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Editorial

# The Complaisant Cannula-Bile Duct Adenoma

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Bile duct adenoma emerges as a benign, biliary neoplasm constituted of uniform dissemination of miniature bile ducts enmeshed within a fibrous tissue stroma. Additionally, designated as peribiliary gland hamartoma, cholangioma or cholangioadenoma, bile duct adenoma configures as a benign epithelial tumour.

Majority (~90%) of neoplasms represent as a solitary nodule confined to sub-capsular hepatic parenchyma. Morphologically, regularly distributed, miniature tubules devoid of cytological atypia are encountered. Mitotic figures are absent.

Commonly, bile duct adenoma incriminates adult population > 20 years. An equivalent gender predilection is observed [1,2].

Bile duct adenoma preponderantly incriminates hepatic parenchyma and is characteristically situated within the sub-capsular region.

Neoplasm was initially contemplated to be a reactive ductular proliferation occurring as an expression of focal bile duct injury with subsequent localized, biliary healing [1,2].

Currently, it is posited that tumefaction emerges as a reaction equivalent to functioning of peri-biliary gland, as pyloric gland metaplasia confined to the foregut or may configure as a hamartoma. Additionally, frequently discernible BRAF V600E genetic mutations appear to postulate a neoplastic genesis [1,2].

Of obscure aetiology, hypothesis of genesis of bile duct adenoma occurring due to focal bile duct injury compounding as a trigger event remains debatable [1,2].

Majority of bile duct adenomas are discerned incidentally upon surgical intervention or autopsy studies. Lesion is frequently evaluated by frozen section in order to exclude distant metastasis from a primary adenocarcinoma [1,2].

Upon frozen section, a solitary, sub-capsular, well circumscribed, non encapsulated tumefaction is observed. Neoplasm is composed of bland proliferation of miniature tubules entangled within preponderantly fibrous tissue stroma. Neoplasm is devoid of significant nuclear atypia or intraluminal secretion of bile. Mitotic figures or focal necrosis are absent [2,3].

Morphological features as cellular stroma, compact glandular architecture, mucin secretion, cytological atypia and emergence of unusual variants as oncocytic features or clear cell change may be misinterpreted as foci of malignant metamorphosis and necessitate additional evaluation [2,3].

Cytological examination exhibits abundant quantities of normal, unremarkable biliary epithelium. Frequently, epithelial cells appear commingled with benign hepatocytes.

Cell block preparations may be beneficially adopted to discern the neoplasm [2,3].

Grossly, tumefaction is predominantly solitary, firm, grey/white, sub-capsular, well circumscribed and non encapsulated. However, multiple lesions may be discerned. Tumour magnitude varies from one millimetre to 20 millimetres with a mean of 5.8 millimetres [2,3].

Upon microscopy, miniature, uniformly disseminated tubules imbued with miniscule or absent lumen may be discerned.

Bile duct adenoma is layered by singular layer of cuboidal cells devoid of cytological atypia. The well circumscribed neoplasm is enmeshed within a preponderantly fibrous tissue stroma. Mitotic figures are absent.

Intraluminal bile is absent as biliary ducts appear non communicative with the biliary tree [3,4].

Exceptionally, neoplasm may display distinct variants as tumours with clear cell change, oncocytic features, neoplastic secretion of droplets immune reactive to alpha-1 antitrypsin or mucin secreting adenoma [3,4].

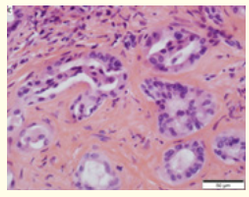

**Figure 1:** Bile duct adenoma depicting uniformly disseminated tubules layered by cuboidal epithelium lacking cytological atypia. Surrounding stroma is fibrotic. Mitotic figures are absent [7].

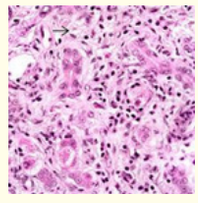

**Figure 2:** Bile duct adenoma delineating tubular structures layered by cuboidal epithelium devoid of cytological atypia. Circumscribing stroma is fibrotic. Mitotic figures are absent [8].

Okuda staging system scripted by K Okuda in 1985 for categorizing hepatocellular carcinoma is constituted of features as

- Carcinoma incriminating >50% of hepatic parenchyma
- Occurrence of ascites
- Serum albumin ≤3 milligrams/decilitre
- Serum bilirubin ≥3 milligrams/decilitre.

Thus categorized, hepatocellular carcinoma is subdivided into

- Stage A comprised of 0 criteria
- Stage B comprised of 1 to 2 criteria
- Stage C comprised of 3 to 4 criteria.

Enhanced tumour stage appears concurrent with inferior prognostic outcomes [3,4].

Bile duct adenoma appears immune reactive to characteristic biliary immune markers as CK7 or CK19.

Besides, neoplasm is immune reactive to MUC5AC, MUC6, BRAF V600E or p16INK4a [5,6].

Ki67 proliferation index is minimal and appears at <10%.

Tumour cells appear immune non reactive to EZH2 [5,6].

Majority of bile duct adenomas may be discerned with albumin in situ hybridization, a feature which segregates the lesion from pancreatic adenocarcinoma associated with distant metastasis [5,6].

Bile duct adenoma requires segregation from neoplasms as cholangiocarcinoma, metastatic adenocarcinoma and Von Meyenburg complex or biliary microhamartoma [5,6].

Bile duct adenoma can be appropriately ascertained upon cogent clinical representation and morphological features ascertained from surgical tissue samples [5,6].

Upon computerized tomography (CT) and magnetic resonance imaging (MRI), a characteristic hyper-enhancement of arterial phase appears to persist into portal venous and delayed phase.

Upon T1 weighted magnetic resonance imaging (MRI), hypo-intense signal intensity is observed. Upon T2 weighted magnetic resonance imaging, hyper-intense signal intensity is delineated [5,6].

Bile duct adenoma is posited to configure as a possible, debatable precursor to intrahepatic cholangiocarcinoma [5,6].

## Bibliography

1. Sasaki M., *et al*. "Bile duct adenoma and small-sized small duct type intrahepatic cholangiocarcinoma show distinct differences in genetic alterations, expression of IMP3 and EZH2 and stromal and inflammatory components". *Histopathology* 83.2 (2023): 298-309.
2. Huang M., *et al*. "Magnetic resonance imaging features of bile duct adenoma". *Frontiers in Oncology* 13 (2023): 1180186.
3. Li W., *et al*. "Clinical study of extrahepatic biliary adenoma". *World Journal of Clinical Cases* 11.24 (2023): 5692-5699.
4. Okuda K., *et al*. "Natural history of hepatocellular carcinoma and prognosis in relation to treatment. Study of 850 patients". *Cancer* 56.4 (1985): 918-928.
5. Kow DK., *et al*. "*In vivo* detection of bile duct pre-cancer with endoscopic light scattering spectroscopy". *Nature Communication* 14.1 (2023): 109.
6. Coucke EM., *et al*. "Biliary Obstruction". Stat Pearls International. Treasure Island, Florida.
7. Image 1 Courtesy: Libre pathology.
8. Image 2 Courtesy: Basic medical key.