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EDITORIAL

# Taming the BEASt: Management of Biliary-Enteric Anastomotic Strictures

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Biliary-enteric anastomoses result from surgery, usually for pancreatico-biliary tumors and related pathology, in which part of the biliary tree must be resected with the remaining portion anastomosed to the intestine. These anastomoses are subject to stricture in 3–17% of cases, with resultant biliary obstruction, that is usually managed endoscopically [1, 2]. Endoscopic management of benign postoperative biliary-enteric anastomotic strictures (BEAS) is challenging on multiple levels. The postoperative anatomical alterations create the initial challenge with regard to endoscopic access to the anastomosis. The advent of balloon enteroscopy-assisted biliary intervention has facilitated endoscopic management of BEAS in situations where percutaneous intervention had previously been the sole non-surgical option. Once the challenge of endoscopic biliary access was overcome, identification of the biliary-enteric anastomosis can often be challenging, especially in the context of a stenosis, where the enteric mucosa can mask the stenosed biliary orifice. Upon identification of the anastomosis, successful biliary cannulation and the decisions regarding the optimal approach to endoscopic management present the endoscopist with a completely new set of questions and challenges.

To date, there has been no standardized endoscopic approach to BEAS. In recent years, there has been increasing literature to support long-term stenting with either plastic or metal biliary stents for management of benign biliary strictures [3, 4]. Since the underlying pathophysiology of BEAS is, however, different than of primary biliary strictures, extrapolation of such data to BEAS would therefore be inappropriate. The additional challenges of endoscopic access to the BEAS add weight to the need to "get it right the first time."

In this issue of *Digestive Diseases and Sciences*, Yamouchi et al. [5] describe a retrospective, single-center study assessing the recurrence rate and risks of developing recurrent BEAS (RBEAS) following a single endoscopic-assisted balloon dilatation. All patients in the study underwent a single balloon dilatation and were evaluated every 3–6 months thereafter. Fifty-five patients were identified over a 9-year period. Follow-up was for a minimum of 1 year, with a median observation period of 3.25 years. Biliary stenting was not performed in any of these cases.

At 12 months, the rate of RBEAS was 32.7%. The rate of RBEAS over the mean follow-up period (3.25 years) was 52.7%, and the median time to RBEAS was 2.78 years. A number of risk factors were identified to predict RBEAS on multivariate analysis, including postoperative bile leak ($p = 0.001$, HR 10.94, CI 2.47–48.39), onset of BEAS within 6 months postoperatively ($p = 0.01$, HR 6.18, 95% CI 1.46–29.21), absence of intrahepatic stones ($p = 0.049$, HR 3.05, 95% CI 1.01–9.22), and remaining balloon waist ($p = 0.0051$, HR 5.71, 95% CI 1.69–19.31). The univariate analysis also included additional risk factors such as preoperative chemotherapy and strictures involving bilateral hepatic ducts.

These results highlight a number of important aspects with regard to balloon dilatation alone as a management option for BEAS. Firstly, that RBEAS following balloon dilatation typically occurs many months—years after initial dilatation. Secondly, that recurrence is common since it is more likely than not that stricturing will recur, especially if life expectancy is > 3 years. Thirdly, and importantly, that there are a number of risk factors that predict the likelihood of recurrence of stricturing after balloon dilatation. In patients who have risk factors for recurrence, the time to recurrence was only 0.88 years, and in patients with no risk factors, the median time to RBBEAS was actually not reached (≥ 3 years). The more risk factors, the greater the probability that stricturing will recur.

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The incidence of BEAS was reported as 4–12%, although this has ranged from as low as 2.6–24% in the published literature [6, 7]. The wide variation in incidence likely reflects the heterogeneity of the underlying pathological entity and again highlights the challenges in standardizing study designs around BEAS. Factors including variations in patient survival due to the underlying disease, a small sample size in many of the reported studies, and variations in surgical techniques and expertise are likely contributing factors. In this study, Yamouchi et al. report a similar time to stricture diagnosis to that reported in the published literature of ~ 17 months [5, 6], supporting the generalizability of their results.

An important aspect of Yamouchi's study is the identification of significant risk factors for predicting RBEAS, in particular, postoperative bile leak, onset of postoperative stricture within 6 months of surgery, remaining balloon waist, and absence of intrahepatic stones. Similar risk factors have been identified as potential etiological factors for onset of BEAS, in addition to other risk factors including preoperative biliary stent placement and drainage, although results have been inconsistent across different studies [6–8]. These additional risk factors were not analyzed in this study. Age was not a significant factor, as has been reported in other case series [9].

How does this study help clinicians decide the most appropriate management strategy for this rare but important and challenging clinical entity? While this study supports endoscopic management of BEAS, it also suggests that balloon dilatation alone is not an adequate treatment option for the majority of patients, especially if survival is anticipated to be > 12 months. Furthermore, and importantly, patient characteristics may help decide which subgroup of patients could potentially be treated with balloon dilatation alone, and who would likely benefit from additional treatment, typically biliary stent placement at the time of dilatation. Tomoda et al., who recently evaluated this question using plastic stents, found a benefit with plastic stent deployment in extending bile duct patency rate compared with balloon dilatation alone [10]. When should self-expandable metal stents (SEMS) be used? Yamauchi's group attempted to answer this question with a published case report using SEMS following recurrence of BEAS managed with balloon dilatation and plastic stent placement [11]. Should SEMS be the primary stent choice, or only be reserved for failed plastic stent cases? This is a question waiting to be answered.

Limitations of this study include its inherent retrospective design and the use of a single center with a relatively small sample size. What is also not known from this study is the decision-making underlying the decision to stent or not to stent at index endoscopy, and what proportion of patients with BEAS underwent non-endoscopic management. Although 11% of patients did not succeed with endoscopic intervention, where there more patients in whom endoscopic intervention was not attempted who proceeded straight to radiological or surgical management? How much selection bias was there to influence the validity of the study findings?

Until more high-quality evidence is available to support clinical decision-making, many questions remain unanswered when dealing with BEAS. Who should undergo routine post-dilatation stenting? Should this be performed on all patients, or patients with risk factors only? Should patients with multiple risk factors have alternative therapeutic interventions? Is stenting likely to change the risk of recurrent stricturing, and are SEMS superior to plastic stents? No doubt, these questions would be best answered with future prospective randomized studies. Nevertheless, given that BEAS will remain a rare clinical entity, the challenge moving forward will be finding a large enough sample size to design a study to answer these questions with scientific rigor. Yet, the logical progression to move forward from this study is to assess whether biliary stenting complements balloon dilatation in BES.

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