STUDY

Rudimentary Meningocele: Remnant of a Neural Tube Defect?

Laila El Shabrawi-Caelen, MD; Wain L. White, MD; Hans Peter Soyer, MD; Bang-Soon Kim, MD; Ilona J. Frieden, MD; Timothy H. McCalmont, MD

Background: Rudimentary meningocele, a malformation in which meningothelial elements are present in the skin and subcutaneous tissue, has been described in the past under a variety of different terms and has also been referred to as cutaneous meningioma. There has been debate as to whether rudimentary meningocele is an atretic form of meningocele or results from growth of meningeal cells displaced along cutaneous nerves

Objective: We reviewed the clinical, histological, and immunohistochemical characteristics of rudimentary meningocele in an attempt to assess the most likely pathologic mechanism for it.

Design: Retrospective study.

Setting: University hospitals.

Patients: Thirteen children with rudimentary meningocele.

Main Outcome Measures: Medical records were reviewed and histopathologic examination as well as immunohistochemistry studies were performed for each case. A panel of immunoperoxidase reagents (EMA, CD31, CD34, CD57, S-100, and CAM 5.2) was used to assess lineage and to confirm the meningothelial nature of these lesions.

Results: Recent evidence indicating a multisite closure of the neural tube in humans suggests that classic meningocele and rudimentary meningocele are on a continuous spectrum.

Conclusion: Rudimentary meningocele seems to be a remnant of a neural tube defect in which abnormal attachment of the developing neural tube to skin (comparable to that in classic meningocele) could explain the presence of ectopic meningeal tissue. In the majority of cases, no underlying bony defect or communication to the meninges could be detected. However, in light of the probable pathogenesis, imaging studies to exclude any communication to the central nervous system should precede any invasive evaluation or intervention.

Arch Dermatol. 2001;137:45-50

From the Department of Dermatology, University Graz, Graz, Austria (Drs El Shabrawi-Caelen and Soyer); the Department of Pathology, North Carolina Baptist Hospital–Bowman Gray School of Medicine, Wake Forest University, Winston-Salem, NC (Dr White); SNU Clinic of Dermatology Seoul, South Korea (Dr Kim); and the Departments of Dermatology (Drs Frieden and McCalmont) and Pathology (Dr McCalmont), University of California, San Francisco.

RUDIMENTARY meningocele is an uncommon developmental anomaly in which meningothelial elements are displaced into the skin and subcutaneous tissue. In the past it has been described under a variety of different terms, such as cutaneous meningioma, hamartoma (of the scalp) with ectopic meningothelial elements, sequestrated meningocele, acoelic meningeal hamartoma, and cutaneous heterotopic meningeal nodules. [1-8] Controversy exists as to whether this entity represents a form of meningocele in which the underlying connection to the meningeal space is obliterated, or whether lesions develop because of proliferation of meningeal cells along the routes of cutaneous nerves.[9]

There is recent evidence for a multisite closure of the neural tube in humans similar to that observed in experimental animals.[10,11] Multiple distinct closure sites of the neural tube explain the most frequent locations of neural tube defects. Failure to achieve complete fusion results in various forms of dysraphia.

We present 13 cases of rudimentary meningocele that show a strikingly similar anatomical distribution to the neural tube fusion sites observed experimentally. The multisite closure model explains the congenital nature and distribution of these lesions and also coincides with the distribution of classic meningoceles.

RESULTS

HISTOPATHOLOGIC FINDINGS

The microscopic findings are summarized in **Table 2**. In general, skin append-

PATIENTS AND METHODS

PATIENTS

The clinical data are summarized in **Table 1**. All 13 patients were children from 0 to 6 years of age (median, 19 months). Eight were boys and 5 were girls. All lesions, variably described as patches, papules, nodules, or exophytic masses, were congenital and ranged in size from 0.5 to 8 cm (**Figure 1**A). Some lesions had increased in size with the growth of the child. All were midline lesions and all but 2 occurred on the scalp, preferentially involving the occiput and vertex. The remaining 2 lesions occurred overlying the cervical spine (C2-C3) and lumbosacral spine (Figure 1B). The "hair collar" sign, a ring of coarse hair surrounding the malformation and thought to be characteristic of cranial dysraphism, was found in 3 infants. Eleven patients had solitary defects. In each of the remaining 2 cases, 2 lesions were apparent. Other clinical abnormalities included port wine stains at a separate location in 2 patients and a congenital melanocytic nevus in patient 1. The clinical diagnoses included aplasia cutis, epidermal inclusion cyst, congenital melanocytic nevus, hemangioma, skin tag, dermal sinus tract, meningocele, and encephalocele. Small bony defects were identifiable in 2 patients and a connecting fibrous tract to the dura was identifiable in one based on radiologic imaging results or direct surgical visualization.

The 2 patients with lesions along the spine showed more complex underlying abnormalities. Both had bifid laminae and tethered spinal cords. In one of these patients, there was associated diastematomyelia and syringomyelia. The patient also had attachment of the penis to the scrotum (penis palma) with vesicoureteral reflux. In the second patient, a fibrovascular bundle pierced the dura and extended into the subdural space (Figure 1C).

All defects were locally excised. No recurrence was observed, and there was no evidence of postsurgical meningitis or cerebrospinal fluid leakage after a median follow-up of 28 months.

METHODS

Formalin-fixed and paraffin-embedded tissue from 13 skin lesions was examined in conventional and immunoperoxidase sections via a light microscope. Immunoperoxidase stains were prepared with an avidin-biotin method using monoclonal antibodies directed against epithelial membrane antigen (EMA) (Dako, Capenteria, Calif), CD31 (Biogenex, San Ramon, Calif), CD34 (Novocastra, Newcastle-upon-Tyne, England), CD57 (Becton-Dickinson, San Jose, Calif), and CAM 5.2 (Becton-Dickinson), as well as polyclonal antibodies directed against S-100 protein (Dako). Two cases were evaluated further with anti–glial fibrillary acidic protein (GFAP) (Boehringer-Mannheim; Indianapolis, Ind) and vimentin (Monosan; Uden, the Netherlands).

ages, vessels, nerves, and melanocytes were quantified, and remnants of brain tissue were sought. Psammoma and collagen bodies and calcification were also sought.

The overall architecture varied in that 8 lesions showed replacement of both the dermis and subcutis by the malformation, while in 4 lesions only 1 of the compartments was involved. In case 5 the biopsy was too superficial to exclude deep involvement. In some specimens a loose network of pseudovascular spaces in the superficial dermis (**Figure 2**A) contrasted with dense collagenous tissue containing clusters of meningocytes in the deeper parts. Cystic structures lined by meningothelial cells were present in 5 cases (Figure 2B). The meningocytes, recognizable by their ovoid to spindle shape with scant eosinophilic cytoplasm and small nuclei with finely stippled chromatin and indistinct cell borders, formed cords, strands, and small nests and had the tendency to encompass collagen fibers and adnexal structures. We observed decreases as well as increases in eccrine glands and hair follicles. There were also distorted follicles and an increase in apocrine glands and smooth muscle bundles. In one case, a follicular cyst was evident. Prominent vessels were noted in 8 cases. Necrotic glial tissue was absent. Calcification was noted either in the form of psammoma bodies (4 patients) or in an unusual reticulate pattern (5 patients) (Figure 2C).

IMMUNOHISTOCHEMISTRY

EMA immunostaining highlighted the meningothelial cells (**Figure 3**) but revealed a heterogeneous pattern. Two cases failed to express EMA in the superficial aspect of the lesion but showed strong expression in the deep portion. Similarly, in specimen 5, which was transected at the level of the midreticular dermis, the loose network of pseudovascular spaces did not express EMA, but vimentin nicely outlined these spaces. CD31 and CD34, known to be vascular markers, did not react with these anastomosing channels, confirming their nonvascular nature.

Antibodies directed against S-100 protein, CD57, and CAM 5.2 failed to label meningothelial cells. S-100 was used to quantify melanocytes, which were increased at the dermo-epidermal junction in 1 specimen and within the rudimentary meningocele in 2 patients. Adjacent nerves were also identified via S-100 immunostaining in 4 patients. Anti-GFAP detected small nests of apparent glial tissue in patient 7 and also stained a few cells in patient 12. However, results of analysis for neurons with neuron-specific enolase and for axons with neurofilament in these 2 cases were negative, unlike what has been reported in most cases of heterotopic glial tissue. Meningothelial elements like those observed in both cases are not a feature of heterotopic glial tissue, but rather favor a diagnosis of rudimentary meningocele.

COMMENT

The pathogenesis of human neural tube defects is complex and poorly understood. Minor deviations not serious enough to significantly arrest or distort the formation of the central nervous system undoubtedly occur. Rudimentary meningocele may be this type of minor deviation and poses a problem to dermatologists in terms of diagnosis and management.

Table 1. Clinical Findings in 13 Patients With Rudimentary Meningocele*†

Patient	Age at Diagnosis/ Sex	Size, cm/ Site of Meningocele	Clinical Diagnosis/ Findings	Other Anomalies	Imaging Studies	Operative Findings	Follow-up Period
1	18 mo/F	0.8×0.5/ Midline occipital	Nevus/Nodule since birth, swelling for 6 mo	Congenital melanocytic nevus—right thigh	MRI: intact skull	No bone defect	No recurrence at 48 mo
2	8 mo/M	1.5×1.5/ Posterior scalp	Congenital nevus vs aplasia cutis/Patch since birth, hair collar sign	None	Not done	Feeding vessel to periostium; no bone defect	No recurrence at 7 mo
3	72 mo/M	0.6/Posterior scalp	Cyst vs neural tumor/Subcutaneous nodule enlarging since birth	None	Not done	No bone defect	No recurrence at 48 mo
4	42 mo/M	3/Posterior scalp	Epidermal inclusion cyst/Firm tumor since birth, rapid growth by 6 y	Not available	Not available	Not available	Not available
5	3 mo/F	0.8×0.8/ Vertex capitis	Aplasia cutis/Hairless patch present since birth	None	Skull radiograph: normal	Not available	No recurrence
6	5 mo/M	0.9×0.8/Scalp	Congenital amelanotic nevus/Irregularly pigmented papule, growth since birth	Port wine stain—left arm	Not done	No bone defect	No recurrence at 9 mo
7	14 mo/M	1.5×1.5/ Cervical C2-C3	Skin tag, dermal sinus tract/Exophytic mass present since birth	None	MRI: cervical dermal sinus tract entering dura, tethering spinal cord	Bifid laminae; fibrovascular bundle piercing dura and extending to subdura	No recurrence at 48 mo
8	78 mo/F	1.2/Scalp	Congenital scalp nodule/Nodule since birth, slowly increasing in size	None	Not done	No bone defect	No recurrence at 36 mo
9	15 mo/F	1.4×0.9, 1×0.5/ Midline scalp (2)	Scalp masses, hemangiomas/ Midline scalp masses since birth	Port wine stain—nose	CT scan: no bone defect	No bone defect	No recurrence at 17 mo
10	4 mo/M	4×2/Occipital scalp	Occipital encephaloceles/ Tumor since birth, hair collar sign	None	MRI: postoperative changes of subcutis and calvarium; brain normal	0.5-cm bone defect	No recurrence at 12 mo
11	3 wk/M	8×2.5/ Lumbosacral	Meningocele, diastematomyelocele/ Epithelialized mass on midlumbar region, congenital	Spina bifida; penis palma with I° vesicoureteral reflux	MRI: tethered cord, diastematomyelia, and syringomyelia	Bifid laminae with central bony spike; spinal cord split around bony spike	No recurrence at 6 mo
12	Newborn/M	1.2×1.1/ Occipital scalp	Dermal sinus tract vs encephalocele/Tender nodule present since birth	None	CT scan: small soft tissue lesion with small hole in skull, dolichocephalic	Small bone defect with soft tissue tract to dura	No recurrence at 72 mo
13	Newborn/F	2×0.8, 0.5×0.5/ Vertex capitis	Aplasia cutis/ 2 Congenital bullous lesions, hair collar sign	None	CT scan: no underlying skull defect	No bone defect	No recurrence at 5 mo

*MRI indicates magnetic resonance image; CT, computed tomographic scan.
†For all of the patients the treatment was local excision.

The 13 cases of rudimentary meningocele presented herein were congenital, situated either over the scalp or midline spine. The clinical appearance was highly variable, leading to difficulty in clinical diagnosis; clinical diagnoses such as melanocytic nevus, epidermal cyst, dermal sinus tract, hemangioma, meningocele, encephalocele, and aplasia cutis were offered. Since the correct clinical diagnosis of cranial/spinal dysraphism was suspected in

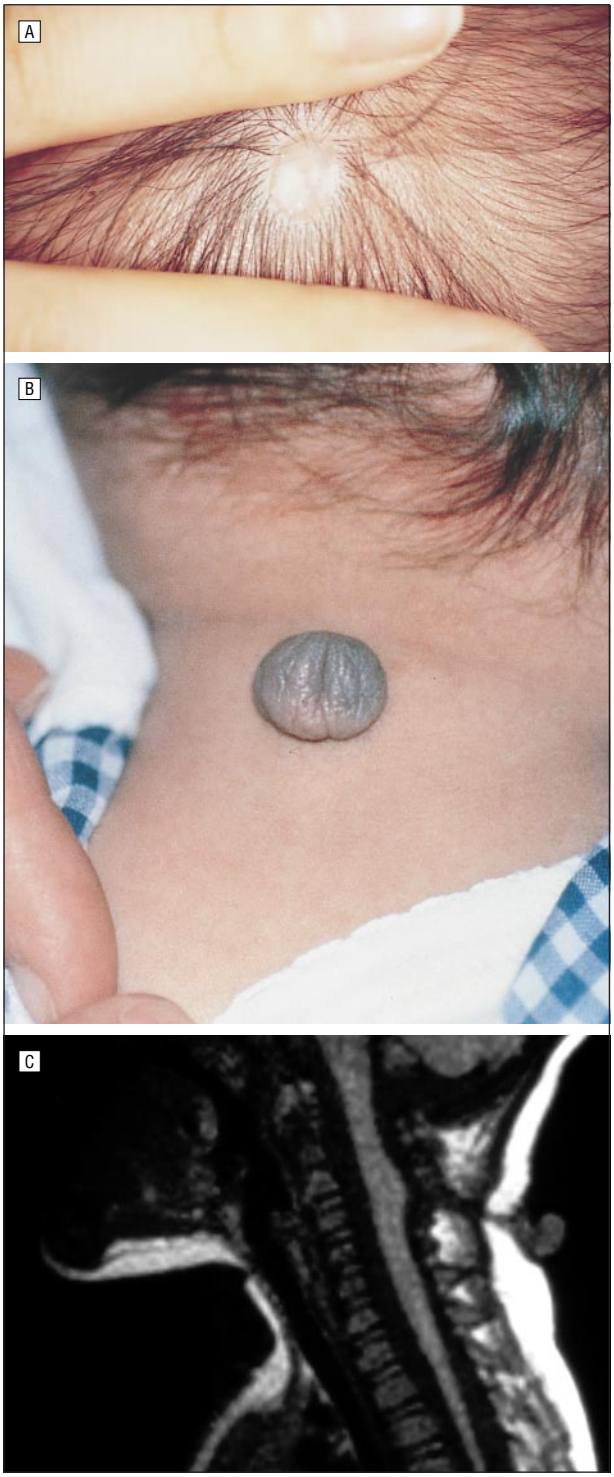

Figure 1. *A, A flesh-colored papule on the vertex capitis. B, Fourteen-month-old boy with a polypoid nodule along the spine (C2-C3). C, On magnetic resonance imaging, a connecting fibrous tract enters the dura and extends into the subdural space.*

only 3 of the 13 patients, there appears to be relative unfamiliarity with these lesions. One of the most useful clinical features of cranial dysraphism, the so called "hair collar" sign, was observed in 3 cases.[10,12-14] Described by Commens et al,[13] this finding consists of a collarette of hair, which encircles the malformation. This finding suggests an underlying meningocele, but the pattern is not specific, since the same clinical pattern occurs in association with an underlying encephalocele, heterotopia of brain tissue, or membranous aplasia cutis.[15] Membranous aplasia cutis refers to congenital, sharply marginated ovoid scalp defects. In contrast to rudimentary meningocele, this condition often presents as multiple lesions with a tendency to regress from an initial cystic or bullous appearance to a scar. Despite these differences, there seems be an overlap with rudimentary meningocele in terms of the clinical picture. One of our patients presented with 2 bullous scalp defects that could not be differentiated from membranous aplasia cutis on clinical grounds. The histopathologic features in our patient, however, were clearly those of rudimentary meningocele based on the presence of meningeal tissue, which by definition is not found in membranous aplasia cutis.

In view of the relative rarity of rudimentary meningocele, the histopathologic diagnosis is difficult. The microscopic features are often subtle, with meningeal tissue simulating the appearance of vascular or connective tissue. Meningocytes tend to encircle collagen bundles and are sometimes accompanied by psammoma bodies; these signs should be sought as clues to the microscopic diagnosis. An additional helpful clue was a decrease or increase in adnexal structures, similar to the pattern that can be seen in some forms of epidermal nevi and aplasia cutis. Cystic structures lined by meningothelial cells, as would be seen in a classic meningocele, were occasionally evident, highlighting the similarity between the classic and rudimentary forms of this disorder. Classic meningocele can be separated from rudimentary meningocele only on the basis of clinical data.[16] In general, meningocytes were few in number, were positioned between collagen bundles, and created a microscopic pattern resembling vascular spaces. These pseudovascular spaces can easily be misinterpreted as a vascular neoplasm such as lymphangioma or even angiosarcoma.[5] Apart from the clinical picture, absence of nuclear pleomorphism and mitotic figures, and the lack of reactivity to vascular markers, such as CD31 and CD34, should enable exclusion of a vascular neoplasm.

Another histopathologic finding that could contribute to error in microscopic diagnosis is the presence of syncytial giant cells. These cells result from fusion of meningocytes and may simulate the pattern of giant cell fibroblastoma, a form of dermatofibrosarcoma protuberans usually observed in children.[16,17] Myxoid stroma noted in either case may further obscure the diagnosis.[18-20] Immunoperoxidase staining is vital in distinguishing these disorders, as dermatofibrosarcoma protuberans typically expresses CD34 and fails to express EMA, while rudimentary meningoceles display opposite reactivity.[16] Although EMA expression is vital to the identification of cells of meningothelial lineage, it is important to note that EMA failed to label the network of pseudovascular spaces located in the superficial dermis in 3 of our patients. Only thorough examination revealed EMA-positive meningocytes in the deeper parts of 2 specimens. Therefore, in specimens from superficial biopsies that do not extend below the level of the middermis, as was the case in patient 5, meningothelial elements may not be detectable. With respect to superficial biopsies, some cases de-

Table 2. Histopathologic Findings in 13 Patients With Rudimentary Meningocele*

Case	Meningothelial Elements in Superficial/Deep Reticular Dermis	Whorled Pattern	Psammoma Bodies/ Collagen Bodies	Calcification Pattern	Skin Appendages	Vessels	Melanocytes	Nerves
1	+/–	+	+/+	PS	EG and HF↓	↑	↑DEJ	NL
2	+/+	–	–/+	RC	HF↓	↑	NL	NL
3	+/+	+	+/+	RC	±	NL	NL	NL
4	+/–	+	++/++	PS	Follicular cyst	↑	NL	NL
5	+/. . .	–	–/–	–	NL	NL	NL	NL
6	+/+	–	–/+	–	EG and HF↓	NL	↑Within RM	NL
7	+/+	–	–/+	RC	AG↑	↑	↑Within RM	↑
8	+/+	+	–/+	–	NL	↑	NL	NL
9	+/+	–	–/–	–	HF↑	NL	NL	NL
10	+/+	+	+/+	PS and RC	HF↓	↑	NL	↑
11	–/+	+	–/+	–	EG↑ and HF–	↑	NL	↑
12	–/+	–	–/–	RC	NL	NL	NL	↑
13	+/+	–	–/+	–	HF↓	↑	NL	NL

*– indicates absent; +, present; ++, abundant; PS, psammoma body; EG, eccrine gland; HF, hair follicle; ↓, decreased DEJ; dermoepidermal junction; NL, normal; RC, reticulate calcification; AG, apocrine gland; ↑, increased; ellipses, not applicable; and RM, rudimentary meningocele.

scribed as membranous aplasia cutis with no meningeal or brain tissue detected by immunohistochemistry may actually be examples of rudimentary meningocele.[15] We observed only focal EMA positivity in normal leptomeningeal tissue, as reported by others.[8,20,21] The diffuse meshwork of subarachnoid cells shares many features with the loose network seen in rudimentary meningocele, and this meshwork does not label at all with EMA. An alternative explanation is that the dilution of the EMA antibody that usually works well with epithelial cells might not work as well in cells of meningothelial lineage.

Speculation about the etiology of rudimentary meningocele started with the first observation of this malformation by Winkler in 1904, in which he described a "peculiar disease of the skin and subcutaneous fat."[1] An increasing number of reports of this entity have appeared in the more recent literature, and with them a variety of divergent opinions on the pathogenesis have been suggested. Some authors consider these lesions to be a form of meningocele with an obliterated intracranial communication, while others refer to them as remnants of the neural crest. A more historical theory suggests that they might represent intradermal nevi containing psammoma bodies.[3,7,16,22-26] In addition, these lesions were also postulated to arise from the sheath cells of cutaneous nerves or to reflect continued growth of meningeal cells within a perineural environment.[8,27]

A fairly new theory about closure of the neural tube may yield greater insight into the pathologic mechanism of rudimentary meningocele, as it seems to explain some of the clinical findings.[10,11] Traditionally, neural tube closure was believed to begin in the cervical region and to proceed from there in a continuous bidirectional way[11] (**Figure 4**A). More recent observations in humans and animals, however, indicate multisite initiation of neural tube closure[10] (Figure 4B). The failure of closure to take place partially or completely would thus result in neural tube defects.[11] Mouse models have shown 4 separate sites of neural tube fusion. Homologies of early embryogenesis in mice and humans as well as previous

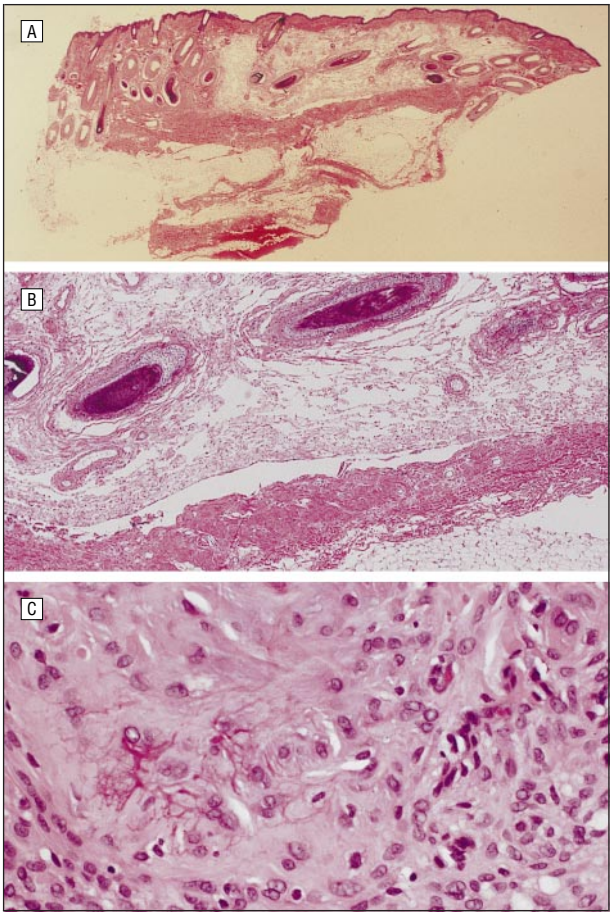

Figure 2. *Scanning magnification shows nearly complete loss of hair follicles, with dense collagenous tissue and prominent vessels in the center of the lesion. A, Low-power magnification shows decreased adnexal structures with distorted hair follicles and a loose network of pseudovascular spaces in the superficial and deep reticular dermis (hematoxylin-eosin, original magnification ×20). B, A network of pseudovascular spaces with a cystic cavity lined by meningothelial cells (hematoxylin-eosin, original magnification ×40). C, Meningocytes recognizable as ovoid to spindle-shaped cells with eosinophilic cytoplasm, small nuclei, and indistinct cell borders. There is a prominent calcification of the reticulate pattern (hematoxylin-eosin, original magnification ×200).*

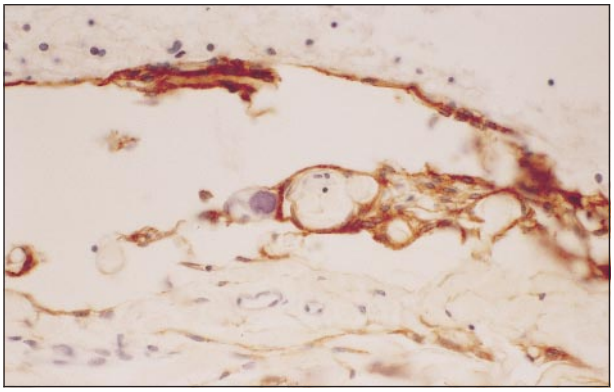

Figure 3. *Meningocytes strongly labeled by immunoperoxidase reagents outline the cystic cavity (EMA immunostaining, original magnification ×200).*

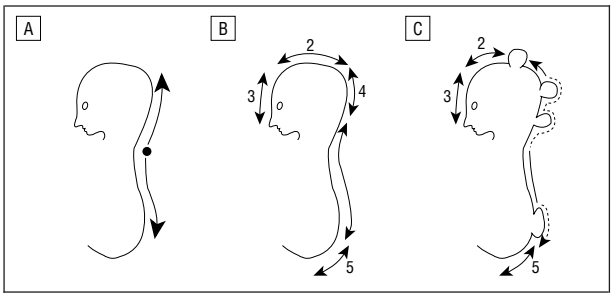

Figure 4. *Potential neural tube closure sites. A, The "zipper" model. B, Multisite closure of the neural tube. C, Common neural tube defects coinciding with the anatomical distribution of rudimentary meningoceles. Reprinted with permission from* Am J Med Genet. *1993;47:723-743.*

illustrations and photographs of human embryos suggest that neural tube fusion occurs at sites similar to those in mice. Of particular interest are the locations of such neural tube defects, as they coincide with the distribution observed in our cases of rudimentary meningocele (Figure 4C). Closure in the occipital area appears to be achieved by a membrane rather than a midline fusion of folds, which explains why such defects may or may not appear in the midline.[10] Another feature of neural tube defects is genetic susceptibility, with a risk of repetitive events in affected families.[10] This suggests that specific genes control individual closure sites.[10] Interestingly, familial occurrence of rudimentary meningocele has been noted in an autosomal dominant inherited pattern and among two siblings.[20,21] Simple displacement of meningocytes along peripheral nerves is an unlikely alternate explanation for such observations.

Another finding that supports classifying rudimentary meningocele as a form of meningocele is the identification of connections, albeit rudimentary ones, that extend from the lesion to dura, and in some instances are associated with minute bony defects.[3,6,23,25] We were able to detect such fibrous tracts in 2 cases and a small osseous defect in 1 infant.

In light of the likely pathogenesis of these lesions, prior to complete surgical excision, we believe that imaging studies should be obtained to exclude the possibility of a communication to the central nervous system. If there is evidence of an underlying connection, the patient should be referred for neurosurgical evaluation. If a communication to the central nervous system can be excluded, simple excision would be appropriate, and neither recurrence nor neurologic consequences would be expected to occur.

The pathologic mechansim of rudimentary meningocele remains enigmatic and has not been completely elucidated. However, our clinical and histopathologic findings and those of other investigators support the conclusion that this entity is a form of dysraphism. We believe this process is a non-neoplastic condition, and thus the designation of cutaneous meningioma should not be used as a synonym for rudimentary meningocele.

Accepted for publication October 11, 2000.

Corresponding author and reprints: Laila El Shabrawi-Caelen, MD, Department of Dermatology, University Graz, Auenbruggerplatz 8, A-8036 Graz, Austria (e-mail: lailaelshabrawi@hotmail.com).

REFERENCES

1. Winkler M. Über Psammome der Haut und des Unterhautgewebes. *Arch Pathol Anat.* 1904;178:322-350.
2. Serwatka LM, Maj MC, Mellette JR. Cutaneous meningioma. *J Dermatol Surg Oncol.* 1984;10:896-900.
3. Lopez DA, Silvers DN, Helwig EB. Cutaneous meningioma: a clinicopathologic study. *Cancer.* 1974;34:728-744.
4. Bain G, Shnitka T. Cutaneous meningioma (psammoma). *Arch Dermatol.* 1956; 74:590-594.
5. Suster S, Rosai J. Hamartoma of the scalp with ectopic meningothelial elements: a distinctive benign soft tissue lesion that may simulate angiosarcoma. *Am J Surg Pathol.* 1990;14:1-11.
6. Bale PM, Hughes L, De Silva M. Sequestrated meningoceles of the scalp: extracranial meningeal heterotopia. *Hum Pathol.* 1990;21:1156-1163.
7. Khallouf R, Fetissof F, Machet MC, Stephanov E, Lechrist J, Lorette G. Sequestrated meningocele of the scalp: diagnostic value of hair anomalies. *Pediatr Dermatol.* 1994;11:315-318.
8. Theaker JM, Fletcher CD, Tudway AJ. Cutaneous heterotopic meningeal nodules. *Histopathology.* 1990; 16:475-479.
9. Penas PF, Jones-Caballero M, Garcia-Diez A. Cutaneous heterotopic meningeal nodules [letter]. *Arch Dermatol.* 1995;131:731.
10. Van Allen MI, Kalousek DK, Chemoff GF, et al. Evidence for multi-site closure of the neural robe in humans. *Am J Med Genet.* 1993;47:723-743.
11. Golden JA, Chernoff GF. Multiple sites of anterior neural robe closure in humans: evidence from anterior neural tube defects (anencephaly). *Pediatrics.* 1995;95:506-510.
12. Drolet BA, Clowry L Jr, McTigue MK, Esterly NB. The hair collar sign: marker for cranial dysraphism. *Pediatrics.* 1995;96:309-313.
13. Commens C, Rogers M, Kan A. Heterotopic brain tissue presenting as bald cysts with a collar of hypertrophic hair: the "hair collar" sign. *Arch Dermatol.* 1989; 125:1253-1256.
14. Stone MS, Walker PS, Kennard CD. Rudimentary meningocele presenting with a scalp hair tuft: report of 2 cases. *Arch Dermatol.* 1994;130:775-777.
15. Drolet B, Prendiville J, Golden J, Enjolras O, Esterly NB. "Membranous aplasia cutis" with hair collars: congenital absence of skin or neuroectodermal defect? *Arch Dermatol.* 1995;131:1427-1431.
16. Marrogi AJ, Swanson PE, Kyriakos M, Wick MR. Rudimentary meningocele of the skin: clinicopathologic features and differential diagnosis. *J Cutan Pathol.* 1991;18:178-188.
17. Fletcher C. Giant cell fibroblastoma of soft tissue: a clinicopathological and immunohistochemical study. *Histopathology.* 1988;13:499-508.
18. Shmookler BM, Enzinger FM, Weiss SW. Giant cell fibroblastoma: a juvenile form of dermatofibrosarcoma protuberans. *Cancer.* 1989;64:2154-2161.
19. Berry AD, Patterson JW. Meningoceles, meningomyeloceles, and encephaloceles: a neuro-dermatopathologic study of 132 cases. *J Cutan Pathol.* 1991;18:164-177.
20. Miyamoto T, Mihara M, Hagari Y, Shimao S. Primary cutaneous meningioma on the scalp: report of two siblings. *J Dermatol.* 1995;22:611-619.
21. Tron V, Bellamy C, Wood W. Familial cutaneous heterotopic meningeal nodules. *J Am Acad Dermatol.* 1993;28:1015-1017.
22. Nochomovitz LE, Jannotta F, Orenstein JM. Meningioma of the scalp: light and electron microscopic observations. *Arch Pathol Lab Med.* 1985;109:92-95.
23. Zaaroor M, Borovich B, Bassan L, Doron Y, Gruszkiewicz J. Primary cutaneous extravertebral meningioma: case report. *J Neurosurg.* 1984;60:1097-1098.
24. Drapkin AJ. Rudimentary cephalocele or neural crest remnant? *Neurosurgery.* 1990;26:667-673.
25. Argenyi ZB. Cutaneous neural heterotopias and related tumors relevant for the dermatopathologist. *Semin Diagn Pathol.* 1996;13:60-71.
26. Lever W. *Histopathology of the Skin.* 6th ed. Philadelphia, Pa: JB Lippincott Co; 1983:677.
27. Penas PF, Jones-Caballero M, Amigo A, Aragues M, Garcia-Diez A. Cutaneous meningioma underlying congenital localized hypertrichosis. *J Am Acad Dermatol.* 1994;30:363-366.