TMJ
Case Report
Published by OJS
Doi: 10.4314/tmj.v32i3.438.g277
OPEN ACCESS JOURNAL

Obstructive Jaundice in a Patient with Portal Hypertension: A Rare Presentation from Northern Tanzania

Jay Lodhia[1,2]*, Elifuraha Mkwizu[2,3], Adnan Sadiq[2,4], David Msuya[1,2]

[1]Department of General Surgery, Kilimanjaro Christian Medical Center, Moshi, Tanzania
[2]Kilimanjaro Christian Medical University College, Moshi Tanzania
[3]Department of Internal Medicine, Kilimanjaro Christian Medical Center, Moshi, Tanzania
[4]Department of Radiology, Kilimanjaro Christian Medical Center, Moshi, Tanzania

***Corresponding author:**

Dr. Jay Lodhia

Kilimanjaro Christian Medical Center

P. O. Box 3010

Moshi, Tanzania

Email: jaylodhia06@gmail.com

TMJ
Case Report
Published by OJS
Doi: 10.4314/tmj.v32i3.438.g277
OPEN ACCESS JOURNAL

Abstract

Introduction

Obstructive jaundice is defined as a condition occurring due to a block in the pathway between the site of conjugation of bile in liver cells and the entry of bile into the duodenum through the ampulla. The block may be intrahepatic or extrahepatic in the bile duct. Majority of the patients with portal biliopathy are asymptomatic and only about 20% presents with symptoms hence management is limited to this group and is dictated by the clinical manifestations and complications.

Case presentation

We are reporting a case of female patient aged 66 years who had features of obstructive jaundice. Abdominal CT-scan revealed obstructive biliopathy due to compression of the extrahepatic common bile duct by dilated periportal collaterals. She underwent bypass cholecystojejunostomy with Braun's anastomosis surgery with no morbidities and her jaundice resolved clinically.

Conclusion

Jaundice due to extra-hepatic biliary obstruction may arise as a result of diverse clinical conditions ranging from those commonly encountered during routine clinical practice to the ones which are rarely seen.

Keywords: *Obstructive Jaundice, Portal Hypertension, Tanzania.*

Published by OJS
Doi: 10.4314/tmj.v32i3.438.g277

Introduction

Portal biliopathy is a condition described as abnormalities arising anywhere in the biliary tree as a consequence of portal hypertension (1). These include bile duct strictures, ischemic injuries, choledochal varices, and they are mostly associated with portal hypertension from portal vein thrombosis (1,2). However, in patients with these anomalies, obstructive jaundice is rarely seen (3).

Many kinds of varices are observed in patients with extra-hepatic portal vein obstruction (EHPVO) due to the extensive network of collaterals involving the paracholecystic, paracholedochal and pancreaticoduodenal veins, of which contribute to the development of portal biliopathy (3). Other less common causes are liver cirrhosis, primary biliary cirrhosis and Budd-Chiari syndrome (3). It is critical to diagnose the extent of portal biliopathy and its consequences in order to plan the correct line of management. Herein we present an uncommon cause of portal biliopathy caused by dilated portal veins from portal hypertension.

Case presentation

A 66-year-old female presented with abdominal pain for two months which had worsened in the last four days. The pain was more pronounced on the right upper quadrant radiating to the right lower quadrant, associated with loss of appetite and vomiting which was non-bilious, non-bloody and non-projectile in nature and character. She also reported mild abdominal distention but denied history of constipation along the course of illness. She is not known to have hypertension or diabetic mellitus, no history of upper or lower gastrointestinal bleeding or any drug or food allergies.

On initial examination, her blood pressure, heart rate and saturation were within normal range, had mild scleral jaundice, conjunctival pallor and no lower limb edema. Her abdomen was slightly distended globally, with tender hepato-splenomegaly otherwise no dilated veins or petechiae (Figure 1).

Her complete blood count revealed borderline anaemia with a hemoglobin level of 10.3 g/dl and a normal platelet count of 184 x 10^9 with normal WBCs and differentials. Rapid test panels for hepatitis B surface antigen and Hepatitis C were negative. Her international normalized ratio was elevated to 1.45 and alpha fetoprotein of 0.50. Her serum creatinine level was 48 µmol/L, conjugated bilirubin of 36.16 µmol/L, raised total bilirubin of 52.41

Published by OJS
Doi: 10.4314/tmj.v32i3.438.g277

µmol/L, albumin of 32.97 g/l, normal aspartate transaminase and alanine aminotransferase of 22.40 U/l and 20.18 U/l respectively.

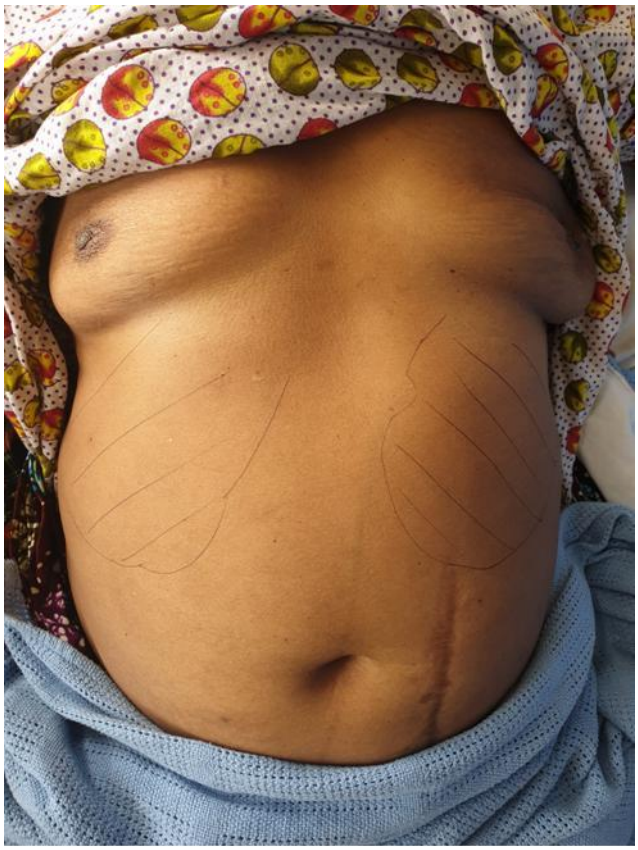
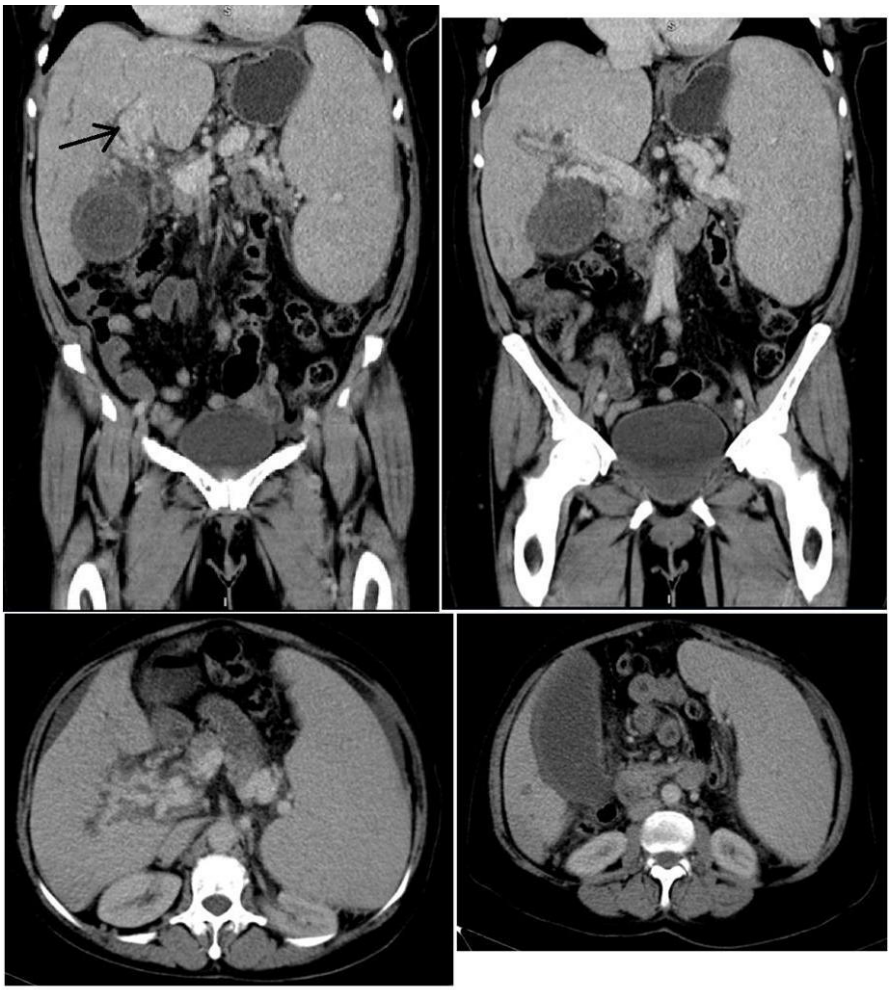


Figure 1: Photograph showing hepato-splenomegaly

Esophagogastroscopy was done that showed grade-A esophagitis due to gastro-esophageal reflux disease and no varices were seen.

Computed-tomography of her abdomen showed features of mild obstructive biliopathy secondary to compression of the extrahepatic common bile duct and common hepatic duct by dilated periportal collaterals and splenomegalyin the portahepatis and splenomegaly of 20 cm (AP) x 10 cm (T) x22 cm (cc) and no radiologic evidence of liver cirrhosis was noted (Figure 2).

Published by OJS
Doi: 10.4314/tmj.v32i3.438.g277



CT-scan images showing multiple dilated periportal and perisplenic collaterals with associated dilatation of the portal and splenic veins. There is associated mild dilatation of the intrahepatic biliary ducts due to obstruction of the common bile duct by the dilated periportal collaterals (arrow)

She was then scheduled for a planned explorative laparotomy, where intra-operatively the gall bladder, common bile duct (CBD) and cystic ducts were dilated and multiple dilated collateral vessels were seen. The pancreas was normal with no masses and no enlarged lymph nodes encountered. Cholecystojejunostomy with Braun's anastomosis was done to bypass bile obstruction at the CBD and prevent cholecystitis / cholangitis respectively. Post operatively, the patient fared well and was discharged on day two with no complications. She was then reviewed at the surgical outpatient clinic at monthly intervals for three months whereby a control abdominal ultrasonography was done at 2 months which only revealed moderately enlarged spleen of 16 centimeters homogenous in texture, and clinically her jaundice had resolved completely. She was advised for regular monitoring and

Published by OJS
Doi: 10.4314/tmj.v32i3.438.g277

esophagogastroscopy to rule out varices. She was counseled for definitive management if at all does appear for which she would be given referral to the hepatobiliary surgeons at the national hospital.

Discussion

The term “biliopathy” originates from a combination of Latin (“bilis” meaning bile) and Greek (“pathos” meaning disease) respectively (4). Portal biliopathy refers to a term used to represent various biliary abnormalities such as stenosis or dilatation of bile duct in patients with portal hypertension. These vessels cause obstructive jaundice but it is not clearly known which one in particular (3).

Chawla et al reported that in all age groups 30% of the causes of portal hypertension is extrahepatic portal venous hypertension. In children the causes are omphalitis, umbilical vein catheterization and intra-abdominal sepsis while in adults the causes are myeloproliferative disorders, local tumor invasion or chronic pancreatitis. The authors continue to state that in more than half of the cases with extrahepatic portal vein obstruction the causes are unknown (5).

Julianov et al presented a rather uncommon cause of obstructive jaundice caused by hepatic artery aneurysm. They reported raised bilirubin levels in a 76-year-old man, where a CT-angiogram revealed a 64mm large hepatic artery aneurysm and was confirmed by endoscopic retrograde cholangiography being the cause of jaundice (6). Another rare manifestation of obstructive jaundice caused by tuberculous lymphadenitis was reported by Baik et al. They reported of a 30-year-old man who developed jaundice after completion of anti-tuberculosis regime. Abdominal computed tomography and cholangiogram showed a narrowing point in the common bile duct due to enlarged lymph nodes, of which biopsy was done and concluded tuberculous lymphadenitis. The patient was managed surgically by Roux-en-Y hepaticojejunostomy and post operatively anti-tuberculosis drugs (7). uSimilarly, in the index case, the patient was managed by surgery to bypass the compressed CBD. Tumor of head of pancreas is the commonest encountered cause of obstructive jaundice in our settling where the patients present in advanced stage hence undergo palliative “triple” bypass (gastrojejunostomy, cholecystojejunostomy and Braun’s anastomosis) surgery.

The etiology of EHPVO in children is idiopathic in about 50% of the cases with morbidity being variceal bleeding, portal biliopathy with obstructive jaundice and hypersplenism from splenomegaly. Miraglia et al outlined some interventional radiologic procedures and surgical

shunting to manage EHPVO and its complications (8). The authors reported of inserting a 'Meso-Rex' shunt in a three-year-old child with EHPVO as a complication due to umbilical vein cannulation with good outcome and patency of the biliary tract. Another case was reported of a ten-year-old with partial liver transplant due to biliary atresia and meso-caval porto-systemic shunt was performed with good outcome. They also stated that the use of multidetector computed tomography (MDCT) confirms the diagnosis and aids plan in the management surgical shunt creation (8).

Franceschet et al concluded that management of portal biliopathy requires combined interventions both on the vascular and biliary system aiming to decompress the portal system. Liver transplant can be considered in patients with liver cirrhosis (2). The authors also continued to mention that only a few will be symptomatic and hence will require management (2). Unlike our patient, liver transplant was not considered as she did not have liver cirrhosis, and due to lack of resources and economic constrains, endoscopic and shunting methods of intervention were not done.

From a different review by Cardoso et al. the authors state that currently therapy is not indicated for asymptomatic patients with normal liver functions, like the index case, otherwise portosystemic shunting surgery is the modality of choice (9).

Conclusion

Obstructive jaundice in association with portal hypertension is an uncommon finding and management is guided by the clinical manifestations and extent of disease. Many patients with portal biliopathy need combined vascular and biliary system interventions. Liver transplant can be considered in patients with secondary liver cirrhosis. However, in those with symptoms, management should be tailored individually.

Consent

Written informed consent was obtained from the patient for publication for this case report; additionally, accompanying images have been censored to ensure that the patient cannot be identified. A copy of the consent is available on record.

Conflict of interest

The authors have no conflict of interests to disclose.

Published by OJS
Doi: 10.4314/tmj.v32i3.438.g277

Authors' contributions

JL conceptualized and prepared the manuscript. DM performed the surgery and along with JL reviewed patient's medical records. AS prepared and reported the radiology films and EM performed the endoscopy. All authors have read and approved the final manuscript.

Acknowledgement

The authors would like to thank the patient for permission for the information to be shared for further learning purposes.

Abbreviations

CBD	Common Bile Duct
WBCs	White Blood Cells
EHPVO	Extra-Hepatic Portal Vein Obstruction
MDCT	Multidetector Computed Tomography

Published by OJS
Doi: 10.4314/tmj.v32i3.438.g277

References

1. Khan MR, Tariq J, Raza R, Effendi MS. **Portal hypertensive biliopathy: review of pathophysiology and management.** Tropical gastroenterology. 2012 Nov 2;33(3):173-8.
2. Franceschet I, Zanetto A, Ferrarese A, Burra P, Senzolo M. **Therapeutic approaches for portal biliopathy: A systematic review.** World journal of gastroenterology. 2016 Dec 7;22(45):9909.
3. Takagi T, Irisawa A, Shibukawa G, Hikichi T, Obara K, Ohira H. **Intraductal ultrasonographic anatomy of biliary varices in patients with portal hypertension**. Endoscopic ultrasound. 2015 Jan;4(1):44.
4. Harmanci O, Bayraktar Y. **How can portal vein cavernous transformation cause chronic incomplete biliary obstruction?** World journal of gastroenterology: WJG. 2012 Jul 14;18(26):3375.
5. Chawla A, Maheshwari M, Parmar H. **Obstructive jaundice in a case of portal hypertension.** The British journal of radiology. 2003 Sep;76(909):667-9.
6. Julianov A, Georgiev Y. **Hepatic artery aneurysm causing obstructive jaundice.** Quantitative imaging in medicine and surgery. 2014 Aug;4(4):294.
7. Baik SJ, Yoo K, Kim TH, Moon IH, Cho MS. **A case of obstructive jaundice caused by tuberculous lymphadenitis: A literature review**. Clinical and molecular hepatology. 2014 Jun;20(2):208.
8. Miraglia R, Maruzzelli L, Cortis K, Caruso S, Luca A. **Interventional radiology procedures in pediatric patients with extra-hepatic portal vein obstruction and complications of portal hypertension.** European Congress of Radiology 2014.
9. Cardoso R, Casela A, Lopes S, Agostinho C, Souto P, Camacho E, Almeida N, Mendes S, Gomes D, Sofia C. **Portal Hypertensive Biliopathy: An Infrequent Cause of Biliary Obstruction.** GE Portuguese journal of gastroenterology. 2015 Mar 1;22(2):65-9.