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REVIEW ARTICLE



Skin diseases of the vulva: eczematous diseases and contact urticaria

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ABSTRACT

Skin diseases in the vulvar area include a variety of disorders many of which have a chronic course with significant morbidity. It is important to be aware of the symptoms, signs and diagnostic tools in order to optimise treatment. Herein, the most common eczematous diseases of the vulvar area, i.e. lichen simplex chronicus, seborrhoeic dermatitis, atopic dermatitis, irritant contact dermatitis, and non-systemic and systemic allergic contact dermatitis are reviewed. Allergic contact urticaria is also described.

KEYWORDS

Vulvar skin diseases; vulvar eczema; lichen simplex chronicus; irritant contact dermatitis; allergic contact dermatitis; systemic contact dermatitis; contact urticaria

Introduction

Eczematous diseases in the vulvar area carry significant chronicity and morbidity (Lynch et al. 2012; Bacchino-Ortiz et al. 2012). A multidisciplinary approach between several medical specialties, particularly gynaecologists, dermatologists, pathologists and urologists is highly needed to optimise diagnosis and therapy. In some countries, this has been achieved by establishing vulvar clinics (Bauer et al. 1999; Sullivan et al. 1999; Tan et al. 2000). Herein, we review the most common eczematous diseases of the vulvar area (Table 1 provides an overview). Onwards the terms eczema and dermatitis are used synonymously to describe the same type of epidermal lymphocytic inflammation with spongiosis.

Lichen simplex chronicus

Lichen simplex chronicus or neurodermatitis/hyperplastic dystrophy is a localised chronic endogenous eczema with predilection to the anogenital area and is one of the most common causes of vulvar pruritus (Lynch 2004; Ozalp et al. 2015). The aetiology is unknown but psychological stress, temperature and humidity of the area as well as contact with synthetic clothes may be aggravating factors. Vulvar lichen simplex occurs most commonly in middle to late adulthood, with the highest prevalence in women aged 30–50 years. However, the exact prevalence is unknown. The eczema initiates a vicious cycle of uncontrollable – often nightly – ‘itch-scratch-itch’ that results in thickened and coarse skin termed lichenification (Figure 1). The erythematous finely scaling lichenified vulvar plaques of lichen simplex chronicus is somewhat ill-defined merging into the normal adjacent skin in contrast to the plaques of vulvar psoriasis, which are sharply defined. The colour of the skin may also change from pink to red and purple and later brown and grey while a

thick layer of keratinocytes is found in the upper layer of the skin. There is a risk for secondary lesions like ulcers and serous secretion as well as the alopecia of the area.

The diagnosis is based on the characteristic clinical presentation, but before accepting the diagnosis it is important to rule out other causes of chronic itch, particularly candidiasis and allergic and irritant contact dermatitis. Histopathological findings are unspecific but demonstrate an acanthotic epidermis with high dermal papillae and a sparse inflammatory infiltrate.

Lichen simplex chronicus may be complicated by allergic or irritant contact dermatitis as the barrier function of the skin is lost in the lichenified area so that ingredients of emollients, rinsing remedies and topical corticosteroids can act as provoking factors (Virgili et al. 2003).

The primary treatment goal is to stop the ‘itch-scratch-itch’ cycle and explain to the patient that symptoms will not resolve as long as this cycle persists (Stewart 2010). However, although these goals are met, symptoms tend to be chronic in a significant portion of the cases. The mainstay of treatment is emollient creams as well as other topical preparations such as tar (external use) and zinc ointment. Medical treatment includes intermittent use of medium to potent topical corticosteroids, or a short course of ultrapotent corticosteroids, however, in women who are in need of continuous treatment it is our experience that tacrolimus ointment or pimecrolimus cream may be of significant benefit in order to decrease the eczema activity (Goldstein et al. 2007; Kelekci et al. 2008).

Seborrhoeic dermatitis

Seborrhoeic dermatitis is a common type of eczema affecting primarily adults aged 30–60 years with a characteristic

Table 1. Characteristics and treatment of common eczematous diseases affecting the vulva.

	Prevalence and age range	Aetiology	Clinical findings	Differential diagnoses	Treatment
Lichen simplex chronicus	>10% in 30–50 year olds	Unknown but psychological factors are involved. Itch-scratch-itch cycle. Temperature and humidity.	Thickened and coarse skin with erythematous finely scaling lichenified plaques somewhat ill-defined merging into the normal adjacent skin.	Psoriasis	Avoid scratching, plus Moderate corticosteroid cream once daily for 4 weeks; twice weekly for maintenance, or Tacrolimus ointment once or twice daily with gradual tapering to twice weekly, or Tar preparations and fat emollient creams.
Seborrhoeic dermatitis	>5% in 30–60 year olds	Malassezia furfur yeast. Flares due to psychological stress, tiredness, obesity, poor hygiene, alcoholism, Parkinson's disease and immunosuppression, including HIV infection.	A diffusely demarcated erythematous and subtly scaling rash. Characteristic distribution also on the scalp, face (eyebrows, glabella, nasolabial folds) and upper trunk.	Atopic dermatitis, Psoriasis	Mild–moderate corticosteroid cream twice weekly, or Tacrolimus ointment or pimecrolimus cream twice weekly.
Atopic dermatitis	<5% in adults, ~10–20% in children	Genetic susceptibility	Acute eczema: lively erythema, oedema and vesicles/papules. Chronic eczema: excoriations and dry scaly lichenification.	Lichen simplex chronicus, Contact dermatitis	Fat emollient creams, plus Moderate-potent corticosteroid cream once daily for 4–8 weeks; twice weekly for maintenance, or Tacrolimus ointment or pimecrolimus cream once or twice daily with gradual tapering to twice weekly. Topical or systemic antibiotics when signs of infection.
Contact dermatitis	<20% in all ages	Repeated contact with an irritant (~80% of cases) or due to sensitisation and contact allergy to a specific allergen (~20% of cases)	Signs are identical in irritant and allergic contact dermatitis. Acute eczema: pruritic and painful erythema, vesicles, oedema and erosions. Chronic eczema: dry, scaly and lichenified plaques.	Lichen simplex chronicus, Atopic dermatitis	Avoidance of irritant or allergen. Moderate-potent corticosteroid cream once daily for 4 weeks; twice weekly for maintenance, or Tacrolimus ointment once or twice daily with gradual tapering to twice weekly.
Allergic contact urticaria	Very rare	IgE-mediated allergy to natural rubber latex or human seminal plasma	Sudden swelling and redness immediately after exposure to suspected allergen (intercourse or vaginal examination).	Symptomatic dermographism and delayed pressure urticaria	Avoidance of allergen, desensitisation.



Figure 1. Lichen simplex chronicus with lichenification.

distribution on the scalp, face (eyebrows, glabella, nasolabial folds) and upper trunk, but also the intertriginous areas, including the vulva (Fischer et al. 1995; Fischer 1996; Crone et al. 2000). A special variety of vulvar seborrhoeic dermatitis may be seen in infancy.

The aetiology is unknown, but colonisation with the lipophilic yeast *Malassezia furfur* may play a role. The disease flares due to psychological stress, tiredness, obesity, poor hygiene, alcoholism, Parkinson's disease and immunosuppression, including HIV infection. Vulvar seborrhoeic dermatitis is characterised by a diffusely demarcated erythematous and subtly scaling rash (Figure 2). The diagnosis is based on the clinical presentation. Pruritus may be minimal or absent so the presence of eczema in extra-vulvar predilection sites supports the diagnosis. Histopathology shows chronic dermatitis with acanthosis, slight spongiosis, parakeratosis and a minimal lymphocytic perivascular infiltrate in the papillary dermis.

Therapy of vulvar seborrhoeic dermatitis is intermittent use of low or mediopotent topical corticosteroids



Figure 2. Seborrhoeic dermatitis.

(Fischer et al. 1995; Fischer 1996; Crone et al. 2000). It is our experience that pimecrolimus cream as well as tacrolimus ointment may also be effective and should be used in more refractory cases. A dermatological evaluation with patch testing should be considered in treatment-refractory cases in order to rule out complicating allergic contact dermatitis.

Atopic dermatitis

Atopic dermatitis is a chronic pruritic skin disease with various clinical presentations according to the age of the patient. In children, predilection sites are the flexural areas of the extremities, whereas in adults, the eczema may be confined to the hands, head and neck although the entire body can be affected. Both in children and in adult women the vulvar area may be affected (Fischer et al. 1995; Crone et al. 2000; Fischer and Rogers 2000). While the lifetime prevalence of atopic dermatitis is around 20% the prevalence of vulvar atopic dermatitis is unknown. The vulva is especially vulnerable in women with atopic dermatitis due to the combination of a deficient skin barrier function and the thin epidermis making this area prone to eczema or irritant contact dermatitis when exposed to irritants. Allergic contact dermatitis resulting, for example, from fragrances or preservatives in creams and rinsing products may complicate the symptoms.

The chief clinical manifestation is pruritus. In women with acute eczema the eczematous lesions are characterised by lively erythema, oedema and vesicles/papules, whereas chronic eczema presents with excoriations and dry scaly lichenification of the vulvar area. The diagnosis is obvious in most women due to the presence of typical atopic eczema on other parts of the body. A history of other atopic diseases such as hay fever and asthma and elevated total serum-IgE and blood eosinophilia support the diagnosis. Histopathology is rarely indicated.

Therapy of vulvar atopic dermatitis is intermittent use of mediopotent topical corticosteroids, but in more chronic cases tacrolimus ointment or pimecrolimus cream may be more appropriate due to the risk of skin atrophy during prolonged corticosteroid therapy. Oozing eczema in the vulvar area should raise suspicion of secondary infection with *Staphylococcus aureus* or streptococci and be treated with appropriate antibiotics.



Figure 3. Allergic contact dermatitis.

Contact dermatitis

Vulvar contact dermatitis is fairly common and may be due either to repeated contact with an irritant or more rarely due to sensitisation and contact allergy to a specific allergen (Brenan et al. 1996; Fischer 1996; Goldsmith et al. 1997; Lewis et al. 1997; Bauer et al. 2000). The presenting signs are identical in irritant and allergic contact dermatitis with pruritus and painful erythema, and, occasionally vesicles, oedema and erosions in the acute and subacute form, and with dry, scaly and lichenified plaques characteristic of the chronic form (Figure 3). A flare-up of the rash following a specific exposure should trigger a suspicion of allergic contact dermatitis. The exact prevalence of vulvar contact dermatitis is unknown.

Irritant contact dermatitis

Irritant contact dermatitis is the most common form of contact dermatitis and several factors have been identified that may cause vulvar eczema (Table 2). Women with known atopic dermatitis have an increased risk of developing irritant vulvar contact dermatitis (Crone et al. 2000). Intertrigo is a non-specific (irritant contact) dermatitis caused by increased moisture and friction in the intertriginous areas including the femoral folds that may spread to the vulvar area, especially affecting middle-aged and older women. Predisposing factors include obesity, sweating, poor hygiene, urine incontinence and occlusion due to diapers. Symptomatic therapy includes intermittent use of medio-potent topical corticosteroids.

Non-systemic allergic contact dermatitis

Primary vulvar allergic contact dermatitis is relatively rare, whereas women with pre-existent chronic vulvar dermatoses have a higher risk of secondary contact sensitisation due to locally applied medicaments and their constituents (Marren et al. 1992; Nardelli et al. 2004). The causes of

Table 2. Aetiological factors in vulvar irritant contact dermatitis.

Factor	Cause
Vaginitis	Persistent vaginal discharge
Urine incontinence	Postmenopausal women
Topical vehicles	Stay-on cosmetics (creams, deodorants) containing ethanol, propyleneglycol and hexylene glycol
Detergents and surfactants	Rinse-off cosmetics (soaps, toiletries, shampoos) containing sodiumlauryl sulphate
Disinfectants	Quaternary ammonium, chlorhexidine, hexachlorophene
Overcleaning	Repeated rubbing of the skin
Poor hygiene	Intertriginous debris
Wart medications	Podophyllotoxin, 5-fluorouracil, imiquimod
Systemic medications	Retinoids (acitretin, isotretinoin)
Salivation-licking	Sexual intercourse with long-standing oro-genital contact
Frictional dermatitis	Prolonged sexual intercourse, masturbation
Dermatitis factitia	Self-imposed by application of caustic or irritant substances

Table 3. Important allergens in vulvar allergic contact dermatitis.

Allergen	Relevance
Medications	
Neomycin	Topical antibacterial creams and ointments
Benzocaine	Antipruritics, analgesics
Quinolines	Antiseptics in topical medications
Ethylendiamine	Stabiliser in topical products
Corticosteroids	Topical corticosteroids, creams, suppositories
Noxonyl	Spermicidal preparation in condoms, lubricants and creams
Preservatives	
Formaldehyde	Preservatives in cosmetics, creams, lotions, soaps, shampoos and wet Quaterniumwipes
Parabenes	
Isothiazolinones	
Imidazolidinylurea	
Perfumes	
Fragrances	Cosmetics, perfumes, toilet paper, wet wipes, soaps, after shaves
Balsam of Peru	Hygiene products
Wool alcohol (lanolin)	Creams, ointments, cosmetics
Rubber	
Thiuram	Condoms, sponges, pessaries, diaphragms, catheters
Carba-mix	
Mercaptobenzothiazole	
Paraphenylenediamine	Black underwear
Vehicles and emulsifiers	
Cetyl alcohol	Vehicles used as emulsifiers and emollients in topical products
Stearyl alcohol	
Propylene glycol	
Hexylene glycol	
Sorbitan sesquioleate	
Triethanolamine stearate	
Nail varnish	
Acryl-sulphonamide	Nail lacquers
Formaldehyde resin	
Colophony	Adhesive tape, paper-based hygiene products

primary non-systemic allergic contact dermatitis are identical to those of secondary allergic contact dermatitis.

The number of allergens implicated in allergic contact dermatitis of the anogenital area is extensive and identification of the one or several relevant allergens for the individual patient requires a dermatological evaluation with patch testing after a careful history is obtained (Table 3). Histopathology is not able to separate irritant from allergic eczema. Women sensitised to perfume may react with flare-up of the vulvar eczema due to contact with scented genital hygienic products or aftershave from their sexual partner



Figure 4. Systemic allergic contact dermatitis.

leading to so-called connubial or consort vulvitis (Jensen et al. 2012). Although rare, the possibility of vulvitis elicited by systemic exposure to allergens (systemic allergic contact dermatitis) should also be borne in mind.

Systemic allergic contact dermatitis

Systemic allergic contact dermatitis is an allergic type IV reaction seen in women previously sensitised to a specific allergen, when this allergen is ingested, inhaled or injected and resulting in an eczematous reaction (Andersen et al. 1984). The hands, flexural and the anogenital areas, including the vulva, are especially high-risk sites for systemic contact dermatitis (Figure 4) (Andersen et al. 1984). Oral intake of food items with a high content of nickel, chromium and cobalt may, in rare instances, induce genital systemic contact dermatitis, and flare-up of anogenital eczema has been described by oral challenge with these metals (Veien et al. 1983, 1987). Although a significant number of women with vulvar eczema are sensitised to metals only few will benefit from a diet with restricted content of nickel (Veien et al. 1985). Food items with high nickel content able to provoke a flare of vulvar eczema include bran, oats, buckwheat, soy, legumes (dried peas and beans), chocolate, cocoa and several nuts and liquorice, although evidence is circumstantial (Veien et al. 1993). Therapy of irritant and allergic contact dermatitis is based on eliminating irritants and/or allergens. Symptomatic therapy is usually mediopotent topical corticosteroids.

Allergic contact urticaria

Most women with allergic contact urticaria of the vulva are atopic individuals with a history of atopic dermatitis, allergic rhinitis or asthma. Two main types of type I (IgE-mediated) allergic reaction have been characterised either against natural rubber latex or against human seminal plasma proteins



Figure 5. Contact urticaria.

(Lavaud et al. 1992; Hamann and Kick 1993; Nardelli et al. 2004).

Contact urticaria to natural rubber may be provoked by direct contact with latex condoms, or surgical latex gloves during a gynaecological examination or surgical procedure. Approximately 25% of women with latex glove contact urticaria and a history of condom use reported local vulvar swelling and pruritus during sexual intercourse (Turjanmaa and Reunala 1989). The amount of antigen may vary in different condom brands (Turjanmaa and Reunala 1989). The primary sensitisation may have occurred by contact with latex rubber gloves used for cleaning. The protein antigens in latex may cross-react with antigens in certain fruits: avocado, banana, celery, chestnut, fig, papaya, passion fruit and peach (Lavaud et al. 1992; Fisher 1993a). Women with contact urticaria to latex may have concomitant delayed hypersensitivity (type IV allergy) and allergic contact dermatitis to rubber chemicals.

The clinical symptoms in women with contact urticaria to rubber latex are acute itching, erythema and swelling during and after sexual intercourse if a condom is used (Turjanmaa and Reunala 1989). A similar reaction can be provoked by vaginal examination from latex gloves worn by medical staff. Latex allergic women may even develop a systemic anaphylactic reaction with generalised urticaria, angioedema, bronchospasm, abdominal pain and diarrhoea – or even respiratory or cardiac arrest (Lavaud et al. 1992).

Type I allergy with development of IgE antibodies to glycoproteins in human seminal plasma is a rare but important cause of vulvar contact urticaria (Weidinger et al. 2005). Combined contact urticaria to both human seminal plasma and latex has been described (Kint et al. 1994). Contact urticaria after exposure to human seminal plasma in a mother and her three daughters has been published (Chang 1976). The clinical presentation of contact urticaria to human seminal plasma is pruritus, a locally burning sensation, erythema and oedema within minutes after ejaculation (Figure 5) (Weidinger et al. 2005). A systemic allergic reaction has been described in women exposed to human seminal plasma with post-coital dyspnoea, dysphagia, rhinoconjunctivitis, generalised urticaria, gastrointestinal symptoms and anaphylactic shock (Chang 1976). A subgroup of women with vulvar vestibulitis have IgE antibodies to seminal fluid and may be a factor in initiation and persistence of symptoms in these women (Babula et al. 2004).

The diagnosis of vulvar contact urticaria caused by either latex or human seminal plasma should be based on history and demonstration of relevant specific IgE antibodies in serum or by skin prick test. An important differential diagnosis of allergic contact urticaria is chronic inducible urticaria, specifically symptomatic dermographism and delayed pressure urticaria with angio-oedema. These inducible urticarias develop reproducibly after repeated physical stimuli such as pressure and rubbing of the skin, and may therefore develop during or immediately after sexual intercourse or vaginal instrumentation.

Therapeutic options include allergen avoidance and desensitisation in women with contact urticaria due to human seminal plasma (Weidinger et al. 2005). Oral antihistamines taken prophylactically may be an option and have shown to reduce symptoms significantly in most affected women (Jones 1991). In women with vulvar contact urticaria due to latex all contact with this allergen should be avoided. Non-rubber male condoms made from processed sheep intestine are available for women who cannot tolerate rubber latex condoms. These condoms may prevent pregnancy whereas they lack protection against HIV infection (Fisher 1993b). Alternatively condoms made of polystyrene may be used (Fisher 1993b). A non-rubber polyurethane female condom has been approved in several countries (FDA Med Bull 1993).

Disclosure statement

No potential conflict of interest was reported by the authors.

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