

A Case of Tubular Apocrine Adenoma

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Tubular apocrine adenoma is a well-circumscribed intradermal benign tumor with tubular structures showing apocrine differentiation. Most of those cases occur on the scalp in middle aged women. Histologically, tubular structure with apparent apocrine decapitation secretion, cystic dilatation of tubule, and connection with epidermis are the characteristic features of tubular apocrine adenoma. We presented a case of tubular apocrine adenoma that had recurred after incomplete excision. It showed apocrine decapitation secretion histologically and both eccrine and apocrine differentiation on immunohistochemical study.

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Key Words : Apocrine and eccrine differentiation, Decapitation secretion

Tubular apocrine adenoma (TAA) is a rare benign sweat gland tumor and occurs as a solitary nodule commonly on the scalp¹. TAA has been thought apocrine in origin, and papillary eccrine adenoma was regarded as its eccrine counterpart because of their histologic similarity except the apocrine secretion². This distinction is difficult in some cases of TAA with both eccrine and apocrine differentiation or uncertain direction of differentiation. Immunohistochemical and electron microscopic studies were not helpful in clarifying apocrine and eccrine differentiation of this tumor. So, these cases were referred to as tubulopapillary hidradenoma or papillary tubular adenoma without further qualification^{3,4}.

We report a case of TAA that recurred after incomplete excision and showed apocrine decapitation secretion and divergent (apocrine and eccrine)

immunohistochemical phenotypes.

CASE REPORT

A 40 year-old Korean woman presented with an asymptomatic papule on the left side of the nasal bridge of about four years duration (Figure 1). Physical examination showed a 0.7cm × 0.7cm sized, round, soft and flat papule. She said that the lesion was removed with a certain electrical method five years ago, but it recurred one year after. She had no other medical problems including past history and family history for skin tumor.

An excision biopsy was done. Histological examination revealed a well-defined and lobulated tumor mass surrounded by compressed fibrous connective tissue in the deep dermis. In the epidermis, mild acanthosis was shown. We could not find the connection of the tumor with the overlying epidermis or adjacent follicles. The tumor consisted of scant stroma and irregularly dilated or small oval tubular structures with two rows of epithelial cells (Figure 2A). The outer layer was composed of cuboidal or flattened cells and the inner layer was composed of columnar cells showing decapitation secretion in the innermost area (Figure 2B). Eosinophilic amorphous material was seen in

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some lumina of tubules. Projections of tubular structures into the lumina were seen in some dilated tubules but did not complete papillary structures. Atypia and mitosis of tumor cells were not observed. PAS stained the luminal border and decapitation secretion. The PAS positive materials were diastase-resistant. Outer cells of the tubular structures and some stromal cells stained for S-100 protein. The luminal borders of most ducts and decapitation secretion reacted with anti-CEA

and anti-EMA antibodies. In tubular cells, keratin and lysozyme positive cells were present. No Leu-M1 immunoreactivity was detected in the tumor cells. In following up for six months, any sign of recurrence has not developed.

DISCUSSION

Tubular apocrine adenoma (TAA), reported only about 20 cases in literature since 1972, is a rare benign skin tumor. Clinicohistologic findings of TAA are the occurrence on the head, tubular structure with apparent apocrine decapitation secretion, cystic dilatation of tubule, and connection with epidermis¹. In our case, the tumor was located on the head of a middle-aged woman. Tumor mass was made up of scant stroma and regular tubular structures with frequent decapitation secretion. The absence of a connection to overlying epidermis might be caused by previous excision because most cases of TAA shows epidermal connection and this suggested that incomplete removal of TAA resulted in local recurrence. Tubular structures and apparent apocrine secretions, and positive reaction to lysozyme made diagnosis of TAA with apocrine origin in our case. But, the positive reactions with S-100, EMA, and CEA

Fig. 1. A flat papule on the left side of the nasal bridge.

Fig. 2. A. A well-defined tumor shows regular tubular structures. (H&E $\times 40$)
B. The inner cells of two rows of tubular epithelium show apparent decapitation secretions in the lumina. (H&E $\times 400$).

stains suggested eccrine differentiation⁴ although these immunohistochemical results could not be clear-cut criteria for distinguishing eccrine from apocrine differentiation⁵.

Papillary eccrine adenoma (PEA), thought to be an eccrine counterpart of TAA, is different from TAA in localization on the extremities, predominance of cystically dilated tubular structures, and absence of both decapitation secretion and epidermal connection⁴. However, controversy exists about the histogenesis of TAA and PEA. They are described as isolated distinct tumors² or regarded as an identical entity⁶. The term, tubulopapillary hidradenoma or tubular adenoma was suggested because of the difficulties in distinguishing the apocrine or eccrine differentiation. In rare cases with both apocrine and eccrine differentiation^{3,5,7,8}, the coexistence of both differentiations was explained by the existence of apoeccrine gland⁸, the spectrum of differentiation⁷ or tumoral acquisition of new antigens in the process of tumor growth⁵. This case also demonstrated the divergent differentiation in histological and immunohistochemical studies. It can be diagnosed as TAA because it showed apparent apocrine decapitation secretion. However the term tubulopapillary hidradenoma or tubular adenoma would be more accurate because it also shows eccrine differentiation. Further case study is needed to clarify this controversy.

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