

Review Article

The Frequency Of Occurrence Of Diabetes Mellitus And Other Autoimmune Diseases In A Sample Of Iraqi Patients With Dermatitis Herpetiformis.

Hayder Riyadh Obayes Al-Hassani^{1*}, Marwah Hamzah Alwan Alfatlawi², Hussein Safaa Abdulameer Alhassany¹

¹Aesthetic and Laser techniques Department, Health and Medical technique, Al-Mustaqbal University, Babylon, 51001, Iraq.

²Consultant family medicine, Arab board of health specializations, Baghdad, Iraq.

Abstract

Background: Dermatitis herpetiformis is a chronic, intensely pruritic, skin condition assisted with gluten-sensitive enteropathy. There is limited research studying the frequency of diabetes among patients with dermatitis herpetiformis.

Aim of study: To find out the frequency of occurrence of diabetes among patients with dermatitis herpetiformis.

Patients and Methods: This is an observational study that included 73 patients diagnosed with dermatitis herpetiformis and was conducted in (Al-Basrah Center of Dermatology and Venereology at AlSadr Teaching Hospital) during the period from 1/8/2023 to 1/8/2024. The patient's socio demographic characteristics were recorded (age, sex). Then, careful clinical history and clinical examination of the lesion were performed to investigate the onset age, and lesion details. Diagnosis was made by the attending consultant dermatologist and recorded for the purpose of this study. All patients were sent for full investigations including complete blood count, glaciated hemoglobin, and serology for celiac disease. The diagnosis was based upon histopathology based on biopsy specimen.

Results: Regarding associated chronic systemic diseases; type-II DM, was diagnosed in 45 (61.6%), celiac disease in 44 (60.2%) patients, autoimmune thyroiditis in 5 (6.8%), and type I DM in 3 (4.1%).

Conclusion: The study showed that diabetes mellitus type-II coexisted with dermatitis herpetiformis in 61.6% of cases. Coeliac disease is the most common autoimmune disease associated with dermatitis herpetiform, followed by autoimmune thyroid disease and type-I diabetes.

Keywords: Herpetiformis; Diabetes Mellitus; Autoimmune Thyroid.

INTRODUCTION

Dermatitis herpetiformis (DH) is a chronic, intensely pruritic, skin condition associated with gluten-sensitive enteropathy (GSE) [1]. The disease is characterized by: (i) symmetrical polymorphic lesions affecting typical regions, including the extensor surfaces of the limbs and sacral area, (ii) a predominant neutrophilic infiltrate in the dermal

papillae histopathological examination, (iii) granular IgA deposits along the dermal-epidermal junction, (iv) a consistent association with celiac disease, and (v) a positive response to a lifelong gluten-free diet [2].

The development of dermatitis herpetiformis usually occurs during the second and fourth decades of life; yet, individuals of any age may be impacted. Dermatitis herpetiformis is uncommon in children [3]. Elevated rates are often seen in

nations like Finland, attributable to this disease's affinity for persons of northern European ancestry. In contrast, DH is infrequent among Asian communities and much more uncommon among African Americans. This condition may manifest at any age, while it is most often diagnosed between 30 and 40 years, with an average age of 43 years. There is a male preponderance with a male-to-female ratio ranging from 1.5:1 to 2 [4].

Dermatitis herpetiformis results from an immunological reaction to the persistent stimulation of the intestinal mucosa by dietary gluten [5].

1). Genetics: Dermatitis herpetiformis and celiac disease has a significant hereditary component. First-degree relatives of individuals with dermatitis herpetiformis and celiac disease have a markedly elevated risk relative to the general population [6].

***Corresponding Author:** Hayder Riyadh Obayes Al-Hassani. Aesthetic and Laser techniques Department, Health and Medical technique, Al-Mustaqbal University, Babylon, 51001, Iraq. Email: hhaiderhaider143@gmail.com.

Received: 15-September-2026, Manuscript No. TAJOPH - 6177; **Editor Assigned:** 21-September-2026; **Reviewed:** 03-October-2026, QC No. TAJOPH - 6177;

Published: 12-October-2026. DOI: 10.52338/tajoph.2026.6177.

Citation: Hayder Riyadh Obayes Al-Hassani. The Frequency Of Occurrence Of Diabetes Mellitus And Other Autoimmune Diseases In A Sample Of Iraqi Patients With Dermatitis Herpetiformis. The American Journal of Public Health. 2026 October; 19(1). doi: 10.52338/tajoph.2026.6177.

Copyright © 2026 Hayder Riyadh Obayes Al-Hassani. This is an open access article distributed under the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original work is properly cited.

- 2). HLA association: Dermatitis herpetiformis and celiac disease are strongly linked to the human leukocyte antigen (HLA) I) Q2 and DQ8 haplotypes; about 90% of cases are related with HLA IX22, while the rest are linked to HLA I)Q8. Both conditions are implicated in the processing of the gluten antigen gliding [7].
 - 3). Deposition of Immune Complexes: High-affinity anti-TG3 antibodies accumulate in the dermal papillae and form complexes with TG3 produced by keratinocytes. This mechanism initiates a neutrophilic inflammatory response in the papillary dermis [8].
 - 4). Environmental factors: Dietary gluten and its component gliadin are the primary environmental factors contributing to the disease's etiology. Interestingly, tissue transglutaminase (tTG) is responsible for converting gliadin in the gut mucosa to a more immunogenic form. Other environmental variables that cause dermatitis herpetiformis include iodine exposure (which may provoke flares of the illness), medicines (such as NSAIDs and progestin), and tobacco smoking (which can improve the condition) [9].
 - 5). Apoptosis may contribute to the pathophysiology of epidermal alterations in dermatitis herpetiformis. Research reveals a considerably higher apoptotic rate inside the epidermal compartment in dermatitis herpetiformis [10].
- The extensor surfaces, including the elbows, dorsal forearms, shoulders, knees, and buttocks, are involved in the distribution. The scalp, neck, upper back, and sacral region are some common areas that are impacted. But it may also affect the groin and face [11-12]. Typical eruption characteristics include a variety of vesicles, urticarial plaques, and clusters of erythematous papules. Many individuals have erosions, crusted papules, and excoriations as a result of intense itching and scratching; these lesions often heal without leaving scars [2, 13-14]. The lack of pruritus, which is so diagnostically significant, clearly suggests a different diagnosis Oral mucosal lesions such as vesicles, macules, and erosions are uncommon in DH, but they have been reported. Petechial and purport, especially on the palms and soles, might appear as DH's sole presenting characteristic or in conjunction with typical symptoms in rare occasions [15].

Figure 1. Dermatitis herpetiformis. A—B, Pruritic pink papules and plaques with occasional erosion.

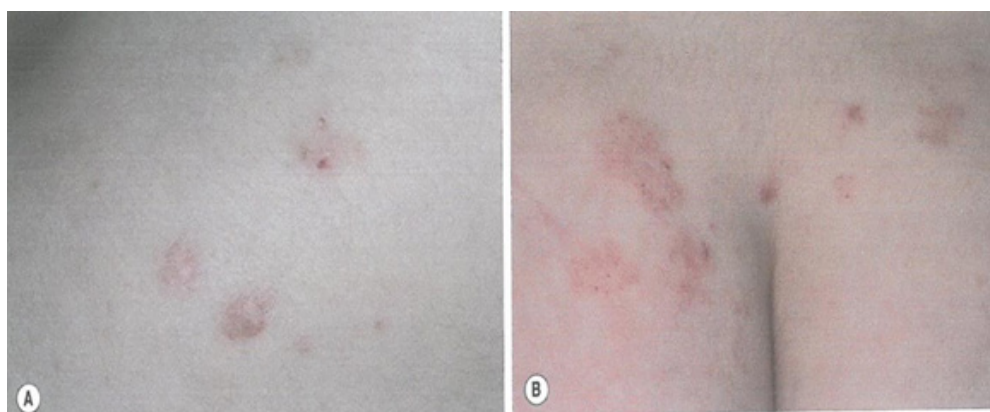
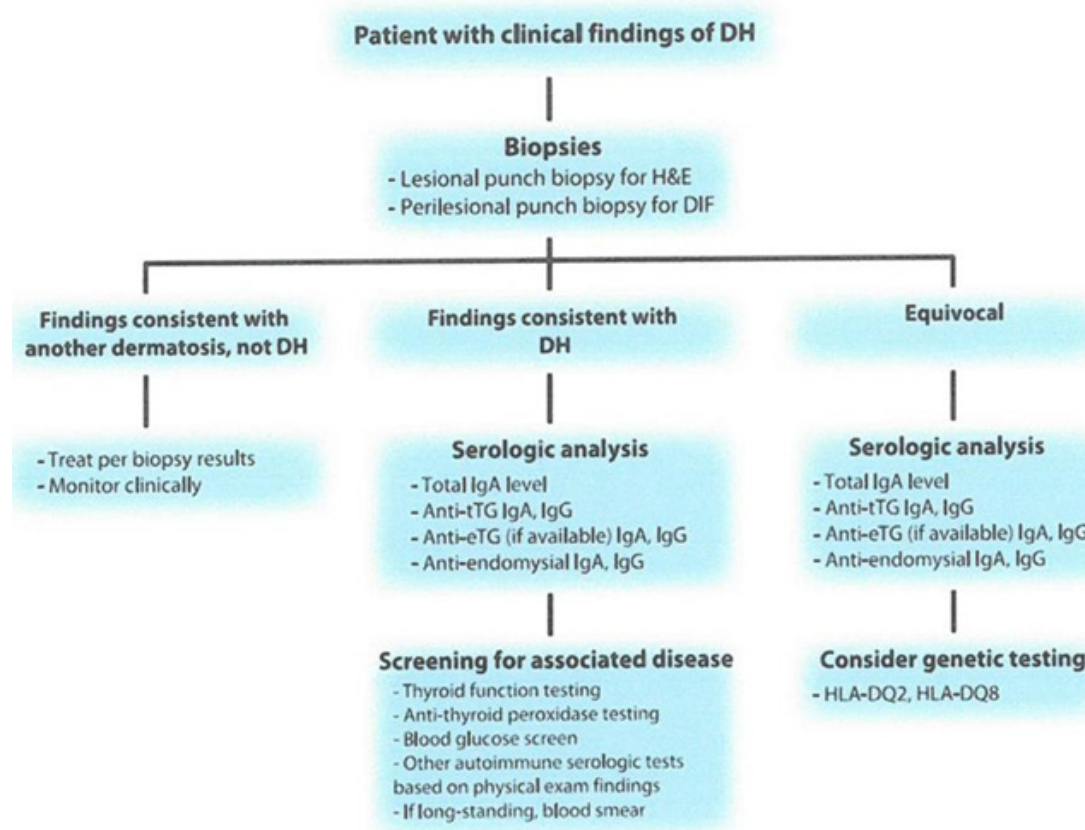


Figure 2. Dermatitis herpetiformis lesions on the a) elbows and, b) knees: observe symmetrical distribution and erosions due to scratching. c) A close-up view of a small blister. d) Direct immunofluorescence finding of uninvolved skin showing pathognomonic granular immunoglobulin A deposits at the dermo-epidermal junction.



Most DH patients have some celiac-type damage in their small intestine, although it is generally less severe than CD. As a result, people with DH may have GI symptoms such as bloating, diarrhea, or constipation, albeit these are often minimal. Patients with DH may have nutritional deficits, osteoporosis, short stature, anemia, and weight loss as a result of their gluten-sensitive enteropathy [16-18]. Approach to the patient with suspected dermatitis herpetiformis shown.

Figure 3. Approach to the patient with suspected dermatitis herpetiformis.



Aim of the study is to find out the frequency of occurrence of diabetes and other autoimmune diseases among patients with dermatitis herpetiformis.

PATIENT AND METHODS

Design of the Study

The study has been conducted in (Al-Basrah Center of Dermatology and Venereology at Al-Sadr Teaching Hospital). The data collection was completed within 12 months, i.e., from 1/8/2023 to 1/8/2024. A total number of 73 patients were enrolled in the study. The patient's sociodemographic characteristics were recorded (age, sex). Then, careful clinical. The age of the studied sample ranged from 13 to 82 years with a mean of (58.66 years $\pm$ 16.73 SD). Most of the studied sample (42.5%) were in the age group (> 60 years). The Sex distribution of the studied sample showed a male predominance, as the male to female ratio was 2.04, patient's data are shown (Table 1). An observational cross sectional descriptive design has been opted for this study. All patients diagnosed with dermatitis herpetiformis were included in the study. Diagnostic criteria for dermatitis herpetiformis were based on clinical evaluation by experienced dermatologist (chronic recurrence of itchy, erythematous papules, urticarial wheals and grouped vesicles that appear symmetrically on the extensor surfaces, buttocks and back), histopathology (subepidermal vesicles and blisters associated with accumulation of neutrophils at the papillary tips), DIF, and therapeutic response to depose.

Table 1. Sociodemographic characteristics of the studied sample.

Sociodemographic characteristics	No. of patients	Percentage
Age Of presentation		
< 18 years	8	11.0
18-40 years	11	15.1
41-60 years	23	31.5
> 60 years	31	42.5
Total	73	100.0
Sex		
Male	49	67.1
Female	24	32.9
Total	73	100.-

History and clinical examination of the lesion were performed to investigate the onset age, and lesion details. Diagnosis was made by the attending consultant dermatologist and recorded for the purpose of this study. All patients were sent for full investigations including CBC, HbA1c, RBS, FBS, thyroid function tests and history and examination. Patients underwent investigations for celiac disease such as serology test and jejuna biopsy for celiac disease by gastroenterologists. The biopsy specimen was obtained by the dermatologist. An internal medicine specialist differentiated between type I and type-II DM by evaluating clinical history (including age of onset, family history).

Selection of the research population

Presentation with ketosis) and the detection of elevated autoantibodies (anti-insulin, anti-GAD65, anti-IA-2, or anti-ZnT8). The patient was prepared for the biopsy procedure by ensuring their comfort. The skin area was prepared with an antiseptic solution. A local anesthetic was injected into the base of the lesion. The lesion was excised using a scalpel or a punch biopsy instrument, including a portion of surrounding unaffected skin. The Specimen was handled carefully and placed in a suitable fixative. It was labeled with the patient's information, date, and location of the biopsy. The specimen was then sent to the pathology laboratory for histological examination. Post-procedure care was provided to the patient, including instructions for wound care, and the patient was advised to report any signs of infection or excessive bleeding. A follow-up appointment was scheduled to discuss the biopsy results and any necessary treatment.

Data entry and analysis

Data entry were done using Microsoft Excel 2019 [19]. Data was recorded into different variables for the purpose of analysis. Analysis were done using statistical package for social sciences (SPSS version 26). Data were summarized using measures of frequency (mean), dispersion (standard deviation).

Ethical Considerations

The study was conducted in compliance with ethical standards. Permission to access patient data and perform diagnostic evaluations was obtained from the respective hospitals. All patient data were anonymized to maintain confidentiality. If required by the institutional review boards, verbal or written consent was obtained prior to inclusion.

Approval and official permission

An approval was taken from the scientific committee of the Scientific Council of Dermatology & Venereology-Arab Board for Health Specializations.

RESULTS

Dermatitis herpetiformis duration

Conceding disease duration, it ranged from 1 month to 10 years with a mean of 1.37 years $\pm$ 1.1 SD, as 53 (72.6%) reported a disease duration of $\leq$ 1 year, 14 (19.2%) reported 1 -5 years, and 6 (8.2%) reported 25 years; as illustrated in (Table 2).

Table 2. Dermatitis herpetiformis duration.

Disease duration	No. of patients	Percentage
$\geq$ 1 year	53	72.6
1-5 years	14	19.2
$\leq$ 1 year	6	8.2
Total	73	100.0

Distribution of cases according to site of lesion

Regarding the site of lesion; elbows were involved in 72 (98.6%) patients, knees in 69 (94.5%), back in 18 (28.1 %); as illustrated in (Table 3).

Table 3. Distribution Of cases according to site of the lesions.

Site Of lesion	No. of patients	Percentage
Elbows	72	98.6%
Knees	69	94.6%
Back	18	28.1%
Buttocks	11	15.1%
Shoulders	8	11.0%
Extensor forearm	6	8.2%
Extensor arm	4	5.5%

Disease characteristics of the studied sample

Regarding disease characteristics; tTG IgA and endomysial IgA serology were detected in 44 (60.3%) patients, skin biopsy showed typical findings in 43 (58.9%) cases; as shown in (Table 4).

Table 4. Disease characteristics of the studied sample.

Disease characteristics	No. of patients	Percentage
ElbowstTG1 serology		
Elevate	44	60.3%
Normal	29	39.7%
Endomysium I gA		
Shoulders	44	60.3%
Elevate	29	39.7%
Skin biopsy		
Typical findings	43	58.9%
Atypical findings	30	41.1%

Distribution of patients with dermatitis herpetiformis according to associated chronic systemic diseases

Regarding associated chronic systemic diseases; type II DM was diagnosed in 45 (90%), celiac disease in 30 (60%) patients, and type I DM in 3 (6%); as shown in (Table 5).

Table 5. Chronic systemic diseases in dermatitis herpetiformis.

Chronic systemic disease	No. of patients	Percentage
Type II DM		
Present	45	61.1%
Absent	28	38.4%
Celiac disease		
Present	44	60.3%
Absent	29	39.7%
Autoimmune thyroiditis		
Present	5	6.8%
Absent	68	93.2%
Type I DM		
Present	2	4.1%
Absent	71	95.9%

Relationship between the age of patient with dermatitis herpetiformis and the presence of DM type-II

(Table 6) shows that older age of patients with dermatitis herpetiformis was significantly associated with the presence of DM type II (p value <0.001).

Table 6. Relationship between the age of patient with dermatitis herpetiformis and the presence of DM type II.

Age	DM patients , Type II		P - Value
< 18 years	0	8	< 0.001
	0%	28.6%	
18-40 years	4	7	
	8.9%	25.0%	
41-60 years	16	7	
	35.6%	25%	
> 60 years	25	6	
	55.6%	21.4%	

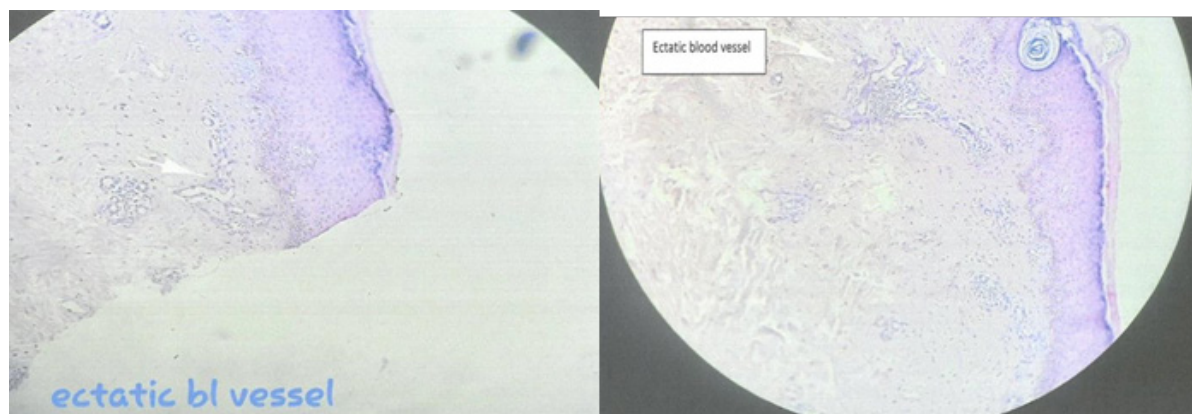
Relationship the duration of dermatitis herpetiformis and the presence of DM type II

(Table 7) shows there is no significant association between the duration of dermatitis herpetiformis and the occurrence of type II DM

Table 7. Relationship between the duration of dermatitis herpetiformis and the presence of DM type II.

Duration of dermatitis herpetiform	DM patients , Type II		P - Value
≤ 1 year	34	19	< 0.001
	75.6%	67.9%	
5 years	7	7	
	15.6%	25.0%	
≥ 5 years	4	2	
	8.9%	7.1%	

Figure 4. Dermatitis herpetiformis on different patient with DM.

Figure 5. Histopathology of dermatitis herpetiformis showing ectatic blood vessel.

DISCUSSION

Dermatitis herpetiformis (DH) is a blistering, autoimmune, and chronic disease that is thought to be an extra intestinal symptom of celiac disease (CD). The forearms, knees, scalp, or buttocks are symmetrically distributed with extremely itchy polymorphic lesions in affected individuals. Subepidermal blisters, neutrophilic infiltrates at the dermal papillae on histology, and IgA deposits along the dermal-epidermal interface on perilesional direct immunofluorescence microscopy (DIF) are the hallmarks of dermatitis herpetiformis [19-20].

Age and sex of DH, in this study the mean age of patients was 58 years, and the sex distribution of the sample under study showed a predominance of men. Zhang (2011) [21], conducted a study in China where the mean age was 44 years Bushra and Rayan (2023) [22], found that the mean age in Saudi Arabia was 43.6 years. [123-25], Handa et al's (2018) [26], study in India found that the average age of onset was in the fifth decade. [361 Similar to our findings, Antiga et al. (2019) [1.37 and Handa et al. (2018), discovered a predominance of men [27].

Disease duration, in the present study the mean disease duration was found to be 1.37 years, (76.0%) which is less than the duration (56.18 months) reported in India by handa et al., and (44.6) months reported by Zhang et al (2011) in china. [28-30]. There are a number of reasons why dermatitis herpetiformis (DH) reported durations vary throughout different research. The length of the condition is also influenced by the degree of adherence to a gluten-free diet and availability of medications like dapsone. Furthermore, different outcomes may arise depending on the sample size and study design (prospective vs. retrospective). Site of lesion, the lesions of DH were mainly found on elbows (98%) and knees (92%) in studied patients. Closely to our finding, Bushra and Rayan (2023) [1.34, found the lesion to be sited on the elbows and knees in 90% of cases and 92% in handa et al 2018 [31].

Diagnostic tMJs, together with DIF and serology (anti-tTG and anti-EMA), clinical and histological results are used to diagnose DH. The gold standard for making a diagnosis is still DIF of uninvolved. We found that tTG IgA and Endomysium IgA was elevated in 60.3% of cases, while skin biopsy showed typical findings in 58.9%. In a study of 24 patients with confirmed DH, 37.5% exhibited nonspecific results when examined using haematoxylin and eosin (H&E) staining.

These results included the presence of a lymphocytic infiltrate, dilated capillaries, and fibrosis in the dermal papillae. Due to the possibility of ambiguous biopsy results, it is crucial to conduct DIF studies [32]. In Bushra and Rayan (2023) study, (77.8%) had positive tTG IgA, IgA was detected on DIF (45%) and typical biopsy finding in 62% of cases. Zhang (2011), study in china showed that tTG IgA and Endomysium IgA were positive in 72.73% of their DH patients and skin biopsy showed typical finding in 95.45%. The first ELISA to identify anti-tTG IgA antibodies was performed by Dieterich et al. (1999), according to their study, the positive rates for anti-tTG and anti-EMA were 70.5% and 75.4% respectively, indicating that these tests can be helpful in identifying individuals with DH. Differences in anti-tTG and anti-EMA positivity in dermatitis herpetiformis (DH) across studies may be influenced by variations in wheat consumption among different cultures. Since these antibodies are produced in response to gluten, higher wheat intake is associated with increased antibody positivity. Conversely, in cultures where gluten consumption is lower or where gluten-free diets are more prevalent, antibody positivity rates may be lower. These dietary variations can impact the prevalence and detection of anti-tTG and anti-EMA antibodies in DH research [33].

Type 1 diabetes mellitus in DH, in the current study, 6% of DH patients had type 1 diabetes mellitus [1331]. This relationship may result from common autoimmune mechanisms or the underlying cause of the relationship could be diabetes-related metabolic dysregulation and decreased skin repair, which aggravates DH's skin symptoms [33]. According to research in Finland by Hernoven et al (2004), 2.3% of individuals with

dermatitis herpetiformis (DH) had type I diabetes mellitus, which is significantly more common in DH patients than in the general population. The prevalence of type I DM was lower (0.7%) in another study from Finland by Tuomilehto et al. [34]. The immune system is assumed to be the cause of type I diabetes, which is most frequently detected in childhood or adolescence and is caused by the destruction of insulin-producing β cells in the pancreatic islets. Moreover, an autoimmune reaction may start concurrently or later in the gastrointestinal system. Even people with asymptomatic celiac disease are diagnosed and treated with a gluten free diet at an early stage because serological screening is now regularly in Finland for patients with Type 1 diabetes. Therefore, it's probable that the rash, or DH, has run out of time to develop in these studies from Finland which perhaps resulted in the lower frequency of type-I, DM there [35].

In regard type 2 mellitus, this study that type 2 DM is present in the majority (61.1 %) of DH cases. Unfortunately, to the best of our knowledge, this association has not been studied in the literature. Thus, the exact frequency of Type II DM in DH remains less clear. Patients with DH may have a higher incidence of type 2 diabetes due to shared autoimmune mechanisms, metabolic dysregulation and inflammatory pathways [36-40]. Another reason may be that type II DM was incidentally associated with older age, which was demonstrated by a significant association between the incidence and type II DM and older age of patient with dermatitis herpetiformis [41].

Autoimmune diseases association with DH, in 76% of the cases in this study, DH was linked to an autoimmune condition, of which 600/0 had celiac disease, 10% had autoimmune thyroiditis, and 6% had type 1 diabetes mellitus. In comparison to other research, Reunala et al (1997) study, which tracked 305 patients with DH for ten years and found that 100/0 of them had autoimmune illnesses. 23% of DH patients in the Handa et al (2018) research also had other autoimmune disorders. 1361 However, research conducted in China or Japan have not found any links with autoimmune diseases.

In 45% of the Saudi Arabian patients with Bushra and Rayan (2023) dermatitis herpetiformis had celiac disease. [34] Nine (13.84%) individuals with DH had thyroid abnormalities, of whom eight had hypothyroidism and one had hyperthyroidism, according to a 2018 Indian study by Handa et al. and 36% of the patients had celiac disease. [42-44].

It is worth mentioning that remarkable improvement with dapsone within less than 72 hours was considered to confirm the diagnosis of dermatitis herpetiformis, as it is mentioned in the These findings necessitate the importance to increase awareness of dermatologists or those practicing medicine, particularly endocrinologists, to search for this association in patients who present with anyone of them [45-47].

CONCLUSION

According to the findings of the current study, the following is to be concluded:

- The study showed that diabetes mellitus type II coexisted with dermatitis herpetiformis in 61.60/o of cases.
- Coeliac disease is the most common autoimmune disease associated with dermatitis herpetiform, followed by autoimmune thyroid disease and type I diabetes.

Based on the study's findings, physicians and dermatologists should raise awareness of and screen for coeliac disease (and risk of celiac lymphoma), autoimmune thyroid disease, and type I diabetes in patients with dermatitis herpetiform. Given the high prevalence of type II diabetes among the study participants, additional research is needed to investigate the potential links and type II diabetes. herpetiform between dermatitis Furthermore, interdisciplinary approaches involving dermatologists, and gastroenterologists should be encouraged to improve overall patient care. Education about the symptoms and implications of these conditions may also aid in early detection and better patient outcomes. Further prospective case control studies with larger sample size are needed in order to validate the findings of this study.

Conflict of Interest

The authors declare no conflict of interest

REFERENCES

1. Griffiths CEM, Tanya O. Bleiker, Daniel Creamer, John R. Ingram. Rook's Dermatology Handbook. first. Wiley-Blackwell; 2022.
2. Antiga E, Maglie R, Quintarelli L, Verdelli A, Bonciani D, Bonciolini V, et al. Dermatitis Herpetiformis: Novel Perspectives. *Frontiers in Immunology* 2019;10.
3. Plotnikova N, Miller JL. Dermatitis herpetiformis. *Skin Therapy Lett.* 2013 Mar-Apr;18(3):1-3. PMID: 23674144.
4. Nguyen CN, Kim SJ. Dermatitis Herpetiformis: An Update on Diagnosis, Disease Monitoring, and Management. *Medicina* 2021;57(8):843.
5. Mirza HA, Gharbi A, Bhutta BS. Dermatitis Herpetiformis. 2022.
6. Bolotin D, Petronic-Rosic V. Dermatitis herpetiformis. *Journal of the American Academy of Dermatology* 2011;64(6):1017-24.

7. Cinats AK, Parsons LM, Haber RM. Facial Involvement in Dermatitis Herpetiformis: A Case Report and Review of the Literature. *Journal of Cutaneous Medicine and Surgery* 2019;23(1):35-7.
8. Salmi T, Hervonen K. Current Concepts of Dermatitis Herpetiformis. *Acta Dermato Venereologica* 2020;100(5):adv00056-121.
9. James G. Dinulos MD. *Habif's Clinical Dermatology: A Color Guide to Diagnosis and Therapy*. Page: 182. 7th ed. Elsevier; 2020.
10. Reunala T, Hervonen K, Salmi T. Dermatitis Herpetiformis: An Update on Diagnosis and Management. *American Journal of Clinical Dermatology* 2021;22(3):329-38.
11. Al-Toma A, Volta U, Auricchio R, Castillejo G, Sanders DS, Cellier C, et al. European Society for the Study of Coeliac Disease (ESSCD) guideline for coeliac disease and other gluten-related disorders. *United European Gastroenterology Journal* 2019;7(5):583-613.
12. Vale ECS do, Dimatos OC, Porro AM, Santi CG. Consensus on the treatment of autoimmune bullous dermatoses: dermatitis herpetiformis and linear IgA bullous dermatosis Brazilian Society of Dermatology. *Anais Brasileiros de Dermatologia* 2019;94(2 suppl 1):48-55.
13. Kárpáti S. An Exception Within the Group of Autoimmune Blistering Diseases: Dermatitis Herpetiformis, the Gluten-Sensitive Dermopathy. *Dermatologic Clinics* 2011;29(3):463-8.
14. Antiga E, Caproni M. The diagnosis and treatment of dermatitis herpetiformis. *Clinical, Cosmetic and Investigational Dermatology* 2015;257.
15. Sewon Kang. *Fitzpatrick's Dermatology, Ninth Edition, 2-Volume Set (Fitzpatrick's Dermatology in General Medicine)*. 9th ed. McGraw Hill /Medical; 2019.
16. Hofmann SC, Juratli HA, Eming R. Bullous autoimmune dermatoses. *JDDG: Journal der Deutschen Dermatologischen Gesellschaft* 2018;16(11):1339-58.
17. Witte M, Zillikens D, Schmidt E. Diagnosis of Autoimmune Blistering Diseases. *Frontiers in Medicine* 2018;5.
18. Betz J, Grover RK, Ullman L. Evaluation of IgA epidermal transglutaminase ELISA in suspected dermatitis herpetiformis patients. *Dermatology Online Journal* 2021;27(4).
19. Borroni G, Biagi F, Ciocca O, Vassallo C, Carugno A, Cananzi R, et al. IgA anti-epidermal transglutaminase autoantibodies: a sensible and sensitive marker for diagnosis of dermatitis herpetiformis in adult patients. *Journal of the European Academy of Dermatology and Venereology* 2013;27(7):836-41.
20. Michael Occidental RHK. Skin nontumor Vesiculobullous and acantholytic reaction patterns Dermatitis herpetiformis. *pathology outlines* 2022.
21. Clarindo MV, Possebon AT, Soligo EM, Uyeda H, Ruaro RT, Empinotti JC. Dermatitis herpetiformis: pathophysiology, clinical presentation, diagnosis and treatment. *Anais Brasileiros de Dermatologia* 2014;89(6):865-77.
22. Ziemer M, Pemler S, Simon J, Treudler R. Acantholytic variant of Duhring's disease. *JDDG: Journal der Deutschen Dermatologischen Gesellschaft* 2016;14(2):186-8.
23. Burbidge T, Haber RM. Topical Dapsone 5% Gel as an Effective Therapy in Dermatitis Herpetiformis. *Journal of Cutaneous Medicine and Surgery* 2016;20(6):600-1.
24. Introduction: Standards of Medical Care in Diabetes-2018. *Diabetes Care* 2018;41(Supplement_1):S1-2.
25. Diagnosis and Classification of Diabetes Mellitus. *Diabetes Care* 2014;37(Supplement_1):S81-90.
26. Banday MZ, Sameer AS, Nissar S. Pathophysiology of diabetes: An overview. *Avicenna Journal of Medicine* 2020;10(04):174-88.
27. Saeedi P, Petersohn I, Salpea P, Malanda B, Karuranga S, Unwin N, et al. Global and regional diabetes prevalence estimates for 2019 and projections for 2030 and 2045: Results from the International Diabetes Federation Diabetes Atlas, 9th edition. *Diabetes Research and Clinical Practice* 2019;157:107843.
28. Corbin KD, Driscoll KA, Pratley RE, Smith SR, Maahs DM, Mayer-Davis EJ. Obesity in Type 1 Diabetes: Pathophysiology, Clinical Impact, and Mechanisms. *Endocrine Reviews* 2018;39(5):629-63.
29. Gurung M, Li Z, You H, Rodrigues R, Jump DB, Morgun A, et al. Role of gut microbiota in type 2 diabetes pathophysiology. *EBioMedicine* 2020;51:102590.

30. Lawrence JM, Contreras R, Chen W, Sacks DA. Trends in the Prevalence of Preexisting Diabetes and Gestational Diabetes Mellitus Among a Racially/Ethnically Diverse Population of Pregnant Women, 1999-2005. *Diabetes Care* 2008;31(5):899-904.
31. Thomas CC, Philipson LH. Update on Diabetes Classification. *Medical Clinics of North America* 2015;99(1):1-16.
32. Solis-Herrera, Carolina Curtis Triplitt, Charles Reasner, RAD. Classification of Diabetes Mellitus. *StatPearls* 2018;
33. Hervonen K, Viljamaa M, Collin P, Knip M, Reunala T. The occurrence of type 1 diabetes in patients with dermatitis herpetiformis and their first-degree relatives. *British Journal of Dermatology* 2004;150(1):136-8.
34. Alasmari B, Alkhodair R. Clinicopathological Profile of Dermatitis Herpetiformis Patients in Saudi Arabia. *Cureus* 2023;15(10).
35. Zhang F, Yang B, Lin Y, Chen S, Zhou G, Wang G, et al. Dermatitis herpetiformis in China: A report of 22 cases. *Journal of the European Academy of Dermatology and Venereology* 2012;26(7):903-7.
36. Handa S, Dabas G, De D, Mahajan R, Chatterjee D, Saika UN, et al. A retrospective study of dermatitis herpetiformis from an immunobullous disease clinic in north India. *International Journal of Dermatology* 2018;57(8):959-64.
37. Antiga E, Maglie R, Quintarelli L, Verdelli A, Bonciani D, Bonciolini V, Caproni M: Dermatitis herpetiformis: novel perspectives. *Front Immunol.* 2019, 10:1290. 10.3389/fimmu.2019.01290.
38. Al-Toma A, Volta U, Auricchio R, et al.: European Society for the Study of Coeliac Disease (ESsCD) guideline for coeliac disease and other gluten-related disorders. *United European Gastroenterol J.* 2019, 7:583-613. 10.1177/2050640619844125.
39. James WD, Berger TG, Elston DM, Odom RB. *Andrews' diseases of the skin clinical dermatology*. 10th ed. Philadelphia SE 961 pages: illustrations (chiefly color); 29 cm: Saunders Elsevier; 2006.
40. Dieterich W, Laag E, Bruckner-Tuderman L et al. Antibodies to tissue transglutaminase as serologic markers in patients with dermatitis herpetiformis. *J Invest Dermatol* 1999; 113: 133-136.
41. Cummins AG, Roberts-Thomson IC. Prevalence of celiac disease in the Asia-Pacific region. *J Gastroenterol Hepatol* 2009; 24: 1347-1351.
42. Holt S, Blackwell JN, Ch B. and diabetes mellitus STEPHEN HOLT. 1980;(January):15-7.
43. Harish MR, Shashikumar BM. Association of Dermatoses with Diabetes- A Case Control Study. 2012 Apr 23;
44. Tuomilehto J, Karvonen M, Pitkaniemi J et al. Record high incidence of type I (insulin-dependent) diabetes mellitus in Finnish children. *Diabetologica* 2000; 43: 682-4.
45. Reunala T, Collin P. Diseases associated with dermatitis herpetiformis. *Br J Dermatol* 1997; 136: 315-318.
46. Ohata C, Ishii N, Hamada T, et al. Distinct characteristics in Japanese dermatitis herpetiformis: a review of all 91 Japanese patients over the last 35 years. *Clin Dev Immunol* 2012; 2012: 562168.
47. Nguyen CN, Kim SJ. Dermatitis Herpetiformis: An Update on Diagnosis, Disease Monitoring, and Management. *Medicina* 2021;57(8):843.