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Clinical Pearls

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Seborrheic dermatitis

By Raj Chovatiya, MD, PhD, MSCI, FAAD

- 1. It's technically a form of eczema. Seborrheic dermatitis (SD) is often described as existing on the psoriasis spectrum of inflammatory dermatoses given cases of clinical overlap, particularly on the scalp and face (so called 'sebopsoriasis'). However, SD is technically considered to be a form of chronic eczema, a family of disorders that also includes atopic dermatitis. Clinical findings of SD (i.e., erythema, flaky scales, and pruritus) can resemble aspects of both eczematous and psoriasiform eruptions. Histopathology may similarly show overlapping features. Acute SD demonstrates spongiosis and superficial perivascular and perifollicular lymphocytic infiltrate (akin to eczematous dermatoses), while more chronic lesions can show irregular acanthosis and focal parakeratosis (more akin to psoriasiform dermatoses). At an immunologic level, SD can demonstrate immune skewing and activation consistent with both eczema (type 2 helper T [Th2] cells) and psoriasis (Th17 cells). Unlike either family of disease, SD uniquely demonstrates a predilection primarily for areas rich in sebaceous glands, as well as intertriginous areas.
- 2. It's not all about the yeast. Since the earliest descriptions of SD, Malassezia furfur (Pityrosporum ovale) has been suspected by some to be the causative etiology. Evidence supporting a primary causative role of yeast in SD includes the distribution of *M. furfur* in highly sebaceous areas, increased density in lesional skin, its contribution to production of inflammatory sebum-derived metabolites, and clinical efficacy of antifungal therapy. The debate continues to this day, and while M. furfur is acknowledged to have a role in pathogenesis, SD is not considered to be an "infectious" process directly driven by yeast. Evidence supporting a more secondary role for yeast in SD pathogenesis includes ubiquity of M. furfur both on healthy skin and healthy individuals, lack of strong correlation between density and either disease presence or severity, and efficacy of primarily anti-inflammatory therapies (like topical corticosteroids and topical calcineurin inhibitors). Emerging data suggest that immune dysregulation and barrier dysfunction may play more important and central roles in SD than previously appreciated, which is supported by clinical data from the newly approved roflumilast 0.3% foam (a novel topical phosphodiesterase-4 inhibitor).
- **3.** Patients experience a greater disease burden than one might expect. SD can be viewed by some (inaccurately) as a secondary issue, benign entity, and/or non-burdensome condition. However, a closer look at

- patient-centered data reveals significant disease-related emotional distress and perceived physical limitations perhaps unsurprising given that SD primarily involves highly visible and cosmetically sensitive areas. Two decades ago, the U.S. combined direct and indirect cost associated with SD was calculated to be nearly a quarter of a billion dollars. However, in reflection of the major psychosocial and quality of life (QoL) burden imposed by the disease, patient willingness to pay for symptomatic relief totaled \$1.2 billion. A recently presented population-based survey of SD patients and clinicians revealed that most SD patients experienced significant mental health impact, had negative QoL impact that was underestimated by clinicians, needed nearly six different treatments per week to manage their symptoms and weren't satisfied with common treatment options, and even felt they would be further along in their career if they didn't have SD.
- 4. Dandruff may be one of several different clinical phenotypes. Dandruff of the scalp is characterized by flaking of the scalp (with or without itch) and commonly referred to as pityriasis sicca. Accompanied by minimal to no clinical signs of inflammation, dandruff is considered by many to be mildest entity on one end of the SD spectrum — though this is yet another debated aspect of SD. Despite limited epidemiology, the prevalence of SD is estimated to be approximately 1-5%, and when including dandruff, this number reaches nearly half the population. Other scalp phenotypes to be aware of with increasing disease severity on this spectrum include pityriasis steatoides ("classic" SD characterized by orange-pink erythema and yellow greasy scales) and pityriasis amiantacea (characterized by concretions of scale around the hair shaft). Beyond the scalp, SD can also commonly involve the forehead, eyebrows, eyelids, nasolabial folds, cheeks, ears, postauricular folds, beard area, neck, and trunk. Distinct patterns include intertrigo, petaloid (polycyclic plaques), seborrheic eczematids (annular plaques with central clearing), pityriasiform, and psoriasiform.

References:

- 1. Borda LJ, Wikramanayake TC. J Clin Investig Dermatol. 2015;3(2).
- 2. Dessinioti C, Katsambas A. Clin Dermatol. 2013;31(4):343-351.
- Prohic A, Jovovic Sadikovic T, Krupalija-Fazlic M, Kuskunovic-Vlahovljak S. Int J Dermatol. 2016;55(5):494-504.
- 4. Pärna E, Aluoja A, Kingo K. Acta Derm Venereol. 2015 Mar;95(3):312-6
- Bickers DR, Lim HW, Margolis D, et al. J Am Acad Dermatol. 2006:55(3):490-500.
- Chovatiya R, Aldredge L, Heath C, et al. J Am Acad Dermatol. 2023 Sep;89(3):AB71.DR

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