Journal of Health and Medical Sciences

Dewi, D. A. R., Firdaus, J., Tasya, K., Winayu, S. T., & Maulida, S. (2026). Gluten-Responsive Probable Pediatric Dermatitis Herpetiformis Mimicking Varicella in a Five-Year-Old Child: A Case Report. *Journal of Health and Medical Sciences*, 9(3), 13-24.

ISSN 2622-7258

DOI: 10.31014/aior.1994.09.03.257

The online version of this article can be found at:
https://www.asianinstituteofresearch.org/

Published by:
The Asian Institute of Research

The Asian Institute of Research *Journal of Health and Medical Sciences* is a peer-reviewed International Journal. The journal covers scholarly articles in the fields of Medicine and Public Health, including medicine, surgery, ophthalmology, gynecology and obstetrics, psychiatry, anesthesia, pediatrics, orthopedics, microbiology, pathology and laboratory medicine, medical education, research methodology, forensic medicine, medical ethics, community medicine, public health, community health, behavioral health, health policy, health service, health education, health economics, medical ethics, health protection, environmental health, and equity in health. As the journal is Open Access, it ensures high visibility and the increase of citations for all research articles published. The *Journal of Health and Medical Sciences* aims to facilitate scholarly work on recent theoretical and practical aspects of Health and Medical Sciences.

ASIAN INSTITUTE OF RESEARCH
Connecting Scholars Worldwide

The Asian Institute of Research
Journal of Health and Medical Sciences
Vol.9, No.3, 2026: 13-24
ISSN 2622-7258

DOI: 10.31014/aior.1994.09.03.257

Gluten-Responsive Probable Pediatric Dermatitis Herpetiformis Mimicking Varicella in a Five-Year-Old Child: A Case Report

Dian Andriani Ratna Dewi[1], Juliandra Firdaus[2], Khufitha Tasya[3], Sendang Tri Winayu[2], Sausan Maulida[4]

[1] Faculty of Military Medicine Republic Indonesia Defense University
[2] Faculty of Medicine YARSI University
[3] Faculty of Medicine UKI University
[4] Faculty of Medicine Hasanuddin University

Correspondence: Dian Andriani Ratna Dewi, Faculty of Military Medicine Republic Indonesia Defense University. Tel: 0819-4631-1759. E-mail: Dianandrianiratnadewi@gmail.com
ORCID ID: https://orcid.org/0000-0002-9541-2790

Abstract
Background: Dermatitis herpetiformis (DH) is a gluten-associated autoimmune vesiculobullous dermatosis that is rare in children and may resemble infectious eruptions, particularly varicella. Case presentation: A 5-year-old boy presented with persistent, painful, and intensely pruritic grouped vesicles and tense bullae, predominantly affecting the posterior trunk and upper back. Although he had a history of varicella at 3 years of age, the current episode was not preceded by known varicella exposure, fever, or prodromal symptoms. The eruption was initially diagnosed as varicella due to its vesiculobullous morphology, and a 5-day course of acyclovir was administered. Over approximately 1 month of close monitoring, some lesions transiently dried and faded, but new grouped vesicles and bullae repeatedly appeared, became more widespread, and remained painful and pruritic. Recurrent flares were subsequently observed after ingestion of gluten-containing foods. Laboratory findings were unremarkable except for eosinophilia of 14% and a serum IgE of 91.88 IU/mL. IgA-based celiac serology, histopathology, and direct immunofluorescence were not performed because the parents declined. The clinical pattern, gluten-associated recurrence, lack of sustained response to acyclovir, and improvement with gluten avoidance supported a working diagnosis of gluten-responsive probable pediatric DH. Clinical improvement was achieved with lesion-directed topical therapy, symptomatic treatment, and a strict gluten-free diet. Conclusion: This case underscores a varicella-mimicking, gluten-responsive probable pediatric DH presentation and highlights the importance of transparent reporting of diagnostic uncertainty when confirmatory testing is unavailable.

Keywords: Case Report, Dermatitis Herpetiformis, Gluten-Free Diet, Pediatric Dermatology, Topical Corticosteroid, Varicella Mimic

1. Introduction

Dermatitis herpetiformis (DH), also known as Duhring disease, is a chronic autoimmune blistering dermatosis strongly associated with gluten-sensitive enteropathy and celiac disease. It classically presents with intensely pruritic grouped papules, vesicles, or small bullae, most often involving extensor surfaces, the trunk, buttocks, scalp, and nuchal area. Although DH is more frequently recognized in adults, pediatric cases are clinically

important because they may present with atypical distribution, prominent excoriation, or secondary inflammation, making the eruption resemble more common infectious or allergic dermatoses (Antiga et al., 2019; Nguyen & Kim, 2021; Reunala et al., 2021; Templet et al., 2007).

The pathogenesis of DH involves gluten-driven autoimmunity, IgA antibodies directed against transglutaminase antigens, and granular IgA deposition in the dermal papillae. Neutrophil-rich inflammation contributes to subepidermal blister formation and severe pruritus. In routine pediatric practice, intact vesicles may be difficult to appreciate because repeated scratching, crusting, and post-inflammatory pigmentary change can obscure the primary morphology. These features may lead to repeated treatment for common childhood infections, including varicella, before a noninfectious vesiculobullous disorder is considered (Görög et al., 2021; Salmi & Hervonen, 2020).

The standard confirmation of DH relies on direct immunofluorescence (DIF) of perilesional skin, demonstrating granular IgA deposition, and is supported by histopathology and celiac-related serology when available. However, confirmatory testing may be limited by access, cost, child-related procedural concerns, or parental refusal of biopsy. In such circumstances, the diagnosis should be reported cautiously as suspected or probable DH rather than definite DH. A careful dietary history, recurrent grouped vesiculobullous morphology, and response to gluten avoidance may provide important clinical clues, but they do not replace IgA-based serology or DIF.

This report presents a 5-year-old boy with a persistent, painful, and pruritic vesiculobullous eruption predominantly involving the posterior trunk and upper back. The patient had a history of varicella at age 3, and the current episode was not preceded by fever, prodromal symptoms, or known contact with a person with varicella. The eruption was initially managed as presumed varicella with a 5-day course of acyclovir. During approximately 1 month of close monitoring, some lesions transiently dried and faded, but this partial improvement was not sustained; new vesicles and bullae subsequently reappeared in larger numbers and became more extensive, particularly after the child consumed gluten-containing foods. Clinical improvement was observed following lesion-directed topical therapy and gluten avoidance. The diagnosis is deliberately classified as probable pediatric DH because IgA testing, histopathology, and DIF were not performed. This transparent terminology aligns with structured case-report principles and prevents overstatement of diagnostic certainty (Gagnier et al., 2013).

2. Case Presentation

2.1 Patient Information

A 5-year-old boy presented with grouped vesicles and large tense bullae accompanied by severe pruritus and pain. The clinically relevant episode began at age 5. The child had previously had varicella at 3 years of age. During the current vesiculobullous episode, the parents reported no known contact with a varicella patient, no fever before lesion onset, and no preceding systemic prodrome. No other household member was reported to have a similar acute vesicular eruption. The eruption was initially treated as presumed varicella with acyclovir for 5 days, followed by approximately 1 month of close clinical monitoring. During this observation period, several lesions appeared to dry and fade gradually; however, this mild improvement was temporary. New bullae then reappeared in greater numbers, remained painful and intensely pruritic, and extended predominantly over the posterior trunk and upper back. After the parents carefully reviewed the child's dietary pattern, recurrent flares were repeatedly noticed after consumption of gluten-containing foods. Importantly, the parents did not report a history of similar chronic or recurrent bullous eruptions since infancy or at age 1. No significant past medical history other than previous varicella was documented, and there was no known family history of autoimmune disease, celiac disease, or gluten-sensitive disorders.

To ensure confidentiality, the patient is described without initials or identifying personal information. Clinical photographs were selected to depict the posterior trunk and back, thereby minimizing the risk of facial identification.

2.2 Clinical Findings

According to the parents, clusters of large, tense bullae appeared repeatedly and were most numerous on the posterior trunk, especially the upper and mid-back. Additional lesions involved the posterior neck, shoulders, and limited areas of the extremities. The lesions were associated with marked itching, pain, excoriation, crusting, and post-inflammatory hyperpigmentation. The current episode was not accompanied by fever before lesion onset, and there was no known varicella contact. During the 1-month close monitoring period, the family initially observed that some lesions gradually became drier and less prominent, but new lesions subsequently appeared in greater numbers. When the child's meals were reviewed more carefully, the recurrence was temporally associated with intake of gluten-containing foods, including bread, noodles, nuggets, and other processed wheat-based foods. Conversely, the eruption tended to improve when these foods were restricted and worsened when they were reintroduced.

Physical examination during an active flare revealed multiple grouped erythematous papules, vesicles, and tense bullae on an erythematous base, with the highest lesion burden on the back. Several lesions showed excoriation, crusting, secondary inflammatory change, and residual hyperpigmentation (Figure 1). No systemic symptoms, urinary complaints, or signs of severe dehydration were documented at presentation. No gastrointestinal manifestations were recorded in the available clinical summary.

2.3 Timeline

Table 1: Clinical timeline

Time point	**Clinical course**
Age 3 years	The child reportedly had varicella at 3 years of age.
Age 5 years	The patient developed persistent painful and intensely pruritic grouped vesicles and tense bullae, predominantly on the posterior trunk, especially the upper and mid-back. There was no known contact with a patient with varicella and no fever or prodromal symptoms before lesion onset.
Initial diagnosis, 5-day antiviral therapy, and 1-month monitoring	The eruption was assumed to represent varicella because of its vesiculobullous morphology. Oral acyclovir was administered for 5 days. After completion of antiviral therapy, the patient was monitored closely for approximately 1 month; during this period, several lesions gradually dried and faded, but new grouped vesicles and bullae subsequently reappeared in larger numbers, remained painful and pruritic, spread predominantly over the posterior trunk, and showed no sustained clinical improvement. The recurrence was later noticed to occur after intake of gluten-containing foods.
Supportive and antimicrobial therapy	The patient received compounded ibuprofen for pain, loratadine syrup for pruritus, and cefadroxil syrup when crusted or suspected secondarily infected lesions were present.
Available laboratory assessment	Urinalysis was within normal limits. Routine blood examination was within normal limits except for eosinophilia of 14%. Serum IgE was 91.88 IU/mL. Total serum IgA, tissue transglutaminase IgA, and endomysial IgA antibodies were not performed.
Diagnostic limitation	Histopathologic examination and DIF were proposed but were not performed because the parents declined skin biopsy; therefore, the case was classified as probable rather than definite pediatric DH.

Therapeutic adjustment	Treatment was adjusted to lesion-directed clobetasol propionate 0.05% cream, mupirocin 2% cream for crusted or suspected secondarily infected lesions, symptomatic therapy, and a strict gluten-free diet.
Follow-up	After reassessment and treatment adjustment, clinical improvement was reported, with drying of lesions and reduced inflammation at approximately 2-4 weeks (Figure 2), followed by sustained improvement during longer follow-up while gluten avoidance was maintained (Figure 3). The family noted recurrence or worsening when gluten-containing foods were reintroduced.

Note. DH = dermatitis herpetiformis; DIF = direct immunofluorescence; IgA = immunoglobulin A; IgE = immunoglobulin E.

2.4 Diagnostic Assessment

The eruption was initially treated as presumed varicella because of its vesiculobullous morphology. The patient reportedly received oral acyclovir for 5 days. After completion of the antiviral course, the child was monitored closely for approximately 1 month. During this period, some bullae transiently dried and became less prominent, but the improvement was incomplete and short-lived. New grouped vesicles and tense bullae then reappeared in greater numbers, remained painful and intensely pruritic, and became more extensive over the posterior trunk. Several clinical details made uncomplicated primary varicella less likely: the child had previously had varicella at 3 years of age, there was no known recent contact with a varicella case, there was no fever or prodromal illness before the eruption, and the course was relapsing rather than self-limited. The family subsequently recognized that repeated flares tended to occur after the child ate gluten-containing foods. The absence of sustained improvement after completion of acyclovir therapy, together with a relapsing course during follow-up and a gluten-related temporal pattern, prompted reconsideration of the diagnosis.

Available laboratory data showed urinalysis and routine blood examination within normal limits, except for eosinophilia of 14%. Serum IgE was recorded as 91.88 IU/mL in the available laboratory document; because laboratory reference ranges may vary, this value was interpreted cautiously. No IgA-based assessment was performed; specifically, total serum IgA, tissue transglutaminase IgA, and endomysial IgA antibodies were not checked. Histopathologic examination and DIF were proposed but not performed because the parents declined a skin biopsy after discussion.

Because IgA-based serologic testing, histopathology, and DIF were unavailable, a definitive diagnosis of DH could not be established. The case was therefore classified as gluten-responsive probable pediatric DH based on grouped vesiculobullous morphology, severe pruritus and pain, dominant posterior trunk involvement, previous varicella at 3 years of age, absence of known varicella contact or fever before lesion onset, recurrent flare after gluten-containing food intake, worsening despite a 5-day acyclovir course and 1-month observation, and reproducible family-reported clinical improvement during gluten avoidance. Differential diagnoses included varicella, bullous impetigo, allergic or contact dermatitis with secondary infection, linear IgA bullous dermatosis, childhood bullous pemphigoid, epidermolysis bullosa acquisita, insect-bite hypersensitivity, and other pediatric autoimmune blistering diseases (Table 2).

The presence of eosinophilia and elevated IgE supported consideration of allergic or hypersensitivity-associated disorders in the differential diagnosis. However, the grouped bullous morphology, posterior truncal clustering, chronicity, and family-reported dietary relationship with gluten made DH a significant clinical consideration. The absence of histopathology and DIF was acknowledged as a major limitation, and the diagnostic terminology was deliberately maintained as probable DH.

Table 2: Differential diagnostic considerations

Differential diagnosis	Findings supporting consideration	Findings less compatible with the final working diagnosis
Varicella	Childhood vesicular eruption; initial vesiculobullous morphology prompted empiric acyclovir.	Previous varicella at 3 years of age, no known varicella contact, no fever or prodrome before lesions, relapsing 1-month course, posterior trunk clustering, recurrent flare after gluten intake, and no sustained response after acyclovir.
Bullous impetigo or secondary bacterial infection	Crusting, erosion, and secondary inflammatory change were present in several lesions.	Grouped recurrent tense bullae and gluten-associated fluctuation were not explained by primary bacterial infection alone; mupirocin and cefadroxil were used only for crusted or suspected secondarily infected lesions.
Allergic or contact dermatitis with secondary infection	Pruritus, eosinophilia, and crusting supported consideration of allergic or hypersensitivity-associated disease.	Large grouped tense bullae on the posterior trunk, recurrent flares after gluten exposure, and sustained improvement during gluten avoidance supported DH within the differential, although not definitively.
Linear IgA bullous dermatosis	Age group and tense bullae may overlap with this diagnosis.	DIF was unavailable, so a linear IgA pattern could not be confirmed or excluded; the family-reported gluten relationship favored probable DH clinically.
Childhood bullous pemphigoid	Tense bullae and pruritus can occur in children.	No confirmatory histopathology or DIF; distribution and gluten-related recurrence were more suggestive of probable DH in this clinical context.
Dermatitis herpetiformis	Grouped vesicles and tense bullae, severe pruritus and pain, posterior trunk predominance, recurrent flares after gluten-containing foods, and improvement during gluten avoidance.	No IgA-based celiac serology, histopathology, or DIF; therefore, the appropriate diagnostic term is probable DH, not confirmed DH.

Note. DH = dermatitis herpetiformis; DIF = direct immunofluorescence.

2.5 Therapeutic Intervention

During the initial presumed varicella episode, the patient received a 5-day course of acyclovir, which did not result in meaningful sustained improvement. Over approximately 1 month of close monitoring, some lesions appeared to dry and fade gradually, but this partial improvement was followed by recurrent crops of new vesicles and bullae that became more numerous and remained painful, pruritic, crusted, and inflamed. The parents subsequently reported that these recurrences were most noticeable after the child consumed gluten-containing foods, which reinforced the rationale for dietary intervention. Symptomatic and supportive medications included compounded ibuprofen powder for pain, loratadine syrup for pruritus, and cefadroxil syrup when crusted and inflamed lesions raised concern for secondary bacterial infection. Following reassessment, treatment was redirected toward controlling inflammation, preventing secondary infection, relieving symptoms, and implementing dietary intervention.

Clobetasol propionate 0.05% cream was applied in a lesion-directed manner to active inflammatory vesiculobullous plaques, while mupirocin 2% cream was used on crusted or suspected secondarily infected lesions. The family was counseled to use potent topical corticosteroid cautiously because of the patient's young age and the risk of local adverse effects with prolonged or extensive use.

A strict gluten-free diet was recommended, with avoidance of wheat, rye, barley, bread, noodles, nuggets, and other gluten-containing or potentially cross-contaminated processed foods. Practical counseling included reviewing food labels, identifying hidden sources of gluten, and preventing cross-contamination at home.

Dapsone was not initiated. Although dapsone can provide rapid symptomatic relief in confirmed DH, its use requires diagnostic confidence and baseline monitoring for hemolysis, methemoglobinemia, and other adverse effects, especially in children (Antiga & Caproni, 2015; Görög et al., 2021). Given the absence of confirmatory IgA testing and parental refusal of biopsy for histopathology and DIF, topical therapy and dietary management were prioritized, with an emphasis on the need for confirmatory evaluation if symptoms recur or worsen.

2.6 Follow-up and Outcomes

After initiation of clobetasol propionate 0.05% cream, mupirocin 2% cream, symptomatic therapy, and gluten avoidance, the patient showed progressive clinical improvement, as reported by the family and documented in serial clinical photographs. This improvement contrasted with the preceding 1-month period of close monitoring after the 5-day acyclovir course, during which lesions had only partially dried and faded before recurring in greater numbers, especially after gluten-containing foods. Within approximately 2-4 weeks after treatment adjustment, the posterior trunk lesions became drier and less inflamed, with reduced pruritus, pain, and crusting. No new large tense bullae were reported during early follow-up when gluten avoidance was maintained (Figure 2).

Longer follow-up showed sustained improvement of the back with minimal residual post-inflammatory change (Figure 3). The family reported better disease control during periods of dietary adherence and worsening after reintroduction of gluten-containing foods. This pattern was particularly noted after consuming foods such as bread, noodles, nuggets, and other wheat-based processed products. No severe systemic complications were documented in the available clinical summary. Because IgA-based serology was not checked, serologic monitoring of disease activity or dietary adherence could not be reported.

3. Discussion

3.1 Diagnostic Challenge and Varicella Mimicry

This case illustrates a practical diagnostic challenge in pediatric vesiculobullous disease. Varicella is a common cause of vesicular eruption in children, and empiric antiviral treatment may be reasonable when the morphology appears infectious. However, several clinical features in this case argued against uncomplicated primary varicella: the child had previously had varicella at 3 years of age, there was no known contact with a patient with varicella, no fever or prodromal symptoms preceded the eruption, and the lesions followed a recurrent course after a completed 5-day acyclovir regimen and approximately 1 month of close observation. Several lesions initially appeared to dry and fade, but recurrent crops of grouped vesicles and bullae subsequently appeared in larger numbers. Posterior trunk clustering, grouped vesiculobullous morphology, increasing lesion burden during follow-up, and a family-observed relationship between gluten exposure and recurrent flares supported consideration of DH or another noninfectious vesiculobullous disorder rather than uncomplicated varicella.

Pediatric DH is uncommon and can be difficult to recognize because primary vesicles are often disrupted by scratching, crusting, and secondary inflammation. Lesions may therefore resemble bullous impetigo, allergic dermatitis, insect-bite hypersensitivity, or other autoimmune blistering disorders. This case reinforces that dietary history can be a useful clinical clue when gastrointestinal symptoms are absent or not prominent (Nguyen & Kim, 2021; Reunala et al., 2021).

3.2 Novelty and Educational Value

The educational value of this report is derived from its real-world diagnostic context rather than from introducing a novel treatment or definitively establishing a rare entity. This case documents a 5-year-old child with a varicella-mimicking vesiculobullous eruption, despite a history of varicella at 3 years of age, no known varicella contact, and no fever before lesion onset. The eruption persisted after a completed 5-day course of acyclovir and approximately 1 month of close monitoring. Although some lesions initially dried and faded, new painful and pruritic bullae subsequently reappeared in larger numbers, predominantly on the posterior back. Careful parental observation identified recurrent flares after gluten-containing foods, and improvement was observed following combined topical anti-inflammatory, antimicrobial, symptomatic, and dietary management. This sequence provides a practical clinical indicator: persistent, recurrent, or progressive grouped bullae with pruritus, pain, lack of sustained response to antiviral therapy, absence of typical varicella epidemiologic or systemic features, and possible dietary fluctuation should prompt reconsideration of noninfectious vesiculobullous disorders, including probable DH.

A second educational point is the transparent classification of the case as probable rather than definite DH. Because IgA assessment, histopathology, and DIF were not performed, the diagnosis was intentionally not overstated. This approach strengthens the scientific integrity of the report and aligns with case-report standards requiring clear diagnostic reasoning, explicit uncertainty, and limitations (Gagnier et al., 2013).

3.3 Diagnostic Limitation and Need for Confirmation

The diagnosis of DH is usually supported by granular IgA deposition in dermal papillae on DIF of perilesional skin. Serologic tests, especially IgA tissue transglutaminase and IgA endomysial antibodies, may support the diagnosis and help monitor adherence to gluten avoidance. Total serum IgA is also relevant because IgA deficiency may cause false-negative IgA-based serologic results (Görög et al., 2021; Salmi & Hervonen, 2020).

In this patient, none of the IgA-based tests were performed. Skin biopsy for histopathology and DIF was proposed but not performed because the parents declined the procedure. Therefore, the case cannot be presented as confirmed DH. The most scientifically appropriate wording is "probable pediatric DH" or "suspected DH" in the clinical differential diagnosis. If the disease recurs, referral for celiac serology (total IgA, tissue transglutaminase IgA, and endomysial IgA) and for a perilesional skin biopsy with DIF would be recommended. If IgA deficiency is suspected or confirmed, IgG-based celiac serology may be required.

The elevated eosinophil percentage and recorded IgE value also require cautious interpretation. Eosinophilia can occur in allergic, parasitic, drug-related, or other inflammatory conditions and is not specific for DH. These findings do not exclude DH but support maintaining a broad differential diagnosis. Their inclusion prevents selective reporting and helps readers understand the uncertainty of the case.

3.4 Management Implications

The main long-term management of confirmed DH is a strict gluten-free diet, which targets both cutaneous disease and underlying gluten-sensitive enteropathy. Dietary treatment can reduce medication requirements over time and may reduce systemic complications associated with celiac disease (Nguyen & Kim, 2021; Pasternack et al., 2021; Reunala et al., 2021). In this patient, the family-reported pattern of slight temporary improvement followed by recurrent flares after gluten-containing foods, together with subsequent improvement during gluten avoidance, supports the clinical suspicion but should be interpreted as supportive rather than diagnostic.

Topical clobetasol propionate 0.05% may reduce inflammation and pruritus in localized inflammatory lesions, but it does not treat the underlying gluten-driven autoimmune mechanism of DH. In children, potent topical corticosteroids should be used carefully, for a limited duration, and under clinical supervision. Mupirocin 2% is appropriate when crusted lesions raise concern for secondary bacterial infection. Ibuprofen and loratadine may help address pain and pruritus, respectively, but they are supportive rather than disease-modifying therapies. These

treatments may explain short-term improvement in inflammation and crusting, whereas gluten avoidance may explain reduced recurrence during adherence.

Dapsone is an established option for rapid symptom control in confirmed DH, but it was not used in this case. This conservative approach was reasonable given the absence of confirmatory testing, parental refusal of biopsy, and the need for safety monitoring in a young child. The case therefore supports a stepwise approach: treat pain, pruritus, secondary infection, and inflammation; document dietary response; counsel the family; and pursue confirmatory testing when available or when disease activity persists.

Importantly, clinical improvement in this case cannot be attributed solely to gluten avoidance, as topical corticosteroid therapy, topical antimicrobial treatment, and symptomatic medications were used concurrently. The gluten-related fluctuation, including recurrence after intake of gluten-containing foods during close monitoring, is therefore best interpreted as supportive evidence for a probable diagnosis, not as a substitute for confirmatory DH testing.

3.5 Limitations

This report has several limitations. First, IgA-based serology, total serum IgA, histopathology, and DIF were not performed. Second, histopathology and DIF were unavailable because the parents declined biopsy, limiting diagnostic certainty. Third, although the parents reported previous varicella at 3 years of age, no known varicella contact, and no fever before lesion onset, varicella vaccination status and virologic confirmation such as polymerase chain reaction testing were not available in the clinical summary. Fourth, serum IgE was recorded as 91.88 IU/mL, but interpretation should follow the reference range of the original laboratory report. Fifth, dietary adherence and response to gluten re-exposure were based on family report rather than standardized dietary assessment or supervised gluten challenge. The observed gluten-related fluctuation should therefore be interpreted as supportive clinical evidence rather than diagnostic confirmation, because topical corticosteroid, topical antimicrobial therapy, and symptomatic medications were administered during the same treatment period.

4. Conclusion

Probable pediatric DH should be considered in children with persistent, intensely pruritic and painful grouped vesiculobullous eruptions that mimic varicella but do not show sustained improvement after appropriate antiviral therapy. In this case, previous varicella at 3 years of age, absence of known varicella contact, absence of fever before lesion onset, posterior trunk predominance, relapsing progression after a 5-day acyclovir course and approximately 1 month of close monitoring, recurrent lesions after gluten-containing food intake, and improvement after clobetasol propionate 0.05% cream, mupirocin 2% cream, symptomatic therapy, and a gluten-free diet supported suspicion of DH. However, the absence of IgA testing, histopathology, and DIF prevented definitive diagnosis. Clear reporting of diagnostic uncertainty is essential, and confirmatory IgA-based serology with perilesional biopsy for DIF should be pursued when available and acceptable to the family.

Author Contributions:
Conceptualization, D.A.R.D.; clinical data acquisition, D.A.R.D., J.F., and S.T.W.; data curation, J.F., K.T., and S.M.; writing – original draft, J.F., K.T., and S.M.; writing – review and editing, D.A.R.D., J.F., K.T., S.M., and S.T.W.; supervision, D.A.R.D. All authors have read and approved the submitted version and agree to be accountable for the accuracy and integrity of the work.

Funding: This research received no external funding. The APC was funded by the authors.

Conflicts of Interest: The authors declare no conflict of interest.

Informed Consent Statement/Ethics Approval: Written informed consent for publication of the clinical information and anonymized clinical photographs was obtained from the patient's parent/legal guardian. Skin

biopsy for histopathology and direct immunofluorescence was proposed but declined by the parents after discussion.

Data Availability Statement: Data supporting the case description are contained within the clinical record and are not publicly available to protect patient confidentiality.

Acknowledgments: The authors thank the patient and family for their cooperation.

Declaration of Generative AI and AI-assisted Technologies: During manuscript preparation, generative AI was used for language editing, structural organization, and formatting assistance. The authors reviewed, edited, and verified the manuscript content and remain fully responsible for the accuracy, integrity, and originality of the submitted work.

References

Antiga, E., & Caproni, M. (2015). The diagnosis and treatment of dermatitis herpetiformis. Clinical, Cosmetic and Investigational Dermatology, 8, 257-265. https://doi.org/10.2147/CCID.S69127

Antiga, E., Maglie, R., Quintarelli, L., Verdelli, A., Bonciani, D., Bonciolini, V., et al. (2019). Dermatitis herpetiformis: Novel perspectives. Frontiers in Immunology, 10, 1290. https://doi.org/10.3389/fimmu.2019.01290

Gagnier, J. J., Kienle, G., Altman, D. G., Moher, D., Sox, H., Riley, D., et al. (2013). The CARE guidelines: Consensus-based clinical case reporting guideline development. Journal of Medical Case Reports, 7, 223. https://doi.org/10.1186/1752-1947-7-223

Görög, A., Antiga, E., Caproni, M., Cianchini, G., De, D., Dmochowski, M., et al. (2021). S2k guidelines (consensus statement) for diagnosis and therapy of dermatitis herpetiformis initiated by the European Academy of Dermatology and Venereology (EADV). Journal of the European Academy of Dermatology and Venereology, 35(6), 1251-1277. https://doi.org/10.1111/jdv.17183

Nguyen, C. N., & Kim, S. J. (2021). Dermatitis herpetiformis: An update on diagnosis, disease monitoring, and management. Medicina, 57(8), 843. https://doi.org/10.3390/medicina57080843

Pasternack, C., Hervonen, K., Mansikka, E., Reunala, T., Collin, P., Kaukinen, K., & Salmi, T. T. (2021). Persistent skin symptoms after diagnosis and on a long-term gluten-free diet in dermatitis herpetiformis. Acta Dermato-Venereologica, 101(9), adv00555. https://doi.org/10.2340/00015555-3914

Reunala, T., Salmi, T. T., Hervonen, K., Kaukinen, K., & Collin, P. (2021). Dermatitis herpetiformis: An update on diagnosis and management. American Journal of Clinical Dermatology, 22(3), 329-338. https://doi.org/10.1007/s40257-020-00584-2

Salmi, T., & Hervonen, K. (2020). Current concepts of dermatitis herpetiformis. Acta Dermato-Venereologica, 100(5), 115-121. https://doi.org/10.2340/00015555-3401

Templet, J. T., Welsh, J. P., & Cusack, C. A. (2007). Childhood dermatitis herpetiformis: A case report and review of the literature. Cutis, 80(6), 473-476.

Figures and Figure Legends

A **B**

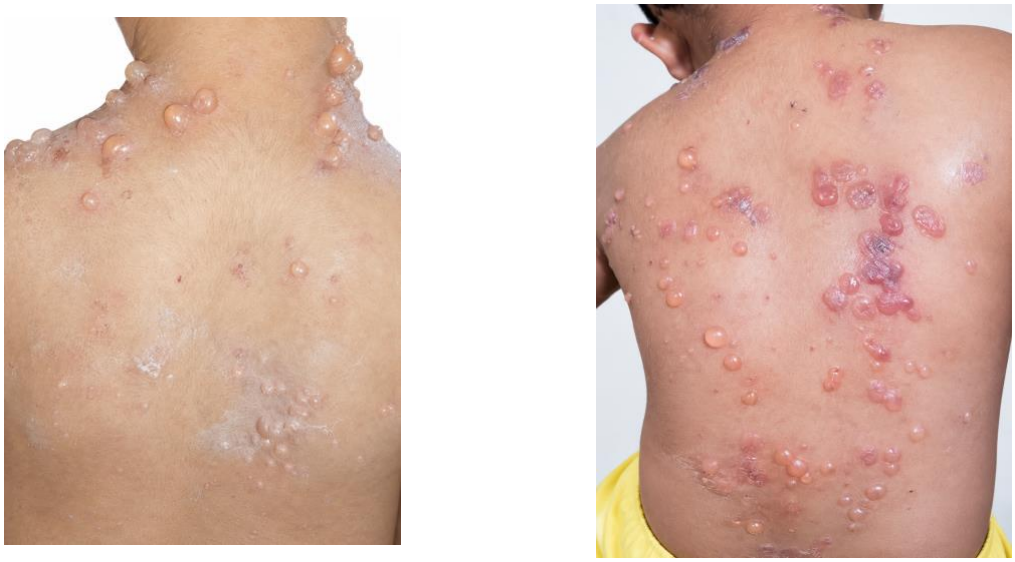

Figure 1. Initial presentation and progression during monitoring after presumed varicella treatment. (A) Initial clinical photograph at 5 years of age showing multiple grouped vesicles and tense bullae over the posterior neck, shoulders, and upper back, accompanied by excoriation, crusting, and early post-inflammatory change. The morphology initially led to clinical consideration of varicella, although the child had previously had varicella at 3 years of age, had no known contact with a patient with varicella, and had no fever before lesion onset. The patient received acyclovir for 5 days. (B) After approximately 1 month of close monitoring following the completed antiviral course, some lesions had transiently dried or faded, but recurrent vesiculobullous lesions remained prominent, reappeared in larger numbers, and became more extensive over the posterior trunk, especially the upper and mid-back. The family later recognized that new crops of lesions tended to occur after gluten-containing foods were consumed.

A B

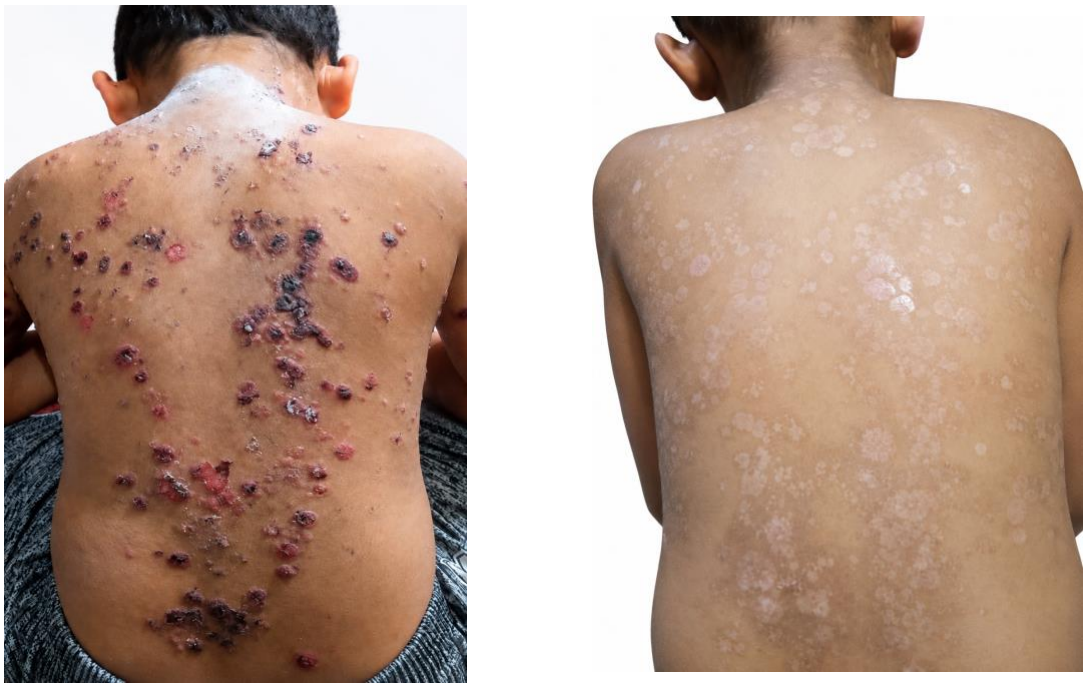

Figure 2. Early response after treatment adjustment. (A) Approximately 2 weeks after treatment was redirected to lesion-directed clobetasol propionate 0.05% cream, mupirocin 2% cream for crusted or suspected secondarily infected lesions, symptomatic therapy, and strict gluten avoidance, active inflammation was still evident; however, compared with the preceding recurrent flare, the lesions showed transition toward crusting, drying, and reduced tense bullous activity. (B) At approximately 4 weeks after treatment adjustment, vesiculobullous activity had largely dried, inflammation and crusting had decreased, and residual post-inflammatory pigmentary change became the predominant finding. This early improvement differed from the earlier monitoring period, in which partial fading was followed by recurrent flares after gluten-containing food intake.

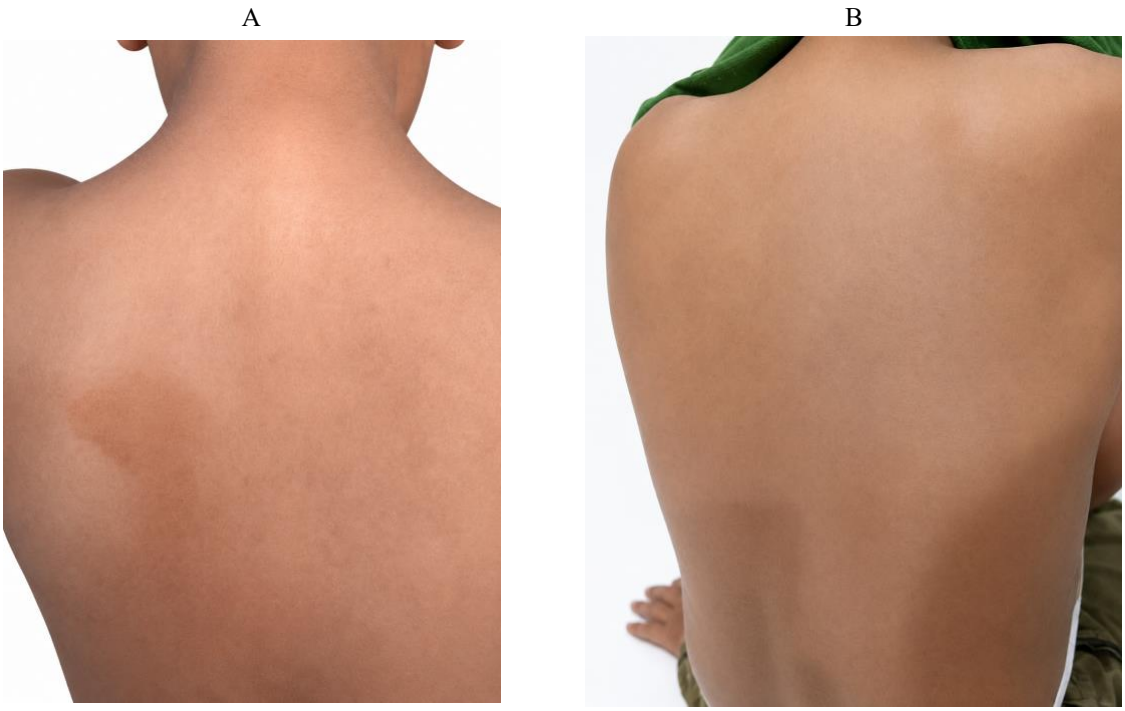

Figure 3. Late and long-term follow-up after treatment adjustment. (A) Approximately 8 weeks after treatment adjustment, the upper back showed marked clearance of active bullae with only mild residual post-inflammatory color change in the photographed area. (B) At approximately 1 year of follow-up, the back remained clinically quiet with minimal residual pigmentation or scarring and no obvious active vesiculobullous lesions.