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**Cardiovascular Reanimation** 

# Persistent Postoperative Hyperbilirubinemia Revealing Pancreatic Adenocarcinoma: A Case Report

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Abstract Case Report

Background: Postoperative hyperbilirubinemia (PHB) is a relatively frequent complication after cardiac surgery, typically transient and multifactorial. Persistent and progressive forms, however, warrant further investigation to rule out serious underlying pathology. Case Presentation: We report the case of an 82-year-old North African woman who developed severe and persistent hyperbilirubinemia following double valve replacement surgery. Initial investigations pointed to common causes such as hemolysis, hepatic congestion, and sepsis, all of which were addressed through appropriate therapeutic measures. Despite these interventions, bilirubin levels continued to rise, with a predominance of the unconjugated fraction. A biliary Magnetic Resonance Imaging (MRI) revealed dilation of the Wirsung duct and intra- and extrahepatic bile ducts, raising suspicion of a pancreatic head tumor or intraductal papillary mucinous neoplasm (IPMN). The patient was subsequently referred to gastroenterology for further evaluation which confirmed a diagnosis of pancreatic adenocarcinoma. This delayed but critical finding underscored the importance of a comprehensive diagnostic approach in atypical cases of postoperative jaundice. Conclusion: This case illustrates the complexity of diagnosing and managing postoperative hyperbilirubinemia in cardiac surgery patients. It highlights the need for high clinical vigilance, structured differential diagnosis, and timely imaging when bilirubin levels remain elevated despite resolution of common perioperative complications.

**Keywords**: Postoperative hyperbilirubinemia, Cardiac surgery complications, Double valve replacement, Hemolysis, Hepatic congestion, Pancreatic neoplasm, Biliary obstruction.

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### BACKGROUND

Postoperative hyperbilirubinemia (PHB) is a known but often underestimated complication following cardiac surgery, particularly in procedures involving cardiopulmonary bypass (CPB) [1]. While mild and transient forms are relatively common, affecting up to 35% of patients, persistent or worsening jaundice may reflect underlying pathology requiring urgent investigation. The pathophysiology of PHB is multifactorial, involving hemolysis, hepatic congestion, ischemia, systemic inflammation, and drug-induced liver injury. Early recognition and differentiation between benign postoperative changes and serious hepatic or biliary conditions are essential for guiding management and improving outcomes [2,3].

We present a case of an elderly north African patient who developed severe, mixed-pattern hyperbilirubinemia after double valve replacement surgery. Despite correction of common perioperative complications, bilirubin levels continued to rise, ultimately revealing a suspected pancreatic lesion. This case illustrates the diagnostic and management challenges posed by persistent PHB and highlights the importance of multidisciplinary evaluation when routine causes are excluded.

## **CASE PRESENTATION**

An 82-year-old North African woman with a history of progressive dyspnea presented for evaluation. Her medical history included bilateral cataract surgery, recurrent tonsillitis, and a previous urinary tract infection treated with ciprofloxacin. She reported worsening

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exertional dyspnea over 10 years, eventually occurring at rest and accompanied by orthopnea. There was no personal or familial history of liver disease, jaundice, or toxin exposure.

Physical examination revealed normal conjunctivae, a regular heart rate of 64 bpm, blood pressure of 130/50 mmHg, and audible systolic and diastolic murmurs suggestive of Aortic stenosis and mitral stenosis There were no signs of right heart failure.

The electrocardiogram showed sinus rhythm, left atrial hypertrophy, incomplete left bundle branch block (LBBB), and T wave inversions in inferior leads.

Transthoracic echocardiography (TTE) revealed severe aortic stenosis (valve area 0.6 cm², Vmax

5 m/s, mean gradient 62 mmHg), moderate aortic regurgitation, and tight mitral stenosis (valve area 1.2 cm², mean gradient 13 mmHg). Left ventricular function was preserved (LVEF 60%). The atria were dilated, the right ventricle was of normal size with preserved function, and mild tricuspid regurgitation was noted with signs of pulmonary hypertension. The estimated STS-PROM score was 6.7%, indicating high surgical risk due to advanced age, symptomatic multivalvular disease, anemia, and moderate pulmonary hypertension.

Due to lack of insurance coverage for TAVI, the heart team recommended double valve replacement with bioprotheses via open-heart surgery. Preoperative labs **Table 1** were notable for hyponatremia (Na 118 mmol/L) and elevated C-reactive protein (26 mg/L).

Table 1: Preoperative laboratory data

Test	Result
Na	118 mmol/l
K	4.3 mol/l
Urea	0.49 g/l
Creatinine	9.4 mmol/l
CRP	26 mg/l
PCT	0.05 ng/ml
TP	87 %
TCA	30/32
Viral serology	Negative

Under general anesthesia with fentanyl, rocuronium, and propofol, the patient underwent median sternotomy. A 25 mm bioprosthesis was implanted in the mitral position and a 19 mm in the aortic position. Total cardiopulmonary bypass and aortic cross-clamp times were 2 hours 50 minutes and 2 hours 30 minutes, respectively. Two units of red blood cells were transfused intraoperatively.

Postoperatively, mild bleeding was managed with protamine, tranexamic acid, and one additional transfusion. The patient remained hemodynamically stable in the cardiac ICU. On postoperative day (POD) 1, she developed acute kidney injury (creatinine 29.8 mmol/L). On POD 2, jaundice appeared with elevated bilirubin (85 mg/L, predominantly unconjugated), and transaminases (AST 577 U/L, ALT 303 U/L). Central venous pressure was elevated (20 mmHg), suggesting hepatic congestion. **Table 2** 

Table 2: Post operative laboratory data

Test	Result
Urea	1.18 g/l
Creatinine	29.8 mmol/l
Hemoglobin	10.5 g/dl
Total bilirubin	85 mg/l
Conjugated bilirubin	30 mg/
Unconjugated bilirubin	55 mg/l
ASAT	577 UI/L
ALAT	303 UI/L

High-dose intravenous furosemide led to improvement in renal function and volume overload. However, bilirubin continued to rise (355 mg/L, predominantly unconjugated), despite hemodynamic stabilization. Abdominal ultrasound showed vesicular sludge without biliary dilation or gallstones.

Laboratory workup suggested hemolysis: anemia (Hb 8.1 g/dL), elevated LDH (1200 U/L), low-normal haptoglobin (0.38 g/L), and schistocytes on blood smear—consistent with postcardiopulmonary bypass hemolysis. **Table 3** 

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Table 5. I onow up laboratory investigations			
Test	Result		
Urea	0.5 g/l)		
Creatinine	5.2 mmol/l		
Total bilirubin	355 mg/l		
Conjugated bilirubin	96 mg/l		
Unconjugated bilirubin	259 mg/l		
GGT	154 UI/L		
Hemoglobin	8.1 g/dl		
Haptoglobin	0.38 g/l (range 0.38-2)		
LDH	1200		
ASAT	98 UI/L		
ALAT	91 UI/L		
Reticulocyte count	54000 (1 %)		
Blood smear	Schizocytes (normal after CPB)		
PCT	0.6 ng/ml		
TP	20%		

Repeat echocardiography confirmed normal mitral prosthesis function (mean gradient 5 mmHg), but moderate aortic prosthetic regurgitation and new right ventricular dysfunction. Mild tricuspid regurgitation persisted.

Sepsis was initially suspected due to elevated procalcitonin (16 ng/mL), but resolved after empirical imipenem therapy and normalization of PCT (0.6 ng/mL). Hepatotoxic drugs were discontinued.

Due to persistent cholestasis and hyperbilirubinemia, magnetic resonance cholangiopancreatography (MRCP) was performed **Figure 1-2**. It revealed dilated intra- and extrahepatic bile ducts and a prominent Wirsung duct (10 mm), suggesting a pancreatic head lesion or intraductal papillary mucinous neoplasm (IPMN). The patient was referred to the gastroenterology department for further assessment.

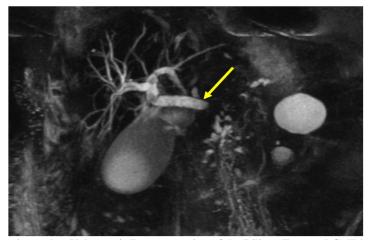


Figure 1: 3D MR Cholangiography: Volumetric Reconstruction of the Biliary Tree and Gallbladder showed intra- and extrahepatic bile duct dilation and an enlarged Wirsung duct

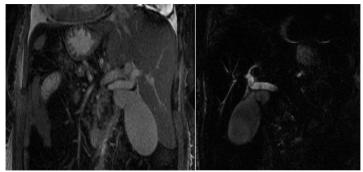


Figure 2: Multisequence MRI Evaluation of the Biliary Tree: Coronal FIESTA and T2

After cardiac stabilization, endoscopic ultrasound (EUS) confirmed the presence of a hypoechoic, irregular mass in the pancreatic head with associated main duct dilatation. Fineneedle aspiration (FNA) biopsy was performed. Cytological analysis revealed clusters of atypical epithelial cells with high nuclear-cytoplasmic ratio and prominent nucleoli, consistent with pancreatic adenocarcinoma. CA 19-9 levels were also elevated (780 U/mL). The diagnosis of pancreatic head carcinoma was established. A multidisciplinary team discussion involving gastroenterology, oncology, and cardiology concluded that curative surgery was not feasible due to the patient's frailty and recent cardiac surgery. Palliative biliary drainage and oncologic management were planned.

## **DISCUSSION**

This case underscores the intricate interplay between severe valvular heart disease and the emergence of postoperative complications, notably hyperbilirubinemia, in elderly patients undergoing cardiac surgery.

Postoperative hyperbilirubinemia is a well-documented phenomenon following cardiac surgeries involving cardiopulmonary bypass (CPB) [4]. Its incidence varies widely, reported between 10% and 40% [5], depending on patient demographics and surgical complexity [2]. The etiology is multifactorial, encompassing hemolysis due to CPB, hepatic ischemia from low cardiac output, systemic inflammatory responses, and pre-existing hepatic congestion

In our patient, the development of hyperbilirubinemia post-surgery was initially attributed to hemolysis and hepatic congestion, common in the postoperative setting. However, the persistence and progression of cholestasis, despite hemodynamic stabilization, prompted further investigation. Magnetic resonance cholangiopancreatography (MRCP) revealed a pancreatic head mass, later confirmed as adenocarcinoma via endoscopic ultrasound and biopsy.

The incidental finding of a pancreatic malignancy in the postoperative period is rare but has been documented. Such discoveries often complicate the clinical picture, as the symptoms of malignancy can mimic or exacerbate postoperative complications. In this case, the pancreatic tumor likely contributed to the persistent hyperbilirubinemia, highlighting the importance of considering alternative diagnoses when standard postoperative recovery does not proceed as expected.

The management of such patients requires a multidisciplinary approach, balancing the risks and benefits of further interventions in the context of recent major surgery and the patient's overall prognosis [6,7]. In our case, the patient's frailty and recent cardiac surgery

precluded curative treatment for the pancreatic cancer, shifting the focus to palliative care.

This case emphasizes the need for vigilance in the postoperative period, especially when complications persist beyond the expected timeframe. Early imaging and interdisciplinary collaboration are crucial in identifying and managing unexpected pathologies that may significantly impact patient outcomes.

### Conclusion

This case illustrates the diagnostic complexity of persistent postoperative hyperbilirubinemia following cardiac surgery. While common causes such as hemolysis, hepatic congestion, and sepsis were initially managed, the persistence and progression of jaundice revealed an underlying obstructive biliary process, likely related to a pancreatic head Adenocarcinoma. This highlights the importance of maintaining diagnostic vigilance and pursuing advanced imaging when standard causes are excluded. A structured, multidisciplinary approach is essential to identify less common etiologies and improve patient outcomes in complex postoperative scenarios.

#### List of abbreviations

**AKI:** acute kidney injury **CPB:** cardiopulmonary bypass **CRP:**C-Reactive Protein

IPMN: papillary mucinous neoplasm. LBBB: Left bundle branch block MRI: Magnetic resonance imaging

**PCT**: Procalcitonin

PHB: Postoperative hyperbilirubinemia

**POD**: postoperative day

STS-PROM: Society of Thoracic Surgeons Predicted

Risk of Mortality

TTE: Transthoracic echocardiography

## **Consent for publication**

Written informed consent was obtained from the patient for publication of this case report and any accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of this journal.

#### Ethics approval and consent to participate

Not applicable.

## Availability of data and materials

All data generated or analyzed during this study are included in this published article.

#### **Authors contributions**

KZ conceived and designed the case report, conducted the literature review, and wrote the initial draft of the manuscript.

LH and OM contributed to the collection and interpretation of clinical and imaging data.

OK and MT participated in patient follow-up and contributed to manuscript editing.

SE and FL assisted with echocardiographic image analysis and figure preparation.

Prof. AB and Prof. SM supervised the clinical management of the case and critically revised the manuscript for intellectual content.

All authors read and approved the final manuscript.

#### **Competing interests**

The authors declare that they have no competing interests.

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