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REVIEW

Cutaneous Symptoms of Cystic Fibrosis: A Narrative Review

Michał Piątkiewicz[1], Barbara Olędzka[1], Anna Wołkowicz[2], Aneta Durmaj[1], Lidia Rudnicka[1]

[1]Department of Dermatology, Medical University of Warsaw, Warsaw, Poland; [2]Cystic Fibrosis Centre, Paediatric Hospital, Dziekanów Leśny, Poland

Correspondence: Lidia Rudnicka, Department of Dermatology, Medical University of Warsaw, Nowogrodzka 59, Warsaw, 02-006, Poland, Tel +48 22 5021324, Email lidia.rudnicka@wum.edu.pl

Abstract: Cystic fibrosis (CF) is caused by a mutation in the Cystic Fibrosis Transmembrane Conductance Regulator (CFTR) gene and is characterized by recurrent pulmonary infections, fat malabsorption, and malnutrition. CF is associated with diverse dermatologic symptoms, including aquagenic palmoplantar keratoderma, nutrient deficiency dermatitis, and vasculitis. Sporadically, these manifestations may represent the initial symptoms of cystic fibrosis. Drug-induced skin reactions are commonly observed in patients with cystic fibrosis (pwCF) due to the chronic use of antibiotics. Therapy with CFTR modulators is considered the gold standard in the treatment of cystic fibrosis. Nevertheless, they are associated with numerous side effects, ranging from rash to Stevens-Johnson syndrome. Identifying the cutaneous manifestations of cystic fibrosis may support comprehensive care for patients. In this regard, dermatologists may play an important role in the management of associated skin complications.

Keywords: cystic fibrosis, CFTR, aquagenic palmoplantar keratoderma, nutrient deficiency dermatitis, vasculitis, antibiotic hypersensitivity

Introduction

Cystic fibrosis (CF) is the most common recessive genetic disorder in the Caucasian population.[1] It is associated with a mutation in the cystic fibrosis transmembrane conductance regulator (CFTR) gene which encodes the ion channel responsible for chloride exchange across the cell membrane. CFTR is expressed on the surface of epithelial cells in the secretory glands of various organs, primarily the respiratory tract, paranasal sinuses, pancreatic ducts, and sweat glands.[2] The most common symptoms of cystic fibrosis include recurrent pulmonary and paranasal sinus infections, steatorrhea, malnutrition, male infertility, and elevated chloride level in sweat.[3] Since 2009, a newborn screening program (NBS) for cystic fibrosis (CF) has been implemented in Poland. Following the introduction of NBS for CS across the country, the diagnosis of CF is primarily established based on an abnormal screening result. However, the test does not eliminate the need for continued clinical vigilance, as cases of "missed" diagnoses may still occur- affecting up to 5% of newborns.[4] In addition, patients with cystic fibrosis (pwCF) frequently develop dermatological manifestations, such as aquagenic palmoplantar keratoderma, nutrient deficiency dermatitis, and vasculitis. The objective of this review is to summarize the dermatological manifestations of cystic fibrosis based on the available literature. Furthermore, cutaneous adverse effects of both antibiotics and CFTR modulators in patients with cystic fibrosis have been described.

Aquagenic Palmoplantar Keratoderma

Aquagenic palmoplantar keratoderma (APK), also known as aquagenic wrinkling of the palms or transient reactive papulotranslucent acrokeratoderma is a condition characterized by the development of oedematous, whitish or yellowish papules and hyperwrinkling of the palms after a few minutes of water exposure (Figures 1 and 2). This phenomenon may present clinical symptoms including pruritus, burning, and sensory disturbances. It is estimated that APK occurs in 44–80% of pwCF and in up to 25% of heterozygous carriers.[5] Any patient with a positive APK sign should be evaluated for CF. It is essential to determine whether there is a family history of CF or if the patient exhibits clinical manifestations

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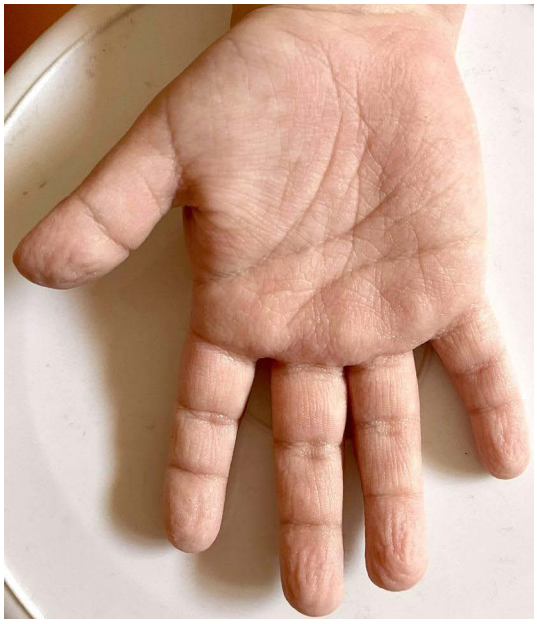

Figure 1 Aquagenic palmoplantar keratoderma caused in a 5-year-old girl with CF by 3-minute immersion in room temperature water.

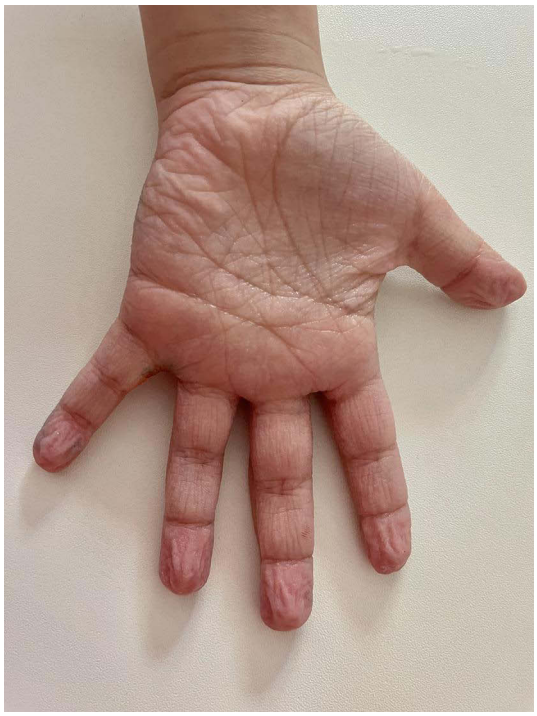

Figure 2 Aquagenic palmoplantar keratoderma caused in a 6-year-old boy with CF by 1.5-minute immersion in room temperature water.

typical of the disease, such as nasal polyps, finger clubbing or pancreatic insufficiency.[6] Typically, histology shows spongiosis of the stratum corneum, orthohyperkeratosis, eccrine acrosyringia dilation, acanthosis, and increased capillary proliferation around sweat glands.[7] The underlying pathophysiology is believed to involve increased transepidermal water loss due to the secretion of hypertonic sweat.[5] It is also suggested that a mutated CFTR channel contributes to the abnormal expression of aquaporin-5 and the dysregulation of the transient receptor potential vanilloid (TRPV) 4 channel, thereby leading to impaired water transport through the epidermis.[8,9] The management of APK includes topical

medications. Therapy with aluminum-based topicals should be considered the first-line treatment such as 20% aluminum chloride. Other therapeutic options include 20% salicylic acid, mometasone furoate ointment and 10% urea cream, as well as botulinum toxin injections.[5]

Cystic Fibrosis Nutrient Deficiency Dermatitis

Cystic fibrosis nutrient deficiency dermatitis (CFNDD) is a rare clinical manifestation resulting from deficiencies in various nutrients, including zinc, essential fatty acids (EFAs), fat-soluble vitamins, and protein. Symptoms typically appear between 2 weeks to 6 months after birth. Before the implementation of newborn screening for cystic fibrosis, CFNDD was often the first clinical indication suggesting specialists consider this diagnosis.[6,10] CFNDD initially manifests in the diaper area, later extending to the periorbital and perioral regions, presenting first as papules and subsequently as burnished plaques.[11,12] This clinical presentation is frequently observed in malnourished patients.[13] Severe immune dysfunction may develop at a later stage.[14] Zinc deficiency is characterized by a rash resembling acrodermatitis enteropathica (AE), initially affecting the buttocks, perianal region, genital area, and mouth, before spreading to other parts of the body.[13,14] It is primarily caused by an impairment in the secretion of pancreatic enzymes into the duodenum.[15] Despite a similar clinical presentation, AE differs from CFNDD. While in AE, zinc supplementation alone is sufficient for complete symptom resolution, in CFNDD, pancreatic enzyme supplementation is also required.[16] Zinc levels do not always accurately reflect the deficiency; in some patients, zinc levels remained within the normal range during symptom onset and only decreased after several weeks.[14,16] The zinc content in human milk declines sharply after three months of breastfeeding, correlating with the timing of symptom onset in pwCF. Therefore, zinc supplementation is crucial in this population.[11] Deficiency of essential fatty acids (EFAs) occurs relatively rarely, most commonly in pwCF who are not receiving treatment.[17] The symptoms, manifesting as a psoriasis-like rash, appear in the same areas as those observed in zinc deficiency. It is characterized by a reduction in the serum level of linoleic acid (LA), a precursor of arachidonic acid (AA) and alpha-linolenic acid (ALA), a precursor of docosahexaenoic acid (DHA), coupled with an elevation in the concentrations of stearic and oleic acids.[14] The altered concentration ratio of these acids leads to disturbances in cell membrane formation, as the incorporation of alpha-linolenic acid into the membrane is reduced, thereby resulting in an intensification of pro-inflammatory processes manifested as dermatitis.[10,18,19] AA and DHA are also crucial for the development of nervous tissue.[10] Protein deficiency represents the most severe of the previously mentioned deficiencies and is a highly unfavorable prognostic indicator, as it signifies severe pancreatic insufficiency.[14,20] It manifests similarly to kwashiorkor, characterized by fluid retention leading to edema, anemia, and dry skin with areas of hypopigmentation in the perineal, inguinal, and extremity regions as well as behind the auricles, contributing to the overall CFNDD presentation.[21,22] Less frequently, alopecia, hepatomegaly, and hair depigmentation occur.[12,23] In pwCF, deficiencies in taurine and tyrosine are very common. Both amino acids participate in critical processes: taurine plays a role in wound healing by influencing cell proliferation and collagen synthesis, while tyrosine is involved in the synthesis of skin and hair pigments.[24,25] After the institution of an appropriate nutritional protocol, pancreatic enzyme replacement, and supplementation with zinc, linoleic acid, taurine, and tyrosine, CFNDD typically resolves within 2 weeks.[6]

Vasculitis

Vasculitis is a rare phenomenon, affecting 2–3% of pwCF. It may be limited to a single organ, most commonly the skin or present in a systemic form. Most pwCF who develop systemic vasculitis are adolescents.[26] It predominantly involves small and medium-sized cutaneous arteries,[27] but venules, capillaries, and large vessels may also be affected.[28] Clinically, vasculitis manifests as purpuric macules or papules, most frequently on the lower extremities, and typically occurs concomitantly with exacerbations of pulmonary symptoms.[29] Additionally, patients may experience joint swelling and arthralgia.[30] In some cases, erythema nodosum may also develop.[31] Histological examination reveals features characteristic of leukocytoclastic vasculitis, including perivascular or interstitial inflammatory cell infiltrates, immune complex deposition in vessel walls, and cellular debris.[26] The pathophysiology of this phenomenon remains unclear; however, bacterial colonization and deposition of immune complexes are major contributing factors. Chronic inflammation predisposes to the formation of antineutrophil cytoplasmic antibodies (ANCA), which form immune complexes and

subsequently lead to vascular damage.[26] Moreover, it is believed that the administration of antibiotics such as penicillins or cephalosporins may contribute to the formation and deposition of immune complexes via a type III hypersensitivity mechanism.[32] Another important factor in the development of vasculitis in pwCF is a reduced lipoxin A4 (LXA4) to the neutrophil ratio. LXA4 is responsible for suppressing the immune response, and its diminished concentration in these patients results in the induction of an inflammatory state.[33] The treatment of vasculitis in cystic fibrosis should primarily focus on identifying the etiologic agent of the infection.[10] Topical steroids in combination with oral steroids, dapsone, or colchicine are considered first-line treatments for CF-associated vasculitis.[34] In case of recurrent vasculitis, it is recommended to use immunosuppressive drugs such as prednisolone in monotherapy or in combination with azathioprine, methotrexate or mycophenolate mofetil.[26] It should be noted that the use of these agents is associated with an increased risk of infection and/or malignancy, both of which are already elevated in pwCF.[35]

Antibiotic Hypersensitivity

Due to frequent and recurrent infections associated with CF, patients with this condition are exposed to prolonged and repeated antibiotic therapy. Colonization with bacteria such as *Pseudomonas aeruginosa* leads to chronic infections that often require long-term antibiotic treatment during exacerbations. Drug hypersensitivity occurs in approximately 30% of pwCF. These patients are particularly susceptible to β-lactam antibiotic-induced hypersensitivity, which occurs three times more frequently than in the general population.[10,36] The increased frequency of drug hypersensitivity in pwCF is likely attributed to the number of cumulative exposures to antibiotics, which appears to be the most significant risk factor, as well as the intravenous route of drug administration, higher rates of atopy, and elevated circulating immune complexes.[10,36,37] While increasing age and prolonged duration of *P. aeruginosa* colonization have been identified as risk factors, sex and CFTR genotype do not appear to influence the risk of drug hypersensitivity in pwCF.[36] β-lactam antibiotics are responsible for most hypersensitivity reactions, likely due to their high immunogenicity and frequent use in pwCF.[37] Piperacillin is the most common culprit, accounting for 33% to 50% of intravenous antibiotic-induced hypersensitivity reactions. Other antibiotic classes frequently used in CF, such as aminoglycosides, are generally better tolerated, with hypersensitivity reactions occurring less frequently.[10] The mechanism of hypersensitivity reactions in pwCF does not significantly differ from that observed in the general population. Delayed allergic reactions, such as morbilliform rash, are relatively common.[10] Cutaneous reactions account for 98% of all hypersensitivity reactions to antibiotics.[36] The most reported symptoms include pruritus, exanthema, and urticaria, though angioedema, morbilliform rash may also occur.[10,36] Although antibiotic hypersensitivity reactions in CF patients are typically mild and confined to the skin, severe and life-threatening reactions, such as Stevens–Johnson Syndrome (SJS), Toxic Epidermal Necrolysis (TEN), and Drug Reaction with Eosinophilia and Systemic Symptoms (DRESS), may occasionally occur.[6,10,36] The management of antibiotic hypersensitivity reactions in pwCF does not significantly differ from that in the general population. However, assessing the risks and benefits of continuing antibiotic therapy in these patients can be particularly challenging for clinicians.

Cutaneous Reactions to CFTR Modulators

CFTR modulators are relatively new drugs that outperform other therapies in the treatment of CF. Elexacaftor-tezacaftor-ivacaftor (ETI) is a triple-combination CFTR modulating treatment. This therapy significantly improves lung function, quality of life, reduces pulmonary exacerbations, and decreases mortality. The mechanism of action of CFTR modulators is based on two key processes: potentiators such as ivacaftor bind to the CFTR channel, causing it to remain open for a longer period, while correctors such as elexacaftor, tezacaftor or lumacaftor improve CFTR protein folding and facilitate their transport to the cell membrane. Triple therapy with ETI is currently considered the gold standard in the treatment of cystic fibrosis.[37] Nevertheless, CFTR modulators are associated with dermatologic side effects. ETI is the most frequently linked to skin reactions and the most common manifestation is a maculopapular rash, which is observed more frequently in women than in men and even more frequently in patients using contraceptives.[38] The less common adverse effect is the development of acne. There is no standard approach to the treatment of acne for pwCF. The use of isotretinoin is controversial due to the risk of pulmonary exacerbations and liver failure. The American Academy of Dermatology recommends topical therapy, which may include retinoids, antibiotics, and benzoyl peroxide, either as

monotherapy or in combination therapy. Systemic therapy may be indicated for moderate to severe cases that do not respond to topical treatment.[39]

Conclusions

CF is associated with diverse dermatologic symptoms, including aquagenic palmoplantar keratoderma, nutrient deficiency dermatitis, and vasculitis. In certain cases, these cutaneous manifestations may precede other systemic signs and serve as important early diagnostic clues, particularly in the small proportion of affected infants not identified by newborn screening. Therefore, awareness of them can be useful in the differential diagnosis of the disease. The presence of these cutaneous changes may correlate with disease activity. Drug-induced skin reactions are commonly observed in pwCF due to the chronic use of antibiotics. Although CFTR modulators are currently the gold standard in the treatment of CF, their use is associated with the risk of dermatological side effects. Nowadays, effective management of CF should be based on a multidisciplinary approach. The dermatologist's role is to prevent and treat skin complications of the underlying disease.

Data Sharing Statement

The data that supports the findings of this study are available from the corresponding author upon reasonable request.

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The authors have no conflicts of interest to declare for this work.

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