Overview of colon polyps

AUTHOR: Finlay A Macrae, MD
SECTION EDITOR: J Thomas Lamont, MD
DEPUTY EDITOR: Shilpa Grover, MD, MPH, AGAF

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INTRODUCTION

A polyp of the colon refers to a protuberance into the lumen above the surrounding colonic mucosa. Colon polyps are usually asymptomatic but may ulcerate and bleed, cause tenesmus if in the rectum, and, when very large, produce intestinal obstruction. Colonic polyps may be neoplastic (eg, adenomas) or non-neoplastic (eg, inflammatory polyps).

This topic will review the clinical features and management of colonic polyps and polypoid lesions of the colon. Colorectal cancer screening and surveillance after colorectal cancer resection are discussed separately. (See "Screening for colorectal cancer: Strategies in patients at average risk" and "Screening for colorectal cancer in patients with a family history of colorectal cancer or advanced polyp" and "Post-treatment surveillance after colorectal cancer treatment" and "Clinical manifestations and diagnosis of familial adenomatous polyposis" and "*MUTYH*-associated polyposis" and "Peutz-Jeghers syndrome: Clinical manifestations, diagnosis, and management".)

INFLAMMATORY POLYPS

Inflammatory polyps are non-neoplastic intraluminal projections of mucosa consisting of stromal and epithelial components and inflammatory cells. Inflammatory polyps include inflammatory pseudopolyps and prolapse type inflammatory polyps.

Inflammatory pseudopolyps — Inflammatory pseudopolyps are irregularly shaped islands of residual intact colonic mucosa that are the result of the mucosal ulceration and regeneration that occurs in response to localized or diffuse inflammation (eg, ulcerative colitis or Crohn disease).

- **Endoscopic features and histology** – Inflammatory polyps may be pedunculated or sessile and are usually smaller than 2 cm. Inflammatory pseudopolyps in patients with inflammatory bowel disease are typically multiple, often filiform, and scattered throughout the involved areas of the colon. They may also be more isolated and semipedunculated in areas of recent inflammation, and have mucus adherent to their apices (picture 1). Inflammatory pseudopolyps are composed of a mixture of inflamed lamina propria and distorted colonic epithelium; surface erosions may or may not be present.

- **Risk of malignancy** – Inflammatory pseudopolyps do not undergo neoplastic transformation. However, they may be associated with surrounding dysplasia in patients with inflammatory bowel disease. Careful attention to the region being biopsied is particularly important when pseudopolyps are present in clusters. (See "Surveillance and management of dysplasia in patients with inflammatory bowel disease".)

- **Management** – Inflammatory pseudopolyps do not require excision unless they cause symptoms (eg, bleeding, obstruction). Treatment is directed at the underlying cause of inflammation. (See "Overview of the medical management of mild (low risk) Crohn disease in adults" and "Clinical manifestations, diagnosis, and prognosis of Crohn disease in adults" and "Medical management of low-risk adult patients with mild to moderate ulcerative colitis" and "Management of the hospitalized adult patient with severe ulcerative colitis".)

Prolapse type inflammatory polyp — Prolapse type inflammatory polyps result from traction, distortion, and twisting of mucosa caused by peristalsis-induced trauma. This results in localized ischemia and lamina propria fibrosis. The classic histologic features of prolapse-induced inflammatory polyps include varying degrees of fibromuscular hyperplasia of the lamina propria, extension of the muscularis mucosae into the lamina propria, crypt elongation, hyperplasia, architectural distortion, and serration. Polyps may be associated with inflammation, ulceration, and reactive epithelial change. Based on the anatomic location of the injury and the underlying cause, these polyps are termed inflammatory cloacogenic polyps of the anal transitional zone, colitis cystica profunda, diverticular disease-associated polyps, or inflammatory cap polyps. Asymptomatic patients do not require treatment unless symptoms develop. In symptomatic patients, we perform endoscopic polypectomy. Surgical repair may be needed in patients with an associated rectal prolapse. The clinical features, diagnosis, and

management of inflammatory cap polyposis are discussed in detail separately. (See "Cap polyposis" and "Overview of rectal procidentia (rectal prolapse)", section on 'Indications for surgical management'.)

HAMARTOMATOUS POLYPS

Hamartomatous polyps are made up of tissue elements that are normally found at that site but are growing in a disorganized mass.

Juvenile polyps — Juvenile polyps are hamartomatous lesions that consist of a lamina propria and dilated cystic glands rather than increased numbers of epithelial cells (picture 2). Juvenile polyps can be diagnosed at any age, although they are relatively more common in childhood. Sporadic juvenile polyps of the colon occur in up to 2 percent of children under the age of 10 years, are usually solitary, and are not associated with an increased colorectal cancer risk [1]. Isolated juvenile polyps are most common in the rectosigmoid colon but can occur in the proximal colon. Juvenile polyps resulting in lower gastrointestinal bleeding or prolapse through the rectum require polypectomy. Asymptomatic patients do not require treatment.

Juvenile polyposis syndrome (JPS) is an autosomal dominant condition characterized by multiple hamartomatous polyps throughout the gastrointestinal tract. Individuals with JPS are at increased risk for colorectal and gastric cancer. The clinical features, diagnosis, and management of JPS is discussed in detail separately.

Peutz-Jeghers polyps — The Peutz-Jeghers polyp is a hamartomatous lesion of glandular epithelium supported by smooth muscle cells that is contiguous with the muscularis mucosa (picture 3). These polyps demonstrate a distinctive, arborizing pattern of smooth muscle derived from the underlying muscularis mucosa. The Peutz-Jeghers polyp is usually, but not always, associated with the Peutz-Jeghers syndrome (PJS), due to *STK11* mutations. Peutz-Jeghers polyps should be resected.

The polyps are usually benign, but may grow progressively and produce symptoms or undergo malignant transformation. Patients with PJS are at increased risk of both gastrointestinal (gastric, small bowel, colon, pancreas) and nongastrointestinal cancers including breast cancer. The clinical manifestations, diagnosis, and management of PJS are discussed separately. (See "Peutz-Jeghers syndrome: Clinical manifestations, diagnosis, and management".)

Cronkhite-Canada syndrome — Cronkhite-Canada syndrome is a rare, nonfamilial disorder associated with alopecia, cutaneous hyperpigmentation, gastrointestinal polyposis, onychodystrophy (picture 4), diarrhea, weight loss, and abdominal pain [2-4]. The polyps are

hamartomas and do not appear neoplastic pathologically. Characteristic features include myxoid expansion of the lamina propria and increased eosinophils in the polyps [1,5]. Cronkhite-Canada syndrome may be immune mediated since it may respond to immunosuppressive therapy and, in some patients, immunostaining of the polyps for IgG4 is positive [6]. Five-year mortality rates as high as 55 percent have been reported with most deaths due to gastrointestinal bleeding, sepsis, and congestive heart failure [7]. Treatment has included nutritional support, glucocorticoids, azathioprine, acid suppression, and antibiotics, but no specific treatment has proven to be consistently effective [6,8,9].

Phosphatase and tensin homolog (PTEN) hamartoma tumor syndrome — The PTEN hamartoma tumor syndrome, which is primarily comprised of the Cowden and Bannayan-Riley-Ruvalcaba syndromes, has also been associated with hamartomatous and other histologic types of polyps. These are discussed in detail separately. (See "PTEN hamartoma tumor syndromes, including Cowden syndrome", section on 'Cowden syndrome' and "PTEN hamartoma tumor syndromes, including Cowden syndrome", section on 'Bannayan-Riley-Ruvalcaba syndrome'.)

SESSILE SERRATED LESIONS

Sessile serrated lesions are a heterogenous group of polyps with variable malignant potential. They include hyperplastic polyps, traditional serrated adenomas, and sessile serrated polyps (SSPs; with and without cytologic dysplasia) [10]. The classification of serrated lesions is evolving and there is significant variability in the histologic classification of these lesions among pathologists [11-13].

Hyperplastic polyps — Hyperplastic polyps are the most common non-neoplastic polyps in the colon.

- **Endoscopic features and histology** – Hyperplastic polyps have an endoscopic appearance of small nodules or polypoid lesions. They are typically located in the rectosigmoid and are less than 5 mm in size [14,15].

  Hyperplastic polyps are serrated polyps with normal architecture and proliferative characteristics. They are composed of normal cellular components, do not exhibit dysplasia, and have a characteristic serrated ("saw tooth") pattern (picture 5). Proliferation is mainly in the basal portion of the crypt of hyperplastic polyps [16]. Three histologic subtypes of hyperplastic polyps have been described (microvesicular, goblet cell, and mucin depleted), but the clinical significance of this distinction is not clear.

- **Risk of cancer** – Small rectosigmoid hyperplastic polyps do not appear to increase the risk of colorectal cancer [17-22]. It is also unclear if small distal hyperplastic polyps are associated with an increased risk of proximal neoplasia and if present, the magnitude of risk appears to be small. A systematic review that included 18 studies estimated that 21 to 25 percent of patients found to have a distal hyperplastic polyp had a proximal neoplasm (including 4 to 5 percent with an advanced neoplasm) [21]. In the four studies in which a colonoscopy was performed irrespective of distal findings, the relative risk of any proximal neoplasia (advanced or not) was 1.3 (95% CI 0.9-1.8). (See "Tests for screening for colorectal cancer", section on 'Evidence of effectiveness'.)

  Although some studies have suggested that serrated lesions that are larger or proximal may be associated with an increased risk of synchronous advanced neoplasia, these studies included hyperplastic polyps, SSPs and sessile serrated adenomas (SSA).

- **Management** – Small hyperplastic polyps are typically biopsied or removed in the process of endoscopy with biopsy forceps because they can be difficult to distinguish from adenomatous polyps (picture 6). Although small left-sided hyperplastic polyps are not a significant marker of colon cancer risk, the biologic characteristics and natural history of large hyperplastic polyps, particularly the microvesicular type, are not well understood. Large lesions that contain some histologic features of a SSL, particularly when located in the right colon, should be resected in entirety. (See "Endoscopic removal of large colon polyps", section on 'Polyp removal techniques'.)

- **Surveillance**

  - In patients with <20 hyperplastic polyps that are <10 mm, surveillance colonoscopy is recommended in 10 years in countries including the United States where screening for colorectal cancer is advocated (table 1) [23]. In other countries, no further colonoscopies are recommended due to the lack of metachronous neoplasia risk. (See 'Sessile serrated polyps and traditional serrated adenomas' below.)

  - In patients with hyperplastic polyps ≥10 mm, a repeat colonoscopy is suggested in three to five years [23]. If there is concern about consistency in the distinction between SSP and HP locally, adequacy of the bowel preparation, or complete excision, we suggest a three-year follow-up interval. Otherwise a five-year interval is favored.

Sessile serrated polyps and traditional serrated adenomas

- **Endoscopic features and histology** – SSPs (synonymous with sessile serrated adenoma [SSA]) are more prevalent in the proximal colon. These polyps have a smooth surface

sometimes with a "cloud-like appearance," are often flat or sessile, and may be covered with mucus. Histologically, SSPs contain significant architectural, proliferative, and maturation abnormalities and may acquire morphologic evidence of dysplasia [16].

Traditional serrated adenomas (TSAs) are more prevalent in the rectosigmoid colon and may be pedunculated or sessile. TSA have diffuse but often mild cytologic dysplasia.

- **Risk of cancer** – SSP frequently exhibit dysplasia. In one study of 110 SSA/Ps, areas of "significant" dysplasia and foci of intramucosal carcinoma (high-grade dysplasia) were found in 37 and 11 percent, respectively [24]. SSPs, particularly those with foci of dysplasia, are considered the likely precursor lesions to sporadic microsatellite instability-high (MSI-H) colon cancer through a molecular pathway characterized by a high frequency of methylation of some CpG islands (CpG island hypermethylation phenotype [CIMP]-positive) (table 2) [25]. The defect may result in hypermethylation of the promoter region of the MMR MLH1 and silencing of gene expression [16,26,27]. Activation of the *BRAF* oncogene (*BRAF* V600E mutation) is also a feature of SSA/Ps, as well as many hyperplastic polyps [28,29]. (See "Molecular genetics of colorectal cancer", section on 'Hypermethylation phenotype (CIMP+) pathway'.)

  Risk factors for a synchronous advanced adenoma in patients with SSPs include SSP/A size ≥10 mm, location in the proximal colon, and the presence of dysplasia [16,26-29].

- **Management** – SSPs are managed clinically like adenomatous polyps and complete excision is recommended. However, due to their sessile nature and indistinct borders, special care is needed to ensure their complete removal endoscopically. There is molecular and clinical evidence that these lesions, either through being missed, incompletely removed, or through a more rapid progression from adenoma to cancer, disproportionally contribute to interval CRCs [30]. (See "Endoscopic removal of large colon polyps".)

- **Surveillance** – The surveillance interval is based on polyp size and histology (table 1).

  - In individuals with one to two SSPs <10 mm in size with no dysplasia, we perform surveillance colonoscopy in 5 to 10 years. (See 'Patients without advanced adenomas' below.)

  - In individuals with three to four SSPs <10 mm, surveillance colonoscopy is repeated in three to five years. In individuals with 5 to 10 SSPs <10 mm, surveillance colonoscopy is performed in three years.

- Individuals with SSP ≥10 mm, a SSP with dysplasia, or TSA are managed as advanced adenomas with a first surveillance colonoscopy in three years. (See 'Patients with advanced adenomas (≥10 mm, villous histology, or high grade dysplasia)' below.)

Our recommendations are consistent with United States Multi-Society Task Force guidelines (table 1) [23]. Other expert consensus recommendations have suggested earlier colonoscopic follow-up (one- to three-year interval) in individuals with two or more SSP larger than 10 mm and in those with any SSP with cytologic dysplasia [10]. However, prospective data to support the surveillance intervals are lacking and these recommendations are based on expert opinion. (See 'Patients with advanced adenomas (≥10 mm, villous histology, or high grade dysplasia)' below and 'Patients without advanced adenomas' below.)

Serrated polyposis syndrome — Serrated polyposis syndrome (SPS) or hyperplastic polyposis syndrome (HPS) is rare condition characterized by multiple, large, and/or proximal serrated polyps [31,32].

- **Endoscopic features and pathology** – Polyps in SPS may be large and flat and are often found along the crests of haustral folds. Mucus and residue frequently adhere to the polyps, a characteristic that may make it difficult to discriminate the polyp from the surrounding normal colonic mucosa.

- **Genetics** – While some patients with SPS carry a mutation in rare polyposis-associated genes including *SMAD4*, *BMPR1A, PTEN, GREM1, RNF43,* and *MUTYH,* these genes are not altered in the majority of individuals with SPS [31,33-35]. *RNF43* where mutated has been most closely related to SPS, but is still very uncommon. However, it is unlikely to be a predisposing mutation in the majority of patients [36]. (See "*MUTYH*-associated polyposis" and "Molecular genetics of colorectal cancer".)

- **Malignant potential** – Patients with SPS have an increased risk of colorectal cancer [37,38]. In a meta-analysis of 36 studies that included 2788 patients with SPS, the overall risk of CRC was 20 percent (95% CI, 15-25 percent) [39]. CRC risk at the time of diagnosis and during surveillance were 15 percent and 3 percent, respectively. SPS patients also had a high incidence of history of CRC prior to SPS diagnosis (7.0 percent; 95% CI, 4.6-11.7 percent).

  This CRC risk is attributable to the presence of serrated polyps with dysplasia, advanced adenomas, and the presence of large or multiple proximal colonic polyps. It is unclear if patients with SPS are also at increased risk for pancreatic cancer [40].

- **Diagnosis** – A clinical diagnosis of SPS requires the presence of one of the following World Health Organization criteria [41]:

  - >20 serrated lesions/polyps of any size, distributed throughout the colon with ≥5 being proximal to the rectum.

  - At least five serrated lesions/polyps proximal to the rectum, all being ≥5 mm, with at least two ≥10 mm in size.

  Genetic testing is not routinely recommended as the genetic basis of SPS is largely unknown. (See "*MUTYH*-associated polyposis", section on 'Colonic manifestations' and "*MUTYH*-associated polyposis", section on 'Diagnosis'.)

- **Management** – Management strategies for patients with SPS and their families have not been well defined. Polyps ≥1 cm should be resected completely [37]. Subsequent colonoscopy intervals are based on the number and size of polyps, as well as the number of concurrent adenomas, but generally should be performed every one to three years [10,42,43].

  Potential indications for colectomy include:

  - Documented or suspected CRC.

  - Severe symptoms related to colonic neoplasia (eg, severe gastrointestinal bleeding).

  - Polyps with high-grade dysplasia or multiple adenomas larger than 6 mm.

  - Marked increases in polyp number on consecutive examinations.

  - Inability to adequately survey the colon because of multiple diminutive polyps.

  - Patient preference to avoid colonoscopic surveillance. The decision to undergo colectomy in such cases requires an informed discussion of the relative risks versus benefits of regular surveillance and polypectomies versus surgical risk of colectomy. Patients with SPS may be at increased risk of missed lesions, incomplete resection, and polypectomy complications given that the polyps are flat and have indistinct color and margins.

  In the absence of evidence to guide colorectal screening recommendations for family members, we begin screening first-degree relatives of individuals with SPS around age 40 (or 10 years earlier than the earliest age at presentation of the SPS in the family) [10,31]. We continue surveillance every five years if no polyps are detected.

ADENOMATOUS POLYPS

Adenomas are the most prevalent neoplastic polyps in the colon.

Epidemiology and risk factors — Approximately two-thirds of all colonic polyps are adenomas. Thirty to fifty percent of colons with one adenoma will contain at least one other synchronous adenoma [44].

Increasing age is a risk factor for the development of colonic adenomas and is associated with the development of high-grade dysplasia within an adenoma, independent of size and histology [19,45]. In colorectal cancer screening studies, the prevalence of adenomas is approximately 25 to 30 percent at age 50 years [45-49]. Autopsy studies have found rates as high as 50 percent by age 70 years (figure 1) but only 1 to 4 percent in those in their twenties or thirties [49,50]. Advancing age is also a risk factor for right-sided polyps [51].

An increased body mass index (BMI) is associated with an increased risk of colorectal adenomas. In a meta-analysis of 36 studies, the risk for colorectal adenomas increased by 19 percent for every 5-unit increase in BMI (summary relative risk 1.19; 95% CI 1.13-1.26) [52]. Abdominal obesity, measured by increasing abdominal visceral adipose tissue volume, may be a better predictor than BMI or waist circumference in both sexes [53]. Lack of physical activity is also a risk factor [54].

Adenomatous polyps are more common in men, and large adenomas may be more common in African-Americans as compared with other ethnic groups [48,55,56]. In addition, African-Americans may have a higher risk of right-sided colonic adenomas and present with colorectal at a younger age (<50 years of age) [57,58]. (See "Colorectal cancer: Epidemiology, risk factors, and protective factors" and "Screening for colorectal cancer: Strategies in patients at average risk".)

Clinical features

Clinical presentation and natural history — Adenomas are generally asymptomatic and are most often detected by colon cancer screening tests. Small adenomas do not typically bleed. The growth rates of adenomatous polyps are variable and do not follow a consistent linear trend. The majority of small polyps exhibit minimal growth (averaging 0.5 mm/year). However, complete regression is uncommon [59]. Only a small minority of adenomas progress to cancer (5 percent or less) over 7 to 10 years. The risk of progression is higher for advanced adenomas (adenoma with high-grade dysplasia, >10 mm in size, or a villous component) [45].

Approximately 5 to 7 percent of patients with adenomas have high-grade dysplasia, and 3 to 5 percent have invasive carcinoma at the time of diagnosis. The proportion of adenomas showing advanced histologic features increases with polyp size from approximately 1 to 2 percent in small adenomas (<5 mm) to 7 to 12 percent for medium-sized adenomas (5 to 10 mm) and 20 to 30 percent for large adenomas (>1 cm) [60-62].

Endoscopic features and classification — The majority of adenomas (60 to 75 percent) are less than 1 cm at endoscopy [19,63]. Based on their gross appearance, adenomas may be pedunculated, sessile, flat, depressed, or excavated.

- **Sessile** – In a sessile polypoid lesion, the base and the top of the lesion have the same diameter. Large, laterally-spreading adenomas may have a granular appearance, which is characteristic of being benign. If the mucosa is smooth and nongranular, with distortion of the microvasculature as seen in high-definition endoscopy, cancer should be suspected.

- **Pedunculated** – In pedunculated polyp the base is narrow. A mucosal stalk is interposed between the polyp and the wall (picture 6).

- **Flat** – Flat lesions are defined as those with a height less than one-half the diameter of the lesion. Up to 27 to 36 percent of adenomas are relatively flat [64-67]. Flat lesions can be difficult to detect on colonoscopy. Some studies have found that flat adenomas tend to be relatively advanced histologically for their size compared with polypoid lesions [65,66,68]. However, their natural history is poorly understood [67].

- **Depressed** – In depressed lesions the entire thickness of the mucosa in the lesion is often less than that of the adjacent mucosa. Depressed lesions appear to be particularly likely to harbor high-grade dysplasia or be malignant, even if small [68-73]. Up to 1 percent of adenomas are depressed [64-67]. Although flat and depressed adenomas were initially thought to be largely in Asian populations, they have now been shown to occur more frequently than previously recognized in western populations [68,74-77].

The Paris classifications of superficial neoplastic lesions of the gastrointestinal tract can be used to classify adenomas into polypoid and nonpolypoid lesions (figure 2 and table 3) [78].

Endoscopic features suggestive of invasive cancer include friability, induration, and ulceration. On probing, a firm consistency or adherence of the polyp to the underlying tissue is also suggestive of malignancy. Features suggestive of deep submucosa invasion include Narrow Band Imaging International Colorectal Endoscopic (NICE) classification type 3 and the Kudo Classification of type V or VI (figure 3) [79]. Features suggestive of superficial submucosal invasion in a polyp include a nongranular laterally spreading tumor (NG-LST) morphology with

sessile shape or depression and granular-LST (G-LST) morphology with a dominant nodule. (See "Endoscopic removal of large colon polyps", section on 'Definitions' and "Endoscopic removal of large colon polyps", section on 'Features suggesting invasive cancer'.)

Histologic features — Adenomas are characterized as tubular, villous, or a mixture of the two [19]. An advanced adenoma is defined as adenomas ≥10 mm in size or with villous components or high-grade dysplasia. While modifications to the presently used definition for advanced adenomas have been proposed (adenoma ≥20 mm or high-grade dysplasia), further validation is required [80].

- **Tubular** – Tubular adenomas account for more than 80 percent of colonic adenomas. They are characterized by a network of branching adenomatous epithelium (picture 7A-B). To be classified as tubular, the adenoma should have a tubular component of at least 75 percent.

- **Villous** – Adenomas with >75 percent villous features are termed villous adenomas. Villous adenomas account for 5 to 15 percent of adenomas (image 1 and picture 8). They are characterized by glands that are long and extend straight down from the surface to the center of the polyp (picture 9). To be classified as villous, the adenoma should have a villous component of at least 75 percent.

- **Tubulovillous** – Adenomas with 25 to 75 percent villous features are considered to be tubulovillous. Tubulovillous adenomas account for 5 to 15 percent of colonic adenomas.

Some degree of dysplasia exists in all adenomas. Based on the degree of dysplasia, polyps are classified as having:

- Low-grade dysplasia
- High-grade dysplasia

High-grade dysplasia (synonymous with intraepithelial carcinoma) represents an intermediate step in the progression from a low-grade dysplasia to cancer. The term is applied to lesions that are confined to the epithelial layer of crypts and lack invasion through the basement membrane into the lamina propria. As there are no lymphatic vessels in the lamina propria, lesions with high grade dysplasia are not associated with metastasis [81,82].

In carcinoma in situ (Tis), or intramucosal adenocarcinoma, cancer cells invade into the lamina propria and may involve but do not penetrate the muscularis mucosa [19,83]. Many pathologists will not differentiate these features from high-grade dysplasia, especially as there

is no need to either from a biologic and management perspective. Invasive adenocarcinoma extends through the muscularis mucosa into the submucosa and beyond.

Management

Polypectomy — Adenomas should be resected completely. Small adenomas (≤2 mm) may be completely removed using biopsy forceps, while larger adenomas require snare resection, with or without electrocautery or advanced endoscopic resection techniques (eg, endoscopic mucosal resection or endoscopic submucosal dissection) [84]. Cold snaring has the advantage of avoiding full thickness injury to the bowel wall that can accompany diathermy, and minimizes delayed postpolypectomy bleeding. Large sessile adenomas often require piecemeal resection. In cases where endoscopic resection is not possible, surgical resection is required. (See "Endoscopic removal of large colon polyps".)

Pedunculated polyps with features of deep submucosal invasion should undergo endoscopic polypectomy with en bloc resection [79].

Nonpedunculated lesions with features of deep submucosal invasion should be biopsied in the area of surface feature disruption, tattooed if the polyps are not at or near the cecum, and patients should be referred for surgical management [79]. Nonpedunculated polyps with features of superficial submucosal invasion (eg, NG-LST morphology) should ideally be resected en bloc instead of piecemeal. In G-LST with a dominant nodule, the dominant nodule should be resected en bloc. (See 'Endoscopic features and classification' above and "Endoscopic removal of large colon polyps", section on 'Features suggesting invasive cancer'.)

Special populations

Incomplete or piecemeal resection — In patients who have undergone piecemeal resection of a large polyp (≥20 mm) or in whom the completeness of resection is uncertain (regardless of polyp size), we perform a repeat colonoscopy to evaluate the site of excision within six months [42,85,86]. Incomplete polyp resection is thought to be a substantial contributor to interval colorectal cancer [87,88]. The rates of incomplete resection appear to be higher for sessile lesions and increases with polyp size. In one study, the rate of incomplete resection was significantly higher for polyps 10 to 20 mm as compared with polyps 5 to 9 mm (17 versus 7 percent) and for sessile serrated polyps (SSLs) as compared with conventional adenomas (31 versus 7 percent) [89]. (See "Endoscopic removal of large colon polyps".)

High-grade dysplasia or cancer — Polypectomy alone is adequate for polyps with high-grade dysplasia and Tis if the resection margins are free of neoplastic tissue [90]. Subsequent surveillance should be performed at the same interval as for patients with high-risk adenomas.

(See 'Patients with advanced adenomas (≥10 mm, villous histology, or high grade dysplasia)' below.)

The management of a polyp containing invasive carcinoma must be individualized. In early-stage (T1) colon cancers, polypectomy alone is usually adequate, particularly in older adults, if the following risk factors for residual cancer and/or nodal metastases are absent [79,91]:

- Poorly-differentiated histology
- Lymphovascular invasion (LVI) or perineural invasion (PNI)
- Tumor budding (foci of isolated cancer cells or a cluster of five or fewer cancer cells at the invasive margin of the polyp) [79]
- A positive margin defined as cancer present at the resection margin [91]
- Submucosal invasion depth >1 mm [79]

After complete resection of a polyp with cancer, we perform follow-up colonoscopy in three months to check for residual abnormal tissue at the polypectomy site if the polyp was sessile [92]. After one negative follow-up examination, care can revert to standard surveillance as performed for patients with advanced adenomas. (See 'Patients with advanced adenomas (≥10 mm, villous histology, or high grade dysplasia)' below.)

The presence of any one of the risk factors for residual cancer and/or nodal metastases should prompt consideration of surgery. The management of invasive carcinoma in a colorectal polyp and surveillance is discussed in detail separately. (See "Overview of the management of primary colon cancer" and "Surgical resection of primary colon cancer", section on 'Malignant polyp'.)

Surveillance

Risk assessment for subsequent colorectal cancer — Assessment of the patient's risks of developing advanced neoplasia is important in guiding colorectal cancer surveillance.

Polyp histology, number, and size are risk factors for metachronous adenomas and colorectal cancer (eg, diagnosed six months after removal of an index cancer or polyp) [93,94]. This was illustrated in a prospective cohort study that included 122,899 participants who underwent flexible sigmoidoscopy or colonoscopy. After a median follow-up of 10 years, there were 491 incident cases of colorectal cancer [95]. Of these, 51 occurred in 6161 individuals with conventional adenomas, 24 in 5918 individuals with serrated polyps, and 427 in 112,107 individuals with no polyp at baseline evaluation. Individuals with advanced adenomas (≥10 mm, high-grade dysplasia, or tubulovillous or villous histology) and large (≥10 mm) serrated polyps

had a significantly higher risk of colorectal cancer as compared with those without adenomas (RR 4.1 and 3.3, respectively). There was no significant difference in the risk of colorectal cancer between individuals with nonadvanced adenomas or small serrated polyps and those without adenomas at baseline.

A prospective cohort study included 15,935 participants of a randomized trial of flexible sigmoidoscopy who underwent colonoscopy for abnormal flexible sigmoidoscopy findings [94]. On colonoscopy, 2882 individuals had an advanced adenoma and 5068 had a nonadvanced adenoma. No adenoma was detected in the remaining 7985 individuals. At a median follow-up of approximately 13 years, incidence rates of CRC in individuals with an advanced adenoma, nonadvanced adenoma, and no adenoma at baseline colonoscopy were 20, 9.1, and 7.5 per 10,000 person-years, respectively. Individuals with advanced adenomas had a significantly higher risk of CRC and CRC death as compared with those without adenomas (RR 2.7 and 2.6, respectively). Although there was no significant difference in the risk of CRC between individuals with nonadvanced adenomas and those without adenomas at baseline, the study may have been underpowered to detect a difference.

- **Histology** – Adenomatous polyps with >25 percent villous histology, high-grade dysplasia or invasive cancer (picture 9) are risk factors for developing metachronous colorectal cancer [60,96]. In some studies, villosity also predicts metachronous advanced adenomas, although villosity is often closely linked with size and is not independently predictive in all studies.

- **Number of polyps** – Numbers of adenomas at colonoscopy and cumulatively over a lifetime is the most consistent risk factor for metachronous colorectal cancer. Studies suggest that patients with a limited number (one or two) of small, tubular, adenomatous polyps removed do not have an increased risk of advanced neoplasia [97]. By contrast, the presence of one or more advanced adenomas predicts a higher rate of both any and advanced metachronous adenomas [96]. In a pooled analysis of eight prospective studies, the absolute risks of metachronous advanced adenomas, colorectal cancer, and their combination (advanced colorectal neoplasia) within three to five years was higher (24 percent) in patients with >4 adenomas at baseline [98]. The risk for metachronous advanced adenomas increased with the number of adenomas at baseline and was 9, 13, 15, 20, and 24 percent for 1, 2, 3, 4, or ≥5 adenomas at baseline, respectively. The risk of metachronous colon cancer also increases with the number of advanced adenomas [60,96].

- **Polyp size** – Patients with one or more adenomas ≥10 mm have an increased risk of advanced neoplasia during surveillance as compared with those with no neoplasia or small

adenomas [60,97,99-101]. The risk of advanced neoplasia increases with adenoma size. As compared with patients with adenomas <5 mm, those with baseline adenoma(s) 10 to 19 mm and ≥20 mm have a significantly higher risk of advanced neoplasia (8, 16, and 19 percent, respectively) [98]. In another study that included 1287 individuals, those with large adenomas (>1 cm) and proximally located adenomas at baseline colonoscopy were significantly more likely to have recurrent high-risk adenomas during a mean follow-up period of 37 months (odds ration [OR] 2.69; 95% CI, 1.3-5.4 and OR 1.7; 95% CI, 1.0-2.7, respectively) [101].

Patients with advanced adenomas (≥10 mm, villous histology, or high grade dysplasia) — An advanced adenomas is defined as any one of the following:

- Tubular adenoma ≥10 mm or
- Adenoma with villous histology, or high-grade dysplasia

Patients with an advanced adenoma at any examination should have short follow-up intervals for surveillance [23,85].

- **First surveillance** – Individuals with an advanced adenoma should undergo a first surveillance colonoscopy in three years (table 4) [23]. For adenomas ≥20 mm that have been resected piecemeal, repeat colonoscopy should be performed at six months.

- **Subsequent surveillance** – The timing of the subsequent surveillance colonoscopy is based on the findings of the first surveillance colonoscopy (table 5) [23].

  - If no adenomas are found on the first surveillance colonoscopy, the next surveillance colonoscopy should be performed in five years. Patients with an advanced adenoma at any examination appear to remain at high risk for CRC and should have short follow-up interval for all subsequent surveillance colonoscopies [85].

  - If the first surveillance colonoscopy is normal (no adenomas, SSP, hyperplastic polyp ≥10 mm, or CRC) or if only one to two tubular adenomas are detected, the next surveillance colonoscopy should be performed at five years.

  - If three to four tubular adenomas <10 mm are detected, the next surveillance colonoscopy should be performed in three to five years.

  - If an advanced adenoma is detected or 5 to 10 adenomas <10 mm are detected, the next surveillance colonoscopy should be performed in three years.

Patients without advanced adenomas — If only one or two small (<10 mm) tubular adenomas are found on baseline colonoscopy, the first surveillance colonoscopy should be performed in 7 to 10 years (table 4) [23].

The timing of the subsequent surveillance colonoscopy is based on the findings of the first surveillance colonoscopy (table 5):

- If no adenomas are found on the first surveillance colonoscopy, the next surveillance colonoscopy should be performed in 10 years in the absence of other factors associated with increased risk for colorectal cancer (eg, colorectal cancer or a high-risk adenoma in a first-degree relative prior to age 60 years or in two first-degree relatives regardless of age). (See "Screening for colorectal cancer in patients with a family history of colorectal cancer or advanced polyp", section on 'Indications for enhanced screening'.)

- If one to two small adenomas are detected, the next surveillance colonoscopy should be performed in 7 to 10 years.

- If three to four small adenomas are detected, the next surveillance colonoscopy should be performed in three to five years.

- If >10 adenomas are detected on single examination, surveillance colonoscopy should be performed in one year.

- Patients with >10 adenomas on a single examination or >10 cumulative adenomas in their lifetime should also be evaluated for a hereditary colorectal cancer syndrome [23,43].

- If an advanced adenoma is detected or if an individual has 5 to 10 low-risk adenomas, the next surveillance colonoscopy should be performed in three years. Patients with an advanced adenoma at any examination appear to remain at high risk and should have short (eg, three to five years) follow-up interval for all subsequent surveillance colonoscopies [23,85].

Our recommendations are consistent with the United States Multi-Society Task Force guidelines [23]. The European Society of Gastrointestinal Endoscopy (ESGE), the European guidelines for quality assurance in colorectal cancer screening and diagnosis, and Australian guidelines recommend that patients with low-risk adenomas at baseline colonoscopy participate in existing national screening programs 10 years after the index colonoscopy [85,102,103]. If no screening program is available, the ESGE recommends colonoscopy 10 years after the index colonoscopy.

OTHER POLYPS AND POLYPOID LESIONS OF THE LARGE INTESTINE

A variety of submucosal lesions, including lymphoid aggregates, lipomas, leiomyomas, pneumatosis cystoid intestinalis, hemangiomas, fibromas, polypoid endocrine tumors, perineuriomas/fibroblastic polyps, Langerhans histiocytosis, rectal carcinoids, lymphomatous polyps, and metastatic lesions may impart a polypoid appearance to the overlying mucosa.

The most common of these, the lipoma, can be diagnosed endoscopically because of its yellow color and softness, assessed by probing the polyp with forceps and eliciting the "pillow sign" of indentation on gentle pressure. Perineuriomas are benign tumors comprised of perineurial cells and they occasionally present as colon polyps [104]. Fibroblastic polyps have identical clinical and histologic features and some have suggested, based upon immunohistochemical staining, that they represent the same entities [105,106]. These polyps are believed to be benign, and surveillance is not recommended.

Endoscopic ultrasound can be useful in defining the site of origin of a polypoid lesion and to biopsy a submucosal lesions if the diagnosis is in doubt. The use of endoscopic ultrasound to evaluate subepithelial lesions is discussed in detail separately. (See "Endoscopic ultrasound for the characterization of subepithelial lesions of the upper gastrointestinal tract".)

INDICATIONS FOR GENETIC EVALUATION

Approximately 5 to 10 percent of colorectal cancers are attributable to a hereditary cancer predisposition syndrome. A hereditary cancer predisposition syndrome should be considered in patients who present with early age at onset of polyps or cancer or unusual numbers or histologies of cancers or premalignant conditions. As an example, in patients with 10 or more cumulative colorectal adenomas or any number of adenomas in combination with duodenal/ampullary adenomas, desmoid tumors, papillary thyroid cancer, epidermal cysts, or osteomas, should raise the possibility of familial adenomatous polyposis (FAP) [23]. The clinical features, diagnosis, and management of other colorectal polyposis syndromes are discussed in detail separately. (See "Clinical manifestations and diagnosis of familial adenomatous polyposis" and "Familial adenomatous polyposis: Screening and management of patients and families" and "*MUTYH*-associated polyposis" and "Peutz-Jeghers syndrome: Clinical manifestations, diagnosis, and management" and "Juvenile polyposis syndrome" and "PTEN hamartoma tumor syndromes, including Cowden syndrome".)

SOCIETY GUIDELINE LINKS

Links to society and government-sponsored guidelines from selected countries and regions around the world are provided separately. (See "Society guideline links: Colon polyps".)

INFORMATION FOR PATIENTS

UpToDate offers two types of patient education materials, "The Basics" and "Beyond the Basics." The Basics patient education pieces are written in plain language, at the 5th to 6th grade reading level, and they answer the four or five key questions a patient might have about a given condition. These articles are best for patients who want a general overview and who prefer short, easy-to-read materials. Beyond the Basics patient education pieces are longer, more sophisticated, and more detailed. These articles are written at the 10th to 12th grade reading level and are best for patients who want in-depth information and are comfortable with some medical jargon.

Here are the patient education articles that are relevant to this topic. We encourage you to print or e-mail these topics to your patients. (You can also locate patient education articles on a variety of subjects by searching on "patient info" and the keyword(s) of interest.)

- Basics topics (see "Patient education: Colon polyps (The Basics)" and "Patient education: Colonoscopy (The Basics)" and "Patient education: Familial adenomatous polyposis (The Basics)")

- Beyond the Basics topics (see "Patient education: Colon polyps (Beyond the Basics)" and "Patient education: Colonoscopy (Beyond the Basics)" and "Patient education: Flexible sigmoidoscopy (Beyond the Basics)")

SUMMARY AND RECOMMENDATIONS

- **Types of polyps** – A polyp of the colon refers to a protuberance into the lumen above the surrounding colonic mucosa. Polyps are usually asymptomatic but may ulcerate and bleed, cause tenesmus if in the rectum, and, when very large, produce intestinal obstruction. Colonic polyps may be neoplastic (eg, adenomas) or non-neoplastic (eg, inflammatory polyps). (See 'Introduction' above.)

- **Inflammatory polyps** – Inflammatory polyps are non-neoplastic intraluminal projections of mucosa consisting of stromal and epithelial components and inflammatory cells.

Inflammatory polyps do not undergo neoplastic transformation and do not require excision unless they cause symptoms. (See 'Inflammatory polyps' above.)

- **Hamartomatous polyps** – Hamartomatous polyps are made up of tissue elements that are normally found at that site but are growing in a disorganized mass. Juvenile polyps and Peutz-Jeghers polyps are hamartomatous polyps. In contrast to patients with sporadic juvenile polyps, patients with juvenile polyposis coli and Peutz-Jeghers syndrome (PJS) are at increased risk for colorectal cancer. (See 'Hamartomatous polyps' above.)

- **Sessile serrated lesions** – Sessile serrated lesions are a heterogenous group of polyps with variable malignant potential. They include hyperplastic polyps, traditional serrated adenomas, and sessile serrated polyps (SSLs; synonymous with sessile serrated adenomas [SSA]). (See 'Sessile serrated lesions' above.)

- **Hyperplastic polyps** – Hyperplastic polyps are the most prevalent non-neoplastic polyps in the colon. Distal small hyperplastic polyps rarely, if ever, develop into colorectal cancers. SSLs, particularly those with foci of classic histologic dysplasia, are considered the likely precursor lesions to sporadic high microsatellite instability (MSI-H) colon cancer through a molecular pathway characterized by a high frequency of methylation of some CpG islands (CpG island hypermethylation phenotype-positive). (See 'Sessile serrated lesions' above.)

- **Adenomatous polyps** – Adenomatous polyps are neoplastic polyps. Clinically, adenomas are generally asymptomatic and are most often detected by colon cancer screening tests. Villous histology, increasing polyp size, and high-grade dysplasia are risk factors for focal cancer within an individual adenoma. (See 'Clinical presentation and natural history' above.)

- **Management** – If a polyp is detected, we recommend colonoscopy to establish the histology, remove the polyp, and search for synchronous lesions (**Grade 1A**). Recommendations for subsequent treatment and surveillance depend on the number and pathologic features of the polyp(s) (table 1 and table 4 and table 5).

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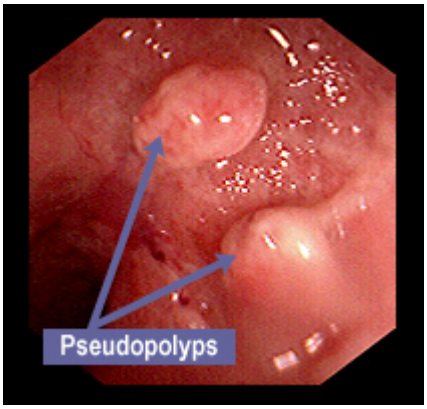
Topic 2594 Version 58.0

GRAPHICS

Colonic pseudopolyps in inflammatory bowel disease

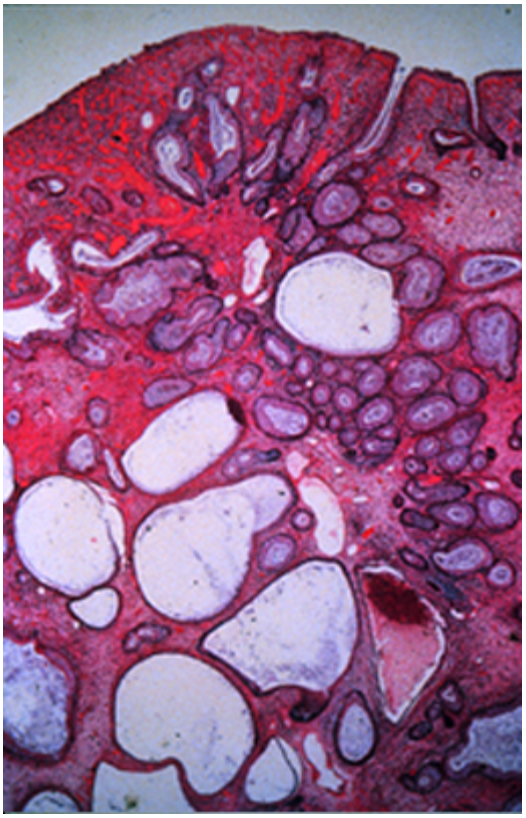


Endoscopy of pseudopolyps; these lesions are not specific to ulcerative colitis, although they are more common in this disorder than in Crohn disease.

Courtesy of James B McGee, MD.

Graphic 76729 Version 3.0

Juvenile colonic polyp

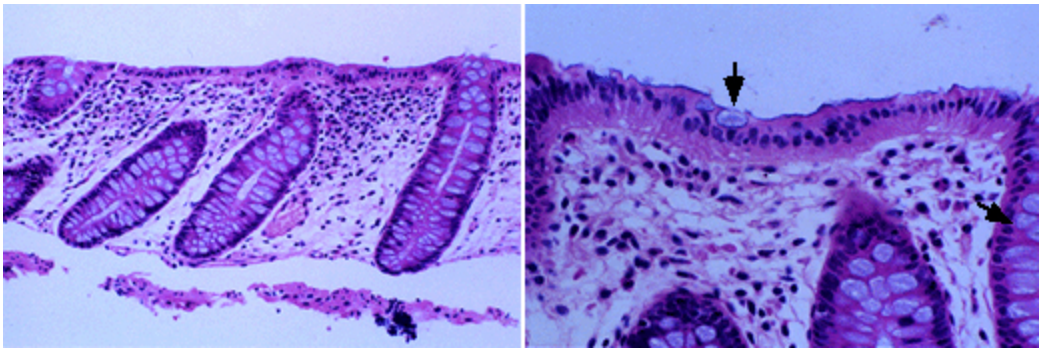


Low power view of a juvenile colonic polyp shows dilated cystic crypts and abundant, mildly inflamed lamina propria.

Courtesy of Robert Odze, MD.

Graphic 72376 Version 1.0

Normal colon

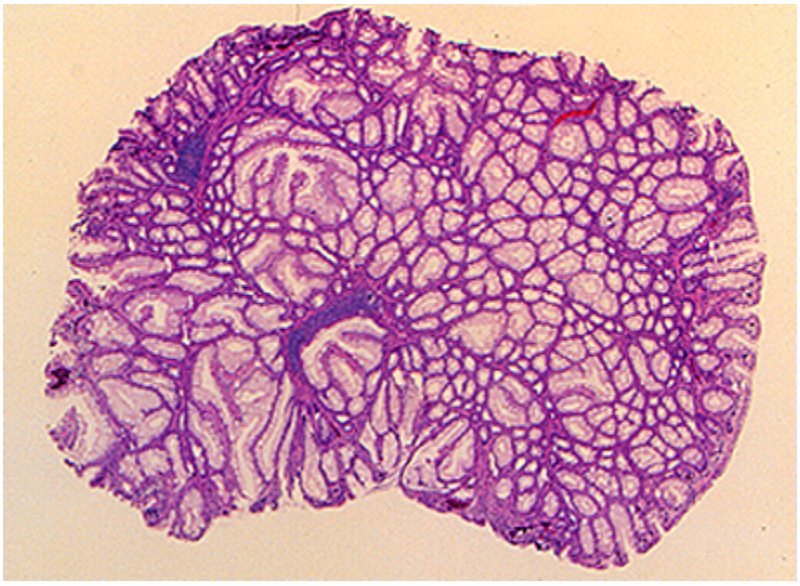


Low (left) and high (right) power views of a biopsy of a normal colon. Low power reveals straight crypts and mild lamina propria mononuclear cell infiltration. High power shows the surface enterocytes with interspersed goblet cells (arrows).

Courtesy of Robert Odze, MD

Graphic 81083 Version 6.0

Colonic Peutz-Jeghers polyp

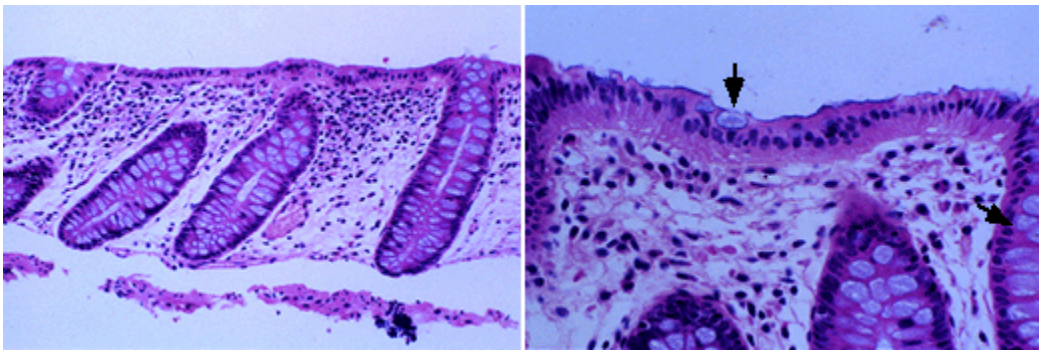


Low power view of a colonic Peutz-Jeghers polyp shows a tree-like proliferation of smooth muscle lined by normal colonic cell types.

Courtesy of Robert Odze, MD.

Graphic 69268 Version 1.0

Normal colon

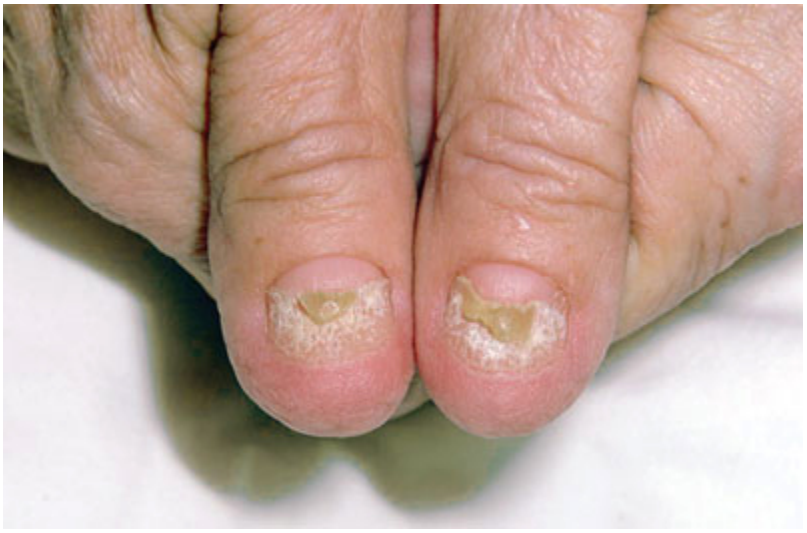


Low (left) and high (right) power views of a biopsy of a normal colon. Low power reveals straight crypts and mild lamina propria mononuclear cell infiltration. High power shows the surface enterocytes with interspersed goblet cells (arrows).

Courtesy of Robert Odze, MD

Graphic 81083 Version 6.0

Cronkhite-Canada syndrome

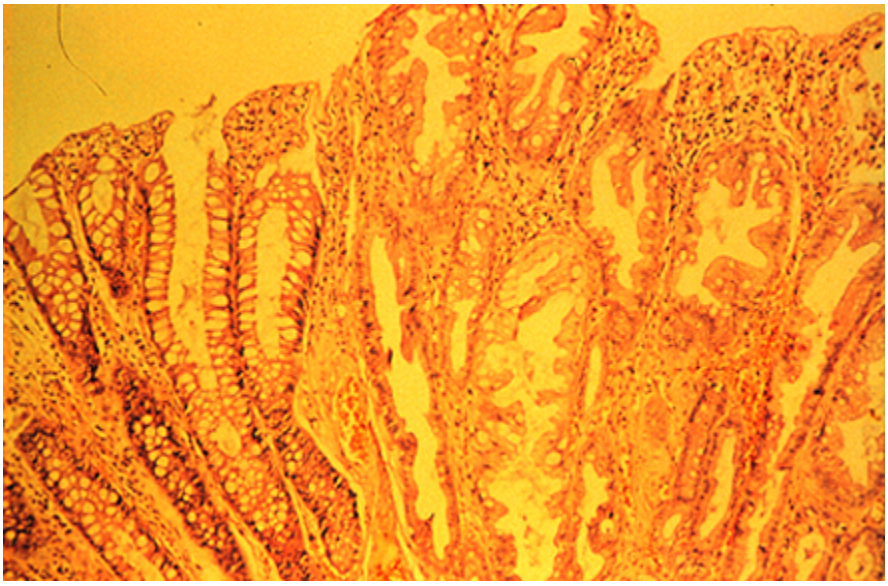


Onychodystrophy in the case patient.

Reprinted by permission from: Macmillan Publishers Ltd: Sweetser S, Alexander GL, Boardman LA. A case of Cronkhite-Canada syndrome presenting with adenomatous and inflammatory colon polyps. Nat Rev Gastroenterol Hepatol 2010; 7:460.

Graphic 76442 Version 2.0

Hyperplastic colonic polyp

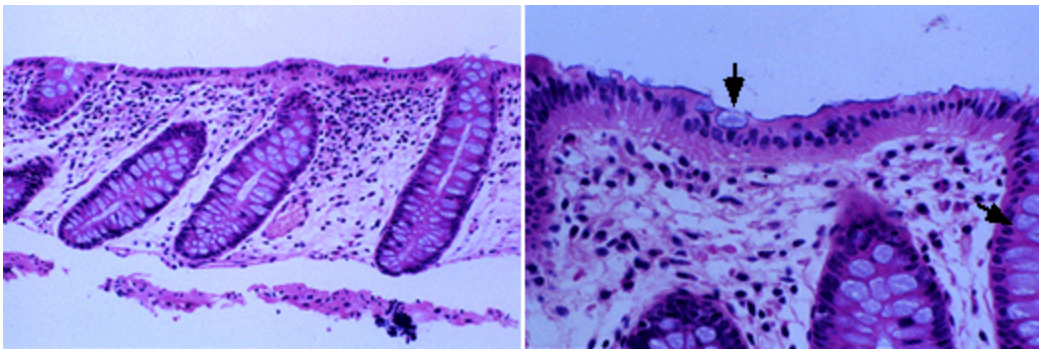


Medium power view of a hyperplastic colonic polyp shows a serrated surface contour and marked luminal infolding of the crypt epithelium.

Courtesy of Robert Odze, MD.

Graphic 59446 Version 1.0

Normal colon

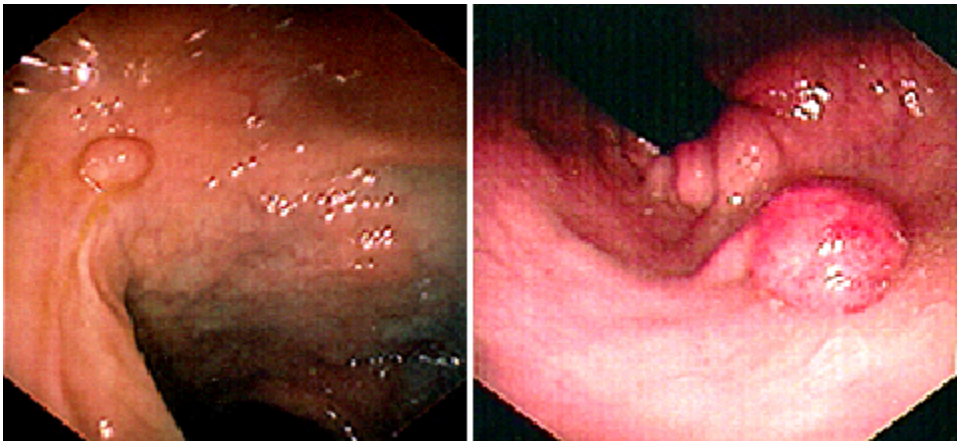


Low (left) and high (right) power views of a biopsy of a normal colon. Low power reveals straight crypts and mild lamina propria mononuclear cell infiltration. High power shows the surface enterocytes with interspersed goblet cells (arrows).

Courtesy of Robert Odze, MD

Graphic 81083 Version 6.0

Colonic polyps

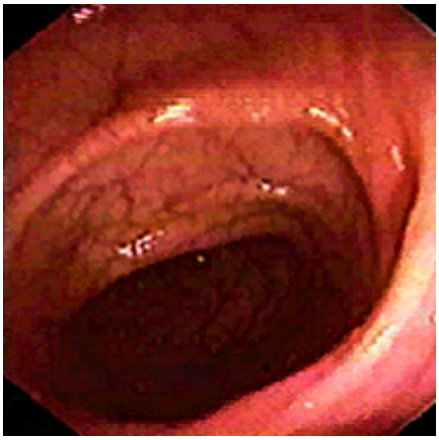


Over 95 percent of colonic polyps are hyperplastic or adenomatous. Although these two types have some distinctive features on gross appearance, they cannot be reliably distinguished endoscopically. Left panel: a typical small sessile hyperplastic polyp that is less than 5 mm in size. Right panel: a typical pedunculated adenomatous polyp.

Courtesy of James B McGee, MD.

Graphic 66254 Version 1.0

Normal sigmoid colon



Endoscopic appearance of the normal sigmoid colonic mucosa. The fine vasculature is easily visible, and the surface is shiny and smooth. The folds are of normal thickness.

Courtesy of James B McGee, MD.

Graphic 55563 Version 1.0

US multi-society task force recommendations for post-colonoscopy follow-up in average-risk adults with serrated polyps*

Baseline colonoscopy finding	Recommended interval for surveillance colonoscopy	Strength of recommendation	Quality of evidence
≤20 HPs in rectum or sigmoid colon <10 mm¶	10 years[Δ]	Strong	Moderate
≤20 HPs proximal to sigmoid colon <10 mm¶	10 years	Weak	Very low
1 to 2 SSPs <10 mm	5 to 10 years	Weak	Very low
3 to 4 SSPs <10 mm	3 to 5 years	Weak	Very low
5 to 10 SSPs <10 mm	3 years	Weak	Very low
SSP ≥10 mm	3 years	Weak	Very low
SSP with dysplasia◇	3 years	Weak	Very low
HP ≥10 mm	3 to 5 years[§]	Weak	Very low
TSA	3 years	Weak	Very low
Piecemeal resection of SSP ≥20 mm	6 months	Strong	Moderate[¥]

SSP: sessile serrated polyp; HP: hyperplastic polyp; TSA: traditional serrated adenoma; CRC: colorectal cancer.

* All recommendations assume examination complete to cecum with bowel preparation adequate to detect lesions >5 mm in size; recommendations do not apply to individuals with a hereditary CRC syndrome, personal history of inflammatory bowel disease, personal history of hereditary cancer syndrome, serrated polyposis syndrome, or malignant polyp, personal history of CRC, or family history of CRC, and must be judiciously applied to individuals with a personal or family history of CRC, favoring the shortest indicated interval based on either history or polyp findings.

¶ Patients with cumulative >20 hyperplastic polyps distributed throughout the colon, with at least 5 being proximal to the rectum, as well as those with 5 serrated polyps proximal to the rectum >5 mm, with at least two ≥10 mm meet criteria for serrated polyposis syndrome and may require specialized management.

Δ Follow-up may be with colonoscopy or other screening modality for average risk individuals.

◇ Assumes high confidence of complete resection.

§ A 3-year follow-up interval is favored if concern about consistency in distinction between SSP and HP locally, bowel preparation, or complete excision, whereas a 5-year interval is favored if low concerns for consistency in distinction between SSP and HP locally, adequate bowel preparation, and confident complete excision.

¥ Refer to US Multi-Society Task Force recommendations for endoscopic removal of colorectal lesions.

Graphic 127124 Version 4.0

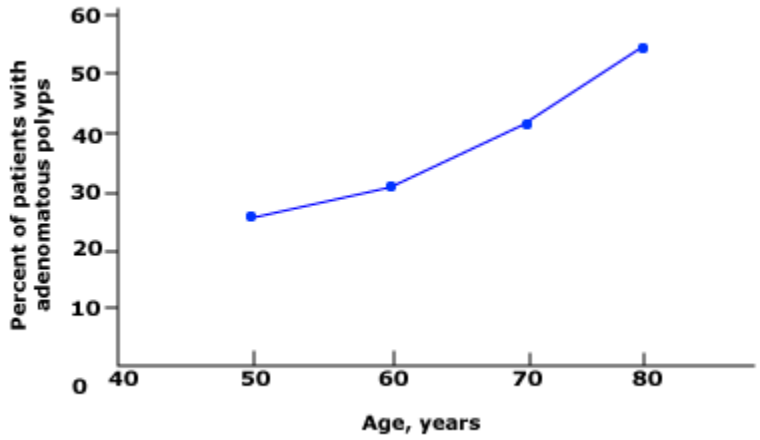
Molecular classification of colorectal carcinoma

	Chromosomal instability pathway (APC)	Mismatch repair pathway	Serrated pathway	
	Hereditary (FAP) and sporadic	Hereditary	Hereditary	Sporadic
CIMP status	Negative	Negative	High	High
MSI status	MSS	MSI-H	MSI-H	MSI-L
Chromosomal instability	+++	---	---	---
KRAS mutation	+++	+/-	---	---
BRAF mutation	---	---	+++	+++
MLH1 status	Normal	Mutation	Methylated	Partial methylation

CIMP: CpG island methylator phenotype; MSS: microsatellite stability; MSI: microsatellite instability; MSI-H: high-level microsatellite instability; MSI-L: low-level microsatellite instability; +++: present; +/-: might or might not be present; ---: absent.

Graphic 77353 Version 5.0

Prevalence of adenomatous colonic polyps increases with age

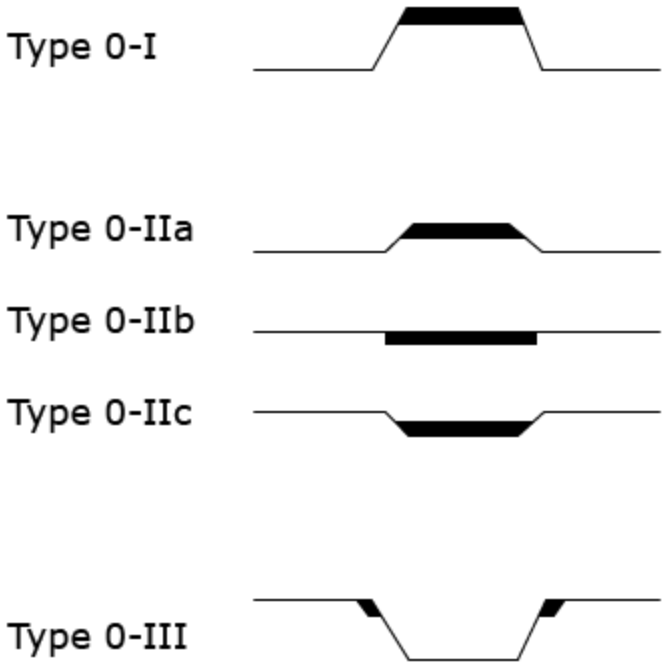


Adenomatous colonic polyps are found in about 25 percent of people by the age of 50; the prevalence continues to increase with increasing age.

Data from: Williams AR, Balasooriya BA, Day DW, Gut 1982; 123:835.

Graphic 55319 Version 2.0

Paris classification system of superficial neoplastic lesions of the gastrointestinal tract



Paris classification system of superficial neoplastic lesions of the esophagus, stomach, and colon. Type 0-I lesions are polypoid (protruded or pendunculated); type 0-II lesions are nonpolypoid and may be slightly elevated (IIa), flat (IIb), or slightly depressed (IIc); type 0-III lesions are excavated.

Based on data from: The Paris endoscopic classification of superficial neoplastic lesions: esophagus, stomach and colon: November 30 to December 1, 2002. Gastrointest Endosc 2003; 58(6 suppl):S3.

Graphic 61277 Version 3.0

Paris classification system of superficial gastrointestinal neoplastic lesions

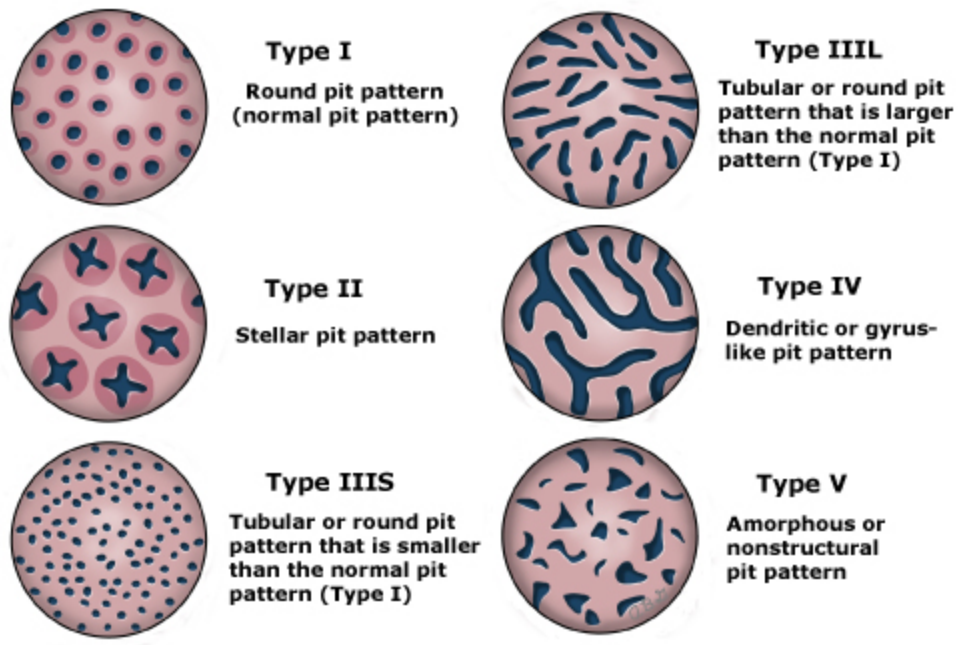
Type	Subclasses
0-I: Polypoid	0-Ip: Protruded, pedunculated
	0-Is: Protruded, sessile
0-II: Nonpolypoid	0-IIa: Slightly elevated
	0-IIb: Flat
	0-IIc: Slightly depressed

0-III: Excavated

The Paris endoscopic classification of superficial neoplastic lesions: Esophagus, stomach and colon: November 30 to December 1, 2002. Gastrointest Endosc 2003; 58(6 suppl):S3.

Graphic 50239 Version 5.0

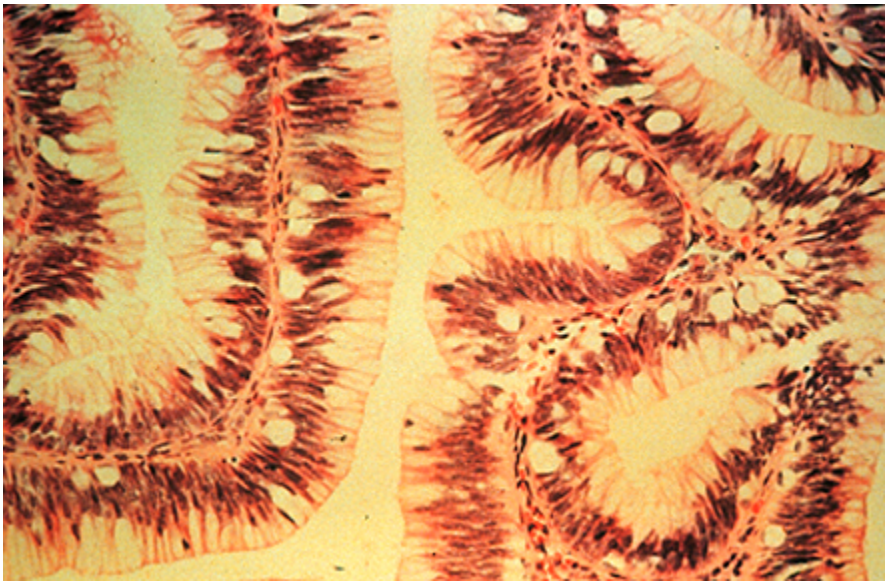
Kudo Pit Pattern Classification of colonic mucosal lesions



Pit pattern classification for colonic mucosal lesions.

Graphic 69425 Version 6.0

Colonic adenoma

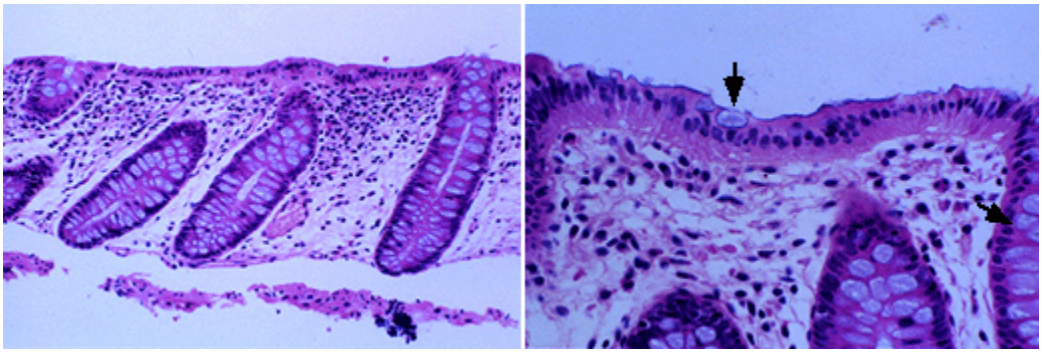


High power view of a colonic tubular adenoma shows mildly dysplastic crypts without invasion of the lamina propria.

Courtesy of Robert Odze, MD.

Graphic 51968 Version 1.0

Normal colon

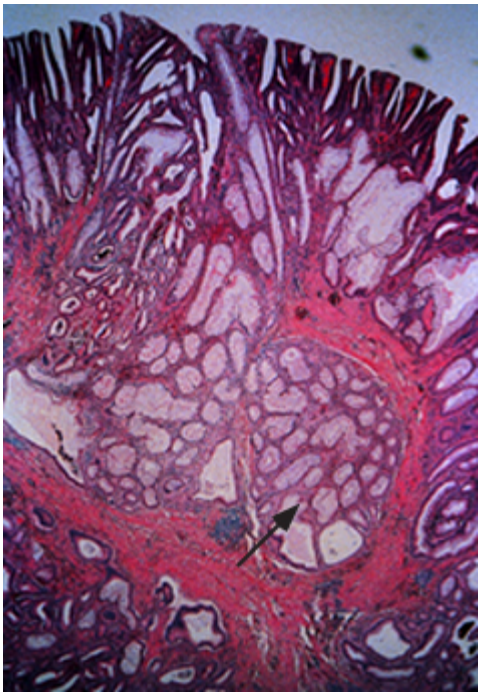


Low (left) and high (right) power views of a biopsy of a normal colon. Low power reveals straight crypts and mild lamina propria mononuclear cell infiltration. High power shows the surface enterocytes with interspersed goblet cells (arrows).

Courtesy of Robert Odze, MD

Graphic 81083 Version 6.0

Colonic adenoma with pseudoinvasion

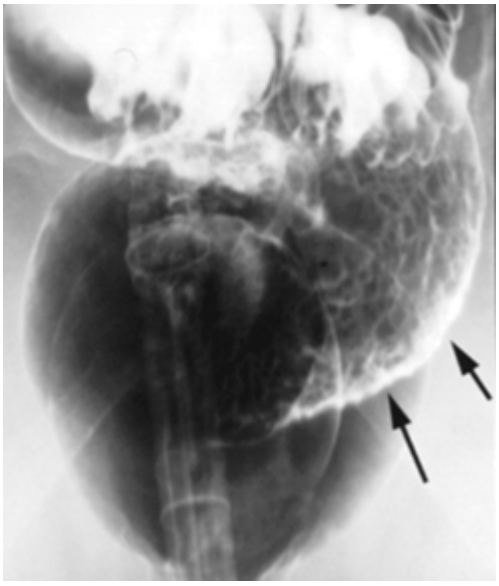


Low power view of a benign colonic adenoma containing a central focus of "misplaced" pseudoinvasive crypts in the polyp stalk (arrow).

Courtesy of Robert Odze, MD.

Graphic 63727 Version 1.0

Villous adenoma

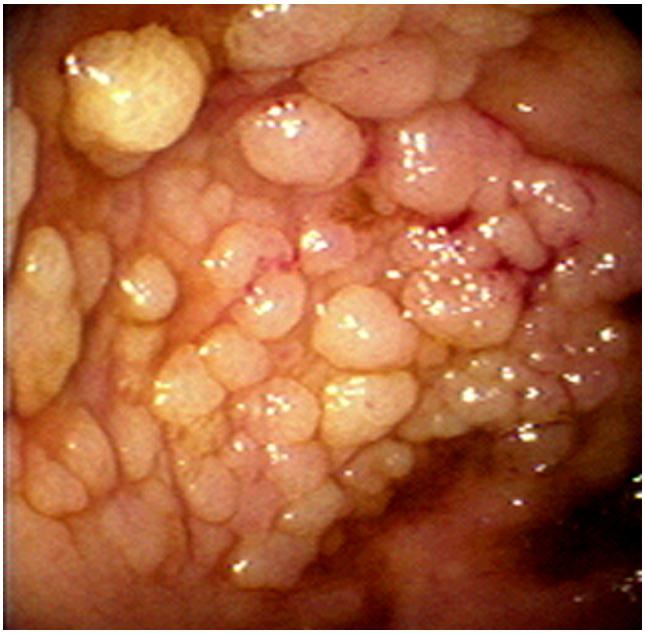


Double contrast barium enema shows a focal region of mucosal irregularity (arrows) caused by a flat villous adenoma. These benign tumors may be pedunculated or sessile.

Courtesy of Jonathan Kruskal, MD, PhD.

Graphic 61015 Version 2.0

Cecal adenoma

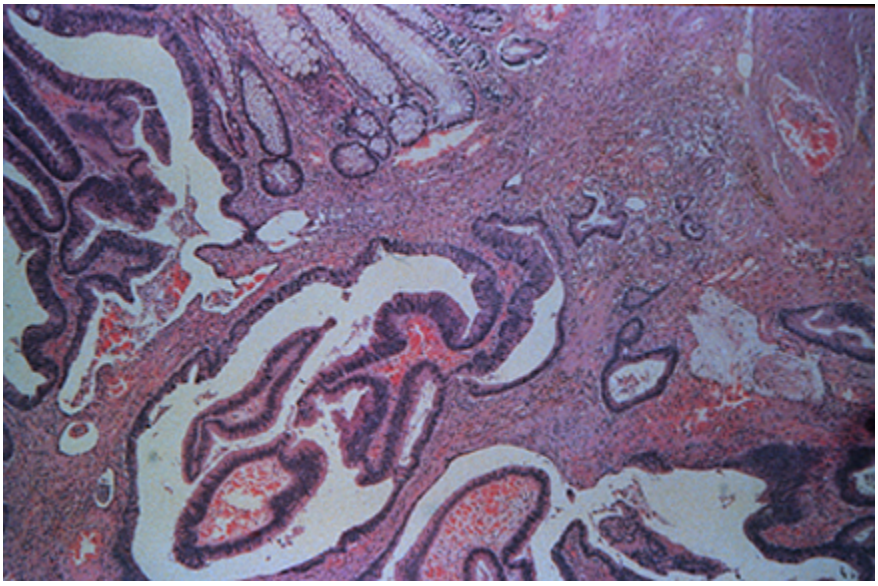


Large, multilobuluated polyp in the cecum with the endoscopic appearance of a villous adenoma, which was confirmed by histology.

Courtesy of Eric D Libby, MD.

Graphic 60933 Version 1.0

Colonic adenoma with malignant transformation



Low power view of a colonic adenoma which has undergone malignant transformation.

Courtesy of Robert Odze, MD.

Graphic 70756 Version 1.0

US multi-society task force recommendations for post-colonoscopy follow-up in average-risk adults with normal colonoscopy or adenomas*

Baseline colonoscopy finding	Recommended interval for surveillance colonoscopy	Strength of recommendation	Quality of evidence
Normal	10 years¶	Strong	High
1 to 2 tubular adenomas <10 mm	7 to 10 yearsΔ	Strong	Moderate
3 to 4 tubular adenomas <10 mm	3 to 5 years	Weak	Very low
5 to 10 tubular adenomas <10 mm	3 years	Strong	Moderate
Adenoma ≥10 mm	3 years	Strong	High
Adenoma with tubulovillous or villous histology	3 years◇	Strong	Moderate
Adenoma with high-grade dysplasia	3 years◇	Strong	Moderate
>10 adenomas on single examination§	1 year	Weak	Very low
Piecemeal resection of adenoma ≥20 mm	6 months	Strong	Moderate¥

CRC: colorectal cancer.

* All recommendations assume examination complete to cecum with bowel preparation adequate to detect lesions >5 mm in size; recommendations do not apply to individuals with a hereditary CRC syndrome, personal history of inflammatory bowel disease, personal history of hereditary cancer syndrome, serrated polyposis syndrome, malignant polyp, personal history of CRC, or family history of CRC, and must be judiciously applied to such individuals, favoring the shortest indicated interval based on either history or polyp findings.

¶ Follow-up may be with colonoscopy or other screening modality for average-risk individuals.

Δ Patients with recommendations issued before 2020 for shorter than 7- to 10-year follow-up after diagnosis of 1 to 2 tubular adenomas may follow original recommendations. If feasible, physicians may re-evaluate patients previously recommended an interval shorter than 10 years and reasonably choose to provide an updated recommendation for 7- to 10-year follow-up, taking into account factors such as quality of baseline examination, polyp history, and patient preferences.

◇ Assumes high confidence of complete resection.

§ Patients with >10 adenomas or lifetime >10 cumulative adenomas may need to be considered for genetic testing based on absolute/cumulative adenoma number, patient age, and other factors such as family history of CRC (refer to UpToDate text).

¥ Refer to US Multi-Society Task Force recommendations for endoscopic removal of colorectal lesions.

Graphic 127123 Version 5.0

US Multi-Society Task Force on Colorectal Cancer recommendations for second surveillance stratified by adenoma findings at baseline and first surveillance colonoscopy

Baseline finding	Recommended interval for first surveillance (years)	Finding at first surveillance	Recommended interval for next surveillance (years)
1 to 2 tubular adenomas <10 mm	7 to 10	Normal colonoscopy*	10
		1 to 2 tubular adenomas <10 mm	7 to 10
		3 to 4 tubular adenomas <10 mm	3 to 5
		Adenoma ≥10 mm in size; or adenoma with tubulovillous/villous histology; or adenoma with high grade dysplasia; or 5 to 10 adenomas <10 mm	3
3 to 4 tubular adenomas <10 mm	3 to 5	Normal colonoscopy*	10
		1 to 2 tubular adenomas <10 mm	7 to 10
		3 to 4 tubular adenomas <10 mm	3 to 5
		Adenoma ≥10 mm in size; or adenoma with tubulovillous/villous histology; or adenoma with high grade dysplasia; or 5 to 10 adenomas <10 mm	3
Adenoma ≥10 mm in size; or adenoma with tubulovillous/villous histology; or adenoma with high-grade dysplasia; or 5 to 10 adenomas <10 mm	3	Normal colonoscopy*	5
		1 to 2 tubular adenomas <10 mm	5
		3 to 4 tubular adenomas <10 mm	3 to 5
		Adenoma ≥10 mm in size; or adenoma with tubulovillous/villous	3

		histology; or adenoma with high grade dysplasia; or 5 to 10 adenomas <10 mm	

* Normal colonoscopy is defined as colonoscopy where no adenoma, sessile serrated adenoma, or colorectal cancer is found.

Graphic 127126 Version 4.0

Contributor Disclosures

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