



## Janssen Pharmaceutical Ltd

Meeting 15 November 1976

### Trends in Candidal Vaginitis

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#### Candidal Vaginitis

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It is astonishing that a disease so easily recognized, so prevalent and so distressing should have evoked such scant comment in medical writings and that it should still prove sometimes intractable to treatment. In this, however, it but shares in the general neglect afforded to fungal diseases. Ajello (1975) has commented that the true incidence and prevalence of mycotic diseases remains unknown and, in consequence, their socioeconomic impact cannot be quantified. All the cutaneous mycoses are common, constituting a real public health hazard and proving costly in terms of medical expertise and of money spent on treatment that is often ineffective.

Candidal vaginitis has been recognized at least since 1792, when Frank observed it developing coevally with aphthæ of the mouth. Its relationship to a fungus was described by Wilkinson (1849) who believed, with Vogel, that epiphytes were not capable of establishing themselves on epithelial surfaces save when 'by peculiar relations, a favourable soil has become prepared for them'. The illustrations accompanying the paper show a dimorphic fungus, with budding yeast forms. Haussman (1875) showed that fungus spores occurred in the mouths of babies whose mothers had vaginal thrush and he demonstrated that the same fungus, *Oidium albicans* (Robin), caused disease in both. Further, he produced the disease in a healthy pregnant woman by inoculating her with material from an infected patient.

Despite this direct demonstration of the noxious effect of the fungus, later to be known as *Candida albicans*, the considerable writings that followed Wilkinson's report had abated by the turn of the



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century, subsiding on a wave of scepticism based on the doubt of the pathogenicity of yeasts. Since species of yeasts, most of which are innocuous, were not always differentiated, the belief in their essential commensalism in the vagina grew until it included the principal pathogen of the genus, *Candida albicans*, and it persisted despite the statement of Castellani & Taylor (1925) that monilia (*C. albicans*) is found neither in smears nor in cultures of the normal vaginal secretion.

Mycotic vulvovaginitis was virtually rediscovered by Plass *et al.* (1931) who had vainly sought *Neisseria gonorrhoeæ* in cultures from two women with severe vulvovaginitis of pregnancy, while consistently demonstrating the blastospores and pseudomycelium of a dimorphic fungus. In their day, candidal vaginitis was regarded as uncommon; presumably, most cases would have been regarded erroneously as gonorrhoea. Only

recently has the specific identification of *C. albicans* become reliable in the laboratories of those who are not committed mycologists, better than 95% agreement being reached by those in the United Kingdom that participate in quality control exercises. This is due, in no small measure, to the germ-tube test, introduced in Taschdjian *et al.* (1960), a test of rarely matched specificity.

Carroll *et al.* (1973) showed that the isolation of *C. albicans* coincided with vaginitis, as judged by direct examination of the vagina, in 84% of cases, and that in 14% it correlated with other signs or symptoms of morbidity of the genital tract. They concluded that *C. albicans* is not part of the normal flora and that its presence indicates morbidity. Since *C. albicans* is often part of the bowel flora, it may occasionally be isolated from the vagina, particularly if the clinical specimen is badly taken.

Other pathogenic species of *Candida* may, rarely, cause vaginitis, as can the closely related yeast *Torulopsis glabrata*. Hurley *et al.* (1973) described the incidence and distribution of these species in the vagina, and their pathogenicity was reviewed by Hurley (1967). None is important in candidal vaginitis, and only *Torulopsis glabrata* is isolated at all frequently. In the five-year period 1966–1970, at Queen Charlotte's Hospital, London, yeasts were isolated from 1538 of 6629 vaginal swabs sent for diagnosis of vaginitis. *C. albicans* comprised 94% of all isolates of yeasts from women with mycotic vulvovaginitis, and *T. glabrata* accounted for 3.5%.

Candidal vaginitis is the most frequent of all infections in maternity practice, and prospective study shows that it afflicts 16–17% of pregnant women. Pregnancy is known to predispose to the disease, but it occurs in women of all ages, has been described in virgins (Mettenheimer 1880), and is not infrequent after the menopause (Davis 1929). It is often exacerbated about the time of menstruation, and growth of candida is more marked at the end of the menstrual cycle (Vaysiere *et al.* 1958). For centuries, vaginitis and vaginal discharge were thought to result from marriage and childbirth, and pregnancy vaginitis seems to be the initiating event in many cases of inveterate vaginal thrush (Hurley 1975).

Treatment during pregnancy is often neglected, or stunted, and is by no means so successful as the claims of manufacturers or the earlier literature indicated. A retrospective survey of some 500 women treated for pregnancy thrush showed that 45% had had more than one course of treatment during pregnancy, and one luckless woman had had nine courses. This does not tally with the

85–95% cure rates claimed for most specific agents by investigators conducting clinical trials, although some workers (Nathanson 1960) have stressed that up to three courses of treatment may be necessary in a few cases. The disease may be extremely persistent, lasting for more than twenty years (Brunsting 1950).

Although some of the factors predisposing to candidal vaginitis (for example, diabetes and pregnancy) are known, it cannot be said that the flaw in the biological system that allows implantation of the fungus has been thoroughly elucidated; nor is it known whether the defect in the host is acquired, or congenital. *Candida albicans* is a frequent commensal of man and can be isolated from various sites, but, principally, from the gastrointestinal tract (Winner & Hurley 1964). There is no evidence of any marked strain variation in virulence, and humoral antibody does not protect. Candidal vaginitis is but one aspect of a relationship between host and fungus that spans all degrees of interaction, from harmless carriage through chronic local granulomatous disease to a disseminated deep-seated rapidly fatal mycosis.

This symposium is designed to consider the pathological mechanisms whereby candidal vaginitis is brought about, and also to consider ways in which the treatment of this common superficial mycosis can be improved.

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