pink-tan tissue pieces. The mucosa was tan-pink and congested with a 2.0 × 2.0 × 0.2-cm area of irregular thickening. The wall thickness measured up to 0.3 cm. Microscopic examination revealed exuberant tubulo-papillary growth with low- to high-grade dysplasia; focal flat dysplasia; oncocytic, pyloric, and goblet cell metaplasia; marked acute and chronic inflammation; and absence formation. No invasion of the gallbladder wall or Rokitansky-Aschoff sinuses were identified. The tumor cells were immunoreactive for CK20 (strong) and patchy positive for CK7, MUC1, and MUC5 AC. Tumor cells were negative for MUC2, MUC6, CDX2, and Hep-par. Based on these findings, a diagnosis of intracholecystic papillary-tubular neoplasm, pancreaticobiliary type, with high-grade dysplasia and clear margins was made (T1 lesion).

**Conclusion:** ICPNs larger than 1 cm, as in this case, are more commonly of the biliary type showing cuboidal cells, prominent nucleoli, and MUC1 positivity and often associated with high-grade dysplasia and invasive cancers. ICPN patients have a propensity to develop biliary tract carcinomas, especially when associated with extensive high-grade dysplasia. These tumors must be sampled adequately, with documentation of tumor characteristics and presence or absence of invasive component.

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**A Rare Case of Langerhans Cell Histiocytosis With Solitary Hepatic Involvement in an Adult Patient: Challenging Diagnosis, Transplantation, and Recurrence**

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Langerhans cell histiocytosis (LCH) rarely involves liver without systemic manifestation, especially in an adult population. We describe a case of LCH with solitary hepatic involvement that was initially diagnosed as anti-mitochondrial antibody (AMA)-negative primary biliary cholangitis (PBC), requiring transplantation. When disease recurred, the allograft was lost and the patient required a second transplantation. We have reviewed histopathologic material from both native and allograft specimens, including all biopsies and explantatectomies, and clinical data accumulated over a 10-year interval. A 46-year-old woman with a clinical history of panhypopituitarism presented with jaundice and pruritus. An initial hepatology workup including liver biopsy led to diagnosis of AMA-negative PBC with advanced fibrosis. Within a year, the patient progressed to decompensated cirrhosis requiring transplantation. Her posttransplantation course was complicated by frequent bouts of markedly elevated alkaline phosphatase (>1,000 mg/dL) and gamma-glutamyl transferase with transaminases elevated above 100 mg/dL. These abnormalities, together with the liver biopsy findings of bile duct injury and portal eosinophilic infiltrate, were thought to be episodes of acute cellular rejection that occurred frequently during the first 2 years after transplantation. Her liver enzymes would show some improvement without complete normalization on high-dose steroid therapy and become elevated once the therapy stopped. Three years after transplantation, the diagnosis of LCH was established, and 4 years later, the patient lost the liver graft. The explanted allograft revealed patchy distribution of disease, confirming difficulty of LCH diagnosis on a core biopsy. A gold standard for LCH diagnosis relies on morphological and immunohistochemical examination. Establishing a diagnosis of LCH in the liver without systemic involvement is challenging due to the patchy nature of the disease and nonspecific inflammatory infiltrate with eosinophils that can mimic PBC and acute cellular rejection. Awareness of morphological features is crucial for pathologists to render the diagnosis when liver presents as solitary involvement.

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**Role of Immunohistochemistry in the Classification of Glioblastoma and Anaplastic Astrocytoma in Kenyatta National Hospital**

*Evelynn Chege, MBChB; University of Nairobi*

**Objectives:** To describe the role of immunohistochemistry in the classification of glioblastoma and anaplastic astrocytoma at Kenyatta National Hospital.

**Methods:** Laboratory-based retrospective descriptive study conducted at KNH/UON. Study included diagnosed glioblastoma and anaplastic astrocytoma cases (46). Patients’ data and histopathological reports were retrieved from the archives and reanalyzed to identify those with a glioblastoma and anaplastic astrocytoma. Histopathology evaluation was done using formalin-fixed, paraffin-embedded blocks and routine H&E and Ki-67, a cell proliferative marker. Immunohistochemical markers isocitrate dehydrogenase (IDH1) and ATRX were done on tissue microarray blocks. The data were processed in STATA. Descriptive analysis and bivariate analyses were performed to correlate ATRX expression with IDH1 in the cases. The results were presented in the form of charts, tables, and figures.

**Results:** The lesions were multisited but most commonly in the cerebral cortex. On review of the previous vs current diagnosis: previous WHO grades III and IV (GBM 89.1%, AA 6.5%, AT/RT 2.2%, oligoastrocytoma 2.2%) and current diagnosis after consensus (GBM 95.6%, AA 2.2%, and one case of no tumor). There were 44 GBM cases: classical subtype (77.3%) and one case of oligodencreytic (2.3%). Forty-five cases underwent IHC. In the majority of the cases (28/44), Ki-67 had a mitotic score (mean [SD] 27.1% [15.1%]) of the GBM cases. The AA case mitotic rate was 7%. IDH1 mutations were present in 11 of 44 GBM cases and in the AA case. ATRX loss was in 17 of 44
GBM cases and the AA case. The anaplastic astrocytoma displayed both IDH1 mutation and ATRX loss.

**Conclusion:** The glioblastoma IDH wild type were the majority in this study and have been shown to have poor prognosis as compared to the longer survival time of the glioblastoma, IDH-mutant, and I would recommend a correlation study of survival time to be carried out within our study population.

**Tall Cell (With Reverse Polarity) Carcinoma of Breast: Molecular Characterization of Six Cases by Next-Generation Sequencing**

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**Objectives:** Tall cell carcinoma of breast (TCCB), also known as “breast carcinoma with reverse polarity,” is a rare entity that shares some pathological features with solid-papillary carcinoma of breast. A few reports (all from 2017) have helped to establish the histological and immunohistochemical features thereof; however, to date, only 16 cases have undergone molecular characterization.

**Methods:** Archived TCCB cases (n = 6), all received in our department for diagnostic consultation (over a 5-year period: 2013–2017), were retrieved and the diagnosis was confirmed. Five cases of non-TCCB solid-papillary breast carcinoma were used as controls. The molecular profiles of all samples were examined through Oncomine (Thermo Fisher Scientific, Waltham, MA), a comprehensive next-generation sequencing assay.

**Results:** All patients were female, with a mean age of 68 years (range, 63–76). Mean tumor size was 1.3 cm (range, 0.7–1.8 cm). To our knowledge, none of the six TCCB cases has recurred or shown axillary nodal or distant metastasis. All cases showed histopathological features characteristic of the entity: architecturally multinodular, solid-papillary growth pattern, with cytologically monotonous tall cells bearing apically placed (ie, reversed polarity) nuclei. By Oncomine assay, all six TCCB tumors demonstrated IDH2 R172 single-nucleotide variants (three R172S, two R172T, and one R172W). Additionally, five (83%) tumors had PIK3CA mutations (four H1047R and one Z545K). No BRAF or RAS mutations were identified. In contrast, none of the five control cases showed IDH2 mutation.

**Conclusion:** In sum, IDH2 mutation was identified in all six TCCB cases. This finding supports the previously reported observation that IDH2 mutation defines this unique subtype of breast cancer.

**The Prevalence of Triple Negative Breast Cancer and Its Androgen Receptor Coexpression in Harare, Zimbabwe**

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**Objectives:** To determine the prevalence of triple-negative breast cancer and its androgen receptor coexpression by way of immunohistochemistry.

**Methods:** The study was conducted on 62 known triple-negative breast cancer cases taken out of 298 total cases, which were further analyzed for their androgen receptor coexpression, including age, race, and tumor characteristics.

**Results:** The study revealed 21.8% triple-negative breast cancer prevalence from cases taken from the last quarter of 2012 to the first quarter of 2016. Of the 298 cases, 65 were triple negative. One male case and two tiny inadequate biopsies were excluded. The study revealed that 45.2% of triple-negative breast carcinoma showed androgen receptor coexpression with a mean (SD) of 5.1 out of 8 total staining score, compared to 54.8% that were negative for androgen receptor coexpression. No significant association was found to exist between triple-negative cancers and race (P = .113), but there was strong association with grade 2 ductal carcinoma (P = .05) and 92% representation and age (P = .032). The triple-negative receptor status was associated with left breast laterality (P = .001) and a 55.4% representation.

**Conclusion:** The prevalence of triple-negative breast carcinoma is 21.8%, and tumors that are triple negative and also coexpressing androgen receptors show a high prevalence of 45.2%.

**PAX-8 Expression in Retinoblastoma and the Eyes of Human Embryos**

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**Objectives:** A previous in vitro study demonstrated that retinoblastoma protein is a transcriptional coactivator of paired box-8 (PAX-8). While PAX-8 immunohistochemical stains are commonly used to identify neoplasms arising from renal tissue or the female genital tract, the expression of PAX-8 in neural lesions is less clear. The objective of our study was to evaluate the expression of PAX-8 in retinoblastomas and embryonal eye tissue.

**Methods:** Using a standard immunohistochemical staining method, formalin-fixed, paraffin-embedded tissues were stained for PAX-8. To assess expression in neural lesions, we stained retinoblastoma, medulloblastoma, and neuroblastoma cases. In addition to the tumors mentioned, we also stained fetal eye tissue at 7 weeks and 13 weeks with PAX-8.

**Results:** PAX-8 expression was positive in the nuclei of the retinoblastoma cases and negative in the nuclei of the medulloblastoma and neuroblastoma cases. The fetal tissue showed that nuclear PAX-8 expression is present as early as 7 weeks. By 13 weeks, the PAX-8 expression is seen only in the posterior portion of the embryonal eye.