

# Bile Duct Stricture – A Sign of things to come?

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**Objective:** The aim of the report is to highlight this difficult to-treat condition hitherto uncommon in our environment and warn of the likelihood of more to come with the rising incidence of gallstone in our sub region.

**Patient and Method:** A case report of an 18-year-old male undergraduate patient.

**Result:** A successful management with clinical, biochemical and radiological evidence (OTC and tubograms) despite inadequate investigative tool.

**Principal Conclusion:** With the rising incidence of gall stones in our environment, it is imperative that the Trainee Surgeon must be schooled in common biliary surgery and help prevent this dreaded complication (bile duct stricture) and other sequelae of gall bladder surgery. A plea is also made for the provision of essential investigative tool to facilitate management of such cases in recognised centers.

**Key words:** gall Stones; post cholecystectomy bile duct stricture; classification; investigations; hepatico-jejunostomy.

Post-cholecystectomy stricture of the bile duct is a recognized sequela of biliary surgery. This is usually encountered in the literature in this part of the world and rightly regarded as a problem in the West and other parts of the world where gall stones and gall bladder surgery are common<sup>1,2&3</sup>.

This complication is reported in an 18 year-old Nigerian male undergraduate student to warn that this problem does happen here too, though occasionally at the moment. The rising incidence of gall stones<sup>4</sup> and the inevitable surgery that follows can only bring this dreaded complication to our door step. The management dilemma in the face of inadequate investigative tool is highlighted and the trainee surgeon is advised to pay more than a passing attention to its management because this complication though infrequent, is nothing short of a tragedy when it happens and imprecise management is associated with disastrous results.

## Case Report

A.E, an 18 year-old Nigerian male university student was referred on 3/06/03 with a 7-week history of deepening and persistent jaundice, dark urine and pale stool. Patient had a cholecystectomy for cholelithiasis a week earlier and developed post-operative bilious ascites four days later. He was re-explored ten days after the cholecystectomy and the only cause of the leak according to the referral "was a loose ligature on the cystic duct stump which was re-tied". The leak stopped but the jaundice, which was initially a tinge, deepened and became persistent hence, the referral.

Physical examination revealed a chronically ill-looking, wasted, pale and jaundiced boy. There was a Mayo-Robson scar on the abdomen with multiple puckered scars on the right lower quadrant—apparently for drains. There was no tenderness and the liver was not palpable. The clinical diagnosis was post-cholecystectomy bile duct stricture. The abdominal ultrasound done showed dilated intra-hepatic duct radicles. A percutaneous trans-hepatic cholangiogram requested could not be done because there was no appropriate needle for the procedure. ERCP (Endoscopic Retrograde cholangiopancreatography) was also not available. The bilirubinuria and elevated serum bilirubin - total = 37.6mg/dl, conjugated = 28.0mg/dl (Reference range for total bilirubin = 0.2-1.0mg/dl, conjugated = 0-0.4mg/l) and alkaline phosphatase 975i.u/L (Reference range = 95 -279 i.u/L) just confirmed obstructive jaundice.

Patient was prepared for surgery over the next two weeks. He was fully hydrated (intravenously and orally) to ensure an adequate daily urinary output of about two litres. He had glucose, protein and vitamin supplements and was given parenteral vitamin K – 10mg daily.

Three units of fresh whole blood were made available for the operation and he had prophylactic antibiotic cover with a third generation cephalosporin (Ceftriaxone). He had his surgery 18 days following admission.

The intra-operative findings were:

- i. jaundiced intra-abdominal viscera,
- ii. extensive dense adhesions involving both inferior and diaphragmatic surfaces of the liver. The

duodenum as well as the hepatic flexure of the colon were stuck to the porta hepatis. The liver was of normal size.

- iii. Dilated common hepatic duct which ended blindly about one centimeter below the liver at the porta hepatis (Bismuth type 2 stricture or type D-Keulemans *et al*).
- iv. Reactive mesenteric adenopathy.

After adhesionolysis and identification of the stump of the hepatic duct, an on-table-cholangiogram (OTC) was performed (Fig.1).

An end-to-side single layer Roux-en-Y retro-colic hepatico-jejunostomy was performed leaving behind a size 8-feeding tube as a trans-anastomotic stent. Patient made an uneventful recovery. Post-operative tubogram done on the 12<sup>th</sup> day revealed free flow of contrast into a well decompressed biliary radicles (Figs.2 & 3). The drain in Morison's pouch was removed on the 17<sup>th</sup> day and the trans-anastomotic stent on the 27<sup>th</sup> day post-operatively. Patient was discharged home three days later.

Two months thereafter, the jaundice had cleared completely. The total serum bilirubin was 1.3mg/dl. The general health was very good and he then weighed 62kg as opposed to 47kg on admission.



**Fig. 1:** On table cholangioram of the patient (per operative study – showing dilated hepatic duct stump and dilated intra-hepatic radicles—complete obstruction).



**Fig. 2:** Tubogram with 30mls of contrast, showing the upper limb of the Roux-en-Y hepatico-jejunostomy demonstrating good decompression of the biliary tree (12<sup>th</sup> post-operative day).



**Fig. 3:** Tubogram with 40mls of contrast demonstrating same good decompression of the biliary tree and the two limbs of the Roux-en-Y (12<sup>th</sup> post-operative day).

#### Discussion

Post-cholecystectomy stricture of the bile duct is not new in the literature both in the open and laparoscopic surgery for gall stones<sup>3,5,6</sup>. It is however not common here because of the low incidence of gallstones and attendant surgery. The rising incidence of gall stones being reported in our sub-region<sup>4</sup> and the attendant surgery will

bring this complication more and more to the fore in our environment. The common causes of benign bile duct stricture are well enumerated in the literature but most are secondary to gall bladder surgery with or without exploration of the common bile duct<sup>3&7</sup>.

The aetiology or rather, the predisposing factor(s) of the gallstone in this boy was obscure. There was no apparent metabolic, infective, or stasis predisposing factors in this 18 year-old Nigerian boy. His haemoglobin genotype is AA and there is no familial history. There was no indication that the excised gall bladder was subjected to histopathology either.

The management of any bile duct injury is predicated on the class of injury. With the advent of laparoscopic cholecystectomy the incidence of bile duct injury has not only increased, the pattern has also changed. This has led to a new classification of bile duct injury<sup>8</sup> as opposed to the old Bismuth classification<sup>3</sup>. The message in either classification is that an anatomical definition of the bile duct injury is a *sine-qua-non* for definitive diagnosis and management. Without this, the strategy for management and success of same becomes a pot luck affair.

Whilst the diagnosis of bile duct injury was fairly straight forward from the history and physical examination in our patient, there was however no way for us to determine the type of stricture pre-operatively. The best radiological study available to us for confirmation of diagnosis and determination of the type of stricture was the common B-mode ultrasonography. Neither ERCP (Endoscopic Retrograde cholangio-Pancreatography) nor PTC (Percutaneous transhepatic cholangiography) which should have been the lynchpin of diagnosis<sup>3</sup> in our type of patient was available to us. With these constraints, it was therefore difficult to know whether much could be done for the patient. The patient and his relatives were well briefed about this shortcoming. The option of an oversea treatment was foreclosed because of finance. Surgery, which they pleaded for in this circumstance, was therefore more or less like a shot in the dark. The chance was however taken and at surgery, the dense adhesion at the porta hepatis and around the entire liver following two previous explorations was not unexpected. The hepatic duct stump just jutting out from the liver was eventually identified by sharp and blunt dissection. The subsequent OTC (Fig. 1) fortunately confirmed a type 2 Bismuth stricture or type D in the recent classification. A retro-colic Roux-en-Y hepatico-jejunostomy (end-to-side) was performed after the fashion of Voyles and Blumgart<sup>9</sup>. Majority of bile duct injury following laparoscopic cholecystectomy are treated endoscopically<sup>8</sup> but a sizeable percentage of surgeons still prefer open surgery. Besides, some strictures- total ductal obstruction (type D in the new classification) like in our patient, are not amenable to endoscopic technique<sup>7,8&10</sup>.

The three parameters recommended for assessing results<sup>3</sup> are clinical (symptoms), biochemistry (serum bilirubin/Alkaline phosphatase) and radiology

(cholangiography/HIDA scan). Our patient has been symptom-free completely. Serum bilirubin is now normal and Alkaline phosphatase is halved already. The post-operative tubogram (Fig.2 & 3) demonstrated good decompression of the obstructive jaundice. It is pertinent to mention that about 30-40ml of contrast may be needed to obtain a good tubogram and this must be shot up into the liver otherwise most of the contrast will run down the upper limb of the Roux-en-Y loop of jejunum.

Duodenitis and concomitant abdominal pain have been reported frequently in this subset of patients but so far our patient has not complained about this. Re-stricture has also been found in a number of patients<sup>3,7&10</sup>. It is too early to tell in our patient.

The need for adequate radiological support especially PTC, ERCP (and lately probably MRCP) in this kind of hepato-biliary surgery cannot be over-emphasised. Without this back up, decisions are reduced to guess work and surgical intervention a pot luck affair. This case emphasises once again that gall bladder surgery is fraught with danger and is not meant for the occasional surgeon and certainly not for the novice. The predisposing factor(s) for gall stone formation in this 18 year-old Nigerian boy is obscure and intriguing. With the rising incidence of gallstones in the sub-region, the trainee surgeon must be competent in routine open and laparoscopic (where available) cholecystectomy. He must also show more than a passing interest in this devastating though infrequent complication – bile duct injury with resultant stricture. This is a surgical disaster, which is better prevented. It is equally important to appreciate that most gallstones are silent and most will have no symptoms whatever throughout their life times<sup>11</sup>. There must therefore be adequate justifications/indications for operation especially on a silent stone<sup>12</sup>.

With proper training in biliary surgery, the hope is that as gall bladder disease and surgery become common place in our environment, the dreaded complication- bile duct injury with stricture, will not be common place too.

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