NEWS, VIEWS, & REVIEWS

An Up-to-Date Approach to the Management of Dissecting Cellulitis

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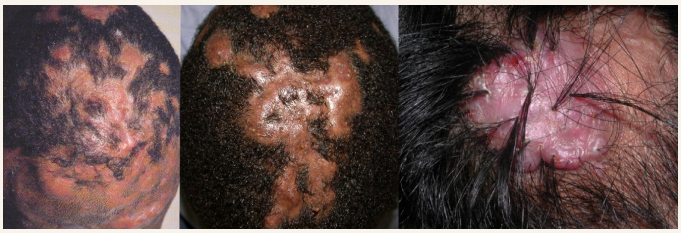
Dissecting cellulitis of the scalp (DCS) also known as perifolliculitis capitis abscedens et suffodiens (PCAS) is a chronic, relapsing, inflammatory dermatosis consisting of edematous sterile pustules and nodules resulting in sinus tracts and scarring alopecia. Although first described as infectious in 1903, DCS is primarily an inflammatory process that can precede a secondary infection; this is similarly true of the other conditions within the follicular occlusion tetrad: hidradenitis suppurativa (HS), acne conglobata, and pilonidal sinus.[1,2]

Figure 1. Dissecting cellulitis of the scalp.



DCS has a greater incidence in black men between the ages of 20 and 40, but it can occur in other demographics, including women and children.[3] DCS has a predilection for the vertex scalp, although the entire scalp can be involved, and produces boggy or fluctuant pustules and nodules Figure 1.[2] Patients commonly complain of pain and of a disfiguring appearance that contributes to the reduction in quality of life. The disease may wax and wane over several years, later producing dermal fibrosis, sinus tracts, and hypertrophic scarring with scarring alopecia. There is an increased risk of squamous cell carcinoma in patients with long-standing disease.[4] The prevalence of DCS is unknown as it is recognized as a rare disease by the National Institute of Health, however research and data on HS indicates a prevalence of 0.3 to 4%, and practice patterns suggest DCS is less common than HS and therefore likely falls on the lower end of that range.[5,6]

The etiology of DCS is undefined, though it certainly is instigated or exacerbated by follicular occlusion, where keratinous plugging of the pilosebaceous apparatus causes dramatic secondary inflammatory changes to proinflammatory stimuli. The follicles in DCS frequently occlude, dilate, rupture, and keratin debris as well as bacteria stimulate relapsing and remitting episodes of inflammatory and infectious nodules, draining abscesses, and ultimately sinus tracts and alopecia. Histology is characterized by infiltration with dense neutrophils, lymphocytes, histiocytes, and plasma cells, as well as granulomas, scarring, and fibrosis seen in later stages. The differential diagnosis includes cysts, furuncles, acne keloidalis nuchae, and folliculitis decalvans, among others.[6] Treatment of DCS is variable depending on severity ranging from topical therapies to systemic monoclonal antibodies, with most patients requiring a multimodal approach as seen in Table 1. This review provides evidence for the treatment of DCS utilizing current literature.

Corticosteroids reduce inflammation in DCS; for mild cases, topical corticosteroids (TCS) like clobetasol, betamethasone, and fluocinolone should be applied twice daily for up to three weeks at a time to avoid long term TCS side effects like hypopigmentation and atrophy. Intralesional triamcinolone acetonide is the standard of care for active DCS lesions. Dosing ranges from 3.33 mg/mL to 10 mg/mL of intralesional triamcinolone, and roughly 0.1 mL to 0.5 mL per lesion.[7] Oral corticosteroids are less commonly prescribed for DCS, and have been shown to be useful at doses up to 50 mg daily and tapered down for flares, or at lower doses as bridging therapy to starting another longer-term treatment option like isotretinoin.[7]

Although no reports specifically for DCS, topical benzoyl peroxide 10% and clindamycin solution 1% daily have shown improvement in HS, another member of the follicular occlusion tetrad with similar pathophysiology. As in HS, benzoyl peroxide and topical clindamycin should be utilized together to prevent colonization with clindamycin resistant bacteria.[8] Oral antibiotics primarily act as anti-inflammatory agents that can also prevent and treat secondary infections caused by DCS, and usually lead to gaps of little to no disease activity without significant flares. Patients anecdotally request antibiotics due to their efficacy, however, clinicians must balance the risk of chronically altering patient's microbiota along with increasing the chance of antibiotic resistance. Case reports indicate success with rifampicin 300 mg twice daily for four months that led to reduction in lesions within weeks prior to starting isotretinoin.[9] Similarly, clindamycin 300mg twice daily also lead to a significant and rapid improvement prior to starting isotretinoin.[10] Combination oral rifampicin 300 mg and clindamycin 300 mg, both twice daily, increases overall efficacy in HS and reduces the risk of antibiotic resistance, and again while not studied in DCS the similar pathophysiology suggests this regimen would be similarly efficacious in DCS[11,12] One case report exists utilizing doxycycline 50 mg every other day in addition to various other therapies for DCS.[13]

Figure 2. Treating dissecting cellulitis of the scalp.

Medication Type	Therapy	Dosing	Reference
Topical	Topical corticosteroids (clobetasol, betamethasone, fluocinolone)	BID for up to 2-3 weeks at a time	Thomas et al[7]
	Prednisone/prednisolone	50 mg taper to 5 mg daily	Thomas et al[7]
	Benzoyl peroxide	10% daily	Thomas et al[7] Nesbitt et al[8]
	Clindamycin solution/gel	1% daily to twice daily	Nesbitt et al[8] Garelli et al[10]
Oral	Doxycycline	20 mg twice daily to 50 mg daily	Di Caprio et al[14] Domonkos et al[13]
	Clindamcyin	300 mg twice daily	Garelli et al[10] Yao et al[12]
	Rifampicin	300 mg twice daily	Georgala et al[9]
	Clindamycin + Rifampicin	300 mg twice daily of each	Ochi et al[11] Yao et al[12]
	Isotretinoin	0.5-1.5 mg/kg/day	Thomas et al[7] Georgala et al[9] Scerri et al[15] Guo et al[17]
	Acitretin	10-20 mg/day	Scheinfeld[3] Jacobs et al[16]
	Zinc	135-405 mg daily	Berne et al[18] Kobayashi et al[19]
Injectable	Intralesional triamcinolone acetonide	3.33 mg-10 mg/cc	Thomas et al[7]
	Adalimumab	40 mg every other week after standard psoriasis loading dose	Navarini et al[20] Martin-Garcia et al[21]
	Secukinumab	150 mg monthly after standard psoriasis loading dose	De Bedout et al[22]
Procedural	5-aminolevulinic acid CO2/ Nd:Yag lasers	3-7 sessions in office procedure at least 2 weeks apart	Thomas et al[7]
	Surgical resection ± skin grafting	Wide local excision, occasionally in stages ± skin grafting	Cuellar et al[6] Hintze et al[23]

Sub-antimicrobial doxycycline (20–40 mg/day) reduces pro-inflammatory cytokines and has been proposed as a safe long term therapy for chronic inflammatory diseases like DCS.[14]

Systemic retinoids, isotretinoin (0.5–1.5 mg/kg/day) and acitretin (10–20 mg/day) have considerable evidence in treating recalcitrant DCS; most patients experienced clinical improvement after a three-month course of isotretinoin and two-month course of acitretin that did not relapse in most patients for multiple years. Treatment for six to eleven months allowed lesions to shrink and resulted in prolonged remission.[3,7,15,16] The overall efficacy of isotretinoin in treating dissecting cellulitis of the scalp was estimated at 0.9 with a 95% confidence interval (0.81–0.97). Recurrence was seen in 24% (6/25) of patients.[17] Oral zinc has also been used as it known to downregulate Nuclear Factor Kappa B (NF-κB) leading to a reduced inflammatory response. Doses of 135–405 mg daily in multiple case reports demonstrated significant improvement in DCS after twelve weeks.[18,19] TNF inhibition with adalimumab (40 mg q2weeks) and infliximab (5 mg/kg q8weeks) both resulted in disease remission within eight weeks that lasted well over a year in most cases.[20,21] One case report indicated that ten weekly doses of secukinumab 150 mg followed by switching to monthly dosing resulted in a durable response seen only after four weeks.[22]

Many patients with DCS have limited regions of the scalp affected, which respond well to traditional medical therapies. For severe cases, areas of the affected scalp may require wide local excision and primary closure or coverage with split-thickness

skin grafting. Multiple reports indicate that surgery achieved both disease remission and excellent aesthetic outcomes in patients who chose this option, however permanent alopecia results in the affected areas.[6,23] Aside from surgery, treatment with ND:Yag lasers, ablative lasers, and photodynamic therapy with 5-aminolevulinic acid have all been reported with varying levels of success, however, each modality currently has very little evidence to support treating DCS.[7]

Each case of DCS is distinct and individualized treatment depends on severity, and more importantly a patient's willingness to treat with combination therapies given what is required to achieve improvement based on the literature. Before considering treatment options, patients' motivation to take oral pills, injections, lasers, and surgery should be assessed. Given there are no FDA approved therapies for DCS, initially recommending zinc, prescribing topical benzoyl peroxide and clindamycin, and oral antibiotics are pragmatic with the consideration of the increased administrative effort required to start oral retinoids or injectable monoclonal antibodies. In addition, some severe DCS patients can benefit from oral steroids initially as a bridge treatment while preparing to start isotretinoin/acitretin and or adalimumab/infliximab. Clinicians and patients should be mindful of antibiotic usage and switch to longer term options like zinc, oral retinoids, or monoclonal antibodies when appropriate. Surgery should be reserved for severe cases and surgical patients should ideally remain on some medical therapy as well. Ultimately, while numerous options exist for DCS and the other members of the follicular occlusion tetrad, further research and clinical trials are needed to supplement the existing evidence.

Disclosure
The authors declare no conflicts of interest.

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