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# Double Primary Cancer of the Pancreas and Gallbladder: Case Report

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**Abstract** **Case Report**

Synchronous tumors occurring in both the pancreas and gallbladder are uncommon. They are frequently linked to an atypical pancreatobiliary junction, which causes a continuous backflow of pancreatic enzymes, leading to ongoing biliary inflammation [1]. In this report, we discuss the case of a 51-year-old woman with concurrent pancreatic and gallbladder lesions, initially regarded as two separate primary cancers and managed with curative treatment.

**Keywords**: Pancreatic Adenocarcinoma, Gallbladder Adenocarcinoma, Pancreaticoduodenectomy.



## INTRODUCTION

Synchronous tumors of the pancreas and gallbladder are rare and often attributed to an abnormal pancreato-biliary junction, which results in a persistent reflux of pancreatic secretions leading to chronic biliary inflammation [1].

## CASE REPORT

The patient was 51-years-old without comorbidities.

The history of the illness dates back to February 2024 with the onset of abdominal pain and cholestatic jaundice. Upon admission, laboratory results indicated elevated transaminase levels (ASAT at 391 U/L and ALAT at 910 U/L) and signs of icteric cholestasis, with GGT at 600 U/L and bilirubin at 64.1 µmol/L. The complete blood count, creatinine, and electrolyte levels were within normal ranges, and Ca 19-9 levels were also normal.

A CT scan of the chest, abdomen, and pelvis revealed a mass located in the pancreatic head, accompanied by intrahepatic bile duct dilation. Additionally, a suspicious thickening of the gallbladder was noted. The patient subsequently underwent a cephalic duodenopancreatectomy and an extended cholecystectomy [figure1].

Intraoperative frozen sections were not performed, and the gallbladder wall thickening was initially attributed to cholecystitis, likely due to obstruction from the carcinoma in the pancreatic head and possibly cholelithiasis. Unexpectedly, the pathological analysis identified two distinct tumors: a moderately differentiated ductal adenocarcinoma of the pancreas [pT2 pN1 (3/14)], with lymphovascular invasion, perineural infiltration, and positive margins at the mesenteric vein; and a poorly differentiated adenocarcinoma of the gallbladder, classified as pT3 pN1 (3/5), with V1, L1, Pn1, and R0 statuses. [Figure 2, 3]

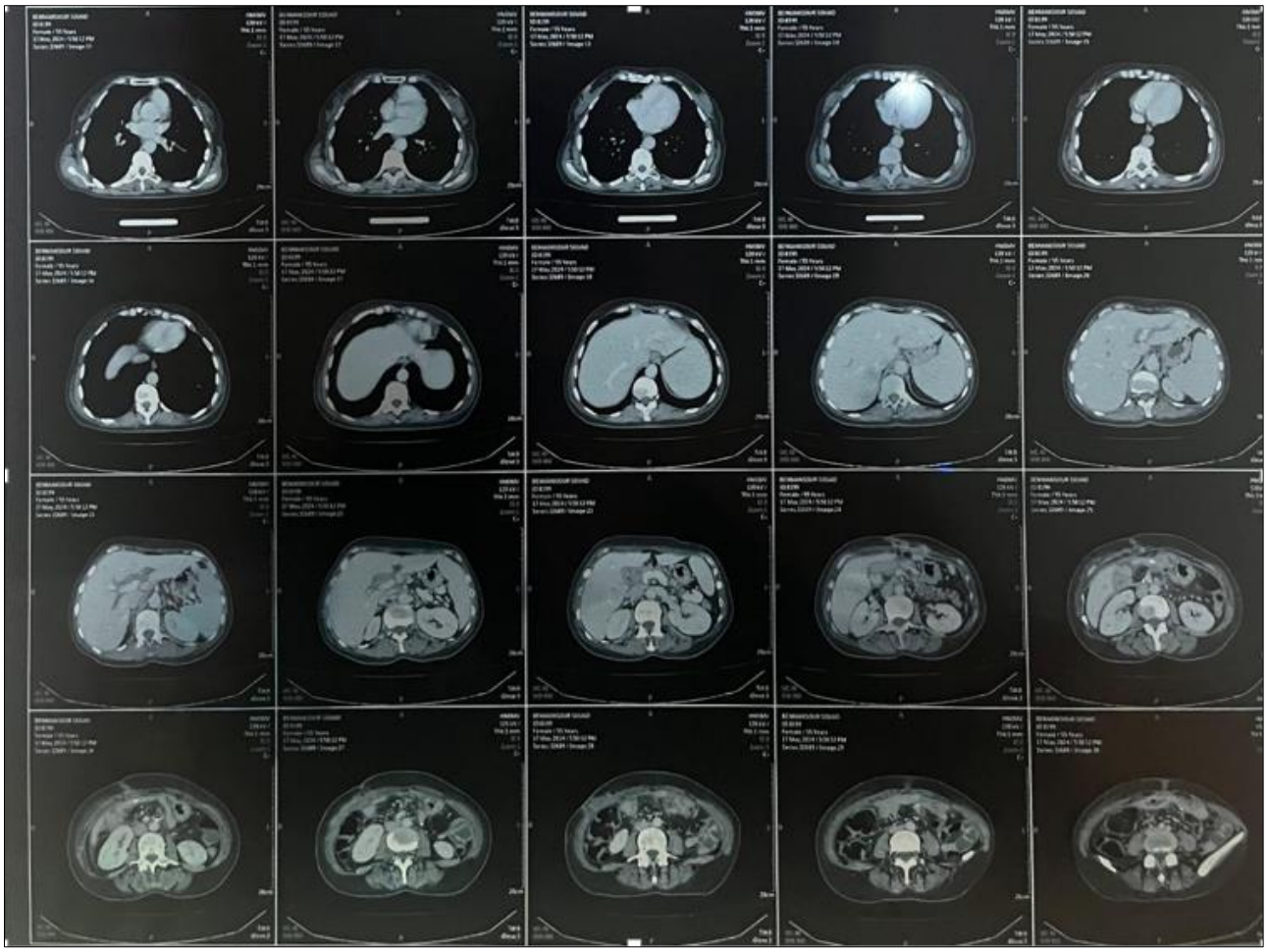

**Fig.1**

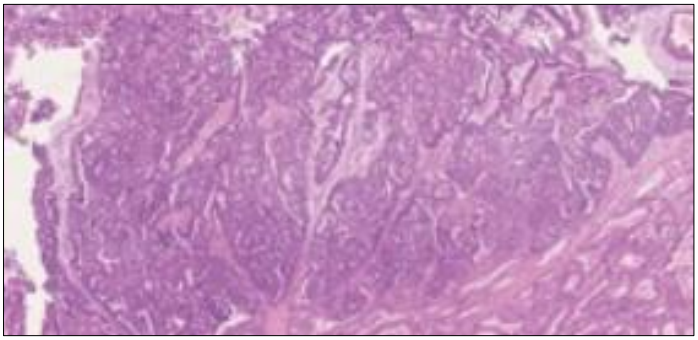

**Figure 2: Gallblader**

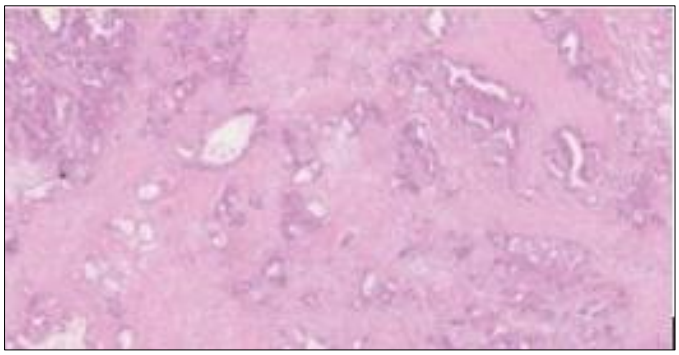

**Figure 3: pancreas**

The patient is under adjuvant chemotherapy with modified folfirinox for six months with excellent clinical tolerance.

## DISCUSSION

Primary pancreatobiliary tumors occurring independently are uncommon and are typically believed to be linked to pancreatobiliary reflux [2]. To date, only seven cases of concurrent gallbladder and pancreatic cancers have been reported [Table 1].

Pancreatic metastases are rare, occurring in just 3–12% of patients with extensive metastatic disease at autopsy. They account for only 2–5% of all pancreatic cancers, with the most common primary sources being the kidney, melanoma, and breast cancer [3].

Recent technological advancements, including next-generation sequencing (NGS), may help determine whether tumors from different organs in the same patient share a clonal origin. Gallbladder cancer (GBC) is associated with various mutations, with TP53, KRAS, and PIK3CA being the most prevalent [10]. These mutations are also observed in pancreatic carcinomas, though their frequencies vary. TP53, a tumor suppressor gene, is frequently mutated in many types of cancer. In pancreatic cancer, TP53 mutations are found in 73.9% of cases, while in GBC, they occur in 47% of patients [11, 12].

KRAS mutations are commonly seen in biliary tract cancers, especially in cases with anomalous pancreaticobiliary junctions. These mutations occur in over 95% of pancreatic ductal adenocarcinomas (PDAC) and are considered an early event in the development of PDAC [16]. KRAS mutations typically arise at codons 12 (54–74%), 13 (3–5%), and 61 (3–5%). In contrast, KRAS mutations are less frequently observed in GBC, particularly in Western populations (0–10%), although the incidence can vary significantly in Japanese studies (0–59%) [12-14]. The lack of KRAS mutations in the analysis of the pancreatic lesion, though not conclusive, suggests that the tumor might not originate from the pancreas. HER2 amplification has been detected in up to 14% of advanced GBC cases. Notably, recurrent mutations in the ErbB pathway have been linked to worse outcomes. Clinical evidence indicates that HER2-targeted therapies may offer a potential treatment option for these patients [15, 16].

## CONCLUSION

In conclusion, while the pancreas is an unusual site for metastasis in gallbladder adenocarcinoma, it remains clinically important when simultaneous lesions are identified. When available, next-generation sequencing (NGS) can offer crucial insights during the evaluation of such cases.

**Statements and Declarations:**

**Ethics Approval and Consent to Participate**

UNIVERSITY Mohammed V souissi RABAT Morocco faculty of medecine approved the study

**Consent for Publication:** The patient consent the publication of the personnal data

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**Radiology departemenent** of the Military Hospital RABAT Morocco

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