Update on colorectal polyps and polyposis syndromes

Laura D Wood

Abstract

Colorectal polyps are frequently encountered in daily pathology practice. The accurate diagnosis of these polyps forms a key component of clinical care, both in determining the malignant potential (and thus the follow-up interval for colonoscopy) as well as raising suspicion for polyposis syndromes. As such, the pathologist plays an instrumental role in the care of patients with colon polyps. This review highlights the histologic features of several commonly (and uncommonly) encountered colon polyps and provides genetic and clinical features of several polyposis syndromes that can be diagnosed (or at least suspected) when evaluating biopsies of colon polyps.

Keywords colon cancer; colonoscopy; colonic neoplasms; colonic polyps; polyposis

Introduction

Because of the use of colonoscopy with biopsy as a screening tool for colorectal cancer, the histologic evaluation of colon polyps is one of the tasks most frequently encountered in daily pathology practice. However, in addition to the neoplastic precursors to colorectal cancer, these biopsies can reveal many other types of polyps, ranging from incidental non-neoplastic lesions to rare polyps that signal the need for follow-up to exclude an inherited polyposis syndrome. Therefore, it is crucial not only to correctly diagnose these polyps but also to indicate when further evaluation is warranted.

Non-neoplastic polyps

Inflammatory polyps

Inflammatory polyps can occur in a variety of clinical settings, ranging from incidental findings at sites of local trauma to sequelae of inflammatory bowel disease. The crypts are often dilated or distorted, and the lamina propria is expanded by acute and chronic inflammatory infiltrate (Figure 1). Epithelial inflammation is also common, and erosion with underlying granulation tissue can be prominent. Marked reactive epithelial and stromal changes are common in inflammatory polyps and must be distinguished from true neoplasia, a distinction that is particularly difficult in patients with inflammatory bowel disease who are at increased risk for inflammatory polyps and colonic epithelial dysplasia. Surface maturation in reactive epithelial changes can be a helpful feature to exclude dysplasia. Of note, inflammatory polyps can be difficult to distinguish from juvenile polyps on histologic grounds alone, and clinical history (such as patient age and history of inflammatory bowel disease) is often helpful in arriving at a diagnosis. If the patient is symptomatic or the flat mucosa appears endoscopically abnormal, biopsies of non-polypoid mucosa may be helpful to exclude inflammatory bowel disease or other diffuse mucosa injury. However, because these polyps are often solitary and incidental, workup for inflammatory bowel disease is not warranted solely on the basis of the presence of an inflammatory polyp.

Filiform polyps, also known as post-inflammatory polyps, occur at sites of prior mucosal injury. These polyps are often noted endoscopically as long finger-like projections. They can be multiple in patients with inflammatory bowel disease but can occur in any clinical setting that leads to ulceration of the colonic mucosa. Histologically, they consist of cylindrical projections of submucosa surrounded by mucosa on all sides — the layers of mucosa often have one or no intervening layers of muscularis mucosae.

Mucosal prolapse changes/polyps

Mucosal prolapse changes can occur throughout the colon, though some sites (such as the ileocecal valve and distal sigmoid and rectum) have a higher tendency towards prolapse changes than others. Prolapsed mucosa often forms a discrete polyp that is identified and biopsied by the endoscopist. Histologically, mucosal prolapse polyps have hypertrophy of the muscularis mucosae, leading to smooth muscle ingrowth into the lamina propria as well as accompanying lamina propria fibrosis (Figure 2). This muscular proliferation and fibrosis leads to distortion of the colonic crypts, which can become diamond-shaped. Serrated changes are common and can lead to diagnostic confusion with sessile serrated adenomas. These polyps can also ulcerate, which can lead to inflammatory changes on the surface and overlap with inflammatory polyps — the muscular proliferation and fibrosis in the lamina propria of mucosal prolapse polyps should help to distinguish them from inflammatory polyps.

Several specific types of mucosal prolapse changes have been described. In solitary rectal ulcer syndrome, mucosal prolapse changes in the distal rectum can lead to ulceration, polyp formation, or both — these lesions are occasionally noted endoscopically to be mass-like and worrisome for an underlying neoplasm. However, the histologic findings are identical to those seen in mucosal prolapse. Inflammatory cloacogenic polyps arise at the anorectal junction and contain both squamous and columnar mucosa. These polyps often display a tubulovillous growth pattern but share the other histologic features of mucosal prolapse, including prominent lamina propria fibrosis and surface ulceration. Mucosal prolapse changes also frequently occur in association with diverticular disease, which occurs most often in the sigmoid colon. The mucosa adjacent to a diverticulum can be thrown up into prominent folds, which then prolapse into the lumen and acquire the histologic changes of mucosal prolapse polyps.

Adenomatous polyps

Tubular adenomas

Identification and removal of tubular adenomas, the dysplastic precursors to colorectal adenocarcinoma, is a major goal of

Laura D Wood *MD PhD Department of Pathology, Johns Hopkins University School of Medicine, Baltimore, MD, USA. Conflicts of interest: none declared.*

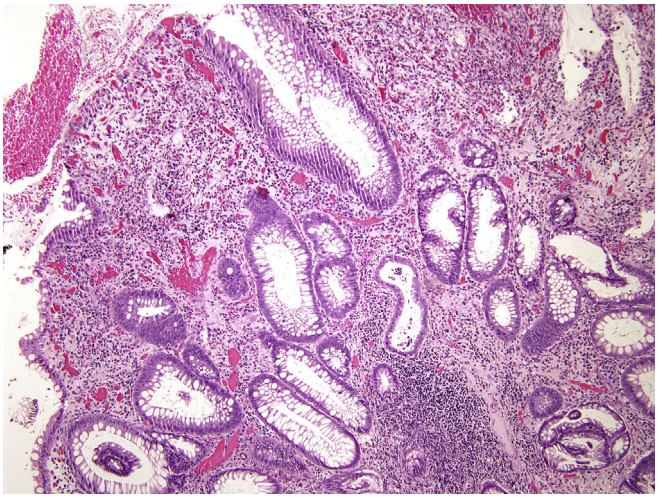

Figure 1 Inflammatory polyp. The lamina propria of this inflammatory polyp is expanded with acute and chronic inflammation, and the crypts are inflamed, dilated, and distorted.

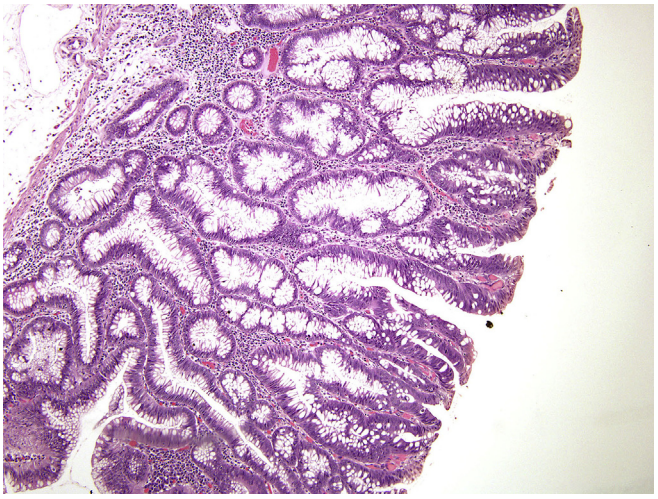

Figure 3 Tubular adenoma. This tubular adenoma shows the typical elongated "pencillate" nuclei with "top—down" dysplasia.

screening colonoscopy. Because subsequent screening practice depends on the pathologic findings after colonoscopy, accurate diagnosis of these polyps is crucial for effective patient care.

Tubular adenomas have at least low grade dysplasia by definition. Histologically they are characterized by hyperchromatic elongated "pencillate" nuclei that maintain their polarity (Figure 3). Their hyperchromasia is uniform throughout the crypts, unlike nondysplastic epithelium that matures at the surface, leading to a description of dysplasia in tubular adenomas as "top—down". In adenomas, the crypts lose their organized architecture and frequently show irregular branched shapes. There are several morphologic variations that can be seen in adenomas, including squamous morules, clear cell change, Paneth cell differentiation, and focal microcarcinoid areas. None of these variations holds any clinical significance, but recognition can prevent misdiagnosis as a more advanced lesion. Tubular adenomas can contain significant inflammation, and the dysplastic epithelium frequently contains intraepithelial neutrophils. As such, inflammation should not be used to favor an inflammatory polyp in polyps with architectural and cytologic features of a tubular adenoma.

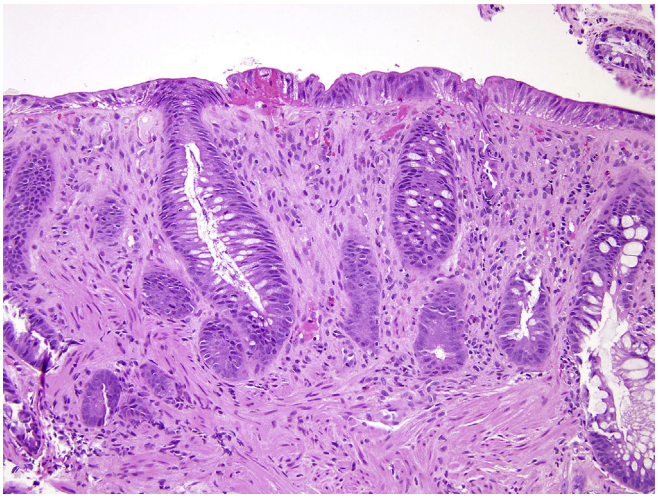

Figure 2 Mucosal prolapse polyp. There is smooth muscle ingrowth and fibrosis in the lamina propria in this mucosal prolapse polyp.

The recommended colonoscopic surveillance interval is ten years for low risk patients over 50 years old. However, there are several pathologic features that impart a shortened surveillance interval and thus are important to include in the diagnosis of adenomas.[1] Specifically, patients with more than three adenomas, with one or more adenomas >10 mm in size, with one or more adenomas with villous morphology, or with one or more adenomas with high grade dysplasia should be screened at a three year interval. The size and number of polyps will be noted by the gastroenterologist, but the presence of villous morphology and high grade dysplasia must be noted by the pathologist. Although their presence should be noted in any tubular adenoma, these features only change the screening interval in patients with few small polyps (as patients with many polyps or large polyps will already have a shortened screening interval). Unfortunately, recent studies have shown a striking lack of reproducibility in the diagnosis of villous morphology and high grade dysplasia in small tubular adenomas.[2] In spite of this, these features still remain a key part of the current surveillance recommendations and should be included in the pathology report when they can be reliably diagnosed. The morphologic criteria for high grade dysplasia can be divided into architectural and cytologic atypia. Architectural complexity, most often noted as cribriform glands, should prompt a diagnosis of high grade dysplasia. In addition, cytologic atypia, including loss of nuclear polarity and pleomorphism, is also indicative of high grade dysplasia, though the degree of atypia required for the diagnosis is inherently subjective (Figure 4). The diagnosis of villous morphology is similarly subjective, though most experts agree that well-formed villi (rather than a small focus that might be interpreted as villous) should be present to diagnose a tubulovillous adenoma — others suggest that an adenoma should be at least 25% villous to diagnose it as a tubulovillous adenoma (Figure 5). Although subjective, these diagnoses on their own often do not change the surveillance interval because both high grade dysplasia and villous morphology correlate with the size of the adenoma.