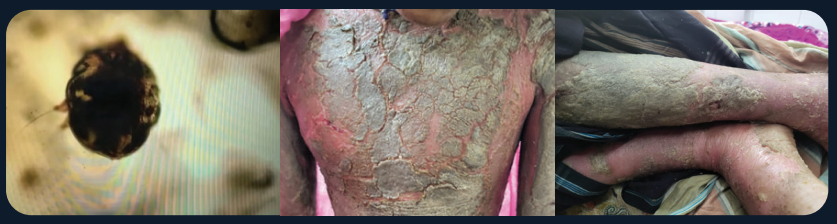
JBAD

Peer Reviewed
ISSN : 2791 - 0725 (paper)
2791 - 0733 (online)

Journal of Bangladesh Academy of Dermatology

Volume 03 Issue 02 July 2023

JBAD Volume 03 Issue 02 July 2023 www.jbadbd.com



Bangladesh Academy of Dermatology (B.A.D.)
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Journal of Bangladesh Academy of Dermatology

Volume 03 Issue 02 July 2023

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Journal of Bangladesh Academy of Dermatology

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Journal of Bangladesh Academy of Dermatology (JBAD) is the official publication of Bangladesh Academy of Dermatology (B.A.D.) scheduled to publish two times (January & July) in a year.

Publisher: Bangladesh Academy of Dermatology (B.A.D.), 153/1 Green road (2nd floor), Dhaka-1205, Bangladesh. Phone: 88-01716259319, 88-01711100552
email: acadbd@yahoo.com, www.jbadbd.com

Subscription: A complimentary copy is provided for all members of B.A.D. Subscription rate for single volume tk. 1500.00 for Bangladesh and USD 30.00 for rest of the world.

Printed by: UNIPACK, 111, Arambagh, Dhaka-1000 ,Bangladesh. Phone: +88 01949-317606

Editorial

Scabies pandemic and treatment failure, a real nuisance

Scabies is an extremely pruritic contagious skin disease caused by the Sarcoptesscabiei var. hominis mite.[1] It spreads mostly via direct skin contact or sharing linens and clothing. Systemic complications of scabies with a disrupted skin barrier due to constant scratching can occur, which may serve as a portal of entry for organisms leading to super infections, rheumatic fever or glomerulonephritis.[2]

It is estimated that more than 200 million people are still suffering from scabies worldwide, where scabies is endemic in some of the subtropical regions. Important risk factors for scabies include young age, a large number of children in the home, low family income, and poor accommodation.

Scabies infestation exhibits a disproportionate effect on children in low-income countries and tropical regions. The prevalence is higher in schools, madras as and boarding houses where they gather in a large mass. The severity of pruritus, which worsens at night, can range from mild symptoms to highly uncomfortable reactions affecting the quality of life (that disturb patients' sleep or impair their concentration at school).[3]

Scabies is treated by topical application of Permethrin either alone or in combination with systemic Ivermectin. In the case of crusted scabies systemic ivermectin is used at a larger dose with multiple applications of permethrin. Other modalities are benzyl benzoate, Crotamiton, Monosulfirum, Gamma benzene hexacloride and ulphur. In April 2021, Spinosad topical suspension 0.9%, was approved by the Food and Drug Administration for treating scabies infestations in adult and pediatric patients 4 years of age and older. Treating all people in contact with the infected patient and preparing clothing and furniture appropriately is of utmost importance to prevent reinfection.[4] Topical scabicides are often applied incorrectly and following instructions are not adapted accordingly, causing a longer course of disease and longer infectiousness.This notable increase in scabies cases even in western countries might be explained by an increase in migration of people there from tropical and sub-tropical countries and the emergence of factors known to favour the disease (e.g. poverty, poor sanitation, high population density and household crowding).[5]

Resistance of mites to permethrin in developed countries was reported as early as 1999 and an in-vitro analysis in 2000 showed protracted survival, with 35% of mites still alive after 3 h and 4% still alive after 18–22 h of constant exposure.[6]

The German guidelines recommend permethrin for common scabies, as it is applied locally and usually needs to be used just once. Whether repeated treatment is needed has not been answered yet but seems worth it even in uncomplicated cases. Repeated application is recommended in cases of crusted scabies, severe scabies (e.g. many papules caused by burrows), immunosuppressed patients, doubt as to whether initial treatment was consistently followed, and scabies outbreaks in care homes and situations in which multiple individuals are affected. When large populations with a high prevalence of scabies are treated, systemic ivermectin seems to be superior to topical treatment.[7] Predictors of treatment failure are associated with the immune status of the host, selection of therapeutic molecule, re-exposure to the mites and drug resistance. Treatment failure is reported low with permethrin and ivermectin used concomitantly. Oral ivermectin administered in two doses one week apart was associated with a significant reduction in treatment failure compared with a single dose.[8] A report of a case series from the SARS-CoV-2 pandemic found a treatment failure rate with permethrin of 73%. However, all patients responded to treatment with oral ivermectin.[9] One of the possible explanations for treatment failure is the lack of response or resistance to topical permethrin.On the other hand, subtle changes in drug formulation cannot be ruled out, leading to less efficacy. Combinations of current treatments and trying other topical preparations may be the options to combat the situation.[10]

To combat this epidemic, the International Alliance for the Control of Scabies (IACS) was launched in 2012. It proposed some criteria to guide the diagnosis of scabies. Subsequently, the World Health Organization classified scabies as a neglected tropical disease in 2017 and currently collaborates

with organisms such as the IACS to develop joint control strategies. In 2020, scabies was included in the roadmap of the World Health Organizationfor neglected tropical diseases 2021---2030.[11]

Proposed reasons that could explain why scabies treatment failure is increasing:[12]

1. Incorrect application or regimens and reinfestation.
2. Decrease in sensitivity to topical treatment.
3. Transmission between animals and humans
4. Residual pruritus: a false therapeutic failure.

It is noteworthy that many patients attend once again a few days after the end of the first treatment due to persistent papules and itching claiming a misdiagnosis or unsuccessful treatment. It is important to remember that pruritus, or some papules can last for another 4 and 6 weeks after the end of treatment, even after the infestation has been eradicated. So, therapeutic failure in clinical practice may be multifactorial - due to application errors, resistance to treatments, presence of poorly identified routes and transmission, and even itching due to immunological reactions to scabies antigen and because of false failures due to residual clinical symptoms.

Studies are ongoing to find out the reasons for treatment failure addressing the susceptibility of mites, genetic typing of mites and finding newer molecules to mitigate the situation. Hope, the pandemic will be over very soon from Bangladesh and other parts of the world with new lights of approaches to mitigate the situation.

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Original Article:

Hidradenitis Suppurativa: Current Trends of Treatment in Bangladesh

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Abstract

Background: Hidradenitis suppurativa (HS) is a chronic, recurrent, debilitating dermatosis. Management of HS remains a challenge owing to lack of a curative medical treatment, potential resistance of recommended antibiotics and higher cost of biologics. **Objectives:** The study aimed to assess the current trends in treatment patterns of Hidradenitis suppurativa in Bangladesh. **Methods:** The cross-sectional study was conducted between January 2023 and March 2023 among 76 dermatologists from all over Bangladesh. A pretested, semi-structured questionnaire containing 13 questions targeted mainly prescribing patterns based on their regular clinical practices. The data has been collected through both online and in-person interviews. **Results:** The study found that topical+systemic antibiotics were the most frequently used treatment modality for HS in all severity groups. Doxycycline was the most preferred systemic antibiotic, followed by Clindamycin. Most of the dermatologists (79%) did not prescribe the combination therapy of clindamycin + rifampicin as a reason for drug resistance (51.7%) and unavailability (46.6%). However,46% prioritized isotretinoin as non-antibiotic treatment and 78% have never given biologics for HS treatment. When it comes to surgical intervention, almost half (43.4%) of the dermatologists never performed any surgical procedure in clinical practices and they referred the patients to a plastic surgeon (30.2%). The study explored the challenges of HS treatment in Bangladesh, including the chronic nature of the disease (76.3%) and patients not compliant with prescribed treatment (52.6%) as challenges in treating HS treatment. **Conclusion:** Identifying and addressing barriers to achieving expertise in the treatment of HS can help provide access to high-quality care for patients with HS.

Key word: Hidradenitis Suppurativa, combination therapy, non-antibiotic therapy, Biologics.

Introduction:

A persistent skin disorder named Hidradenitis suppurativa (HS) can have a detrimental effect on a person's physical, psychological, and social well-being. HS is a persistent, recurring, inflammatory skin disorder that affects intertriginous skin and is frequently accompanied by a number of systemic comorbidities.[1] Globally, the true prevalence of HS remains unclear; in Europe and the US, the estimated HS general frequency varies from 0.00033% to as high as 4.1 percent.[2] The majority of HS prevalence estimates came from the US, Australia, and Europe; especially in Asia, a small number of HS studies were conducted. Given that HS is still a condition that is commonly misdiagnosed and underdiagnosed, the true worldwide prevalence may be higher. The development and maintenance of HS are influenced by several genetic, environmental, and immunologic parameters. It is

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Cite this Article:
Jesmin A, Rashid MM, Hamid MA, Hoq AJMS, Snigdha KSR, Ahamed ARS, Alam AKMM, Ahmed I, Noor T, Nishat F, Chowdhury AH, Khan MZH. Hidradenitis Suppurativa: Current Trends of Treatment in Bangladesh. Ban Acad Dermatol. 2023; 03 (02): 50-57

Available at: www.jbadbd.com
An official publication of Bangladesh Academy of Dermatology (B.A.D.)

mostly connected to follicular occlusion, which may arise from a variety of biological processes, such as follicular hyperplasia and hyperkeratinization.[3]

The clinical course is quite unpredictable and can range from mild clinical presentation, which is defined by the development of papules, pustules, and nodules, to severe cases with deep abscesses, draining sinus tracts, and keloid scars.[4] The intertriginous regions, specifically the axillae and anogenital regions in both sexes, as well as the submammary folds in women, are the areas most frequently affected. The detrimental impacts on a patient's quality of life frequently lead to social disengagement, unemployment, despair, and suicidal ideation due to intense pain, pruritus, unpleasant discharge, sleep, sexual dysfunction, and low self-esteem.[5-6] Additionally, numerous comorbidities have link to HS, including cardiovascular disease, obesity, anxiety, depression, diabetes, polycystic ovarian syndrome, inflammatory bowel disease, spondyloarthritis, and other inflammatory disorders.[7-8] Delay in diagnosis, improper or inadequate illness management, and unpredictable disease development all contribute to the disease's late recognition.

Healthcare professionals face difficulties in managing patients with HS, particularly when it comes to the long-term care of a condition that is frequently resistant to treatment. A wide range of therapeutic methods, including topical medications, systemic medicines, and procedural techniques, are used in HS management strategies. Antiseptic washes, steroid injections, topical and oral antibiotics (as single agents or in combination), retinoids, dapsone, oral immunomodulators, oral contraceptive medicines, and antitumor necrosis factor (anti-TNF) therapy are examples of common medical therapy techniques. The surgical procedure includes a skin flap or graft, full excision with closure by secondary intention, narrow margin excision, and incision and drainage for acute flares.[9] It is often necessary to use two or more of these medicines in combination to achieve a therapeutic response.[10]

Modern modalities like biologics are particularly beneficial in moderate-to-severe HS. However, there are many constraints to employing modern treatment techniques in Bangladesh, such as the availability of drugs, the expense of treatment, insufficient experts, and patient noncompliance with treatment. Dermatologists in our country are also literarily little known about the treatment preferences and challenges of HS. The present study aimed to observe the management strategies of HS and treatment preferences among dermatologists, along with the reported challenges in treating HS in Bangladesh.

Materials & Method:

It was a descriptive type of cross-sectional study conducted between January 2023 and March 2023. The study got responses from 76 dermatologists across Bangladesh to observe dermatologists' treatment preferences for treating Hidradenitis Suppurativa. The study instrument was a questionnaire containing 13 questions targeting mainly prescribing patterns of treatment modalities, which was developed and pretested by expert dermatologists. The data has been collected through both online and in-person interviews. Informed written consent was obtained using the cover letter enclosed with the questionnaire. Respondents were asked to give their responses based on their regular clinical practices. The BMDC (Bangladesh Medical and Dental Council) recognized consultant groups' dermatologists who gave the comprehensive HS management pattern were included and the dermatologists who were not willing to participate were excluded from the study. Collected data were analyzed descriptively by the statistical software SPSS (Version 26).

Results:

Characteristic	n (%)
Practicing experience (After post- graduation)	
< 5 years	18(23.7)
5 – 10 years	18(23.7)
11-15 years	19(25)
16-20 years	8(10.5)
>20 years	13(17.1)
Practice setting	
Government	15(19.7)
Private	30(39.5)
Both	31(40.8)
Region of practice	
Barisal	nil
Chittagong	5 (6.6)
Dhaka	47 (61.8)
Khulna	6 (7.9)
Mymensingh	3 (4)
Rajshahi	7 (9.2)
Sylhet	8 (10.5)
Rangpur	nil
Number of patients seeing (in last one year)	
≤ 10	55(72.4)
11-20	13(17.1)

Table I found the demographic characteristics of the participants. The findings revealed that, after post-graduation, 19 (25%) dermatologists are practising with their experience for 11–15 years, followed by 18 (23.7%) respondents for both <5 years and 5–10 years. Among the participants, 31 (40.8%) are both government and private practitioners, and 47 (61.8%) are practising in the capital, Dhaka. The findings revealed that topical + systemic antibiotics were the most frequently used by around 50% of the treatment modality for HS in all severity groups. In mild cases, only topical (35%) was used as a second preferred treatment modality, whereas in severe and moderate HS cases, isotretinoin was used as a second treatment modality Figure 1.

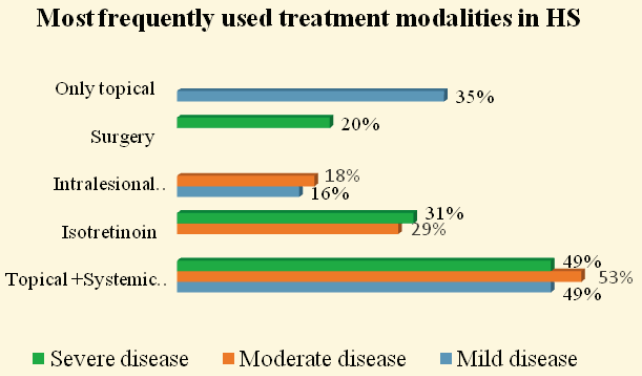

Figure 1: Commonly used treatment modalities in Hidradenitis Suppurativa.

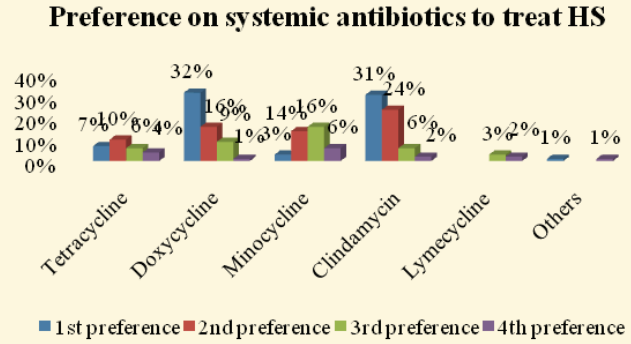

Figure 2: Preference on systemic antibiotics for the treatment of Hidradenitis Suppurativa

Figure 2 reported that, among systemic antibiotics, as the first preference, Doxycycline was mostly preferred, followed by Clindamycin. As a second preference, clindamycin was mostly preferred, followed by doxycycline. Minocycline was more effective in the circumstances of the third preference.

Among all the participants, 79% mentioned that they did not prescribe the combination therapy of clindamycin + rifampicin, while 21% of participants preferred the combination treatment. The reason for not using combination therapy has been explored in Figure 3.

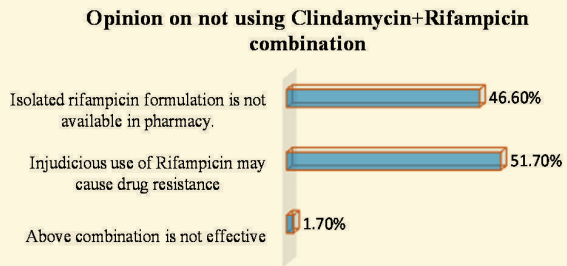

Figure 3: Opinion on not using Clindamycin+Rifampicin combination

Figure 3. Most of the dermatologists (51.7%) narrated that injudicious use of Rifampicin might cause drug resistance, followed by 46.6%, who mentioned that the isolated Rifampiicin formulation was not available in pharmacies. The remaining 1.7% of dermatologists said that the combination of Clindamycin+Rifampicin was not effective for the treatment of HS.

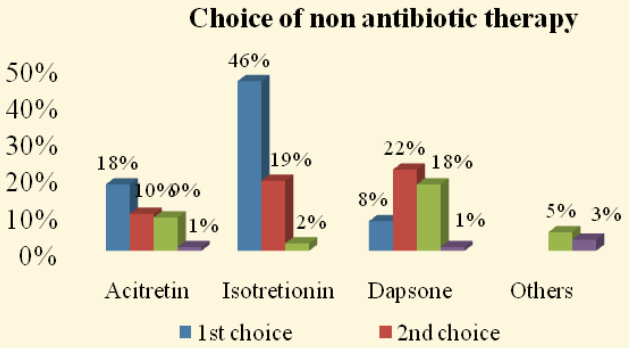

Figure 4: Choice of non-antibiotic therapy (n=76)

Figure 4 documented that the majority of dermatologists (46%) prioritize isotretionin when selecting a non-antibiotic treatment. Dapsone was the second choice among the participants (22%), followed by isotretionin (19%). Nonetheless, the dermatologist recommended Acitretin between the first and third choice, but the recommendation ratio was low in comparison to other treatments.

However, 78% of dermatologists have never given biologic prescriptions for HS Figure 5.

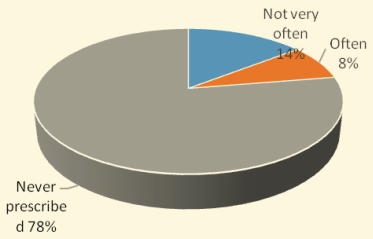

Figure 5: Frequency of prescribing Biologics

When it comes to surgical intervention, the study found that the majority of participants (43.4%) never performed any surgical procedure in clinical practice and referred the patients (30.2%) to a plastic surgeon. A minimum portion of participants performed surgical intervention; those were incision and drainage (15.80%), and deroofing (9.20%), whereas wide excision (1.40%) was the least clinical practice among all dermatologists Figure 6.

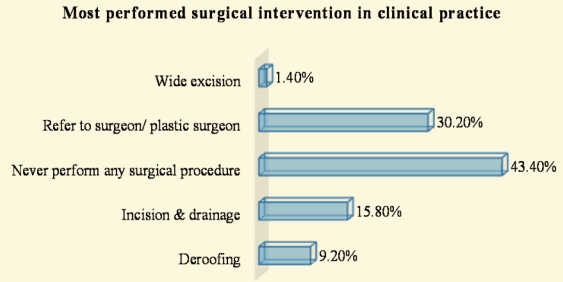

Figure 6: Surgical intervention in clinical practice

The study explored the challenges of HS treatment in Bangladesh. Most of the participants (76.30%) mentioned the nature of the disease (chronicity) as a challenge in treating HS; 34.2% narrated a lack of head-to-head comparative trials; 30.3% stated the high cost of treatment; 23.7% reported delayed diagnosis and those newer biologics are not available; and only 14.5% mentioned the side effects of treatments Figure 7.

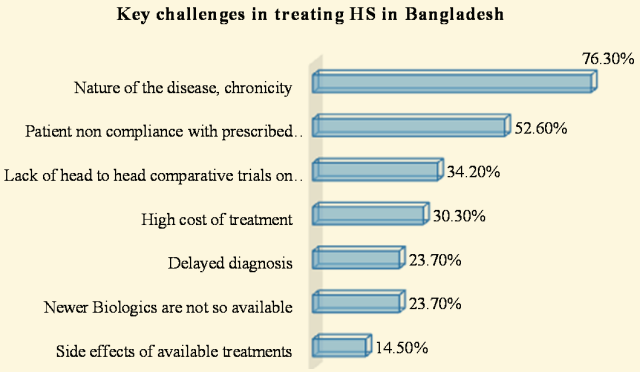

Figure 7: Challenges in treating Hidradenitis Suppurativa

Discussion:

Therapeutic approaches to HS are challenging due to the wide clinical manifestations of the disease, drug resistance, and complex pathogenesis.[11] Expert groups in North America, South America, and Europe have recently proposed a number of HS management guidelines that cover a wide range of therapy methods, including topical medications, systemic therapies, and surgical approaches. Treatment availability, cost, patient's age,

co-morbidities, and severity of the disease all influence the decision of treatment choice.[10]

Considerable variation might exist in the current management of HS in our country. Hence, this study describes the current treatment pattern and challenges in HS management. Most of the dermatologists in the present study, 31 (40.8%), were both government and private practitioners; however, among them, 47 (61.8%) practicing in Dhaka, while no practitioner participated in the study from Barisal, the south-centred division, or Rangpur, Bangladesh.

Skin cleansers, keratolytic agents, and topical antibiotics are among the treatments used topically for HS.[12] Topical treatments are useful as a supplement to systemic treatments for moderate-to-severe disease and are particularly beneficial for mild or localized disease, especially in Hurley stages one and two.[13] For many years, systemic antibiotics have been the cornerstone of treatment for HS; several regimens have been documented. Mild diseases may respond to monotherapy; tetracycline is the preferred first line of treatment and clindamycin + rifampicin is the second. Because of the higher recurrence rate and lesser response rates in late disease, their role is supplementary.[12]

When oral Tetracycline fails, the Clindamycin+Rifampicin combination works well as a second-line treatment for mild to moderate disease.[13] or as a first-line or adjunct treatment for severe disease.[12]

The present study revealed that, in mild, moderate, and severe cases, topical + systemic antibiotics were the most frequently used treatment modality by the study participants, with Doxycycline (32%) and Clindamycin (31%) being the most preferred systemic antibiotics. In the USA, the most frequently prescribed treatments were oral antibiotic therapy (83.9%), topical antibiotic therapy (74.5%), intralesional Kenalog (63.1%), and biologic therapies (TNF-α inhibitors; 49%). Oral and topical antibiotics were the most commonly prescribed treatments in this cohort.[14] Another study showed that both topical and systemic antibiotic therapies were the mainstay of care for HS patients.[10]

In an answer to a question about prescribing a combination of Clindamycin and Rifampicin for the treatment of Hidradenitis Suppurativa, almost 80% mentioned that they did not prescribe the combination therapy of Clindamycin and Rifampicin. The reason behind not prescribing the Clindamycin+Rifampicin combination is that about 52% of participants said that injudicious use of Rifampicin might cause drug resistance as Bangladesh is a tuberculosis-endemic country and Rifampicin is a potential drug for an anti-TB regimen. Some others (46%) mentioned that the isolated Rifampicin formulation was not available in pharmacies. However, Brazilian guidelines recommend using rifampicin carefully as it is the first-line medication for the treatment of tuberculosis, which is common in the area and is becoming increasingly resistant to drugs15. Wayne Gulliver and colleagues considered clindamycin and rifampicin combination therapy for moderate-to-severe HS. Saunte et al. found that antibiotic treatment with combinations of clindamycin and rifampicin was effective.[16]

More research has been done in HS on the combination of clindamycin and rifampin than on most other antibiotics. A recent study revealed that using clindamycin and rifampicin for more than 10 weeks was safe.[17] nevertheless, rifampicin resistance should be considered when thinking about extending therapy.[18] For an 8–12-week therapy term, the recommended dosage is 300 mg twice daily of clindamycin combined with 300 mg twice daily of rifampicin or 600 mg once daily.[18]

The 300 mg formulation of rifampicin is not sold in commercial pharmacies or drug stores in Bangladesh. The 150 mg and 450 mg formulations have been introduced by very few pharmaceutical companies, but they are also not regularly available. Therefore, the patient must pick up Rifampicin from the National Tuberculosis Control Program's TB DOT (Direct Observe Treatment) corner.

In treating HS patients who are not responding to topical or oral antibiotics, acitretin is advised by all guidelines as a second or third line of treatment. Because of its greater response rates, acitretin is favoured over isotretinoin; nevertheless, women of childbearing age should not take acitretin, and if prescribed, they must utilize effective contraception.[13] Even though acitretin is more effective than isotretinoin, isotretinoin is still preferred for female patients who are of childbearing age, according to the guidelines. In the present study, almost half of the dermatologists chose isotretinoin as their first choice as second-line therapy for moderate-to-severe HS treatment, while Acitretin and Dapsone were also

recommended.

Immunomodulation is quickly evolving into the mainstay of treatment for HS, ranging from moderate to severe. According to international standards, biologics should be taken into account for treating moderate-to-severe HS patients who are not responding to systemic conventional medication. The sole licensed biologic for HS is adalimumab, which is advised as the first-line biologic treatment in all guidelines. Infliximab is included as the suggested second-line treatment in most guidelines.[19] Currently, patients with a moderate-to-severe degree of the disease are thought to be benefited most from biologic therapy.[20] When HS patients undergoing biologic therapy were compared to alternative treatments, Peterson GC et al. observed a greater overall response rate. How often do dermatologists in Bangladesh prescribe biologics in HS? Only 22% of dermatologists in our sample prescribed biologics for moderate-to-severe HS; among them, 14% stated that they did prescribe, but not frequently. 78% of dermatologists have never prescribed biologics for HS. In contrast to developed countries, biologics are not commonly prescribed here. The main problems are the scarcity of biologics, the cost of therapy, and the lack of qualified professionals using biologics in HS.

Surgical intervention, laser therapy, and light-based therapy are all part of the procedural management of HS. Except extensive local excision, deroofing, and carbon dioxide (CO2) ablative laser treatment, which are mentioned as successful therapies in almost all guidelines, international recommendations for these modalities are a little erratic.[13] To perform surgical treatments in HS, meticulous qualification is required. In the present study, in a question about performing surgical intervention, about 44% of participants mentioned that they had never performed any surgical procedure in clinical practice, and 30.20% referred the patients to a plastic surgeon. As surgical interventions, incision and drainage (I&D), deroofing, and wide excision were performed by 15.80%, 9.20%, and 1.40% of participants, respectively. In their daily clinical practice, a small number of dermatologists carried out surgical treatments alone; these primarily involved incision and drainage (I&D) and deroofing.

According to a study, surgical interventions connected to HS were comparatively rare throughout the post-index decades, with incision and drainage being the most commonly reported procedures.[21] A different study revealed that, with only 2.7% of patients obtaining surgery or surgical referrals,[10] their providers also used surgical therapy at a significantly lower rate. More than half of the dermatologists questioned regarded experience with surgical therapies, training and education on surgical therapies, insurance coverage, and equipment availability as very major hurdles.[21]

The procedures that are necessary for dermatological training do not include surgical techniques for dermatologists treating HS, and no dermatology intern is exposed to these treatments throughout their residency training. Increasing the number of opportunities to acquire HS surgical skills both during and after residency can help address these barriers.[22]

The present study identified dermatologists who reported key challenges in treating HS. The nature of the disease, chronicity was revealed by 76.30% of participants as a key challenge in treating HS, while 34.2% reported insufficient head-to-head comparative trials, 30.3% noted treatment high cost, delayed diagnosis, and that newer biologics are not available, as reported by 23.7% of the study population, and 14.5% narrated treatment side effects as the challenges for the treatment of HS.

Dermatologists found that the main obstacles to providing appropriate therapy for patients with HS were the high cost of treatments (53.3%), patient compliance with prescribed treatments (60.0%), and restricted health insurance coverage of accessible therapies (70.0%).[23] One of the main unmet needs for HS patients, according to the report, is the limited possibilities for therapy. The lack of randomized controlled trials makes it difficult to compare treatment outcomes between medicines. It is necessary to research novel therapy options because there are few effective treatments for HS. According to a study, HS-specific total healthcare costs made up 4–15% of all healthcare costs.[21] For patients who were adults and adolescents alike, outpatient medical expenses accounted for a larger share of the financial burden. To improve health and lower medical costs, patients with HS may need comprehensive care methods involving a multidisciplinary team of specialists.

Conclusion:

It is expected that more research on the HS treatment pattern would improve the assessment of illness. Identifying and addressing barriers to achieving expertise in the treatment of HS can help provide access to high-quality care for patients with HS. Future research should incorporate benefit-risk ratio analysis and long-term evaluation of efficacy and safety to support appropriate medication and long-term evidence-based treatment.

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Original Article:

Prescription pattern of dermatologists for alopecia areata in Bangladesh: A cross-sectional survey.

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Abstract

Background: Alopecia areata (AA) is a chronic, immune-mediated disease characterized by acute or chronic non-scarring hair loss, with heterogeneity in clinical manifestations ranging from patchy hair loss to complete scalp and body hair loss. Though before June 2022 there was no FDA-approved systemic medication for alopecia areata dermatologists prescribe a good number of topical and systemic agents for the treatment of alopecia areata (AA), and in Bangladesh all treatment modalities are not available and prescribed. **Methodology:** This cross-sectional survey was conducted on 120 dermatologists to see the prescription pattern in alopecia areata. **Results:** The responses of 120 dermatologists were analyzed. The most common treatments prescribed for AA were topical Steroids (91%), topical calcineurin inhibitors (88%), biotin (73%), Zn (73%) minoxidil (60%)tofacitinib (46%), methotrexate (3%), cryotherapy (2%), PUVA/NBUVB, anthralin. At the time of the survey, 46% of patients were being prescribed a combination of corticosteroids (23%), injectable corticosteroids(12%), and topical corticosteroids/topical calcineurin inhibitor. **Conclusions:** This analysis provides a snapshot of the different local and systemic treatment options currently being used in a real-world treatment setting.

Keywords: Alopecia areata; Cross-sectional survey; Treatment patterns.

Introduction:

Alopecia areata is a common, nonscarring, autoimmune disease that can affect any hair-bearing area.[1] The exact pathophysiology of alopecia areata remains unknown. The most widely accepted hypothesis is that alopecia areata is a T-cell–mediated autoimmune condition that is most likely to occur in genetically predisposed individuals.[2] Alopecia areata has a reported incidence of 0.1-0.2%, with a lifetime risk of 1.7%.[3] The disease can begin at any age, but the peak incidence is between 20 and 50 years of age.[4] Both the sexes are equally affected and there is no racial variation reported.[5] Clinically, alopecia areata may present as a single well-demarcated patch of hair loss, multiple patches, or extensive hair loss in the form of total loss of scalp hair (alopecia totalis) or loss of entire scalp and body hair (alopecia universalis).[6] Histopathologically, alopecia areata is characterized by an increase in the number of catagen and telogen follicles and the presence of perifollicular lymphocytic infiltrate around the anagen phase hair follicles.[7] The condition is thought to be self-limited in the majority of cases, but in some cases, the disease has a progressive course and needs active treatment in the form of oral or topical therapeutic options.[8] Progressive alopecia

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Cite this Article:
Zaman MS, Ahmed F, Noor T, Kalam A, Jahan S & Akter M. Prescription pattern of dermatologists for alopecia areata in Bangladesh: A cross-sectional survey. Ban Acad Dermatol. 2023; 03 (02): 58-61

Available at: www.jbadbd.com
An official publication of Bangladesh Academy of Dermatology (B.A.D.)

areata is associated with severe social and emotional impact.[9]

Materials and Methods

The responses of a total of 120 dermatologists were included. This was an observational, multicenter, descriptive cross-sectional study. This study was self-administered questionnaires autocompleted by dermatologists working in Bangladesh. The questions were treatment of different stages of alopecia area with multiple treatments. Participating dermatologists were asked over the telephone, email and directly to fill out a standardized form of treatment on alopecia areata. Children and adults, all types of alopecia areata patients were included. Then asked to observe any side effects of prescribed medication. Consulting for alopecia areata first visit or a follow-up visit was from January to December 2022.

The study was conducted in accordance with local laws and regulations.

Statistical analyses were performed using Stata, version 11 (Stata Inc., College Station, TX, USA). All tests were two-tailed and p-values were considered statistically significant.

Result

The responses of 120 dermatologists were analyzed. The most common treatments prescribed for Alopecia areata were topical corticosteroid (91%), topical calcineurin inhibitor (88%), oral biotin & zinc (73%), topical minoxidil (60 %), systemic steroid (46%), Tofacitinib (36%), intralesional triamcinolone acetonide (23 %), Methotrexate (3%) cryotherapy (2%).

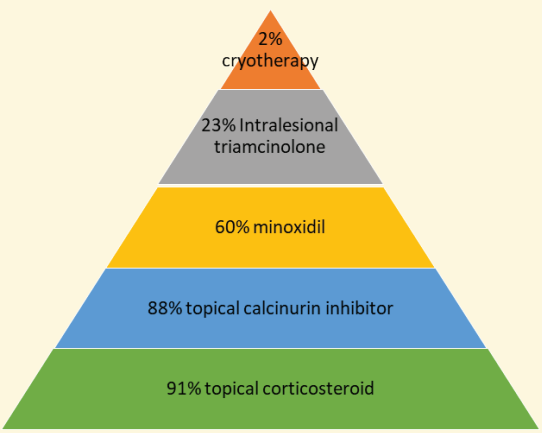

Figure 1: Topical Medication use for AA by dermatologists in Bangladesh.

Figure 2: Oral Medication use for AA by dermatologists in Bangladesh.

Discussion

Alopecia areata occurs worldwide. The estimated prevalence is approximately 1 in 1000 people, with a lifetime risk of approximately 2 per cent. Both children and adults may develop alopecia areata, and the disorder occurs at similar rates in males and females. More than 80% show signs of the disease before age 40, and 40% experience symptoms by age 20.Steroids help control alopecia areata by reducing inflammation and suppressing the immune system, to stop the body from attacking the hair follicles. Topical corticosteroids with varying levels of efficacy have been used to treat alopecia areata. These include fluocinolone acetonide cream, fluocinolone scalp gel, betamethasone valerate lotion, and clobetasol propionate ointment.[10] Our study showed 91% of dermatologists treated with topical steroid,46% systemic steroid and 23% intralesional steroid with variable efficacy.Triamcinolone acetonide is the first-line therapy for adult patients with less than 50% scalp involvement. The preferred concentration for the scalp is 5 mg/mL and for the face and eyebrows it is 2.5 mg/mL.[11] The use of systemic corticosteroids for the treatment of alopecia areata is under much debate. The suggested dosages are 0.5-1mg/kg/day for adults and 0.1-1 mg/kg/day for children.[12] Treatment course ranges from 1-6 months, but prolonged courses should be avoided to prevent the side effects of corticosteroids. The side effects profile of corticosteroids in conjunction with the long-term treatment requirements and high relapse rates make systemic corticosteroids a more limited option.Minoxidil appears to be effective in the treatment of alopecia areata. Its mechanism of action has yet to be determined, but it is known to stimulate DNA synthesis in hair follicles and has a direct action on the proliferation and differentiation of the keratinocytes.[13] Sixty present dermatologists prescribed topical minoxidil for alopecia areata. Price et al reported an 11-patient study in which none of the patients had terminal hair growth in response to tacrolimus ointment 0.1 % applied twice daily for 24 weeks.[14] Emma Andrus in her recent study showed that combined with a low dosage of prednisone, methotrexate provided nearly complete

or complete hair regrowth in up to 31.2% of patients with alopecia areata.[15] In our study, 88 % of dermatologists used for patients. Methotrexate either alone or in combination with prednisolone has been used in the treatment of alopecia areata in various studies with variable success rates. Serum zinc levels are lower in patients with alopecia areata than in the control population in a study on 15 patients, hair regrowth was observed in 9 patients (67%) after oral zinc gluconate administration.[16] Eleven patients diagnosed with AA universalis or totalis were treated with oral tofacitinib.[17] Myungsoo Jun and Won-Soo LeeNineteen patients with AA were treated with cryotherapy successfully treated for 1 month.[18] Another observation is cryotherapy is used for only 3% of patients of alopecia areata. Baricitinib is an oral, reversible, selective JAK1/JAK2 inhibitor.On 13th June 2022, Baricitinib oral tablets were officially approved by the Food and Drug Administration as a systemic treatment for adult patients with severe Alopecia Areata.[19] Alopecia areata (AA) is a relapsing, chronic, immune-mediated disease characterized by nonscarring, inflammatory hair loss that can affect any hair-bearing site. AA clinical presentation is heterogeneous. Pathogenesis of Alopecia areata involves immune and genetic factors and several pro-inflammatory cytokines including interleukin-15 and interferon-γ, as well as Th2 cytokines, such as IL-4/IL-13, that signal through Janus kinase (JAK) pathway. JAK inhibition has been shown to stop hair loss and reverse alopecia. Baricitinib is a Janus kinase inhibitor that is approved to treat AA in several countries, based on results from two studies, BRAVE-AA1 and BRAVE-AA2. One study showed adults with at least 50% scalp hair loss were treated with baricitinib for 36 weeks.[20]

Conclusions

This analysis provides a snapshot of the different local and systemic treatment options currently being used in a real-world treatment setting. Unfortunately, none of these treatments provides a sustainable, safe, and relapse-free solution, which leads to high treatment dissatisfaction rates and hence indicates a significant unmet need for new and advanced treatment options for patients with AA.

Conflict of Interest

None

Funding source

None

Patient Consent

Taken

IRB approval status

Not applicable

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Original Article:

Topical Steroid Abuse in Children: A Glimpse from Bangladesh

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Abstract

Introduction: Irrational use of topical steroids in the pediatric age group is common in Bangladesh. Before consulting with a dermatologist for skin disease, parents tend to use medicine from a previous prescription, consult a quack or take medicine from the pharmacy shopkeeper. As there is no regulation on dispensing steroids over the counter, it is really hard to control steroid abuse.

Aim: The study aimed to observe how steroid abuse occurred, which steroids were abused more and common side effects due to abuse.

Material and method: This was a cross-sectional study conducted at the outpatient department of different government and non-government hospitals in Dhaka, Bangladesh over 3 months. A total of 150 steroid-abused children were enrolled.

Results: The most affected age group was 2 to 6 years. About 96% of the abuse occurred due to recommendation by non-dermatologists, 58% of which was by Quacks and pharmacy salesman, 19% by general practitioners and 17% by paediatricians. The most common topical steroid used by patients was potent e.g. betamethasone dipropionate (46%). Out of 150 patients, 103 (68.6%) used pure steroid cream while 47 (31.4%) used steroid cream in combination with either antifungal or antibacterial or both. The most common side effect observed was tinea incognito, impetigo, and eczema herpeticum.

Conclusion: Topical steroid abuse in children is very common in our country. The problem is worsening due to the easy availability of these medications even without a proper prescription. Every physician should have a good knowledge of steroids before prescribing them. Education of the general public through different communication media should be taken to reduce this abuse.

Keywords: Topical Steroids, Abuse, Children, Bangladesh

Introduction:

Topical steroids was first introduced in dermatology by Sulzberger and Witten in 1952. They first described the effect of compound F in selected dermatoses.[1] This compound F was later renamed as hydrocortisone. With the advancement of time, topical steroids are now available in different potencies and also in the form of combination with antibiotics and antifungals. Effective prescription of topical steroids requires adequate knowledge of steroid potency, duration of application and where to apply in which formulation.

Comparing the anatomy of pediatric and adult skin,

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Cite this Article:
Noor T, Chowdhury S, Jahan S, Wahab F, Afreen H, Chowdhury MM, Guha A, Sayem NN, Haque F. Topical Steroid Abuse in Children: A Glimpse from Bangladesh Ban Acad Dermatol. 2023; 03 (02): 62-66

Available at: www.jbadbd.com
An official publication of Bangladesh Academy of Dermatology (B.A.D.)

the epidermis is thinner and the rate of water absorption, desorption and trans-epidermal water loss is higher in pediatric skin.[2] So they require low-potency steroids. The inappropriate potency of steroids results in the appearance of side effects.

In 1963, the first publication on the side effects of steroids was published.[3] The first side effect in the pediatric age group was documented in a child with psoriasis where parents applied potent topical steroids so compulsively that the child developed atrophy of the skin and died of adrenal failure.[4]

Steroid abuse in children is not so rare in Bangladesh. Before being referred to a dermatologist for the skin problem, they are misled by quacks, pharmacy salesmen, general practitioners, as well as paediatricians. Without adequate knowledge of potency and efficacy, steroids should not be advised. This results in unwanted adverse effects and sometimes iatrogenic Cushing. This is mostly seen in infants where potent steroids are prescribed for diaper dermatitis. Satter H et al published a study on iatrogenic cushing in children using diapers and found out that using potent steroids as diaper rash cream was responsible. This is a major problem in developing countries as there are no guidelines on dispensing over-the-counter drugs.[5]

It was really sad to learn that, there is still no research article or any case report on topical steroid misuse in children of Bangladesh. This is unfortunate to publish this research work as the very first paper on topical steroid misuse in the country.

Materials and Methods

We conducted a cross-sectional study, over 3 months (from January 2023 to March 2023) at different dermatology outpatient departments and clinics in Dhaka. We used a pre-validated structured questionnaire. A total of 150 children of steroid abuse were enrolled. Steroid abuse was diagnosed by a dermatologist by history of steroid application as evidenced by either previous prescription, appearance of steroid-induced adverse effects or the medicine brought by the parents. The children were divided into neonates, infants, young and older children, and adolescents. The person who prescribed the medicine first was documented along with the potency of the steroid applied, duration of application and adverse effects of the steroid that were observed. Striae, atrophy, telangiectasia, acne, Rosacea, hypertrichosis, perioral or periocular dermatitis, tinea incognito, majocchi's granuloma, infantile gluteal granuloma, eczema herpeticum, molluscum contagiosum, warts, exacerbation of impetigo, folliculitis and none were taken into account. The potency of the steroids was classified as mild potent: hydrocortisone, fluocinolone acetonide; moderate potent: clobetasol butyrate, betamethasone valerate 0.025%, mometasone, potent: betamethasone valerate 0.1%, betamethasone dipropionate; very potent: clobetasol propionate, halobetasol.

After completion of data collection, analysis for descriptive statistics of the responses was done by IBM SPSS statistics (version 25.0).

Result

The most affected age group was young children and adolescents. Out of 150 patients, 96% of the patients recommended topical steroids by non-dermatologists, among them 58% by quacks and pharmacy shopkeepers (Fig1). Adverse effects were observed in 85 (56.6%) patients out of 150. The most common side effects were tinea incognito (20%), impetigo (14.6%) and eczema herpeticum (12.6%) (Table 3). The most common topical steroid used by patients was potent e.g. betamethasone dipropionate (46%) (Fig 2). Out of 150 patients, 103 (68.6%) used pure steroid cream while 47 (31.4%) used steroid cream in combination with either antifungal or antibacterial or both.

Table 1: Distribution of the patients according to age

Age group	Frequency	Percent
Neonates (birth up to 1 month)	3	2.0
Infants (1 month up to 2 years)	40	26.7
Young children (2 up to 6 years)	43	28.7
Older children (6 up to 12 years)	21	14.0
Adolescent (12 up to 18 years)	43	28.7
Total	150	100.0

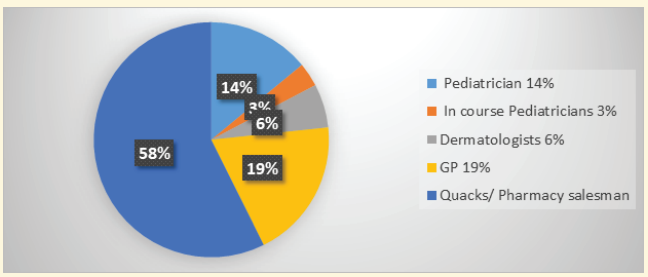

Fig: 1 Frequency of topical steroid prescription inappropriately

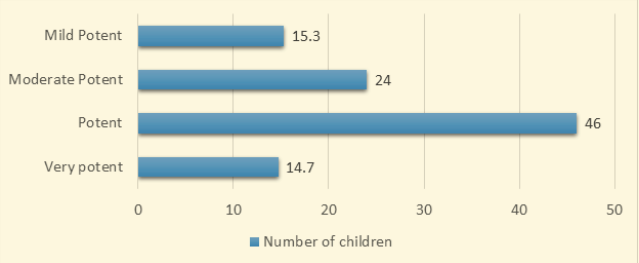

Fig: 2 Distribution of topical steroid abuse according to potency

Table 2: Distribution of patients according to adverse effects observed:

	Adverse effects	Frequency	Percent
1	Atrophy	6	4
2	Striae	4	2.6
3	Telangiectasia	7	4.6
4	Hypertrichosis	7	4.6
5	Acne	18	12
6	Roasacea	2	1.3
7	Perioral or periocular dermatitis	6	4
8	Tinea incognito, majocchi's granuloma, infantile guteal granuloma	30	20
9	Eczema herpeticum, molluscum contagiosum, warts	19	12.6
10	Exacerbation of impetigo, folliculitis	22	14.6
11	Hypopigmntation	12	8
12	None	17	11.3

Discussion

Topical steroids have mostly been used by dermatologists since their introduction in 1951. Their anti-inflammatory action works like magic in several dermatoses. However, they are also considered as double-edged sword as they require careful handling by the provider and the recipient also for safe and effective use.

Uncontrolled use (abuse) of steroid medications has led to many different adverse reactions from the very beginning. Rebound vasodilatation and proinflammatory cytokine release have been proposed as the mechanism for most adverse reactions.[6]

Misuse of steroids in children is common in Bangladesh though adverse effects and safety of topical steroids are clearly stated in the medical literature. The main problem is that patients can buy steroids as OTC drugs without any prescription. Patients also try to save money by consulting with quacks or pharmacy shopkeepers instead of doctors and end up spending more money to overcome the adverse effects. They also think that all skin diseases are the same and same medicine can do magic for every disease. This situation is not so different from our neighbouring country India.[7] If they were fortunate, they might get cured but most patients had to suffer the unwanted effects. Topical steroid is widely used as a whitening cream in adolescents. Continued use of topical steroids results in acneiform eruption, hypertrichosis, hypopigmentation and rosacea. The appearance of side effects depends on the potency of the steroid used, duration and site of application.[6]

A total of 150 children were included in this study. To define topical steroid abuse or unjustifiable use following criteria were taken into account: wrong indication, undiagnosed dermatoses, inappropriate potency, and using more than prescribed duration. The children were divided into neonates, infants, young and older children, and adolescents' age groups. The most common age groups were young children (28.7%) and adolescents (28.7%). This is probably because young children start to move around more, and begin their school, while adolescents begin to have their androgen effects and become conscious about their appearance. A similar study was done in Iraq and the common age was 10-19 years.[8-9] The most common indication was dermatophytosis, scabies, eczema, and acne. The use of steroids as skin-lightening cream was the most common reason for use in Iraq and India.[8-9] Adverse effects were observed in 85 (65.6%) patients. The most frequent side effect which was seen in our study was Tinea incognito (20%) followed by an exacerbation of impetigo (14.6%), acneiform eruption (12%) and eczema herpeticum (12.6%). Similar studies done by Saraswat et al and Al-Dhalimi et al had acne as the most common side effect as topical steroids were commonly used as a fairness cream.[8-9]

Betamethasone dipropionate was used in 46% of the children and Betamethasone valerate in 24% of the total cases. Hydrocortisone was the other common steroid used in combination with antifungals and antibiotics. Bet CL, Betameson and Betnovate were the popular brand names which were used by parents. In Iraq, clobetasol propionate (42.1%) was the most commonly used steroid while betamethasone valerate (26.4%) was the second most common.[9] Again the study done by Saraswat et al. found betamethasone valerate (50.1%) was the most commonly used topical steroid.[9] Another study in India by Mishra et al. showed that clobetasone

propionate was mostly abused followed by betamethasone velarate. Again Santwana Mahar et al conducted a study and found that betamethasone valerate (72.8%) followed by the use of a topical combination of clobetasol propionate, anti-biotics and antifungals (18.4%) were commonly abused.[10]
Most parents used topical steroids for a period of 1 to 2 months and then consulted Dermatologist due to either no improvement or aggravation of the primary disease. The principal portion of inappropriate topical steroids was prescribed by Quacks or a pharmacy salesman and is about 57.3% and the rest by a medical practitioner. Out of the rest, 14% of patients used topical steroids recommended by a paediatrician, 22.6% by a general practitioner, and 6% by a dermatologist. In India, Mishra et al revealed that 29% of patients using topical steroids were recommended by a friend or pharmacist and 71% by a medical practitioner. Out of these 71%, most of the prescriptions were by dermatologists (54%) followed by physicians (16%) and general practitioners (1%). A study done on topical steroid abuse on the face by Saraswat et al showed that 59.3% of the total patients had used topical steroids on recommendation without a valid medical prescription8 In the study of Mishra et al., it was observed that adverse effects were more often seen in patients of non-dermatologists. Similar findings were observed by Al Dhalimi et al. in Iraq where potent steroids were prescribed by both the groups.[9] Sheth et al also found out that most of the abuse was caused by general practitioners.[11] This is due to a lack of knowledge of the potency of topical steroids, where to apply and how long can be used. This leads to misuse of steroids.
There are very few studies on steroid abuse, especially in children. Because most of the developed countries have regulations and steroids cannot be sold without a prescription. The steroid abuse is more prevailing in Southeast Asian countries like India, Bangladesh, Nepal and some Middle east countries due to lack of government regulation on selling steroids. A large portion of people in Bangladesh take treatment primarily from quacks or pharmacy salesmen. Most of these quacks and salesman use to imitate dermatologist's prescription anyhow without knowing why steroid is given. On the other hand, there is a tendency not to refer patients to dermatologists for skin problems in paediatricians. Again one can buy a steroid preparation without having a prescription and this has allowed many of these brands to become household names. Parents are unaware of the side effects posed by these drugs and tend to use them for long periods before consulting a dermatologist.[12]

Conclusion

Topical steroid abuse in children is worsening due to the easy availability of these medications even without a proper prescription. Abuse is mostly done by quacks and pharmacy salesmen and also paediatricians and dermatologists to some extent. This study was to be aware of how steroids are being abused in the pediatric age group. Along with educating the general public through different communication media, regulations should be made to stop over-the-counter selling of steroids.

Financial support and sponsorship

Nil.

Conflicts of interest

There are no conflicts of interest.

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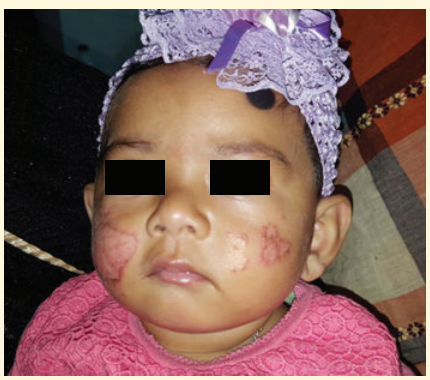

Fig 03(b): Tinea incognito

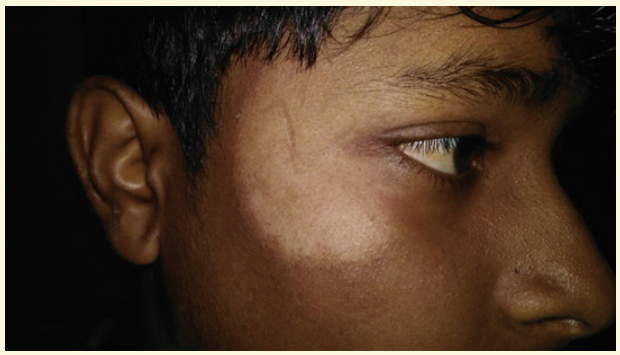

Fig 04. Steroid induced hypopigmentation

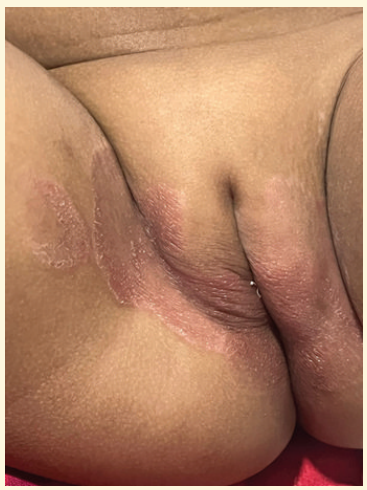

Fig. 05. Aggravation of tinea cruris following application of steroid for diaper dermatitis

Original Article:

Clinico-epidemiological Profile of Hidradenitis Suppurativa in Bangladesh

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Abstract

Background: Hidradenitis suppurativa (HS) is a painful and difficult to manage inflammatory disease presenting with deepseated abscess, discharging sinuses and scarring. It is relatively uncommoninAsiaand there is dearth of published data on HS from Bangladesh.
Objective: To describe the epidemiological and clinical profiles of HS in Bangladesh.
Methods: This multicenter cross-sectional observational study was conducted with diagnosed cases of HS in 3 tertiary level hospital of Bangladesh. Demographic and clinical features were recorded and were compared with published data from Bangladesh other countries. Descriptive statistics in terms of mean, standard deviation, percentage, median, and percentiles were calculated for all parameters in the study. Obtained data were compared with the published articles of home and abroad.
Result: Male outnumbered female in HS cases and male to female ratio was 1.6:1.The disease was started after puberty in majority of cases. The mean age of participants was 28.4±3.7 years that ranging from 16-74 years. Mean duration of disease was9.7 ±3.4years ranging from 2 to 25 years. Comparing with general population the rate of diabetes and obesity were significantly higher ($p<0.01$) in patients with HS whereas smoking and hypertension was comparable($p>0.05$).Axillae was the common (73.9%) affected anatomical site followed by groin. Acne was the commonest comorbidity and most of patients.
Conclusion: In this clinic-epidemiological study males are more prone to develop HS and delayed diagnosis is an important issue which may leads to improper treatment.Acne, diabetes and obesity were the commoncomorbidities of HS.

Keywords: Hidradenitis suppurativa, Acne inversa, Epidemiology of hidradenitis suppurativa

Introduction:

Hidradenitis suppurativa (HS) or acne inversa is a chronic inflammatory dermatological disorder mostly involves hair follicle ofthe apocrine-bearing areas of the body including axillary, inguinal, and anogenital regions characterized by recurrent deep-seated nodules, abscesses and discharging sinuseswith unpleasant odor. , , Typically patient suffersmore than two recurrences in 6 months. It is presently described as an inflammatory disease of the pilosebaceous follicle with an underlying system immune dysregulation in genetically susceptible persons, the course of which modified by exogenous triggers or aggravating factors. The process starts with follicular occlusion in the folliculo-pilosebaceous unit, followed by rupture and an ensuing immune response, where the immune response involves the activation of neutrophilic granulocytes, macrophages, and plasma cells, as

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Cite this Article:
Bhuiyan MSI, Sultana A, Rahman L, Bhiyan I, Sultana L, Jannat N, Jesmin A, Mahmud M. Clinico-epidemiological Profile of Hidradenitis Suppurativa in Bangladesh. 2023; 03 (02): 67-72

Available at: www.jbadbd.com
An official publication of Bangladesh Academy of Dermatology (B.A.D.)

well as innate pro-inflammatory cytokines such as interleukins (IL-1β, IL-17), tumor necrosis factor (TNF-α), and interferon (IFN-γ), which leads to a vicious cycle of tissue destruction. HS is associated with a range of comorbidities, including acne, polycystic ovary syndrome (PCOS),metabolic syndrome, diabetes mellitus type 2, inflammatory bowel disease (IBD), psoriasis and many autoimmune disorders.

The persistent inflammatory nature of the disease significantly affects patient's quality of life, making patient vulnerable for social stigma, low mental health, and increase suicidal ideation in comparison to the general population. HS is diagnosed on the basis of clinical features of typical HS lesions located mainly in the intertriginous areas with additional supportive evidence of radiological (high-frequency ultrasonography and magnetic resonance imaging) and histopathological features. HS reasonably mimics with acne, follicular pyoderma, furuncles, carbuncles, abscesses, scrofuloderma, actinomycosis, lymphogranuloma venereum, granuloma inguinale, Crohn's disease, Bartholin cysts, metastasis and many other diseases. The level of awareness regarding this disease among general population and non-dermatologist physicians is low even in high prevalent countries. Delayed diagnosis, wrong diagnosis, under treatment of HS and drop out from treatment is a consequence. Hidradenitis suppurativa has a wide range of prevalence ranging from 0.00033% to 4.10%. Though the exact epidemiological data of HS from South Asia, especially from Bangladesh is not availableand according to our practical experience HS can be considered a rare condition in Bangladesh. The current study was conducted to analysis demographic, epidemiological and clinical profile of Bangladeshi patients of HS.

Methods

It was a multi-center cross sectional observational analytic study conducted from May 2022 to April 2023 in Bangabandhu Sheikh Mujib Medical University (BSMMU), Mugda Medical College, Dhaka and Central skin and social hygiene center, Agrabad, Chittagong. HS case detection was made on the basis of following criteria. A) Obligatory criteria: i) Typical history: Recurrent painful or purulent lesions more than twice/6 months; ii) Typical location: Groin, armpit, perineum, buttocks area and submammary/intramammary fold, iii) Typical clinical signs: Follicular papule/pustule (folliculitis), nodule (inflammatory or noninflammatory), abscess, cyst, fistula/sinus (exudative or nonexudative), double pseudocomedone, scar(atrophic, net-like, erythematous, hypertrophic, linear or bridged). B) Additional criteria (not obligatory) – HS-positive family history, no evidence of pathogens or presence of normal skin microflora at the predominant primary type of lesions. If all three obligatory criteria are present,or one or more obligatory locations are involved, one or more types of obligatory lesions are present then the diagnosis of HS was confirmed.13Information on age, sex, history of affected family members, smoking habit, age at onset of the disease, age at diagnosis, diagnostic delay, body weight (BMI), comorbidities, aggravating factors, anatomical area involved, and severity of the disease (Hurley stage) were recorded.Family history was considered positive ifany of first- or second-degree relatives had found as sufferer of HS.Comorbidities were also diagnosed on the basis of previous document, history, clinical and laboratory findings. BMI (kg/m2) values were divided as underweight (BMI < 18.5), normal (BMI 18.5–23.5), overweight (BMI 23.5–27.5) and obese (BMI > 27.5) according to the WHO guidelines for the Asian population.

HS was categorized according to the severity as three stages by Hurley classification system. Stage I – solitary or multiple isolated abscess formation without scarring or sinus tracts,Stage II – recurrent abscesses, single or multiple widely separated lesions, with sinus tract formation, and Stage III – diffuse or wide involvement, with multiple interconnected sinus tracts and abscesses.This study was approved by the Institutional Review Board (BSMMU/2022/4085; Date 23-4-2022) of BSMMU.

Result

Demographic and clinical data were collected from 23 patients, males were predominantly sufferer (male to female ratio 1.6:1) (Table I). In 16(69.6%) patients the disease was started after puberty (≥18 years) and the mean age of patient at the time of diagnosis was 28.4±3.7 years ranging from 16 to 74 years. The mean of delay for the diagnosis was 9.7 ±3.4years ranging from 2 to 25. A positive history of HS among first- or second-degree relatives was found in 2(8.7%) (Table I). Among male patients 6 (35.7%) were current or ex-smoker and65.2% were obese or over weight with a mean BMI 29.0±7.0.

Axillae was the most frequently (73.9%) affected site followed by groin (39.1%). HS severity was mild (Hurley I) in (52.2%), moderate (30.4%) and severe (17.4%). Acne was the most common (30.4%) comorbidity followed by diabetes mellitus, hypertension and hyperlipidemia. The rate of smoking and hypertension has no significant difference with normal population and obesity anddiabetesare significantly prevalent(Table II).

Table I: Demographic and clinical profiles hidradenitis suppurativa patients (n=23)

Variables		Frequency/percentage/Mean/Ratio
Sex (male:female)		14:9(1.6:1)
Age at disease onset (years)		
	<18 years	7(30.4%)
	≥18 years	16(69.6%)
Age at diagnosis: Mean, range (Years)		28.4±3.7,16-74
Diagnostic delay: Mean, range (Years)		9.7 ±3.4, 2-25
Family history		2(8.7%)
Smoker (current or ex-smoker)		
	Male (n=14)	6 (35.7%)
	Female (n=9)	0
BMI (Mean±SD)		29.0±7.0
	Normal	8 (34.8%)
	Over weight	9 (39.1%)
	Obese	6 (26.1%)
Involved site Obese		
	Axillae	17 (73.9%)
	Inguinal	9 (39.1%)
	Gluteal	3 (13.0%)
	inframammary	2(8.7%)
	Genital	2 (8.7%)
	Perianal	2(8.7%)
	Abdominal	2 (8.7%)
Disease severity		
	Hurley I	12 (52.2%)

Table 2. Comparison of smoking rate,BMI, diabetes and hypertension with the general population

Variables	HS patients(n=23)	General population	P value
Smoking rate	35.7%	36.0%[1]	>0.05
BMI (kg/M^2)	29.0±7.0	22.6± 3.7[2]	<0.01*
DM	21.7%	12.8%[3]	<0.01*
Hypertension	17.4%	17.9%[4]	>0.05

Discussion

The epidemiological studiesfromdifferent parts of the world has created controversies regarding the gender distribution of patients with hidradenitis suppurativa. Although in many of those it has been described as a female predominant disease and often attempted to justify this by disease onset, fluctuation and flare during the menstrual cycle, pregnancy, and menopause. In European and North American population HS is three times more prevalent among women. In our study male to female ratio was 1.6:1 which is consisted with previous Asian studies. - HS usuallyaffects after puberty with an average onset at age 23 years.

Average age of onset of HS among our patients were 28.4±3.7 years ranging.ThoughHS is particularly rare in young and prepubescent children,20in the current study 30.4% manifested before the age of 18 years and the youngest at the age of 14. Diagnosis of HS is often challenging, very often they misdiagnosed by general physicians or other specialists before a correct diagnosis as HS, patients also feel ashamed to present the disease as it mostly involves relatively sensitive areas including axillae, groins, buttock, breast and genitals. Globally an average delay before a correct diagnosis of HS,patient suffersfrom 7 to 10 years.7In this study the mean delay of diagnosis was 9.7±3.4, one patient of 74-year-old age suffered about 25 years before diagnosis as HS.

Bangladesh has one of the largest tobacco consuming populations (37.8million adults) in the world where 36% of the adult male and 0.8% women smoke tobacco. ,16 In the current study none of the women and 35.7% of male were smoker. The rate of smoking was indifferent among patients of HS comparing with general population.16,23In the current study 8.4% had affected family member though in the previous cohort 30-40% of patients of HS have shown positive family history ofthe disease. Genetic susceptibility and environmental influences of shared microbiome, diet, and obesity among related subjects could explain familial clustering. Obesity is considered as a risk factor of HS though the precious mechanism is not yet clear and the relationship between BMI and impact of HS is non-linear. - Approximately two thirds of HS patients were overweight or obese and the mean BMI was significantly higher than the general population.17Axilla was the exclusively commonest site of hidradenitis suppurativafollowed by groin which is consistent with previous studies,2 but in some studies groin is the mostly affected site. Schrader et al, found that lesions on axillae, groins and breast areas are associated with more severe disease.24Among our patients about fifty percent was mild (Hurley I), similarly in previous studies in

Argentina and South Korea majority of patients of HS were offollowed by Hurley II and Hurley III to a lesser extent. , Patient of HS often suffer with different comorbidities which make patients dissatisfied with their treatment. Acne was found as a comorbidity in 30.4% of our cases and one patient (4.3%) had pilonidal sinus. In previous studies pilonidal sinus was foundin 4.6% to 31% cases of HS and acne were associated in 13% to 36%. , In comparison with common people, individuals with HS are nearly 3 times more prone to develop diabetes mellitus. The overall age-standardized prevalence of diabetes in Bangladesh is 12.8%.18 whereas diabetes was significantly higher (21.7%) among patients with HS ($p<0.01$). The rate of hypertension in patient with HS was very comparable to the national prevalence.

Conclusion

In Bangladesh, HS affects predominantly males after puberty and the diseaseis diagnosed at late part of third decadeof age usually after a suffering of approximately ten years. Diabetes mellitus and obesity are significantly higher among patients with HS but rate of smoking and hypertension were comparable with the national prevalence. Majority of the lesions were located at axillae and severity was mild (Hurley I).

Limitations

Small number of samples.

Conflicts of interest

None

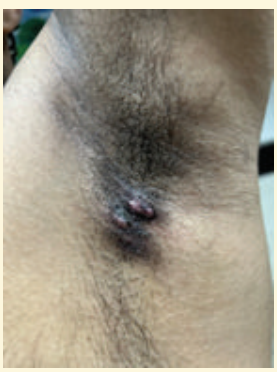

Figure 1: HS lesion on axilla

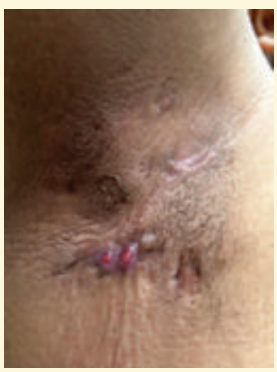

Figure 2:HS with interconnecting fistulae

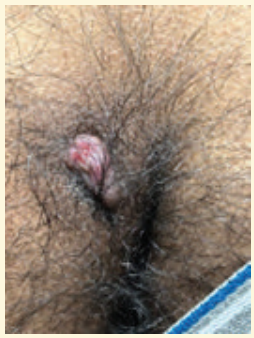

Figure 3: HS with Pilonidal sinus

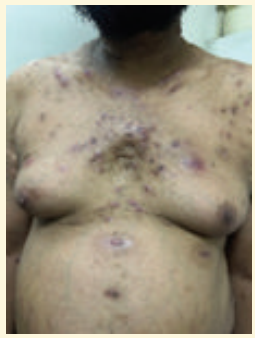

Figure 4: HS lesion on intermammary area

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Case report:

Congenital Erythropoietic Porphyria (CEP): A rare disease

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Abstract

Congenital Erythropoietic porphyria (CEP or Gunther disease) is a rare autosomal recessive inborn error of heme biosynthesis with a mutation in the gene that codes for uroporphyrinogen III synthase (UROS), resulting in severe loss of activity of UROS and elevations of uroporphyrin I and coproporphyrin I. This deficiency is associated with the accumulation of porphyrins and porphyrin precursors in the erythrocytes, plasma, and, urine which, in turn, causes blistering over sun-exposed areas and chronic severe photosensitivity. Here, we reported a 10-year-old boy, born of parents of consanguinous marriage presented with recurrent blistering over sun-exposed skin from the age of 2 and reddish urine from birth. There was erythrodontia, hyperpigmentation, atrophic scarring, hypertrichosis predominantly in the face and extremities, and deformities of fingers. Bright red fluorescence was noted in the urine and teeth under the wood's lamp. Histopathology revealed subepidermal bulla. Based on history, clinical examinations, wood lamp examination, and histopathology we diagnosed the case as congenital erythropoietic porphyria. We recommended absolute photoprotection, using high sun protection factor sunscreens, vitamin D supplementation to compensate for the lack of sun exposure, and psychotherapy to minimize the psychosocial impact.

Keywords: Congenital Erythropoietic Porphyria, Uroporphyrinogen III synthase (UROS), Cutaneous blistering, Subepidermal bulla

Introduction

The first documented human porphyria is congenital erythropoietic porphyria (CEP). It is related to the disturbance of the porphyrin metabolism.[1] Gunther defined the disease as 'haematoporphyria congenital' in 1911–12.[2] Congenital erythropoietic porphyria (CEP) is a rare genetic disease inherited as an autosomal recessive trait.[1-2] The main pathology is the remarkable deficiency of the fourth enzyme of the haem biosynthetic pathway, uroporphyrinogen III synthase.[3] This enzyme defect results in the accumulation of pathogenic porphyrin precursors, uroporphyrin I and coproporphyrin I.[1-3] The accumulation of porphyrins results in unique cutaneous manifestations, such as bullae and vesicles on sun-exposed regions of skin, scarring, erythrodontia, hypo or hyperpigmentation and hypertrichosis.[4] Visceral complication, hemolysis, and growth retardation are also reported to complicate the disease.[3-4] Congenital erythropoietic porphyria is an extremely rare disease; estimated prevalence is around 1 or less in 10,000,00 population.[5] To our knowledge, only one case was documented in Bangladesh before. However, another case of this rare porphyria was reported in Bangabandhu Sheikh Mujib Medical University Hospital (BSMMU), Dhaka, Bangladesh at the

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Cite this Article:
Chowdhury MAH, Malabeka S, Tahnin NAT, Uddin MJ, Islam MA. Congenital Erythropoietic Porphyria (CEP): A rare disease. Ban Acad Dermatol. 2023; 03 (02): 73-77

Available at: www.jbadbd.com
An official publication of Bangladesh Academy of Dermatology (B.A.D.)

outpatient department of Dermatology & Venereology in 2023.

Case report

Our patient is a 10-year-old boy who presented to the outpatient department of the hospital with complaints of reddish urine from birth and recurrent blisters over sun-exposed skin from the age of two. Blisters were tense and non-itchy, prominent over the dorsum of hands, feet, and face which were exacerbated by exposure to sunlight. Some blisters ruptured to form erosion, and some became ulcerated, leaving atrophic scar and hypopigmentation on healing. Since birth, his parents noticed red urine staining the diaper without any noticeable discomfort during micturition. Parents also noticed eruption of red teeth at the age of 1 year of their child. The patient was born into a consanguineous marriage. He had one younger brother who was healthy. Both his parents were phenotypically normal. His gestational period and delivery remained uneventful. He was reported to be healthy at birth, made normal progress, and achieved age-appropriate developmental milestones. The boy was vaccinated as per the national EPI schedule. A detailed history revealed that the child had multiple visits to the local doctor for his complaints and was given multiple antibiotics without any significant improvement. He lives with his family and comes from a low socio-economic status.

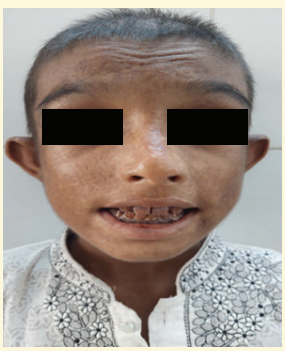

Fig. 1: Erythrodontia

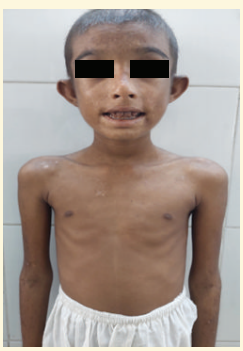

Fig. 2: Scarring, hypopigmentation, and hypertrichosis

The complete general examination showed that the patient was well-oriented with normal vital parameters. The patient was anemic and had reddish teeth, and facial scars with thin, sparse hair. There were multiple atrophic scars, hypo, and hyperpigmented macules over the face, dorsum of hands, feet, and V area of the chest and back with facial hypertrichosis. No intact blisters were found, but there were some erosions and crusting more marked over the dorsal hands and elbow. There was flexion deformity of the distal interphalangeal joints of both hands. There was discoloration of toenails with increased nail fragility and subungual hyperkeratosis of great toenails. However, the fingernails were normal. He had a soft, non-tender abdomen with a just palpable spleen. All other systems revealed no abnormality. Based on the history and clinical examination, our primary diagnosis was congenital erythropoietic porphyria.

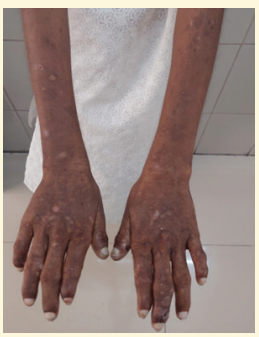

Fig. 3: Atrophic scarring, hypopigmentation, crusting and deformity of hands

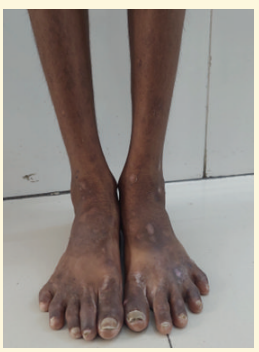

Fig. 4: Atrophic scarring and nail changes of both feet

To assist with the clinical diagnosis, further laboratory investigations were performed to confirm the diagnosis. Wood's lamp examination revealed coral red fluorescence of teeth and urine. Lab investigation revealed moderate microcytic hypochromic anemia, Moreover, a higher red cell distribution width (RDW), and a normal white blood

cell and platelet count. The peripheral blood film revealed microcytic hypochromic red blood cells (RBCs) with anisopoikilocytosis, few target cells, and marked rouleaux formation. Ultrasonography revealed splenomegaly without hepatomegaly. Histopathology revealed subepidermal bulla, dermal edema, extravasated RBC, thickened and hyalinized dermal blood vessels which were suggestive of erythropoietic porphyria. Porphyrin levels were not measured and genetic testing was not performed due to unavailability.

Fig. 5: Red urine

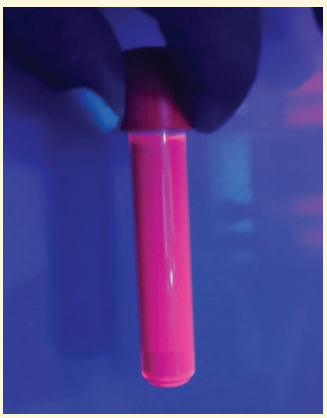

Fig. 6: Coral red fluorescence of urine

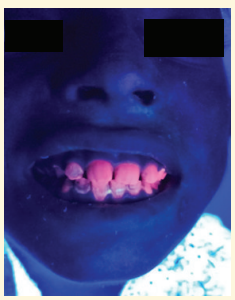

Fig. 7: Coral red fluorescence of teeth

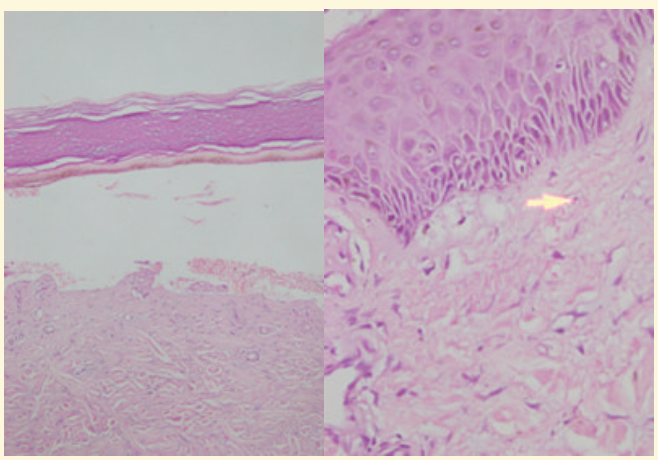

Fig.8: Sub-epidermal bulla, dermal edema, extravasated RBC, thickened and hyalinized dermal blood vessels with few chronic inflammatory cell infiltrates in the dermis.

Discussion

CEP is a very rare form of porphyria inherited by an autosomal recessive trait that causes the skin to be highly sensitive to sunlight.[5] The underlying defect is mutations of two allelic genes encoding the enzyme uroporphyrinogen III synthase that leads to the accumulation of the porphyrin precursors, coproporphyrin I and uroporphyrin I.[6] The C73R mutation is the most frequently mutated genes. The co-inheritance of gain of function mutation in ALAS2 can lead to a more severe phenotype.[7] The accumulated precursor porphyrinogens are spontaneously oxidized to their corresponding porphyrins. These porphyrins are biologically inert but cause cutaneous features especially photosensitivity which leads to the formation of blisters, erosions, scarring, and mutilating deformity of photo exposed skin.[6-7] These porphyrins are released from the maturing erythrocytes into the plasma and are excreted in urine, thereby producing a reddish color urine.[7] The primary dentition of patients with CEP shows a deep red-brown discoloration due to porphyrin accumulation.[7-8] Porphyrin concentration is much higher in the dentin than in the enamel due to the affinity of porphyrins to calcium phosphate.[8] The reddish-brown color of urine together with red teeth is the characteristic feature for early diagnosis of CEP.[8] CEP presents soon after birth, but uncommonly may present in adulthood. Symptoms that appear in adulthood tend to be milder than symptoms that begin early in life.[7-8] In our context, the 10-year-old boy manifested the symptoms since birth with red urine. Characteristic erythrodontia was present of both permanent and deciduous teeth since its eruption. Photosensitivity started from the age two which also suggested the early childhood manifestations of the disease. However, photo- sensitivity was not severe enough to cause immediate pain and burning leading to screaming when exposed to sun. Blistering and scarring were more marked on the photo exposed area with limited involvement of scalp. In severe cases, ectropion with corneal damage and loss of vision may result.[9] But in our case the child did not complain any eye symptoms, probably due to less severity of the disease. Hypertrichosis of the cheeks and long eyelashes were evident. Atrophic scarring was present on dorsal hands and face with flexion deformity of fingers without any mutilating scar. Onset at birth, absence of GIT and neurological symptoms (seizure,

psychosis) aided us to exclude other variants of porphyria (porphyria cutanea tarda, variegate porphyria, erythropoietic porphyria).

Systemic features of CEP include growth retardation, hemolytic anemia, thrombocytopenia, porphyrin gallstones, organomegaly, osteopenia, and increased rate of fracture.[10] However, in our case patient did not have any growth retardation and achieved developmental milestones in time. He had microcytic hypochromic anemia, probably due to iron deficiency which could be a cause of nutritional deficiency, as nutritional anemia is more prevalent in developing countries among those with low socio-economic status.[10] The Patient had splenomegaly without hepatomegaly. Apart from anemia and splenomegaly, he didn't have any other systemic features. Wood's lamp examination of both urine and teeth showed coral red fluorescence, which is a unique and characteristic feature of CEP. This unique finding led us to diagnose the disease in the very first place. Skin biopsy showed subepidermal bulla and extravasation of RBC which was consistent with the biopsy findings of CEP. However, the porphyrin assay, which is an important diagnostic marker for CEP, could not be performed due to unavailability. Moreover, the severity of the disease is directly related to the plasma porphyrin level and the residual activity of UROS. Abnormal and high levels of uroporphyrin I and coproporphyrin I are found in urine, stool, and RBC.[8-10] Therefore, in our patient severity of the disease could not be determined. But clinically it seemed to be a case with mild to moderate severity. Therefore, based on the characteristic's clinical findings, laboratory investigations, wood's lamp, and biopsy findings we have labeled the case as CEP.

Treatment of CEP is challenging and there is no standard therapy exists. Treatment is based mainly on the clinical manifestation. Currently, the only curative treatment option is allogeneic bone marrow transplantation.[9-11] In patients who do not undergo this procedure, strict avoidance of sunlight use of sunscreens containing zinc oxide or titanium oxide, vitamin D supplementation, and use of sunglasses to avoid ocular complications is the mainstay of therapy. Other options include activated charcoal, presumably impairing the absorption of endogenous porphyrins, phlebotomy, and repeated transfusion of packed RBC to maintain the hematocrit level.[10–12] In some patients with hypersplenism and excessive circulating RBC trapping and destruction, splenectomy may reduce the need for transfusions.[11-12] In our case, since the patient had mild to moderate symptoms, we opted for general management options which include, sun avoidance, the use of sunscreen, vitamin D and Zinc supplementation. The patient was advised for regular follow-up to observe the course of the disease and assess the prognosis.

We understand that early detection of CEP symptoms is crucial for accurate diagnosis and treatment. Careful observation and diagnostic approach can help us to differentiate it from other overlapping porphyrias at an early stage. It is also important to note that CEP is a lifelong condition, and patients will require lifelong monitoring. Recognizing the disease at its earliest stages is important to assist clinicians in providing appropriate patient-centered treatment and to address the quality of life by educating and counseling on preventive measures and providing psycho-social support.

Conclusion

CEP is a very rare form of porphyria. Therefore, we reported this case to highlight the varied clinical presentation and useful diagnostic approach despite having limitations in performing porphyrin assay and genetic testing. We understand this can aid in raising awareness among fellow clinicians. We hope our findings can further advance the understanding of this rare disease and treatment options. The rarity of the disease and the chance of under-reporting prompted us to present this case report.

Conflict of Interest

None

Funding source

None

Patient Consent

Taken

IRB approval status

Not applicable

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Case report:

Crusted Scabies

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Abstract

Crusted scabies (Norwegian scabies) is an extremely contagious variety of scabies in which millions of mites inhabit in skin. It occurs more frequently in people who have immune suppression due to a disease or medical treatment. Patients with crusted scabies typically have thicker, fissured hyperkeratotic crust. In the context of the application of topical corticosteroids, we describe the case of a 17-year-old patient who had hyperkeratotic scabies. He was successfully treated by using a combination of topical permethrin and oral Ivermectin.

Introduction

A skin condition known as scabies is brought on by the infection of the human-specific ectoparasite Sarcoptes scabiei var. hominis, which has been classified as a neglected tropical illness.[1]

Crusted scabies is a severe and contagious form of scabies in which millions of mites proliferate and cause widespread hyperkeratotic crusting of the skin.[2] The clinical appearance of crusted scabies differs from typical scabies in that it is characterized by extensive local or diffuse hyperkeratosis on an erythematous background, as well as crusting and fissures on various body areas.[3]

Scabies spreads globally and has an impact on all social groups and communities. Because these individuals continue to be contagious for a very long time, it is very difficult to get rid of the mites from the parts of the skin that are heavily crusted.4 It spreads through close personal contact or indirectly through fomites (clothes or bed linens).[2] Crusted scabies is more likely to affect patients who have systemic or powerful topical glucocorticoids, organ transplant recipients, immunocompromised, malnourished, and people with human immunodeficiency virus (HIV).[1]

Clinical observations and microscopic inspection of scales obtained by skin scraping are used to confirm the diagnosis. The burrow ink test, video-dermatoscopy, recently developed serologic assays like PCR/ELISA, and specific IgE targeted toward key mite components are further diagnostic techniques. To stop an outbreak of scabies, patients with crusted scabies must be strictly isolated. Ivermectin has been successfully utilized in the treatment of crusted scabies, either alone or in conjunction with other scabicidals.[5-7]

In view of the delayed diagnosis and incorrect application of topical corticosteroids, we report a patient who had crusted scabies.

Case report

A 17-year-old boy from Rajbari, Bangladesh, complained of 2-month-long crusting, widespread erythema, and itching when he visited the department of Dermatology and Venereology at BSMMU. He had a small red papule and vesicle covering his entire cutaneous surface two months prior. His general physician had prescribed him topical steroids for his pruritus for an extended period. His symptoms initially got better, but after a few days they came back with a thick crusted lesion. When the skin was examined, it showed widespread erythema, numerous fissured hyperkeratotic lesions, extensive scales, primarily across the trunk and limbs, and pedal oedema Fig1(a, b).

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Cite this Article:
Pudel H, Uddin MJ. Crusted Scabies. Ban Acad Dermatol. 2023; 03 (02): 78-80

Available at: www.jbadbd.com
An official publication of Bangladesh Academy of Dermatology (B.A.D.)

For a time, the patient was unable to attend school. The lesions were painful and tender hampering his regular activities. His family members similarly experienced excoriations and pruritic popular lesions. After admitting the patient, we immediately scraped the hyperkeratotic lesion, placed the sample on a slide, and added a drop of mineral oil. Microscopic examination revealed Sarcoptes scabiei mite, fig 2. It was determined that the patient had crusted scabies. He received a combined treatment that included oral ivermectin (200 mcg/kg/dose) and topical permethrin 5%.

Oral ivermectin was given on day 1,2,8,9, and 15. Permethrin 5 % cream was applied every two to three days for two weeks. Antihistamine was administered for pruritus. His family member was also treated with anti-scabietic therapies. After one week of treatment, the patient significantly improved (fig. 3a-b). We chose to continue treatment with outpatient care when the lesion began to regress.

Discussion

We describe the case of a 17-year-old boy without any comorbidity but had a history of long-term use of steroids, which may contribute to the host's susceptibility to infestation by Sarcoptes scabiei. Crusted scabies is more common in immunocompromised, underweight and handicapped individuals, but this case is of a young boy with crusted scabies due to delayed diagnosis and inappropriate use of topical steroids.

Crusted scabies is a serious public health issue, and misdiagnosis can have further negative health and economic effects, particularly when outbreaks occur in medical facilities.[4] Topical corticosteroids reduce local cell-mediated immune responses when used for an extended period, allowing one to develop this severe form of human scabies.6 Scabies epidemics can cause long-term health effects (such as heart and kidney problems) that can have a major financial impact on national health services.[4]

The basis of therapy is an early diagnosis and appropriate care. The systemic medication of preference is ivermectin, and many treatments may be necessary. Never disregard the contact individual's prophylactic treatment.[5] Since mites die over 72 hours when they are isolated from their human host, clothing and linens should either be kept in a plastic bag or machine-washed at a temperature of at least 50 degrees Celsius to avoid re-infestation. If there are no signs of active scabies (no active lesions, no nocturnal pruritus) one week following the completion of therapy, the infestation is considered to be under control. Itching following therapy could last for up to 2-4 weeks.[7-8]

Conclusion

It is essential to suspect someone having scabies if they have a pruritic popular lesion and a favourable family history. Its outbreaks and life-threatening complications can be avoided with prompt diagnosis and appropriate treatment. The hospital staff or household contacts should be advised to take the necessary steps to avoid becoming infected.

Conflict of Interest

None

Funding source

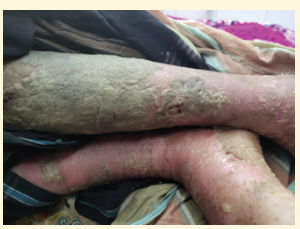
None

Patient Consent

Taken from parents.

IRB approval status

Not applicable



a.

b.

Fig 1(a) hyperkeratoticlesions with fissuring on the trunk and upper extremities; 1(b) hyperkeratotic lesions on the lower extremities with bilateral pedal edema.

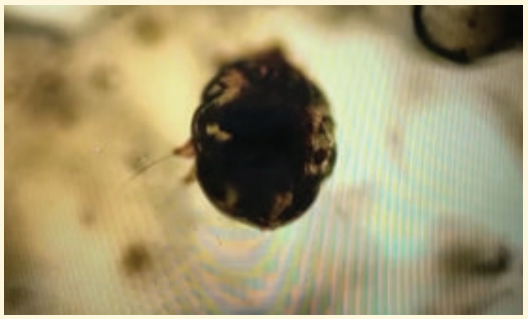
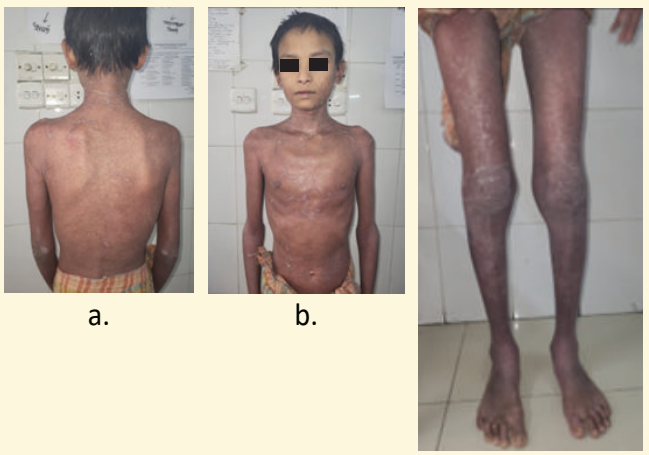


Fig 2. Sarcoptes scabiei on microscopic examination
(Photo: Bhuiyan Mohammed Saiful Islam Bhuiyan)



a. b. c.

fig 3(a,b,c) regression of the lesions after a week.

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Case report:

Disseminated superficial actinic porokeratosis - a rare case report

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Abstract

Porokeratosis is a heterogeneous group of keratinization disorders characterized by papules and plaques with central atrophy and a hyperkeratotic margin. We describe a woman who had DSAP or disseminated superficial actinic porokeratosis, whose lesions appeared as itchy plaques and papules. Clinical evidence suggested the diagnosis and histologically confirmed. The pruritus brought on by the condition was treated symptomatically, Her skin lesions will be clinically monitored. This rare dermatosis requires clinical and histopathological correlation to diagnose at an early stage and to avoid under-reporting.

Keywords: Cornoid lamella, disseminated superficial porokeratosis, porokeratosis

Introduction

Porokeratosis is a rare keratinization disorder that has the clinical appearance of papules encircled by a clearly defined peripheral keratotic wall, corresponding histologically to a "cornoid lamella".[1] Disseminated superficial actinic porokeratosis (DSAP), disseminated superficial porokeratosis, linear porokeratosis, porokeratosis of Mibelli, porokeratosis palmaris et plantaris disseminata, and punctate porokeratosis are some of the common clinical variations of porokeratosis; less common porokeratosis subtypes are Eruptive bullous, follicular, genitogluteal, lichen planus-like, porokeratotic acanthoma, porokeratotic adnexal ostial nevus, and pruriginous. The most prevalent clinical form of porokeratosis is DSAP, characterized by the bilateral appearance of numerous papules and plaques over sun-exposed areas, particularly on the distal limbs. [2] The lesions are slightly reddish or brownish in colour, and the surrounding ridge usually appears more accentuated than the circumscribed interior. As the lesions centrifugally expand, an annular or irregular configuration is typical.[3] DSAP has been reported in Patients with AIDS, cirrhosis, Crohn's disease, and organ transplant recipients.[1] DSAP is considered a rare disorder; no data exist on the prevalence of the disease in our country.

Case report

A 31-year-old woman, garments worker, presented to the outpatient department with a history of multiple brownish annular macules, patch and plaques over face, trunk, and upper limbs and lower limbs for the past 14 years. The lesions started appearing as patch, brown in colour, then slowly progressed in size as a plaque with a keratotic rim. initially over the face, followed by the chest, back, and upper limbs. The lesions gradually increased in size and number. There was no photosensitivity. Her

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Cite this Article:
Haque AKM R, Mahmud MM, Ashadullah SM, Haque MA, Mia MT. Disseminated superficial actinic porokeratosis - a rare case report. Ban Acad Dermatol. 2023; 03 (02): 81-83

Available at: www.jbadbd.com
An official publication of Bangladesh Academy of Dermatology (B.A.D.)

younger brother has same kind of illness for the last 7 years.

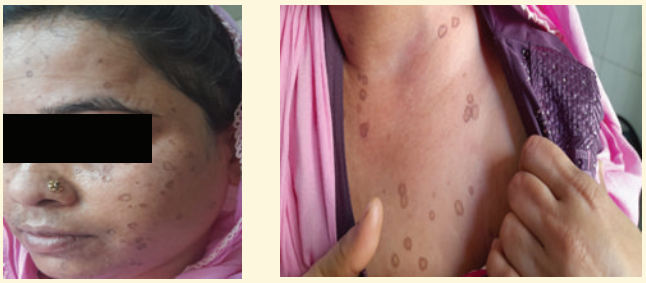

Fig 1: Multiple hyperpigmented annular macules,patchs & plaques over the face,chest

On physical examination the lady appeared to have normal well-being, vitals are within normal limit, cutaneous examination shows hyperkeratotic annular plaques with raised margin over the face, neck, trunk and arms. Other systemic findings were normal. Laboratory findings including Complete Blood Picture, Urine Routine Examination, SGPT, Serum creatinine, HBsAg, Anti HIV (1 and 2), CXR, USG of whole abdomen, were normal. Histopathological study showed epidermis is thin, show mild hyperkeratosis, parakeratosis and focal vacuolar alteration of basal layer. The dermis shows mild perivascular infiltration of chronic inflammatory cells including a few melanophages (Fig. 2).

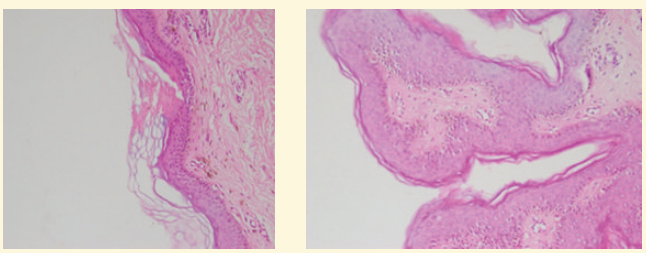

Fig 2: Histopathology of the trunk lesion showing vertical layered parakeratosis, the cornoid lamella and vacuolated keratinocytes

Discussion

Porokeratosis is an abnormal differentiation of keratinocytes rather than hyperproliferation. It can appear as a single lesion or multiple lesions, and as a localized or disseminated form. The cornoid lamella, which histopathologically represents the raised border, is made up of a thin vertical tier of parakeratoses.[4-5] Different forms of porokeratosis frequently appear in particular age groups. Classical porokeratosis of Mibelli and linear porokeratosis typically appear during infancy or childhood, whereas disseminated palmoplantar porokeratosis and punctate palmoplantar porokeratosis typically appear during adolescence. DSAP typically first appears in adulthood. Males are more likely to develop porokeratosis of Mibelli, genital porokeratosis, and punctate porokeratosis, whereas females are more likely to develop DSAP.[6] Our patient is an adult female. Although the pathogenesis of porokeratosis is still not fully understood, DNA polyploidy, as well as keratinocyte and fibroblast sensitivity to ionizing radiation in the affected skin, have been shown to play a role in the pathogenesis.[7] Porokeratosis has been linked to a number of risk factors, including genetic predisposition, exposure to UV rays, and immunosuppression.[8] Our patient has a positive family history. Disseminated superficial actinic porokeratosis lesions begin as asymptomatic or mildly itchy pink to brown papules and macules with raised borders in sun-exposed areas. The most frequently affected body parts are the legs, forearms, shoulders, and back, the face can rarely be affected, the palms and soles are unaffected. When exposed to sunlight, the lesion typically gets worse, and pruritus can get worse.[9] But, in our case, the lesions 1st appear on the face and has no photosensitivity. A skin biopsy should be performed that includes the lesion's border. The cornoid lamella is a column of parakeratotic cells that corresponds to the raised border of the lesion. The granular layer beneath this column can be thin or absent. There is dyskeratosis in the epidermis beneath the cornoid lamella. Spongiosis can be present. Our patient's histopathology report shows a thin epidermis, mild hyperkeratosis, parakeratosis and focal vacuolar alteration of the basal layer. The dermis shows mild perivascular infiltration of chronic inflammatory cells including few melanophages which is suggestive of DSAP. Precancerous status is assigned to these lesions. The likelihood of malignant transformation into basal cell carcinoma or squamous cell carcinoma is between 7.5 and 10%.[10]

Conclusion

The case in this report had late-onset DSAP. Her diagnosis was clinically suspected, and microscopic analysis of a tissue biopsy specimen confirmed it. The lesions run the risk of developing into malignancies. photoprotective measures should be encouraged to reduce the risk of further skin

damage . Patients should also visit frequently so that their lesions can be monitored.

Conflict of Interest

None

Funding source

None

Patient Consent

Taken from parents.

IRB approval status

Not applicable

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Case report:

Glomus Tumour On The Hypothenar Eminence Of Hand: A Case Report

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Abstract

Glomus tumour is an uncommon vascular tumours, usually presenting with painful bluish-red lesions with localized point tenderness and hypersensitivity to cold. Glomus tumours are located mostly in the subungual region of the digit, but involvement in other sites has also been reported. We here describe a 21-year-old male presenting with a painful plaque on the hypothenar eminence of the left hand, diagnosed as glomus tumour. It is a treatable condition, and proper diagnosis & treatment can significantly improve a patient's quality of life.

Keywords: Glomus tumour, hypothenar eminence, glomus body, extradigital

Introduction

Glomus tumours are mostly benign vascular tumours that develop from a neuromyoarterial structure named a glomus body.[1] Glomus body is a particular form of arteriovenous anastomosis commonly located in the reticular layer of the dermis, which carries glomus cells that are modified vascular smooth muscle cells and under normal circumstances regulate temperature and blood flow to the skin.[2] Common clinical presentation of glomus tumour includes a triad of paroxysmal intense pain, localized point tenderness, and marked sensitivity to cold.[3] Glomus tumours represent only 1%-5% of all hand tumours.[4] Though some Studies have described that glomus tumours may located in different sites of the hand and body, the most frequent site is a subungual area of the digit.[5] As the prevalence of glomus tumours is low, an uncommon presentation of glomus tumours may cause a delayed diagnosis, especially for extradigital glomus tumours.[3] This case report describes a glomus tumour presented on the hypothenar eminence of the hand.

Case Presentation

A 21-year-old male patient presented to the dermatology outpatient department with a painful plaque on the palmer aspect of his left hand. He first noticed the lesion about two years back. Initially, it was small in size and associated with mild pain. Recently his pain became more intense. Though he could not remember any incident of injury at that site, he thought that it might be due to a throne entry. His pain was persistent, aggravated by touching. On asking patient mentioned that the severity of pain increased during winter. Cutaneous examination revealed a well-defined, rounded erythematous

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Cite this Article:
Shaila KN, Nasim R, Wahab F, Amin MSTZ, Akter N, Tabashum T. Glomus Tumour On The Hypothenar Eminence Of Hand: A Case Report. Ban Acad Dermatol. 2023; 03 (02): 84-87

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plaque on the hypothenar eminences of the left hand. It was measuring about 1.5 cm x 1.5 cm.

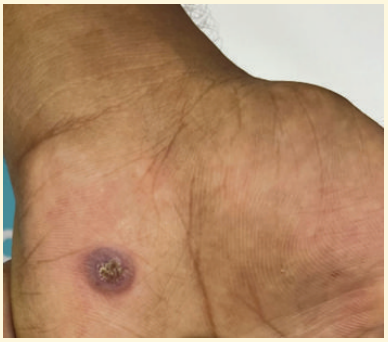

Fig 1: Shows an erythematous plaque on the hypothenar eminence of the left hand

The plaque had a thickened- scaly center, with a blanchable peripheral erythema. On touching the lesion, it felt firm, and remarkable tenderness was found by applying pressure to the lesion. With these clinical findings, we considered the following differential diagnoses: foreign body reaction, glomus tumour, viral wart, and neuroma. As the patient had complained of intense pain, excision of the lesion followed by histopathological examination was planned. The patient was not suffering from any other medical illness and his all vitals were normal. An excisional biopsy was performed.

Microscopic examination of the specimen revealed a benign tumour composed of glomus cells arranged in nests associated with vascular channels with connective tissue stoma.

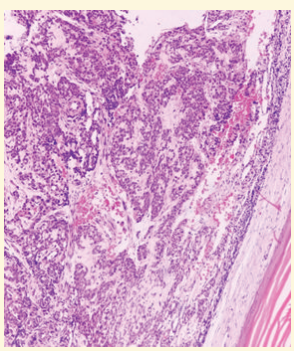

Fig 2: Shows clusters of glomus cells with varying sizes of blood vessels in a hyalinized stroma.

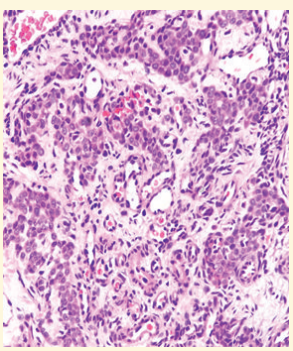

Fig 3: Shows rounded uniform tumour cells with scanty cytoplasm.

With these clinical and histopathological findings, he was diagnosed with a case of extra digital glomus tumour. The patient was advised to have a follow-up visit 2 weeks following the procedure, at that time his pain was completely disappeared.

Discussion

Glomus tumour was first reported by Wood in 1812 as a subcutaneous tubercle that was painful, and its common location as a subungual region was described by Kolaczek in 1878. Later on, Barre and Masson defined that it originated from the glomus body.[6]

Glomus tumours are uncommon, subcutaneous benign neoplasms.3 Glomus bodies play an important role in thermoregulation.[7] Though glomus bodies are located in the fingers, palms, and soles, higher densities of glomus bodies are found in the nail bed.[8]

The exact cause of pain in glomus tumour is not known yet, although various hypothesis exists: various substances such as heparin, histamine, 5-hydroxytryptamine, etc. are released from mast cells present in the glomus body in response to pressure and cold, producing severe pain even with minimal stimulus.[9] Glomus tumour is penetrated by several non-myelinated nerve fibres that also contribute to the pathogenesis of pain.[10] Besides, the contraction of myofilaments due to temperature changes increases intracapsular pressure that produces pain.[9] Glomus tumours are usually benign tumours. In the recent past, The World Health Organization has described the presence of malignant glomus tumour and classified the tumour as benign glomus tumour, intermediate glomangiomatosis, and malignant glomus tumour.[11] A benign glomus tumour commonly presents with a single lesion. But in the case of hereditary glomus tumours, which come into the autosomal dominant way, multiple tumours may present.[6] Glomangiomatosis is a diffuse glomus tumour.[11] Glomangiosarcoma is another name for malignant glomus tumours, they can appear as individual cancerous tumours or less frequently from a pre-existing benign glomus tumour.[12]

Sites of involvement of these vascular tumours can be extensively diverse and even have been found in mucosa or viscera.[5,7] Although glomus tumours are commonly seen in the skin, also found in other sites of the body, such as in the trachea, lungs, stomach, bones, mucous membrane, etc..[7] In the case of hand glomus tumours, they usually appear in the subungual region, the pulp and lateral aspects of fingers, and the palm.[8] Based on location, glomus tumours are categorized into- digital and extradigital, where sub ungual digital tumours are more common.[7] Regarding age and sex distribution, digital glomus tumours, particularly subungual tumours occur very often in women of younger age, whereas extra digital

tumours are common in elderly men.[7,13] Classic clinical findings of glomus tumours that comprise painful bluish-red lesions with localized pinpoint tenderness, and cold hypersensitivity are more frequently seen in digital subungual glomus tumours. Hence, in the case of extra digital tumours, there is often a long lag period before diagnosis as a glomus tumour.[3] In our study, an extradigital glomus tumour was noted in a young male patient.

Several studies were analyzed to outline the clinical presentation of a glomus tumour in the hand.[5] A case series by Saaiq et al. shows that in all patients with glomus tumours on hand, only digits were involved. 82.35% of patients had subungual glomus tumours and 17.64% had glomus tumours on the volar pulp of the finger.[4] Another study conducted by Hamdi et al also revealed the localization of glomus tumours on digits.[14] In this case report, the location of the glomus tumour was the hypothenar eminence of the hand.[3] Although in literature a small number of cases with glomus tumours in the wrists have been described.[12,15-16] There was a report of a case with a glomus tumour that happened in the hypothenar area of the palm. Typically, clinical characteristics of glomus tumours include a triad of paroxysmal pain, marked pinpoint tenderness, and cold hypersensitivity, mostly appearing as a red or blue-red colored, painful nodule localized in the subungual area of the digits.[16-17] In this case, the patient was presented with a painful erythematous plaque with a thickened-hyperkeratotic centre on the hypothenar eminence of his left hand and severe tenderness was present on touching. To aid the diagnosis of glomus tumours some physical examinations can be performed that include Love's pin test, Hildreth's test, and the cold sensitivity test.[17] In Love's pin test, a pin is used to apply pressure to the tumour, and that causes severe pain in patients with glomus tumours.[5] Hildreth's test is positive if pain and tenderness suddenly disappear when the affected areas become ischemic by applying a proximal tourniquet.[3] Cold sensitivity test reveals severe pain is felt in the affected area, in response to submerging the area in ice-cold water.[3]

To diagnose glomus tumours imaging modalities may help, such as plain X-ray, Doppler ultrasound, magnetic resonance imaging, etc..[5] Although with these clinical and radiographic findings, a possibility of glomus tumours can be considered, a microscopic analysis of lesions is needed to confirm the diagnosis of glomus tumour.[6] Histologically, glomus tumours are composed of a varying proportion of glomus or tumour cells, blood vessels of varying sizes, and smooth muscle cells.[3] Depending on these findings, histologically glomus tumours are categorized into three subtypes- solid tumour, glomangioma, and glomangiomyoma.[6] The common variant is solid glomus tumour, where glomus cells are numerous, with a few blood vessels and smooth muscle cells. In the case of glomangioma blood vessels are predominant, while in glomangiomyoma, there are few glomus cells, but the proportion of blood vessels and smooth muscle cells both are high.[6] Immunohistochemical evaluation of glomus tumours reveals, that tumour cells are usually positive for SMA, calponin, vimentin, and H-caldesmon.[18] Our patient's histopathology report shows, a benign tumour composed of glomus cells arranged in nests associated with vascular channels with connective tissue stoma. The tumour cell was small, round, and uniform with scanty cytoplasm. No malignancy was seen.

Glomus tumours are commonly painful tumours.[6] As a result of suspicion of other painful conditions, such as neuroma, etc., there may be a recurrent misdiagnosis.[19] Diagnosis of glomus tumour is often delayed, especially for atypical locations. The case report shows the presence of a glomus tumour on the hypothenar eminence, which is not a common site. From the reviewed literature, it was seen that a delay in diagnosis of glomus tumour ranged from 3 months to 15 years.[19] For our patient, the duration was 2 years. No medical therapy exists for glomus tumours, the only treatment option is surgical excision.[20] In this case, the pain was completely relieved following surgery and at follow-up, the patient did not complain of any pain.

Conclusion

Diagnosis of glomus tumour can be delayed significantly especially when located outside a typical subungual region. So high index of suspicion is needed for early diagnosis of this potentially curable tumour.

Conflict of Interest

None

Funding source

None

Patient Consent

Taken

IRB approval status

Not applicable

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Case report:

Rosacea and Demodex: A Comprehensive Case Study Validated by Dermoscopy

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Abstract

Human Demodex, a widely known ectoparasitic mite, affects mainly pilosebaceous units of the face and head. Human Demodicosis is a skin disease that develops when the follicles become heavily infested, or when the mites penetrate the dermal tissue. Here we have reported a 38-year-old female, who was presented with persistent erythema of the nasal bridge accompanied by pustules. Dermoscopic examination revealed Demodex tails and Demodex follicular openings both specific features of this entity. Histopathological features of skin biopsy were consistent with rosacea in the context of infection with Demodex folliculorum.

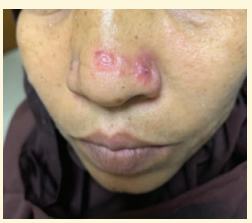
Introduction

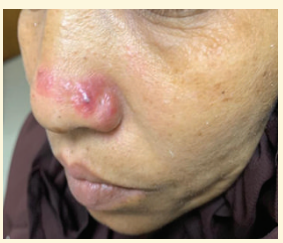
Rosacea is a chronic and relapsing inflammatory skin disorder characterized by a varied clinical presentation, including congestion, flushing, telangiectasia and rhinophyma. It commonly occurs in adults over 30 years of age, with a female preponderance.[1] Dermoscopy has become a popular tool to diagnose and monitor many dermatological diseases. The specific dermoscopic patterns can be used to diagnose rosacea, especially demodicosis. The dermoscopic feature of Demodicosis is specific and it can be diagnosed without a superficial surface biopsy. Demodex mites must be investigated in drug-resistant Rosacea.[2]

There are four main types of Rosacea: erythematotelangiectatic, papulopustular, phymatous, and ocular rosacea. Variants of rosacea may also occur which do not strictly match the morphologic patterns or combinations seen in these subtypes.[1] The exact pathophysiology of rosacea is poorly understood. Inflammation and vascular abnormalities are two main factors.[3] Vascular abnormalities result in dilatation of blood vessels with increased capillary permeability and oedema, which in turn provide a favourable setting for the Demodex mites to proliferate and colonize in that area. Demodex mites then stimulate inflammation, increasing the likelihood of papulopustular or granulomatous lesions.[4]

Case Report

A 38-year-old Bangladeshi village woman came with the complaints of chronic persistent asymptomatic facial rashes for three years, without any prior triggers.Physical examination revealed fixed erythematous coalescing papules and plaques studded with very few pustules over the nasal bridge and part of Ala Nasi. No ocular involvement was noted. Cheek, chin forehead were unaffected





All baseline laboratory investigations including the purified protein derivative (PPD) skin test, serum anti-nuclear antibody (ANA) and QuantiferronTB gold test were unremarkable. Under dermoscopy

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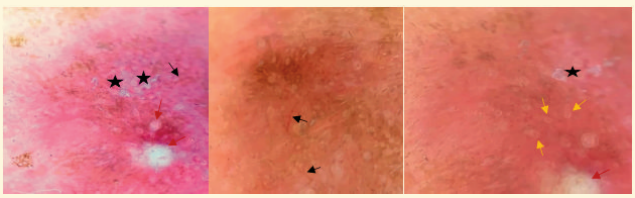
Cite this Article:
Wahab F, Hossain MM, Siddika M, Nasim R. Rosacea and Demodex: A Comprehensive Case Study Validated by Dermoscopy. Ban Acad Dermatol. 2023; 03 (02): 88-90

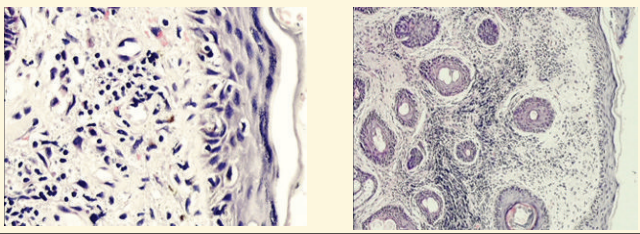
Available at: www.jbadbd.com
An official publication of Bangladesh Academy of Dermatology (B.A.D.)

(Dermlite dl4) we observed non-follicular and perifollicular gelatinous threads or filaments protruding out of follicular openings known as "Demodex tails." They account for the presence of the mite itself. Demodex follicular openings were also identified as dilated follicular openings containing round, brown plugs surrounded by an erythematous halo. They are both specific features of Demodicosis.



Dermoscopic features: Erythematous background, scales(black stars), linear blood vessels (black arrows), follicular pustules(red arrow), follicular plugs and demodex tail (yellow arrows).

A skin biopsy was performed and the histology report was compatible with rosacea.



Histopathological features: Mild exocytosis of the epidermis, dense perifollicular & perivascular infiltration of chronic inflammatory cells in the dermis, dilated capillaries & few dilated follicular infundibula, some of the latter contains demodex.

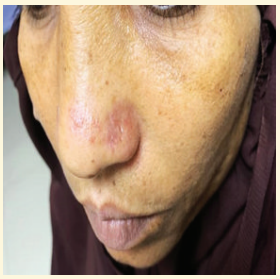
Discussion

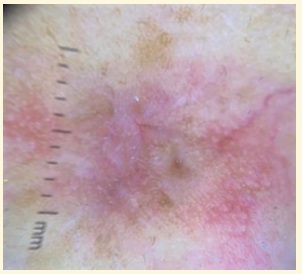
Rosacea is a common chronic skin and ocular condition,[5] characterized by intermittent periods of exacerbation and remission.[6] Reported prevalence rates of rosacea range from 0.09% to 10%.[7] The clinical criteria of rosacea are divided into primary and secondary categories. Primary criteria include transient facial erythema (flushing), no transient erythema, papules, pustules, and telangiectasia. The presence of one or more of these signs with a central facial distribution is considered highly indicative of rosacea. Secondary diagnostic criteria include burning or stinging skin sensations, elevated red plaques without epidermal changes, dry skin appearance, oedema, peripheral location, ocular manifestations, and phymatous changes are often met with one or more of the primary features of rosacea.[1] In this case,a few co-existent clinical criteria (non-transient erythema, papules, pustules, plaques) were present from both categories.

Polygonal vascular structures made up of linear blood vessels are the main findings in Rosacea. The clinical examination is not sufficient to discriminate the polygonal vasculature, whereas papules and pustules are seen clearly with dermoscopy. Follicular plugs, comedones, and dilated follicles are more prominent in the papulopustular subtype of rosacea.[8] In the case of demodicosis, the dermoscopic examination reveals a "Demodex tail" that protrudes from the follicle orifice, which is surrounded by a "Demodex follicle opening" that appears as a grey circle 1–3 mm in length. It is often confused with open comedones, but the open comedones can be seen with the naked eye, and they are more brownish and surrounded by a thin, hyperpigmented ring.[9] Here, we have found the linear vasculature in the form of polygonal networks,follicle plugs and Demodex tails, compatible with histopathological findings.[10]

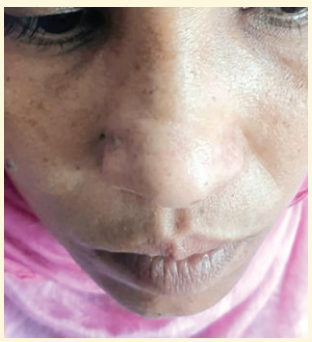
The diagnosis of Rosacea may be difficult due to the clinical similarities to several skin disease conditions such as acne vulgaris, seborrheic dermatitis, contact dermatitis, and photodermatitis.[6] In this case, other probable differentials were Lupus vulgaris, Sarcoidosis and Tumid Lupus Erythematosus. Here, dermoscopy (Dermlite dl4) was used as a rapid tool for the submacroscopic diagnostic evaluation before we could reach ANA, Qantiferron TB Gold Test and histopathological report.

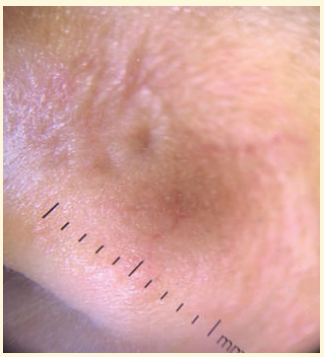
Various medical options are there to treat Rosacea. Currently, there is no cure for the condition,[11] however, a wide range of medications are used for the treatment of purpose. Topical medications include metronidazole, azelaic acid, benzoyl peroxide, antibiotics, sulfacetamide/sulfur, and retinoids, while oral medications used for this purpose include tetracyclines, metronidazole, isotretinoin, and macrolides.[12] Our patient was treated with oral minocycline 100 mg daily and topical dapsone, retinoid, and sunblock for three months and she remained in remission.





Clinical and dermoscopic changes after two months of therapy.Dermoscopy shows a reduction of background erythema, few linear blood vessels, reduction of scales, no follicular pustules & plugs, and no demodex is seen.





Clinical pictures after three months of therapy show almost complete recovery. Dermoscopy shows mild background erythema, and few linear blood vessels, all other previous dermoscopic findings have subsided.

Conclusion

The dermoscopic method can be used as a reliable noninvasive, easy and faster method for the diagnosis of rosacea and the accompanying Demodicosis without the requirement of a skin biopsy.

Conflict of Interest

None

Funding source

None

Patient Consent

Taken

IRB approval status

Not applicable

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Review Article:

Loss of fingerprints: A hidden problem facing during biometric identification

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Abstract

In the era of globalization as well as digitalization fingerprinting is the most acceptable form of digital recognition. It is the most available unique method for identification and individuality. But in recent years people from different corners of the world have noticed to have no fingerprinting incidentallywhen they are facing problems in digital identification and authentication systems. A thorough literature review was conducted involving different medical databases, using the following terms: loss of fingerprints, adermatoglyphia, and biometrics both individually and in combination. Surprisingly, no articles were found discussing specific guidelines for adermatoglyphic patients who have to undergo mandatory fingerprint verification. However, few case reports and scientific studies reveal that it will be a burning issue within the next decade. This article highlighted the epidemiology, types of adermatoglyphia, and challenges in diagnosing the cases and the importance of imposing other biometric methods to overcome the problem.

Keywords: Adermatoglyphia, fingerprints,

Introduction

The scientific and methodical study of complex patterns andfingerprints from palms, fingers, soles and toes isreferred to as dermatoglyphics.[1,2,3] Dermatoglyphics (or fingerprints) is a Greek word meaning skin (derma) and carvings (glyphe). These ridges are distinctive carvings of nature especially prominent over the palmar surface of hands and plantar surface of feet.[4] More than a century ago, Sir Francis Galton discovered that these ridge patterns areincredibly constant throughout the lifespan of an individual.[5] Later, the term“dermatoglyphics” wasintroduced by Dr. Harold Cummins in 1936.[6] All over theworld, fingerprinting is the most widely used method for individual identificationand authentication (I&A). Dermatoglyphics form a major portion of mass data collection,biometric surveillance, and verification.Adermatoglyphia is defined as the congenital or acquired loss of the epidermal ridge pattern.[4] It can be complete or partial, reversible or irreversible. It is also referred to as "Immigration Delay Disease".[7] Adermatoglyphia, or simply, lossof fingerprints attributedto a medical cause, represents a taxing situation for such biometric scrutiny systems requiring afingerprint scan as a mandatory phase in identification and authentication procedure. The scenario can be extremelydebilitating for adermatoglyphic patients, especially when the condition is permanent orirreversible.[8]

The disparityin ridge pattern of every individual is partly determined by genetics during gestational weeks 7th to 21st and partly by changes, trauma and diseases acquired during life.[9,10] Biometric methods which are used for identification inrecent days are complex, however, the use of fingerprints is less complex, cost-effective, readily available and therefore, universally used compared to other modalities. However, numerous hereditary and acquired causes of adermatoglyphia pose a huge

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Cite this Article:
Nasim R, Sikder MS. Loss of fingerprints: A hidden problem facing during biometric identification. Ban Acad Dermatol. 2023; 03 (02): 91-95

Available at: www.jbadbd.com
An official publication of Bangladesh Academy of Dermatology (B.A.D.)

hurdle in individual identification.[11]

Epidemiology

Adermatoglyphia is not a known topic and no extensive work has not been done on it. So, there is a dearth of literature. However, nowadays the need for biometrics in identification, authentication and forensics application have exposed cases of hidden adermatoglyphiaand the field of interest is increasing. Haber et al. conducted a national survey in Lebanon in collaboration with the Ministry of internal affairs in 2014 which recorded 0.18%

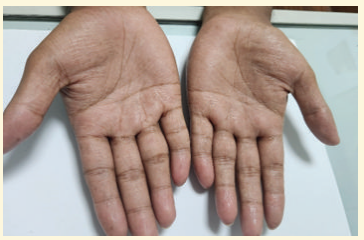
cases of adermatoglyphia with a female predominance and an increased incidence in the ageing population with dermatitis as thecommonest cause.[12]

A cross-sectional survey was carried out in the Department of Dermatology, Services Hospital, Lahore. All patients were referred by the National Database and Registration Authority (NADRA) through the Medical Examination Department (MED) of Services Hospital for verification of adermatoglyphia. A total of 152 patients with adermatoglyphia were enrolled over two years. Out of them, 96 (63.2%) were males while 56 (36.8%) were females. The mean age of the patients was 41.89 ± 23.72 years. Among their study population, 20 (13.2%) patients had isolated congenital (idiopathic) adermatoglyphia, 52 (34.2%) patients suffered from Congenital adermatoglyphia associated with a syndrome, while 80 (52.6%) patients presented with acquired causes of adermatoglyphia.[11]

Loss of fingerprints can be congenital or acquired.[8,13] The congenital form can be part of a complex syndrome and the prevalence will mirror that of the syndrome. Individuals with completely missing fingerprints as an isolated finding is extremely rare. Four generations were reported to have this isolated form of adermatoglyphia inherited in an autosomal dominant fashion.[14]

The acquired forms are more common andcould result from other dermatological conditions.[8,14-15]

A 27-year service holder visited in a private chamber referred by a travel agency in July 2023 for medical evaluation. He realizedthe loss of his fingerprints when he was unable to give his fingerprint during passport issuing, requiring his to be ten-printed (i.e., all fingers and thumbs of both hands were scanned/printed). Physical examination reveals hyperhidrosis of both palms andlaboratory reports reveal no abnormalities.He was treated as a case of idiopathic palmar hyperhidrosis and adviced to follow up after 2 months.



Loss of fingerprints of a 27 years male due to palmar hyperhidrosis

BBC reported in December 2020,that a Bangladeshi family where 3 generations had no fingerprints faced obstacles in issuing national ID cards, driving licences, buying SIM cards and office attendance systems. A dermatologist in Bangladesh has diagnosed the family's condition as congenital palmoplantar keratoderma. More genetic testing would be needed but these were not available in this resource-poor country.They got their NID card issued by the Bangladesh Governmentafter presenting a medical certificate.The cardused other biometric data too-retina scan and facial recognition.16

Congenital palmoplamterkeratodrma of a Bangaladeshi male[16]

The rationale of the review

Clinically adermatoglyphia is defined as the absence of an epidermal ridge pattern that may be due to congenital or acquired causes.6A few or all digitscan be affected. It may also refer to the absenceof the ridge patterns formed on the plantar aspects of the feet. Furthermore, adermatoglyphiacan refer to a partial loss of the ridges (i.e., ridges are unnoticeable on general evaluation but detectable on deep inspection or under a magnifying lamp) or a complete absence (depictingtotal effacement) of epidermal ridges. These are time-consuming challenges not only for the concerned authorities but also for the individual who has to face the problem in completing verification procedures. Face recognition and fingerprinting are the primary modes ofbiometric I&A

all over the world. Any technical problem or error hindering these steps of verification can cause the entire verification process to come to a halt.[12] Mostpatients with adermatoglyphia are unaware of the fact that they cannot produce their fingerprints and are surprisingly shocked when biometricanalysis fails.Nothing can be done except resorting to other modalities of identification. This is also achallenge in other areas such as forensics and criminal identification as it slows the process.Diagnosing adermatoglyphia is quite challenging.Therefore, a reliable alternativeto fingerprinting that is cheap, unique and readily available must be sought shortly.

Table1. Congenital causes of adermatoglyphia and their associated genes[17]

S/no	Disease condition	Associated Gene
1	Basans syndrome	Smarcad1
2	Naegeli-franceschetti-jadassohn syndrome	Krt14
3	Dermatopathia pigmentosa reticularis	Krt14
4	Reticulate acropigmentation of Kitamura	Krt14, dkc1
5	Rothmund thomas syndrome	Wrn,blm
6	Dyschromatosis universalis hereditaria	Smarcad1,dkc1

Table 2. Dermatological and non-dermatological causes of acquired adermatoglyphia[17]

Dermatological	Non dermatological
Allergic and irritant contact dermatitis	Trauma
Atopic dermatitis	Burn
Dyshidrotic eczema	Amputation
Cutaneous LE	Caustic abrasion
Epidermolysis bullosa	Denervation injuries
Pemphigus vulgaris	Capecitabine
Psoriasis	Topical steroid
Keratoderma blennorrhagica	Retapamulin
Palmar wart	Atorvastatin
Leprosy	
Pyoderma/impetigo	
Coxsackie	
Tinea mannum	
Erythema multiforme	
Steven johnson syndrome	
Toxic epidermal necrolysis	
Serum sickness	
Primary hyperhidrosis	
Lichen	
Acrodermatitis	

Methods of human identification and authentication

The necessity of a second-line substitute I&Asystem, especially for patients suffering from irreversible adermatoglyphia can only be realized when people get into the hassle of identification. Imperatively a medicalcertificate can be medico-legally issued but only provides a temporary solution, as the validityand the authenticity of such certificates might not be acceptable by other organizations.Different set-ups demand different types of biometric verification, often involvingmandatory, multi-modal biometric functions operating without a backup plan.[18,19] Additionally, some corporations require only thumb-printing for monitoring office attendanceor bank account verification. Other agencies granting citizenship, driver's licenses, passports,and immigration papers require a more stringent policy, requiring individuals to be ten-printed.Therefore, a substitute I&A system is recommended that can function globally as a defaultprogram for patients suffering from irreversible adermatoglyphia.

Biometrics

Over the past few decades, many advancements have been made in biometric technology withthe introduction of new measurable biological traits as potential biometric targets. Table 3presents a concise review of the indispensable characteristics required for a biometric indicator.

While many of these biological traits fulfil the necessary characteristics required toqualify as a biometric indicator, feasibility becomes the most important factor based on
efficacy, social acceptability, the technical complexity of systems, ease of applicability, andcost-effectiveness. Consequently, there is no single biometric indicator that ideally achieves allthese parameters. Therefore, hybrid or complex multi-modal biometric systems are used toenhance the accuracy and efficacy of I&A.[18,19]

TABLE 3: Essential qualities of a biometric indicator

Characteristic	Definition
Generality	Universally present in all individuals
Uniqueness	No individuals share the same configuration
Stability	Unchanging throughout the lifespan
Quantifiable	Measurable for comparison

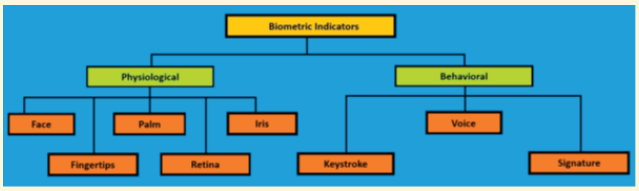


Fig: Biometric Subtypes

Classification is based on physiological and behavioural traits

In addition to facial recognition and fingerprinting technology, extensive research has beenconducted on other biometric recognition techniques, namely, iris/retinal scanning, handgeometry and palm prints, DNA fingerprinting, voice recognition, signature scanning, gait
patterns, and keystroke patterns.[20-23] Future endeavours will involve the exploration of otheranatomical organs and parts, with the potential for I&A to fulfil essential biometric features. These domains include but are not limited to, finger-vein patterns,[21] foot-printing,[24] dorsal finger pattern,[25] lip-printing,[26] and tongue-printing. [27]

Challenges

Diagnosing adermatoglyphia in Bangladesh is quite challenging. Patients usually present with difficulty in capturing fingerprints duringbiometrics. Further evaluation to enable the Attending physicians cannot get a chance to find out a specific cause because of the lack of molecular genetic testing.Thisis the primary investigation to ascertain whetherthe patient has smarcad1gene and keratin[14] mutation which are the major gene mutations seen in adermatoglyphia. [28,29] Theseinvestigations are not readily available anddefinitive diagnosis is halted. Other investigative modalities like volar pad biopsy are usually refuged by patients as they see the absence of fingerprints as a variant of normal and will resist attempts for invasive procedures. Another challenge is tracing family members to ascertain whether they also have adermatoglyphia.

The paucity of data on adermatoglyphia is also an issue as most patients with the condition do not present to a health care facility except when in need of a medical report. Patients are usually seen during biometrics in the banks, borders or immigration offices and access to
these data is met with some bureaucracies. The psychosocial impact patients with adermatoglyphia face during biometric screening is also enough trauma to make them avoid further analysis.

Conclusion

Adermatoglyphia, especially due to acquired causesisnot an uncommon finding in the geriatric age group and manual labourers. It is also found in the young population. Despite itsbenign nature, the persistent invasion of biometric identification in modern life via fingerprintverification creates huge hassles for patients with irreversible adermatoglyphia. As fingerprinting is the most available and cheaper method for biometric identification, it provides the identification and authentication system quicker and is applied in different systems and institutions.However, aperson with absent or permanent loss of fingerprinting has to undergo enormous hardships due to lack of any alternative options. Therefore, specific guidelines or a substitute I&A system is recommendedthat can function worldwide, as a default program, facilitating I&A for patients suffering from irreversible adermatoglyphia.

Conflict of Interest

None

Funding source

None

Patient Consent

Taken

IRB approval status

Not applicable

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