

IATROGENIC CHYMOBILIA — A DISEASE OF THE NINETIES?

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The last two decades have seen a strong swing towards preservation of some of the sphincters of the alimentary tract, namely, the pyloric and anal sphincters, while at the same time there has been increasing "vandalism" of the sphincter at the distal end of the common bile duct.

Endoscopic papillotomy, first introduced in 1974, is a very useful procedure in selected patients but too liberal use of the technique may result in a harvest of new problems in years to come. The intact sphincter of Oddi prevents the entry of duodenal contents into the biliary tree; its division allows their free reflux. By the term 'chymobilia' we mean the presence of chyme in the biliary tree. The Greek word chyme (juice) is combined with the Latin word bilis (bile) in a manner similar to the term haemobilia introduced by Sandblom in 1948¹. Following endoscopic sphincterotomy reflux of duodenal chyme occurs in most patients; aerobilia is seen in half and bacterobilia in all². The results of surgical sphincterotomy are similar. The majority of these patients with bacterobilia do not develop classical symptoms of ascending cholangitis, namely pain, pyrexia, rigors and jaundice but 20 per cent have bouts of upper abdominal pain and associated elevation of the serum Gamma glutamyltranspepside³. Following biliary-enteric anastomosis both aerobic and anaerobic bacteria are involved⁴. Bacterobilia may not be as innocent as was initially thought, and for patients with immunosuppression, the risk of cholangitis is markedly increased⁵. In addition the reflux of chyme may set up chemical changes with resultant inflammatory reaction in the duct epithelium and periductal area. The clinical syndrome of ascending cholangitis often indicates outflow obstruction usually of an incomplete nature due to residual or recurrent stones or restenosis. However we have seen cholangitis in patients following sphincterotomy and other types of biliary-enteric anastomosis where a free-flowing, unobstructed biliary tree was demonstrated by percutaneous cholangiography and by retrograde barium and air studies of the ductal system.

Goldman and colleagues reported on six patients who suffered repeated episodes of cholangitis despite widely patent biliary-enteric anastomoses⁶. Debris is frequently seen in the duct following fenestration whether it be choledochoduodenostomy where the sump syndrome can occur, or after simple surgical or endoscopic sphincterotomy. These ductal filling defects are sometimes referred to as false calculi images. Escourrou and colleagues found evidence of reflux from the duodenum into the biliary tree in 65 per cent of patients after endoscopic sphincterotomy but state that they never observed clinical symptoms related to

reflux in the absence of recurrent or retained stones⁷. However the authors did not give any results of liver function studies in their patients following sphincterotomy. The reflux of chemical and bacterial irritants is probably a major factor in the production of the elevated serum bilirubin and hepatic enzymes seen in 36 per cent of patients with choledochoduodenostomy and 17 per cent of patients with transduodenal sphincterotomy as noted in a surgical series of 246 biliary fenestrations reported from Leicester⁸.

Although reflux may not always produce a clinical syndrome, the biochemical changes indicate at least some degree of continuing low grade damage within the liver parenchyma. Greenfield and colleagues found periportal fibrosis and inflammation on liver biopsy in four out of five patients with raised transaminase levels³. Eleftheriadis and others biopsied the bile duct mucosa one to twelve years after choledochoduodenostomy in nine asymptomatic patients. In all nine patients they found hyperplasia of the epithelial cells, metaplastic goblet cells, pseudopyloric gland formation, dense inflammatory cell infiltration with lymphocytes, plasma cells and polymorphonuclear cells and also fibrosis⁹. Not surprisingly, similar findings were noted in an experimental study using sphincterotomy instead of choledochoduodenostomy¹⁰. Some consider that the changes in the main biliary ducts are adaptive and therefore beneficial. However the liver biopsies show that even the smaller ducts suffer from this inflammatory reaction. Fibrosis and inflammation in these small calibre ducts is likely to cause obstruction, biliary stasis and ultimately parenchymal change. My contention is that iatrogenic chymobilia with its accompanying bacterobilia can produce secondary sclerosing cholangitis in the absence of any obstructive element. The reason that we have not seen a lot of secondary cholangitis and secondary biliary cirrhosis in the past is that many of the surgically created biliary-enteric anastomoses or sphincterotomies were performed in elderly patients with recurrent stones or as bypass procedures in patients with malignancy, both groups having relatively short life expectancy. However much younger patients with benign disease are now being subjected to endoscopic sphincterotomies and I believe that we will see a crop of problems secondary to chymobilia in the decade in which we have entered and subsequently.

Particularly worrying is the use of sphincterotomy for ductal stones without cholecystectomy in some relatively young patients. The residual gallbladder can act as a focus of infection following the ascent of bacteria from the gut. Certainly choledochoduodenostomy without cholecystectomy in the dog results in inevitable chronic inflammation of the gallbladder with the formation of concretions in two thirds of the animals¹¹. Even a Roux-en-Y anastomosis does not protect the dog from ascending cholangitis if the gallbladder is left *in situ* during biliary-enteric anastomosis. Safraney and colleagues noted that acute cholecystitis occurred in 11 per cent of patients subjected to endoscopic sphincterotomy without cholecystectomy¹³. When doing surgery for benign disease of the biliary tree surgeons often go to great lengths to try and prevent reflux of chyme. Creation of valved conduits, interposed jejunal segments and Roux-en-Y procedures have all been used^{14,15} but the outcome is not always satisfactory. The Roux-en-Y operation which was first introduced for gastric surgery using a 20 cm limb has been adapted for biliary-enteric anastomoses¹⁶. Initially the length of the Roux limb which was advocated in biliary surgery was quite short and it has been fascinating to see the "lengthening Roux-en-Y syndrome" (Table 1). Although the currently recommended length is 70 cm, we have demonstrated reflux of oral barium into the biliary

Table 1 The lengthening roux-en-y limb

<i>Author</i>	<i>Length in cm</i>	<i>Year</i>
Smith	20	1964
Thorbjarnarson	25	1975
Warren	30	1980
Takahasi	35	1988
Kune	40	1980
Gadacz	60	1983
Bismuth	70	1978
Blumgart	70	1988
Who Knows?	90	2000

(References 17–24)

tree with 70 cm Roux-en-Y limbs. Perhaps an 80 cm or longer Roux-en-Y limb is required to prevent reflux of orally ingested material. This may reduce the incidence of clinically significant ascending cholangitis but can not stop chymobilia. Once the sphincter mechanism is destroyed nothing prevents the potentially dangerous reflux of bowel contents into the unprotected biliary tree. When Dr Summerfield argued that biliary obstruction is best managed by endoscopists, he wisely added, “Probably only a proportion of the new techniques will stand the test of time!” Perhaps we clinicians need to exercise more caution if we are to avoid the potential hazards of chymobilia.

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