Patients attending a vulval clinic in a genitourinary medicine department

The first vulval clinic was established in the USA in the 1960s.[1] Few data have been published regarding diagnoses or efficiency of vulval clinics especially in the United Kingdom. A vulval clinic at the Genitourinary Medicine (GUM) Department of the City Hospital, Nottingham, has been conducted jointly by a GUM consultant and a consultant dermatologist since July 1991. Data of 61 consecutive patients seen at the vulval clinic between July 1991 and January 1994 were retrieved and analysed. Patients showing subclinical human papilloma virus (HPV) infection without vulval intraepithelial neoplasia (VIN) on biopsy and having symptoms of vulval pain were grouped into the diagnosis of vulvodynia.

The mean age of the patients was 32·1 years. The mean duration of symptoms at first presentation to the vulval clinic was 28·9 months. The mean duration of previous attendance at regular GUM clinics was 24·3 months. Twenty nine patients (47%) had a history of previous lower genital infection.

A total number of 66 diagnoses were made at the vulval clinic. These are summarised in the table. In 48 patients (83%) a previous diagnosis was changed. Four patients were excluded from evaluation of a possible change in diagnosis. Eight patients could not be assessed regarding disease outcome. A complete resolution of symptoms was seen in eight out of 53 patients (15%), a partial resolution in 55% (29/53) and in 16 out of 53 patients (30%) there was no change in disease severity. The table shows the number of patients who were successfully or unsuccessfully treated in the various diagnostic categories.

Treatment plans for vulval vestibulitis syndrome (VVS) at our vulval clinic include topical anaesthetics or topical emollients, followed by topical anti-inflammatory drugs. The next step is the use of topical corticosteroids and then the administration of low dose amitriptyline. Our treatment regimen for vulvodynia comprises topical emollients, followed by topical corticosteroids if necessary. Eventually, low dose amitriptyline might be needed. Treatment of vulval dermatoses consists of avoidance of irritants and allergens and the use of topical emollients and topical corticosteroids. Therapeutic regimens were changed in 44 out of 53 patients (83%).

Diagnoses made at the vulval clinic and therapeutic outcome*

	Diagnoses made		*Therapeutic outcome*	
Diagnosis	*No.*	*(%)*	*improved*	*not improved*
CID/CAD	13	20	9	1
VVS	13	20	9	2
Vulvodynia	9	14	5	4
PFF	6	9	2	4
Recurrent VVC	5	7	5	0
VIN	4	6	2	2
Psoriasis	4	6	3	1
AD/LSX	4	6	3	0
Lichen sclerosus	2	3	2	0
Folliculitis/SC	2	3	1	0
Behcet's disease	1	1.5	0	1
Nerve injury	1	1.5	0	1
Tinea cruris	1	1.5	1	0
Atrophic vulvitis	1	1.5	0	0

*multiple diagnoses were made in some patients.
CID/CAD = contact irritant or contact allergic dermatitis; VVS = vulval vestibulitis syndrome; PFF = posterior fourchette fissure; VVC = vulvovaginal candidiasis; AD/LSX = atopic dermatitis or lichen simplex; Folliculitis/SC = folliculitis or sebaceous cyst.

Forty out of 53 patients (75%) were seen at the vulval clinic only once and 13 patients (25%) had one or more follow-up appointments at the vulval clinic. Twenty seven patients (44%) were referred to other specialties.

The distribution of diagnoses in our study, with nearly 80% of diagnoses being either dermatological conditions or vulval pain syndromes (VVS or vulvodynia) is different from that of earlier reports which showed a higher incidence of lower genital infection.[1-3] This is probably because our patients have already been treated for STDs and had already attended a GUM clinic for an average of two years.

Our data show that a majority of patients had their diagnoses revised in our vulval clinic and required a change in treatment strategy. As a result symptomatic improvement or resolution was seen in a majority of patients. This treatment outcome is particularly encouraging given that patients with vulval disease present with problems that are very difficult to treat.[4]

In view of the good clinical response in our patients and of the high rate of changes in diagnosis in our vulval clinic we would advocate the use of vulval clinics, provided such services are adequately resourced. Nearly half of our vulval clinic patients were referred to other specialties. This emphasises the necessity of a multidisciplinary approach to vulval disease, with co-operation between gynaecologists, dermatologists and GUM physicians.[5]

K A WOLPERT
K E ROGSTAD
I H AHMED
Department of Genitourinary Medicine, City Hospital, Nottingham
K L DALZIEL
Dermatology Department, Queen's Medical Centre, Nottingham

Address correspondence to: Dr K A Wolpert

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Vaginal colonisation by *Candida lipolytica*

We wish to report a case of a 25 year old woman who attended the dermatology outpatient clinic of our hospital in order to be screened for sexually transmitted diseases.

At the initial visit in April 1994, she complained of excessive vaginal discharge. On questioning she also gave a past history suggestive of recurrent genital herpes. At the time,

cervical smear for gonococci was negative. Smears for *Trichomonas vaginalis* and *candida* were also negative. Cervical pap smear showed cervicitis with squamous metaplasia and was negative for malignant cells. She was not investigated for chlamydial infection as facilities were not available at the time. However, a previous study of antenatal women in our hospital showed a low prevalence of chlamydial infection.[1] ELISA for HIV and VDRL were non reactive. She was not given any specific treatment and her symptoms resolved. At her next visit 5 months later, she was asymptomatic. On examination she was found to have minimal mucoid vaginal discharge. The KOH preparation showed yeast like forms with an unusual morphology. Hence culture on Sabouraud's dextrose agar was done which showed a pure growth of a yeast like organism. This was identified to be *Candida lipolytica*, based on morphology, growth characteristics, sugar fermentation and assimilation tests.[2] Repeat smears for *Trichomonas vaginalis* and gonococci were negative. Her husband complained of dysuria, although his urine microscopy did not reveal any abnormality.

C lipolytica has been previously isolated from respiratory including bronchial secretions, urine and cases of oral and oesophageal candidiasis.[3 4] It has also been implicated in ocular candidiasis, persistent fungaemia with catheter associated *candida* thrombophlebitis, polymicrobial sinusitis, and tissue colonisation.[5 6] However, colonisation of the female genital tract by this species has not been previously reported.

Based on a murine model of disseminated candidiasis caused by this species, it has been suggested that *C lipolytica* is a weakly virulent pathogen.[6] This may explain the fact that this patient, who was otherwise healthy, was asymptomatic. However since her husband gave a history of extramarital sexual contacts, she may be at risk of acquiring HIV infection. If she subsequently becomes immunocompromised, the organism may assume the role of an opportunistic pathogen.

Our patient was not given any specific treatment since she was asymptomatic. She was advised to return if she developed any symptoms. However, she has not come to our clinic since then.

B RAJAGOPALAN
Department of Dermatology
M S MATHEWS
Department of Microbiology
M JACOB
Department of Dermatology
Christian Medical College Hospital
Vellore 632 004, India

Address correspondence to: Dr M Jacob

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