

A clinicomicrobiological study of chronic paronychia

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Abstract

Accumulating evidence suggests that chronic paronychia is an eczematous condition with multifactorial aetiology and questions the role of *Candida* as a pathogen. The objectives were to assess the clinical pattern, predisposing factors, and microbial status of chronic paronychia and to compare the efficacy of topical steroids, antiseptics, and antifungals as treatment.

Clinical pattern was assessed and swabs were taken from the gap between the nail fold and cuticle of the most severely affected and normal fingers for microbial studies. The sample was randomly divided into two groups. One group was treated according to the culture with Povidone iodine ointment for bacteria, and with Miconazole tincture for *Candida*. The other group was given topical 0.1% Betamethasone Valerate ointment irrespective of the culture. Patients were reassessed after six weeks.

Of 75 patients the male-female ratio was 1:2 and 38% were house wives. Single hand was affected in 56.66%, which was the dominant in 84%. Water and soap were the commonest predisposing factor (64.4%). The commonest association was eczema (29%). Coliforms were predominantly isolated (83.6%) and *Candida* was isolated in 51%. Steroids indicated a statistically significant improvement compared to the culture specific treatment ($p < 0.001$). For *Candida* positive cases treatment with steroids showed better outcome than antifungals but this difference was not statistically significant ($p > 0.05$).

Our results favour that chronic paronychia is an inflammatory disorder caused by environmental hazards. Topical steroids are an effective treatment option irrespective of the microbial status.

Background

Chronic paronychia is a disease characterized by swelling and erythema of the proximal and lateral nail folds¹. The cuticle is damaged by chemical or mechanical trauma leading to barrier impairment. The damaged cuticle creates a space between the nail fold and nail plate where irritants, allergens, and micro-organisms can penetrate¹. All these factors contribute to the inflammation of the nail fold.

Candida has been isolated in the proximal nail fold in a variable percentage in different studies^{2, 3, 4}. Although it is commonly identified its pathogenic role is now debated⁵. Accumulating evidence suggests it is

an eczematous condition with multifactorial aetiology^{5, 6, 7}. The role of *Candida* could be an opportunistic pathogen rather than the primary etiological agent.

Treatment of chronic paronychia is often disappointing. Most of the patients undergo a chronic disabling course¹. A clear idea of pathogenesis will help in elimination of the predisposing factors and identifying the treatment modalities. Different clinicians use different modes of treatment ranging from topical antibacterials, antiseptics, antifungals, and steroids to systemic antifungals.

Method

The study was conducted at the Department of Dermatology at National Hospital of Sri Lanka during a period of 6 months. Ethical approval was granted from the Ethical Clearance Committee of the National Hospital of Sri Lanka.

The objectives of the study were,

1. To assess the clinical pattern, predisposing factors, and microbial status of chronic paronychia.
2. To compare the efficacy of topical steroids, antiseptics, and antifungals in the treatment of chronic paronychia.

All patients with clinical evidence of chronic paronychia in the fingers were included. Informed consent was taken from the participants.

Acute or chronic paronychia (point tenderness or discharge of pus), active hand dermatitis and known allergy to Povidone iodine were criteria for exclusion.

Data were collected using an interviewer administered questionnaire.

Clinical severity was assessed by grading the inflammation as mild, moderate and severe by the first author. Additionally digital photographs were taken for comparison.

Hand washing using tap water and soap was done before microbial studies. Sterile saline soaked swabs were taken from the space between the proximal nail fold and cuticle of the most severely affected finger and a clinically normal finger as the control. Gram stain and culture in blood agar were done for both samples. Advice on hand care was given to all patients and the sample was randomly divided into two groups for treatment.

1. 0.1% Betamethesone Valerate ointment irrespective of microbial status.
2. According to the possible pathogen isolated.
Bacteria – Povidone iodine ointment.
Candida – Miconazole tincture.

All applications were used twice a day. The treatment was prescribed by the co-author. Response to treatment was assessed by the first author after six weeks.

The outcome was defined as:

Cured – normal cuticle with no swelling of the nail fold.

Complete improvement – no swelling with damaged cuticle.

Partial improvement – reduction in swelling.

Stable – no reduction in swelling.

Worsened – increased swelling.

The statistical significance of the response was assessed by the Chi Square test on selected co-factors.

Results

Study sample consists of 75 patients. 59% were females.

Most (38%) were of 51-60 years. 38% were house wives, 18% were cooks, 14% had an occupation with exposure to a chemical and 10% were in the medical field. Duration of symptoms ranged from 1 month to 10 years with a mean of 2.7 years. Single hand was affected in 56.66% and it was the dominant in 84%. In 45.33% only one finger was affected and this was the thumb in majority. 37 (50%) had other associations, out of which atopy was the commonest (25.33%). 65% identified soap and water as a predisposing factor. The use of steel wool for dish washing in 36.36% of the sample is an interesting feature which we thought could have lead to the mechanical damage of the cuticle.

When considering the bacteriological studies gram stain did not reveal pus cells in any. But all were culture positive with organisms as Staphylococcus, Coliforms, Pseudomonas and Proteus. Only 4 (5.33%) had a growth of a single organism. Similar range of organism was found in the diseased as well as the control. In the diseased sample Coliforms were the most frequently (83.6%) isolated whereas it was Staphylococcus in the control. Candida was isolated in 51% of the diseased and in none of the control fingers.

70% of participants (with 130 affected fingers) turned up for the follow up 6 weeks after treatment. The response to treatment on individual fingers is illustrated in Table 1.

The improvement is statistically significant ($p < 0.001$) in topical steroid group compared to the culture specific treatment groups.

The outcome of the study sample according to the culture positivity of Candida is illustrated in Table 2.

Table 1. Response to different treatment modalities

<i>Treatment</i>	<i>Stable</i>	<i>Partial improvement</i>	<i>Complete improvement</i>	<i>Total</i>
Steroids	19 (20.0%)	38 (40.0%)	38 (40.0%)	95 (100.0%)
Povidone iodine	12 (60.0%)	4 (20.0%)	4 (20.0%)	20 (100.0%)
Micanazole	35 (54.7%)	20 (31.2%)	9 (14.1%)	64 (100.0%)
Total	36 (27.7%)	47 (36.2%)	47 (36.2%)	130 (100.0%)

Table 2. The outcome of the study sample according to the culture positivity of *Candida*

<i>Culture Status</i>	<i>Treatment</i>	<i>Stable improvement</i>	<i>Partial improvement</i>	<i>Complete</i>	<i>Total</i>
Culture negative	Steroids	6 (11.1)	21 (38.9)	27 (50.0)	54 (100.0)
	Povidone iodine	12 (60.0)	4 (20.0)	4 (20.0)	20 (100.0)
Culture positive	Steroids	13 (31.7)	17 (41.5)	11 (26.8)	41 (100.0)
	Miconazole	35 (54.7)	20 (31.2)	9 (14.1)	64 (100.0)

For *Candida* negative cases treated with steroids show a very significant improvement ($p < 0.001$). For culture positive cases the improvement with steroids is better than with antifungals but this difference is not statistically significant ($p > 0.05$).

Discussion

In our study male-female ratio was 0.7:1. However, the incidence of males in our series was comparatively higher (41%) than other studies^{2,3,4}. Men with occupations with water and chemical contact may contribute to this change.

The commonest association was atopy (25%) and many identified soap and water as predisposing factors. This favours the current concept of paronychia to be an eczematous condition aggravated by exposure to irritants or allergens^{6,7}.

Coliforms was the predominant organism found (83.6%) in the diseased finger and *Staphylococcus* was seen in the majority of the control sample. This microbiological findings have been reported by some^{1,4} while others reported *Staphylococcus* as the predominant organism^{2,3}. Coliforms are also found in normal intestinal flora which would have colonized the nail fold by contamination with stools. This would be possible when considered the toilet habits of the local community. We believe that bacteria are most likely to be colonizers. Absence of pus cells in gram stain, presence of multiple growths, presence of similar bacterial spectrum in the clinically normal control finger and poor response to Povidone iodine treatment are all in keeping with this concept.

80% showed a clinical (complete/partial) improvement in the steroid treated group which was statistically significant (Table 1). In an Italian study at 9-week follow up, clinical improvement occurred in

about half of the nails treated with oral antifungal agents, compared with 85% of the nails treated with the topical corticosteroid⁸.

Candida was isolated in 51% in our study. *Candida* has been isolated in the proximal nail fold in 40%, 64%, and 97% of patients with chronic paronychia in different studies^{2,3,4}. Although *Candida* commonly inhabits the nail fold in chronic paronychia whether it is the primary etiological agent is a matter of debate. In our study when *Candida* was positive, 68% showed a partial/complete improvement in the steroid group compared to 45% in miconazole group. Although this difference was statistically not significant, this raises the possibility of *Candida* acting as a colonizer or causing a hypersensitivity reaction contributing to the inflammation and not the primary infection. Nevertheless a long term follow up to detect recurrences are needed.

Conclusion

Our results favour the possibility of chronic paronychia being an inflammatory disorder caused by environmental hazards and that the role of microbial agents is one of colonization. Topical steroids can be regarded as an effective mode of treatment irrespective of the bacteriological and mycological status.

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