Polyps and polypoid lesions of the anus

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Abstract

Anal polyps are a relatively rare and neglected part of pathology practice. Similar to other parts of the gastrointestinal tract, anal polyps are predominantly epithelial in origin, but mesenchymal lesions do occur. The aetiology of anal polyps is diverse and includes infectious, reactive, developmental and neoplastic conditions. Thus an awareness of the clinical scenario can be very informative in difficult cases. Diagnostic pitfalls are perhaps more common than is generally realized and misdiagnosis can have important management and social implications. Herein, we discuss the most common and interesting anal polyps and polypoid lesions with a particular focus on diagnostic pitfalls. Careful attention to the clinical scenario and histological features, along with judicious use of immunohistochemistry will resolve most diagnostic dilemmas.

Keywords anus; diagnosis; human papilloma virus; pathology; polyps

Introduction

Anal polyps are uncommon, accounting for significantly less than one percent of gastrointestinal (GI) polyps. As such they are a neglected part of pathological practice. Recently there has been a renewed interest in anal pathology, driven largely by the rise in human papilloma virus (HPV) related anal pathology. A by-product of anal surveillance for HPV is the increasing identification of incidental anal lesions that then require pathological diagnosis. Usually these diagnoses are straightforward; however some entities create diagnostic dilemmas. This review focuses on the more common polyps and polyp-like lesions of the anus and particularly emphasizes areas where misdiagnosis occurs.

Anal embryology and anatomy

The anus is an anatomically and embryologically complex region and a comprehensive discourse of these topics is beyond the scope of this review. We refer interested readers to excellent dedicated reviews of the subjects by Pandey and Dujovny et al.[1,2] However, a working knowledge of the embryology and the basic anatomical compartments is essential for accurate reporting of anal specimens.

The proximal portion of the anus is derived from the endodermal hindgut and the distal portion from the ectodermal proctodeum.[3] The dentate (pectinate) line represents the point of fusion of these structures and also roughly corresponds to the mid point of the anal canal. It is an important landmark as the lymphatic's proximal to the dentate line drain to the inferior mesenteric lymph nodes, whereas the distal drainage is to the internal iliac and inguinal nodes.[2] The endodermal hindgut has a larger calibre than the proctodeum and as such the anus above the dentate line is thrown into folds to accommodate this discrepancy. These folds are recognized as the columns of Morgagni and the intervening anal crypts.

Anatomically the anus begins at the level of the levator ani muscle and ends 5 cm external to the anal verge. The anus is then divided into three key regions namely; the anal transformation zone (ATZ), encompassing the 1–2 cm of mucosa proximal to the dentate line; the intra-anal compartment, extending from the dentate line to the anal verge and the peri-anal region which extends 5 cm external to the anal verge. The anal canal includes the transformation zone and the intra-anal compartment and is about 4 cm in length.

Precise localization of anal pathology is of critical importance. In particular, the management of peri-anal lesions can be vastly different to intra-anal lesions. Confusion tends to occur when a lesion crosses an anatomical boundary. Currently, if a lesion cannot be completely visualized with gentle traction on the buttocks it is considered to be intra-anal. If a lesion can be completely visualized with gentle traction on the buttocks then it is considered to be peri-anal.[4]

Anal histology

The upper extent of the anus is composed of rectal type mucosa. This quickly changes to the ATZ, which represents the transition from glandular rectal type mucosa to squamous anal mucosa. The transition zone itself is lined by a metaplastic squamous epithelium that resembles urothelium. The ATZ ends at the dentate line to become non-keratinizing squamous mucosa. The squamous mucosa of the intra-anal compartment is devoid of adnexal structures and this allows distinction from the peri-anal skin that begins at the anal verge. Melanocytes are present in the squamous mucosa of the intra-anal compartment and in lesser numbers in the ATZ but are not normally present in rectal type mucosa.[5] The anal ducts enter the anus via the anal crypts just proximal to the dentate line. The anal glands are lined by stratified columnar epithelium and are most often found in the submucosa of the anterior anus.

Anal polyps

For the purposes of this review we divide the discussion into; 1. 'true' anal polyps, that is, lesions that essentially always present as a discrete elevated lesion, and 2. 'polypoid' anal lesions, that is, lesions that can present as a polyp or polyp-like mass in some patients but can also present as non-discrete mass lesions. Table 1 represents a comprehensive list of entities that can theoretically present as an anal polyp, only the more relevant of these will be discussed.

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All entities that can potentially present as an anal polyp

Entity	Presentation in the anus	Presentation as a polyp
Neoplastic		
Epithelial		
Squamous cell carcinoma	+++	++
Adenocarcinoma	+	+
Adenoma	++	++++
Neuroendocrine carcinoma	+	+
Basal cell carcinoma	+	+++
Adnexal type tumours	+	++
Mesenchymal		
Neurofibroma	+	++
Granular cell tumour	+	+++
Lipoma	+	++
Fibrolipoma	+	++
Lymphangioma	+	+++
Kaposi sarcoma	+	+
Angiosarcoma	+	+
Aggressive angiomyxoma	+	++
Leiomyoma/sarcoma	+	+++
Fibrohistiocytic	+	+
Inflammatory fibroid tumour	+	++
Gastrointestinal stromal tumour	+	++
Melanoma	++	++
Haematolymphoid	+	+
Non-neoplastic lesions		
Fibroepithelial polyp	++++	++++
Inflammatory cloacogenic polyp	+++	++++
Haemorrhoids	+	++
Endometriosis	+	+
Heterotopia (prostate, breast, gastric)	+	+++
Amyloid	+	+
Infectious		
HPV (condyloma accuminatum)	+++	+++
Treponema pallidum (condyloma lata),	+	+
Granulomatous (*Mycobacterium tuberculosis*, Enterobius)	+	+
Cysts		
Epidermoid, dermoid, tailgut, median raphe, anal gland, duplication	++	++

+ Rarely; ++ sometimes; +++ usually; ++++ essentially always.

Table 1

True anal polyps

Fibroepithelial polyp

The fibroepithelial polyp (anal tag, hypertrophied anal papilla) is the most common anal polyp. They are non-neoplastic and typically arise from the anal papillae at the base of the columns of Morgagni. This occurs on a background of chronic injury and inflammation. In particular, fissures of the anal valves are frequent antecedents to FEPs, and thus they are common in patients with Crohn's disease.

Clinical findings: the majority of FEPs come to clinical attention because of local irritation related to incontinence and discharge. Rarely they can bleed secondary to trauma. They may be attached to the surrounding mucosa by either a narrow pedicle or a broad base.

Pathological features: FEPs are variable in size and are soft and fleshy when in situ. They are lined by non-keratinizing squamous epithelium. Because they are subjected to recurrent trauma, areas of hyper and parakeratosis are universal and ulceration will sometimes be present. Serum lakes are often seen in the superficial layers of the epithelium and are useful diagnostically, as this feature is rare in condylomata. The stroma is variable; in most it is myxoid with abundant ectatic vessels, however over time it becomes pauci-cellular and collagenized.[6] The myxoid appearance is more prominent in subepithelial regions. At the base of FEPs, vessel walls may exhibit hyaline change, but no true vasculitis. Bi or multinucleate atypical stromal cells can be identified in many cases. CD34 is characteristically expressed in these cells and they are reactive for desmin in one third of cases, but are of no significance. Most cases contain some smooth muscle that is usually continuous with the underlying muscularis mucosa. An infiltrate of Mast cells is often present but unless ulcerated or arising in a setting of Crohn's disease, significant inflammation is not seen.

FEPs sometimes arrive with a clinical query of Crohn's disease. The only histological feature useful in this distinction are granulomas, which are reported in 30% of patients with Crohn's disease but never in patients without (Figure 1).[7]

Diagnostic pitfalls: despite the general lack of interest surrounding FEPs, problems with diagnosis are surprisingly common. In particular, over-diagnosis of reactive epithelial changes as HPV-induced koilocytosis is a recurring issue. In response to injury, the superficial squamous cells of FEPs can develop voluminous clear cytoplasm, accompanied in some cases by centrally placed nuclei. This can be erroneously interpreted as koilocytosis, leading to the diagnosis of condyloma acuminatum. This is not a trivial mistake, as it may result in inappropriate follow-up and treatment and can also have significant social implications. In one series, 40% of polyps diagnosed as condyloma acuminata of the anus were actually FEPs.[8]

Attention to the architectural and cytological features helps avoid this misdiagnosis. FEPs do not have the verrucous pattern of a condyloma acuminatum and lack the genuine HPV-induced nuclear changes, such as binucleation, chromatin margination, nuclear indentation and central clearing. True HPV effect in FEPs is exceedingly rare and probably only occurs in the setting of HPV in the adjacent anal epithelium. In unresolved cases, immunohistochemistry can be applied. Ki67 staining will typically be negative in the upper two thirds of the epithelium in FEPs, whereas condyloma will usually show some positive staining in the upper layers. P16 is generally not helpful as it will usually be negative in both lesions.