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Case study

Acquired adenomatous hyperplasia of the rete testis: an immunohistochemical study of its pathogenesis☆,☆☆

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Summary Adenomatous hyperplasia of the rete testis (AHRT) is an uncommon abnormality first described by Nistal and Paniagua in 1988. Congenital and acquired forms of AHRT are recognized. We present a case of AHRT in a patient who underwent bilateral orchiectomy for penile carcinoma; he had received chemotherapy for Hodgkin lymphoma 18 years prior. Proposed causes for this disorder include developmental, hormonal, and paracrine induction by adjacent testicular tumors and exposure to chemicals, based on clinical contexts but without experimental support. We performed immunohistochemical studies using markers of cell cycle (cyclin D1, p16), proliferation (Ki-67), apoptosis (bcl2), senescence (γ-H2AX), and androgen receptors to try to provide scientific support for or refute existing hypotheses. Our results indicate that, in this case of acquired AHRT after chemotherapy, the process is neither adenomatous nor hyperplastic but rather represents abnormal accumulation of rete testis cells with acquired senescence.
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1. Introduction

Adenomatous hyperplasia of the rete testis (AHRT) is an uncommon, usually incidental finding originally described in an autopsy case by Nistal and Paniagua in 1988 [1]. The patient had received chemotherapy for breast cancer. Histologically, the lesion is characterized by expansion of the septal rete testis, which retains a lobular architecture but shows prominent branching, with solid, tubular, tubulopapillary, or cribriform architecture. The rete testis cells become columnar and show large ovoid to spindle cell nuclei and frequent nuclear folds. Mitosis, apoptosis, or necrosis is absent [1,2]. Nistal et al [2], in a subsequent publication, proposed classifying cases associated with cryptorchidism and developmental anomalies as "congenital" and the remaining cases as "acquired."

We present a case of AHRT in a patient who underwent bilateral orchiectomy for penile carcinoma. The patient had received chemotherapy for Hodgkin lymphoma 18 years prior. Proposed causes for this disorder include estrogenic therapy, androgen blockade, germ cell tumors, and exposure to chemicals, based on clinical contexts but without experimental support [1-3]. We performed immunohistochemical studies using markers of cell cycle, proliferation, apoptosis, senescence, and androgen receptors to try to provide scientific support for or refute existing hypotheses. Our results indicate that, in acquired

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☆☆ All procedures were performed in compliance with institutional guidelines (*Veterans Health Administration Handbook* 1200.05).

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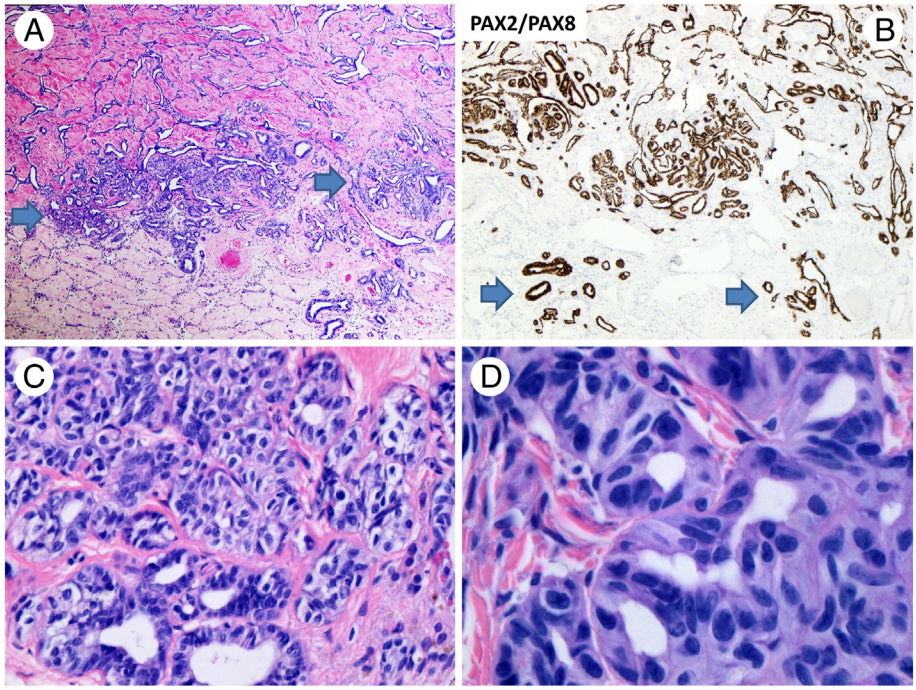

Fig. 1 AHRT. A, Low power showing abnormal complex architecture of the septal rete testis (RT) (arrows) (hematoxylin and eosin [H&E], original magnification ×5). B, PAX2/PAX8 immunostains confirm the rete testis lineage of the abnormal area and partial extension of the process into the adjacent seminiferous tubules (arrows) (×5). C, High power shows abnormal stratification, abnormal gain of abundant cytoplasm, and nuclear enlargement of RT cells (H&E, ×40). D, Oil-immersion micrograph showing enlarged irregular nuclei with smudged chromatin but absent mitotic/apoptotic activity (H&E, ×100).

cases after chemotherapy, the process is neither adenomatous nor hyperplastic but rather represents abnormal accumulation of cells with acquired senescence.

2. Clinical history

A 64-year-old patient underwent palliative total penectomy and bilateral orchiectomy for management of pain and obstruction of stage IV penile squamous cell carcinoma. Medical history included ABVD chemotherapy (doxorubicin, bleomycin, vinblastine, dacarbazine) and radiotherapy of the neck area for Hodgkin lymphoma 18 years prior and local radiotherapy (patient declined chemotherapy) for cT2N2M0 tonsillar squamous cell carcinoma 1 year before this surgery. Exposure history also included heavy tobacco and alcohol use with documented alcoholic cardiomyopathy and a suspected malignant pulmonary lesion for which he declined additional diagnostic workup. The patient did not have history of cryptorchidism.

3. Materials and methods

Orchiectomy specimens were submitted for gross examination. Representative sections of spermatic cord, testis, and epididymis were submitted for histologic evaluation. Immunohistochemical studies were performed in selected slides including AE1/AE3 cocktail (Novacastra, Newcastle Upon Tyne, UK; catalog #NCL-L-AE1/AE3, dilution 1:1600), CAM 5.2 (Cell Marque, Rocklin, CA; catalog #452 M-94, dilution 1:400), calretinin (Novacastra; catalog #NCL-L-CAL-RET-566, dilution 1:1200), androgen receptor (Bio SB, Santa Barbara, CA; catalog #BSB 6074, dilution 1:10), PAX2 (Bio SB; catalog #BSB 2566, pre-dilute), PAX8 (Cell Marque, Rocklin, CA; catalog #363 M-14, dilution:1:200), β-catenin (Cell Marque; catalog #224 M-14, dilution 1:300), bcl2 (Cell Marque, catalog #226R-15, dilution 1:40), p53 (Thermo Scientific, Fremont, CA; catalog #MS-738-P0, dilution: 1:1250), cyclin D1 (Cell Marque; catalog #241R-16, dilution 1:10), p16 (Ventana Medical Systems, Tucson, AZ; catalog #9517, dilution 1:10), Ki-67 (Cell Marque; catalog #275R-15, dilution 1:100), and γ-H2AX (Abcam, Cambridge, UK; catalog #ab11174, dilution 1:5000 and Cell Signaling, Danvers, MA; catalog #9718, dilution 1:50 and 1:100 using SignalStain Antibody Diluent, catalog #8112). Immunohistochemistry was performed on a Leica BOND-III automated stainer (Leica Biosystems, Melbourne, Australia) using EDTA buffer antigen retrieval protocols, except for AE1/AE3, which used enzyme retrieval.

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