**ABSTRACT**

The vulvar skin in the adult comprises about one percent of the body surface and it is a highly specialised and adaptable skin. The competency of the vulvar and vaginal skin to withstand injury and infection is a remarkable feat of humankind. However, a number of allergic conditions may affect this region of the female body. The major symptomatology of vulvar disease can be summarised as pain, pruritus, swelling, local masses and dyspareunia and most of these are common in allergic diseases in this area.

Symptoms of contact dermatitis include severe pruritus, swelling and usually a date of onset. Several causes of contact dermatitis are known, including allergy to condom contents. Atopic vulvar dermatitis is the vulvar component of systemic atopy and the vulva displays the symptoms of pruritus and burning. Allergens may produce the condition known as painful vulva syndrome, which presents as vulvodynia. The vulva is sometimes the only affected part of the skin in women with eczema.

**INTRODUCTION**

The vulvar skin in the adult comprises about one percent of the body surface covering. It is a highly specialised and adaptable skin. The competency of the vulvar and vaginal skin to withstand injury and infection is a remarkable feat of humankind. However, a number of allergic conditions may affect this region of the female body. The major symptomatology of vulvar disease can be summarised as pain, pruritus, swelling, local masses and dyspareunia and most of these are common in allergic diseases in this area.

Due to the nature of sexual contact, the vulvar skin is exposed to foreign contact including a whole host of organisms. The vulvar epithelial thickness is only 1 mm. There is no other difference between the vulva and the vagina. The vagina hosts more than 90 types of micro-organism and vaginal discharge flows distally over the vulva. The competency of the vulvar and vaginal skin to withstand injury and infection is a remarkable feat of humankind.

It is not surprising that the vulvar skin reacts immunologically and that disease patterns can be ascribed to such reactions. In this review focus will be placed on local disease where allergy is the most likely cause of disease or where allergy is a main contributor to local disease.

**TYPICAL DISEASE PATTERNS IN THIS GROUP OF DISORDERS**

The major symptomatology of vulvar disease can be listed as follows:

- Pain;
- Pruritus;
- Oedema/swelling;
- Local masses;
- Dyspareunia.

In the group under discussion, masses are rarely present.

**VULVAR CONTACT ALLERGIC DISEASE**

**CONTACT DERMATITIS**

Symptoms include severe pruritus, swelling and usually a date of onset. Several types or causes of contact dermatitis are known:

- Dermatitis medicamentosa (e.g. ‘pile’ medication);
- Chemicals present in sanitary products and perfumes;
- Cleaning products and soaps;
- Accidental chemical contact from partner.

The findings include localised redness, swelling and demarcation between normal and abnormal skin. The clinical picture is sufficient to make a diagnosis and install treatment. The most important factor in treatment is to avoid the irritant. Symptomatic treatment includes salt water sitz baths daily and application of water based emollients. A short course of medium potency topical corticosteroids may be needed.
CONDOM ALLERGY
This variant of contact dermatitis results in pruritus, slight burning and peri-introital redness in sexually active patients using condoms. Possible irritants include spermicides in or on the condoms as well as cosmetic products used on the condom itself.

The management is the same as for contact dermatitis. Patients should refrain from using cosmetically charged condoms.

ATOPIC VULVAR DERMATITIS
This is the vulvar component of systemic atopy. The vulva displays the symptoms of pruritus and burning. The skin may be oedematous and dry out and even form fissures. Local management is the same as described but the systemic management of the atopy will be important to resolve the problem.

VULVODYNIA
This very important disorder was described for women suffering from ‘Painful Vulva Syndrome’. Over the years, major aetiological classes were described:2

- Infectious - Bartholinitis, candidiasis, herpes, human papilloma virus infections, molluscum contagiosum infections;
- Trauma - Prior surgery to introitus, prior vaginal delivery, prior sexual assault;
- Systemic illness - Crohn’s disease, systemic lupus erythematosus, Behcet’s disease (vasculitis);
- Neoplasia - Vulvar intra-epithelial neoplasia;
- Allergens - Soaps, douches, antiseptics, creams or suppositories, medications including podophyline and thrichloroacetic acid;
- Dermatologic conditions - Dermatitis, eczema, lichen sclerosus, lichen planus, psoriasis;
- Urinary tract syndromes - Interstitial cystitis, painful urethra syndrome;
- Neurologic conditions - Neuralgias, referred pains, dysesthesias after herpes zoster infection or spinal problems;
- Psychological conditions - Vulvar pain syndromes, sexual abuse history.

With time, vulvodynia has found a place in the vulvar pain syndrome group and is included as such in the DSM5. To fulfill the diagnosis of vulvodynia none of the aetiological classes, as listed above, can be present. There must be symptoms and very limited signs of disease.

The typical complaints include severe vulvar pain which could be provoked (such as with intercourse or clothes or exercise), unprovoked where the pain is present all the time, or a mixed variety. The pain makes normal life impossible. It can be generalised over the whole of the vulva, or localised.3,4 The majority of patients will be in the 20-30 age group but all ages may be affected.

The typical history is of severe pain following on an event. In most cases, the event would prove to be a severe infection with Candida albicans. However, there is no single aetiology. The proposal that previous infection or allergy could lead to this pain syndrome is still a causative option.5 A dysfunctional immune response to previous candidiasis is mentioned as a potential cause.6

The clinical features include redness of the vestibule and tenderness to touch. A described entity of vestibulitis includes redness, in particular, at vestibular gland openings and also localised oedema at the vestibule.

As other diseases have to be excluded, the patient must be examined including colposcopy and cytology. If no disease can be found, vulvodynia is the remaining diagnostic disease option.

Treatment is initiated with local hygiene and sitz baths (with salt water). Initial management must include potent anti-candidal medication, preferably oral fluconazole. After 1-2 weeks of sitz baths and fluconazole, a topical medium potency corticosteroid cream is added every day for 2 months after which the patient must be reassessed and the length of treatment reconsidered.

Pain relief is almost always necessary. Analgesics can be taken orally. For severe pain amitriptyline can be added.

The expected success rate for medical management is in the order of 75%. Resistant and persistent cases should be considered for surgery. Excision of the vestibule or part of the vestibule (the “Woodruff operation”) is generally successful in symptomatic control for such patients.

ECZEMA
This disorder commonly affects the vulva where lesions are red, scaly and with a thickened skin. The vulva is sometimes the only affected part of the skin.7 Vulvar management should specifically be offered by using salt water sitz baths, water based emollients and low potency corticosteroid creams.

Care should be taken not to use topical corticosteroid creams for prolonged periods as this will lead to symptomatic thinning of the vulvar skin.

OTHER SYSTEMIC DISEASES WITH VULVAR COMPONENTS
Systemic diseases may have a presentation on the vulvar skin with or without systemic presentation at the same time. This includes Crohn’s disease, chronic bullous disease of childhood and the dermatoses. The management of the disease is systemic. Vulvar supportive therapy includes salt water sitz baths and emollients.
NEWS: CONGRATULATIONS!

Congratulations to Dr Claudia Gray who was awarded a PhD in December 2014 at the University of Cape Town’s Faculty of Health Sciences under the Department of Paediatrics for her thesis entitled “The prevalence and patterns of IgE-mediated food sensitisation and allergy in South African children with atopic dermatitis”. She also has diplomas in Allergy and Nutrition from the University of Southampton, and is a specialised paediatrician and subspecialist allergologist. Dr Gray’s interest in food allergy and eczema in children stimulated the research on which her PhD is based.

Her research is on South African children with atopic dermatitis, who are investigated for IgE-mediated food allergy. This is the first study in South Africa to utilise food challenges, component resolved diagnostics and microarray technology in food allergy diagnosis and is unique for its comparison of food allergy patterns between ethnic groups in the same geographical area. Prior to this study, it was believed that food allergy is rare in South Africa, particularly in Black African subjects, even in children with atopic dermatitis (AD) who are at higher risk for food allergy. She performed a prospective, observational study on children with AD, aged 6 months to 10 years, randomly recruited from the dermatology clinic at Red Cross Hospital. They were assessed for sensitisation and allergy by questionnaire, skin prick tests, Immuno Solid Phase Allergen Chip (ISAC) test and incremental food challenges. Sensitised patients were also tested for specific IgE by ImmunoCAP test.

The study showed a high prevalence of sensitisation and allergy equivalent to that in affluent countries. Early onset AD (< 6 months), severe eczema, and young age (< 2 years) were significant risk factors for food allergy. Egg and peanut were the most common allergens. Ethnic differences were evident with Black Africans demonstrating lower allergy rates despite similar sensitisation rates, indicating that other factors are protective against manifesting with food allergy in this subgroup. This data has been published in the prestigious journal “Paediatric Allergy and Immunology”. (Gray CL, Levin ME, Zar HJ, Potter PC, Khumalo NP, Volkwyn L, Fenemore B, du Toit G. Food allergy in South African children with atopic dermatitis. Pediatric Allergy and Immunology 2014;25(6):572-9).

The thesis also describes component patterns and predictive values of tests in food allergy diagnosis. Widely used 95% positive predictive values for peanut, egg and cow’s milk allergy performed poorly, particularly in Xhosa patients. Skin tests were found to be an excellent test in both ethnic groups for diagnosing peanut allergy, and SPT to raw egg white and fresh milk were the superior test in egg and milk allergy diagnosis. The data on peanut diagnosis has also been submitted to an international journal. (Gray CL, Levin ME, Zar HJ, Potter PC, Khumalo NP, Volkwyn L, Fenemore B, du Toit G. Ethnic differences in peanut allergy patterns in South African children with eczema. Submitted). It suggests new values for more accurate interpretation of laboratory tests for food allergy diagnosis in this population that differ according to ethnicity. The findings in Dr Gray’s research thus have practical implication in allergy diagnosis, indicate the possible rise in food allergy in South Africa, which could become a significant public health burden and have inspired further research into food allergy in an unselected African cohort.

The thesis was supervised by Mike Levin and co-supervised by George du Toit.

Dr Gray’s research was supported by an ALLSA Aspen/GSK Research Award which was awarded to Dr Gray at the ALLSA Congress in Durban in August 2014.