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Chronic paronychia, and oral and vaginal candidal carriage

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Introduction

In a previous study (1, 2) a higher incidence of *Candida albicans* was found in the mouth in individuals with recurrent candidal vaginitis than in persons without vaginal pathology. Concomitant infection of the skin — *erosio interdigitalis*, chronic paronychia, and intertriginous *Candida* infection was however significantly low. As an extension of the previous study we re-evaluated the incidence of *Candida* carriage in the mouth and in the vagina in cases of paronychia chronica. Conflicting opinions about this aspect of the problem have been published. WHITTLE et al. (3) found *C. albicans* in the vagina in 1 out of 18 patients. MARTEN (4) found *C. albicans* in the vagina in 5 out of 20 patients, in the rectum in 9 out of 18 patients and in the oral cavity in 13 of 17 patients. FORMAN (5) on the other hand found that in 63 female patients with chronic paronychia, 50 were associated with vaginitis, vulvitis or angular cheilitis. Since oral carriage of *Candida* in one series of hospitalized patients was as high as 58 % (*C. albicans* 54 %) (6) we decided to include a control group as was done in our study of candidal vaginitis.

Materials and Methods

Sixty patients with chronic paronychia were examined. The age of the patients varied from 18 to 65 years. From each of these patients material was taken from the paronychia, the mouth and the vagina. The material from the paronychia was taken by inserting a sterile paper-clip under the affected nail. The smears from mouth and vagina were taken with a sterile cottonwool swab. The material was inoculated immediately onto taurocholate agar and glucose peptone agar. *C. albicans* was identified as described previously (1). In all patients a careful history was taken with regard to predisposing factors: pregnancy, hormonal disturbances and systemic therapy with broad spectrum antibiotics, corticosteroids, the use of oral or vaginal metronidazol and contraceptive pills. For the evaluation of the effect of the drugs on candidal carriage, only those patients who had received a particular drug less than 3 months before examination were regarded as having been treated with it. Fasting blood sugar was examined routinely in all cases, and a glucose tolerance curve was done when indicated.

Sixty female patients with various skin diseases served as controls. These patients were chosen randomly by selecting the first patient to attend the dermatological out patient clinic who was in the same age group as the preceding paronychia patient. In these control patients the following diagnoses were made: tinea, eczema, pityriasis versicolor, pruritus, psoriasis, seborrheic dermatitis, defluvium capillorum, furuncle, solar keratosis, clavus, callositas, combustio, hidradenitis, herpes zoster, lichen amyloidosis, syringoma, discoid lupus erythematosus and xanthelasma. A history was taken from these patients, a fasting blood sugar was done and swabs were taken from the mouths, in the same way as from the paronychia patients. Vaginal smears were not taken from the control patients because of technical difficulties.

Results

Clinical candidosis of the angles of the mouth was found in one, of the vagina in two, and of the mouth in none of the paronychia patients.

Figure 1 shows the incidence of *C. albicans* and other *Candida* species in the nail fold, the mouth and the vagina in patients with chronic paronychia.

C. albicans was cultured from the paronychia in 50% and another *Candida* species in 15% of the cases. From the mouth *C. albicans* was cultured in 67% and another *Candida* species in 12%, while from the vagina *C. albicans* was cultured in 12% and another *Candida* species in 2% of the cases.

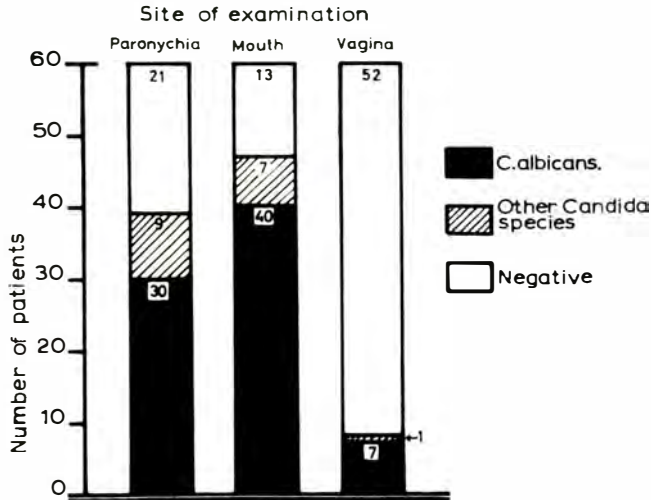


Figure 1. Incidence of *C. albicans* and other *Candida* species in different sites in patient with chronic paronychia

Figure 2 shows the incidence of *Candida* species in the oral cavity of patients with paronychia, and controls. The difference between the incidence of *C. albicans* in the two groups is statistically significant ($p < 0.01$).

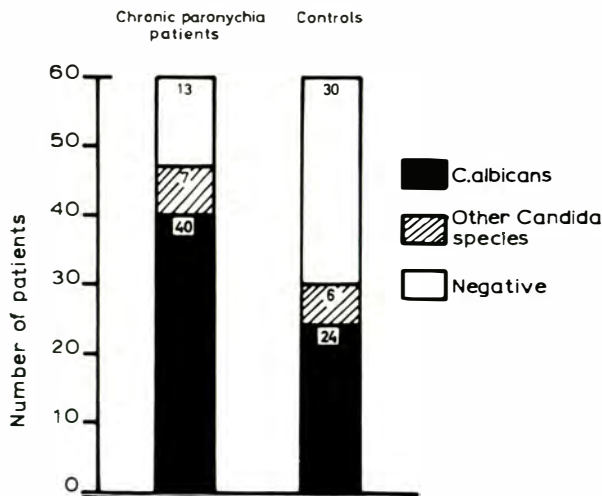


Figure 2. Incidence of *Candida* in the mouths of paronychia and control patients

Figure 3 shows that there is no significant difference in the carriage rate of individuals with and those without a predisposing factor. Nor is the difference in incidence of predisposing factors in the patient and in the control group significant

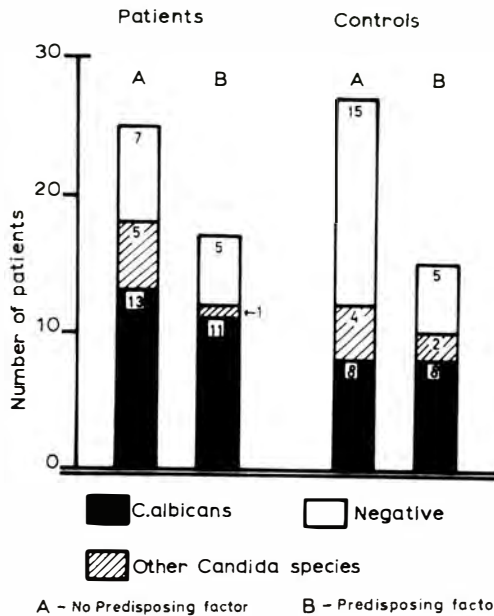


Figure 3. Incidence of *Candida* in the mouths of patients and controls with and without a known predisposing factor

Table 1: Incidence of predisposing factors in 42 patients with chronic paronychia and 42 controls

	Patients	Controls
diabetes mellitus	7	3
corticosteroid treatment	2	1
broad spectrum antibiotics (chloramphenicol, tetracyclin, ampicillin)	7	6
recurrent candidal vaginitis	1	1
oral contraceptives	0	2
Total	17	13

Table 1 shows the predisposing factors which were present in the paronychia and in the control groups (Immersion in water is not mentioned as virtually all patients and controls stated that they did so frequently).

Diabetes mellitus was the only predisposing factor which was more often present in the paronychia patients than in controls. In fact three of the cases of diabetes mellitus in the paronychia patients were detected as a result of the routine blood glucose examination.

Table 2: Other diseases, and drugs taken by 42 patients with paronychia and 42 controls

	Patients	Controls
hypertension	2	5
anemia	2	1
rheumatic fever	0	1
ascaris infestation	0	1
amoeba infestation	0	1
use of erythromycin	0	1
use of meproamate	4	0
use of valium	0	1
antipyretics (more than once a month)	2	1

In **table 2** are listed other diseases and drugs taken by the paronychia patients and the controls. The only drug more frequently used by paronychia patients was meproamate.

No acrocyanosis, Raynaud's disease or phenomenon (except perhaps one patient who complained about white hands in winter) were seen. Local changes of the skin, as in housewife dermatitis, were frequently seen both in paronychia patients and controls, but the incidence was not recorded. Other local factors possibly of influence (3) were also disregarded.

Table 3: Incidence of Candida cultured from different sites in paronychia patients in fertile period and menopause

	paronychia		mouth		vagina	
	fertile period	meno-pause	fertile period	meno-pause	fertile period	meno-pause
<i>C. albicans</i>	17	13	20	20	7	0
Other <i>Candida</i> species	6	3	7	0	0	1
Negative	14	7	10	3	30	22
Total	37	23	37	23	37	23

Table 3 shows the incidence of *Candida* species in paronychia, mouth and vagina in patients in the fertile period and in the menopause. There was no difference between the carriage rate of *C. albicans* in the mouths of patients in the fertile age and those in the menopause. In the vagina, however, *C. albicans* was found only in patients in the fertile period.

Table 4: Age incidence of chronic paronychia

Age:	under 21	21—30	31—40	41—50	51—60	61—70	Total
number of patients	1	11	19	6	21	2	60

The age incidence of chronic paronychia in this study is shown in **table. 4**. A lower incidence of cases in the 41—50 years age group is obvious.

Discussion

The incidence of vaginal *Candida albicans* in patients with chronic paronychia (12 %) was within the normal limits as quoted in the literature (7) and was similar to those found in the series of WHITTLE et al. (3) and of MARTEN (4). In patients in the fertile period vaginal *C. albicans* was found in 7 out of 37 patients, which is still within normal limits. It seems, therefore, that, in contrast to the findings of FORMAN (5), vaginal carriage of *C. albicans* and of other *Candida* species, is of limited importance in the epidemiology of chronic paronychia. Similarly, chronic paronychia does not appear to be of epidemiological importance in recurrent candidal vaginitis. In our previous study of candidal vaginitis (1, 2) and in the present study we found only three out of 92 patients who had both chronic paronychia and candidal vaginitis. It seems that local factors, such as constant wetting of the fingers in patients with paronychia, play some role in the clinical appearance of the candidosis. The role of local factors may also be seen experimentally in the ability to infect mice vagina only when the epithelium is completely cornified and polymorphonuclear cells are absent in vaginal smears (8). Biochemical changes may also be of importance and may be associated either with the epithelium (8) or with the cervical or vaginal secretions. Our finding that the damaged uterine cervix may harbour *C. albicans* (9) may support this hypothesis. Another possible factor is the inhibitory influence of the prostatic fluid of the patients partner (10). There may be hormonal factors which are disturbed in pregnancy and by the use of contraceptive pills.

The incidence of *Candida* species in the mouth in chronic paronychia is in agreement with the findings of MARTEN (4) and is comparable to the incidence found in recurrent candidal vaginitis (1, 2). Predisposing factors do not seem to be a significant factor in the higher incidence of oral carriage in patients with chronic paronychia. Thus, it may well be that oral candidal carriage plays a role as the source of chronic paronychia in a way similar to that suggested for recurrent candidal vaginitis (1, 2). On the other hand it may be that the higher incidence of *C. albicans* in the mouth is a result of the clinical infection, or that both mechanisms are present. In contrast to the experience of FORMAN (5) clinical candidosis of the mouth angles was found only once in the present series.

The incidence of *Candida* species in the paronychia was lower than in the studies of MARTEN (4) and of STONE and MULLINS (11). We assume that our techniques was not as good as theirs, but this does not explain the higher incidence of other *Candida* species, which were present in both the mouth and the paronychia in 5 instances, in the paronychia only in 4, and in the mouth only in two instances. As the formation of pseudo-germ tubes in human serum is a very sensitive test for the detection of *C. albicans* (12, 13, 14), it may be assumed that these patients did indeed have other *Candida* species. Unfortunately, because of the work-load in the routine laboratory, sugar fermentation reactions could not be performed and so no further differentiation was possible. However, other *Candida* species were also found in chronic paronychia by RAUBITSCHKE (15) and by WHITTLE et al. (3). Although paronychia formation in humans has been produced experimentally by *C. albicans* endotoxin only (11), other *Candida* species could have properties similar to *C. albicans*, as has been proven in animal experiments (16). Alternatively, the hypothesis of RAUBITSCHKE (15) that they are only secondary invaders may be true.

In contrast to the findings of FRAIN-BELL (17) and of WHITTLE et al. (3) fewer cases were present in the fifth decade. This may be coincidental.

Whether the immediate contacts of the patient, especially her close family constitute a source of reinfection remains to be established.

It may be concluded from this study that vaginal candidal carriage has no influence on the epidemiology of chronic paronychia whereas oral candidal carriage may be an important source of either primary infestation or reinfection.

Summary

In 60 patients with chronic paronychia, *Candida* was found in the paronychia in 39 patients (*C. albicans* in 30 of them), in the vagina in 8 patients (*C. albicans* in 7 of them) and in the mouths in 47 patients (*C. albicans* in 40 of them). In the mouths of a control group of 60 female patients of comparable age, *Candida* was found in only 30 (*C. albicans* in 24). The incidence of *Candida* in the mouth was not affected by the presence of "predisposing factors" nor was there any difference in candidal carriage rate in paronychia and mouths of subjects in the fertile period and those in the menopause. *C. albicans* was present in the vagina in the fertile period only.

The incidence of chronic paronychia was less in the 5th decade than in either the 4th or 6th decades.

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