasty. Postoperatively, no *de novo* neurological abnormalities were observed. An Ommaya's cerebrospinal fluid (CSF) reservoir was placed on the 23rd day after birth due to ventricular dilatation, which ceased after intermittent drainage of CSF.

Histopathologically, the severed TF-LS consisted of GFAP immunopositive neuroglial tissues with ependymal canal abutting on a fibrocollagenous tissue [Figures 1c-e]. CK 5/6 and CK14 immunopositive squamous epithelium and keratinizing cells were focally embedded inside the fibrocollagenous tissue [Figures 1d-f]. These findings indicated the presence of cryptic dermoid elements in the TF-LS.

We repeated follow-up MRI, and 3D-hT2WI at 2 months after surgery confirmed successful untethering [Figure 2a]. However, at 1 year and 2-months-old, a small hyperintense lesion was observed in the caudal end of the reconstituted spinal cord [Figure 2b], which increased in size and showed strong high signal intensity on the diffusion-weighted image at 2 years and 6 months of age, indicating the development of a dermoid or epidermoid cyst [Figure 2c]. Since her mild neurological symptoms remained unchanged, a second surgery was performed at 3 years of age.

Laminoplastic laminotomy of L3-4 revealed a pearly tumor in the CM with partial exophytic extension. Opening of the cyst

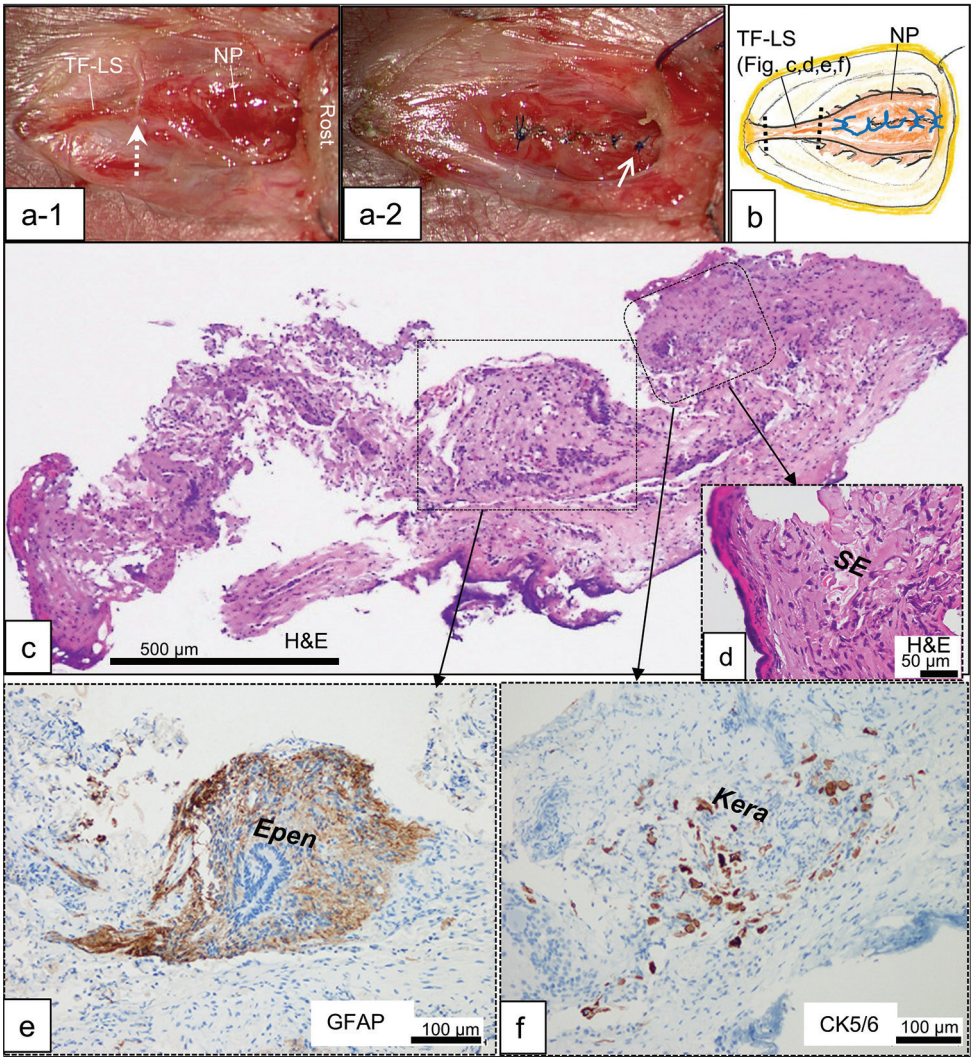

Figure 1: Case 1: First surgery (a-1) intraoperative photograph of the open neural placode (NP) and terminal filum-like structure (TF-LS) in the open spinal canal. The TF-LS is severed at the border to the neural placode (dotted arrow). Rost: rostral side. (a-2) Reconstituted neural placode with pia-arachnoidal suture using unabsorbable monofilament. Note the location of the most rostral knot (arrow). (b) Schematic drawing of the surgery showing the relationship between the severed lines of TF-LS (dotted lines) and the knots (blue lines). Histopathological findings of the transverse sections of the TF-LS stained with hematoxylin and eosin (H&E; c and d), and immunostained with glial fibrillary acidic protein (GFAP; e) and cytokeratin marker CK5/6 (f). Higher magnification views of the area are indicated by dashed squares in (c). The specimen composed of GFAP immunopositive neuroglial tissues with ependymal canal-like structure (Epen: e) abutting on a fibrocollagenous tissue. Squamous epithelium (SE; d), and CK5/6 immunopositive keratinizing cells associated with keratin debris (Kera; f) are focally embedded inside of the fibrocollagenous tissue.

revealed the tumor capsule and keratin substance existing around and rostral to the most rostral knot of unabsorbable monofilament used in the first surgery, but not in the vicinity of the caudal knots, where the morphological border to the filum terminale had been at the first surgery [Figure 2d]. The capsule was totally resected. Postoperatively, her neurological status remained unchanged.

Histopathologically, the capsule consisted of stratified squamous epithelium associated with keratin debris, but without hair shafts or skin appendage, abutting on a gliotic central nervous tissue, and the diagnosis of epidermoid cyst were made [Figures 2e and f]. These findings indicated that the inclusion tumor did not develop from the previous caudal border of the NP to the TF-LS [Figure 2g].

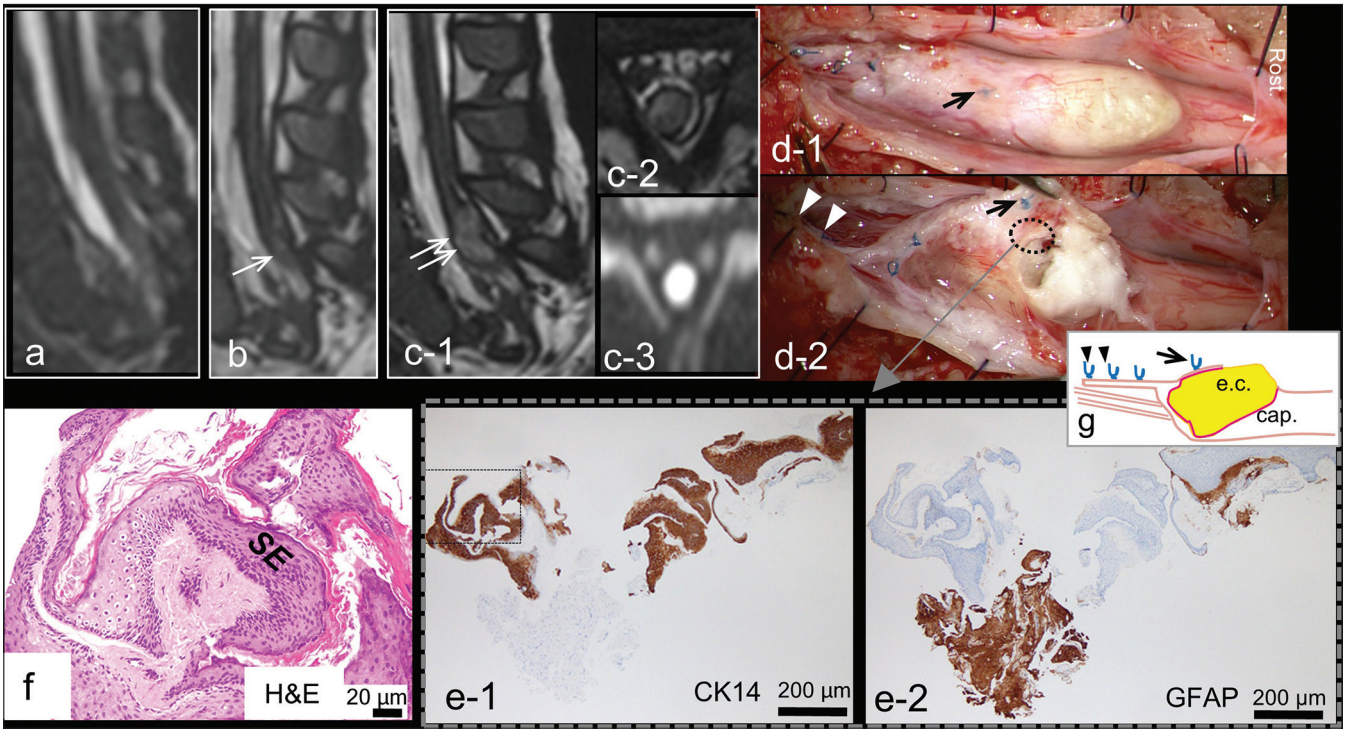

Figure 2: Case 1: Second surgery (a-c) follow-up 3D-heavily T2-weighted magnet resonance imaging in chronological order. (a) A sagittal image at 2 months after the first surgery showing reconstituted spinal cord in the dural cul-de-sac. (b) An image at 1 year and 2 months of age demonstrating small hyperintense lesion (arrow) in the caudal end of the spinal cord. Sagittal (c-1) and axial (c-2) images at 2 years and 6 months of age revealing mass lesion (double arrow; c-1) with strong hyperintensity on diffusion-weighted image (c-3), indicating a dermoid or epidermoid cyst. (d-1) Intraoperative photograph showing a pearly tumor in the conus medullaris (CM) with partial exophytic extension, located rostrally to the most rostral knot (arrow) used in the first surgery. Rost: rostral side. (d-2) Opening of the cyst revealing the tumor capsule and keratin substance located around and rostral to the most rostral knot (arrow), but not in the vicinity of the caudal knots (arrowheads). Histopathological findings of the resected capsule indicated by dotted ellipse in (d-2) immunostained with CK14 (e-1) and glial fibrillary acidic protein (GFAP) (e-2). (f) Higher magnification view of the area indicated by dashed square in (e-1) stained with H&E. Stratified squamous epithelium (SE; f) which is immunopositive for CK14 (e-1), associated with keratin debris but without hair shafts or skin appendage, are abutted on a GFAP immunopositive gliotic central nervous tissue (e-2). (g) Schematic drawing of the CM from a lateral view indicating the epidermoid cyst (e.c.) with capsule (cap.) beneath the most rostral knot (arrow) and the absence of epidermoid cyst in the vicinity of the caudal knots (arrow heads).

Case 2

Case 2 was a girl who had a cord-like structure in the open spinal canal, which appeared as dysplastic CM [Figures 3a and b]. She had severe motor dysfunction below the L5 level on both sides and anal dysfunction. At repair surgery 1 day after birth, the dysplastic CM was resected at the rostral and lateral intermediate zones [Figures 3c and d]. The caudal resection line of the CM was placed at its most rostral non-functional portion confirmed by IONM. The nerve roots in the open spinal canal were covered with yellowish viscid tissues. Postoperatively, her neurological status remained unchanged.

Histopathologically, the resected caudal margin of the CM composed of fibrocollagenous tissues focally embedded with arachnoid cells and CK5/6 and CK14 immunopositive squamous epithelial cells [Figures 3e and f]. Viscid yellowish tissues between the nerve roots contained a keratinous material with CK5/6 and CK14 immunopositive squamous epithelial cells. No hair shafts were present. These findings suggested that the resected caudal portion of the dysplastic CM harbored cryptic dermoid elements.

She underwent a ventricle-peritoneal shunt at 6 months of age due to hydrocephalus. Follow-up MRI performed at 1 year and 6 months of age demonstrated absence of the dermoid cysts at the lumbosacral region.

Case 3

Case 3 was a male infant who exhibited a NP in the open spinal canal [Figures 4a and b]. He had motor dysfunction below the L5 level on both sides, and mild anal dysfunction. At repair surgery, the NP was circumferentially resected

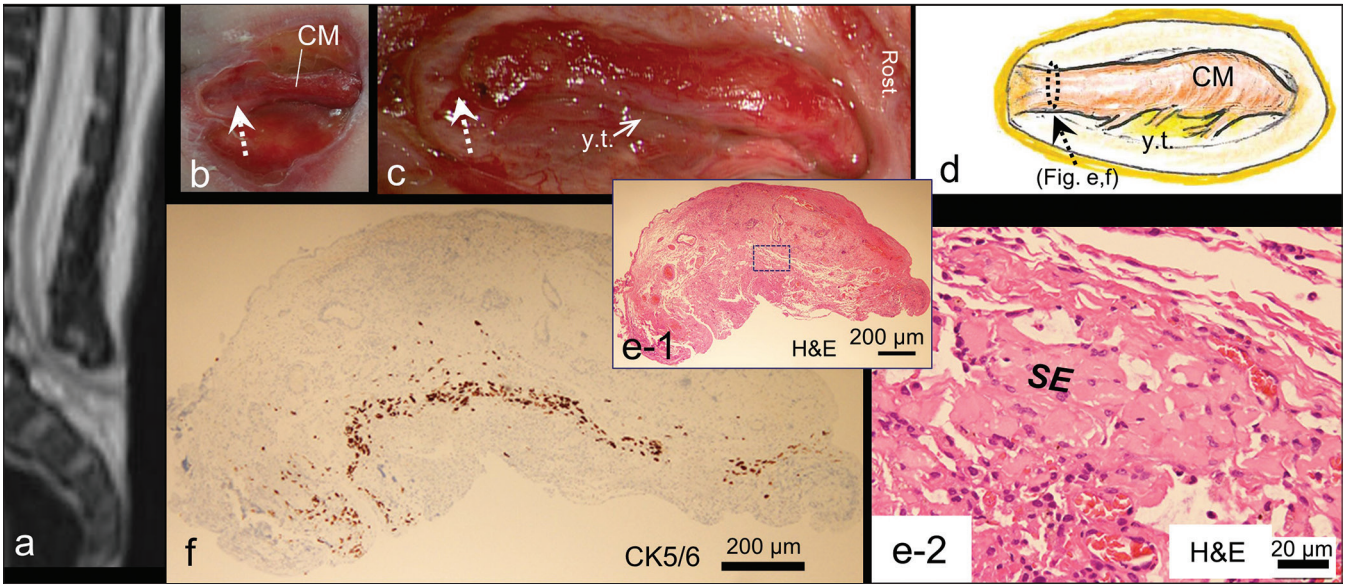

Figure 3: Case 2 (a) 3D-heavily T2-weighted image at birth indicating a neural structure extending dorsally from the spinal cord. Intraoperative photograph (b and c) and schematic drawing (d) of the dysplastic conus medullaris (CM) in the open spinal canal. Rost: rostral side. The caudal resection line of the CM (dotted arrow in (b-d) is located at its most rostral non-functional portion, where the border to the skin is blurred. After untethering of the CM (c), yellowish viscid tissues (y.t. in [c and d]) are observed between the nerve roots. Histopathological findings of the resected caudal margin of the CM stained with H&E (e-1 and 2) and immunostained with CK5/6 (f), of which location is indicated by dashed ellipse in (d). Higher magnification view of the area is indicated by dashed square in (e-1). Squamous epithelium cells (SE: e-2), which are CK5/6 immunopositive (f), are focally embedded in the fibrocollagenous tissues.

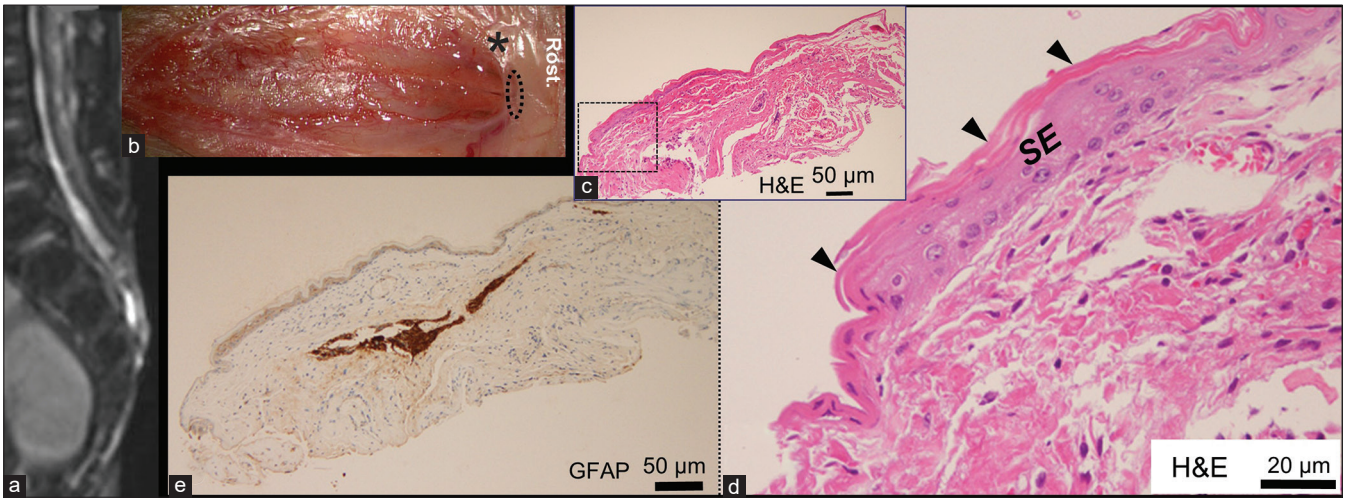

Figure 4: Case 3 (a) 3D-heavily T2-weighted image at birth indicating a neural placode at the sacral level. (b) Intraoperative photograph of the open neural placode. Rost: rostral side. The rostral portion of the neural placode is located in close proximity to the dysplastic skin layer (*). Histopathological findings of the resected rostral margin of the neural placode, of which location is indicated by dotted ellipse in (b), stained with H&E (c and d) and immunostained with glial fibrillary acidic protein (GFAP) (e). Higher magnification view (d) of the area is indicated by dashed square in (c). Stratified squamous epithelium (SE; d) overlapped by keratinizing layer (arrowheads; d) partly lines fibrocollagenous tissue embedding a small GFAP immunopositive neuroglial tissue (e).

from the skin. At the rostral portion, the NP was in close proximity to the dysplastic skin layer, and therefore, the resection line had to be placed on the margin of the NP [Figure 4b]. Postoperatively, his neurological status remained unchanged.

Histopathologically, the resected tissue of rostral margin of the NP consisted of a fibrocollagenous tissue partly lined by keratinizing stratified squamous epithelium, embedding a small neuroglial tissue immunopositive for GFAP [Figures 4c-e]. These findings suggested that the resected

specimen was a rostral portion of the NP overlapped by a thin squamous epithelial layer continuous from the skin layer.

Follow-up MRI performed at 1 year and 6 months of age showed the absence of dermoid cysts.

DISCUSSION

In Cases 1 and 2, dermoid elements were embedded in the TF-LS caudal to the NP and in the caudal margin of the dysplastic CM, respectively, suggesting prenatally existing cryptic dermoid elements. The origin of the dermoid element located inside these structures might be prenatal migration of dermal tissue or cutaneous ectoderm from the caudal margin of the structures, where skin edges were attached.[2,13] As far as Case 1 goes, another theory may be inferred from the histopathological finding that the ependymal canal surrounded by the neuroglial tissues was observed in the TF-LS. This feature is reminiscent of retained medullary cord (RMC), that is, a closed spinal dysraphism thought to be caused by regression failure during late secondary neurulation which consists of a non-functional medullary cord with a central canal-like ependyma-lined lumen in the GFAP immunopositive neuroglial tissues.[15,17] Cases of RMC associated with congenital dermal sinus, which consists of a tract lined by stratified squamous epithelium, have been reported.[13,14] The pleuripotential caudal cell mass in the medullary cord, which disappears during secondary neurulation, but persists and differentiates,[13,17] might have relevance to the formation of the dermal elements in the TF-LS of Case 1.

Rostral margins of the NP might be where dermal elements tend to be left behind at the initial surgery, as shown in Case 3. Because the dermal layer can grow over a pia-arachnoid membrane of the intermediate zone and sometimes a NP,[5,6] when the NP is in close proximity to the skin margin, it might be practically difficult to separate it totally from the skin. A way to avoid remnants of dermal elements is by increasing the degree of resection of the NP, which should be determined by considering pre-existing neurological state of each patient, because NPs of MMC reportedly possess neural capacity.[18,22]

An epidermoid cyst occurred in delayed fashion in Case 1, in which the capsule did not exist in the vicinity of the previous border to the TF-LS presumed from the location of the knots used at the first surgery, but were located more rostrally. This finding suggests that the epidermoid cyst might not have developed from the dermoid element that was continuous with that in the TF-LS. In that case, one possible origin is a remnant of dermal element on the rostral margin of the NP at the first surgery, as shown in Case 3. A second possible origin is another congenital dermoid element in the NP that had existed independently of the one in the TF-LS, because dermoid elements are speculated to exist anywhere in MMC site at birth due to common embryological origin for MMC and the dermoid elements during neural tube formation.[2,11] Another theory is that the positional relationship between the reconstituted NP and the knots might have changed after the first surgery; we speculate one possibility that the caudal part of the NP had atrophied due to an effect of electrocoagulation used for the resection of the TF-LS at the initial surgery, and the arachnoid membrane including the knots dragged down caudally. In this case, the epidermoid cyst could have developed from the dermoid element continuous with that in the TF-LS.

The present findings suggest that dermoid/epidermoid cysts occurring later could be derived from both prenatally existing cryptic dermoid elements in neural structures and remnants of dermal elements overlapping the margins of NP. Total removal of dermal elements on or in functional neural structures might be sometimes difficult. It has been reported that the delayed occurrence of dermoid cyst was observed in patients who underwent prenatal MMC repair surgery with no significant difference or a slightly increased rate compared to those underwent postnatal repair.[1,3] The technical challenge of removing dermal elements during intrauterine surgery is assumed to be related to the incidence of dermoid cysts occurring later.[4] This might suggest that meticulous removal of tiny dermoid element is still required at the initial repair surgery for MMC to reduce the risk of dermoid cyst occurrence.

The incidence of dermoid/epidermoid cysts after repair surgery for MMC has been reported to be 9–16%.[5,21] In the present study, histopathological presence of dermal element at the initial repair surgery is found in 22% of patients with MMC, and delayed occurrence of epidermoid cyst was observed in one case among three. It is impossible to discuss the incidence of dermoid/epidermoid cysts from our experience, because only a small part of the tissues was investigated. Duration between initial repair surgery for MMC and radiological diagnosis of dermoid cysts has been reported to vary from 14 to 196 months.[5] Hence, as to Case 2 and 3, although no delayed occurrence of dermoid/epidermoid cysts was observed during the follow-up period of 1 year and 6 months, longer follow-up is necessary. Although postoperative histopathological observation of the resected specimens might be recommended, it is sometimes difficult to make a correct histopathological diagnosis of dermoid elements with routine H&E staining alone, and it is thought to be important to combine immunohistochemical staining for cytokeratin such as CK5/6 and CK14, as shown in Case 1 and 2.

CONCLUSION

Prenatally existing cryptic dermoid elements in the caudal portion of neural structures and remnants of dermal elements overlapping the rostral margin of the NP are associated with delayed occurrence of dermoid/epidermoid cysts. Postoperative histopathological investigation of the resected specimens is recommended. Once dermal elements are revealed, repeated MRI examination including 3D hT2WI and additional surgery, when required, should be considered.

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Ethics statement

The authors confirm that written informed consent was obtained from the families of the infants described in this report.

The authors declare that this work complies with the guidelines for human studies, and the research was conducted ethically in accordance with the World Medical Association Declaration of Helsinki.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent.

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Conflicts of interest

There are no conflicts of interest.

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