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Endoscopic management of benign bile duct strictures

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ABSTRACT

Endoscopic treatment for benign biliary strictures has largely replaced surgical and percutaneous approaches because of lower morbidity and mortality. However, endoscopic therapy often requires multiple procedures and serial stenting for 1 year or longer. Although the optimal algorithm for endoscopic therapy is unknown, most experts agree that maximal dilation and stenting for a period of at least 3 months will result in the best short- and long-term outcomes. Dominant strictures related to sclerosing cholangitis are more challenging to manage and typically respond best to dilation alone or with shorter-term stent therapy. When considering endoscopic treatment, the location and etiology of the stricture have important prognostic implications that may prompt earlier referral to a different subspecialist or alter the endoscopic strategy. Newer stent technologies, such as fully covered self-expandable metallic stents, may alter the treatment paradigm, although future studies are needed before this strategy can be endorsed. This article reviews the current evidence supporting endoscopic therapy for benign biliary strictures, reviews the clinical predictors of long-term success, underscores the technical aspects of dilation and stent placement, and considers future directions for endoscopic treatment.

1. Introduction

Although "benign bile duct strictures" are often discussed in toto, this disorder can occur from a broad spectrum of iatrogenic and pathologic etiologies. Endoscopic treatment via retrograde cholangiopancreatography (ERCP) has largely replaced surgery and percutaneous therapy as the first-line approach, given its favorable short- and long-term efficacy along with reduced morbidity and mortality. Most experts agree that an aggressive course of therapy to achieve maximal sustained dilation of a stricture will result in the best long-term outcome; unfortunately, this approach often requires 3 or more procedures to accomplish, increasing costs and likely reducing patient satisfaction. Furthermore, it is important for endoscopists to recognize stricture characteristics that portend a poor prognosis because this may affect their threshold for initiating or persisting with endoscopic therapy versus referral to a different subspecialist (eg, interventional radiology, hepatobiliary-pancreatic surgery) earlier in the course. Newer stent technologies may affect the standard approach to stricture therapy. This review outlines the categories of benign bile duct strictures, discusses the evolution of endoscopic therapy to its current paradigm, details the techniques of stricture dilation and stenting, and considers future directions for endoscopic treatment.

2. Stricture characteristics

2.1. Location

Benign bile duct strictures can be classified based on their relationship with the hepatic bifurcation, originally described in the setting of postoperative bile duct injuries by Bismuth et al. [1]. Strictures ≥2 cm below the bifurcation are type I, whereas those within 2 cm are type II. If the hepatic bifurcation is involved, strictures are subgrouped into those with separation of the left and right intrahepatic ducts (type IV) and those without (type III). Of note, most outcome data for endoscopic therapy are limited to Bismuth type I (strictures located ≥2 cm below the hepatic bifurcation) and, to a lesser degree, type II (within 2 cm, but not directly involving the bifurcation) strictures. In general, strictures having a Bismuth class of III or higher are more challenging to manage via endoscopic therapy alone. In these cases, the unaffected upstream duct(s) often limits the dilation that can be achieved via endoscopic stenting, particularly when multiple hepatic segments are independently involved (type IV). Unfortunately, the upstream duct may not be visualized via retrograde opacification in the setting of a complete obstruction, requiring a percutaneous or surgical approach for first-line therapy. Because maximal dilation, defined as having more stents in parallel across a stricture, correlates with the greatest probability of complete and permanent stricture resolution, cases where only a single 10-F stent can bridge the level of obstruction are less likely to completely resolve and more likely to recur during follow-up [2].

Competing interests: Consultant, Boston Scientific, Corp.

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Table 1
General classification of benign bile duct strictures based on etiology.

Benign, Intrinsic	Benign, Extrinsic
Postoperative	Chronic pancreatitis
Liver transplant (duct-to-duct anastomosis)	
Cholecystectomy	
Partial hepatectomy	
Biliary reconstruction (eg, hepaticojejunostomy)	
Primary sclerosing cholangitis	Organized pancreatic fluid collection/pseudocyst
Gallstones	Acute pancreatitis
Stent-associated	Gallbladder (eg, Mirizzi syndrome)
Ischemic	Vascular (portal vein thrombosis, periductal varices)
Autoimmune cholangiopathy	
Lymphoplasmacytic sclerosing pancreatitis/cholangitis	
Infectious (HIV, parasites)	

2.2. Etiology

Another crucial step in characterizing a benign stricture is defining its etiology (Table 1). Benign strictures can be subgrouped into intrinsic and extrinsic etiologies. Most strictures intrinsic to the bile duct result from postoperative injury (eg, postcholecystectomy) or a local fibroinflammatory and ischemic event at the level of a biliary anastomosis (eg, post-liver transplant with a duct-to-duct biliary anastomosis). Postoperative strictures, primarily as a result of cholecystectomy and orthotopic liver transplant, usually result in a focal narrowing at the site of biliary anastomosis (liver transplant) or where a surgical clip was placed partially or completely across the common hepatic duct. Postcholecystectomy injuries involving the intrahepatic biliary tree are beyond the scope of this review; therefore, the discussion is focused on the management of Bismuth type I-II postoperative injuries. In this subgroup, additional predictors of a poor response to endoscopic therapy include having a delayed clinical presentation or a tight stricture on presentation, concomitant bile leak, and the presence of a surgical T-tube [3].

A second important subgroup of intrinsic strictures relates to primary sclerosing cholangitis (PSC). Unlike postoperative cases that are typically localized areas of narrowing, PSC-induced strictures are usually multifocal, longer and irregular in their contour, and frequently involve the hepatic bifurcation. As a result, the approach to endoscopic therapy for a "dominant" PSC stricture is distinct from a postoperative stricture, with a greater emphasis on dilation and short-term stenting. This will be discussed later in this review. Autoimmune cholangiopathy represents an uncommon but medically treatable disease that can mimic PSC or occult cholangiocarcinoma (Fig. 1) [4]. This is classically, but not unequivocally, an IgG4-mediated entity that will respond to medical therapy. Early consideration of this entity may avoid unnecessary endoscopic and surgical interventions.

In patients with chronic pancreatitis, a fibrotic pancreas surrounding the bile duct leads to a persisting force extrinsic to the bile duct, causing it to narrow and potentially obstruct. Therefore, chronic pancreatitis-induced biliary strictures have much less favorable long-term response rates because of underlying dense fibrosis and calcifications in the pancreatic head [5–7]. The stricture can be easily bridged with stents, but these strictures have a higher probability of recurrence after all stents are removed [5–7]. By contrast, secondary bile duct strictures in the setting of acute pancreatitis, with or without fluid collections abutting the bile duct and other acute pathology such as an obstructed gallbladder, will often resolve after treatment of the underlying source of extrinsic compression. In these cases, simply bridging the narrowed segment with a single plastic stent can temporarily relieve the obstruction to allow for the underlying process to resolve with or without additional therapy (eg, drainage of a fluid collection, cholecystectomy). Although these strictures represent a narrowing of the bile duct lumen, the bile duct itself is intrinsically normal, and treatment of the source of biliary compression should be the emphasis for long-term resolution.

3. Principals of endoscopic management

3.1. Dilation

Early reports of nonoperative treatment for bile duct strictures described the use of balloon catheters to dilate strictures [8–10]. Initial strategies described a percutaneous transhepatic approach, which was then extrapolated to endoscopy. These early observations reported favorable results in terms of fluoroscopic resolution of the stricture, particularly in the setting of biliary reconstruction (eg, choledochojejunostomy). The balloon dilation technique is fairly intuitive: a balloon catheter should be centered across the location of the stricture and inflated at least until the balloon waist is obliterated, or the diameter of the balloon is nearly equivalent to the diameter of the unaffected up- or downstream duct (Fig. 2). The optimal duration of balloon dilation is unknown, but most endoscopists keep the balloon inflated across the stricture for 30 to 60 seconds. In cases where the stricture is very long, particularly in the setting of sclerosing cholangitis, a graduated passage catheter can be used to progressively stretch open the affected segment. Alternatively, a balloon can be reinflated in a proximal-to-distal fashion across the stricture, marching the catheter down to fully treat its entire length.

In patients with early (<4 weeks) postoperative strictures, dilation should be avoided whenever possible, to minimize the likelihood of disrupting the anastomosis or surgical clips and causing a secondary leak. In these cases, simply bridging the obstruction with a single, small (7- or 8.5-F) plastic stent for 6 to 8 weeks is usually sufficient. This will allow the patient to recover from surgery and then return for subsequent dilation and stenting. Many cases of early postoperative strictures result from edema; on repeat cholangiography 6 to 8 weeks after surgery, many of these early strictures will resolve as the edema and acute inflammatory response cedes.

Fig. 1. Autoimmune cholangiopathy: a medically treatable type of biliary stricture. An 84-year-old man presented with obstructive jaundice and multiple strictures of the common hepatic duct and right main hepatic duct. Subsequent biopsy confirmed the presence of plasma cell infiltration with IgG4 positivity, consistent with autoimmune cholangiopathy.