18724

Tolerance and subject satisfaction of an over the counter colloidal oatmeal (*Avena sativa*) lotion in patients with psoriasis and sensitive skin

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Psoriasis is an autoimmune skin disease that is associated with significant impairment in quality of life including skin discomfort and dissatisfaction of skin appearance. The purpose of this study was to determine the tolerability and satisfaction in skin appearance of a 1% colloidal oatmeal lotion on individuals with psoriasis. Sixty women with psoriasis and self-reported sensitive skin were enrolled into the study. Subjects applied only the assigned 1% colloidal oatmeal lotion to their entire body at least once per day for 4 weeks. Subjects assessed their skin condition on a 100-point VAS and completed product-satisfaction and Quality of life questionnaires at baseline and after 4 weeks of use. After 4 weeks of application, subjects perceived a significant improvement in each of the skin parameters queried including dryness, desquamation, roughness, smoothness, softness, suppleness, redness, discomfort, and itching with an average of 38% improvement over all the parameters. The greatest improvements included itch and desquamation with a 45% improvement over baseline scores for both. More than 96% of subjects thought the product helped to reduce both the severity and number of patches on the body. In addition, 100% of subjects felt the product was suitable to use daily for their skin type. The product was well tolerated and no product related skin adverse events were reported during the study. In conclusion, the over-the-counter colloidal oatmeal moisturizing lotion improves the skin appearance and discomfort associated with psoriasis and is a suitable adjunctive moisturizer for daily skincare in this patient population.

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18763

Validation of case identification for alopecia areata using International Classification of Diseases (ICD) coding

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The aim of this study was to assess the validity of the International Classification of Diseases (ICD) 9 and 10 codes for the identification of alopecia areata (AA) in a large clinical database. A multicenter retrospective review was performed at Columbia University Irving Medical Center and Brigham and Women's Hospital to determine whether patients with an ICD-9 code (704.01, Alopecia Areata) or ICD-10 codes (L63.0, Alopecia Totalis; L63.1, Alopecia Universalis; L63.2, Ophiasis; L63.8, Other Alopecia Areata; L63.9, Alopecia Areata, Unspecified) met diagnostic criteria for AA. Out of the 880 charts reviewed, 97.5% had physical exam findings consistent with AA and the diagnosis was unequivocal in 90% of charts. AA was diagnosed by a dermatologist in 87% of the charts. The positive predictive value of the ICD-9 code 704.01 for AA was 248/255 (97%), while the positive predictive value for the ICD-10 codes for AA were 75/118 (64%) for L63.0, 130/151 (86%) for L63.1, 1/2 (50%) for L63.2, 81/89 (91%) for L63.8, and 247/265 (93%) for L63.9. Overall, 782/880 (89%) of the patients with a single ICD-9 or 10 code for AA were determined to have a true diagnosis of AA. Patients whose medical records contain an ICD code associated with AA have a high probability of having the condition. This data can be used to support the identification of AA cohorts among clinical databases to determine disease burden and associated comorbidities.

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18745

Neurotoxin impurities and flagellin: A threat to efficacy

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Botulinum neurotoxin A (BoNT/A) products recently introduced for medical esthetic use, including Nabota/Jeuveau, Meditoxin/Neuronox, and Botulax, contain nontoxin accessory proteins (NAPs) and excipients. Our objective is to show that there is scarce, publicly-available, clinical evidence of these toxins' purity, potential immunogenicity or their links to treatment failures. This study aimed to review the issues surrounding BoNT/A-induced immunogenicity and antibody-induced treatment failures. We examined reports on the immunogenicity of BoNT/A and its complexing proteins, and the impact of toxin-specific (eg contaminating antigenic and accessory proteins) and treatment-related factors (eg total toxin dose and injection frequency). We discuss multiple patients who presented to a single clinic for resolution of previous treatment failures caused by the use of toxins containing complexing proteins elsewhere. It is important to understand a toxin's true composition and the justifications for the presence of any non-toxin elements or high neurotoxin quantities in some formulations, which increases the risk of neutralizing antibody (NAB) formation and treatment failures. We highlight the inadequate clinical evidence supporting the recently introduced toxins, as well as their potential antigenic protein loads and the impacts of these entities on NAB formation. Our patient cases illustrate an increasing and worrying incidence of such treatment failures. If suboptimal clinical outcomes occur, physicians must consider the possibility of immunogenicity. The most prudent approach is to prevent NAB formation and treatment nonresponsiveness from the start by using only formulations with no adjuvant proteins and only the active neurotoxins without inactive components, such as incobotulinumtoxinA.

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18781

A familial case of white sponge nevus and review of the literature

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White sponge nevus (WSN) is an autosomal dominant skin disorder characterized by white corrugated and diffuse plaques mainly affecting the non-keratinised stratified squamous epithelium. Although WSN is usually seen in the oral mucosa, other sites, such as the nasal, esophageal, laryngeal, and anogenital mucosa, may also be involved. We describe a case a 11-year-old girl who presented with asymptomatic rough, diffuse, white patches in the bilateral buccal mucosa gingiva with extension to the esophagus that was evident on esophageal gastroduodenoscopy. A biopsy of esophageal tissue confirmed the presence of WSN. Her father had a similar biopsy-proven but more widespread appearance in the buccal mucosa and esophagus as well. WSN is caused by germline variants of the keratin genes KRT4 or KRT13, located, respectively, at chromosomes 12q13 and 17q21-q22. Extra-oral affected sites have been shown to arise only from KRT13 variants. It is important to recognize this benign entity and to differentiate it from other acquired causes of intra-oral white plaques such as leukoplakia, candidiasis and lichen planus. A thorough history and physical examination would help to exclude other genodermatoses like pachyonychia congenita, hereditary benign intraepithelial dyskeratosis, Darier disease, and dyskeratosis congenita.

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