

A Case of Neurilemmoma Associated with Transepidermal Elimination

Gil Ju Yi, M.D., Soo Nam Kim, M.D.

*Department of Dermatology, College of Medicine, Korea University,
Seoul, Korea*

Many cutaneous disorders have been associated with transepidermal elimination (TEE). But there has not been a case with neurilemmoma. We report a 20-year-old man who shows a neurilemmoma on right index finger tip associated with transepidermal elimination.

To our knowledge, this is the first case of the simultaneous occurrence of TEE associated with neurilemmoma. (*Ann Dermatol* 4:(2) 87-90, 1992)

Key Words: Transepidermal elimination, Neurilemmoma, Schwannoma.

Schwannomas (neurilemmomas) are uncommon, benign tumors of nerve sheath (Schwann's cell) origin¹, and these occur almost invariably as solitary tumors along the course of peripheral or cranial nerves. In the skin, Schwannomas are usually solitary subcutaneous, often tender nodules ranging from a few millimeters to a few centimeters in size.

Transepidermal elimination (TEE) is a well-recognized histologic phenomenon. It is characterized by the elimination of exogenously introduced or endogenous matter via the epithelium without gross disturbance of epidermal integrity². TEE as a cathartic mechanism of skin has been reported mostly in conditions displaying some infectious disorders, granulomatous disorders, and rarely benign or malignant tumors³.

This report presents our observation on a patient who had neurilemmoma that displayed TEE.

REPORT OF A CASE

A 20-year-old man had a painful dome shaped, erythematous, translucent papule on the distal, ventral surface of his right thumb. The lesion was an 1 cm translucent papule of 2 month's duration (Fig. 1). He was in good general health and

the review of system was negative. He had no trauma history relevant to his skin lesion nor any systemic disease at the time of his visit. Excisional biopsy specimen from the lesion was fixed in 4% formaldehyde solution, serially sectioned, and stained with hematoxylin-eosin (Fig. 2, 3) and S-100 protein (Fig. 4). H-E stained sections show Antoni type A tissue composed of cells whose nuclei are elongated. The nuclei are arranged in a wavy pattern, revealing a neurilemmoma with a transepidermal canal surrounded by hyperplastic epidermis overlying the tumor.

DISCUSSION

TEE, sometimes termed the perforating dermatoses, has involved (1)inflammatory cells and erythrocytes; exocytosis, black heel, thromboses of capillary hemangioma, and cells in lichen nitidus, (2)products of keratinization; epidermal cysts and porokeratosis of Mibelli, (3)altered connective tissue components; granuloma annulare, necrobiosis lipoidica, rheumatoid nodules, pseudoxanthoma elasticum, chondrodermatitis, cutaneous sclerosis, bone, acne keloidalis, healing wounds, keratoacanthoma, discoid lupus erythematosus, and collagenome perforant verruciforme, (4)extracellular substances; amyloid protein, calcium, uric acid, mucin in papular mucinosis and sialomucin, (4)foreign materials or reactions thereto; calcium nitrate, calcium chlo-

Received November 1, 1991

Accepted for publication November 19, 1991

Reprint request to: Gil Ju Yi, M.D., Department of Dermatology, College of Medicine Korea University, Seoul, Korea.

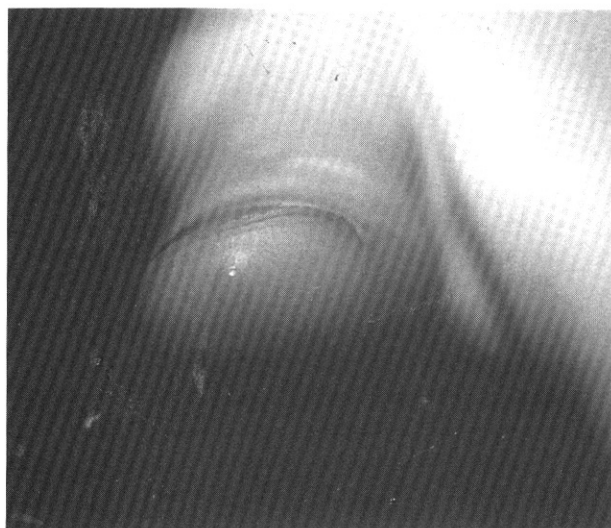


Fig. 1. An erythematous papule on the tip of his right thumb.

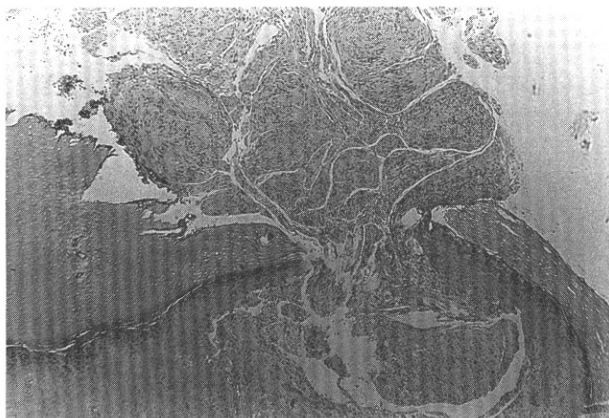


Fig. 2. A cellular nest is removed through a transepidermal canal. (H&E, ×40)

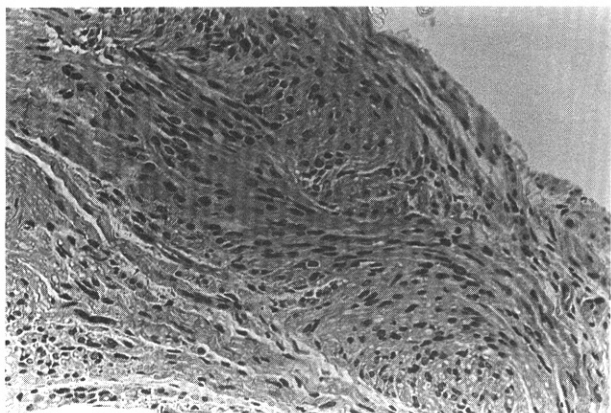


Fig. 3. Numerous elongated nuclei are arranged in a wavy pattern. (H&E, ×200)

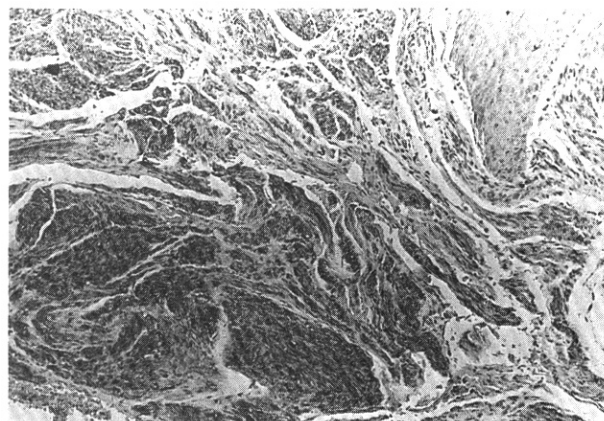


Fig. 4. It shows the presence of S-100 protein in this tumor. (S-100 protein, ×100)

ride, silica and beryllium granulomas, suture, Monsel's solution and tick parts, (5) organisms in infectious disease; leprosy, cryptococcosis, leishmaniasis, tuberculosis, schistosomiasis, blastomycosis, and aspergillosis, (6) tumors and their by-products; pilomatricoma, nevi, malignant melanoma, and nevus sebaceus, and (7) primary perforation disorders; Kyrle's disease, perforating collagenosis, and reactive perforating collagenosis⁴. Mehregan categorized the TEE process into three types depending on the quality and particle size of the foreign materials. In type-I TEE, cellular elements such as lymphocytes, erythrocytes, spirochetes, melanin granules, and chromomycosis organisms transigrate the epidermis with the flow of maturing keratinocytes. In type-II TEE, cell aggregates such as lakes of hemorrhage, small abscesses, or granuloma (sarcoidosis, deep mycoses) are exfoliated with maturing keratinocytes. No clinical lesions are visible in type-I or type-II TEE. The clinical prototype of type-III TEE is a 2 to 5mm, umbilicated, centrally crusted or open papule that histologically reveals a transepidermal canal. This canal is bilaterally encompassed in hyperplastic epidermis and connected with an underlying foreign substance. Because of its compressive histologic presentation, type III is the most readily recognized, and therefore the most often reported form of the TEE phenomenon⁵. Our case shows type-III TEE.

Histologically, it is characterized by varying degrees of pseudoepitheliomatous hyperplasia of the epidermis or follicular epithelium. Elongated tongues of newly formed epithelium extend into

the cornium surrounding the irritation materials. Once the material is partially or completely taken into the perforating epithelium, it is slowly moved upward by the force of maturing keratinocytes and eventually eliminated. A continuous flow and elimination of foreign material to the surface will result in formation of multiple transepithelial perforating canals⁶.

TEE was studied extensively by Bayoumi et al⁷, who injected charcoal particles subepidermally into guinea pig flank skin. They concluded that: (1) the most "sensitive" zone was located above the level of the hair papillae in the dermis; (2) increased epidermal mitotic activity and active epidermal cell movement were involved in the response; and (3) the material was expelled via the hair follicle or intact epidermis. Son and Kim⁸ showed experimentally that the rabbit could be used as another animal model for the investigation of dermatosis showing TEE. This expulsion of particulate matter from the dermis into the exterior is probably mediated by a complex series of interrelated events. The first event may involve recognition of a foreign body by uncharacterized receptors. Recognition may be followed by the elaboration of a substance by dermal components, which induces hyperproliferation of the epidermis. The resultant formation of perforating channels requires the coordination of activities such as enhanced epidermal growth, cell movement, and phagocytosis by individual epidermal cell⁹.

In contrast to an abundance of reports of the TEE conditions, there is a paucity of reports of TEE in benign or malignant tumors. Aside from a few cases of perforating pilomatricoma¹⁰, perforating ossified trichilemmal cyst¹¹, perforating mucinous digital cyst, perforating juvenile xanthogranuloma², and perforating verruciform angioma¹² have been reported. TEE in malignant disorder is a common, yet poorly recognized event. It is frequently seen in mycosis fungoides, Paget's disease of the mammary or extramammary type¹³, Wöringer-Kolopp syndrome¹⁴, and malignant melanoma¹⁵. These are exfoliated for type II-TEE. TEE of type-III in malignant tumors has been reported only in malignant melanoma, squamous cell carcinoma, and basal cell carcinoma².

Schwannomas typically are well circumscribed

tumors composed of spindle cells that are often arranged in cellular (Antoni A) and paucicellular (Antoni B) foci. Vertical palisading of the nuclei in Antoni A tissue is a frequent finding and when present, it is a helpful diagnostic feature. However, nuclear palisading schwannomas may be difficult to differentiate from other spindle cell mesenchymal neoplasms including neurofibromas¹⁶. They are most commonly found in a subcutaneous location on the head or extremities and only rarely on the trunk¹. If present intradermally, they may be multiple¹⁷. Neurilemmomas may be asymptomatic, but they are not infrequently associated with pain, as our case was, which may be localized to the tumor or radiate along the nerve from which the neurilemmoma arises¹⁸.

REFERENCES

1. Lever WF, Schaumburg-Lever G: *Histopathology of the skin*, 7th Ed, JB Lippincott Co, Philadelphia, 1990, pp743-744.
2. Goette DK: Transepithelial elimination of benign and malignant tumors. *J Derm Surg Onco* 13(1):68-73, 1987.
3. Malak JA, Kurban AK: "Catharsis": An excretory function of the epidermis. *Br J Dermatol* 84:516-522, 1971.
4. Patterson JW: Progress in the perforating dermatoses. *Arch Dermatol* 125:1121-1123, 1989.
5. Mehregan AH: Transepithelial elimination. In: Mali JW(ed), *Current Problems in Dermatology*, Vol. 3. Basal, Karger, 1970, pp124-147 (cited from 2)
6. Mehregan AH: Elastosis perforans serpiginosa: A review of the literature and report of 11 cases. *Arch Dermatol* 97:381-393, 1968.
7. Bayoumi AH, Gaskell S, Marks R: Development of a model for transepidermal elimination. *Br J Dermatol* 99:611-620, 1978.
8. Son SJ, Kim SN: An experimental study on tissue reactions to foreign materials. *Kor UMJ* 23(3):191-198, 1986.
9. Bayoumi AH, Marks R: Transepidermal elimination: Studies with an animal model. *Br J Exp Pathol* 61:560-566, 1980.
10. Ter Poorten HJ, Sharbaugh AH: Extruding pilomatricoma. Report of a case. *Cutis* 22:47-49, 1978.
11. Civatte J, Tsoitis G, Le Roux P: "sebacé" (trichilemmal) ossifié perforant, A propos d'un cas. *Ann Dermatol syphiligr (Paris)* 101:155-170, 1974. (cited from 2)
12. Laugier P: Angiome perforant verruciforme. *Ann Dermatol Venereol* 104:492-493, 1977. (cited from 2)
13. Jones RE, Jr Austin C, Ackerman AB: Extramammary Paget's disease. *Am J Dermatopathol* 1:101-132, 1979. (cited from 2)
14. Degreef H, Holvoet C, Van Vloten WA, Desmet V, De Wolf-Peters: Wöringer-Kolopp disease. *Cancer* 38:2154-2165, 1976.

15. Kornberg R, Harris M, Ackerman AB: *Epidermotropically metastatic malignant melanoma. Differentiating malignant melanoma metastatic to the epidermis from malignant melanoma primary in the epidermis.* Arch Dermatol 144:67-69, 1978.
16. Enzinger FM, Weiss SW: *Soft tissue tumors, 2nd Ed.* The C.V. Mosby Company, St. Louis, 1988, pp725-735.
17. Shilshiaba T, Niimura M, Ohtsuka F et al: *Multiple cutaneous neurilemmomas as a skin manifestation of neurilemmomatosis.* J Am Acad Dermatol 10:744-754, 1984.
18. Izumi AK, Rosato JE, Wood MG: *Von Recklinghausen's disease associated with multiple neurilemmomas.* Arch Dermatol 104:172-176, 1971.