MICRO LABS LIMITED

COSMODERMA TIMES

Volume 2 | Issue 12|2023

Dear Doctor,

Greetings from Micro Labs. We are happy to bring you "COSMODERMA TIMES" which contains latest and interesting news in the field of Dermatology.

This issue contains:

Should you require any article or, medical information, please feel free to contact us medicalservices@microlabs.in

Looking forward to your valuable feedback.

Best Regards,

Medical Team, Micro Labs Limited.

Determination of the characteristics of contact sensitization in allergic contact dermatitis (ACD) patients

Introduction:

Atopic dermatitis (AD) is a relapsing inflammatory skin disease that is characterized by recurrent pruritus and a characteristic age-related lesion distribution.[1] Allergic contact dermatitis (ACD) is a T-cell mediated delayed-type (type IV) hypersensitivity reaction in response to an exogenous substance that has been investigated using patch testing, the gold standard diagnostic method.[2] It is difficult to distinguish between AD and ACD where both exhibit similar histological characteristics, such as epidermal spongiosis and superficial perivascular inflammatory cell infiltration. There has been some dispute on whether people with atopic dermatitis (AD) have a higher prevalence of contact allergen sensitivity. Increased exposure to topical medicines and moisturizers, as well as compromised skin barrier function, enhance the risk of contact sensitization, but the Th2-skewed inflammatory pathway of AD is linked with a lower risk.[1] The purpose of this study was to identify the features of contact sensitization in individuals with allergic contact dermatitis (ACD) who have a current or previous history of AD.

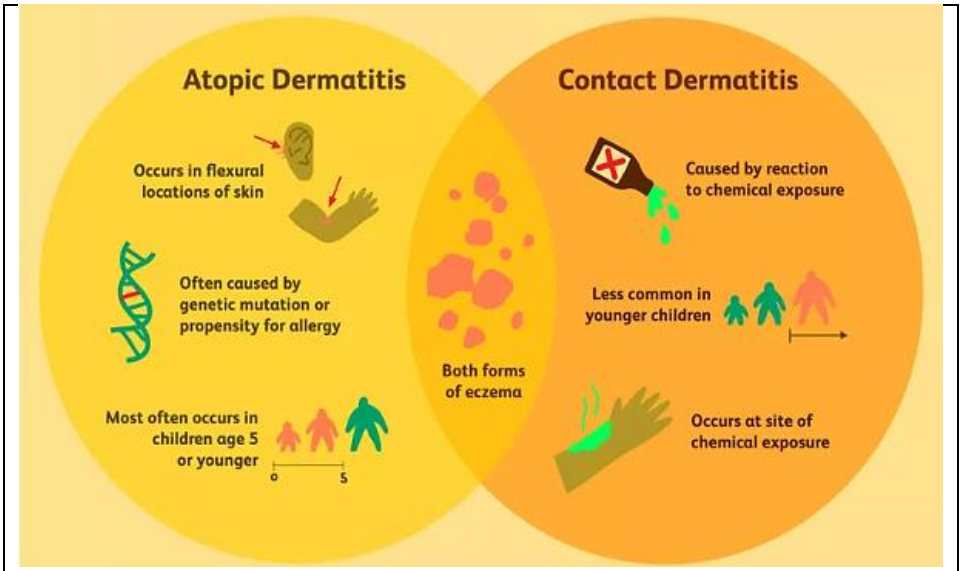


Figure 1. Similarities and differences between atopic and contact dermatitis

Methods:

This retrospective analysis included 515 individuals who were patch-tested and suspected of having ACD. Patch testing was carried out in accordance with the International Contact Dermatitis Research Group (ICDRG)/European Society of Contact Dermatitis (ESCD) recommendations. The frequencies of contact sensitization among ACD patients with and without AD were examined in this study. Statistical analyses were carried out with IBM SPSS

26.0, utilizing Pearson's chi-square test and Fisher's exact test for binary variables between patients with and without AD.

Results:

A total of 515 patch test results were examined and classified into two groups: those with AD (n = 53) and those without AD (n = 462). Compared to the non-AD group (72.9%), the AD group had lower any-allergen positivity (1+, 2+, or 3+) (56.6%) (p=0.013). The AD group had a considerably greater budesonide positivity rate (p=0.011), but the non-AD group had a higher frequency of positive results for balsam of Peru (p=0.036). Nickel (II) sulfate hexahydrate (26.4%), cobalt (II) chloride hexahydrate (24.5%), potassium dichromate (9.4%), budesonide (5.7%), and thimerosal (5.7%) were the most prevalent reactions in the AD group. Nickel (II) sulfate hexahydrate (38.1%), cobalt (II) chloride hexahydrate (27.5%), potassium dichromate (13.9%), Myroxylon pereirae resin (Peruvian balsam) (11.0%), thimerosal (10.8%), and scent mix I (8.0%) were the most frequently occurring sensitizing antigens in the non-AD group. (Figure 2)

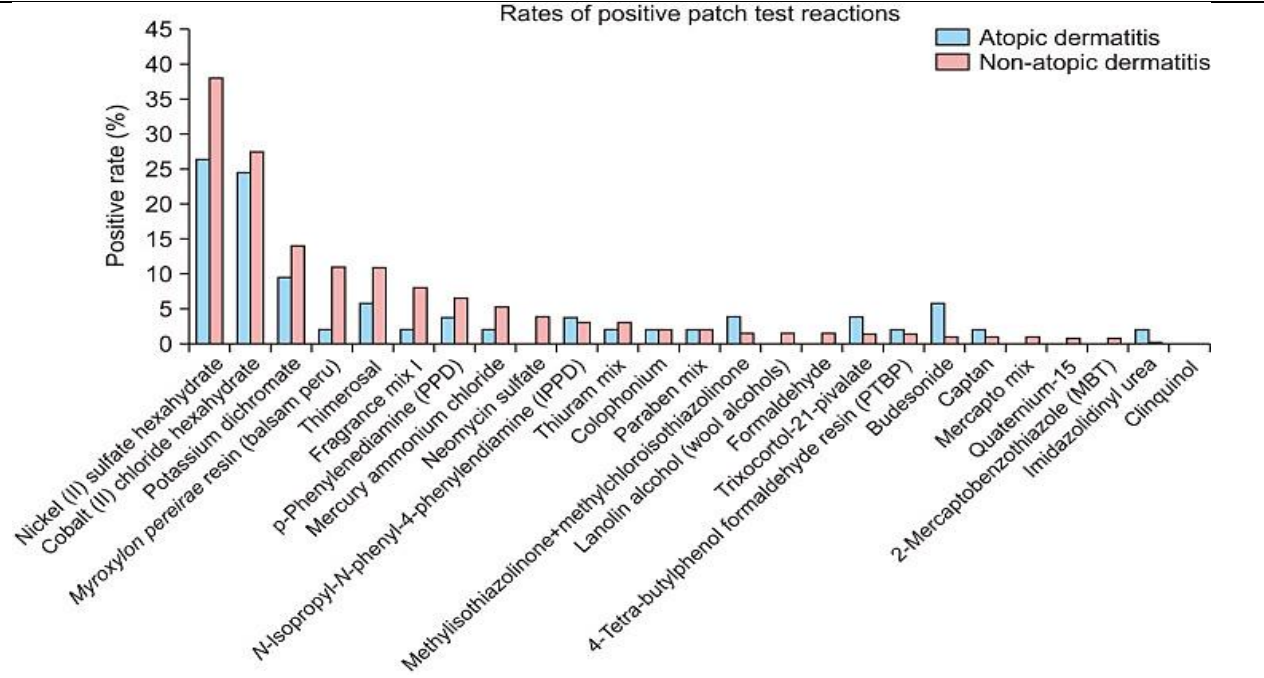


Figure 2. Rates of positive patch test reactions

Discussion:

In this investigation, the rates of at least one allergen response (1+, 2+, or 3+) were substantially lower in the AD group (56.6% vs. 72.9%, respectively, p=0.013) than in the non-AD group, suggesting that individuals with AD had a reduced incidence of contact sensitization.[1] This is consistent with the findings of Hamann et al., which showed that in patients referred for patch testing, there was an inverse relationship between AD and contact sensitization.[3]

Conclusion:

This study found that contact sensitization was less common in AD patients than in non-AD individuals. Physicians should be aware that individuals with ACD who have a history of AD may be allergic to corticosteroids.

The results of this investigation showed that compared to people without AD, contact sensitization was less prevalent in AD patients.

Reference:

1. Choi YJ, Byun JY, Choi YW, Roh JY, Choi HY. Analysis of Positive Patch Test Allergens in Allergic Contact Dermatitis Patients with Atopic Dermatitis. Ann Dermatol. 2023 Aug;35(4):303-312.
2. Watts TJ, Watts S, Thursfield D, Haque R. A Patch Testing Initiative for the Investigation of Allergic Contact Dermatitis in a UK Allergy Practice: A Retrospective Study. J Allergy Clin Immunol Pract. 2019 Jan;7(1):89-95.
3. Hamann CR, Hamann D, Egeberg A, Johansen JD, Silverberg J, Thyssen JP. Association between atopic dermatitis and contact sensitization: a systematic review and meta-analysis. J Am Acad Dermatol 2017;77:70-78.

Assessment of serum level of survivin in acne and post-acne scarring patients along with the possible effect of isotretinoin therapy on its level

Introduction:

Acne vulgaris is a prevalent chronic inflammatory pilosebaceous unit disease. It is a pleomorphic condition characterized by microcomedones, pustules, papules, and nodules that mostly affect the face, upper chest, and back, followed by regions that are rich in sebaceous glands.[1] Acne vulgaris is the eighth most common condition worldwide and nearly all people in the 15–17 age range are vulnerable. Acne can cause long-term adverse effects such as facial scarring, feelings of poor self-esteem, isolation from society, and depression. Survivin is a member of the inhibitors of apoptosis (IAP) gene family and is a a 16.5 kD protein that affects cell division, proliferation, and survival as well as inhibiting apoptosis. It is made up of 142 amino acids in total and is inversely regulated by p53, expressed in both tumor cells and fetal tissue, and encoded by the BIRC5 gene, which is found on chromosome 17 (17q25). Neoplasia and sebaceous hyperplasia have been linked to nuclear survivin expression. Acne formation may result from abnormal apoptosis and elevated sebocyte survival, both of which can be regulated by survivin, affecting infundibular keratinocyte differentiation and sebum production.[1] The purpose of this study was to determine the serum level of survivin in patients with acne and acne scarring, as well as to evaluate any potential effects of isotretinoin medication on this level.

Methods:

This case-control prospective study involved sixty individuals, including 40 patients with acne (Groups IA, IB) and 20 age- and sex-matched controls (Group II). Group IA had 20 patients with active moderate-to-severe acne without scarring, and this group was given oral isotretinoin treatment for three months. Group IB had 20 individuals with post-acne scars that lasted no longer than 6 months and no visible current acne lesions. An enzyme-linked immunosorbent assay (ELISA) was used to determine the serum survivin levels in each of the three groups. The data were entered into a computer and assessed using the IBM Statistical Package for the Social Sciences (IBM SPSS) software package version 20.0.

Results:

All three groups (IA, IB, and II) had a higher proportion of females than males, with the ratios being 3:1, 2.5:1, and 2.5:1, respectively. The active acne group (IA) experienced a disease duration ranging from 4 months to 3 years, whereas the acne scar group experienced a disease duration of 2 to 6 months. The scar group had a statistically significant greater survivin level than the active acne group ($p = 0.008$) and control group ($p < 0.001$) when serum survivin levels were measured using ELISA. Additionally, the group with active acne had a statistically significant greater amount of survivin than the control group. (Figure 1) Furthermore, following therapy, survivin levels decreased significantly and positively linked with a decrease in the global acne grading system (GAGS) in the group of patients with active acne.

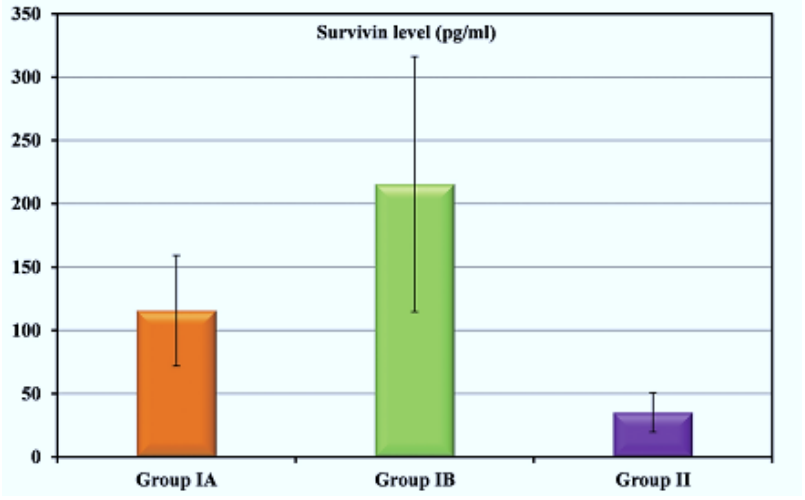


Figure 1. Serum survivin levels in the control (II), acne scarring (IB), and acne (IA) groups

Discussion:

This study identified that there was a statistically significant difference in serum survivin levels across the three groups investigated, with the scar group having the highest levels, followed by the active acne group and finally the control group.[1] In parallel to this result, Mohammed et al, found a statistically significant rise in serum survivin levels in the active acne and post-acne scar groups when compared to the control groups in their investigation.[3] According to El-Tahlawi et al, patients with active acne exhibit greater levels of survivin compared to those with acne scars.[4]

Conclusion:

The findings of this study support the hypothesis that survivin may play an important role in the development of acne vulgaris, as well as more substantial involvement in the pathogenesis of post-acne scar formation. Furthermore, survivin may be a target for oral isotretinoin treatment.

Survivin may have a significant role in the development of acne vulgaris, as well as a pivotal role on the pathogenesis of post-acne scar formation.

Reference:

1. Hussein TM, Tawfik NT, Abd-Rabboh MM, Taha AA. Serum survivin in acne versus post-acne scarring and the possible effect of isotretinoin therapy on its level. Acta Dermatovenerologica Alpina, Pannonica, et Adriatica. 2023 Mar 1;32(1):7-10.
2. El Mokadem SM, Khashaba SA, Said NM, Mostafa AM. Serum survivin level as a novel biomarker in acne vulgaris patients. The Egyptian Journal of Hospital Medicine. 2020 Oct 1;81(3):1565-70.
3. Mohammed S, Elfatah A, Mokadem E, Abd S, Khashaba E, Said N, et al. Serum survivin level as a novel biomarker in acne vulgaris patients. Egypt J Hosp Med. 2020;81:1565–70.
4. El-Tahlawi S, Mohammad NE, El-Amir AM, Mohamed HS. Survivin and insulinlike growth factor-I: potential role in the pathogenesis of acne and post-acne scar. Scars Burn Heal. 2019;5:2059513118818031.

Evaluation of reconstructive surgery for the management of hidradenitis suppurativa

Introduction:

Hidradenitis suppurativa (HS/Acne Inversa) is a chronic and severe multifactorial inflammatory cutaneous illness of the pilosebaceous. Normal skin in HS becomes altered, resulting in the creation of abscesses, inflammatory nodules, tunnels, and scars. The body's axillae, groins, ano-genital area, buttocks, and sub-mammary region are among the intertriginous regions primarily get affected.[1] HS has a major influence on both physical and mental health-related quality of life. Given the discomfort, discharge, odor, and itching that are connected with HS, it has been established that these factors adversely affect patients' quality of life in relation to their health. The clinical manifestations of hidradenitis suppurativa range from rare, benign inflammatory nodules to widespread abscesses, sinus tracts, and scarring. Pharmacological treatment has no long-term therapeutic benefits. One potential therapeutic option for severe stages of the disease is surgery. Deroofing, primary closure, or reconstructions using skin flaps or split thickness skin grafts are common surgical treatments for HS.[2] So, this study assessed the surgical therapy for patients who underwent surgery and had a six-month follow-up.

Methods:

Classical reconstructive techniques were used during surgery on thirty-one HS patients. Both male and female patients with Hurley Scale grades I–III hidradenitis suppurativa were included in this study. Women who are pregnant, breastfeeding, have had cancer within the last five years, including basal cell carcinoma (BCC), and are currently receiving biological and pharmacological treatment for HS were among the exclusion criteria. The following fundamental reconstructive surgical approaches were employed in this study: 1) rotation flap reconstruction, 2) autogenous skin grafts, 3) mixed treatment (split thickness skin graft and skin flap reconstruction), 4) vacuum aided closure (VAC), and split thickness skin grafts. The patients were followed at the outpatient clinic for 6 months. The statistical analysis was conducted with Statistica 13.0 software. The normality of the distribution was evaluated using the Shapiro-Wilk test.

Results:

Of the patients, 83.87% had recovered totally. A statistically significant positive association was seen between the age, BMI, duration of disease, and time of diagnosis of the patients ($p < 0.050$). In addition, there was a correlation between the BMI value and the duration of the condition as well as the time of diagnosis. (Table 1) Compared to patients without metabolic syndrome, those with metabolic syndrome had a statistically longer duration of disease (15.00 ±5.00 vs. 7.00 ±4.00, $p = 0.048$). Patients in Hurley Stage I were considerably younger than patients in Hurley Stage II ($p = 0.026$) and Hurley Stage III ($p = 0.019$). Patients in Hurley Stage I also had a shorter illness duration than patients in Hurley Stages II and III ($p = 0.025$ and $p = 0.019$, respectively). Surgical treatment evaluation (6-month follow-up) revealed that surgical therapy results in a very high percentage of complete wound healing following broad excisions (83.87%) employing surgical reconstructive procedures. Keloids (6.45%) and small injury dehiscence during healing (6.45%) are the most frequent consequences of healing. Only one patient had a recurrence at six months' observation in the surgical location, accounting for 3.23% of cases.

	Age	BMI	Duration of disease	Time from first symptoms to final diagnosis	Time of hospitalization
Age	–	0.66*	0.78*	0.77*	0.07
BMI	0.66*	–	0.61*	0.59*	−0.09
Duration of disease	0.78	0.61	–	0.95*	0.10
Time from first symptoms to final diagnosis	0.77*	0.59*	0.95*	–	0.19
Time of hospitalization	0.07	−0.09	0.10	0.19	–

Table 1. Spearman's rank correlation coefficient (r_s) between quantitative factors

Discussion:

The first line of therapy for mild HS (Hurley I) is medical treatment with topical or systemic antibiotics. However, surgical procedures should be taken into consideration in extreme situations. A full recovery was achieved in 83.87% of the patients. The study found that HS recurrence at the surgical site occurred in just one (3.23%) patient after 6 months of follow-

up.[2] In Ovadja et al., a total of 107 surgical procedures were carried out on 54 patients and after a median follow-up of 30 months, the total recurrence rate was 31.8%.[3]

Conclusion:

As demonstrated in this study, HS is currently an under-recognized condition. Systemic therapies are frequently ineffective, and it appears that in severe cases of HS, surgical therapy can have good clinical effects. One successful strategy for treating HS is surgery. The good therapeutic impact of surgical therapy is supported by the comparatively low recurrence rate after 6 months and the majority of patients' complete recovery.

One successful strategy for treating Hidradenitis suppurativa (HS) is surgery. The good therapeutic impact of surgical therapy is supported by the comparatively low recurrence rate after 6 months and the majority of patients' complete recovery.

Reference:

1. Gierek M, Kitala D, Łabuś W, Szyluk K, Niemiec P, Ochała-Gierek G. Impact of hidradenitis suppurativa surgical treatment on health-related life quality. Journal of Clinical Medicine. 2022 Jul 26;11(15):4327.
2. Gierek M, Bergler-Czop B, Łabuś W, Ochała-Gierek G, Niemiec P. Reconstructive surgical treatment of hidradenitis suppurativa with a 6-month follow-up. Advances in Dermatology and Allergology/Postępy Dermatologii i Alergologii. 2023 Apr 1;40(2):268-72.
3. Ovadja ZN, Bartelink SAW, van de Kar AL, et al. A multicenter comparison of reconstruction strategies after wide excision for severe axillary hidradenitis suppurativa. Plastic Reconstr Surg Glob Open. 2019;7:e2361.

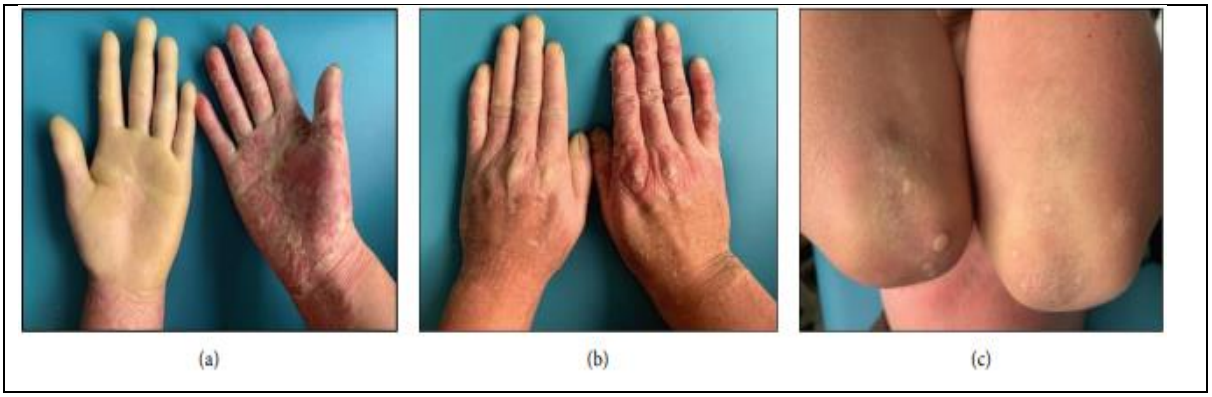
Case report on Atypical Mal de Meleda in a Hispanic Patient

Introduction:

Mal de Meleda (MDM) is a rare autosomal recessive form of palmoplantar keratoderma (PPK) caused by mutations in the gene encoding secretory lymphocyte antigen 6/urokinase-type plasminogen activator receptor related protein 1 (SLURP1). This condition is characterized by inflammation that begins in early infancy and gradually spreads to the dorsal surface of the hands and feet, as well as diffuse erythema and hyperkeratosis of the palmoplantar regions.[1] There is no specific therapy for MDM, however oral systemic retinoids have been shown to be effective.[2] The current clinical case presented a Hispanic patient with asymmetric MDM who was treated with isotretinoin.

Case Presentation:

A male Hispanic patient of 42 years old, had diffuse PPK that persisted and extended to the dorsum of his hands, feet, wrists, elbows, and knees. His mother noticed hyperkeratotic plaques on the tips of both hands when he was three months old. Early in childhood, the lesions expanded to the dorsal surfaces of the hands, feet, ankles, and wrists, as well as the surface of the palms and soles. A clinical examination revealed yellowish waxy hyperkeratotic transgressive plaques on the left palm. There was desquamation on the right palm with an erythematous background and interphalangeal hyperkeratotic rings. There were hyperkeratotic lichenifed plaques on the elbows, and the fngertips had lost their dermatoglyphics. (Figure1)



(a) (b) (c)

Figure 1: Clinical characteristics associated with Mal de Meleda of the hands and elbows include: (a) hyperkeratotic plaque in the left hand and desquamation in the right hand; (b) keratoderma on the dorse of the hands limited to proximal phalanges and nail involvement with dystrophy, koilonychia, onycholysis, and yellowish chromonychia; and (c) hyperkeratotic-lichenifed plaques on the elbows with a slight desquamation.

There were hyperkeratotic plaques on the soles and desquamation on the fingertips. In the dorse of the foot, periungual and interphalangeal regions displayed scaly hypochromic plaques with desquamation. The knees exhibited hyperkeratotic plaques and scaly spots, as well as hair loss on the body. (Figure 2) The rest of the physical evaluation was normal. The patient has been treated with ointments, steroids, and keratolytic medicines all of his life without seeing any improvement in his condition. However, he had acitretin (unknown dosage) for three years while living abroad fifteen years ago, and he claimed overall improvement. The patient was diagnosed with palmoplantar pityriasis rubra pilaris (PRP) a year ago and was treated with fluconazole (150 mg orally once weekly) and clotrimazole cream 1% (topically administered daily). Because the patient did not have coalescing reddish-orange keratotic follicular plaques, PRP was diagnosed. Topical therapy was started twice a day with Burrow's solution (aluminum triacetate) immersion, clobetasol 0.1% + salicylic acid 20% + lactic acid 2% cream, and urea 5% moisturizing cream. Due to the lack of clinical response, a punch biopsy on the right palm and right knee was done one month later. A thicker corneal layer, a noticeable stratum lucidum, a noticeable acanthosis, spongiosi, and a noticeable perivascular lymphohistiocytic infiltrate were all seen upon histopathological investigation. The histological report showed a nonepidermolytic hyperkeratosis with a thick plug of compact keratin compressing the epidermis, resulting in a thick granular layer and acanthosis with regular hyperplasia of the networks. There was also a sparse lymphocytic infiltrate surrounding the papillary vessels. The findings of the laboratory tests were normal. The MDM diagnosis was made based on the earlier condition, clinical presentation with no systemic symptoms, history of inbreeding, and histological results. A genomic study of the

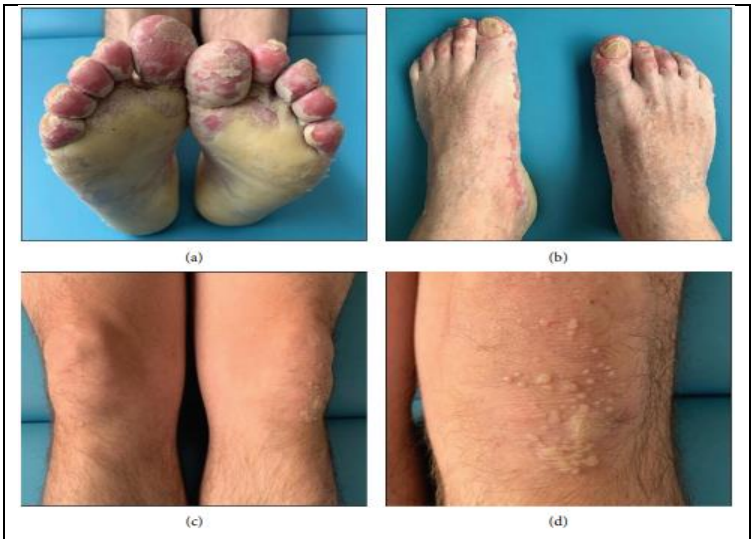


Figure 2: Clinical characteristics of Mal de Meleda in the foot, soles, and knees.

SLURP1 mutation was not performed. To evaluate tolerance, isotretinoin was started at low dosages (10 mg orally daily) and gradually increased in dosage. After a period of two months, the patient exhibited a decrease in the thickness of PPK lesions, a reduction in desquamation, and the restoration of tactile and thermal feeling in both hands. Because of the patient's better tolerability and lack of side effects, the dose of isotretinoin was increased to 20 mg orally once a day.

Discussion:

MDM has an estimated incidence of 1/100,000 people and was first recorded in 1826 on the Croatian island of Meleda because of the severe risk of inbreeding. MDM lacks a well-established therapy procedure; nonetheless, some therapies have received more attention than others. Oral retinoids, dermabrasion, topical keratolytic medicines, and the management of complications were the general recommendations for the treatment of PPKs. In the present case MDM was managed by isotretinoin.[2] In previous studies by Pospischil I et al. and Cebeci D et al., MDM was treated by oral acitretin.[3,4]

Conclusion:

Despite the fact that MDM has been recorded worldwide, there were no examples among Hispanics. Therefore, in order to further and broaden the understanding of this illness, this case describes a Hispanic patient with asymmetric MDM in this case report. The diagnosis of MDM was confirmed based on a personal history of consanguinity, clinical presentation with no systemic symptoms, and a transgredien pattern of the lesions. After starting systemic therapy with low dosages of isotretinoin (10 mg orally every day), slight clinical improvement was seen after two months.

In this case, after starting systemic therapy with low dosages of isotretinoin (10 mg orally every day), slight clinical improvement was seen after two months.

Reference:

1. Kudo M, Ishiura N, Tamura-Nakano M, Shimizu T, Kamata M, Akasaka E, Nakano H, Okuma Y, Tada Y, Okochi H, Tamaki T. Abnormal keratinization and cutaneous inflammation in Mal de Meleda. J Dermatol. 2020 May;47(5):554-558. doi: 10.1111/1346-8138.15296. Epub 2020 Mar 10.
2. Guevara M, Mafla M, Miño C. Atypical Mal de Meleda in a Hispanic Patient. Case Reports in Dermatological Medicine. 2023 Sep 14;2023.
3.] I. Pospischil, K. Enzelsberger, S. Gross, W. Hoetzenecker, and T. W. Fischer, "Mal de Meleda: diagnostic work-up and therapy with low-dose acitretin," Acta Dermato-Venereologica, vol. 102, p. 758, 2022.
4. D. Cebeci, S. Karasel, and G. Bayraktaroglu, "Two sisters with mal de Meleda: case series report," International Journal of Applied and Basic Medical Research, vol. 12, no. 2, pp. 151–154, 2022.