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## Short Communication

# Gum, sap and canker-colloid carcinoma -pancreas

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## Abstract

Colloid carcinoma pancreas is an infiltrative ductal epithelial neoplasm of the 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 the inverse polarization of cells with stromal mucin glycoproteins facing the intrinsic cellular surface. Cogent clinical symptoms such 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 extracellular 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.

## Introduction

Colloid carcinoma pancreas manifests as a ductal epithelial neoplasm of the 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 the head of the 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 the exocrine pancreas wherein around 27% to 70% of intra-ductal papillary mucinous neoplasm (IPMN) with associated invasive adenocarcinoma exhibit a colloid component [1,2].

Colloid carcinoma pancreas is posited to arise from inverse polarization of cells with stromal mucin glycoproteins facing the intrinsic surface rather than the luminal cellular surface. Besides, neoplastic cells frequently express MUC2, gel-forming mucin. Additionally, the absence of external lamina or basement membrane may contribute to the accumulation of extracellular mucin, a feature which restricts neoplastic dissemination and manifests tumour suppressor activity [1,2].

Colloid carcinoma pancreas is a microsatellite stable tumefaction and exhibits KRAS genetic mutation confined to codon 12 in ~33% of instances. Besides, TP53 genomic mutation may ensue. Somatic mutations confined to GNAS are frequently encountered.

The mean age of disease emergence is 61 years. An equivalent gender predisposition is observe [1,2]. Colloid carcinoma pancreas represents cogent clinical symptoms such 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 [1,2].

Adoption of diagnostic manoeuvres as an incisional biopsy may contribute to the emergence of complications

as a thromboembolic phenomenon. Exceptionally, colloid carcinoma pancreas delineates complications such as pseudomyxoma peritonei [2,3].

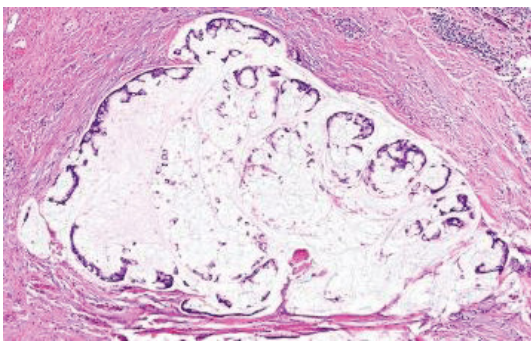
Grossly, colloid carcinoma pancreas emerges as an enlarged, well-demarcated tumefaction with a mean tumour diameter of 5 centimetres. The Cut surface is solid, firm and gelatinous [2,3]. Cytological smears of the abundantly mucoid colloid carcinoma pancreas may be challenging to disperse upon glass slides. Besides, a malignant component may delineate minimal cellularity [2,3].

Upon microscopy, the neoplasm is predominantly (~ 80%) comprised of enlarged, extracellular accumulates of stromal mucin. The mucoid component may appear nodular and invasive with a magnitude of  $\geq 1$  centimetre.

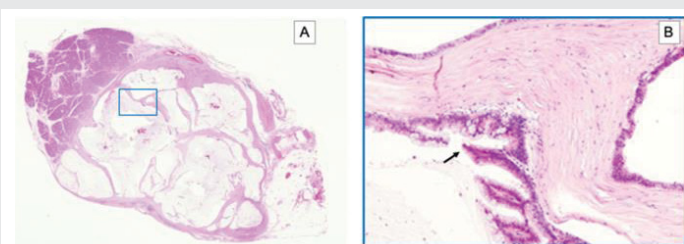
Carcinoma cells are minimal and appear suspended within extra-cellular mucin pools. Generally, tumour cells manifest as cuboidal or columnar epithelial cells and configure cribriform or stellate cellular clusters or miniature tubules or strips of columnar cells. Signet ring cells may be discerned (Figure 1).

Articulated mucin lakes frequently demonstrate incomplete layering with neoplastic epithelial cells. Besides, mucin secreted by tumour cells may be retained during histological processing [3,4] (Figure 2).

Colloid carcinoma pancreas frequently concurs with tubular adenoma, tubulovillous 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



**Figure 1:** Colloid carcinoma demonstrating large pools of extracellular stromal mucin disseminated with and layered by a scanty population of neoplastic cells [5].



**Figure 2:** Colloid carcinoma delineates a solid, firm and gelatinous cut surface with large aggregates of extracellular stromal mucin with minimal quantities of floating and layering carcinoma cells [6].

circumscribing glandular articulations or neoplastic aggregates is absent [3,4].

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 [3,4].

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 and non-reactive to HER2 and MUC1 [3,4]. 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 [3,4].

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 [3,4].

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, the 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 [3,4].

## Conclusion

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. Tumour diameter,



precursor lesions of intra-ductal Papillary Mucinous Neoplasm (IPMN) or Mucinous Cystic Neoplasm (MCN), the status of surgical margins, regional lymph node, vascular or perineural invasion and chromosomal mutation within KRAS or TP53 do not influence prognostic outcomes.

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5. Image 1 Courtesy: Pathology outlines.
6. Image 2 Courtesy: Springer link.