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**Review**

# Dissecting cellulitis (Perifolliculitis Capitis Abscedens et Suffodiens): a comprehensive review focusing on new treatments and findings of the last decade with commentary comparing the therapies and causes of dissecting cellulitis to hidradenitis suppurativa

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## Abstract

Dissecting cellulitis (DC) also referred to as to as perifolliculitis capitis abscedens et suffodiens (Hoffman) manifests with perifollicular pustules, nodules, abscesses and sinuses that evolve into scarring alopecia. In the U.S., it predominantly occurs in African American men between 20-40 years of age. DC also occurs in other races and women more rarely. DC has been reported worldwide. Older therapies reported effective include: low dose oral zinc, isotretinoin, minocycline, sulfa drugs, tetracycline, prednisone, intralesional triamcinolone, incision and drainage, dapsone, antiandrogens (in women), topical clindamycin, topical isotretinoin, X-ray epilation and ablation, ablative $C0_2$ lasers, hair removal lasers (800nm and 694nm), and surgical excision. Newer treatments reported include tumor necrosis factor blockers (TNFB), quinolones, macrolide antibiotics, rifampin, alitretinoin, metronidazole, and high dose zinc sulphate (135-220 mg TID). Isotretinoin seems to provide the best chance at remission, but the number of reports is small, dosing schedules variable, and the long term follow up beyond a year is negligible; treatment failures have been reported. TNFB can succeed when isotretinoin fails, either as monotherapy, or as a bridge to aggressive surgical treatment, but long term data is lacking. Non-medical therapies noted in the last decade include: the 1064 nm laser, ALA-PDT, and modern external beam radiation therapy. Studies that span more than 1 year are lacking. Newer pathologic hair findings include: pigmented casts, black dots, and "3D" yellow dots. Newer associations include: keratitis-ichthyosis-deafness syndrome, Crohn disease and pyoderma gangrenosum. Older associations include arthritis and keratitis. DC is likely a reaction pattern, as is shown by its varied therapeutic successes and failures. The etiology of DC remains enigmatic and DC is distinct from hidradenitis suppurativa, which is shown by their varied responses to therapies and their histologic differences. Like HS, DC likely involves both follicular dysfunction and an aberrant cutaneous immune response to commensal bacteria, such as coagulase negative staphylococci. The incidence of DC is likely under-reported. The literature suggests that now most cases of DC can be treated effectively. However, the lack of clinical studies regarding DC prevents full understanding of the disease and limits the ability to define a consensus treatment algorithm.

## Introduction

In the last decade, a number of new treatments have been suggested for dissecting cellulitis (DC) also called perifolliculitis capitis abscedens et suffodiens (PCA or PCAES) or Hoffman disease, which manifests with keloids, perifollicular and follicular pustules, nodules, abscesses, and sinuses that evolve into scarring alopecia (Figure 1, Figure 2 and Figure 3). DC has been reported worldwide. In the U.S., DC predominantly occurs in African American men between 20-40 years of age. DC also affects other races and also women, albeit more rarely. DC can occur with acne conglobata, hidradenitis suppurativa (HS), and pilonidal cysts (PC); these cases are referred to as the follicular occlusion duad, triad, or tetrad. DC, HS and AC can occur concurrently with arthritis or spondyloarthropathy.

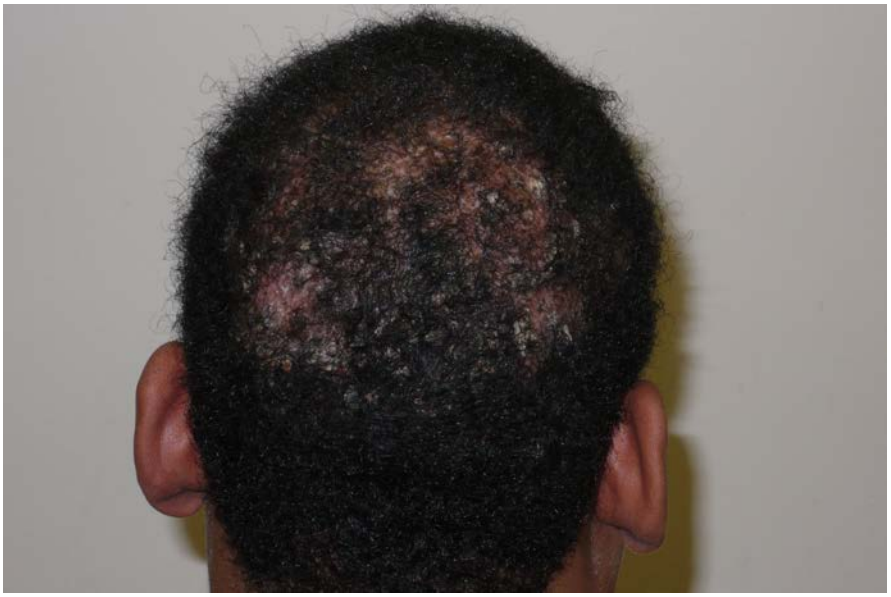

**Figure 1**. Dissecting cellulits treated with clindamycin, rifampin and intralesional triamcinolone, 10mg/kg, after 3 treatments

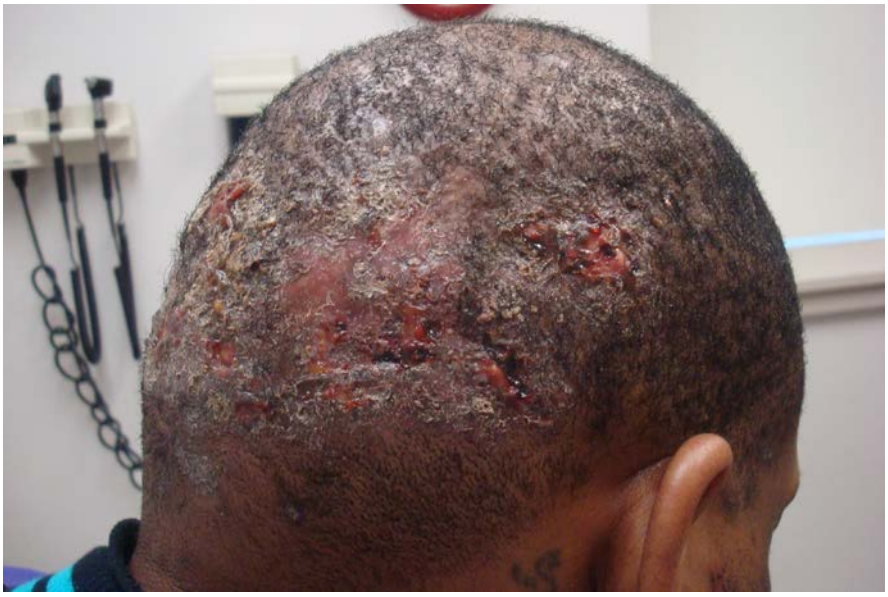

**Figure 2**. Untreated DC (Picture Provided by John Hall)

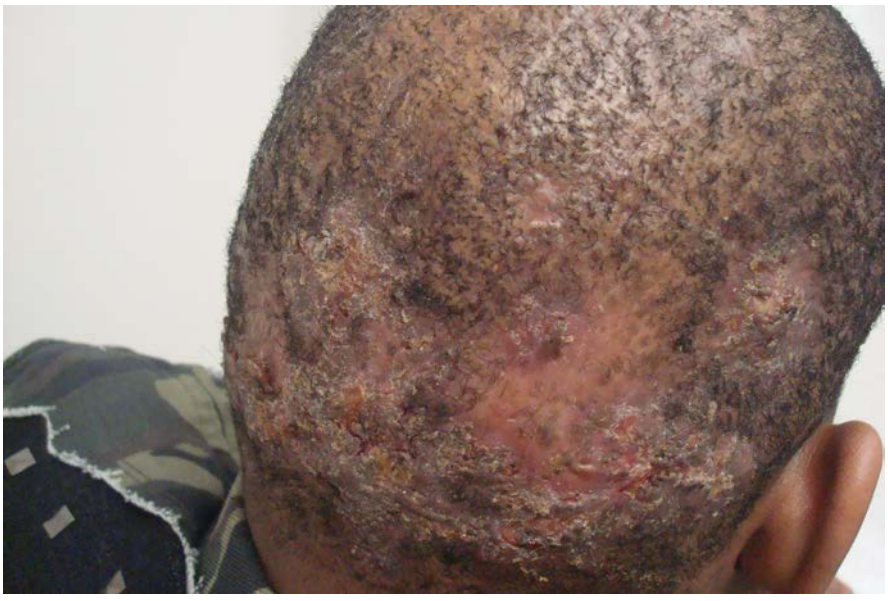

**Figure 3**. DC after 4 weeks of treatment with clarithromycin (Picture Provided by John Hall)

This review will focus mostly on the literature published about medical and light treatment noted since 2003 with a historical review of DC and its treatments. I published an earlier review of DC treatments in 2003 [1], but treatments have advanced since that time. In 2014, treatments still widely vary for DC. Fewer cases (if any) seem beyond medical treatment than in 2003 when eradication of DC required radical surgical excision, X ray, or $CO_2$ laser ablation to control cases that failed isotretinoin [1].

HS has been the subject of significant attention; this is evidenced by the fact that as of April 20, 2014, Pubmed listed 995 publications searching the term "hidradenitis suppurativa" or "acne inversa," but DC has received much less medical attention. Only 88 articles listed in Pubmed relating to "dissecting cellulitis" and 57 to "perifolliculitis capitis" were found as of April 1, 2014. An additional 5 articles were identified that discussed DC in the context of scarring alopecias,discovered by searching "scarring alopecia," "scalp and isotretinoin," and "acne kelodalis nuchae." Nevertheless the therapeutic options for DC seem to parallel and partly mirror those for HS. The parallels include the utility of tumor necrosis factor blockers (TNFBs), rifampin, the 1064nm laser, and oral alitretinoin [2]. However, the promotion of multiple treatments, such as the use of monotherapy with isotretinon, isotretinon with antibiotics (e.g. dapsone), quinolones, sulfa drugs, penicillin, macrolide antibiotics, oral corticosteroids, and modern external beam radiation show that the treatment algorithm for DC diverges from that of HS.

# Historical Review of DC

DC was first described by Spitz in 1903 who termed the disease "dermatits folliculais capitis et perifolliculitis conglobata" [3]. Hoffman [4] was the first to term the disease "perifolliculitis capitis abscedens et suffodiens." McMullan and Zeligman in 1956 were the first to report successful treatment of DC with X-ray epilation in 4 patients [5]. Moyer and Williams reported another 6 cases of DC treated with X-ray in 1962 [6]. Enrenreich was the first to link interstitial keratitis to DC in 1953 [7]. Wasserman was the first to link DC to arthritis [8]. Cornbleet and Kagen were the first to report successful treatment of DC with penicillin [9]. Musumeci was the first to compare tinea capitis and DC in 1956 [10]. Moschella *et al* were the first to report that removal of the scalp with grafting to abate DC in 1967 [11]. Barney in 1931 was the first to use the term dissecting cellulitis to describe the disease [12]. Adrian and Arndt were the first in 1981 (in an age before isotretinoin was on the market) to note the growth of *Staphylococcus epidermidis* from DC and report the remission with oral corticosteriods [13]. Connon (1944) [14] and Esterin (1948) [15] attempted treatment with intralesional steroids and topical and oral antibiotics with limited success. Cormie was the first to comprehensively focus on the histology of DC in 1962 [16].

# The differential diagnosis of DC

The differential diagnosis of DC is outlined in Table 1. Tinea capitis, which is rare in adults, may be confused with DC [17]. An interesting entity referred to as alopecic and aseptic nodules of the scalp (AANS), also calledpseudocyst of the scalp,that is distinct from DC, has been described [18]. AANS exhibits non-scarring alopecia, a cystic, circular appearance of nodules that can be solitary or small in number, which distinguishes it clinically from DC. In addition, the histology of AANS shows either multinucleated cells mixed with neutrophils, lymphocytes, histiocytes, and sometimes plasma cells or nonspecific deep lymphohistiocytic inflammation. However, there is always a lack of fibrosis and doxycycline 100 daily for 1-3 months gives excellent results in AANS. AANS has been reported mostly in Japan and France, almost always in Caucasians, but occasionally in those of African descent.

**Table 1 Disease coincident with and Mimics of DC**

| Diseases linked to DC | Mimics of DC [1] |
|---|---|
| Hidradenitis Suppurativa | Tinea capitis |
| Pilonidal Cysts | Pseudopelade of Brocq |
| Acne Conglobata | Folliculitis (spinulosa) decalvans |
| Pyoderma gangrenosum | Tufted folliculitis |
| Keratitis-Ichthyosis-Deafness Syndrome | Lichen planopilaris |
| Spondyloarthritis [76] | Alopecic and aseptic nodules of the scalp (AANS) [18] |
| Osteomyelitis [1] | Malignant proliferating pilar cysts (squamous cell cancer (SCC))[78] |
| Sternocostoclavicular hyperostosis [1] | Squamous cell cancer |
| Arthropathy, Arthritis [1] | Folliculotropic mycosis fungoides with large-cell transformation [79] |
| Crohns disease [53] | Acne keloidalis nuchae |
| Pyoderma vegetans [1] | Cutis verticis gyrata [80] |
| Pityriasis rubra pilaris [1] | Erosive pustular dermatosis of the scalp [81] |
| Marginal keratitis [1] | Metastatic cancer of the scalp |

| | |
|---|---|
| SCC in long standing DC [77] | |
| Malignant proliferating pilar cysts, HS, and KID syndrome [78] | |

# Associations with DC

DC, like HS, has now been associated with keratitis-ichthyosis-deafness syndrome [19]. In the last decade, a report has linked DC to pyoderma gangrenosum [20], a linkage already made with HS on numerous occasions. The follicular occulsion tetrad of HS, DC, acne conglobata, and pilonidal cysts has already been well reported [2]. In Table 2, the conditions that have been reported to occur with DC are listed. Arthritis [8] and keratitis have been reported [1, 7].

**Table 2 Treatments of DC**

| Medical: new | Medical: older | Light Based | Destructive |
|---|---|---|---|
| Adalimumab loading dose of 80mg followed by at week 1 40mg and then 40 mg QOW [23, 24] | Isotretinoin 0.5-1mg/kg QD for three months to one year | 1064 nm long-pulsed Nd:YAG laser | Modern external beam radiation therapy |
| Infliximab 5mg/kg dosed every 8 weeks [25, 26] | Dapsone 25-100mg BID | 800 nm pulsed diode | Scalp removal with graft |
| Ciprofloxacin 250-500 mg BID [43, 44] | Corticosteroids oral starting at 60mg with taper | 694nm laser | Older Full X-ray treatment of scalp$^{o}$ |
| Rifampin 300mg bid for 3M followed by isotretinoin for 3M[28] | Intralesional Triamcinolone acetate 5-10mg [1, 31] | ALA-PDT | $CO_2$ laser ablation of scalp [83] |
| Alitretinoin 10-20mg [19] | Colchicine [1] | | |
| Azithromycin, amoxicillin-clavulanate, fluconazole, followed by a long period of oral isotretinoin with local wound skin care [48] | Clindamycin 300mg BID -600 mg TID [65] | | |
| Minocycline 100mg BID | Trimethoprim/sulfamethoxazole DS BID [31] | | |
| Dapsone 50-100 mg with isotretinoin [36, 77] | Zinc in varying doses: oral zinc sulphate 135 mg TID to 220 mg three times daily with food and then tapered down* | | |
| Metronidazole plus clindamycin, dermatosurgical approach, and high-dose isotretinoin [47] | Tetracycline 500 mg BID [82] | | |
| Acitretin 10mg and 30 mg prednisolone daily tapered to 5 mg, 100 mg zinc aspartate QD topical glucocorticoids and tacrolimus 0.1% were alternated [22] | Minocycline 100mg BID for 3 months with cyproterone acetate 100mg orally on days 5-14 inclusive of the menstrual cycle and ethinyl estradiol 50μ orally on days 5-25 inclusive in a female patient [46] | | |
| Clarithromycin 500mg bid | | | |

*Zinc: give with copper. QD=Daily BID=Twice a day TID=Three times a day QOW=every other week. M=Month $^{o}$Disfavored with advances in x-ray treatment of skin disease.

# New treatments for DC-Retinoids-Oral Alitretinoin

Oral alitretinoin [21], dosed between 10-30mg per day, has been approved in the United Kingdom and many states in the European Union for chronic hand dermatitis that does not response to conventional therapies. Oral alitretinoin has been reported to be a useful treatment for DC in a case report [19]. This patient had KID syndrome with HS and DC and had failed treatment with

acitretin 20mg daily for 2 months followed by a partial improvement with two months of isotretinoin 20mg daily, prednisolone 25mg daily, and dicloxacillin. Photodynamic therapy was also tried with no effect. However, better improvement was seen after systemic treatment with clindamycin 300mg BID and rifampicin 300mg BID given for 3 months. Itraconazole and then use of acitretin at 20mg a day did not work at all. Alitretinoin, off-label, was given at an initial dose of 10 mg daily for 2 months, which did not fully control the disease, followed by a maintenance dose of 20 mg daily for five months. The only side effect was slight erosions of the auditory canals. The patient experienced a remission and was tapered to alitretinoin 10 mg daily. Unlike acitretin, alitretinoin has a much shorter washout period: women need only use contraception for 6 weeks post-treatment [2].

## New treatments for DC-Retinoids-Oral Acitretin

Oral acitretin has been reported to be an effective treatment for HS [2]. One report [22] notes DC treated with 10 mg acitretin and 30 mg prednisolone daily as an inpatient. The prednisolone was reduced to 5 mg per day during the course of treatment. Because of the patient's decreased level of zinc, 100 mg zinc aspartate was given daily. In addition, topical glucocorticoids and tacrolimus 0.1% were alternated. This regimen produced almost total healing within 11 days. The systemic corticosteroids were further tapered and the topical corticosteroids were stopped. Six months after discharge the patient's skin condition was stable on a regimen of 10 mg acitretin and 100 mg zinc aspartate daily, supplemented by topical therapy with topical tacrolimus 0.1%.

In sum, acitretin seems less effective than alitretinoin and isotretinoin for treating DC. This therapeutic observation is the opposite of the observation in HS, in which isotretinoin has low effectiveness and acitretin, although less well tolerated, seems efficacious. Alitretinoin seems useful for treating both HS and DC, but it is off-label and reports, both articles and abstracts, are sparse as of April 1, 2014. Oral alitretinoin is not available in the United States.

## New treatments for DC-TNFB

The TNFB, adalimumab [23, 24], has been reported to be useful for DC when isotretinoin and antibiotics failed. In one series [23], three white male patients, 27, 29, and 30 years of age with DC who had failed isotretinoin and antibiotics were given adalimumab at a dose of 80 mg administered subcutaneously, followed by a dose of 40 mg 1 week later and an additional 40 mg every second week. During treatment with adalimumab, clinical symptoms subsided within 8 weeks of treatment in all 3 patients. After 3 months, clinical activity and patients' subjective symptoms were effectively reduced. However, biopsy findings during treatment demonstrated that although the inflammatory infiltrate was reduced in 2 of the 3 patients (patients 1 and 3), preexisting pathologic residual structures such as subcutaneous sinus tracts remained unchanged. Ultimately, when treatment with adalimumab was paused, in patient 3, after 4 months of successful treatment, disease activity returned within 4 weeks and adalimumab had to be restarted. In the other report, a 39-year-old man who relapsed after treatment with multiple courses of antibiotics, intralesional glucocorticoids, surgical excision, and isotretinoin was given adalimumab at standard dosing of 80 mg as a loading dose at week 0, followed by 40 mg SC at week 1, and then 40 mg SC every other week and achieved an appreciable response. At a one-month follow-up visit, the patient reported that the pain and purulent discharge had ceased. At two months, the patient had hair growth and at five months, the DC had abated and hair was normalized. Adalimumab was continued every other week [24] with no report of recurrence noted, but follow up was not reported in subsequent years.

The TNFB, infliximab, has also been used to treat DC in two cases using a dosing of 5mg/kg. In one patient, this was done every 8 weeks for one year with full remission of the disease [25]. Another patient [26] who also had lesions on his neck and face was given infliximab 5mg/kg body weight at weeks 0 and 2. This produced a good response but a psorisiform eruption occurred. TNFB have been reported to have the paradoxical effect of causing or worsening psoriasis. The eruption was controlled with topical prednicarbate ointment and the infliximab was given at week 6, at which point surgery was performed successfully on the DC and the eruption disappeared.

TNFB are now commonly and successfully used for HS, usually at double or more of the psoriatic dose i.e. adalimumab 40-80 mg every week. The data on DC and TNFBs show the differences of HS and DC as diseases and as therapeutic dilemmas. TNFB can be a bridge to surgery for severe DC, HS, and even complex perianal fistulas in Crohn disease, which seem to heal faster and relapse later when infliximab precedes surgery [26]. It seems that DC responds better than HS to TNFB and can have a more durable response, but the small number of cases makes generalizations difficult to draw.

## Older treatments of DC

The first report of isotretinoin as an efficacious treatment for DC was published in 1986 [27]. Reports continue to be published that suggest isotretinoin, at 0.5-1mg/kg, is useful for the treatment of DC [27, 28, 29, 30, 31, 32, 33, 34, 35] for between 3 months and 1 year. The optimal duration and dose of therapy with isotretinoin and useful combination therapies that include isotretinoin have yet to be defined. One report noted that treatment with 0.8mg/kg of isotretinoin for 1 year put the disease into remission

[29]. One report noted a 19-year-old man with DC for 2 years. Multiple systemic antibiotic therapies and surgical approaches had shown no effect. Monotherapy with isotretinoin 80 mg daily for 4 weeks had not been successful. Combination therapy with dapsone 100 mg daily and isotretinoin 80 mg daily produced significant improvement. During 4 weeks of treatment significant clearing was achieved. Dapsone was reduced to 50 mg daily after 6 months and isotretinoin was discontinued gradually; the patient remained on dapsone 50 mg every other day and has remained free of recurrences for 6 months. [36]. Most of these reports, however, suggest that monotherapy with isotretinoin (3-12 months) can put DC goes into remission. In 2008, Korfitis in Greece reported four cases of DC put into remission; 3 were culture positive for *Staphylococcus aureus* and treated with rifampin 300 mg twice daily for 3-4 months. This was followed by isotretinoin, 0.5mg/kg for 3-4 months; one patient was still clear after 10 months. [28]. The affect on long-term remission of the previous antibiotic treatment followed by isotretinoin has not been defined.

An interesting question is why DC seems to respond much better to isotretinoin than does HS. The durable HS response rate to isotretinoin reported in one trial of 68 patients [37] and in another case series of 358 [38] patients was 16%. One reason why DC might respond to isotretinoin better than HS is that sebaceous gland number and volume are significantly reduced in uninvolved hair follicles from patients with hidradenitis suppurativa, whereas in DC the sebaceous glands are not reduced in size or number [39]. No reports have noted changes of the sebaceous glands with untreated DC. However, the success of isotretinoin in DC must be considered critically and not assumed because no controlled clinical studies support a durable success and the reports noted above have not discussed follow up longer than one year.

## Newer therapies with longer histories of use-Zinc.

A number of reports note the efficacy of zinc for DC [22]. One authority recommends starting patients on zinc sulphate 220mg TID as an effective solo treatment [31]. A Japanese report [40] notes a patient with DC and acne conglobata who failed minocycline 100mg BID and clindamycin lotion but was then successfully treated with oral zinc sulphate 135 mg three times a day. After 4 weeks of this treatment, the nodules stopped growing and became flat; after 12 weeks, hair regrowth was noted in the involved scalp skin. Thereafter, zinc sulphate was reduced to 260 mg per day and kept at this dose for 7 weeks. During that period, there were no signs of recurrence 1 week after stopping the oral zinc therapy. The DC recurred despite restarting minocycline 100 mg daily. Oral zinc sulphate therapy in addition to minocycline was then started and the DC diminished within 8 weeks. Afterwards, the lesions were well controlled for 1 year by oral zinc sulphate 135 mg once or twice daily without minocycline. During this period, the patient's plasma zinc and copper levels remained within the normal range. A 24-year-old man with a one-year history of DC was treated with 400 mg of oral zinc sulfate (equivalent to 90 mg Zn++) TID. After 12 weeks healing was complete, with almost full regrowth of hair. The zinc dose was then halved and after an additional 2 1/2 months of administration therapy was stopped. Despite the high initial dose of 270 mg of zinc sulfate, this patient suffered no nausea, which is quite a common side effect of zinc therapy owing to the irritative effect on the gastric mucosa [41]. Zinc (90 mg of zinc gluconate per day) has also been reported to be useful for the treatment of HS [42]. It should be recalled that zinc chelates copper out of the body, so if high dose zinc is taken then copper levels should be tested to determine a need for supplementation.

## Ciprofloxacin

Reports of the utility of ciprofloxacin for DC have been noted in the last 10 years [43, 44]. One involved a sub-continental patient from India who had a good response of his DC to gatifloxacin while he was living in India. The DC relapsed after he moved to the United Kingdom. After failing isotretinoin, minocycline, dapsone, and zinc, there was a mild response to 40 mg daily of prednisolone. The patient was changed to ciprofloxacin 250 mg twice daily. This produced flattening of the nodules in one month [43]. In a comment published on this article, a patient was noted with DC who had tolerance issues with isotretinoin, but was started on ciprofloxacin 500 mg BID for 4 weeks and then tapered to 250 mg for 3 weeks with good response and virtual remission at 3 months [44]. The commentators who have used ciprofloxacin believe ciprofloxacin works because of the anti-inflammatory effect; the patient's DC was culture negative. It is just as likely that commensal bacteria triggered the DC and that ciprofloxacin inhibits their growth; commensual organisms may not be noted on culture reports. Similar speculation regarding why antibiotics are effective for treating acne and HS exists. A combination of rifampin-moxifloxacin-metronidazole has been used with success to treat HS [45]. The wide spectrum and anti-inflammatory effects of quinolones make them interesting agents for diseases of follicular occlusion. The potential side effects of quinolones, such as the rare incidence of tendon rupture (estimated at about 1:10,000), dictate that physicians should use them with care.

## Other antibiotic and hormonal treatments for DC

The list of antibiotics used to treat DC is long. Some clinicians prefer to initiate treatment with trimethoprim/sulfamethoxazole DS BID for 2 weeks followed by tetracycline 500mg BID to maintain response [31]. Successful therapy of the follicular occlusion triad in a young woman consisted of cyproterone acetate 100mg orally on days 5-14, inclusive of the menstrual cycle, ethinyl estradiol 50µ orally on days 5-25, and minocycline 100 mg BID for 3 months [46]. Antibiotics can act as adjuncts or

preparation regimens for isotretinoin. A patient in India required metronidazole plus clindamycin, drainage, and high-dose isotretinoin to get his DC under control [47]. It is likely that the primary agent that exerted the positive effect was the isotretinoin. The combination of sulfa drugs and rifampin has been noted as effective treatment for DC [31]. Preparation therapy for isotretinoin was noted in a case of DC treated with systemic azithromycin, amoxicillin-clavulanate, and oral fluconazole, followed by a long period of oral isotretinoin. This combination along with local skin care produced resolution [48]. It is likely that the primary agent that exerted a curative affect was the isotretinoin. I brought a case of DC under control with oral rifampin 300 mg BID, clindamycin 300mg BID over 6 weeks, and intra-lesional corticosteroids, 10mg/cc (3 cc at each visit) twice weekly over 4 visits. The patient was not cleared but his pain was much decreased and the amount of pus produced was much decreased (Figure 1). The use of rifampin with other antibiotics has utility because rifampin suppresses *Clostridium difficile*. Unfortunately, rifampin has many drug interactions.

## Prednisone

Prednisone can be helpful for DC and is often combined with other agents. A case of DC in a 22-year-old woman was put into remission with 60mg per day. She was gradually tapered to 5 mg every other day one year later [49]. Prednisone has been used with acitretin [21]. A number of other reports in this article have noted the use of prednisone, but since the advent of isotretinoin its use seems less necessary. However, a short course can be a bridge to success with isotretinoin or other therapies because of its rapid anti-inflammatory effect.

## Topical therapy and DC

Some reports note that topical care is important in the treatment of DC. Some authorities recommend an avoidance of pomades in the hair [31]. Although it is true that pomade acne is a real entity, no evidence exists that DC started after pomade use. One case report noted remission of the disease with topical isotretinoin [50]. Another noted that topical tacrolimus and topical steroids helped to bring the disease under control [22]. Others have used topical clindamycin with other oral antibiotics [1, 40]. The use of topical treatments, however, seems a weak arrow to add to the therapeutic quiver and those who treat DC should likely focus on the use of systemic treatments.

## Laser, Photodynamic and Modern External Radiation Therapy for DC

Ablation of the follicle could also play a role in the treatment of DC. In my 2003 paper I noted that the 800nm diode laser [51] and 694nm long-pulse non-Q-switched ruby laser have been used successfully to treat DC [52]. One report in the last decade notes that the 1064nm laser can treat DC [53]. Four patients with long-standing dissecting cellulitis were treated with consecutive laser treatments with the long-pulsed Nd:YAG laser without epidermal cooling. One year after initiating laser treatment, patients achieved decreased pus formation, a reduced reliance on systemic treatments, and a controlled or terminated disease process without dyspigmentation. Three patients had regrowth of terminal hairs in treatment sites [53]. On a similar note to HS, the 1064nm seems the most effective laser for treatment [54].

A female DC patient was treated successfully with 6 sessions of topical ALA-PDT [55]. ALA-PDT seems to be less successful in the treatment of HS; there are a number of conflicting reports on its efficacy in the literature. It should be noted that PDT breaks up the biofilm of coagulase negative Staphylococcus i.e. Staphylococcus epidermidis [56, 57] and if this bacteria appears to aggravate a particular case of DC, PDT with its mild side effect profile could be a first line treatment. It could be that stage, sex, and race might play a role in the utility of ALA-PDT for DC.

X-ray therapy might still have a role in 2014 in the physician's therapeutic armamentarium. Modern external beam radiation therapy for refractory dissecting cellulitis of the scalp that had failed antibiotics, dapsone, corticosteroids, isotretinoin, and laser therapy was found effective, with follow-up ranging from 4 to 13 years [58]. Hair does not re-grow after radiation therapy.

## Surgery

As is true in HS, sometimes patients fail medical therapy and surgery is needed. This was the case as noted above [26] in a patient treated with infliximab effectively that was resistant to antibiotics and isotretinoin. The infliximab produced the side effect of a psoriasiform eruption and could not be continued, but infliximab use was a bridge to successful surgery. Williams *et al* noted 4 DC patients who had failed conventional therapy who had complete scalp excision followed by successful split-thickness skin grafting [59]. However, surgery is not always curative and the DC can return. Of note, there is a report of a case of localized DC that was treated with surgical excision, but recurred one year later at the site of the surgical scar [24].

# Histology and Hair Pathology of DC

The histology of HS and DC is similar but not identical. Granulomas seem more common in DC (and for that matter Crohn disease) than HS but occur in both. The histopathologic picture of DC varies based on its stage. Early lesions are characterized by a dense neutrophilic, lymphocytic, histiocytic, and plasma cellular infiltrate. Abscesses may be present in the dermis and even in the subcutaneous tissue. In later stages, chronic granulomas can be observed that consist of lymphocytes, plasma cells, and foreign-body giant cells. Scarring and fibrosis are frequently seen in the late stages [60]. Subcutaneous sinus tracts can be present in DC [23].

There are many anomalies in the hair and hair shafts within areas of HS. An interesting study of scalp diseases, including DC, showed that many of these diseases manifest with pigmented casts. The pigmented casts were present in 21 of 29 cases of alopecia areata (AA) (72%), 7 of 7 cases of trichotillomania (100%), 1 case of friction alopecia, 4 of 28 cases of central centrifugal cicatricial alopecia (14%), and 4 of 4 cases of DC (100%) [61].

Black dots are macrocomedo-like round structures localized to the follicular ostium, which are considered a specific trichoscopic feature of alopecia areata (AA). In fact they were found in 11% (22/107) of patients with hair loss, including 53.3% (16/30) with AA, 40% (2/5) of patients with severe chemotherapy-induced alopecia,100% of patients with DC of the scalp (n = 2), hypotrichosis simplex (n = 1), and congenital aplasia cutis (n = 1). No black dots were seen in patients with androgenetic alopecia or telogen effluvium [62].

Trichoscopy [63] was performed in a total of 1,884 consecutive patients presenting with hair loss. In this group, 84 patients were diagnosed with cicatricial alopecia and 1,800 patients with non-cicatricial alopecia. Sixty healthy persons served as controls. Researchers performed trichoscopy using the Fotofinder II videodermoscopy system. Researchers identified the following unique or characteristic features: scattered dark-brown discoloration of the skin, large yellow dots and thick arborizing vessels in cutaneous (discoid) lupus erythematosus (n=20), tubular perifollicular scaling and elongated blood vessels in lichen planopilaris (n=28), minor perifollicular scaling in frontal fibrosing alopecia (n=19), tufted hairs with starburst pattern perifollicular hyperplasia in folliculitis decalvans (n=9), and large, "3D" yellow dots imposed over dystrophic hairs in DC (n=8). Rakowska *et al* [64] stated that in DC characteristic findings are "3D" yellow dots imposed over dystrophic hairs, large, yellow amorphous areas, and pinpoint white dots with a whitish halo.

# Bacteria and DC

Although the abscesses of DC are usually sterile, one report notes that needle aspiration of a large pustule yielded non-foul-smelling purulence that was cultured for aerobic and anaerobic bacteria. Gram staining revealed numerous polymorphonuclear leukocytes and Gram-positive cocci in chains. The culture grew a heavy load of *Prevotella intermedia* and *Peptostreptococcus asaccharolyticus*. Because the lesions of DC and HS are usually sterile, it is hard to know what to make of this report. Treatment with oral clindamycin 600 mg TID was given for 6 weeks along with topical isotretinoin [65]. The patient's condition improved significantly over 12 months of follow-up. In one case of DC, discharged pus revealed coagulase negative *Staphylococcus epidermidis* [66], which has been noted in other cases [33, 49]. The presence of coagulase negative staphylococci (CNS) has been reported several times in HS. One report notes the growth of *Pseudomonas aeruginosa* in a 10-year-old girl [67]. The presence of coagulase negative staphylococci (CONS) has been reported several times in HS. The significance of bacterial culture and its effect on the treatment of DC remains to be defined. In a report in which patients were successfully treated with adalimumab after failing antibiotics and isotretinoin, microbiologic findings showed colonization of pus with CONS in all 3 cases and *Propionibacterium acnes* in 1 case [23].

Whether or not antibiotics work for DC because of anti-inflammatory effects, antibacterial effects, or both, is unclear. The role of bacteria, specifically CONS in DC, remains to be defined as does the exact follicular defect in DC. DC, even more than HS, remains an enigmatic disease with no genes yet identified that are linked. However, the disease can be familial as was reported by Bjellerup and Wallengren in 1990 [33], who noted two brothers aged 20 and 33 with DC; both responded to isotretinoin and had no recurrence 6 months after the therapy was stopped. DC is the least common disease of the diseases of follicular occlusion. The histology of DC is distinct but similar to HS. Its hair pathology is unique among dermatological diseases that affect the scalp. New treatments i.e. TNFB and alitretinoin appear promising in the treatment of patients that fail isotretinoin, without the use of radical surgery or X-ray.

**Table 3 First, Second, Third and Fourth Line Therapies for DC**

| First Line Treatments | Second Line Treatment | Third Line Treatment | Fourth Line |
|---|---|---|---|

| | | | |
|---|---|---|---|
| | | | Treatments |
| High dose zinc sulphate 135mg -220 mg TID for months | **Isotretinon 0.5-1mg/kg for 3 months -12 months** | TFAB i.e. Adalimumab or Infliximab | Radical Surgery with grafting |
| **Ciprofloxacin 250-500mg BID** | Isotretinoin with dapsone 50-100 mg | Alitretinoin | X-ray treatment |
| **Clindamycin and rifampin 300mg each bid** | Rifampin and than isotretinoin | | $CO_2$ laser ablation of scalp[83] |
| Avoidance of pomades | Acitretin with oral prednisone and zinc | | |
| Trimethoprim/sulfamethoxazole DS BID | Other antibiotic combinations followed by isotretinoin | | |
| Minocycline 100mg BID | | | |
| Minocycline with antiandrogens | | | |
| Minocycline with clindamycin | | | |
| Hair removal 1064 nm laser in Fitzpatrick type 3-6 skin | | | |
| Hair removal 694, 800nm laser in Fitzpatrick type 1-2 skin | | | |

**Bold are Preferred.** Duration of all therapies should likely be a given for a minimum of three months. Intralesional Triamcinolone acetate 5-10 mg/kg give every 2-4 weeks (to a total dose of up to 40mg) can be with any 1st, 2nd or 3rd line therapy. Zinc can also be combined with any therapy above.

# Conclusion

Like most dermatological diseases DC is likely a reaction pattern rather than a single entity, which is why different treatments work or fail for patients whose clinical appearance is similar. Again the animating theory to date contained in this article suggests that bacteria, in particular CONS, elicit an aberrant cutaneous immune response. Isotretinoin might normalize the skin and the follicular apparatus to decrease the aberrant cutaneous immune response. It could be that cases of DC that fail isotretinoin are better classified as HS, but taxonomy is tricky, just as clinicopathological correction is needed to separate metastatic Crohn disease from HS. If a follicular occlusion disease occurs on the scalp there is no easy way to distinguish DC from HS except by therapeutic trial. Luckily, new therapies like TNFB, alitretinoin, quinolones, ALA-PDT, 1064 nm laser, and modern external beam radiation therapy might work in cases that fail older therapies. A therapeutic ladder is provided in Table 3.

The incidence of DC is unknown and it is likely under-reported, confused with other entities, or not fulminant enough to attract patients to seek medical care. DC has no ICD-9 code. Of note, ICD-10, used in Europe and soon to be used in the U.S in 2015, codes DC/PCA as L66.3 [68]. DC occurs in populations whose dermatology care is more likely to be provided by non-dermatologists [69]. HS, which has an ICD-9 code (705.83), has an incidence of 0.053%, which certainly underestimates its incidence [70]. HS experts, such as Jemec, think it might occur in 1-3% of the population [71]. Under-diagnosis of DC is reinforced because 2/3 of dermatology care is provided by non-dermatologists [72].

Treatment and definition of DC have advanced beyond X-ray epilation or ablation and radical surgery since Brunsting first linked DC with other diseases of follicular occlusion in 1939 [73]. In the age before isotretinoin, which first came on to the market in middle 1980s, oral corticosteroids, penicillin, and tetracycline were used with some good, but inconsistent effect. Antibiotics or prednisone alone sometimes are able to control the disease, putting it into remission. It is not clear whether antibiotics followed by isotretinoin gives the patient a different clinical course than use of isotretinoin alone. Sometimes the addition of dapsone or clindamycin and rifampin can ameliorate DC when isotretinoin alone cannot.

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