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A Case of Iatrogenic Choledochocolic Fistula with Malabsorption

ANDERS LJUNGSTRÖM and IVAR WERNER

From the Department of Internal Medicine, University Hospital, Uppsala, Sweden

ABSTRACT

A case of choledochocolic fistula, unintentionally created by surgery, with no communication between the biliary tract and the intestine proximal to the anastomosis at the hepatic flexure of the colon was studied before and after surgical correction. The patient was almost symptomfree, without diarrhea, but a metabolic study showed severe malabsorption of fat and vitamin K. The malabsorption was completely abolished by corrective surgery.

INTRODUCTION

Internal biliary fistulas are uncommon but not extremely rare. In autopsy material an incidence as high as 0.12–0.22% has been reported and in various series of biliary surgery an incidence of 0.15–5%. More than 90% are enterobiliary fistulas while fistulas to pleura, pericardium, bronchi renal pelvis, arteries etc. are very uncommon (5, 7, 14, 17, 18, 19).

The cause of internal biliary fistulas are in about 90% of the cases calculous biliary tract disease, sometimes peptic ulcer or malignancy, seldom other conditions (5, 7, 18).

The most common type of spontaneous enterobiliary fistula are the cholecystoduodenal, which is said to account for 60–75%, and the cholecystocolic in about 20% of the cases. Fairly common are the cholecystogastric and the choledochoduodenal fistulas, the latter in most cases due to peptic ulcer (5, 7, 21). The choledochocolic fistula seems to be extremely rare, and only a few cases are found in the literature (13).

The case reported here is a patient with a malabsorption syndrome due to a choledochocolostomy unintentionally created by surgery.

CASE REPORT

The patient was a 64-year-old carpenter with a history of duodenal ulcer and periods of gastritis since about 35 years.

For one year the patient had experienced slight symptoms of biliary tract disease, when silent icterus supervened and the patient was admitted to the surgical department. An upper gastrointestinal series showed postulcerous deformation of the duodenal bulb and reflux into the biliary tract, where a defect in the barium was seen. Serum bilirubin and alkaline phosphatase were high, indicating biliary tract obstruction. Hemoglobin and prothrombin index were normal. The jaundice spontaneously subsided and the patient was in a good general condition. Cholecystectomy was performed and a T-tube was inserted into the common bile duct. Because of hypotension during the operation further surgery was postponed. A cholangiography through the T-tube showed contrast defects. Three weeks later a choledocholithotomy was performed without complications. Because of a firm cicatricial stenosis of the common bile duct at the sphincter of Oddi a choledochoduodenostomy was brought about at the same time.

Eight months later the patient was readmitted to the surgical department for checking up on the supposed choledochoduodenal anastomosis. In the meantime the patient had felt quite well. No abdominal pain, fever or jaundice had been noted and no signs of bleeding diathesis. He had had 2–3 fairly bulky stools daily and had not found anything remarkable about his defaecations, no diarrhea. In spite of a good appetite his body weight remained at about 70 kg, although his body weight before the first operation was 76 kg.

Physical examination on admission revealed nothing remarkable except a slow atrial fibrillation, which also had been noted at previous admissions. The hemoglobin was 8.6 g/100 ml, the prothrombin index below 10% and stool specimens gave positive tests for occult blood. Vitamin K was given parenterally for two weeks and the prothrombin index went up to normal within a few days. At the same time stool specimens started to show negative reaction for occult blood and a normal hemoglobin was easily restored. Six weeks later the prothrombin index had once again fallen to 28% and vitamin K medication was repeated with rapid effect on the prothrombin index.

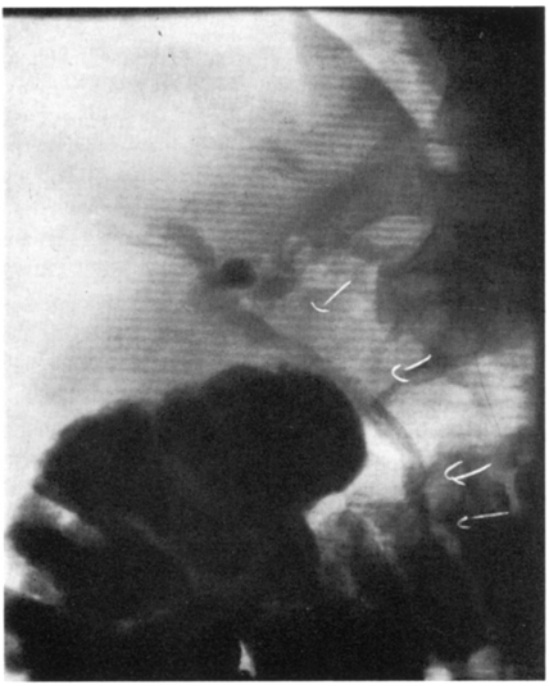

Fig. 1. Barium enema showing passage from the hepatic flexure of the colon to the biliary tract.

Table I. *Absorption and excretion data before and after corrective surgery*

	Before correction	After correction
Fat (diet 75–80 g/day)		
Absorption	10%	90–95%
Stool fat		
Neutral fat	68.0 g/day	6.2 g/day
Free fatty acids	4.3 g/day	0.8 g/day
Absorption of vitamin K	Poor	Normal
Protein (diet 12.4 g N/day)		
N in stool	1.55 g/day	1.53 g/day
N in urine	10.80 g/day	9.30 g/day
Balance	Slightly negative	Positive
Calcium (diet 1030 mg/day)		
Ca in stool	1052 mg/day	954 mg/day
Ca in urine	29–45 mg/day	56–69 mg/day
Balance	Slightly negative	Slightly positive
Excretion of urobilinogen and urobilin		
In stool	0, 35, 48 mg/day	40, 41, 58 mg/day
In urine	0.2–1 mg/day	0.2–1 mg/day
Bilirubin in stool	Positive	Negative

An upper gastrointestinal series showed the biliary tract filled with gas indicating good function in the anastomosis but no barium entering the biliary tract. The bile ducts were not visualized by an excretory cholangiography, but when a barium enema was given they were filled through an anastomosis at the hepatic flexure of the colon (Fig. 1).

It was now evident that a choledochocolic anastomosis had been created by mistake. The patient underwent a metabolic study in the medical department and three months later a new operation was performed, at which the choledochocolostomy was closed and a choledochoduodenostomy was created without complications. At the operation the liver seemed macroscopically normal and nothing pathological except the expected postoperative changes was found. Postoperatively the patient underwent once again a metabolic study in the medical department.

Laboratory examination before the correction of the choledochocolic fistula showed hemoglobin 11.0 g/100 ml, RBC-morphology normal, WBC 6.000 with normal differential, thrombocytes 155.000, reticulocytes 8‰, sedimentation rate 24–10 mm/h, RN 31 mg/100 ml, urinanalysis negative, urobilinogen in urine negative, urobilin in urine trace negative. Total serum bilirubin 0.8 mg/100 ml, thymol 0.1 U, Hanger negative, Takata-Ara negative, alkaline phosphatase 8.3–6.1 King-Armstrong U, serum calcium 9.8–10.0 mg/100 ml, serum phosphorous 3.8–3.9 mg/100 ml, serum cholesterol 173 mg/100 ml, total serum lipoids 755 mg/100 ml (Schoenheimer-Sperry). No changes in the bone structure were seen on X-ray pictures of the lumbal columna, pelvis, left and right humerus and femur. Aspiration with a Miller-Abbot sond in duodenum showed no trace of bile but signs of good activity of trypsine, amylase and lipase.

A study of absorption and excretion of fat, protein and calcium on a balanced diet before and after the corrective operation of the choledochocolic fistula was performed. The results are seen in Table I.

After the corrective operation with creation of a choledochoduodenostomy the patient was in good health with no signs of malabsorption and no symptoms of biliary tract disease except a short episode of fever attacks 6 years later, which were suspected to be due to cholangitis. During the last 4 years of life he suffered from recurrent gastrointestinal haemorrhage from duodenal ulcers and the last year he showed increasing signs of cardiac failure due to mitral insufficiency. He died 10 years after his first operation in cardiac failure with pulmonary oedema. At autopsy the choledochoduodenostomy was in good function. The liver showed no changes except a slight stasis due to circulatory failure. Apart from duodenal ulcers the abdominal organs were normal.

COMMENT

The symptoms of spontaneous enterobiliary fistulas are often unimpressive, i.e. usually the patient only shows symptoms of the disease causing the fistula, e.g. calculous biliary tract disease or peptic ulcer. Often the condition is first sus-

pected on X-ray pictures showing gas in the biliary tract or reflux of barium into the bile ducts from an upper gastrointestinal series or a barium enema, like the case discussed above. Often it is unexpectedly found at operation or autopsy. In singular cases more spectacular complications like gallstone ileus or massive haemorrhage occur (5, 6, 7, 17, 19). In cases of biliary-colic fistulas (mostly cholecystocolic since choledochocolic and other fistulas are extremely rare) weight loss, protracted diarrhea and attacks of cholangitis are sometimes found. Of 32 patients from the Mayo clinic with cholecystocolic fistulas 19 had considerable loss of weight and 2 protracted diarrhea (21), and of 11 cases with biliary-colic fistulas Moreaux found 3 cases with profuse diarrhea (15). Bergner states that biliary-colic fistulas are often accompanied by diarrhea but rarely by cholangitis (5).

The patient described here showed very few signs or symptoms of disease, but laboratory and metabolic studies disclosed a considerable malabsorption of fat and vitamin K, which disappeared completely after surgical reconstruction of the anatomy. The important part in the digestion and absorption of fat and fat soluble vitamins played by the bile in the upper gastrointestinal tract seems to be exercised through several physiological mechanisms, especially micellar formation (20). The substantial increase of almost solely neutral fat in stool and the comparatively insignificant increase of free fatty acids raise the suspicion that the complete absence of bile in the upper gastrointestinal tract leaves the dietary fat essentially undissolved and thus also fairly inaccessible to the pancreatic lipase. In this patient with recurrent duodenal ulcers, hyperchlorhydria may also have exerted a negative effect on the lipolysis. There was no sign of pancreatic insufficiency that could have explained the faecal fat findings (2).

The patient showed clear evidence of malabsorption of vitamin K. Malabsorption of vitamin D and associated bone disease are also well-known to occur in steatorrhea, e.g. caused by absence of bile in cases of long-standing obstructive jaundice (3). The absence of signs of bone disease in this case is well explained by the short duration of the condition. According to Nordin bone disease in steatorrhea is caused by a combination of vitamin D malabsorption and negative calcium balance caused by increased faecal loss of calcium (16). Even if the calcium balance was slightly negative before and slightly positive after the correction of the choledochocolic fistula, the figures are unimpressive and could not be used as a basis for any conclusions.

The study of protein balance did not verify any sign of malabsorption. This is in agreement with the general conception, that absence of bile does not influence the faecal content of nitrogen (1, 10).

In the cases of cholecystocolic fistulas accompanied by malabsorption and diarrhea studied by Gross & d'Alcey (11), Elsas & Gilat (8), Augur & Gracie (4), and Grossman (12) the degree of fat malabsorption seems to have been moderate compared with our case. Only in one of the cases the prothrombin time was appreciably prolonged as a sign of malabsorption of vitamin K. This could perhaps be explained by the possibility that the communication between the common bile duct and the duodenum in these cases was not completely obstructed, even if bile was not found in duodenal aspirations.

On the other hand diarrhea was a predominant feature of the cases of Gross & d'Alcey (11), Elsas & Gilat (8), Augur & Gracie (4) and Grossman (12) in contrast to the case presented here. The mechanism of diarrhea in these cases is thought to be the same as in so-called "cholerheic enteropathy", i.e. diarrhea caused by the noxious effect of increased amounts of bile salts entering the colon (9). In our case the bile salts were also directly shunted into the colon, and why there was no diarrhea is obscure.

REFERENCES

1. Annegers, J. H.: Faecal excretion of nutrients in impaired absorption. Quart Bull Northwestern Univ Med Sch *23:* 198, 1949.
2. — Function of pancreatic juice and of bile in assimilation of dietary triglycerides. Arch Int Med *93:* 9, 1954.
3. Atkinson, M. et al.: Malabsorption and bone disease in prolonged obstructive jaundice. Quart J Med *25:* 299, 1956.
4. Augur, N. A. & Gracie W. A.: Cholecystocolonic fistula associated with malabsorption. Amer J Gastroent *53:* 558, 1970.
5. Berger, L. H.: Internal biliary fistulas. Amer J Gastroent *43:* 11, 1965.

6. Christiansen, E. & Petersen, E. A.: Galdestensileus og fistula biliaris interna. Nordisk Medicin *56:* 97, 1961.
7. Dowse, J. L. A.: Spontaneous internal biliary fistulae. Gut *5:* 249, 1964.
8. Elsas, L. J. & Gilat, T.: Cholecystocolonic fistula with malabsorption. Ann Int Med *63:* 481, 1965.
9. Garbutt, J. T. et al.: The enterohepatic circulation of bile salts in gastrointestinal disorder. Amer J Med *51:* 627, 1971.
10. Gross, J. B. & Power, M. H.: Total solids, fat and nitrogen in faeces. V. A study of patients with primary parenchymatous hepatic disease. Gastroenterology *16:* 140, 1950.
11. Gross, L. & d'Alcey, L. A.: Cholecystocolonic fistula simulating malabsorption syndrome. Amer J Gastroent *42:* 301, 1964.
12. Grossman, E. T.: Cholecystocolic-fistula—an unusual cause of diarrhea. Amer J Gastroent *55:* 277, 1971.
13. Hampson, L. G. & Lehman, G.: Choledochocolic fistula. Arch Surg *79:* 746, 1959.
14. Madsen, C. M. et al.: Spontane interna galdevejsfistler. Nordisk Medicin *56:* 103, 1961.
15. Moreaux, J. et al.: Les fistules bilio-coliques. Arch Franc Mal Appar Dig *56:* 515, 1967.
16. Nordin, B. E.: Effect of malabsorption syndromes on calcium metabolism. Proc Roy Soc Med *54:* 497, 1961.
17. Porter, J. M. et al.: Spontaneous biliary-enteric fistulas. Surgery *68:* 597, 1970.
18. Schäfer, H. & Gruner, H.-J.: Zur Diagnostik und Klinik der inneren Gallenwegfisteln. Materia Medica Nordmark *14:* 461, 1964.
19. Stull, J. R. & Thomford, N. R.: Biliary intestinal fistula. Amer J Surg *120:* 27, 1970.
20. Tyor, M. P. et al.: Metabolism and transport of bile salts in the intestine. Amer J Med *51:* 614, 1971.
21. Wakefield, E. G. et al.: Cholecystoenteric fistulas. Surgery *5:* 674, 1939.

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Address for reprints:

Anders Ljungström
Department of Internal Medicine
University Hospital
S-750 14 Uppsala 14
Sweden