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Development of Desmoid Tumors After Ileorectal Anastomosis Versus Ileal Pouch-Anal Anastomosis in Familial Adenomatous Polyposis

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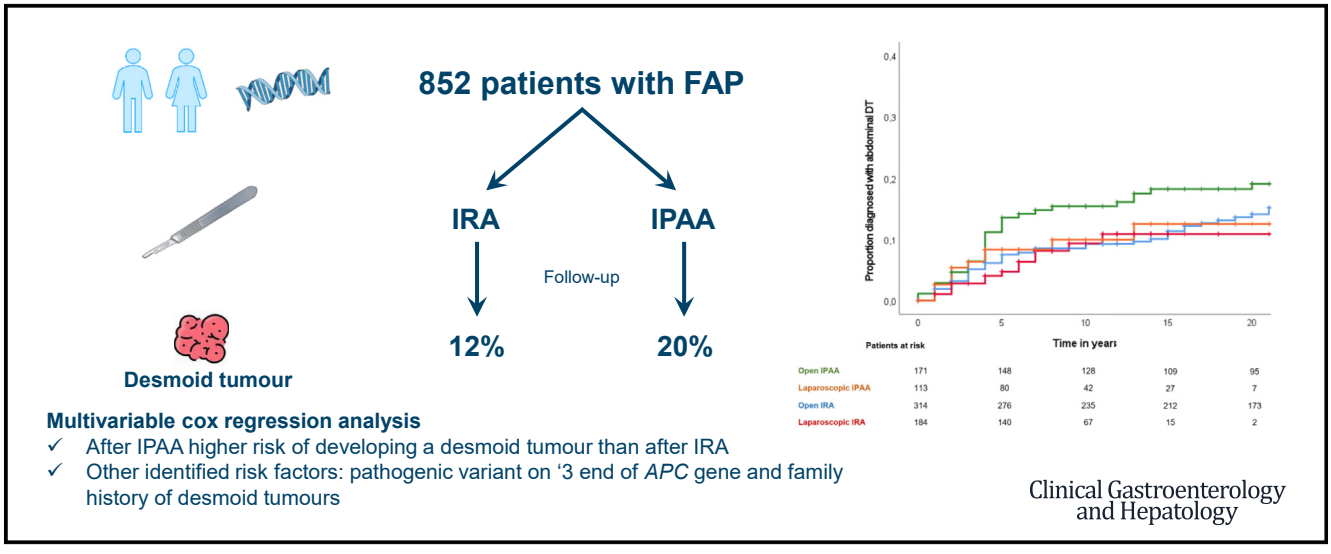

Abbreviations used in this paper: CT, computed tomography; DT, desmoid tumors; FAP, familial adenomatous polyposis; IPAA, ileal pouch-anal anastomosis; IRA, ileorectal anastomosis; MRI, magnetic resonance imaging.

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Most current article

BACKGROUND & AIMS: **Desmoid tumors (DT) are an important cause of morbidity and mortality in patients with familial adenomatous polyposis (FAP). DT development might be related to the type and approach of colectomy. We aimed to compare DT development after colectomy with ileorectal anastomosis (IRA) and proctocolectomy with ileal pouch-anal anastomosis (IPAA).**

METHODS: **We performed an international historical cohort study in patients with FAP who underwent IRA or IPAA between 1961 and 2020. The primary outcome was the incidence of abdominal DT (either mesenteric, retroperitoneal, or abdominal wall). Patients with a DT diagnosis before or at colectomy were excluded. Time to DT was considered censored at an eventual secondary proctectomy after IRA. We used multivariable Cox regression modelling to adjust for potential confounders.**

RESULTS: **We analyzed data from 852 patients: 514 after IRA and 338 after IPAA (median follow-up, 21 and 16 years, respectively). DTs were diagnosed in 64 IRA patients (12%) and 66 IPAA patients (20%). The cumulative DT incidence at 5 and 10 years was 7.5% and 9.3% after open IRA and 4.7% and 10.9% after laparoscopic IRA. These estimates were 13.6% and 15.4% after open IPAA and 8.4% and 10.0% after laparoscopic IPAA. The postoperative risk was significantly higher after IPAA ($P < .01$) in multivariable analysis, whereas approach did not significantly influence the risk.**

CONCLUSIONS: **The risk of developing an abdominal DT was found to be significantly higher after IPAA than after IRA. Postoperative DT risk should be taken into account when choosing between IRA and IPAA in FAP.**

Keywords: Familial Adenomatous Polyposis; Colectomy; Desmoid Tumors.

Patients with familial adenomatous polyposis (FAP) undergo colectomy at a young age followed by lifelong endoscopic surveillance, which helps to prevent colorectal cancer in most patients.[1] After colectomy, desmoid tumors (DT) are among the most common causes of death in patients with FAP.[2] DT are myofibroblastic proliferations that occur in 10%–20% of patients with FAP and most often develop in the small bowel mesentery or abdominal wall.[3,4] DT are benign nonmetastasizing tumors but they can invade into surrounding tissues, which can cause major morbidity and mortality. Figure 1 shows an example of an intra-abdominal DT on cross-sectional imaging and a photograph of a resected DT.

Reported risk factors for the development of DT in FAP include female sex, a germline pathogenic variant on the 3' end of the *APC* gene, a family history of DT, and a history of abdominal surgery.[3,5] Of these factors, abdominal surgery might be the only modifiable factor. Although refraining from colectomy is not feasible in most patients with FAP, choosing the type of colectomy and subsequent restorative procedure may be informed by the subsequent DT risk.

In daily practice, patients generally undergo a total colectomy with ileorectal anastomosis (IRA) or proctocolectomy with ileal pouch-anal anastomosis (IPAA). Up to 85% of DT in FAP are diagnosed after colectomy.[3] Usually, these tumors arise shortly after surgery, after a median of 3.2 years,[3,5] implying a relationship to surgery.

One could argue that a more extensive procedure, including both abdominal and pelvic surgery and lengthening maneuvers with stretching of the mesentery when constructing an IPAA, could trigger DT formation. Two meta-analyses demonstrated no difference in the DT incidence in patients undergoing either IPAA or IRA. These meta-analyses pooled reported incidence ratios and did not adjust for confounders.[6,7] Because the etiology of DT in FAP is multifactorial, it is of importance to take into account other known risk factors when studying the relationship between type of colectomy and DT risk. Three studies reported a multivariable analysis in which IPAA was associated with DT formation when adjusting for other variables.[8–10] Patients who underwent laparoscopic surgery were found to have a lower risk of developing DT.[8,10] These studies were relatively small or did not adjust for all known DT risk factors. We believed that a large multicenter study could add robust evidence on DT risk after different types of colectomy. We compared total colectomy and IRA with proctocolectomy and IPAA in terms of postoperative intraabdominal, retroperitoneal, and abdominal wall DT risk, taking into account all known risk factors.

Methods

Design and Study Population

We performed a multicenter historical cohort study in 7 expert centers for FAP, located in the Netherlands,

United Kingdom, Denmark, Spain, and Italy, all members of the European FAP Consortium. Patients with FAP who had undergone total colectomy and IRA or proctocolectomy and IPAA between 1968 and 2022 were included. A diagnosis of FAP was confirmed genetically by the presence of a germline pathogenic variant in the *APC* gene or clinically by the presence of at least 100 colorectal adenomas. Patients who were diagnosed with an intraabdominal or abdominal wall DT before or during colectomy were excluded. The institutional review boards of all centers approved the study. Informed consent of patients was required only in centers in the Netherlands, Denmark, and Italy.

Data Collection

Demographics and details on FAP diagnosis, including the pathogenic variant site, were collected. Mutation site was classified into 3 groups: at the 5' end of codon 1250, in between codon 1250 and 1450, and at the 3' end of codon 1450. A patient with a pathogenic variant at the 3' end of codon 1450 was considered to be at increased risk of developing DT.[5] Surgical data included the type of colectomy (IRA or IPAA) and approach of colectomy (open or laparoscopic). The total number of abdominal operations was also collected.

An abdominal DT diagnosis was defined as a radiologic diagnosis, a histologic diagnosis, or an optical diagnosis of DT during abdominal surgery. Data on DT included the year of diagnosis, and the location of the DT divided into intraabdominal DT (mesenteric or retroperitoneal) and abdominal wall DT. A positive family history of DT was defined as having 1 or more first-degree relatives with a DT diagnosis.

In patients without a diagnosis of DT, information was collated on whether they had had imaging, computed tomography (CT), or magnetic resonance imaging (MRI) for any indication at least 5 years after colectomy without signs of DT. Because DT develop usually shortly after (procto)colectomy, we thereby aimed to lower the chance of labelling patients as having no DT while they might have asymptomatic DT. In none of the participating hospitals do patients with FAP have (regular) CT or MRI scans to actively screen for DTs. However, cross-sectional imaging was performed in some patients for other reasons, such as the development of new symptoms, or because of concern due to a family history of DT.

What You Need to Know

Background

Patients with familial adenomatous polyposis (FAP) undergo colectomy and ileorectal anastomosis (IRA) or proctocolectomy with ileal pouch-anal anastomosis (IPAA) to prevent colorectal cancer. Patients are also at risk of desmoid tumors (DT), usually developing after surgery. The aim of this study was to compare IRA to IPAA in terms of post-operative abdominal DT risk.

Findings

After IRA, 12% of patients developed an abdominal DT compared to 20% of patients after IPAA. After adjusting for potential confounders in multivariable analysis, the post-operative risk of developing DT was significantly higher after IPAA, while surgical approach (open or laparoscopic) did not significantly influence DT risk.

Implications for patient care

In FAP, performing IRA instead of IPAA is preferred in terms of post-operative DT risk. However, an IRA should only be performed when the rectal polyposis can be safely managed endoscopically.

Statistical Analysis

The proportion of patients developing DT was estimated using the Kaplan-Meier method in 4 groups: after open/laparoscopic IRA versus open/laparoscopic IPAA. For patients with IRA who underwent a secondary proctectomy, time to event was considered censored at

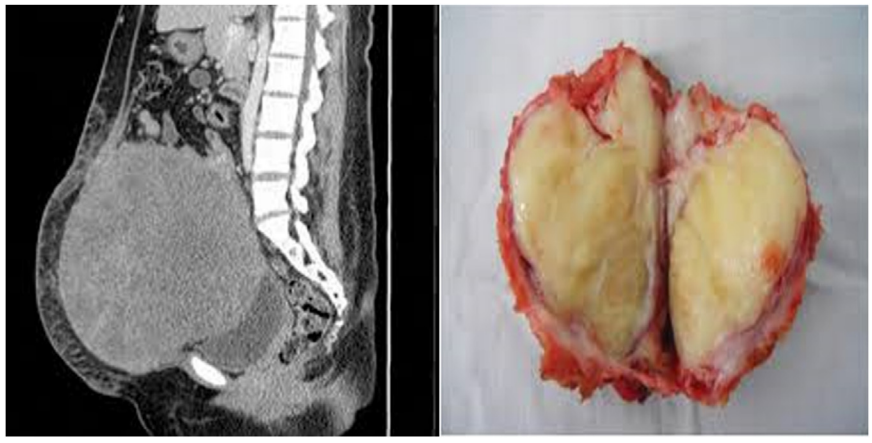

Figure 1. An intraabdominal desmoid tumor on cross-sectional imaging (*left*) and a photograph of a resected desmoid tumor (*right*).

the time of proctectomy. A multivariable Cox proportional hazard regression analysis was performed to evaluate differences between groups, adjusting for the following potential confounders: sex, *APC* pathogenic variant site, family history of DT, number of undergone abdominal operations, age at (procto)colectomy, and country. In the first model we included 4 groups: open IRA, laparoscopic IRA, open IPAA, and laparoscopic IPAA. In the second model we excluded approach and only included IRA and IPAA. To study the potential effect of abdominal operations on DT risk, the number of previous surgeries was included as a time-dependent variable. All analyses were performed using SPSS version 28.0 (IBM SPSS Statistics for Windows, Armonk, NY).

Results

Patient Characteristics

Table 1 summarizes the characteristics of the included patients. In total, 852 patients (52% female sex) without an abdominal DT diagnosis before or at time of (procto)colectomy were included, of which 514 had undergone total colectomy with IRA and 338 proctocolectomy with IPAA. Sixteen patients with a DT diagnosis before (procto)colectomy (n = 5) or at time of (procto) colectomy (n = 11) were excluded. A pathogenic variant at the 3' end of codon 1450 was present in 35 (9%) IRA and 41 (16%) IPAA patients. The IPAA group underwent surgery at a significantly higher age than the IRA group (median, 27 years vs 21 years; $P < .01$). Laparoscopic surgery was performed in 185 (37%) IRA patients and 114 (37%) IPAA patients. Two hundred and thirty-two (45%) and 231 (68%) patients underwent 1 or more other abdominal operations before or after (procto) colectomy in the IRA and IPAA group, respectively. Of note, closure of an ileostomy after IPAA was also counted as abdominal operation. Significantly more IRA than IPAA patients had 1 or more first-degree relatives with DT: 123 (24%) versus 45 (13%). The median duration of follow-up was significantly longer in the IRA group (21 vs 16 years; $P < .01$). One patient in the IRA group and 1 patient in the IPAA group were diagnosed with an extraabdominal DT before surgery.

Abdominal Desmoid Diagnosis After Ileorectal Anastomosis Versus Ileal Pouch-Anal Anastomosis

Table 2 shows the characteristics of diagnosed DTs. Within the entire cohort, 130 patients (15%) were diagnosed with 1 or more abdominal DT after (procto) colectomy: 64 (12%) in the IRA group and 66 (20%) in the IPAA group. Five of 96 (5%) patients developed a DT in Spain/Italy, 5/78 (6%) in Denmark, 25/196 (13%) in the Netherlands, and 95/481 (20%) in the United

Table 1. Patient Characteristics

	IRA (n = 514)	IPAA (n = 338)	*P* value
Female sex, n (%)	274 (53)	165 (49)	.22
Known pathogenic variant in *APC*, n (%)	482 (94)	310 (92)	.45
Pathogenic variant location, n (%)			
5' end of codon 1250	319 (81)	165 (63)	
Between codon 1250 and 1450	42 (11)	55 (21)	< .01
3' end of codon 1450	35 (9)	41 (16)	
Median age at (procto)colectomy (IQR)	21 (17–32)	27 (19–35)	< .01
Year of (procto)colectomy, n (%)			
1968–1980	38 (7)	4 (1)	< .01
1981–1990	150 (29)	37 (11)	< .01
1991–2000	91 (18)	89 (26)	< .01
2001–2010	105 (20)	117 (35)	< .01
2011–2022	130 (25)	91 (27)	.60
Approach of (procto)colectomy, n (%)			
Open	316 (63)	194 (63)	1.00
Laparoscopic	185 (37)	114 (37)	
Total number of abdominal operations, n (%)			
1 operation ([procto]colectomy)	282 (55)	107 (32)	< .01
2 operations	93 (18)	121 (36)	
3 or more operations	139 (27)	110 (33)	
First-degree relative with DT, n (%)	123 (24)	45 (13)	< .01
Median duration of follow-up, y	21 (10–32)	16 (9–25)	< .01

DT, desmoid tumors; IPAA, ileal pouch-anal anastomosis; IQR, interquartile range; IRA, ileorectal anastomosis.

Table 2. Characteristics of DT Diagnosed After (Procto)Colectomy

	IRA (n = 514)	IPAA (n = 338)	*P* value
Postoperative abdominal DT, n (%)	64 (12)	66 (20)	< .01
Location of abdominal DT			
Mesentery	57	52	.81
Retroperitoneal	0	2	
Abdominal wall	27	25	
Abdominal symptoms and intraabdominal DT, n (%)[a]	38 (59)	44 (67)	.39
Imaging screening for DT, n (%)	8 (2)	11 (3)	.15
No DT diagnosis and CT/MRI >5 y after surgery without DT, n (%)	144/450 (32)	116/272 (43)	< .01

CT, computed tomography; DT, desmoid tumors; IPAA, ileal pouch-anal anastomosis; IRA, ileorectal anastomosis; MRI, magnetic resonance imaging.
[a]Abdominal symptoms at time of DT diagnosis that might have been caused by the DT.

Kingdom. The median time from surgery to DT diagnosis was 6 years after IRA and 4 years after IPAA. Of the 163 abdominal DTs that were diagnosed in this cohort, 109 (67%) were located in the mesentery, 52 (32%) in the abdominal wall, and 2 (1%) in the retroperitoneal space. At time of DT diagnosis, 38 (67%) IRA patients and 44 (83%) IPAA patients had abdominal symptoms that may have been caused by DT. One hundred forty-four (32%) IRA patients and 116 (43%) IPAA patients without a diagnosis of DT had undergone 1 or more CT/MRI scans at least 5 years after IRA/IPAA that did not show DTs.

Fourteen patients (2%) developed an extraabdominal DT after a median of 18 years after surgery. Given the median time to extraabdominal DT development, we believe that they are unlikely to be related to surgery and therefore have not been included in further analyses.

Figure 2 shows the Kaplan-Meier curves, reflecting the estimated proportion of patients diagnosed with abdominal DT after open IRA, laparoscopic IRA, open IPAA, and laparoscopic IPAA. The unadjusted proportions at 5 and 10 years were 7.5% and 9.3% in the open IRA group and 4.7% and 10.9% in the laparoscopic IRA group. Within the IPAA group, the unadjusted proportions at 5 and 10 years were nonsignificantly higher in the open group: 13.6% and 15.4% in the open IPAA group and 8.4% and 10.0% in the laparoscopic IPAA group.

Adjustment for Potential Confounders

In the first Cox proportional hazard regression model, including the different type of operations (open IRA, laparoscopic IRA, open IPAA, and laparoscopic IPAA),

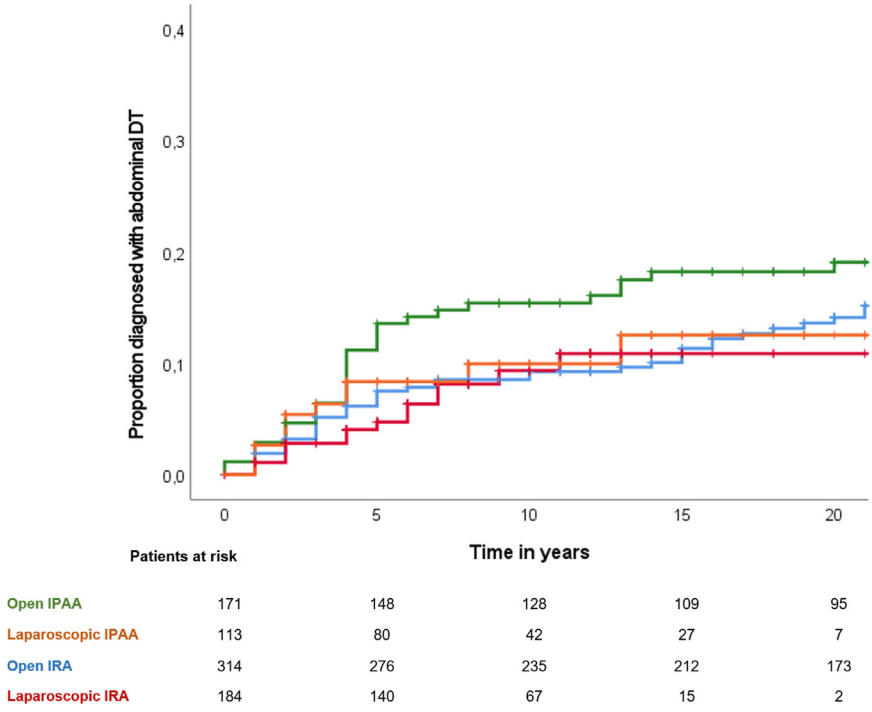

Figure 2. Proportion of patients diagnosed with abdominal DT.

approach (open or laparoscopic) did not significantly influence the DT risk in both the IRA group and in the IPAA group. When not including approach in the second model, the risk of an abdominal DT diagnosis was significantly higher after IPAA than after IRA (hazard ratio, 1.75; 95% confidence interval, 1.12–2.75; $P = .02$). In this analysis, the risk was found to be also higher in case of a pathogenic variant at the '3 end of the *APC* gene and having a first-degree relative with DT (Table 3).

Discussion

In this large multicenter study, we evaluated the risk of abdominal DT after colectomy with IRA and proctocolectomy with IPAA. The 5-year risk varied between 5% and 14%. The risk of an abdominal DT diagnosis was significantly higher after IPAA than after IRA, whereas approach (open or laparoscopic) did not significantly influence this risk. Highest DT risks were observed in patients with a pathogenic variant at the 3' end of *APC* gene and in patients with a family history of DT. Most DTs were observed in the mesentery and were diagnosed in the first years after surgery. Most patients had abdominal complaints at time of DT diagnosis that may have been caused by the DT; these were the primary reason to perform imaging.

Overall, 15% of patients in our study cohort were diagnosed with 1 or more abdominal DT after IRA/IPAA. The previously reported incidences of DT after IRA/IPAA vary. In a large study from Nieuwenhuis et al,[3] a comparable percentage of 11% (163/1546 patients) developed DT after IRA/IPAA, whereas in a recent study 29% (100/345) developed DT after IRA/IPAA when only including abdominal DT with abdominal symptoms. This is a high incidence keeping in mind that more recent studies might have lower DT incidences because of more laparoscopic procedures, because laparoscopic surgery was shown to result in a lower DT risk.[8,11] The higher incidence in the Sommovilla et al[10] study might be caused by a study population that is more prone to develop DT, such as epidemiologically or because of the expertise of the center (referral bias).

In 3 other studies including a multivariable analysis, undergoing IPAA was found to be significantly associated with developing a DT after surgery.[8–10] Whether surgical approach (open or laparoscopic) influences DT risk is debatable. Two studies found that laparoscopic surgery resulted in lower DT risks,[8,11] whereas 2 other studies including this study did not find differences between open and laparoscopic procedures.[10]

There were several differences in baseline characteristics between the IRA and IPAA group in our study. The IRA group was significantly younger than the IPAA group at time of (procto)colectomy. There has been discussion on whether the timing of colectomy influences DT risk and studies show contrary results.[3,5,9,12] Most studies did not show such an association in a multivariable analysis, except for Durno et al,[12] who found that in female patients, early colectomy ($\leq$18 years) is associated with DT development.

We found a higher risk of an abdominal DT diagnosis after IPAA. IPAA surgery policies differ between centers, which might also influence DT risk. Some centers advocate a 1-stage IPAA to lower the number of operations, whereas other centers prefer a 2-stage IPAA with temporary loop ileostomy to lower the risk for early anastomotic leakages and mitigate the severity of morbidity related to these early leaks. Closure of the ileostomy in a 2-stage IPAA might result in additional surgical trauma and therefore increase DT risk.

The introduction of transanal minimal invasive surgery during proctocolectomy has resulted in a less invasive procedure and thereby might result in a lower DT risk. More studies on different IPAA techniques and the influence on DT risk should be performed to minimize DT risk in patients who require IPAA surgery.

Some centers consider DT risk when choosing the type of colectomy, most preferring IRA in patients at increased risk of developing DT,[13] such as young female patients with a positive family history of DT or a pathogenic variant on the '3 end of the *APC* gene. This might result in selection bias and could partly explain the observed differences in baseline characteristics between

Table 3. Multivariable Cox Proportional Hazard Regression Analysis Adjusting for Potential Confounders

	Hazard ratio	95% CI	*P* value
Female sex	1.50	0.99–2.27	.06
Pathogenic variant at 3' end of *APC* gene	2.28	1.27–4.09	< .01
First-degree relative with DT	3.00	1.96–4.62	< .01
Number of undergone abdominal operations (increasing by 1)	1.17	0.88–1.55	.29
IPAA vs IRA	1.91	1.26–2.90	< .01

NOTE. Besides the variables included in the table, the model adjusted for age at (procto)colectomy and country of the centers.
CI, confidence interval; DT, desmoid tumors; IPAA, ileal pouch-anal anastomosis; IRA, ileorectal anastomosis.

our IRA and IPAA group. A multivariable analysis, considering these factors, is crucial to reduce bias in documenting the effect of colectomy type and approach on DT risk. Yet, we acknowledge that residual confounding should be considered when interpreting our findings.

We cannot be certain that all DT diagnosed in our cohort developed after surgery. There is a chance that (small) DT were not detected during (procto)colectomy and were diagnosed years later. In that case, surgery was not the trigger for DT development in the first place, although it might have triggered further progression. Another limitation is that not all patients had CT/MRI after surgery. We cannot claim that patients without a DT diagnosis did not have asymptomatic DT, which might have resulted in an underestimation of DT incidence. Performing imaging in every patient before undergoing (procto)colectomy and at 5 years after surgery could attenuate these limitations. Although this would help in further studying DT development after surgery in FAP and might result in earlier detection of DTs, it also results in an additional burden for the patient and costs. Another limitation might be that we did not have information on pregnancy or other causes of hormonal changes that might influence DT risk. Although in studies in FAP there is no robust evidence that these factors result in higher DT risk with even 1 study showing an improved desmoid course after pregnancy,[14–16] studies in the sporadic setting suggest an increased desmoid risk.[17]

Postoperative DT risk is a factor to take into account when deciding between IRA and IPAA. Nevertheless, rectal polyp burden and colorectal cancer risk probably remain the most important factors when choosing the type of surgery. Traditionally, for patients with FAP, having >20 rectal polyps at time of colectomy was an indication to perform IPAA. Recent studies show a low risk of rectal cancer in patients with IRA,[1,18] even in a cohort of patients with up to 50 rectal polyps at time of surgery.[1] When high-quality postoperative endoscopic surveillance is available and patients are motivated, it might be possible to safely perform IRA in patients with >20 rectal polyps at time of surgery, which might be favorable in patients with FAP in general but especially for those patients at risk of DT having a high rectal polyp burden. Before providing recommendations, further research is needed to evaluate whether this is a safe strategy.

DT are a major cause of morbidity and mortality in patients with FAP. Minimizing the risk of developing DT should be an element in the clinical management of these patients. Abdominal surgery is the only known modifiable risk factor for DT. We observed differences in risk after the respective surgical procedures, with a significantly higher risk of postoperative DT detection after IPAA. Additional efforts are needed to understand how to reduce postoperative DT risk in patients with FAP.

References

1. Anele CC, Xiang J, Martin I, et al. Regular endoscopic surveillance and polypectomy is effective in managing rectal adenoma progression following colectomy and ileorectal anastomosis in patients with familial adenomatous polyposis. Colorectal Dis 2022;24:277–283.
2. Ghorbanoghli Z, Bastiaansen BA, Langers AM, et al. Extracolonic cancer risk in Dutch patients with APC (adenomatous polyposis coli)-associated polyposis. J Med Genet 2018; 55:11–14.
3. Nieuwenhuis MH, Lefevre JH, Bulow S, et al. Family history, surgery, and APC mutation are risk factors for desmoid tumors in familial adenomatous polyposis: an international cohort study. Dis Colon Rectum 2011;54:1229–1234.
4. Heiskanen I, Jarvinen HJ. Occurrence of desmoid tumours in familial adenomatous polyposis and results of treatment. Int J Colorectal Dis 1996;11:157–162.
5. Sinha A, Tekkis PP, Gibbons DC, et al. Risk factors predicting desmoid occurrence in patients with familial adenomatous polyposis: a meta-analysis. Colorectal Dis 2011;13:1222–1229.
6. Aelvoet AS, Struik D, Bastiaansen BAJ, et al. Colectomy and desmoid tumours in familial adenomatous polyposis: a systematic review and meta-analysis. Fam Cancer 2022; 21:429–439.
7. Xie M, Chen Y, Wei W, et al. Does ileoanal pouch surgery increase the risk of desmoid in patients with familial adenomatous polyposis? Int J Colorectal Dis 2020;35:1599–1605.
8. Vitellaro M, Sala P, Signoroni S, et al. Risk of desmoid tumours after open and laparoscopic colectomy in patients with familial adenomatous polyposis. Br J Surg 2014; 101:558–565.
9. Saito Y, Hinoi T, Ueno H, et al. Risk factors for the development of desmoid tumor after colectomy in patients with familial adenomatous polyposis: multicenter retrospective cohort study in Japan. Ann Surg Oncol 2016;23(Suppl 4):559–565.
10. Sommovilla J, Liska D, Jia X, et al. IPAA is more "desmoidogenic" than ileorectal anastomosis in familial adenomatous polyposis. Dis Colon Rectum 2022;65:1351–1361.
11. Sinha A, Burns EM, Latchford A, et al. Risk of desmoid formation after laparoscopic versus open colectomy and ileorectal anastomosis for familial adenomatous polyposis. BJS Open 2018; 2:452–455.
12. Durno C, Monga N, Bapat B, et al. Does early colectomy increase desmoid risk in familial adenomatous polyposis? Clin Gastroenterol Hepatol 2007;5:1190–1194.
13. Chittleborough TJ, Warrier SK, Heriot AG, et al. Dispelling misconceptions in the management of familial adenomatous polyposis. ANZ J Surg 2017;87:441–445.
14. Nieuwenhuis MH, De Vos Tot Nederveen Cappel W, Botma A, et al. Desmoid tumors in a Dutch cohort of patients with familial adenomatous polyposis. Clin Gastroenterol Hepatol 2008; 6:215–219.
15. Church JM, McGannon E. Prior pregnancy ameliorates the course of intra-abdominal desmoid tumors in patients with familial adenomatous polyposis. Dis Colon Rectum 2000; 43:445–450.
16. Gurbuz AK, Giardiello FM, Petersen GM, et al. Desmoid tumours in familial adenomatous polyposis. Gut 1994;35:377–381.
17. Johner A, Tiwari P, Zetler P, et al. Abdominal wall desmoid tumors associated with pregnancy: current concepts. Expert Rev Anticancer Ther 2009;9:1675–1682.

18. Pasquer A, Benech N, Pioche M, et al. Prophylactic colectomy and rectal preservation in FAP: systematic endoscopic follow-up and adenoma destruction changes natural history of polyposis. Endosc Int Open 2021;9:E1014–e1022.

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Patrick M.M. Bossuyt (Conceptualization: Equal; Investigation: Equal; Methodology: Equal; Supervision: Equal; Writing – review & editing: Equal)

Andrew Latchford (Conceptualization: Equal; Investigation: Equal; Methodology: Equal; Writing – review & editing: Equal)

Roel Hompes (Conceptualization: Equal; Investigation: Equal; Methodology: Equal; Writing – review & editing: Equal)

Evelien Dekker, MD, PD (Conceptualization: Lead; Funding acquisition: Lead; Investigation: Lead; Methodology: Lead; Supervision: Lead; Writing – original draft)

Conflicts of interest

These authors disclose the following: Maria Pellise reports endoscopic equipment on loan from FujiFilm and Olympus; research grant from FujiFilm, ZiuZ, and Casen recordati; consultancy for FujiFilm and Olympus; and speakers' fee from Olympus, Fujifilm, Norgine, and IPSEN. Barbara A.J. Bastiaansen reports speakers' fee from Olympus, Tillotts Pharma AG, and Ovesco Endoscopy AG. Monique E. van Leerdam reports editorial fee from Elsevier. Rodrigo Jover reports consultancy for CPP Pharmaceuticals. Francesc Balaguer has received an honorarium for consultancy from Sysmex and CPP Pharmaceuticals; speaker's fees from Norgine; and editorial fee from Elsevier. Michal F. Kaminski received speaker's fee from Olympus, Fujifilm, Boston Scientific, Medtronic, AlfaSigma, IPSEN; and consultancy fee from Olympus, ERBE, and AlfaSigma. Luigi Ricciardiello reports consultancy and unrestricted research grant from SLA Pharma AG. John G. Karstensen reports consultancy for Boston Sci, SNIPR BIOME, and AMBU; and speakers' fee from Norgine. Roel Hompes reports speaker's fee from Medtronic and Stryker; and consultancy fee from Applied Medical. Evelien Dekker reports endoscopic equipment on loan from FujiFilm; research grant: FujiFilm; honoraria for consultancy from Olympus, Fujifilm, Ambu, and InterVenn; and speaker's fees from Olympus, GI Supply, Norgine, IPSEN, PAION, and FujiFilm. The remaining authors disclose no conflicts.

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