Review

Benign “lumps and bumps” of the vulva: A review

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ABSTRACT

Vulvar dermatology represents a challenge for many providers. Given that the vulva is both a gynecologic and dermatologic organ, patients with cutaneous lesions involving the vulva may present to primary care, gynecology, or dermatology. Particularly within dermatology, the vulva remains understudied, which can lead to anxiety among providers regarding appropriate next steps in the diagnosis and management of vulvar lesions. Thus, the purpose of this review is to highlight commonly encountered anatomic variants and benign neoplasms of the vulva, distinguish them from key pathologic mimickers, and provide guidance to practicing dermatologists on what may constitute normal vulvar variations.

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Contents

Introduction .................................................................................................................................................. 383
Anatomic variants ..................................................................................................................................... 384
Vestibular papillomatosis .......................................................................................................................... 384
Variations in vulvar dimensions ............................................................................................................... 384
Benign neoplasms ..................................................................................................................................... 385
Urethral caruncle ...................................................................................................................................... 385
Seborrheic keratosis .................................................................................................................................. 385
Syringoma .................................................................................................................................................. 387
Hidradenoma papilliferum ....................................................................................................................... 388
Cystic lesions ............................................................................................................................................ 388
Epidermal inclusion cysts ......................................................................................................................... 388
Vestibular gland cysts ............................................................................................................................. 389
Conclusion ................................................................................................................................................ 389
Declaration of Competing Interest ........................................................................................................ 389
Funding ....................................................................................................................................................... 389
Study approval ......................................................................................................................................... 389
Supplementary materials ....................................................................................................................... 389
References ................................................................................................................................................ 390

Introduction

The vulva refers to the external female genitalia and includes the mons pubis, labia majora, labia minora, clitoris, vulvar vestibule, urethral meatus, vaginal introitus, and Bartholin’s and Skene’s vestibular glands (Nguyen and Duong, 2020). As both a gynecologic and dermatologic organ, the vulva lies well within the purview of the dermatologist’s practice. That said, vulvar lesions can often elicit an elevated level of anxiety for patients and dermatologists alike—this is likely due to the broad range of etiologies of vulvar lesions, some of which carry significant risk of morbidity, but many of which are benign (Kelekci et al., 2016). Dermatologists’ ability to recognize benign variants of the vulva is critical, not only to reduce patient anxiety, but also to minimize unnecessary workup.
Unfortunately, the current literature on benign neoplasms and anatomic variants of the vulva is both limited in scope and principally found in gynecologic journals not widely read by dermatologists. Thus, the purpose of this review is to highlight some of the most commonly encountered anatomic variants and benign neoplasms of the vulva, distinguish them from key pathologic mimickers, and provide reassurance as to what may constitute normal vulvar variations. These entities include vestibular papillomatosis (VP), variation in vulvar dimension, urethral caruncle (UC), seborrheic keratosis (SK), syringoma, epidermal inclusion cyst (EIC), vestibular gland cyst (VGC), and hidradenoma papilliferum (HP).

**Anatomic variants**

**Vestibular papillomatosis**

VP was first described in 1982 by Altmeyer et al. (1981) as “pseudocondylomata of the vulva” and has since been reported under a variety of names, including hirsutoid papillomas of the vulva, vulvar squamous papillomatosis, micropapillomatosis labialis, and squamous vestibular micropapilloma (Altmeyer et al., 1981; Sarifakioglu et al., 2006). The exact prevalence of VP remains unknown, but current estimates suggest that it ranges from 1% to 33% of the population (Welch et al., 1993). Importantly, despite the suggestion of its monikers and its clinical resemblance to human papillomavirus (HPV)-associated condyloma acuminata, VP is considered a benign anatomic variant of the vulva (Fig. 1; Moyal-Barracco et al., 1990).

Clinically, VP is characterized by the presence of multiple frond-like mucosal papillae in a linear and symmetric distribution within the vulvar vestibule and inner labia minora. The presence of grouped papules in the vulva invokes HPV-associated condylomas as part of the differential, but VP can be distinguished from condylomas by two key features. First, whereas the filiform projections of condylomas tend to fuse at their base, the bases of individual projections of VP remain separate and distinct. Second, whereas condylomas tend to be firmer, randomly distributed, and not confined to the vulvar vestibule, VP tends to be softer and symmetrically distributed within the confines of the inner labia minora and vestibule (Muhammed et al., 2019).

Although clinical appearance and workup alone are usually sufficient to make a diagnosis of VP, should histopathological analysis be pursued, prominent fibrovascular cores covered by mature squamous epithelium are characteristic of VP. Epithelial cells often demonstrate clear vacuoles containing glycogen, rather than the true koilocytic changes that are seen in condylomas (Sarifakioglu et al., 2006). Importantly, because VP represents a normal physiologic variant, it does not require treatment beyond patient education and reassurance.

**Variations in vulvar dimensions**

Although not a specific clinical entity, variation in vulvar dimension and appearance can be challenging for dermatologists to contextualize, especially when lacking a robust background in performing vulvar examinations. Moreover, despite representations of female nudity being a common feature in popular culture, surprisingly few depictions of normal female genitalia and vulvar anatomy exist. More recently, a limited number of observational, cross-sectional studies within the gynecologic literature have sought to better characterize “normal” vulvar variations. Lloyd et al. (2005) described the genital dimensions of 50 healthy premenopausal women and found wide ranges in clitoral size (0.5–3.5 cm), labial length (7.0–12.0 cm for the labia majora, 2.0–10.0 cm for the labia minora), and rugosity (ranging from smooth to marked). Other studies in both pre- and postmenopausal women have demonstrated a similarly broad range of normal vulvar dimensions (Verkauf et al., 1992; Weber et al., 1995).

From the dermatologist’s perspective, it is important not only to appreciate these normal variations in vulvar dimensions, but also to contrast them with physical signs of scarring or erosive vulvar pathology. For example, both lichen sclerosus and erosive...
lichen planus are inflammatory lichenoid dermatoses of the vulva that, when left untreated, can lead to irreversible scarring, sexual dysfunction, and, in extreme cases, interference with urination (Fruchter et al., 2017). Although these dermatoses typically manifest with overt symptomatology (most commonly, pruritus or pain), in some instances early anatomic changes are the only clue that a scarring process is underway. Notably, although vulvovaginal atrophy in the setting of age-related hypoestrogenism may manifest with shrinkage of the labia minora and vulvar vestibule, overt signs of inflammation (including the presence of erosions, dyspigmentation, fusion of the labia minora with the labia majora, fusion and loss of mobility of the clitoral hood, and/or scarring of the vaginal introitus) should prompt a thorough inquiry into vulvar symptomatology and possible biopsy to evaluate for an inflammatory vulvar process (Fig. 2; Fruchter et al., 2017; Schlosser and Mirowski, 2015).

Benign neoplasms

Urethral caruncle

UCs are the most common benign tumor of the female urethra (Chiba et al. 2015; Ferrier, 1926). Most often arising from the posterior urethral meatus, these exophytic, polypoid masses are typically seen in postmenopausal women, although isolated reports in prepubertal women exist (Burkland, 1952; Conces et al., 2012; Haley et al., 1998). UCs are typically solitary and small in size (Fig. 3), but some patients may present with multiple lesions up to 1 to 2 cm in diameter. Their marked variation in size and morphology can make clinical recognition of UCs quite challenging (Ferrier, 1926; Lobo et al., 2017).

Although their etiology remains incompletely understood, UCs are thought to arise in the setting of hypoestrogenemia and vaginal atrophy. Inflamed urethral mucosa everts and ultimately progresses to localized prolapse of the posterior urethral wall (Haley et al., 1998; Lobo et al., 2017). Growth of the caruncle is thought to occur secondary to chronic irritation and inflammation. Moreover, antiestrogen therapies (e.g., tamoxifen) have been found to accelerate UC formation (Bachmann and Nevadunsky, 2000).

UCs may be asymptomatic or can present with pain, dysuria, bleeding, or other irritative urinary symptoms (Burkland, 1952; Coban and Biyik 2014; Conces et al., 2012). Early excisional biopsy must be considered should features suspicious for malignancy (e.g., changing size, irregular consistency, or failure to respond to topical estrogen) be noted (Chiba et al., 2015; Tunitsky et al., 2012). Histopathologic examination typically reveals a proliferation of transitional and stratified squamous epithelium with vascular connective tissue and an inflammatory infiltrate of lymphocytes and plasma cells (Haley et al., 1998). Benign entities, including pyogenic granuloma, urethral prolapse, varicosities, or perirethral gland abscesses, are common UC mimickers (Table 1; Lobo et al., 2017). Urethral prolapse typically can be distinguished from UC because a prolapse tends to manifest as an annular, circumferential lesion resembling a donut (Lobo et al., 2017). Notably, malignant neoplasms, including urothelial carcinoma, squamous cell carcinoma, melanoma, lymphoma, and sarcomas, can also share features with UC and must be considered in the differential (Conces et al., 2012).

If symptomatic, management of UC can be divided into a conservative treatment approach versus surgical resection. Initial therapy includes topical estrogen or steroidical ointments. If the caruncle does not regress, grows further, or symptoms continue, excisional biopsy is indicated (Lobo et al., 2017).

Seborrheic keratosis

First described by Freudenthal in 1927, SKs are exceedingly common, benign, epidermal tumors whose prevalence increases with age (Kwon et al., 2003; Yeatman et al., 1997). Although a link between HPV and genital SKs has also been suggested, several re-
| Clinical appearance | Histopathologic features | Treatment |
|---------------------|--------------------------|-----------|
| **Table 1**         |                          |           |
| **Characterization of benign vulvar lesions** |                         |           |
| **Cystic lesions**  |                          |           |
| Bartholin gland cyst | Small cystic swelling and/or palpable mass in the vulvar vestibule at 4 and 8 o’clock positions | Cyst lined by columnar squamous or flattened epithelium | For cysts that do not spontaneously resolve, catheterization or marsupialization can be considered |
| Canal of Nuck cyst  | Painless, reducible, inguinal-labial swelling | Hydrocele lined with columnar, transitional, and stratified squamous epithelium | Refer to gynecology and/or general surgery for surgical excision and closure of persistent canal |
| Epidermal inclusion cyst | Firm, round, yellow-white papulonodules | Cyst lined with keratinizing stratified squamous epithelium with an intact granular layer filled with laminated keratin | Reassurance; incision and drainage if infected; intraluminal steroids if inflamed; excision |
| Mesonephric cyst | Small cystic swelling and/or palpable mass on the lateral aspects of the vulva | Cyst lined by cuboidal cells | Reassurance; larger cysts may be excised |
| Milia | Waxy, firm, white to flesh-colored dome-shaped papules 1–3 mm in size | Homogenous deposition of eosinophilic colloid in the papillary dermis | Reassurance; electrodessication or expression of keratin contents after incision can be pursued for cosmesis |
| Skene’s duct cyst | Small cystic swelling and/or palpable mass adjacent to the urethral meatus, occasionally with drainage | Cyst lined by columnar, transitional, or squamous epithelium | For cysts that do not spontaneously resolve, marsupialization can be considered |
| Steatocystoma multiplex | Small, flesh-colored to yellowish cystic papules or nodules | Cyst lined by stratified squamous epithelium with sebaceous glands attached to cyst epithelium | Reassurance; incision and drainage if cosmetically unfavorable |
| Vestibular gland cyst | Translucent soft, smooth, and round cysts | Cyst lined by simple mucous-secreting columnar epithelium | Reassurance |
| **Glandular neoplasms** |                          |           |
| Hidradenoma papilliferum | Firm, smooth-surfaced, red, blue, or skin-colored nodule with well-defined capsule, 0.5–2.0 cm | Papillary and glandular hyperplasia | Excision |
| Poroma | Single, slow-growing, well-circumscribed, pink-to-red papule, nodule, or plaque | Proliferation of small, round, monotonous cuboidal cells (poroid cells) | Excision only indicated if malignancy suspected |
| Spiradenoma | Slow-growing, flesh-colored nodule, occasionally painful | Multilobulated basophilic dermal nodules surrounded by hyalinized collagen capsule | Excision only indicated if malignancy suspected |
| Syringocystadenoma Papilliferum | Firm, smooth-surfaced or verrucous red nodule or plaque | Papillary and glandular hyperplasia | Excision |
| Syringoma | Multiple 1–4 mm, firm, skin-colored to brownish-pink papules in symmetric, bilateral distribution on labia majora | Dermal proliferation of eccrine duct-like tubular structures lined with two layers of cuboidal epithelium | Reassurance; surgical excision, carbon dioxide laser vaporization, cryotherapy, or electrosurgery if symptomatic |
| **Melanocytic neoplasms** |                          |           |
| Melanocytic nevus | Pink to dark black-brown macules or papules with well-demarcated borders and uniform pigmentation | Groups of benign nevi cells in basal epidermis, dermis, or both | Excision only indicated if malignancy suspected |
| **Lymphovascular proliferations** |                          |           |
| Angiokeratoma | Dark-red to purple papules 2–5 mm in diameter | Large dilated blood vessels in the superficial dermis with overlying epidermal hyperkeratosis | Reassurance; electrocautery or pulsed dye laser can be employed for cosmesis |
| Endometriosis | Tender, reddish-brown papules or nodules that cause cyclical pain in association with menses | Dermal proliferation of endometrial glands lined by pseudostratified columnar epithelium | Referral to gynecology; wide local excision |
| Lymphangioma | “Frog egg”-like grouped thin-walled vesicles | Dilated lymphatic channels with flat endothelium | Reassurance; recurrence common after surgical excision, laser, or sclerotherapy |
| Pyogenic granuloma | Glistening, friable, bright-red papule or nodule that bleeds spontaneously or after trauma | Lobular proliferation of capillary size blood vessels | Excision is recommended to rule out malignancy |
| **Keratinocytic neoplasms** |                          |           |
| Epidermolysis acanthoma | Single or multiple, variably pigmented (flesh-colored to white to erythematous) papules | Hyperkeratosis, papillomatosis, and focal vacuolar epithelial degeneration | Diagnostic biopsy to rule out condyloma acuminata |
| Seborrhoeic keratosis | Firm, “stuck-on” appearing, well-demarcated papules and plaques often with “waxy” or oily texture | Sharply demarcated proliferation of monotonous epidermal keratinocytes | Reassurance |
| **Pilar/sebaceous lesions** |                          |           |
| Pilar cyst | Smooth, mobile nodule, variable size; when ruptured, significant associated inflammation can occur | Cyst lined with multilayered epithelium with characteristic absence of the granular layer | Excision; incision and drainage in setting of cyst rupture |
| Pilomatrixoma | Firm, subcutaneous nodules 1–3 cm in size, often with bluish-red hue | Circumscribed dermal nodule with shadow/ghost cells (basophilic cells resembling hair matrix cells with absent nuclei) | Excision only indicated if malignancy suspected |
| Trichoepithelioma | Flesh-colored or slightly erythematous, smooth and round papules, 1–5 mm in size | Small cords of basoloid cells in fibroblast-rich collagenous stroma | Excision only indicated if malignancy suspected |

(continued on next page)
cent studies have largely debunked this association (Reutter et al., 2014; Tardio et al., 2012).

SKs may be found on all keratinized skin surfaces, including the vulva (Fig. 4). SKs are characterized by their sharply demarcated, “stuck-on” appearance; color and size can vary considerably and the surface is often uneven and may appear oily or waxy (Hafner and Vogt, 2008). Of note, vulvar SKs may be less keratotic and exhibit less follicular plugging than SKs found on nonintertriginous sites (de Giorgi et al., 2005; Venkatesan, 2010). Although SKs typically can be identified macroscopically, dermoscopic evaluation can reveal characteristic keratotic invaginations, known as “comedone-like openings” and “milia-like cysts,” that reinforce the diagnosis. These clinical and dermoscopic findings are echoed by the histologic features of SKs, which include papillomatous acanthotic epithelium consisting of basaloid cells with small regular nuclei, intraepidermal horn pseudocysts, and varying degrees of hyperpigmentation and hyperkeratosis (Heller, 2015).

SKs are benign neoplasms without malignant potential and thus do not require removal or clinical monitoring. However, in certain instances, darkly pigmented SKs may be difficult to distinguish from tumors of melanocytic origin. Dermoscopy serves as a valuable tool in these cases because melanocytic lesions feature pigment networks, streaks, and globules that are not present in SKs (de Giorgi et al., 2005; Hafner and Vogt, 2008). Should diagnosis still be unclear or a suspicious change be noted, biopsy is strongly encouraged because 0.5% of clinically suspected vulvar SKs are histologically proven melanomas (Edwards, 2010; Venkatesan, 2010). Furthermore, multiple or clustered SKs are uncommon on the vulva, particularly in patients without other cutaneous SKs. Such lesions also merit biopsy to rule out pigmented condyloma acuminata, HPV-associated vulvar intraepithelial neoplasia, or other malignancies (Edwards, 2010; Venkatesan, 2010).

**Syringoma**

Initially described as lymphangiomas by Kaposi and Biesiadeki (1872), syringomas have since been recognized as adnexal in origin—specifically, as benign tumors of the eccrine sweat glands (Mahajan et al., 2012). Although most commonly seen on the face and neck of young women, vulvar syringomas have also been described on the labia majora, typically appearing as multiple 1- to 4-mm, firm, skin-colored to brownish-pink papules in a symmetric, bilateral distribution (Fig. 5; Corazza et al., 2017; Dereli et al., 2007; Hoffman et al., 2020). These lesions are usually asymptomatic and diagnosed incidentally during routine examination; however, pruritus can be a complaint, with some patients ex-
periencing exacerbations during menstruation or warmer months (Gerdsen et al., 2002; Huang et al., 2003). Syringomas often arise during puberty and enlarge during pregnancy and with the use of oral contraceptives (Mahajan et al., 2012). It has therefore been suggested that the growth of syringomas is hormonally influenced, although the literature has remained inconclusive to date (Huang et al., 2003).

Unfortunately, given the rather banal appearance of vulvar syringomas, the differential diagnosis can be broad and includesFordyce disease, EICs, sebaceous cysts, condyloma acuminata, milia, senile angiomas, lichen simplex chronicus, and lymphangioma circumscription (Table 1; Corazza et al., 2017; Mahajan et al., 2012). Thus, a biopsy with microscopic examination is often warranted (Hoffman et al., 2020). Histopathologically, the dermis features eccrine, duct-like, tubular structures lined with two layers of cuboidal epithelium embedded in a fibrous stroma (Corazza et al., 2017; Gerdsen et al., 2002; Mahajan et al., 2012).

If patients endorse significant pruritus, short courses of topical steroids and oral antihistamines may be trialed. Although topical atropine and topical tretinoin have both emerged as treatments for eruptive syringomas on the face and chest, their efficacy in vulvar syringomas remains to be seen (Hoffman et al., 2020; Huang et al., 2003). Definitive removal of the syringomas can be accomplished with surgical excision, carbon dioxide laser vaporization, cryotherapy, or electrosurgery (Huang et al., 2003).

**Hidradenoma papilliferum**

HP is a benign adenosomatous neoplasm of anogenital, mammary-like glands, first described in 1878 by Worth (Baker et al., 2013). HP is most commonly seen in postpubertal women and typically affects the labia majora and labia minora with roughly equal frequency (Fig. 6). HP is rarely found on the clitoris or in the perineal region (Hernández-Angeles et al., 2017; Scurry et al., 2009). HP presents as a firm, solitary, smooth-surfaced, red, blue, or skin-colored nodule with a well-defined capsule, ranging in size from 0.5 to 2.0 cm (Kazakov et al., 2011; Lobo et al., 2017; Scurry et al., 2009). HP is usually asymptomatic but may become ulcerated, leading to pruritus, pain, and/or bleeding (Maldonado, 2014).

Histologically, HP shows both papillary and glandular architecture with pronounced glandular hyperplasia (Baker et al., 2013). There is a complex, “labyrinthine” pattern of branching tubules and acini forming the papillae, lined by cuboidal or columnar epithelial cells with pale eosinophilic cytoplasm and surrounded by a thin myoepithelial layer (Kazakov et al., 2011). Although there is prominent glandular proliferation, the mitotic index is usually low (Lobo et al., 2017). Adjacent normal mammary-like glands are often present (Scurry et al., 2009). Due to the proclivity of HP to ulcerate, clinically differentiating HP from adenoscarcinoma may be difficult (Lobo et al., 2017). Furthermore, malignant transformation of HP has been described (Baker et al., 2013). Biopsy is therefore indicated to establish a diagnosis and rule out malignancy (Hernández-Angeles et al., 2017). The differential diagnosis should include mammary-like gland adenocarcinoma, Bartholin’s cysts, lipomas, and syringocystadenoma papilliferum (Table 1; Hernández-Angeles et al., 2017; Maldonado, 2014). Treatment of HP is complete surgical excision, and recurrence is uncommon (Maldonado, 2014).

**Cystic lesions**

**Epidermal inclusion cysts**

EICs, also referred to as epidermal cysts, epidermoid cysts, epidermoid inclusion cysts, infundibular cysts, and sebaceous cysts, are the most common cutaneous cyst. Of note, the term “sebaceous cyst” is a misnomer because EICs are derived from follicular infundibulum and do not feature sebaceous gland differentiation (Hoang et al., 2019). EICs arise when keratinizing squamous epithelium becomes trapped in the dermis, which can occur spontaneously or after trauma to the pilosebaceous unit (Heller, 2015; Nigam et al., 2017). The development of vulvar EICs is a well-described complication of female genital mutilation (Rouzi, 2010).

EICs typically present as firm, round, yellow-white papulonodules. Vulvar EICs are most often found on the labia majora, can be multicystic, and range in size from a few millimeters to several centimeters in diameter (Fig. 7; Apostolis et al., 2012; Pehlivan et al., 2015; Yang et al., 2012). In rare cases, EICs may also affect the labia minora and clitoris (Pehlivan et al., 2015). The clitoral location is most closely associated with previous female genital mutilation (Heller, 2015). Notably, epidermoid cysts may occur at any age—tiny superficial epidermoid cysts, termed milia, have even been described in neonates (Hoang et al., 2019).

EICs are lined with stratified squamous epithelium, which produces keratin and causes the cyst to fill with caseous debris (Heller, 2015). Histopathologic examination reveals a cyst lined with keratinizing stratified squamous epithelium with an intact granular layer filled with laminated keratin. If the cyst ruptures, the keratin can cause a foreign-body reaction with a dense inflammatory infiltrate featuring multinucleated giant cells and histiocytes (Cuda et al., 2019; Nigam et al., 2017). EICs are generally slow growing and asymptomatic but may become secondarily infected, inflamed, or rupture (Cuda et al., 2019; Rouzi, 2010). EICs are be-
nign and do not require removal or treatment. If symptomatic or if the patient requests removal, complete excision of the cyst lining is required to prevent recurrence and should be performed when the cyst is not inflamed (Endrizzi, 2017). Incision and drainage may be required if the cyst becomes infected, purulent, or painful (Cuda et al., 2019). For smaller cysts that become inflamed, intralesional steroids may be considered (Weir and St. Hilaire, 2020).

The differential for vulvar EICs includes Bartholin’s gland cysts, lipomas, Skene’s duct cysts, VGCs, cysts of the canal of Nuck, syringomas, and endometriomas (Table 1) (Pehlivan et al., 2015; Yang et al., 2012). There have been reports of squamous cell carcinoma, basal cell carcinoma, and other malignancies arising from EICs, including in the vulva (Delacretaz 1977; Sze et al., 2016). Malignancy should be suspected if the EIC is rapidly enlarging, recurring, or not responding to treatment. In these situations, excisional biopsy should be pursued.

**Vestibular gland cysts**

VGCs, also termed vestibular cysts or mucinous cysts, are benign cysts of the vulva. As the name suggests, VGCs are found within the vestibule of the vulva on the medial labia minora and are of minor vestibular gland origin (Maldonado, 2014). The cysts are soft, smooth, round, and range in size from 2 to 30 mm (Scurry and McGrath, 2012). VGCs can be distinguished from other vulvar cysts by their translucent nature, which is attributable to the clear, liquid mucin they contain (Fig. 8; Karakaya et al., 2018; Maldonado, 2014; Scurry and McGrath, 2012). The lining of these cysts is a simple mucous-secreting columnar epithelium, sometimes with squamous metaplasia and rarely with ciliated epithelium (Anderson, 2017; Maldonado, 2014). Hormonal involvement in the formation of VGCs has been suggested, given that onset is most frequently between puberty and the fourth decade in parous women and those exposed to contraceptives. There are also reports of cysts being strongly estrogen receptor positive (Scurry and McGrath, 2012).

VGCs are generally asymptomatic; thus, reassurance to patients can be provided. However, if there is associated pain, discomfort, or cosmetic distress, surgical excision can be performed (Karakaya et al., 2018; Maldonado, 2014). Although there is evidence of marsupialization of gland cysts in the vulvar vestibule (e.g., Bartholin’s and Skene’s gland cysts), limited data on this approach in VGCs exist (Campbell et al., 2019). As with EICs, VGCs may be mistaken for Bartholin’s gland cysts, Skene’s gland cysts, Gartner’s duct cysts, and cysts of the canal of Nuck (Table 1; Campbell et al., 2019). Biopsy can be deferred unless features suspicious for malignancy are identified (Lee et al., 2014).

**Conclusion**

Vulvar dermatology lies at the cross-section of gynecology, dermatology, and women’s health. Although traditionally underrecognized and understudied, the diagnosis and management of benign vulvar lesions lies firmly within the purview of the dermatologist’s practice. Herein, we highlighted some of the most commonly encountered anatomic variants and benign neoplasms of the vulva of which every dermatologist should be aware. We hope this manuscript serves as a framework for practicing dermatologists to help distinguish benign vulvar lesions from their pathologic mimickers.

**Declaration of Competing Interest**

None.

**Funding**

None.

**Study approval**

None.

**Supplementary materials**

For patient information on skin cancer in women, please click on Supplemental Material - Patient Page. Supplementary data associated with this article can be found, in the online version, at doi:10.1016/j.ijwd.2021.04.007.
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