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Obstructive Jaundice Unmasking Suspected Cholangiocarcinoma: Diagnostic Challenges and Early Multidisciplinary Intervention

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ABSTRACT

Introduction: Perihilar cholangiocarcinoma is a rare but aggressive malignancy arising at the biliary confluence and often presents late due to its subtle and insidious progression. Obstructive jaundice is the most frequent initial manifestation, typically accompanied by pruritus and cholestatic biochemical abnormalities. Early recognition and timely intervention are critical to prevent complications and enable multidisciplinary oncologic planning.

Case Presentation: A 67-year-old male with a history of systemic hypertension presented with progressive jaundice for 20 days and pruritus with sleep disturbance for one week, without fever or constitutional symptoms. Laboratory evaluation revealed total bilirubin 26.3 mg/dL, direct bilirubin 17.8 mg/dL, alkaline phosphatase 497 U/L, and leukocytosis with a total count of 12,800 cells/µL. Tumor marker assessment showed CEA 2.3 ng/mL and CA 19-9 of 59.40 U/mL. Multislice contrast-enhanced CT of the abdomen demonstrated an ill-defined 2.3 × 1.4 cm enhancing lesion involving the confluence of the right and left hepatic ducts and the common bile duct, with bilateral intrahepatic biliary dilatation measuring 10.6 mm. The distal common bile duct appeared normal. The gallbladder was contracted.

Discussion: This case highlights the typical yet often delayed presentation of perihilar cholangiocarcinoma. The combination of progressive jaundice, cholestatic liver function abnormalities, and characteristic imaging features supported the diagnosis. Although tumor marker elevation was mild, CA 19-9 can be variably increased in obstructive states. Early biliary decompression remains essential to improve hepatic function, facilitate subsequent therapy, and reduce morbidity. Multidisciplinary input is crucial due to the complex anatomical involvement and limited resectability rates in hilar tumors.

Conclusion: Perihilar cholangiocarcinoma should be considered in elderly patients presenting with progressive obstructive jaundice. Prompt evaluation, early biliary drainage, and coordinated multidisciplinary care are essential for optimal management and improved outcomes.

KEYWORDS: Case Presentation, Investigations, Management, & Discussion

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INTRODUCTION

Cholangiocarcinoma is an uncommon but highly aggressive malignancy arising from the biliary epithelium and represents the second most common primary hepatobiliary cancer worldwide. Its incidence has shown a gradual rise globally, partly attributed to improved diagnostic capabilities and a growing burden of risk factors such as chronic biliary inflammation, hepatolithiasis, primary sclerosing cholangitis, liver fluke infection, and metabolic disorders **(1,3,7)**. Perihilar cholangiocarcinoma, also known as Klatskin tumor, accounts for nearly 50–60% of all cholangiocarcinoma cases and frequently presents late due to its insidious onset and anatomically concealed location within the biliary confluence **(2,5)**.

Obstructive jaundice is the most common initial clinical presentation, often accompanied by pruritus, pale stools, dark urine, and cholestatic biochemical abnormalities. These features generally reflect tumor-related obstruction of the biliary tract rather than hepatic parenchymal dysfunction **(9)**. Despite advancements in imaging modalities and tumor marker assays, early diagnosis remains challenging due to the nonspecific nature of symptoms and overlapping patterns with benign biliary obstruction **(6,8)**. Here, we present a case of a 67-year-old male who developed progressive jaundice and pruritus, later diagnosed as suspected perihilar cholangiocarcinoma. This case highlights the importance of early evaluation, appropriate biliary drainage, and timely referral for multidisciplinary oncologic decision-making.

CASE PRESENTATION

A 67-year-old male presented to the hospital with a 20-day history of progressive yellowish discoloration of the eyes and skin. The jaundice had gradually worsened, and over the past week he developed diffuse itching severe enough to disturb sleep. He also reported an irregular sleep pattern for the same duration owing to nocturnal pruritus. He denied fever, abdominal pain, constipation, vomiting, changes in appetite, or recent weight loss. There was no history of pale stools or dark urine reported at the time of presentation.

The patient was a known case of long-standing systemic hypertension for which he had been taking telmisartan 40 mg once daily and a single daily dose of CPM. He had no documented history of chronic liver disease, gallstone disease, alcohol abuse, viral hepatitis, or prior hepatobiliary surgery. His family and social history were non-contributory.

On examination, he was icteric with excoriation marks over the extremities suggestive of cholestatic pruritus. There was no evidence of fever, abdominal tenderness, hepatosplenomegaly, or ascites. The cardiovascular and respiratory systems were unremarkable, and neurological examination revealed no signs of hepatic encephalopathy. Based on clinical presentation, the patient was initially evaluated for obstructive jaundice.

2.1 Investigations

Initial laboratory investigations revealed significantly deranged liver function tests. The total bilirubin level was markedly elevated at 26.3 mg/dL, with direct bilirubin at 17.8 mg/dL, indicating predominantly conjugated hyperbilirubinemia. Alkaline phosphatase was raised at 497 U/L, consistent with cholestasis. Complete blood count showed a total leukocyte count of 12,800/µL, suggestive of mild inflammatory response but without evidence of overt infection. Coagulation parameters were not detailed initially, although vitamin K was administered prophylactically given the severe cholestasis.

Table 1. Laboratory Profile on Admission

Investigation	Result	Interpretation
Total bilirubin	26.3 mg/dL	Markedly elevated
Direct bilirubin	17.8 mg/dL	Conjugated hyperbilirubinemia
Alkaline phosphatase	497 U/L	Cholestatic elevation
Total leukocyte count	12,800/µL	Mild leucocytosis
CEA	2.3 ng/mL	Within normal limits
CA 19-9	59.40 U/mL	Mildly elevated

Serum tumor markers were obtained to further delineate the etiology of obstruction. Carcinoembryonic antigen (CEA) was 2.3 ng/mL, within normal limits, whereas CA 19-9 was moderately elevated at 59.40 U/mL. Although CA 19-9 elevation may occur in both benign and malignant biliary obstruction, the presence of progressive jaundice and cholestasis prompted advanced imaging.

A multislice contrast-enhanced CT scan of the abdomen revealed a normal-sized liver with homogeneous parenchymal enhancement. However, an ill-defined enhancing lesion measuring approximately 2.3 × 1.4 cm was identified at the confluence of the right and left hepatic ducts and the proximal common bile duct. This lesion was associated with bilateral intrahepatic biliary duct dilatation, with the dilated segment measuring up to 10.6 mm. The distal common bile duct was normal in caliber at 4 mm, and the gallbladder appeared contracted. These findings were highly suggestive of a hilar obstructive lesion, most likely perihilar cholangiocarcinoma.

An MRCP was subsequently performed for further characterization. The study demonstrated a T1 hypointense and predominantly T2 hyperintense ill-defined diffusion-restricting lesion measuring approximately 2.3 × 1.8 cm at the hilar region. There was severe dilatation of both right and left hepatic ducts as well as intrahepatic biliary radicles, with non-visualization of the ductal confluence. An enlarged periportal lymph node measuring 1.3 × 1.2 cm was also noted. No significant atrophy of the liver lobes was observed, and the scan showed no ascites or features of cholangitis. The pancreas, spleen, and kidneys were normal. The radiologist's impression was strongly suggestive of a mass-forming perihilar cholangiocarcinoma, and PET-CT with histopathological evaluation (HPE) was recommended.

Table 2. MRI–MRCP Findings

Parameter	Result
Lesion characteristics	T1 hypointense, T2 hyperintense, diffusion-restricting
Lesion size	2.3 × 1.8 cm
Location	Hilar region
Right hepatic duct	Dilated (13.2 mm)
Left hepatic duct	Dilated (17.1 mm)
Intrahepatic radicles	Severely dilated
Confluence	Not visualized
Lymph nodes	Enlarged periportal node (1.3 × 1.2 cm)
Ascites	Absent
Adjacent organs	Pancreas, spleen, kidneys normal
Radiologic impression	Mass-forming perihilar cholangiocarcinoma

2.2 Management

Upon admission, the patient was initially stabilized with intravenous normal saline and dextrose normal saline to maintain hydration and correct any electrolyte disturbances. On the second day of hospitalization, he was started on intravenous cefotaxime 1 g and metronidazole thrice daily as prophylaxis against ascending cholangitis. Additional supportive medications included intravenous ranitidine and ondansetron to prevent stress-related mucosal irritation and nausea.

Given the severe hyperbilirubinemia and risk of coagulopathy, the patient was administered vitamin K 10 mg intravenously once daily. To alleviate cholestatic symptoms, ursodeoxycholic acid (UDCA) 300 mg twice daily was prescribed. Lactulose substitutes and bowel-cleansing agents such as bisacodyl and Dulcolax were given to prevent hepatic encephalopathy, while rifaximin 550 mg was administered for ammonia-lowering effects. Vitamin A supplementation was included to counteract fat-soluble vitamin deficiencies associated with obstructive jaundice.

After reviewing the imaging results, the surgical oncologist emphasized the need for urgent biliary drainage due to severe obstructive jaundice and the impending risk of cholangitis, hepatic dysfunction, and the inability to initiate systemic anticancer therapy with such elevated bilirubin levels. The patient was advised to undergo either percutaneous transhepatic biliary drainage (PTBD) or endoscopic stenting at the earliest opportunity to decompress the biliary system. Following decompression, a treatment plan involving chemotherapy and further oncologic evaluation was recommended. The patient was subsequently referred to the medical oncologist for systemic therapy planning, with PET-CT and tissue diagnosis scheduled as the next steps in management.

DISCUSSION

Perihilar cholangiocarcinoma is frequently diagnosed at an advanced stage owing to its silent progression and delayed onset of clinically apparent biliary obstruction. The patient's presentation with jaundice and pruritus over several weeks, coupled with significant cholestatic liver enzyme elevation, was consistent with malignant obstructive jaundice—a hallmark of cholangiocarcinoma **(3,9)**. His imaging findings of an ill-defined enhancing lesion at the confluence of the right and left hepatic ducts with intrahepatic biliary dilatation further supported the diagnosis, aligning with classic radiologic characteristics described in current guidelines **(2,10)**.

Tumor markers such as CA 19-9 may support the diagnostic process but lack absolute specificity. The mild elevation in CA 19-9 observed in this patient is a common but nonspecific finding, as cholestasis itself may elevate this marker **(5,7)**. Thus, integration of imaging, biochemical testing, and clinical presentation remains essential.

Management of perihilar cholangiocarcinoma is complex and requires a coordinated multidisciplinary approach. Early biliary drainage is often required to relieve cholestasis, optimize liver function, and prepare the patient for systemic therapy or potential surgical resection. This step is particularly emphasized in expert consensus recommendations for hilar cholangiocarcinoma **(10)**. In this case, referral to both surgical and medical oncology enabled prompt planning for biliary stenting or percutaneous transhepatic biliary drainage (PTBD), followed by evaluation for chemotherapy.

Despite therapeutic advances, outcomes for perihilar cholangiocarcinoma remain poor, with resection offering the best chance for long-term survival but being feasible only in selected patients with localized disease **(4,8)**. The presented case underscores the need for early recognition of malignant obstructive jaundice and rapid progression to definitive diagnostic and therapeutic interventions to improve clinical outcomes.

CONCLUSION

This case highlights the importance of promptly evaluating progressive jaundice and recognizing its potential malignant causes. Early imaging and multidisciplinary intervention are vital for timely diagnosis and optimal management of perihilar cholangiocarcinoma. The patient described here presented with classical features of obstructive jaundice, and detailed imaging established the presence of a hilar mass consistent with cholangiocarcinoma. Appropriate stabilization, biliary drainage, and

oncologic referral formed the foundation of his management plan. Continued follow-up and histopathological confirmation remain essential to guide definitive therapy.

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