Perforation in colorectal stenting: a meta-analysis and a search for risk factors CME

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Background: Recent studies suggest that there is a substantial risk of perforation after colorectal stent placement.

Objective: To identify risk factors for perforation from colonic stenting.

Design: A meta-analysis of 86 studies published between 2005 and 2011.

Setting: Multicenter review.

Patients: All patients who underwent colorectal stent placement.

Intervention: Colorectal stent placement.

Main Outcome Measurements: The occurrence of perforation with subgroup analyses for stent design, stricture etiology, stricture dilation, and concomitant chemotherapy, including the use of bevacizumab.

Results: A total of 4086 patients underwent colorectal stent placement; perforation occurred in 207. Meta-analysis revealed an overall perforation rate of 7.4%. Of the 9 most frequently used stent types, the WallFlex, the Comvi, and the Niti-S D-type had a higher perforation rate (>10%). A lower perforation rate (<5%) was found for the Hanarostent and the Niti-S covered stent. Stenting benign strictures was associated with a significantly increased perforation rate of 18.4% compared with 7.5% for malignant strictures. Dilation did not increase the risk of perforation: 8.5% versus 8.5% without dilation. The subgroup of post-stent placement dilation had a significantly increased perforation risk of 20.4%. With a perforation rate of 12.5%, bevacizumab-based therapy was identified as a risk factor for perforation, whereas the risk for chemotherapy without bevacizumab was 7.0% and not increased compared with the group without concomitant therapies during stent therapy (9.0%).

Limitations: Heterogeneity; a considerable proportion of data is unavailable for subgroup analysis.

Conclusions: The perforation rate of colonic stenting is 7.4%. Stent design, benign etiology, and bevacizumab were identified as risk factors for perforation. Intraprocedural stricture dilation and concomitant chemotherapy were not associated with an increased risk of perforation. (Gastrointest Endosc 2014;79:970-82.)

Abbreviations: CI, confidence interval; SEMS, self-expandable metal stent.

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The use of self-expandable metal stents (SEMSs) for colorectal obstruction has evolved over the past decades. Their applications have extended to the management of acute malignant colorectal obstruction, palliation of inoperable obstructing colorectal cancer, and treatment of benign colonic strictures.[1,2] In the setting of emergency acute colorectal obstruction, colorectal SEMS placement has several advantages over surgery. SEMS placement allows (1) the possibility to improve the patient's clinical condition to allow for elective surgery (also referred to as a bridge to surgery) and (2) accurate tumor staging to prevent surgery in patients with incurable disease or those with an unacceptable surgical risk.[3] The potential benefits reported after SEMS placement are decreased mortality, morbidity, number of temporary and permanent colostomies, and hospital stay. These benefits are supported by several uncontrolled and comparative studies.[4-12] However, some recently published randomized, controlled trials failed to confirm advantages of SEMS placement over surgery for patients with malignant colonic obstruction.[13-16]

Clinical failure after successful colorectal SEMS placement is mainly caused by stent occlusion (16%), stent migration (uncovered SEMSs, 3%-12%; covered SEMSs, 30%-50%), and perforation of the tumor and/or normal colonic wall.[17] The latter is the most feared adverse event of colonic stenting because of its serious consequences. According to current literature, perforation occurs in 3.8% to 6.9% of the patients undergoing colonic stent placement,[18-20] requires surgical management in the majority of patients (73%), and leads to death in 16.3% of cases.[21] Despite the severity of this adverse event, details on perforation are poorly reported in literature. Therefore, little is known about the etiology of colonic perforation in patients undergoing colonic stent placement. Van Hooft et al[13] prematurely closed their randomized study because of an unexpected high perforation rate in the SEMS group compared with the surgical group and suggested that the type of stent and administration of chemotherapy could have played a causative role. Cennamo et al[22] reported an increased risk of colonic perforation during bevacizumab-based therapy. Studies are lacking to definitively confirm the risk factors for colonic perforation after SEMS placement. Therefore, the primary objective of our study was to extensively review the published data and to assess the effects of different types of colorectal stents on the occurrence of colonic perforation in patients undergoing colorectal SEMS placement for malignant and benign colorectal obstruction. Secondary objectives were to assess the effects of chemotherapy, particularly bevacizumab administration, stricture dilation, and the etiology of stenosis on the occurrence of perforation.

Take-home Message

- The perforation risk in colorectal stenting is 7.4%; almost 70% of perforations occur in the first week after stent placement.
- This meta-analysis suggests that certain factors influence the risk of perforation, such as the type of stent, a benign stricture etiology, and concomitant bevacizumab therapy.

METHODS

This study was designed as a literature review with additional retrospective data collection and a meta-analysis. On March 7, 2011, the MEDLINE database was searched beginning with data published from January 2005 forward. Only publications in English were reviewed. To avoid missing relevant citations, reference lists of reviews on colonic stenting were also checked. Figure 1 shows the selection criteria and the results of the search process. The search and selection process was conducted by the first author under the direct supervision of 2 other authors (A.R., J.v.H.). The reviewers had no affiliation with other authors, institutions, or journals of the articles ultimately included in the analysis. After fulfillment of inclusion criteria, we identified 4 duplicate publications that were excluded.[23-26] The study by Kim et al[27] included 55 patients from the study by Song et al.[28] However, both studies were included because they described large study populations and reported different cases of perforations.

A total of 86 studies met eligibility criteria and were included in this review. The study designs were retrospective (n = 46, 53.5%); prospective (n = 22, 25.6%); case report (n = 7, 8.1%); randomized, controlled trials (n = 5, 5.8%); both retrospective and prospective (n = 2, 2.3%); and undefined (n = 4, 4.7%). Malignant lesions were the primary stenting indication in 77 studies (89.5%), whereas 9 (10.5%) focused on benign colonic stenting. Stents were inserted endoscopically under fluoroscopic guidance in 62 studies (72.1%), purely radiologically in 8 studies (9.3%), and purely endoscopically in 2 studies (2.3%); combinations were used in 11 studies (12.8%), and the technique for stent deployment was not reported in 3 studies (3.5%). Study characteristics are presented in Table 1 (available online at www.giejournal.org). Data extracted regarding the total study population and the specific cases of perforation are depicted in Table 2. As previously mentioned, details on perforation are poorly reported in literature. When the required data (Table 2) were missing from publications, the corresponding authors were contacted by e-mail to request these data. The data provided in the literature were sufficient for inclusion in our review in only 8 articles, including 6 case reports. Therefore, request letters for additional data were sent to the corresponding authors of the 78 remaining studies. When authors were queried about the site of perforation pertaining to the stent, they could choose one of the following options: proximal end of the stent, stent body, distal end of the stent, both ends of the stent, at the site

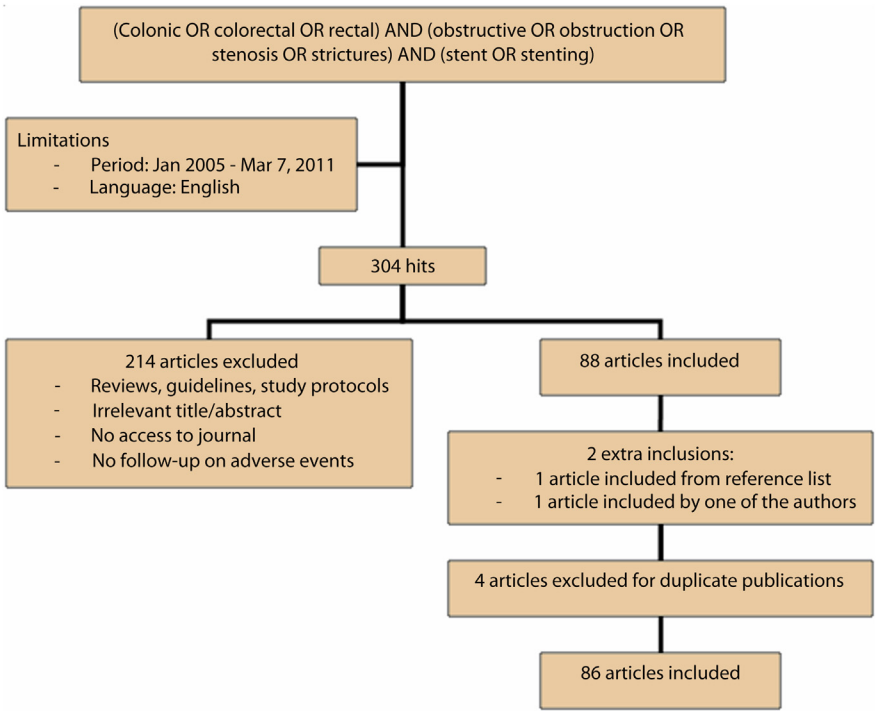


Figure 1. MEDLINE search and study selection.

of the stent, proximal to the site of the stent, distal to the site of the stent, tumor perforation, cecal perforation, or unknown. A perforation was considered *stent related* when it was reported as such or when the end of the stent protruded through the intestinal wall and *stent unrelated* when it was reported as such or when the perforation occurred in an area remote from the stent. When the relationship between perforation and stent was missing, the authors were queried about their assessment and could choose from stent related, stent unrelated, or unknown. Over a period of 3 months, 75.6% (59/78) of the authors responded to our request; we received additional data in 59.0% (46/78), and the overall required data for analysis were complete in 50.0% (43/86) of the included studies. All useful data from the 86 articles supplemented with the provided data from the request letters were used for statistical analysis.

Statistical analysis

To determine the pooled stent perforation rate, a meta-analysis was performed. We calculated the natural log (ln [odds]) with its standard error (SE ln[odds]) of the perforation rate for every separate study using the stent design concerned. These calculations were performed with the Microsoft Excel 2002 program (Microsoft Corporation, Redmond, Wash). The ln(odds) and its SE of each study were pooled by using Review Manager (RevMan) software, version 5.1 (The Nordic Cochrane Centre, The Cochrane Collaboration, 2011 Copenhagen, Denmark). RevMan estimated the pooled ln(odds) with 95% confidence interval (CI) by applying weight to every separate study by using a fixed model. To assess heterogeneity of the pooled results, the I^2 test was calculated. Heterogeneity was defined as low, moderate, and high with I^2 values of less than 25%, 25% to 50%, and greater than 50%, respectively.[29] Finally, the overall pooled perforation rate of an individual stent design with 95% CI was calculated from the overall pooled ln(odds) with 95% CI using the Microsoft Excel 2002 program. Secondary endpoints were pooled and analyzed by using the same method as described. When a perforation rate related to a specific variable was missing, the study was excluded from analysis. To avoid the inclusion of samples with a perforation rate of 100%, only series that reported on subgroups of 3 or more patients were included for meta-analysis. In the search for homogeneity among perforation rates of pooled studies, a meta-analysis was also performed for 2 subgroups according to the study design: retrospective and prospective. Statistical significance was reached when the 95% CI of the point estimate for a group did not overlap with the point estimate for the other group and vice versa.

RESULTS

Overall population

Of the 86 studies included, a total of 4086 patients underwent colonic stent placement. A total of 3864

TABLE 2. Data extraction

Total study population	Perforation cases
Study design	Stent type
Year of publication	Stent length and diameter
Country	Location of the stent in the colon
Stricture etiology	Site of the perforation pertaining to the stent
Deployment technique	Stricture dilation
Patients subjected to colonic stenting procedure	Stricture etiology
Patients receiving a stent for malignant and benign indications	Concomitant chemotherapy and bevacizumab
Patients on concomitant chemotherapy and bevacizumab	Days to perforation
Stricture dilations Types of stents used Perforations	Stent related

(94.6%) patients had a colonic stent inserted, whereas in the remaining patients, the stent could not be placed due to inability to traverse the stricture with the guidewire or to pass the stent deployment system across the stenosis (n = 176, 4.3%). In some cases, it was unclear whether the stent was deployed (n = 46, 1.1%). Colonic perforation occurred in 207 patients. Meta-analysis showed an overall perforation rate of 7.4% (95% CI, 6.5%-8.5%) with high heterogeneity (I^2 = 52%) between the reported perforation rates. Further details on the overall study population are summarized in Table 3.

Perforation rates based on stent type

The characteristics of the 207 cases of perforation are presented in Table 4. Colonic perforation occurred after a median of 3 days (range 0-960 days) after stent placement and was deemed stent related in 44.9% (93/207). Nine different types of colorectal stents were used most frequently: the Bard Memotherm colorectal stent (Angiomed [now Conmed], Swindon, UK), the Comvi Stent (Taewoong Medical Co, Gimpo, South Korea), the Dual Stent (S&G Biotech, Seoul, Korea), the Enteral Wallstent (Boston Scientific, Natick, Mass), the Hanarostent (M.I. Tech, Seoul, Korea), the Niti-S covered colorectal stent (Taewoong Medical Co), the Niti-S D-type colorectal stent (Taewoong Medical Co), the Ultraflex Precision colonic stent (Boston Scientific), and the WallFlex colonic stent (Boston Scientific). Three studies using the Bard Memotherm stent were excluded because of missing data

TABLE 3. Baseline characteristics of patients undergoing stent placement

Characteristics	Total	%
Patients undergoing stent placement procedure	4086	100
Patients having a colonic stent inserted	3864	94.6
Inability to pass the stricture*	176	4.3
Unclear whether stent was deployed	46	1.1
Reported perforations	207	5.1
Etiology of patients receiving stent		
Malignant stenosis	3982	97.5
Benign stenosis	104	2.5
Concomitant treatment		
None	1433	35.1
Patients on chemotherapy without bevacizumab	637	15.6
Patients on chemotherapy with bevacizumab	86	2.1
Missing data	1930	47.2
Stricture dilation		
None	2511	61.5
Stricture dilation	335†	8.2
Intraprocedural prestent dilations	146	3.6
Intraprocedural poststent dilations	190	4.7
Endoscopic reintervention dilations	25	0.6
Missing data	1240	30.3
No. of stents inserted into the colon	4221	
Uncovered stents	3114	73.8
Covered stents	633	15.0
Missing data	474	11.2

*With guidewire or stent delivery system due to nonnegotiable stricture or tortuous anatomy.
†Patients undergoing intraprocedural pre- and poststenting dilation (n = 19), patients undergoing intraprocedural dilation and reintervention dilation (n = 7).

on causes of perforation.[30-32] Meta-analyses showed perforation rates varying from 3.1% to 10.9% (Table 5). Two groups could be identified that differed significantly: stent designs with a high perforation rate (>10%) versus designs with a low perforation rate (<5%). High perforation rates were found for the WallFlex (10.9%; 95% CI, 7.4%-16.0%), the Comvi (10.8%; 95% CI, 4.4%-24.2%), and the Niti-S D type (10.3%; 95% CI, 5.7-18.1%). The

TABLE 4. Perforation characteristics (n = 207)

Characteristics	Perforations (No.)	%
Days until perforation (n = 176, missing n = 31)		
Median, 3 (range 0-960)		
0	51	29.0
1-3	41	23.3
4-7	25	14.2
8-14	19	10.8
15-30	12	6.8
>30	28	15.9
Description of perforation		
No descriptive details	139	67.1
Procedure related	32	15.5
Guidewire perforation	8	3.9
Reintervention related	2	1.0
Multiple stents in situ	8*	3.9
Silent perforation	15*	7.2
Fractured stent	1	0.5
Migrated stent	1	0.5
Perforation after restenting	1	0.5
Stent transversely inserted	1	0.5
Stent-related perforation		
Yes	93	44.9
No	47	22.7
Unknown	67	32.4
Stent length, mm (n = 122; missing, n = 85)		
Median 90 (range 40-160)		
<90	47	38.5
90	42	34.4
>90	33	27.0
Stent diameter, mm (n = 122; missing, n = 85)		
Median 22 (range 18-30)		
<22	12	9.8
22	53	43.4
>22	57	46.7
Stent location in colon (n = 158, missing n = 49)		
Rectum	8	5.1
Rectosigmoid junction	39	24.7
Sigmoid	70	44.3
Sigmoid-descending junction	6	3.8
Descending colon	13	8.2
Splenic flexure	3	1.9
Transverse colon	8	5.1
Hepatic flexure	1	0.6
Ascending colon	2	1.3
Not applicable†	8	5.1
Site of reported perforation n = 170; missing, n = 37)		
Proximal end of the stent	23	13.5
Stent body	8	4.7
Distal end of the stent	9	5.3
Both ends of the stent	6	3.5
At the site of the stent	24	14.1
Tumor perforation	38	22.4
Cecal perforation	16	9.4
Proximal to stent	11	6.5
Distal to stent	2	1.2
Unknown	25	14.7
Guidewire perforations	8	4.7

*One patient with a silent perforation had multiple stents inserted.
†Guidewire perforations

Hanarostent (4.7%; 95% CI, 2.8-7.9%) and the Niti-S covered (3.1%; 95% CI, 1.1-8.6%) were found to have a low perforation risk (see Table 5 for details).

Identification of risk factors

Stricture etiology. The etiology of the stricture was malignant in 97.5% and benign in 2.5% (Table 3). A rate of 89.9% (186/207) of perforations occurred in patients with a malignant stenosis, 5.3% (11/207) in patients with a benign stenosis, and 4.8% (10/207) of cases in which the stricture etiology could not be defined because of missing data. In the meta-analysis, patients with benign strictures were found to have a significantly higher rate of perforation compared with patients with malignant strictures, 18.4% (95% CI, 10.0%-31.2%; $I^2 = 20\%$) versus 7.5% (95% CI, 6.5%-8.6%; $I^2 = 53\%$), respectively (Table 6).

Stricture dilation. Data on the use of stricture dilation during the course of stent therapy were identified in 2846 patients (69.7%). Stricture dilation was performed in 11.8% (335/2846) of patients and was determined to

be (1) intraprocedural before stent placement, (2) intraprocedural after stent placement, or (3) reintervention with the stent in situ. Twenty-six patients underwent stricture dilation for more than 1 of the above scenarios as presented in Table 3. Perforations occurred when stricture dilation was performed in 13.5% (28/207), including intraprocedural prestent dilation (n = 8), intraprocedural poststent dilation (n = 12), reintervention dilation with stent in situ (n = 4), and dilation unspecified (n = 4). Meta-analysis (Table 7) showed no statistically significant difference in perforation when stricture dilation was performed: 8.5% (95% CI, 5.5%-12.8%; I^2 = 25%) versus a perforation rate of 8.5% (95% CI, 7.2-10.0%; I^2 = 60%) when no dilation was performed. However, the subgroup of patients who underwent postprocedural reintervention dilation with the stent in situ had a significantly increased perforation risk of 20.4% (95% CI, 6.5%-48.8% with I^2 = 0%) compared with the nondilation group. Details are presented in Table 7.

Concomitant chemotherapy

In 2156 patients (52.8%), it was explicitly mentioned whether chemotherapy was administered; 29.5% (637/2156) of patients received chemotherapy without bevacizumab during stent therapy, 4.0% (86/2156) received a bevacizumab- (Avastin) based regimen, and 66.5% (1433/2156) of patients did not receive concomitant therapy. In the 207 cases of perforation, 44.0% (91/207) occurred in patients without concomitant chemotherapy, 13.0% (27/207) occurred in patients receiving concomitant chemotherapy, and 5.3% (11/207) occurred in patients receiving concomitant bevacizumab-based therapy. In 37.7% (78/207) of perforations, the use of concomitant therapy was unknown. In a meta-analysis (Table 8), concomitant chemotherapy without bevacizumab was associated with a significantly lower perforation rate of 7.0% (95% CI, 4.8%-10.0%; I^2 = 5%) compared with 9.0% (95% CI, 7.2%-11.1%; I^2 = 52%) when no concomitant therapy was administered during stent therapy. The perforation risk of bevacizumab-based chemotherapy (12.5%; 95% CI, 6.4%-22.8%; I^2 = 0%) was significantly increased compared with the no concomitant therapy group.

DISCUSSION

This meta-analysis of colorectal stent data revealed significant differences in perforation outcome between the most frequently used stent designs. The primary objective of this analysis was to identify the perforation rates between different stent designs, and the impetus was based on the trial by van Hooft et al.[13] The authors speculated that there was a correlation between the WallFlex colonic stent and the unexpectedly high number of perforations. Indeed, our meta-analysis showed the WallFlex as a type of stent associated with a higher risk (>10%) of perforation. However, recently published results of a large multicenter, prospective cohort using the WallFlex colonic stent did not support this and showed lower perforation rates of 3.0% to 5.1%.[33-35] The one caveat is the relatively short follow-up in which a maximum follow-up of 12 months was reached in only 14.1% of patients.[35] A recently published randomized, controlled trial from Korea also showed a WallFlex perforation rate of 5.6% (4/71).[36] Other stent designs associated with a higher perforation risk in our meta-analysis were the Niti-S D type and the Comvi stents. Compared with the stent designs with lower perforation rates (Hanarostent and Niti-S covered), we could not find striking differences between stent designs to explain perforations. All stents are made of a woven nitinol frame. The WallFlex has a proximal flange, just as the Niti-S covered has at both ends, to minimize migration. All stents have looped ends to prevent tissue injury. The percentage of foreshortening during expansion of the stent differs in both groups.[3] Differences among expansive force of the stents could be of some importance; however, to our knowledge, no data are available on the radial and longitudinal forces of the stents considered in our study. Although of interest, the correlation between stent diameter and perforation could not be analyzed because of insufficient data in the literature. The finding that the majority of perforations occurred within the first days after stent placement is in accordance with the literature.[19,21] This raises the suspicion that a significant number of perforations result from the stent placement itself. In our review, however, procedural perforations were reported in 19.4% of cases, including 8 guidewire perforations (3.9%). It could be that the remaining group of early perforations was caused by failure of the colonic wall to adapt to the expanding forces of the stent. With regard to the different stent designs, perforation within the first week after stent placement varied from 65.2% to 100% with exception of the WallFlex (36.4%, 8/22) and the Comvi (25.0%, 1/4) stents. The relatively high occurrence of delayed perforations with the WallFlex suggests that a difference in duration of follow-up between stent designs could have influenced the perforation rate because longer follow-up may identify more delayed perforations.

We identified an increased risk of perforation in patients with benign strictures compared with malignant strictures. More recent publications reporting on the application of SEMSs in the treatment of benign colorectal strictures do not report increased perforation rates.[37-40] These series mainly included anastomotic and Crohn's disease strictures rather than diverticular strictures, and all concluded that the application of SEMSs is safe for these indications. The benign strictures included were mainly diverticular.[41-43] These strictures might be more susceptible to perforation during stent therapy than anastomotic and Crohn's strictures. An explanation for this could be that diverticular strictures contain more active inflammation, which weakens the bowel wall, whereas anastomotic

TABLE 5. Meta-analysis of perforation rate per stent design

Stent design	No. of studies (R:P)	Events	Total	Perforation rate (95% CI)
Bard Memotherm*	5 (3:2)	13	42	Excluded
Comvi stent	3 (1:2)	4	126	10.8% (4.4%-24.2%)
Dual stent	3 (1:2)	23	283	8.7% (5.9%-12.8%)
Hanarostent	10 (6:4)	11	388	4.7% (2.8%-7.9%)
Niti-S covered	5 (4:1)	2	125	3.1% (1.1%-8.6%)
Niti-S D-type	10 (7:3)	9	221	10.3% (5.7%-18.1%)
Ultraflex	14 (10:4)	16	338	7.2% (4.7%-11.0%)
WallFlex	19 (13:6)	22	423	10.9% (7.4%-16.0%)
Wallstent	27 (20:7)	53	959	7.7% (6.0%-9.9%)

R, the number of retrospective studies, including, case reports, series with both retro- and prospective, data and studies without description of the design; *P*, the number of prospective studies, including, randomized, controlled trials; CI, confidence interval.
*Excluded from analysis due to small numbers and heterogeneous data.

TABLE 6. Meta-analysis of perforation rate for stricture etiology

Stricture etiology	No. of studies (R:P)	Events	Total	Perforation rate (95% CI)
Benign	8 (7:1)	8	71	18.4% (10.0%-31.2%)
Malignant	73 (47:26)	185	3689	7.5% (6.5%-8.6%)

R, number of retrospective studies, including case, reports, series with both retro- and prospective data, and studies without description of the design; *P*, number, of prospective studies, including randomized, controlled, trials; *CI*, confidence interval.

TABLE 7. Meta-analysis of perforation rate for stricture dilation

Stricture dilation	No. of studies (R:P)	Events	Total	Perforation rate (95% CI)
None	47 (30:17)	134	2415	8.5% (7.2%-10.0%)
Overall	10 (5:5)	19	308	8.5% (5.5%-12.8%)
Intraprocedural prestent	7 (2:5)	5	137	8.2% (4.0%-16.1%)
Intraprocedural poststent	6 (3:3)	12	188	7.3% (4.2%-12.3%)
Reintervention	4 (3:1)	2	16	20.4% (6.5%-48.8%)

R, number of retrospective studies, including case, reports, series with both retro- and prospective data, and studies without description of the design; *P*, number, of prospective studies, including randomized, controlled, trials; *CI*, confidence interval.

stenoses are fibrotic rings, and Crohn's strictures usually develop after remission of the disease activity and are typically associated with fibrotic enteral wall thickening. In case series using the SX-ELLA biodegradable stent (ELLA-CS, Hradec Kralove, Czech Republic) for anastomotic and Crohn's strictures, colonic perforation did not occur.[44-46]

Stricture dilation before stent placement is believed to cause an increased risk of colonic perforation, although our meta-analysis did not confirm this. The increased risk found in the literature is mainly based on the results of 2 pooled analyses with data from before the year 2004.[19,47] Khot et al[47] reported a pooled perforation rate of 9.5% (10/105) in the dilation group, whereas we found a pooled perforation rate of 6.2% (19/308). Differences in characteristics of the stenosis and/or diameter and dilation techniques might explain this disparity. The hypothesis of dilation-related perforation has been studied by Tanaka et al.[48] They investigated the effect of balloon dilation in excised colorectal cancer specimens by slowly inflating an 18-mm balloon and maintaining the maximum diameter for 1 minute. Perforation occurred in 17% (8/47). They found that the stenoses of specimens in which perforation occurred were of greater annularity and smaller diameter and had more peritumoral fibrosis. We found a significantly increased perforation risk for the subgroup of postprocedural reintervention dilation, which

TABLE 5. Continued

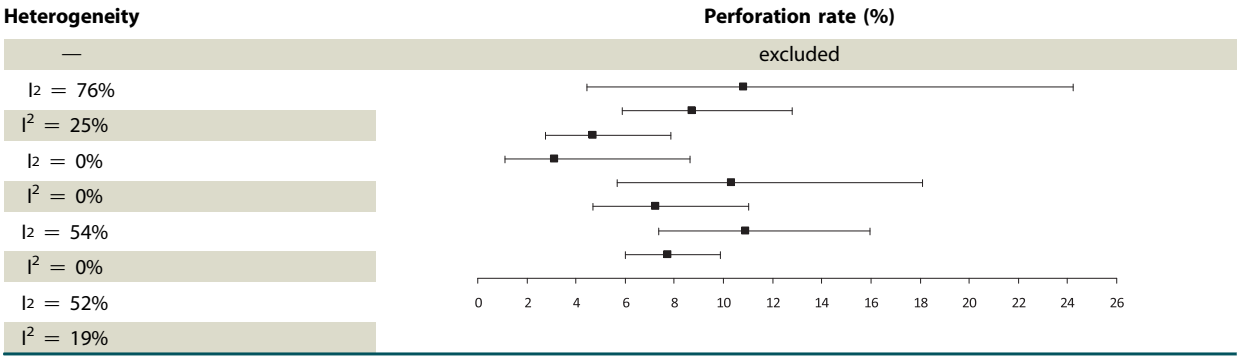


TABLE 6. Continued

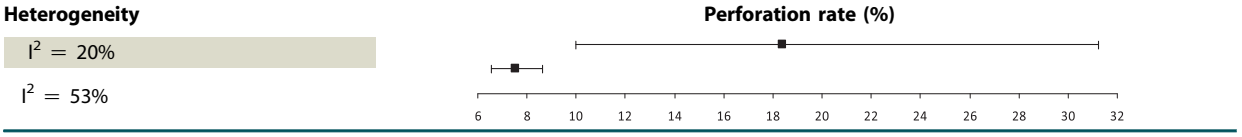
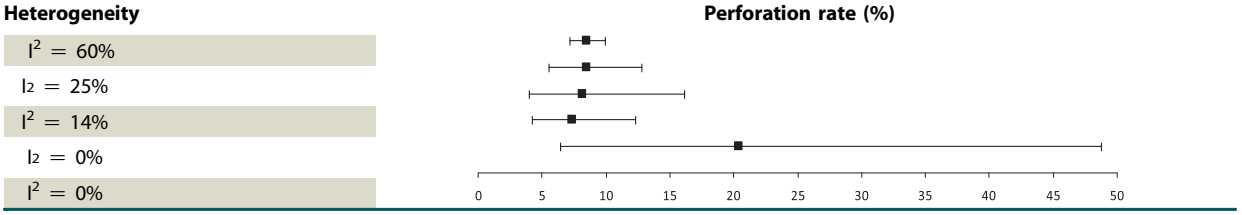


TABLE 7. Continued



was usually performed because of stent reocclusion by tumor ingrowth. A possible explanation could be that the expanding tumor process weakens the bowel wall, making it more vulnerable to perforation. We therefore advise against performing reinterventional dilation in colonic stenting.

We found a reduced risk of perforation when concomitant nonbevacizumab-based chemotherapy was administered during stent treatment. It has been speculated that chemotherapy could contribute to perforation from colonic stenting because of the destructive effect on the proliferating cancer cells in the bowel wall.[13,49] We do not have an explanation for this finding, and because of the small difference in perforations, we do not consider chemotherapy without bevacizumab to be clinically relevant. In this meta-analysis, a higher perforation rate was seen in patients receiving concomitant bevacizumab compared with the population without concomitant therapy (12.5% vs 9.0%). Bevacizumab, a monoclonal antibody that binds to vascular endothelial growth factor, results in reduced and delayed angiogenesis. It is known to increase the risk of GI perforation in patients with various types of cancer.[50] A more recent series reporting on bevacizumab therapy during colonic stent placement also found increased incidences of perforation.[51] The previous data indicate that bevacizumab-based therapy should be always

TABLE 8. Meta-analysis of perforation rate for concomitant therapy

Concomitant therapy	No. of studies (R:P)	Events	Total	Perforation rate (95% CI)
None	39 (24:15)	74	1347	9.0% (7.2%-11.1%)
Chemotherapy without bevacizumab	25 (17:8)	22	578	7.0% (4.8%-10.0%)
Bevacizumab-based therapy	8 (5:3)	6	80	12.5% (6.4%-22.8%)

R, number of retrospective studies, including case reports, series with both retro- and prospective data and studies, without description of the design; *P*, number of prospective studies, including randomized, controlled trials; *CI*, confidence interval.

considered with great caution or avoided if possible in patients previously treated with colonic stenting.

This meta-analysis of literature data has several limitations. First, data on perforation were insufficient in the vast majority of included studies. Even after the data collected from the request letters, a significant number of studies were excluded from analyses. Second, to collect a large series of perforations, all studies on colonic stent placement, regardless of study design, were included. Therefore, the aggregated data presented in this review are based on heterogeneous patient populations. The heterogeneity shown as the I^2 test in the meta-analysis is based on the difference in perforation rates between the included studies, but does not provide details about the differences in patient populations. When results are presented with moderate to high heterogeneity in combination with a large range in the 95% CI, one must be cautious when interpreting the findings. Third, the statistical model does not correct for confounders. Therefore, if 2 variables influenced the outcome of perforation, their effect was biased by confounding. An individual patient data meta-analysis would be the next step to disentangle the independent contributions of predictors of perforation. Fourth, it is difficult to precisely define whether a perforation is stent related. In cases in which the wires at the stent ends protrude through the tumor-free intestinal wall, an association with the stent can be assumed. However, a perforation in the tumor area at the body of the stent could be a consequence of the expanding tumor process or may be caused by the stent. This makes it challenging to directly relate perforation to stent design. We suggest that future research studies distinguish perforations in a manner proposed by Baron et al[3]: "(1) guidewire or catheter malpositioning, (2) dilation of the stricture before or after stent placement, (3) stent-induced perforation (subclassified as tumor and nontumor local perforation), (4) perforation caused by proximal colonic distention away from the site of stent placement because of inadequate colonic decompression or excessive air insufflation." Last, this meta-analysis was based largely on observational studies, given the paucity of randomized, controlled trials on colonic stenting. Therefore, there is the possibility of an unmeasured confounder influencing our results. Irrespective of the aforementioned limitations, the choice to perform this meta-analysis provides a possible estimation of perforation incidence and outcome from available published data. Instead of a pooled analysis in which all data from studies are simply accumulated, a meta-analysis applies weight to the studies according to population size and number of events, providing insight into the heterogeneity.

In conclusion, this extensive literature review provides insight into perforation after colorectal stent placement. These aggregated data show that the perforation rate after colonic stent placement is 7.4%. In the search for risk factors, we found significant differences in perforation between the most frequently used stent designs. Benign strictures, specifically diverticular, and bevacizumab-based chemotherapy for malignant strictures increase the risk of perforation. Stricture dilation and concomitant chemotherapy were not identified as risk factors. Perforation in the subgroup of postprocedural reintervention dilation was increased almost threefold compared with the nondilation group. To learn more about perforation in colorectal stenting, we call for more detailed reporting on perforation in future research, including subgroup analyses, pathological reports, and further data on the circumstances in which perforation occurs.

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TABLE 8. Continued

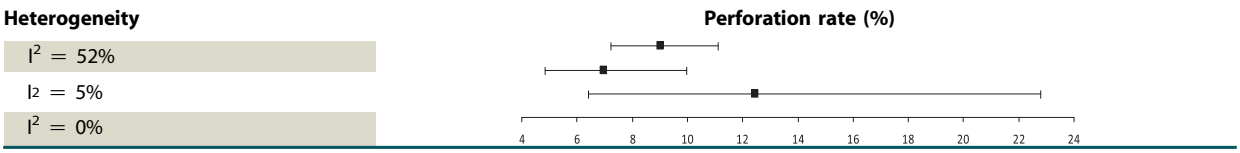

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TABLE 1. Study characteristics

Study	Country	Design	Stent pop.(N)	Follow-up with stent in situ Median (range)	Etiology (N)	Deployment technique (N)	Inability to pass stricture N (%)	Inserted stents (N)	Perf. N (%)	Stricture dilation N (%)	Concomit. chemo N (%)	Concomit. bevacizu N (%)
Abbas 2009[52]	USA	CR	2	6 and 22mo	B	Endo/fluor	0	Polyflex 4	0	0	0	0
Alcantara 2007[53]	Spain	P	95	BTS: 5d (3-30) PAL: missing	M: 92 B: 3	Fluoroscopic	0	Wallstent 21 Esophacoil 5 Hanarostent 32 Wallflex 45	4 (4.2)	missing	missing	missing
Al Samaraee 2010[54]	UK	R	38	BTS: 20d (2-69) PAL: 315d	M	Endo/fluor	3 (7.9)	Wallstent 37	1 (2.6)	missing	missing	missing
Athreya 2006[30]	UK	R	102	Survival: (14d-2y)	M: 99 B: 3	Fluor 67 Endo/fluor 20	15 (14.7)	Memotherm 66 Wallstent 26 Ultraflex 2	4 (3.9)	Intrapr *mis* Reint 1 (1.0)	missing	missing
*Baerlocher 2008[55]	Canada	R	11	mean 81.9d (5-352)	M	Fluoroscopic	2 (18.2)	Ultraflex 2 Wallflex 9 Unkown 4	0	0	0	0
Baraza 2008[56]	UK	P	63	(2d-40mo)	M	Endo/fluor	missing	Niti-S covered 3 Niti-S D-type (*mis*) Memotherm (*mis*) Total 63	1 (1.6)	Intrapr 0	10 (15.9)	missing
*Bielawska 2010[57]	Canada	R	30	mean 15.2mo (6-26)	M	Endo/fluor	0	Wallflex 31 Wallstent 2	0	0	10 (33.3)	0
*Branger 2010[58]	France	P	93	BTS: 15mo (12-42) PAL: 7mo (3d-37mo)	M	Endo/fluor	3 (3.2)	Hanarostent 68 Wallstent 25	3 (3.2)	Intrapr 93 (100) Reint 7 (7.5)	0	0
*Brehant 2009[59]	France	P	30	BTS: mean 7.5d (5-14)	M: 28 B: 2	Endo/fluor	3 (10.0)	Wallflex 27	5 (16.7)	0	0	0
*Caceres 2008[60]	USA	R	35	7.7mo (3.19-11.9)	M	Endo/fluor	8 (22.9)	Wallstent 29	0	Intrapr 29 (82.9)	27 (77.1)	0
Cennamo 2009[22]	Italy	*mis*	28	BTS: (4-78d) PAL: 131d (11-324)	M	Endo/fluor	0	Wallflex 28	2 (7.1)	0	9 (32.1)	2 (7.1)
Chang 2011[61]	Korea	R	77	8mo (3-13)	M	Fluoroscopic	1 (1.3)	Design missing Total 78	1 (1.3)	missing	missing	missing
*Cheung 2009[62]	China	RCT	24	BTS: 10d (2-16)	M	Endo/fluor	4 (16.7)	Wallstent 20	0	0	0	0
Choi 2007[63]	Korea	*mis*	37	mean 116d (3-319)	M	Fluor 32 Endo/fluor 5	4 (10.8)	Choostent cov 21 Choostent unco 12	0	missing	missing	missing

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TABLE 1. Continued

Study	Country	Design	Stent pop.(N)	Follow-up with stent in situ Median (range)	Etiology (N)	Deployment technique (N)	Inability to pass stricture N (%)	Inserted stents (N)	Perf. N (%)	Stricture dilation N (%)	Concomit. chemo N (%)	Concomit. bevacizu N (%)
Chung 2008[64]	Korea	R	17	BTS: 7d (2-11)	M	Endo/fluor	0	Wallflex (*mis*) Niti-S D-type (*mis*) Niti-S Comvi 1 Total 18	0	missing	missing	missing
*Crosta 2006[65]	Italy	P	24	9.8mo (<1-27)	M	Endo/fluor	1 (4.2)	Wallstent 22 Ultraflex 5 Niti-S covered 1	1 (4.2)	0	18 (75.0)	0
Dafnis 2007[66]	Sweden	CR	1	Until death	B	Endo/fluor	0	Wallstent 5	0	0	1	0
Dai 2010[67]	Germany	R	14	mean 41.2mo (3.5-123)	B	Endo/fluor	0	Polyflex 15 Ultraflex cov 8	0	Reint 3 (21.4) Intrapr *mis*	missing	missing
Dastur 2008[68]	UK	R	19	mean 21 ± 25mo	M	Endo/fluor	3 (15.8)	Wallstent (*mis*) Memotherm (*mis*) Total 16	1 (5.3)	missing	2 (10.5)	missing
Davies 2005[69]	UK	R	21	12mo (1-30)	M	Endo/fluor	4 (19.0)	Memotherm 17	1 (4.8)	missing	missing	missing
*Donatelli 2008[70]	Italy	CR	2	24mo	B	Endo/fluor	0	Niti-s covered 2	0	0	0	0
Donnellan 2010[71]	Ireland	R	43	Survival: 113 and 135d	M	Endo/fluor	3 (7.0)	Wallstent 40	2 (4.7)	0	missing	missing
Dronamraju 2009[72]	UK	R	16	BTS: mean 6w (1-12) PAL: 9mo	M	Endo/fluor	1 (6.3)	Wallstent cov 15	0	missing	missing	missing
Dulucq 2006[73]	France	P	11	BTS: mean 6.2d	M	Endo/fluor	0	Wallstent 8 Hanarostent 2 Unknown 1 (*excl*)	1 (9.1)	Intrapr 1 (9.1)	1 (9.1)	missing
*Elsberger 2008[74]	UK	P + R	7	4.3mo (3-12)	M	Endo/fluor	0	Wallstent 7	0	0	0	0
Faragher 2008[75]	Australia	R	29	14mo	M	Fluor *mis* Endo/fluor *mis*	0	Design missing Total 33	2 (6.9)	missing	missing	missing
*Fernández-Esparrach 2010[76]	Spain	R	47	130d (4-945)	M	Endo/fluor	1 (2.1)	Wallstent 41 Wallflex 3 Hanarostent 2	5 (10.6)	0	28 (59.6)	0
*Foo 2011[77]	China	R	130	BTS: mean 12d (4-69) PAL: 2mo (1.2-2.7)	M: 129 B: 1	Endo/fluor	0	Wallstent 66 Wallflex 22 Ultraflex 3 Wallstent eso 34 Choostent 5	2 (1.5)	missing	missing	missing

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TABLE 1. Continued

Study	Country	Design	Stent pop.(N)	Follow-up with stent in situ Median (range)	Etiology (N)	Deployment technique (N)	Inability to pass stricture N (%)	Inserted stents (N)	Perf. N (%)	Stricture dilation N (%)	Concomit. chemo N (%)	Concomit. bevacizu N (%)
Forshaw 2006[78]	UK	R	5	29mo (3-75)	B	Endo/fluor	0	Wallstent 1 Memotherm 4	0	0	missing	missing
*Frago 2010[79]	Spain	R	49	4.4mo (0.2-32.0)	M	missing	missing	Wallflex 38 Wallstent 7	2 (4.1)	0	37 (75.5)	6 (12.2)
Fregonese 2008[80]	Several (Europe)	P + R	36	BTS: 11d (7-15)	M	Fluor *mis* Endo/fluor *mis*	missing	Ultraflex 40	4 (11.1)	0	missing	missing
Galizia 2008[81]	Italy	R	3	16mo (5-61)	M	missing	0	Design missing Total 3	1 (33.3)	missing	3 (100)	missing
*García-Cano 2006[82]	Spain	R	175	BTS: 11d (1-149) PAL: 100d (5-246)	M	Endo/fluor 134 Endo 41	7 (4.0)	Wallstent 110 Hanarostent 23 Ultraflex 29 Unknown 2	7 (4.0)	0	30 (17.1)	0
Geiger 2008[83]	USA	CR	1	9mo	B	Endo/fluor	0	Polyflex 1	0	Intrapr 1 (100)	0	0
*Im 2008[84]	Korea	P	49	mean 331d (23-655)	M	Endoscopic	0	Hanarostent 51	2 (4.1)	0	23 (46.9)	8 (16.3)
*Iversen 2011[85]	Denmark	R	34	BTS: 35d (6-100)	M	Endo/fluor	0	Wallstent 11 Wallflex 6 Niti-S D-type 4 Unknown 13	4 (11.8)	0	0	0
*Jost 2007[41]	Switzerland	R	67	BTS: mean 7.2d (2-22) PAL: mean 92d (10-285)	M: 59 B: 8	Endo/fluor	7 (10.4)	Wallstent 73	8 (11.9)	Intrapr 10 (14.9) Reint *mis*	missing	missing
*Jung 2010[86]	Korea	R	39	111d	M	Endo/fluor	0	Niti-S covered 5 Hanarostent 1 Wallflex 16 Niti-S D-type 17	2 (5.1)	0	10 (25.6)	0
Karoui 2010[49]	France	P	35	6.3mo (0.5-51)	M	Endo/fluor	0	Design missing Total 41	2 (5.7)	0	19 (54.3)	0
*Keswani 2009[87]	USA	R	49	missing	M	Endo/fluor	4 (8.2)	Wallstent 34 Wallflex 11	3 (6.1)	Reint 1 (2.0)	17 (34.7)	3 (6.1)

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TABLE 1. Continued

Study	Country	Design	Stent pop.(N)	Follow-up with stent in situ Median (range)	Etiology (N)	Deployment technique (N)	Inability to pass stricture N (%)	Inserted stents (N)	Perf. N (%)	Stricture dilation N (%)	Concomit. chemo N (%)	Concomit. bevacizu N (%)
*Kim 2008[88]	Korea	R	42	BTS: (6-41d) PAL: 62d (0-1014)	M	Fluoroscopic	0	Hanarostent 33 Dual Stent 12	0	0	2 (4.8)	0
*Kim 2009[27]	Korea	P	122	mean 453d (3-2370)	M	Fluoroscopic	missing	Dual stent 124	7 (5.7)	Total 56 (45.9) Intrapr 55 (45.1) Reint 1 (0.8)	missing	missing
*Kim 2009[89]	Korea	R	35	BTS: mean 8.6 ± 5.5d	M	Endo/fluor	0	Niti-s D-type 18 Niti-s covered 13 Wallflex 4	0	0	0	0
*Kim 2010[90]	Korea	R	99	BTS: mean 10.3d PAL: mean 100d (2-455)	M	Fluoroscopic	1 (1.0)	Hanarostent 73 EGIS *eso* 43	0	0	81 (81.8)	21 (21.2)
Lee 2007[91]	Korea	P	80	BTS: *missing* PAL: (1-238d)	M	Endo/fluor	missing	Niti-s D-type 59 Niti-s covered 64	1 (1.3)	0	0	0
Lee 2010[92]	USA	R	46	mean 126d (2-1210)	M	Endo/fluor	0	Wallstent 32 Wallflex 14 Ultraflex 6 Polyflex 4	2 (4.3)	Intrapr 1 (2.2)	missing	missing
*Lee 2011[93]	Korea	R	71	9.63mo (0.6-43.2)	M	Endo/fluor	1 (1.4)	Wallflex 25 Comvi Stent 19 Niti-s D-type 17 Unknown 9	9 (12.7)	0	49 (69.0)	5 (7.0)
Li 2010[94]	China	P	52	BTS: mean 8d (4-11)	M	Endo/fluor	2 (3.8)	Micro-Tech 56	0	0	0	0
Lopes 2008[95]	Brazil	R	36	17w (0-138)	M	Endo/fluor	0	Hanarostent 29 Wallstent 13 Choostent 10	3 (8.3)	Intrapr 0 Reint *mis*	25 (69.4)	missing
Mates 2008[96]	Canada	CR	1	5mo	M	Endo/fluor	0	Wallflex 2	0	0	1 (100)	0
Modarai 2008[97]	UK	CR	1	Until surgery	B	Endo/fluor	0	Memotherm 1	1 (100)	0	0	0
Moon 2010[98]	Korea	P	68	BTS: 10.8d (5-28) PAL: (23-847d)	M	Endo/fluor	0	Niti-s D-type 37 Comvi Stent 33	1 (1.5)	missing	missing	missing

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TABLE 1. Continued

Study	Country	Design	Stent pop.(N)	Follow-up with stent in situ Median (range)	Etiology (N)	Deployment technique (N)	Inability to pass stricture N (%)	Inserted stents (N)	Perf. N (%)	Stricture dilation N (%)	Concomit. chemo N (%)	Concomit. bevacizu N (%)
*Nagula 2010[99]	USA	P	38	24w	M	Endo/fluor	6 (15.8)	Wallstent 26 Ultraflex 12	0	missing	missing	missing
*Park 2010[100]	Korea	RCT	151	(3.6-10.3mo)	M	Endo/fluor	2 (1.3)	Wallflex 75 Comvi Stent 74	0	Reint 1 (0.7)	81 (53.6)	8 (5.3)
*Park 2011[101]	Korea	R	103	(1-630d)	M	Endo/fluor	0	Wallstent 27 Niti-s D-type 20 Niti-s covered 24 Bonastent 28 Hanarostent 4	1 (1.0)	Total 14 (13.6) Intrapr 13 (12.6) Reint 1 (1.0)	27 (26.2)	0
*Phillips 2011[102]	UK	R	28	mean 10.7mo (5d-28mo)	M: 26 B: 2	missing	1 (3.6)	Ultraflex 13 Wallstent 9 Wallflex 2 Memotherm 3 Unknown 2	2 (7.1)	missing	missing	missing
*Pirlet 2011[15]	France	RCT	30	BTS: 7d (5-19)	M	Fluoro 13 Endo/(*mis*) 17	13 (43.3)	Memotherm 15 Wallstent 1	12 (40.0)†	0	0	0
*Pommergaard 2009[42]	Denmark	R	45	mean 162.9d BTS: mean 23.3d	M: 38 B: 7	Endo/fluor	missing	Wallstent 18 Wallflex 14 Ultraflex 2 Niti-s D-type 3 Other 4 Unknown 4	5 (11.1)	Intrapr 1 (2.2)	missing	missing
Ptok 2006[103]	Germany	P	48	251d (8-1120)	M	Endo/fluor	0	Wallstent (*mis*) Choostent (*mis*) Memotherm (*mis*) Total 56	0	missing	missing	missing
*Rayhanabad 2009[104]	USA	R	36	11mo (1-42)	M: 28 B: 8	Endo/fluor	8 (22.2)	Ultraflex (*mis*) Wallstent (*mis*) Z-stent (*mis*) Polyflex (*mis*) Ultraflex *eso* (*mis*) Total 41	1 (2.8)	Intrapr 4 (11.1)	missing	missing

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TABLE 1. Continued

Study	Country	Design	Stent pop.(N)	Follow-up with stent in situ Median (range)	Etiology (N)	Deployment technique (N)	Inability to pass stricture N (%)	Inserted stents (N)	Perf. N (%)	Stricture dilation N (%)	Concomit. chemo N (%)	Concomit. bevacizu N (%)
*Repici 2007[105]	Several (Europe)	P	44	6mo	M: 43 B: 1	Fluor *mis* Endo/fluor *mis*	1 (2.3)	Ultraflex 47 Wallstent 1	0	Total 8 (18.2) Intrapr 7 (15.9) Reint 1 (2.3)	26 (59.1)	3 (6.8)
*Repici 2008[106]	Italy	P	42	BTS: 5.0d (4.4-5.6) PAL: 208d (93-323)	M	Endo/fluor	1 (2.4)	Wallflex 48	1 (2.4)	0	missing	missing
*Reza 2009[107]	Iran	P	8	Until death	M	Endo/fluor	0	Niti-s D-type 8	0	Intrapr 8 (100)	6 (75.0)	0
*Shin 2008[108]	Korea	R	39	81d (2-640)	M	Endo/fluor	missing	Niti-s covered 19 Niti-s D-type 38	0	missing	14 (35.9)	missing
Shrivastava 2008[31]	UK	R	91	63d (IQR 20-270)	M	Fluor 81 Endo/fluor 10	10 (11.0)	Wallflex 37 Memotherm 44	10 (11.0)	Reint 2 (2.2)	missing	missing
*Small 2008[43]	USA	R	23	6mo (0.75-75)	B	Endo/fluor	0	Ultraflex 11 Wallstent 11 Ultraflex *eso* 4	2 (8.7)	Total 5 (21.7) Intrapr 2 (8.7) Reint 3 (13.0)	0	0
*Small 2010[109]	USA	R	233	BTS: 6d (4-10) PAL: 33.5d (1-2837)	M	Endo/fluor	2 (0.9)	Wallstent 129 Ultraflex 101 Wallflex 3 Unknown 34	18 (7.7)	Total 25 (10.7) Intrapr 22 (9.4) Reint 3 (1.3)	84 (36.1)	26 (11.2)
*Song 2007[28]	Korea	P	151	BTS: mean 7d (1-30) PAL: 152d (108-196)	M	Fluor 138 Endo/fluor 13	6 (4.0)	Dual Stent 147 PTFE-cov *eso* 8	16 (10.6)	Intrapr 39 (25.8)	missing	missing
*Soto 2006[110]	Spain	R	62	BTS: mean 7.7d (3-20) PAL: 197d (13-300)	M	Endo/fluor	3 (4.8)	Wallstent 63	3 (4.8)	0	26 (41.9)	0
Stefanidis 2005[111]	USA	R	21	BTS: 5d (1-12) PAL: 11mo (5-15)	M: 19 B: 2	Endo/fluor 16 Fluor 5	4 (19.0)	Wallstent 17 Wallstent *eso* 1	1 (4.8)	missing	missing	missing
Stenhouse 2009[112]	UK	P	72	missing	M	Endo/fluor	5 (6.9)	Wallstent 70 Memotherm 3	2 (2.8)	missing	missing	missing
*Stipa 2008[113]	Italy	*mis*	31	BTS: mean 11d (1-21) PAL: mean 3mo (2-10)	M	Endo/fluor	0	Ultraflex 31	1 (3.2)	Intrapr 31 (100)	3 (9.7)	0

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TABLE 1. Continued

Study	Country	Design	Stent pop.(N)	Follow-up with stent in situ Median (range)	Etiology (N)	Deployment technique (N)	Inability to pass stricture N (%)	Inserted stents (N)	Perf. N (%)	Stricture dilation N (%)	Concomit. chemo N (%)	Concomit. bevacizu N (%)
*Súarez 2010[114]	Spain	R	45	7.3mo	M	Endo/fluor	0	Hanarostent 45	2 (4.4)	0	25 (55.6)	0
Suh 2010[115]	Korea	R	55	211d (151-271)	M	Endo/fluor	0	Hanarostent 56	1 (1.8)	0	missing	missing
*Syn 2005[116]	UK	R	17	(3-197d)	M: 14 B: 3	Endo/fluor	2 (11.8)	Wallstent 10 Ultraflex 6 Z-stent 1	0	0	6 (35.3)	0
Trompetas 2010[117]	UK	R	11	2mo	M	Fluoroscopic	missing	Design missing Total 5	1 (9.1)	missing	missing	missing
*Tsurumaru 2007[118]	Japan	*mis*	12	mean 133d (9-534)	M	Endo/fluor	0	Ultraflex 12	0	0	3 (25.0)	0
*Van Hooft 2008[13]	Netherlands	RCT	10	360d (IQR 86-593)	M	Endo/fluor	1 (10.0)	Wallflex 10	6 (60.0)	0	7 (70.0)	1 (10.0)
*Van Hooft 2011[14]	Netherlands	RCT	43	BTS: missing	M	Endo/fluor	8 (18.6)	Wallstent 31 Wallflex 8	9 (20.9)	0	1 (2.3)	1 (2.3)
*Varadarajulu 2011[119]	USA	R	12	Until death	M	Endo/fluor	0	Ultraflex 12	0	missing	missing	missing
*Varpe 2008[120]	Finland	P	26	mean 178d (3-675)	M	Endo/fluor	7 (26.9)	Ultraflex 16 Hanarostent 3	3 (11.5)	0	11 (42.3)	2 (7.7)
Vemulapalli 2010[121]	USA	R	53	24w (2-196)	M	Endo/fluor	3 (5.7)	Wallstent (*mis*) Wallflex (*mis*) Total 55	6 (11.3)	missing	missing	missing
Vitale 2006[122]	Italy	P	57	missing	M	Endo/fluor	3 (5.3)	Wallstent 23 Ultraflex 33	1 (1.8)	Intrapr 0 Reint *mis*	missing	missing
Wada 2005[123]	Japan	CR	1	32mo	B	Endoscopic	0	Other 1	1 (100)	0	0	0
Watson 2005[32]	UK	R	107	28mo (2w-6y)	M: 100 B: 7	Fluoroscopic	5 (4.7)	Memotherm 78 Wallstent 2 Ultraflex 2 Wallstent *eso* 30	2 (1.9)	0	missing	missing
Young 2011[124]	Australia	P	100	34.5mo (1-64)	M: 93 B: 7	Fluor 8 Endo/fluor 92	7 (7.0)	Wallstent (*mis*) Ultraflex (*mis*) Wallflex (*mis*) Total 91	5 (5.0)	missing	missing	missing

pop. = population; *perf.* = perforations; *concomit.* = concomitant; *chemo* = chemotherapy; *bevacizu* = bevacizumab; *CR* = case report; *P* = prospective; *R* = retrospective; *Endo* = endoscopic guidance; *Fluor* = fluoroscopic guidance; *M* = malignant; *B* = benign; *BTS* = bridge to surgery group; *PAL* = palliative group; *d* = days; *w* = weeks; *mo* = months; *y* = years.

*= received additional data; *mis* = missing; *cov* = covered; *uncov* = uncovered; *excl* = excluded from analysis; *eso* = esophageal; *Intrapr* = intraprocedural stricture dilation (pre- or poststenting); *Reint* = stricture dilation as reintervention with stent in situ.

†one perforation occurred in a nonrandomized patient.