Best Practice & Research Clinical Gastroenterology 31 (2017) 389–399

Contents lists available at ScienceDirect

Best Practice & Research Clinical Gastroenterology

journal homepage: https://ees.elsevier.com/ybega/default.asp

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Management of duodenal polyps

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Keywords:
Duodenum
Adenoma
Epidemiology
Diagnosis
Detection
Endoscopic resection
Polypectomy
Endoscopic mucosal resection

A B S T R A C T

Duodenal adenomas are the most common type of polyp arising from the duodenum. These adenomas can occur within and outside of genetic syndromes, and are broadly classified as non-ampullary or ampullary depending on their location. All adenomas have malignant potential and are therefore appropriately treated by endoscopic resection. However, the unique anatomical properties of the duodenum, namely its relatively thin and vascular walls, narrow luminal diameter and relationship to the ampulla and its associated pancreatic and biliary drainage, pose an increased degree of complexity for any endoscopic interventions in this area. This review will discuss the epidemiology of duodenal adenomas, their endoscopic detection and diagnosis, and techniques for safe and effective endoscopic resection of ampullary and non-ampullary lesions.

Introduction

Ampullary and non-ampullary polyps of the duodenum are diagnosed within and outside of genetic syndromes. Adenomas are the most commonly encountered duodenal polyps, although hyperplastic polyps and other mucosal tumours such as Brunner's gland adenomas and hamartomas also occur. Similar to that of colonic adenomas, duodenal adenomas have malignant potential and endoscopic resection (ER) is the first line treatment. However, unique challenges to the management of duodenal adenomas exist, particularly with regard to heightened risks of procedural complications, largely owing to the duodenum's relatively thin, fixed and vascular walls. Endoscopic identification of duodenal adenomas can also be hampered by the similarly appearing, adjacent normal villiform duodenal mucosa. The optimal method of ER depends upon a range of factors including lesion location, size and morphology. In this review, we discuss the management of ampullary and non-ampullary duodenal adenomas with respect to their diagnosis, resection, management of complications and follow-up. Management of other small bowel polyps are discussed in a separate review in this issue.

Epidemiology of duodenal adenomas

Sporadic duodenal adenomas are uncommon, but are becoming more readily detected with greater adoption of diagnostic endoscopy and increasing use of high definition endoscopes. The majority of patients with duodenal adenomas are asymptomatic and these lesions are often detected incidentally during endoscopic evaluation for other gastrointestinal issues. Previous series have reported duodenal polyps to occur in up to 4.6% of patients presenting for gastroscopy, with the prevalence of sporadic non-ampullary adenomas being <0.5% [1–3]. The prevalence of sporadic ampullary adenomas is even lower, and estimated to be between 0.04 and 0.12% [4]. Most non-ampullary duodenal adenomas are found on the posterior or lateral walls of the descending duodenum, at the level or below the ampulla of vater. This may reflect a true anatomical distribution or be the result of a detection bias from forward viewing endoscopes and the anatomical configuration of the duodenum [5].

Although considered benign, management of duodenal adenomas (ampullary and non-ampullary) are of importance given their potential for progression to carcinoma, in a sequence similar to that occurring in colonic adenomas [6,7]. The progression from duodenal adenoma with low grade dysplasia to adenocarcinoma can take up to 15–20 years [8], slower than that for colonic adenomas. However larger lesions (≥20 millimetres [mm]) and those with high grade dysplasia have a high risk of harbouring invasive disease [5,9]. In addition, ampullary adenomas have a risk of malignancy much

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http://dx.doi.org/10.1016/j.bpg.2017.04.015
1521-6918/

higher than that of non-ampullary sporadic duodenal adenomas [10,11]. Therefore, it is recommended that all duodenal adenomas be resected when detected. However, given that the majority are only very slowly progressive and often occur in elderly, comorbid patients, the patient's overall health and anticipated longevity should be continuously factored into the clinical decision making. In addition, the presence of a duodenal adenoma is associated with colorectal neoplasia (relative risk [RR] 2.5–7.8 compared with average risk age-matched controls), and screening colonoscopy is also recommended for these patients [12,13].

Epidemiology of duodenal polyps associated with genetic syndromes

Duodenal adenomas occur in up to 90% of patients with familial adenomatous polyposis (FAP), an autosomal dominant polyposis syndrome characterised by germline mutations in the adenomatous polyposis coli (*APC*) tumour suppressor gene, located on chromosome 5q21-q22 [14,15]. Duodenal adenomas in patients with FAP are most commonly found in D2, often distal or adjacent to the papilla, although they may be encountered in the duodenal cap or third part. It is postulated that such a distribution could be related to the mucosal exposure to bile flow and its growth-promoting properties [5]. Similar to that of sporadic adenomas, the rate of progression to carcinoma for FAP associated adenomas is generally slow. Risk of carcinoma can be stratified according to the Spigelman system, which was developed to quantify the severity of duodenal adenomatosis (Table 1). The classification is based upon a 5 grade scale (0–IV) according to adenoma number (1–4, 5–20, or >20), size (1–4, 5–10, or >10 mm), histologic type (tubular, tubulovillous, or villous) and severity of dysplasia (mild, moderate, or severe) [16]. Those with stage IV disease have the highest risk [17–19], with a recent study of 218 registry patients enrolled over a 30 year follow-up period reporting the risk of developing duodenal carcinoma to be 2.1% (95% confidence interval [CI]: 0–5.2) at 15 years [17].

MUTYH-associated polyposis (MAP) is another polyposis syndrome associated with duodenal adenomas. MAP is an autosomal recessive condition caused by biallelic pathogenic variants in *MUTYH*, located on chromosome 1p32.1-p34.3. Patients with this syndrome have an increased risk of colorectal and other cancers. Duodenal adenomas are found in 17%–25% of patients with MAP, with a lifetime duodenal cancer risk of about 4% [20]. The data on prevalence and risk of progression to cancer for MAP is less comprehensive than that for FAP, although a recent contemporary study of 92 patients with MAP found duodenal polyposis (34% of cohort) developed less frequently than in patients with FAP, and developed at a later age (median age of 50 years at duodenal adenoma detection) [21]. Similar to FAP, increasing lesion size and villous change appears to promote adenoma progression in MAP [21].

Principles of endoscopic resection in the duodenum are generally the same for sporadic or non-sporadic, non-ampullary adenomas, and are discussed in detail below. Specific surveillance recommendations exist for duodenal polyposis associated with FAP and MAP [8,22]. For FAP (classic or attenuated) patients, upper endoscopic screening using a forward-viewing gastroscope and side-viewing duodenoscope should be initiated at the time of onset of colonic polyps or around age 25–30 years (whichever comes first). Surveillance should be repeated every 0.5–4 years depending on the Spigelman stage (0 = 4 years, I = 2–3 years, II = 1–3 years, III = 6–12 months, and IV = surgical evaluation) [22]. Guidelines suggest similar recommendations for duodenal adenoma surveillance in MAP [22].

Table 1
Grading system for duodenal polyposis (Spigelman classification) [16].

	Number of points		
	1	2	3
Polyp number	1–4	5–20	>20
Polyp size (mm)	1–4	5–10	>10
Histology	Tubular	Tubulovillous	Villous
Dysplasia	Mild	Moderate	Severe

Spigelman stage 0: 0 points; stage 1: 1–4 points; stage 2: 5–6 points; stage 3: 7–8 points; stage 4: 9–12 points.

Detection and diagnosis of duodenal adenomas

Early and accurate detection of duodenal adenomas allows optimal outcomes from ER. In recent years, advances in diagnostic endoscopy through introduction of high definition endoscopes, digital chromoendoscopy such as narrow band imaging (NBI), as well as conventional dye based chromoendoscopy have improved the endoscopic detection of duodenal adenomas [23–26]. For complete views of the duodenum including peri-ampullary regions, a side-viewing duodenoscope in addition to a forward-viewing scope is usually required. Use of a transparent distal cap attached to a gastroscope may also aid examination, particularly in potentially difficult to inspect areas such as behind mucosal folds of the posterolateral wall at the junction of the first and second parts of the duodenum.

On white light endoscopy (WLE), duodenal adenomas typically are flat or sessile, solitary and with a whitish surface villi appearance. In one study of 118 consecutive patients, conventional dye based chromoendoscopy with indigo carmine detected significantly more duodenal lesions than standard WLE (98 vs 28 lesions; $P = 0.0042$) [27]. Dye based chromoendoscopy however, is time-consuming and has not conclusively been shown to reliably differentiate duodenal adenomas from non-adenomas [28]. Magnification endoscopy with NBI may differentiate adenomatous from non-adenomatous ampullary lesions based upon villi appearance in a small pilot study of 14 patients [23]. The authors proposed that type I (oval-shaped villi) predicted inflammatory or hyperplastic changes, and type II (pinecone/leaf-shaped villi) or type III (irregular/non-structured) predicted adenoma and adenocarcinoma, respectively [23]. However, a recent prospective study of 37 FAP patients found examination with NBI resulted in no increased duodenal adenoma detection compared with high definition WLE [26]. Furthermore, the only endoscopic feature that predicted advanced histology in duodenal adenomas was size ≥10 mm, although this may be due to under-powering [26]. As such, the optimal imaging modality for duodenal adenoma detection and diagnosis remains to be defined. Use of NBI seems logical and practical, based on data elsewhere in the gastrointestinal tract, however further data is needed to confirm its role.

Every duodenal adenoma should be assessed for features of submucosal invasion (SMI) and suitability for ER. Features suggestive of SMI include a depressed component within the lesion (Paris 0-IIc), type 5 Kudo pit pattern, surface ulceration, and non-lifting sign following submucosal injection. Other factors that will assist in planning of endoscopic resection include lesion size, extent of duodenal circumference involved, and relationship to the ampulla of vater. Lesion size *per se* does not restrict suitability of ER for duodenal adenomas, with multiple series reporting technical success and acceptable safety profile for even very large lesions [29–32].

Work-up of ampullary adenomas prior to endoscopic resection

Additional work-up of ampullary adenomas is required before ER given their relationship to the common bile duct (CBD) and pancreatic duct (PD). We prefer the term endoscopic papillectomy