Dissecting cellulitis (DSC) after interferon beta-1a treatment and scalp trauma

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Dissecting cellulitis (DSC) is a chronic inflammatory disease of hair follicles. Affected patients develop painful scalp nodules and sinus tracts. Although the precise cause is unknown, it is proposed that collapse of the hair follicle wall secondary to defective follicular support plays a role not only in DSC but also in conditions that make up the so-called follicular occlusion tetrad: DSC, hidradenitis suppurativa (HS), acne conglobata, and pilonidal cysts.[1]

Altered innate and adaptive immune responses may also be involved in the pathogenesis of DSC and HS.[2] In HS, increased levels of tumor necrosis factor (TNF)-alfa, interleukin (IL)-10, IL-1β, and IL-17a have been documented. Treatments that inhibit the action of TNF-alfa, such as infliximab and adalimumab, are helpful for many patients with DSC and HS.[3-5] How other immune mediators affect these diseases is not well understood.

Here we present a case of DSC developing at site of trauma in a patient receiving interferon beta-1a therapy for multiple sclerosis. This case provides further insight into the possible pathogenesis of DSC.

Abbreviations used:

DSC:	dissecting cellulitis
HS:	hidradenitis suppurativa
IFN:	interferon
IL:	interleukin
TNF:	tumor necrosis factor

CASE REPORT

A 25-year-old man of Middle Eastern descent presented with a 7-month history of scalp nodules, abscesses, and sinus tracts with purulent discharge. Affected areas were predominantly left-sided and the area of involvement had slowly increased in size over time (Fig 1). Biopsy specimen confirmed the diagnosis of DSC.

Further history revealed that the scalp papules and papulonodules first developed at the site of a previous traumatic injury to the scalp. The patient was in a car accident 7 years prior and at that time required scalp stitches. The area, however, healed completely with no residual pain, itching, redness, discharge, or obvious hair loss. Medical history included a 3-year history of multiple sclerosis. Interferon beta-1a (Rebif, EMD Serono, Mississauga, ON) subcutaneous injections were started 6 months before the development of DSC. The patient was also a smoker.

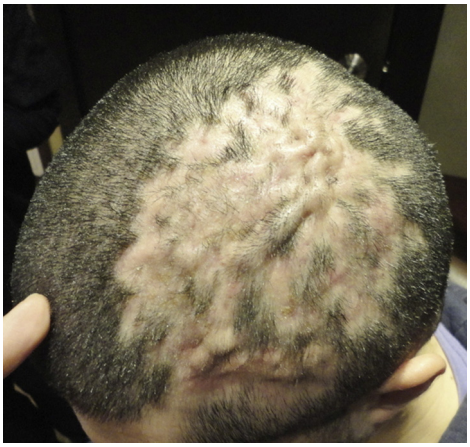

Fig 1. Dissecting cellulitis at the site of previous scalp injury in a patient receiving interferon beta-1a treatment.

Scalp swabs were negative for bacteria. The patient partially responded to treatment with

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isotretinoin 1 mg/kg. Further reduction and flattening of the lesions was achieved with an 8-week tapering course of prednisone (40 mg to 0 mg, reducing 5 mg per week) and cephalexin.

DISCUSSION

DSC is an uncommon suppurative scarring alopecia that typically affects young men. Although the precise pathogenesis of DSC is unknown, it is hypothesized to be related to follicular occlusion, secondary infection, and deep-seated inflammation. Of all the cytokines and chemokines studied in HS and DSC, the best understood role is for TNF-alfa; TNF-inhibitory medications are helpful in both DSC and HS.[3,4]

The short duration between initiation of interferon beta-1a therapy and the development of DSC supports a possible association. Interferon (IFN) beta plays an important role in the innate immune system. It functions as a counter-regulatory cytokine and opposes the production of proinflammatory cytokines. For example, IFN beta inhibits TNF-alfa secretion and this would likely be beneficial to inhibit the development or progression of DSC. However, IFN also enhances IL-10 production, which would theoretically be detrimental and promote development of DSC-type lesions.[5] IFN may also impair neutrophils, which would be significant given that impaired neutrophilic function may be contributory to the pathogeneis.[6] There have been anecdotal reports submitted to the US Food and Drug Administration of HS-type conditions developing in patients using various types of interferon beta-1a therapy[7,8]; however the strength of these associations is not clear, nor are the precise details of each of the cases. DSC has not been previously reported with interferon beta-1a therapy to our knowledge.

Further study is needed to determine if alterations in the immune system induced by interferon beta-1a are associated with follicular occlusion in predisposed patients. It appears that the specific immune system changes that occur in patients with multiple sclerosis are not commonly associated with the entities of the follicular occlusion tetrad. To the author's knowledge, DSC has not been reported with MS. However, HS has been reported in association with multiple sclerosis in 1 report.[9]

The development of DSC precisely at the site of the patient's traumatic injury suggests a role for trauma. However, the role of trauma in the pathogenesis of DSC is unknown. It appears, at least for HS, that reducing trauma is important in preventing new lesions. For the other entities in the follicular occlusion tetrad, trauma does not appear to be a major mechanism in the pathogenesis of these conditions.

The patient's smoking may also play a role in his susceptibility to DSC. Nicotine may inhibit normal glandular duct secretion and promote epithelial hyperplasia and is hypothesized to play a role in DSC and HS.[10]

Taken together, it is possible that interplay between interferon beta-1a therapy and smoking on a background of multiple sclerosis favored the development of DSC at the site of trauma. As we gain more experience with the use of interferon beta-1a, it will be important to evaluate if any of the entities that comprise the follicular occlusion tetrad become increasingly reported.

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