Parathyroid Hemangioma: A Report of Two Cases

Maria J. Merino, MD, Rodrigo Chuaqui, MD, and Pedro Fernandez, MD

Abstract

Two cases of intraparathyroid hemangioma, associated with hyperparathyroidism, are reported. The first case showed a typical capillary hemangioma morphology with small branching vascular channels, almost completely replacing the gland’s architecture. The second case was a 2-mm cavernous hemangioma associated with glandular hyperplasia. This is, to our knowledge, the first time that this type of lesion is described.

Key Words: Parathyroid glands; hemangioma; angioma.

Introduction

Benign vascular tumors are frequent neoplasms that have been known to occur in almost every tissue type. Because of their function, endocrine organs usually have abundant vascularity, thus being sites where vascular proliferations develop [1-6]. On the parathyroid gland, however, these lesions have not been recorded. We herein present two cases of hemangioma arising in parathyroid glands associated with hyperparathyroidism.

Case Reports

Case 1

A 68-yr-old woman had a history of polymyalgia rheumatica, osteoporosis, treated hypothyroidism, hypertension, and hypercalcemia secondary to hyperparathyroidism. She underwent right inferior parathyroidectomy. Although she had persistent hypercalcemia and hyperparathyroidism after surgery, she has remained without specific complaints.

Case 2

A 62-yr-old male had a 10-yr history of recurrent duodenal ulcer and renal calculi. Five years before, he had been diagnosed as having primary hyperparathyroidism. On computed tomography (CT)-scan, a right upper (RU) neck mass was found. He underwent surgery and a diagnosis of 1.8 x 1.5 cm “parathyroid adenoma” was obtained. Histopathology of that specimen was not available for review. Postoperatively calcium and parathyroid hormone (PTH) levels, however, remained unchanged. Three years later he had a neck re-exploration that showed three enlarged glands. Right lower (RL), left lower (LL), and left upper (LU) parathyroids were removed. After surgery, calcium and PTH levels dropped down to normal, and the patient had no further evidence of hyperparathyroidism.

Materials and Methods

Tissues were fixed in 10% neutral buffered formaldehyde solution and embedded in paraffin. Five-micron sections were obtained in each case and stained with H&E.

Immunohistochemical evaluation was performed using the biotin-streptavidin complex technique with commercially available antibody antifactor 8 (Dako, Carpinteria, CA) and CD34 (Immunotech, Westbrook, ME). Factor 8 stain was per-
Fig. 1. (left) Case 1: Low-power view showing proliferation of small branching vascular channel replacing the endocrine parenchyma. A thin rim of normal parathyroid tissue is also present.

Fig. 2. (right) Case 1: Stroma was loose and edematous in some areas. Branching capillary channels sometimes contained blood cells. Small aggregates of chief cells were present in the stroma (arrows).

Fig. 3. Case 1: High-power view showing branching capillary channels and some aggregates of chief cells.

formed after 30 min 0.1% trypsin digestion at 37°C, using a 1:800 antibody dilution. For CD34, a 1:200 dilution was used after microwave antigen retrieval. Adjacent normal endocrine parenchyma was used as positive control.

Results

Pathologic Findings

In case 1, the specimen consisted of a 1.5 × 0.7 × 0.3 cm oval fragment of tan-red tissue. Microscopically, the architecture of the gland had been almost completely replaced by a diffuse proliferation of small, branching vascular channels (Fig. 1). Some of them were slightly dilated at the center and the periphery of the lesion. Also at the periphery, a thin rim of preserved normal parathyroid parenchyma was present (Fig. 1). The stroma between the vascular channels was loose and edematous in some areas and contained sparse small aggregates of chief cells (Figs. 2 and 3). Endothelial nuclei were prominent, but neither atypia nor mitotic activity were present. Some of the vascular structures contained red blood cells. Immunostaining for factor 8 and CD34 showed intense expression in endothelial cells (Fig. 4).

In case 2, specimens consisted of three fragments of tan soft tissue measuring 1.2 × 0.7 cm (RL), 1.3 × 0.7 (LL), and 2.3 × 0.6 cm (LU). Microscopically, the LU gland showed a 2-mm nonencapsulated discrete lesion displaying large vascular spaces filled with blood (Fig. 5). Between them, entrapped endocrine cells with nuclear polymorphism and hyperchromasia could be seen. No mitotic activ-