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Gum, Sap and Canker-Colloid Carcinoma-Pancreas

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Abstract

Colloid carcinoma pancreas is an infiltrative ductal epithelial neoplasm of pancreas characteristically denominating a preponderant (>80%) component of enlarged pools of extracellular stromal mucin pervaded with suspended neoplastic cells. Colloid carcinoma pancreas is a microsatellite stable tumefaction and exhibits KRAS genetic mutation confined to codon 12. Tumefaction is posited to arise from inverse polarization of cells with stromal mucin glycoproteins facing intrinsic cellular surface. Cogent clinical symptoms as abdominal or epigastric pain, pancreatitis, diarrhoea, hyperbilirubinemia or loss of weight are discerned. Tumefaction emerges as an enlarged, well demarcated lesion with a mean diameter of 5 centimetres and a solid, firm, gelatinous cut surface. Neoplasm is predominantly comprised of enlarged, extracellular accumulates of stromal mucin with minimal carcinoma cells suspended within extra-cellular mucin pools. Cuboidal or columnar epithelial cells configure cribriform or stellate cellular clusters or miniature tubules and strips of columnar cells along with signet ring cells. Colloid carcinoma pancreas is intensely immune reactive to CDX2, MUC2 and CEA. Neoplasm requires segregation from tumours as extravasation of benign stromal mucin, intra-ductal papillary mucinous neoplasm, mucinous cystic neoplasm or conventional pancreatic ductal adenocarcinoma. Colloid carcinoma pancreas is devoid of specific therapeutic guidelines or recommended treatment.

Keywords: epithelial, stromal mucin, inverse polarization

Colloid carcinoma pancreas manifests as a ductal epithelial neoplasm of pancreas. As defined by World Health Organization (WHO), the infiltrative neoplasm characteristically denominates a preponderant (>80%) component of enlarged pools of extracellular, stromal mucin pervaded with suspended neoplastic cells. Colloid carcinoma pancreas commonly incriminates head of pancreas.

Tumour volume is predominantly (>80%) comprised of accumulated mucin with disseminated scanty, floating carcinoma cells. The neoplasm is associated with intra-ductal papillary mucinous neoplasm, preponderantly an intestinal subtype, mucinous cystic neoplasm and ampullary or duodenal tubulovillous adenomas.

Colloid carcinoma pancreas is additionally designated as mucinous non-cystic carcinoma or gelatinous carcinoma. Colloid carcinoma pancreas demonstrates superior prognostic outcomes, in contrast to conventional pancreatic ductal adenocarcinoma.

Colloid carcinoma pancreas manifests as a

malignant neoplasm of exocrine pancreas wherein around 27% to 70% of intra-ductal papillary mucinous neoplasm (IPMN) with associated invasive adenocarcinoma exhibit a colloid component (Chen CH, Yeh HZ & Li HN, 2022; Yasuoka H, Kato H, Asano Y et al., 2022).

Colloid carcinoma pancreas is posited to arise from inverse polarization of cells with stromal mucin glycoproteins facing intrinsic surface rather than luminal cellular surface. Besides, neoplastic cells frequently express MUC2, a gel forming mucin. Additionally, absence of external lamina or basement membrane may contribute to accumulation of extracellular mucin, a feature which restricts neoplastic dissemination and manifests tumour suppressor activity (Chen CH, Yeh HZ & Li HN, 2022; Yasuoka H, Kato H, Asano Y et al., 2022). Colloid carcinoma pancreas is a microsatellite stable tumefaction and exhibits KRAS genetic mutation confined to codon 12 in ~33% instances. Besides, TP53 genomic mutation may ensue. Somatic mutations confined to GNAS are frequently encountered.

Mean age of disease emergence is 61 years. An equivalent gender predisposition is observed (Chen CH, Yeh HZ & Li HN, 2022; Yasuoka H, Kato H, Asano Y et al., 2022). Colloid carcinoma pancreas represents with cogent clinical symptoms as abdominal or epigastric pain, pancreatitis (50%), diarrhoea, hyperbilirubinemia or loss of weight. Enlarged tumours ~6.0 centimetre magnitude display a decimated tumour stage and superior survival outcomes, in contrast to conventional pancreatic ductal adenocarcinoma (Chen CH, Yeh HZ & Li HN, 2022; Yasuoka H, Kato H, Asano Y et al., 2022). Adoption of diagnostic manoeuvers as an incisional biopsy may contribute to emergence of complications as thromboembolic phenomenon. Exceptionally, colloid carcinoma pancreas delineates complications such as pseudomyxoma peritonei (Yasuoka H, Kato H, Asano Y et al., 2022; Fujii M, Okamoto Y, Fujioka SI et al., 2022).

Grossly, colloid carcinoma pancreas emerges as an enlarged, well demarcated tumefaction with a mean tumour diameter of 5 centimetres. Cut surface is solid, firm and gelatinous (Yasuoka H, Kato H, Asano Y et al., 2022; Fujii M, Okamoto Y, Fujioka SI et al., 2022). Cytological smears of the abundantly mucoid colloid carcinoma pancreas may be challenging to disperse upon glass slides. Besides, malignant component may delineate minimal cellularity (Yasuoka H, Kato H, Asano Y et al., 2022; Fujii M, Okamoto Y, Fujioka SI et al., 2022).

Upon microscopy, the neoplasm is predominantly (~80%) comprised of enlarged, extracellular accumulates of stromal mucin. Mucoid component may appear nodular and invasive with a magnitude of ≥ 1 centimetre. Carcinoma cells are minimal and appear suspended within extra-cellular mucin pools. Generally, tumour cells manifest as cuboidal or columnar epithelial cells and configure cribriform or stellate cellular clusters or miniature tubules or strips of columnar cells. Signet ring cells may be discerned. Articulated mucin lakes frequently demonstrate incomplete layering with neoplastic epithelial cells. Besides, mucin secreted by tumour cells may be retained during histological processing (Fujii M, Okamoto Y, Fujioka SI et al., 2022; Orcutt ST, Coppola D, Hodul PJ., 2016).

Colloid carcinoma pancreas frequently concurs with tubular adenoma, tubulo-villous adenoma, intra-ductal papillary mucinous neoplasm or mucinous cystic neoplasm. Tumefaction is frequently accompanied by perineural tumour infiltration or regional lymph node metastasis. Upon ultrastructural examination, mucigen granules appear disseminated upon the stromal surface. A distinctive basement membrane circumscribing glandular articulations or neoplastic aggregates is absent (Fujii M, Okamoto Y, Fujioka SI et al., 2022; Orcutt ST, Coppola D, Hodul PJ., 2016).

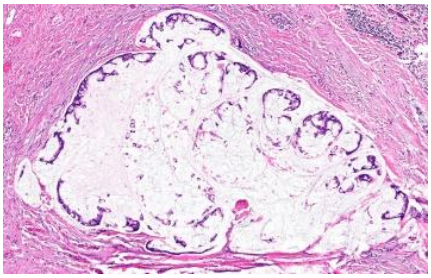

Figure 1. Colloid carcinoma demonstrating large pools of extracellular stromal mucin disseminated with and layered by a scanty population of neoplastic cells

Source: Pathology outlines

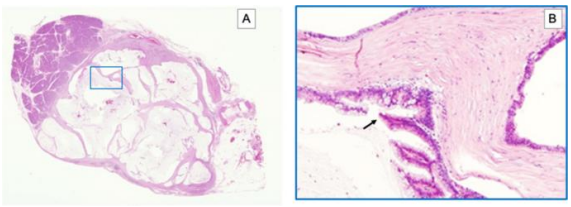

Figure 2. Colloid carcinoma delineating a solid, firm and gelatinous cut surface with large aggregates of extracellular stromal mucin with minimal quantities of floating and layering carcinoma cells

Source: Springer link.

Grading of colloid carcinoma pancreas contingent to microscopic evaluation is denominated as

•GX: Tumour grade cannot be evaluated

•G1: Low grade, well differentiated neoplasm simulating normal pancreatic architecture with minimally aggressive biological behaviour and superior prognostic outcomes

•G2: Moderately differentiated neoplasm with cytology and architecture intermediate to grade 1 and grade 3

•G3: High grade, poorly differentiated neoplasm with significant cytological atypia, aggressive biological behaviour and inferior prognostic outcomes (Fujii M, Okamoto Y, Fujioka SI et al., 2022; Orcutt ST, Coppola D, Hodul PJ., 2016).

Colloid carcinoma pancreas is intensely immune reactive to CDX2 and MUC2, thereby indicating intestinal differentiation. Besides, tumour cells are immune reactive to carcinoembryonic antigen (CEA) and focally reactive to synaptophysin or chromogranin.

Colloid carcinoma pancreas is immune non reactive to HER2 and MUC1 (Fujii M, Okamoto Y, Fujioka SI et al., 2022; Orcutt ST, Coppola D, Hodul PJ., 2016). Colloid carcinoma pancreas requires segregation from neoplasms such as extravasation of benign stromal mucin, intra-ductal papillary mucinous neoplasm, mucinous cystic neoplasm or conventional pancreatic ductal adenocarcinoma (Fujii M, Okamoto Y, Fujioka SI et al., 2022; Orcutt ST, Coppola D, Hodul PJ., 2016).

Colloid carcinoma pancreas can be appropriately discerned upon histological examination of extensive tissue sampling obtained with surgical resection specimen. Upon radiographic examination, colloid carcinoma pancreas demonstrates dilated ductal articulations. Pancreatic parenchyma may occasionally be pervaded with nodular lesions (Fujii M, Okamoto Y, Fujioka SI et al., 2022; Orcutt ST, Coppola D, Hodul PJ., 2016).

Colloid carcinoma pancreas is devoid of specific therapeutic guidelines or recommended treatment. Colloid carcinoma pancreas demonstrates a 5 year survival of 57% to 72%. Factors such as tumour diameter, concurrence of intra-ductal papillary mucinous neoplasm (IPMN) or mucinous cystic neoplasm (MCN) as a precursor lesion, status of surgical margins or regional lymph nodes, vascular or perineural invasion and chromosomal mutation within KRAS or TP53 do not influence prognostic outcomes (Fujii M, Okamoto Y, Fujioka SI et al., 2022; Orcutt ST, Coppola D, Hodul PJ., 2016).

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