Atypical Ductal Hyperplasia: A Diagnostic Term Whose Days May Be Numbered

Patricia Delgado, MD,1 Jorda Merce, MD, PhD, MBA,2 Taraneh Hashemi-Zonouz, MD,3 Jasmin Sibai,2 and Carmen Gomez-Fernandez, MD, FASCP,4 University of Miami Miller School of Medicine, and 2University of Miami School of Medicine, and 1Yale University School of Medicine

Background: Atypical ductal hyperplasia (ADH) refers to an intraductal clonal epithelial proliferation (≤2 spaces or ≤2 mm). On core needle biopsies (CNBs), ADH is associated with surgical upgrade rates ranging from 0% to 62%. Poor interobserver reproducibility for ADH vs usual ductal hyperplasia (UDH) and low-grade ductal carcinoma in situ (LG-DCIS) may in part explain variable upgrade rates. Immunohistochemistry with CK5/6 and ER is useful for distinguishing the polyclonal UDH from clonal ADH and LG-DCIS. Ductal intraepithelial neoplasia (DIN) has been suggested as alternative terminology for clonal intraductal proliferations: DIN1 (ADH and LG-DCIS), DIN2 (IG-DCIS), and DIN3 (HG-DCIS).

Design: The primary objective was to determine the upgrade rate for a diagnosis of ADH based on CK5/6 and ER and size ≤2 mm on CNBs obtained from 2014 to 2017. The secondary objective was to study the impact that use of DIN1 for ADH and LG-DCIS would have on upgrade rate. Upgrade rate for ADH was determined using the subsequent excision diagnoses: negative for residual disease (NEG), ADH, DCIS, and invasive ductal carcinoma (IDC). Upgrade rate was then recalculated replacing DIN1 for ADH on CNB and comparing against the excision diagnosis of NEG, DIN1, DIN2, DIN3, and IDC.

Results: Thirty-five patients had image-guided, vacuum-assisted CNB for BI-RADS 4 or 5 target lesions (calcifications/mass densities) using 9g(23), 12g(10), or 14g(2) needles, followed by surgical excision for ADH. There was a diagnostic upgrade of 34% (9 LG-DCIS, 2 IG-DCIS, 1 IDC). Size of upgraded lesions ranged from 3 mm to 8 mm. When ADH and LG-DCIS were termed DIN1, the upgrade rate decreased to 8.6%.

Conclusion: Strict criteria for the diagnosis of ADH on CNB, as well as terminology like DIN that addresses a clonal relationship to LG-DCIS, decrease rates of surgical upgrade. Unification of terms and removal of the “malignant” connotation for these small, low-grade intraductal clonal proliferations is timely.

Nonfunctioning Adrenal Cortical Carcinoma Presenting as Flank Pain in a 35-Year-Old Female: A Case Report

Jonathan Newsom, MD,1 Roshanak Derakhshandeh, MD,1 and John Lazarchick, MD,2 1University of South Alabama and 2Mobile Infirmary Medical Center

Adrenal cortical carcinoma (ACC) is a rare malignant neoplasm (0.5–2 cases/million inhabitants per year) originating in the adrenal cortex with a poor prognosis, slight female predilection, and a bimodal age distribution with peaks in the first and fifth decades of life. However, nonfunctioning ACCs usually affect older patients in the fifth to seventh decades of life and occur in males twice as often as females. Although most cases are sporadic, ACCs are also occasionally associated with a variety of hereditary cancer syndromes. Clinical symptoms are most frequently related to an excess of steroid hormones. We report a 35-year-old previously healthy female who presented with a 1-month history of intermittent left flank pain. A CT scan revealed a 16-cm complex, solid mass with scattered calcifications and circumscription by a thin peripheral capsule. The mass was superior to the upper pole of the left kidney and appeared to be separated from the adjacent organs. On gross examination, the mass measured 16.7 cm in greatest dimension and weighed 993 g. Serial sectioning of the mass revealed solid and cystic cut surfaces with a heterogeneous appearance and areas of extensive necrosis. Microscopically, the mass consisted of sheets of large, pleomorphic cells with a hyperchromatic chromatin pattern, frequent bizarre nuclei, eosinophilic cytoplasm, and high mitotic activity. Extra-adrenal extension into surrounding soft tissue was present. Three regional lymph nodes were negative for metastatic disease. The neoplastic cells were positive for Melan A (weak), steroidogenic factor 1, and synaptophysin. Ki-67 expression was estimated at 75%. Immunohistochemical stains for chromogranin, cytokeratin CAM 5.2, desmin, pan-keratin, PAX-8, S-100, SMA, and SOX10 were negative. Our case is unusual because the patient was a younger female who presented with symptoms related to mass effect rather than the hormonal-related changes more commonly seen as a result of a functional ACC.

Sevelamer-Associated Appendicitis

Andrew Jones, MD,1 and Kristin Olson, MD2; 1UC Davis Pathology & Lab Med and 2University of California Davis Health

A 34-year-old woman with a medical history of end-stage renal disease on home peritoneal dialysis secondary to systemic lupus erythematosus and lupus nephritis was admitted to the hospital for abdominal pain. A lengthy hospital stay and extensive workup revealed a gangrenous and perforated appendix with diffuse peritonitis and paracolic gutter abscess. Gross examination of the appendix revealed distension of the appendiceal lumen by finely granular gray-brown material, with an associated full-thickness perforation and fibrinopurulent exudate. Histopathologic examination revealed innumerable crystalline bodies within the appendiceal lumen. Review of her