

# Dermatitis cruris pustulosa et atrophicans

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## ABSTRACT

Dermatitis cruris pustulosa et atrophicans (DCPA) is a distinctive type of chronic superficial folliculitis, primarily affecting the lower limbs. It is characterized by symmetrical follicular pustules of both legs, with cutaneous edema, resulting in alopecia, atrophy and scarring. It was first described by Clarke, from West Nigeria, in 1952 and well illustrated in his book "Skin diseases in the African," under the initial label of "Nigerian shin disease." Subsequently, it was described in India as well, in 1964, and continues to be a problem in dermatology clinics across the country. It is predominantly a disease of men and has a high prevalence in some geographical regions; up to 3-4% in Madras, South India. Some unique features that distinguish DCPA from banal pustular folliculitis include its peculiar localization to the legs, extreme chronicity, resistance to therapy and inevitable alopecia and atrophy of the involved skin, with little postinflammatory hyper- or hypopigmentation. Further, even in the presence of extensive lesions, there are no systemic features. Coagulase-positive *Staphylococcus aureus* is known to have a role in the etiology of DCPA, but the exact etiopathogenesis still needs to be elucidated. Immunological postulates such as hypergammaglobulinemia have been put forward to explain the chronicity of the condition. A number of therapeutic agents have been tried in various studies, including cotrimoxazole, psoralen with ultraviolet A (PUVA) therapy, ciprofloxacin, pentoxifylline, rifampicin, dapsone, minocycline and mupirocin (topical) with variable success rates. Although a well-recognized entity in dermatology clinics in tropical countries, DCPA has received little attention in the dermatological literature and has only a few studies to its credit. Its unique clinical picture, unclear etiopathogenesis and resistance to therapy afford a vast scope for further investigation and study.

DOI: 10.4103/0378-6323.53129

**Key words:** Chronic folliculitis, Dermatitis cruris pustulosa et atrophicans, *Staphylococcus aureus*

## INTRODUCTION

Pyodermas are common purulent infections of the skin caused mainly by *Staphylococcus aureus* and *Streptococcus pyogenes*.<sup>[1]</sup> Primary pyodermas may be follicular or nonfollicular. The common causes of superficial folliculitis are *S. aureus*, coagulase-negative staphylococci and physical or chemical irritation. *S. aureus* is among the most important causes of skin infections and serious, sometimes fatal, systemic disease.<sup>[1]</sup>

Folliculitis of the skin may be superficial or deep, as in pyodermas caused by pyogenic organisms like *S. aureus* or due to other factors like irritation or occlusion. However, there are a number of inflammatory diseases involving the hair follicle, the etiology of which are complex or uncertain, with the role of bacterial

infection being partial, secondary, not known or absent. For example, these include conditions such as sycosis barbae and folliculitis decalvans.<sup>[1]</sup>

Dermatitis cruris pustulosa et atrophicans (DCPA) is a distinctive type of chronic superficial folliculitis, of complex etiology, seen primarily in tropical countries. It primarily affects the anterior shins, with recurrent crops of itchy follicular pustules, which resolve either spontaneously or with treatment, resulting eventually in atrophy and loss of hair over the affected skin. The disease typically responds to a variety of topical and systemic antibiotics, but is characterized by recurrences that may be triggered by a variety of factors. Thus, DCPA has several unique features that distinguish it from banal pustular folliculitis, such as its peculiar localization to the legs, extreme chronicity, resistance to therapy and inevitable alopecia and atrophy of the

**How to cite this article:** Kaimal S, D'Souza M, Kumari R. Dermatitis cruris pustulosa et atrophicans: A review. Indian J Dermatol Venereol Leprol 2009;75:348-55.

**Received:** October, 2008. **Accepted:** January, 2009. **Source of Support:** Nil. **Conflict of Interest:** None declared.

involved skin, without any systemic symptoms.<sup>[2,3]</sup>

**HISTORICAL PERSPECTIVE**

Follicular diseases resulting in permanent alopecia and scarring have been known to physicians for over a 100 years. In the last century, French dermatologists described a number of these conditions under various names: pseudopelade (Brocq, 1885), folliculitis decalvans (Quiquaud, 1888) and acne decalvante (Lailler, 1884).<sup>[2]</sup>

DCPA first earned a mention under the terminology “epilating folliculitis of the glabrous skin,” described by Arnozan and Dubreuilh as “therapy-resistant symmetrical follicular pustules of both legs resulting in alopecia, atrophy and scarring.” Although many such cases were reported in the European dermatological literature, it was an English dermatologist, Clarke, who is credited with describing a similar condition in the dark-skinned Africans of Nigeria. He coined the term “Dermatitis Cruris Pustulosa et Atrophicans” in the year 1952.<sup>[2]</sup> Miller described the first case from North America in 1961. However, he suggested the name “Lupoid Sycosis of the legs.”<sup>[2]</sup> In India, Desai *et al.*,<sup>[4]</sup> in 1966, studied what they called “therapy-resistant pyogenic folliculitis of the legs” in 30 patients. Subsequent studies by the same authors showed these cases to be identical to DCPA. However, they also reported the occurrence of immunological abnormalities in these patients in the form of hypergammaglobulinemia.

Harman,<sup>[3]</sup> in 1968, reviewed DCPA and studied it extensively among 15 outpatients in the Ibadan province of Nigeria. He gave a detailed clinical description of the lesions of DCPA, calling it the “Nigerian shin disease.” He noted that although *S. aureus* was isolated from the lesions, they were uniformly resistant to antibacterial agents. He also suggested that the traditional practice of applying vegetable oils, especially coconut oil, may be an essential causative factor.

In India, the disease has been termed “chronic folliculitis of the legs” (CFL) by Prasad *et al.*<sup>[5]</sup> The disease has thus acquired various names over the years, including “Nigerian shin disease,”<sup>[3]</sup> “lupoid sycosis of the legs,”<sup>[2]</sup> “therapy-resistant pyogenic folliculitis of the legs,”<sup>[4]</sup> “folliculitis cruris pustulosa et atrophicans”<sup>[2]</sup> and CFL.<sup>[5]</sup>

**EPIDEMIOLOGY**

There is very little published epidemiological data on DCPA. As only hospital-based data are available, it

serves at best as an approximate guide to the incidence of the disease in the community.

DCPA is fairly commonly encountered in outpatient clinics in humid tropical climates. Shrank and Harman<sup>[6]</sup> reported a frequency of 2.2% in the Ibadan province of Nigeria from 1961 to 1963, while the Figure in a skin clinic in Kaduna, Northern Nigeria, was 4.8% among all outpatients in 1974.<sup>[7]</sup> However, a recent study from another skin clinic in Kaduna, Nigeria, reported a reduced frequency of DCPA (0.4%).<sup>[8]</sup> DCPA was reported to occur in 3–4% of dermatology outpatients in Madras, South India, while the corresponding Figure was 2.9% in Matara, Sri Lanka.<sup>[9]</sup> In general, DCPA is seen primarily in West Africa, Trinidad, India and Malaysia, whereas no definite cases have been reported after Miller’s original report in 1961 from any part of the United States or Europe till date.<sup>[3]</sup>

DCPA is a disease predominantly of males,<sup>[2,9,10]</sup> although some studies have reported an equal incidence in males and females.<sup>[3,7,11]</sup> Males constitute anywhere between 50 and 91% in various studies.<sup>[2,7]</sup> Although it has been reported to occur up to the sixth decade,<sup>[2]</sup> it is largely a disease of the second to third decade.<sup>[2,3,9,11]</sup> The disease is invariably chronic, the duration lying anywhere between a few months to 22 years,<sup>[2,5,12]</sup> with some studies reporting a duration of 1-2 years in the majority of their patients.<sup>[5,7]</sup> Sugathan *et al.* found that a third of their patients had a disease duration greater than 5 years.<sup>[2]</sup>

**CLINICAL FEATURES OF DCPA**

The main features of the eruption are shown in Table 1.<sup>[2,3]</sup>

The general health is not affected and there is a notable

**Table 1: Clinical features of dermatitis cruris pustulosa et atrophicans**

• Pustular folliculitis
• Pruritus
• Cutaneous pitting edema
• Wiry roughness of hair in the involved area
• Scale formation
• Loss of skin marking
• Remarkable shininess of the surface
• Inevitable alopecia and atrophy of the affected skin
• Complete absence of postinflammatory hypo- or hyperpigmentation
• Symmetrical involvement of both legs between the knee and ankle, mainly on the anterior surfaces of the shins

absence of systemic symptoms such as fever as well as regional lymphadenopathy, even in the presence of extensive lesions. The state of nutrition and hygiene is usually satisfactory.<sup>[2,3]</sup>

Some Indian studies have noted a unilateral onset of disease in 25-75% of the patients.<sup>[2,12]</sup> Among these patients, the time taken for the involvement of the other leg varied from 1 month to 8 years.<sup>[2]</sup> The degree of pruritus in DCPA ranges from mild to severe. It may be the most prominent and distressing symptom in some,<sup>[5,13]</sup> while it is common but mild in others.<sup>[9]</sup> Pruritus may persist even after resolution of the lesions and sudden onset of pruritus may be a marker of relapse.<sup>[5]</sup>

Invariably, the eruption begins on the lower third of both legs. The borders are well defined, especially the lower margin, which is very sharp, but conforms only approximately to the lower limit of the terminal hairs at the ankle.<sup>[3]</sup> Even in advanced cases, some totally unaffected hair follicles can be seen on the ankle distal to the eruption. The dorsa of the feet and other areas of glabrous skin are never affected.<sup>[3,12]</sup> Sugathan *et al.*<sup>[2]</sup> observed in their study that 52% of patients had involvement of the front of the thighs as well. Also, involvement of the forearms (22.8%) and the face (3.8%) was noted by them among Indian patients. The trunk, scalp and pubic region were never affected.<sup>[2,3]</sup> Some other studies have also recorded involvement of areas other than the legs, such as the thighs and forearms, ranging from 13.7 to 35% of the cases.<sup>[3,5,7,9]</sup>

Nearly all follicles in the affected area contain a pustule, from the center of which a single hair can be seen to be emerging. The leg usually becomes scaly and shiny, with a variable amount of pitting edema. Normal skin marking is lost and there is some atrophy.<sup>[2]</sup> In long-standing cases, the direction in which the terminal hairs emerge is haphazard such that running the fingers lightly over them gives a sensation of wiry roughness.<sup>[3]</sup> Also, with chronic cases, there may be a reduction in hair-bearing follicles and the remaining hair may also be short and irregular. The disease may undergo a natural cure once all the hair in the affected area is lost.<sup>[9]</sup> Further, in contrast to many inflammatory dermatoses, there is little postinflammatory hyper- or hypopigmentation.<sup>[2]</sup> However, one study from Sri Lanka did record postinflammatory hyperpigmentation.<sup>[9]</sup> Eczematous changes have been described in a few patients, whereas in others, signs and symptoms such as minimal pain, burning, oozing, bleeding and scaling have been seen.<sup>[5,9]</sup>

The individual manifestations of the condition vary from case to case and in the extent to which they dominate the clinical picture. Pustular folliculitis, scaling, shiny edema and follicular atrophy with scarring may each be the major feature in different patients. Owing to this pleomorphic dominant picture, Sugathan *et al.*<sup>[2]</sup> suggested a clinical grading for DCPA [Table 2].

Thus, DCPA has a number of peculiar clinical features that justify its status as a distinct clinical entity: The appearance of follicular pustules associated with pruritus, symmetrically localized to the legs, leading to alopecia and atrophy, with occasional, although rare, involvement of the forearms, face and scalp; the trunk and pubic region never being affected in spite of these sites fulfilling the prerequisite of possessing well-developed terminal hair.

#### AGGRAVATING FACTORS

Subjective and objective aggravating factors have been reported in most studies. In early studies by Harman in Ibadan, application of vegetable oils, especially coconut oil, was considered an essential causative factor.<sup>[3]</sup> The application of oil for cosmetic or medicinal purposes is a common practice in Western Nigeria, where four types of oils are commonly used, namely, coconut oil, palm oil, palm kernel oil and shea butter.<sup>[3,11]</sup> In India, coconut oil and mustard oil are commonly used.<sup>[12,13]</sup> Jacyk suggested that occlusion, leading to increased hydration of the stratum corneum, makes DCPA worse because moist skin has a higher friction coefficient than dry skin and is hence more vulnerable to trauma.<sup>[11]</sup> Sugathan *et al.* reported a wide variety

**Table 2: Clinical grading of DCPA proposed by Sugathan *et al.*<sup>[2]</sup>**

Grade I	Patients having follicular pustules only, a few of them showing well-defined perifollicular erythema [Figure 1]
Grade II	Predominant lesions are follicular pustules but there are also a significant number of infiltrated papules around broken hairs. The papules show excoriation marks, crusting or a peripheral rim of white scales. Alopecia is observable, but not marked. Palpating hands can detect a wiry roughness due to broken or irregular hair [Figure 2]
Grade III	This stage is characterized by a number of infiltrated scaly papules, although a few scattered pustules can be seen in the periphery. There is also atrophy, shininess and marked alopecia [Figures 3 and 4]
Grade IV	This is characterized by almost complete alopecia and atrophic, shiny and scaly skin. Pustules are no longer seen. However, a few discrete scaly papules can be seen at the periphery



Figure 1: A close-up view of follicular pustules in DCPA



Figure 2: Grade II DCPA - many pustules, few papules, early alopecia and atrophy



Figure 3: Grade III DCPA - alopecia, atrophy and few pustules. The lower limit of shininess and atrophy is well-defined.



Figure 4: Forearm involvement by grade III DCPA with numerous papules. The margin near the left elbow is well-defined.



Figure 5: Biopsy showing an intrafollicular abscess, containing polymorphs, with extravasated erythrocytes in the dermis (H and E, x40)

of factors, including the ingestion of animal protein, consumption of alcohol, sleeplessness, excessive sweating and contact with wet soil and cowdung.<sup>[2]</sup>

They, however, found that application of coconut oil did not in any way influence the course of the disease, a finding similar to that of Desai *et al.* in another study.<sup>[4]</sup> Some patients who came in contact with oil at the work place (cooks, drivers, mechanics) reported improvement during leave at home.<sup>[13]</sup> Later studies pointed out that while oil application was all over the body, lesions were localized to the lower limbs. Also, patch and usage tests with coconut and mustard oil were negative.<sup>[12]</sup> Allergic contact dermatitis to topically applied antibacterial agents has been postulated as a perpetuating factor for the folliculitis.<sup>[12]</sup>

Physical or chemical injury may also precipitate this condition, a possibility reflected by the finding that most of the patients were farmers in one Indian study by Prasad *et al.*<sup>[5]</sup> In another study, most patients were sugarcane harvesters and they attributed the cause of their folliculitis to the stiff needles on the bark of the

sugarcane.<sup>[10]</sup> Although patients may be from diverse walks of life,<sup>[4,7]</sup> outdoor work<sup>[10]</sup> is an important predisposing factor. A field survey of epilating folliculitis among fishermen in Calicut, Kerala,<sup>[14]</sup> revealed an incidence of 20.4%, which is much higher than that reported in the general population. Constant wetting of the skin with sea water was considered an important predisposing factor.<sup>[14]</sup> Additional relevant etiologic factors may be scrubbing of the legs with rough objects like pumice stones, ichthyosis and occupational exposure to irritants.<sup>[9,11]</sup> The disease shows a pronounced seasonal variation, with most exacerbations occurring in the summer and monsoon months.<sup>[2,11]</sup>

Conditions known to be associated with resistant pyogenic infections, such as diabetes, malnutrition, tuberculosis and malignancy, have not been found in patients with DCPA.<sup>[2,4,11]</sup>

### IMMUNOLOGICAL ABNORMALITIES

One of the most important features of the disease has been its striking chronicity, with disease duration of up to 22 years being reported.<sup>[2]</sup> This would seem to suggest an underlying immunological mechanism. As early as 1969, Desai *et al.*<sup>[4]</sup> had reported the occurrence of hypergammaglobulinemia in 30 patients. Sugathan *et al.*<sup>[2]</sup> further found that the presence of hypergammaglobulinemia had no correlation with the extent of involvement or duration of illness and, therefore, reflected only the presence of a chronic infection. Subsequent investigations by Tiwari *et al.*<sup>[13]</sup> in 15 soldiers showed that immunoglobulin (Ig) G was increased in all cases. The levels varied from 1650 to 1973mg/100 ml. IgA was marginally increased in 20% of the cases, while no increase in IgM was seen.<sup>[13]</sup>

A systematic study was undertaken by Parikh *et al.*<sup>[12]</sup> to address the various lacunae in the immunology of DCPA. They studied polymorphonuclear (PMN) percentage phagocytosis, intracellular killing (ICK) capacity and Ig and complement levels in 20 patients. They found that both the parameters of PMN function, i.e. percentage phagocytosis and ICK, were significantly raised above normal in the initial stage and returned to normal after treatment, suggesting that there was no abnormality of PMN function. There was no significant rise in the Ig levels, suggesting a depressed immunological response. Further, they found that C3 levels were also decreased. They felt that these two factors could play a decisive role in the chronicity of this condition. However, these findings are in contrast to earlier studies. Hence, more

studies are needed to clarify the immunological role in the etiopathogenesis of this enigmatic condition.

### MICROBIOLOGY OF DCPA

All studies on DCPA have shown primarily isolates of *S. aureus* from the pus cultures, making it virtually the prime pathogen responsible for DCPA.<sup>[3,11-13]</sup> Additional bacteria that have been grown in culture from DCPA pustules in a small percentage of cases include *Pseudomonas* spp., *Klebsiella* spp. and beta hemolytic *Streptococcus*.<sup>[5,15]</sup> Although *Trichophyton rubrum* has been considered by some authors as a possible pathogenic agent, no evidence to support this theory has been documented.<sup>[3]</sup>

In an early landmark study by Harman, swabs from two-thirds of the patients grew *S. aureus*, of which half were penicillin resistant.<sup>[3]</sup> In three patients, *S. aureus* was grown from nasal swabs as well. However, no link was established between the nasal and leg pustule flora. Phage typing of staphylococci from 12 cultures from the same series showed a heterogeneous collection representative of organisms typically found as secondary invaders in lesions due to some other cause, similar to any skin department in Britain.<sup>[1]</sup>

Tiwari *et al.*<sup>[13]</sup> isolated *S. aureus* from pustular lesions in all their patients; however, a majority (87%) were resistant to penicillin but sensitive to erythromycin, chloramphenicol, ampicillin and other antibiotics. Similar results were also obtained by Parikh *et al.*, who studied 20 patients of DCPA and isolated *S. aureus* from all their pus samples.<sup>[12]</sup> Predictably, all strains were resistant to penicillin but sensitive to erythromycin, chloramphenicol and cotrimoxazole.

### HISTOPATHOLOGY OF DCPA

Some authors consider DCPA a combination of superficial and deep folliculitis,<sup>[1]</sup> while others are of the opinion that inflammatory changes are confined to the ostium or slightly below it and healing correspondingly occurs without scar formation.<sup>[16]</sup> The epidermis shows mild to moderate hyperkeratosis and parakeratosis in loose bands of stratum corneum.<sup>[13]</sup> Findings in early lesions include only nonspecific focal round cell infiltration of the dermis and focal infiltration of the mouth of the hair follicle by polymorphs.<sup>[2]</sup> Intraepidermal and subcorneal pustule formation containing leukocytes is seen,<sup>[4]</sup> with adjacent areas of the skin heavily infiltrated by

neutrophils, lymphocytes and occasional eosinophils. Well-developed pustular lesions show a “wine glass”-shaped abscess involving the upper part of the hair follicle without involvement of the bulb or root of the hair [Figure 5]. The hair shaft can be seen passing through the abscess cavity.<sup>[2]</sup> Some of the pustules in grade I and II disease were found to be unassociated with hair follicles in one study by Jacyk.<sup>[11]</sup> Flakes of keratin and acantholytic cells can also be seen in the abscess cavity. Lymphocytic and plasma cell infiltration of the sweat glands and hair follicles can also be seen. There is, however, an absence of foreign body giant cells.<sup>[2]</sup> Another notable feature is the

presence of marginal acanthosis around the abscess.<sup>[2,3]</sup> A recent report noted the histopathologic presence of an intraepidermal neutrophilic pustule along with eosinophilic cellulitis and panniculitis with flame figures in a female patient with lesions of both DCPA and prurigo nodularis.<sup>[17]</sup>

In the cicatricial stage, the rete ridges are atrophic, with atrophy of the appendages, upper dermal fibrosis and focal collection of round cells around the appendages.<sup>[11]</sup> Additional epidermal and dermal changes that have been described include hyperkeratosis, parakeratosis, acanthosis, spongiosis and occasional red blood cell-containing vesicles.<sup>[2,3]</sup>

**Table 3: Treatment of DCPA: Regimens used in previous studies and their efficacy**

Author(s)	Regimen	Follow-up period	Efficacy
Jacyk <sup>[7]</sup>	Cotrimoxazole: 1 <sup>st</sup> week – 2 tabs BID 2 <sup>nd</sup> week – 1 tab TID 3 <sup>rd</sup> week – 1 tab BID Next 4 weeks – 1 tab QD	6 months	Response seen in 23 of 24 patients (95.83%); remission for 6 months in 19 patients (79.17%)
Parikh <i>et al.</i> <sup>[12]</sup>	Cotrimoxazole (double strength): 2 tabs BID for 6 weeks	Not mentioned in the study	17 of 20 patients (85%) showed a good response
Shenoy <i>et al.</i> <sup>[18]</sup>	Cotrimoxazole 1 tab BID + PUVASOL (20 mg 8-MOP at 8 AM followed by exposure to sunlight from 10 AM to 10.15 AM). Duration of therapy not mentioned in the study	90 days	100% showed complete clearance at 15 days, 90.48% at 45 days and 62.5% at 90 days
Das and Gogoi <sup>[19]</sup>	PUVA therapy for a period of 10 weeks along with a course of cotrimoxazole for 2–3 weeks	6 months	Complete improvement in 68 patients (85%), partial improvement in 12 patients. Recurrence in five patients during the follow-up period
Kumar and Muralidhar <sup>[20]</sup>	Topical mupirocin 2% ointment twice daily after washing	6 months	50% improvement in 7 days and complete clearing in 4 weeks for all three patients. The first patient showed no recurrence over follow-up while other patients were still on follow-up
Balachandran <i>et al.</i> <sup>[21]</sup>	Ciprofloxacin one tab BID for 2 weeks	24 weeks	21 of 23 patients (91.30%) improved; average remission period was 44.5 days
Prasad <sup>[16]</sup>	50 patients; 25 patients each: rifampicin 10 mg/kg and dapsone 100 mg per day for 8 weeks. Patients not showing any improvement within 8 weeks crossed over to the other drug for eight more weeks	6-9 months	Rifampicin group: Complete resolution in 18 patients (72%), moderate response in 8% and no response in 4% Dapsone group: Complete resolution in none, moderate response in 20%, mild response in 4% and no response in 44%. Dapsone nonresponders crossed over to rifampicin: Complete resolution in seven of 17 (41.16%), moderate improvement in nine patients and no response in one Relapse: 15 of 42 (35.71%) on rifampicin
Prasad <i>et al.</i> <sup>[22]</sup>	Group I (19 patients): Ciprofloxacin BID + placebo TID x 2 weeks, followed by placebo TID x 4 weeks Group II (19 patients): Ciprofloxacin BID for 2 weeks + pentoxifylline 400 mg TID followed by pentoxifylline 400mg TID x 4 weeks	6 months	Group I: Complete resolution in 12 of 18 patients (66.7%), good response in three (16.7%) and no response in three (16.7%). Relapse in 15 of 18 patients (83.3%); average remission period: 42.5 days Group II: Complete resolution in 15 patients (88.3%), good response in one (5.9%) and no response in one (5.9%). Relapse in three (17.7%); average remission period: 103 days
Prasad <sup>[23]</sup>	Minocycline 100 mg QD for 21 days	6 months	Complete clearance in all eight patients; mild clinical recurrence in two (25%)

## TREATMENT OF DCPA

Treatment for this condition is unsatisfactory, leading to the usage of the term “therapy-resistant folliculitis.” Both Harman and Sugathan, in their studies, have remarked about the resistance to various systemic and topical therapies.<sup>[2,3]</sup> Modalities of therapy for DCPA that have been studied include systemic and topical antibacterial agents (cotrimoxazole, ciprofloxacin, rifampicin, dapsone, minocycline, topical mupirocin), pentoxifylline and PUVA. Table 3 shows a summary of the treatment regimens used for DCPA in various studies.

Ultimately, combination therapy with antiseptic lotions, long-term systemic antibiotics, topical antibiotics and avoidance of risk factors are essential in controlling the disease. However, the disease is generally refractory to most forms of therapy and it is difficult to halt its inexorable progression to alopecia and atrophy of the affected site.

## CONCLUSION

Although a common entity, DCPA has received little attention in dermatological literature and has only a few studies to its credit. Its unique clinical picture, unclear etiopathogenesis and resistance to therapy afford a vast scope for further investigation and study.

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## Multiple Choice Questions

- Most common site of involvement of DCPA is:
  - Anterior shin
  - Beard
  - Fore arms
  - Scalp
- All of the following terms are synonymous with DCPA except:
  - Folliculitis cruris pustulosa et atrophicans
  - Nigerian shin disease
  - Lupoid sycosis of legs
  - Dissecting folliculitis of legs

3. All the following factors predispose to DCPA except:
  - a. Tropical humid climate
  - b. Female gender
  - c. Friction
  - d. Occlusion
4. Which of the following statements regarding DCPA are false:
  - a. Wiry roughness of hair is seen in the involved area
  - b. Pruritus is characteristically absent.
  - c. Scaling is present
  - d. Atrophy and alopecia is inevitable
5. Presence of infiltrated scaly papules with mild atrophy, shininess and alopecia in a patient classifies him to be in which grade:
  - a. Grade I
  - b. Grade II
  - c. Grade III
  - d. Grade IV
6. Most common primary isolate in cases of DCPA is:
  - a. *Pseudomonas* spp.
  - b. *Beta hemolytic streptococcus*
  - c. *Trichophyton rubrum*
  - d. *Staphylococcus aureus*
7. Modalities of therapy for DCPA include all except:
  - a. Cotrimoxazole
  - b. Rifampicin
  - c. Azithromycin
  - d. Topical mupirocin
8. Involvement with DCPA is characterized by all except:
  - a. Usually bilateral
  - b. Symmetric eruption
  - c. Ill defined borders
  - d. Scaling and shininess of surface
9. Histopathology of DCPA shows all except:
  - a. Intraepidermal or subcorneal pustule
  - b. Neutrophilic periappendageal infiltrate
  - c. Presence of foreign body giant cells
  - d. Atrophy of appendages
10. Immunological abnormality commonly associated with DCPA is:
  - a. Hypergammaglobulinemia
  - b. Hypercomplementemia
  - c. Decreased phagocytosis by neutrophils
  - d. Increased lysosomal activity

**Answers**  
1. a, 2. d, 3. b, 4. b, 5. c, 6. d, 7. c, 8. c, 9. c, 10. a