

13. Seymour E, Mallory G, Morales-Demori R. Surgical and Cardiac Catheterization Outcomes of Scimitar Syndrome Patients: A Three Decade Single-Center Experience. *Pediatr Cardiol.* 2023 Mar;44(3):579-586. doi: 10.1007/s00246-022-02965-2.
14. Vida VL, Guariento A. A sword threatening the heart: The scimitar syndrome. *JTCVS Tech.* 2020 Jan 25;1:75-80. doi: 10.1016/j.jtc.2020.01.017.
15. Wang H, Kalfa D, Rosenbaum MS, Ginns JN, Lewis MJ, Glickstein JS, et al. Scimitar Syndrome in Children and Adults: Natural History, Outcomes, and Risk Analysis. *Ann Thorac Surg.* 2018 Feb;105(2):592-598. doi: 10.1016/j.athoracsur.2017.06.061.

Conflict of interest. The author has no conflicts of interest to declare.

ORCID: Salekhova G.B. <https://orcid.org/0000-0002-2434-2377>, Polukhova A.A. <https://orcid.org/0009-0005-5647-4840>, Rahimova N.J. <https://orcid.org/0000-0002-1551-2979>, Alkhasova A.F. <https://orcid.org/0009-0005-9095-6405>, Abbasaliyeva A.I. <https://orcid.org/0009-0000-1679-1810>, Aghayeva I.A. <https://orcid.org/0009-0007-0435-8666>, Iskenderli G.N. <https://orcid.org/0009-0004-9481-8280>.

Article received: 14.08.2025

DOI 10.26724/2079-8334-2026-3-97-284-289

UDC 616.5-007.21-071-053.2

**Tsvirenko S.M., Pokhylyko V.I., Cherniavska Yu.I., Shcherban O.A., Soloviova H.O.,
Zhuk L.A., Liulka A.R.**
Poltava State Medical University, Poltava, Ukraine

CLINICAL CASE OF APLASIA CUTIS CONGENITA HIGHLIGHTING THE DIAGNOSTIC SIGNIFICANCE OF THE HAIR COLLAR SIGN IN A NEWBORN

e-mail: yu.cherniavska@pdmu.edu.ua

Aplasia cutis congenita is a rare congenital disorder characterized by the absence of skin at birth, most commonly affecting the scalp. The aim of the study is to present a clinical observation of a case of congenital aplasia of the skin in a child. The article describes the case of girl K., hospitalized on the 4th day for examination and determination of management tactics due to the presence of 2 blisters filled with clear fluid in the parietal area, which were determined from birth. The skin around them was not changed, along the head to the right of the sagittal suture there was a strand of long dark hair. At 3 months of life one of the blisters has become less tense, its volume has decreased, a white scar begins to form in the center. At 6 months – it was observed that atrophic flat scars covered with thin epidermis formed in place of both bullae. ACC is rare and therefore the awareness of neonatologists, pediatricians and dermatologists is extremely important for early clinical diagnosis and conducting studies to exclude a concomitant brain defect, especially in the presence of a "hair collar" symptom, in order to prevent further complications.

Key words: aplasia cutis congenita, newborn, blisters, hair collar symptom.

**Цвіренко С.М., Похилько В.І., Чернявська Ю.І., Щербань О.А., Соловійова Г.О.,
Жук Л.А., Люлька А.Р.**

КЛІНІЧНИЙ ВИПАДОК ВРОДЖЕНОЇ АПЛАЗІЇ ШКІРИ У НОВОНАРОДЖЕНОГО З АКЦЕНТОМ НА ДІАГНОСТИЧНЕ ЗНАЧЕННЯ СИМПТОМУ ВОЛОСЯНОГО КОМІРЦЯ

Вроджена аплазія шкіри – це рідкісне вроджене захворювання, що характеризується відсутністю шкіри при народженні, найчастіше вражаючи волосисту частину голови. Метою дослідження є представлення клінічного спостереження за випадком вродженої аплазії шкіри у дитини. У статті описано випадок дівчинки К., госпіталізованої на 4-й день для обстеження та визначення тактики ведення у зв'язку з наявністю 2 пухирів, заповнених прозорою рідиною, утім'яній ділянці, які визначалися з народження. Шкіра навколо них не змінена, вздовж голови праворуч від сагітального шва було пасмо довгого темного волосся. На 3 місяці життя один з пухирів став менш напруженим, його об'єм зменшився, у центрі почав формуватися білий рубець. На 6 місяці життя було виявлено, що на місці обох булл утворилися атрофічні плоскі рубці, покриті тонким епідермісом. Вроджена аплазія шкіри зустрічається рідко, тому обізнаність неонатологів, педіатрів та дерматологів надзвичайно важлива для ранньої клінічної діагностики та проведення досліджень з метою виключення супутнього дефекту мозку, особливо за наявності симптому волосяного комірка, щоб запобігти подальшим ускладненням.

Ключові слова: вроджена аплазія шкіри, новонароджений, пухирі, симптом волосяного комірка.

The study is a fragment of the research project "To develop an improved algorithm for diagnostic and genetic testing for the early assessment of metabolic disorders and diseases, as well as a system of preventive and therapeutic measures for newborns and children", state registration No. 0120U102856.

Aplasia cutis congenita (ACC) is a rare and clinically heterogeneous congenital disorder characterized by the complete or partial absence of skin at birth. The condition most frequently affects the scalp, although lesions may occur on virtually any anatomical site and may vary substantially in number, morphology, size, and depth of tissue involvement

[10]. ACC may present as a small superficial defect, an ulceration, a membranous lesion, or a fluid-filled/bullous lesion, while extensive forms may involve the subcutaneous tissues, calvarium, dura mater, and intracranial structures [2, 6]. The rarity of the disorder and its marked phenotypic variability may contribute to diagnostic difficulties, particularly

in the neonatal period.. A large European population-based study conducted within the EUROCAT network identified its prevalence of approximately 5.10 per 100000 births. The scalp was the predominant site of involvement, accounting for 96.4 % of cases, while associated congenital anomalies were identified in approximately one-third of affected children [6]. These findings emphasize, that systematic evaluation for associated abnormalities may be clinically relevant. The pathogenesis of ACC remains incompletely understood and is considered multifactorial. Proposed mechanisms include disturbances of embryonic vascular supply, impaired tissue development, intrauterine infection, teratogenic exposure, mechanical factors, placental abnormalities, and genetic predisposition [2, 7]. ACC may occur as an isolated congenital defect or as part of a broader developmental or genetic disorder. Associations with Adams–Oliver syndrome, epidermolysis bullosa, fetus papyraceus, chromosomal abnormalities, and other congenital malformation syndromes have been described [7, 13]. Experimental and genetic studies have demonstrated an important role of the KCTD1 and KCTD15 genes in cranial neural crest cells, cranial suture development, and skin morphogenesis. [9]. The clinical classification proposed by Frieden remains widely used for characterization of ACC. [13]. Lesions may differ not only in their anatomical distribution but also in morphology and association with extracutaneous abnormalities. A particularly important clinical finding is the presence of a hair-collar sign, defined as a localized ring or tuft of unusually long, dark, coarse hair surrounding a congenital scalp lesion. This sign has attracted considerable attention because it may indicate an underlying neuroectodermal or cranial developmental abnormality. A multicenter study demonstrated that the hair-collar and hair-tuft signs may be associated with underlying neurovascular abnormalities and emphasized the importance of appropriate neuroimaging in affected children [14, 16]. Similarly, a retrospective analysis of 90 patients with ACC demonstrated that vertex or midline localization, the presence of a hair-collar sign, vascular stains, and nodules were significant clinical indicators of underlying skull or cerebrovascular involvement [12, 13]. Therefore, recognition of these signs during the neonatal examination may have implications beyond the diagnosis of the skin lesion itself [7]. Dermoscopic and clinical studies have further demonstrated that ACC may display characteristic patterns of scarring alopecia and follicular abnormalities, supporting the value of careful morphological assessment in selected cases [11, 17]. Management of ACC depends primarily on lesion size, depth, anatomical localization, associated abnormalities, and the clinical condition of the infant. Small superficial lesions without evidence of underlying structural abnormalities can frequently be

managed conservatively with protection of the affected area, appropriate wound care, and monitoring for infection or bleeding [7]. In contrast, extensive defects involving the calvarium, dura mater, or major intracranial vessels may require multidisciplinary surgical management [4, 10]. Despite increasing recognition of the clinical features and potential complications of ACC, the condition remains uncommon, and evidence concerning its diagnosis and management is largely derived from retrospective studies, case series, and individual case reports. This is particularly relevant for unusual clinical variants, including bullous ACC, which may initially be misinterpreted as a primary vesiculobullous disorder. The described case highlights the importance of early recognition of characteristic cutaneous signs and appropriate evaluation for potential associated cranial abnormalities in newborns with congenital scalp lesions.

The purpose of the study was to present a clinical observation of a case of congenital aplasia of the skin in a child.

Materials and methods. The present publication represents a detailed clinical case report of a newborn with a rare bullous phenotype of aplasia cutis congenita. The child was observed at the Department of Pediatrics #1 with Neonatology, Poltava State Medical University, Ukraine. The clinical observation included assessment during the early neonatal period and subsequent follow-up at 3 and 6 months of age. The patient was hospitalized on the fourth day of life for comprehensive clinical evaluation and determination of further management because of two congenital bullous lesions located in the parietal region of the scalp. According to the parents, both lesions had been present from birth. The clinical assessment was aimed at characterizing the morphology and anatomical localization of the lesions, evaluating the general condition of the newborn, identifying potential extracutaneous abnormalities, and determining the need for additional diagnostic investigations and specialist follow-up.

A structured neonatal clinical examination was performed. The assessment included collection of antenatal, intrapartum, and early postnatal medical history, evaluation of maternal health and pregnancy course, characteristics of delivery, gestational age, Apgar scores, anthropometric parameters at birth, neonatal adaptation, feeding, and early neonatal morbidity [8]. The maternal history included information regarding previous pregnancies and deliveries, maternal chronic diseases, acute illnesses during pregnancy, medication exposure, and hereditary history. The neonatal history included gestational age, mode of delivery, indications for cesarean section, condition at birth, Apgar scores, birth weight, body length, head circumference, chest circumference, early neonatal adaptation, feeding pattern, and physiological neonatal conditions. A

comprehensive physical examination was performed with particular attention to the morphology of the scalp lesions and the presence of associated cutaneous abnormalities. The lesions were evaluated according to their number, location, shape, size, surface characteristics, content, surrounding skin, and relationship to adjacent hair growth. The presence of the hair-collar sign was specifically documented. The general condition of the infant was assessed according to neurological status, motor activity, quality of crying, posture, skin color, hydration, respiratory and cardiovascular parameters, abdominal examination, urine output, stool characteristics, and neonatal reflexes.

Laboratory and instrumental investigations. Laboratory evaluation included a complete blood count, assessment of coagulation parameters, biochemical blood analysis, determination of serum electrolytes, bilirubin fractions, glucose, liver enzymes, renal function parameters, C-reactive protein, blood group and Rh status, and urinalysis. Instrumental assessment included abdominal ultrasonography and neurosonography. Neurosonography was performed to evaluate the structural characteristics of the brain, ventricular system, interhemispheric fissure, subarachnoid spaces, periventricular regions, and choroid plexuses, with particular attention to findings suggestive of intracranial developmental abnormalities. The diagnostic strategy was based on the clinical characteristics of the lesion and the potential association between congenital midline scalp abnormalities and underlying cranial developmental defects. The decision to perform neuroimaging was therefore considered an integral component of the diagnostic assessment. The infant underwent clinical follow-up after discharge. The assessment at 3 months included evaluation of general health, neuropsychological development, weight gain, feeding, and local evolution of the scalp lesions. A subsequent assessment was performed at 6 months of age, with particular attention to the morphology of the residual lesions, epithelialization, scar formation, and the presence or absence of neurological or developmental abnormalities. The clinical case was evaluated in accordance with the principles of biomedical ethics and the requirements for research involving human subjects. According to the decision of the Bioethics Commission of Poltava State Medical University No. 217 dated 06 December 2023, the materials of the study complied with the relevant ethical principles, including the principles of the Declaration of Helsinki and applicable Ukrainian legislation. Written informed consent for clinical examination and publication of the clinical information was obtained from the child's parents.

Results of the study. Perinatal and neonatal history. A female newborn was admitted to the early childhood department on the fourth day of life because of two congenital bullous lesions localized in

the parietal region of the scalp. According to the parents, the lesions had been present since birth. The pregnancy was the mother's second pregnancy and the delivery was her second. The mother was 24 years old and reported no chronic diseases or significant hereditary disorders. Her first pregnancy had resulted in cesarean delivery, and the first child was reported to be healthy. The current pregnancy was complicated by grade I anemia, which was treated. At 12 and 24 weeks of gestation, the mother experienced respiratory illnesses clinically manifested by rhinopharyngitis. She reported no medication use during these episodes. No significant hereditary predisposition was identified. The infant was delivered at 38 weeks of gestation by cesarean section because of a uterine scar. The condition of the newborn at birth was satisfactory. The Apgar score was 8 points at 1 minute and 9 points at 5 minutes, indicating satisfactory neonatal adaptation immediately after delivery. Birth weight was 3300 g, body length was 51 cm, head circumference was 35 cm, and chest circumference was 34 cm. The early neonatal period was characterized by physiological jaundice beginning on the third day of life and physiological breast enlargement. The infant was exclusively breastfed on demand. No clinically significant respiratory, cardiovascular, neurological, or gastrointestinal complications were documented during the early neonatal period.

Clinical examination at hospitalization. At admission, the general condition of the infant was relatively satisfactory. The newborn responded to examination with increased motor activity, and the cry was loud. The posture was predominantly flexor. The skin was pink with a mild subicteric coloration. The most prominent clinical finding was represented by two oval-shaped bullous lesions in the parietal region of the scalp (Fig. 1).



Fig. 1. ACC lesions in newborn.

The lesions measured approximately 3 cm and 2 cm in diameter, respectively, and were filled with transparent, straw-colored fluid. The surrounding skin appeared clinically unchanged, without evident

inflammatory infiltration, hyperemia, or edema. An additional characteristic cutaneous finding was identified along the scalp: a strand of long, dark hair located to the right of the sagittal suture and adjacent to the area of the congenital lesions. This finding corresponded to the clinically recognized hair-collar sign and was considered an important diagnostic feature in the context of the congenital scalp lesions. The mucous membranes were moist and clean. Peripheral lymph nodes were not enlarged. Subcutaneous tissue was satisfactorily developed. The anterior fontanelle measured 1.5×1.5 cm. Neonatal reflexes were preserved, symmetrical, and lively, while muscle tone remained predominantly flexor. Respiratory examination revealed symmetrical air entry throughout both lungs without pathological additional sounds. Respiratory rate was 42 breaths per minute. Cardiac examination demonstrated rhythmic and loud heart sounds with a heart rate of 144 beats per minute. The abdomen was soft and accessible to deep palpation, with active intestinal peristalsis. The liver edge was palpable approximately 1 cm below the costal margin, while the spleen was not enlarged. The umbilical cord remnant was dry, mummified, and dense, and the skin surrounding the umbilical ring showed no signs of inflammation, edema, or hyperemia. Urine output was sufficient, and stools were yellow and appropriately digested. Thus, apart from the congenital scalp lesions and mild physiological neonatal jaundice, no clinically significant abnormalities were identified on systemic examination.

Laboratory findings. The complete blood count demonstrated hemoglobin of 124 g/L, erythrocyte count of $3.4 \times 10^{12}/L$, hematocrit of 34.4 %, platelet count of $330 \times 10^9/L$, and leukocyte count of $9.6 \times 10^9/L$. The erythrocyte sedimentation rate was 8 mm/h. The differential leukocyte count included 1 % band neutrophils, 35 % segmented neutrophils, 4 % eosinophils, 52 % lymphocytes, and 8 % monocytes. Coagulation-related assessment showed a bleeding duration of 1 minute, with clotting beginning at 2 minutes and ending at 3 minutes. These findings did not indicate a clinically apparent coagulation abnormality. Biochemical testing revealed total protein of 61 g/L, urea 5.1 mmol/L, creatinine 42 $\mu\text{mol}/L$, direct bilirubin 30.1 $\mu\text{mol}/L$, and indirect bilirubin 92.1 $\mu\text{mol}/L$. Serum potassium was 4.9 mmol/L, sodium 145 mmol/L, and chloride 108 mmol/L. Alanine aminotransferase was 25 IU/L and aspartate aminotransferase 43 IU/L. C-reactive protein was negative, while blood glucose was 3.4 mmol/L. The blood group was B(III), Rh-positive. Urinalysis did not demonstrate pathological abnormalities. The laboratory profile was generally consistent with a clinically stable newborn. The bilirubin pattern corresponded to the physiological neonatal jaundice documented during the early neonatal period, while the absence of an inflammatory response and the preserved blood count did not suggest systemic infection. *Neuroimaging.*

Abdominal ultrasonography did not demonstrate structural abnormalities. Neurosonography showed symmetrical positioning of the cerebral structures. The cerebral sulci and gyri were clearly visualized. The interhemispheric fissure was not widened and measured up to 2 mm. The subarachnoid space was also not enlarged, measuring up to 2 mm. The anterior horns of the lateral ventricles measured 2 mm, while the ventricular bodies measured 3 mm. The third and fourth ventricles measured 3 mm each, with the fourth ventricle maintaining a regular triangular configuration. The cavity of the transparent septum was not enlarged and measured approximately 2 mm. The choroid plexuses had homogeneous echogenicity and smooth, clearly defined contours without evidence of thrombosis or subependymal cysts. The periventricular echogenicity was within the expected range, with no focal signs suggestive of ischemia or leukomalacia. Importantly, no structural abnormalities of the brain were identified at the time of examination.

Clinical diagnosis and early outcome. Based on the characteristic congenital bullous lesions, their localization, the clinical evolution, and the associated hair-collar sign, a diagnosis of congenital bullous aplasia cutis congenita was established. The infant remained clinically stable and was discharged on the seventh day of life in satisfactory condition. No acute complications such as bleeding, secondary infection, neurological deterioration, or hemodynamic instability were observed during hospitalization. Since the child was born by cesarean section, the destruction of the bullae on the head did not occur, so observation tactics were chosen.

Follow-up at 3 months. At 3 months of age, the child's general condition remained satisfactory. Neuropsychological development was appropriate for age, and adequate weight gain was documented. The infant continued to receive unrestricted breastfeeding. The local evolution of the scalp lesions demonstrated a gradual transition from the initial bullous morphology toward scar formation (Fig. 2).



Fig. 2. 3-month-old patient. ACC bullous formation has decreased in size.

One of the lesions had become less tense, with a significant reduction in its volume. A white scar had begun to develop centrally. This evolution was consistent with the previously described natural course of bullous/membranous ACC, in which a congenital cystic or bullous lesion progressively involutes and is replaced by an atrophic scar covered by thin epithelium.

Follow-up at 6 months. At 6 months of age, both congenital lesions had undergone further involution. At the sites of the original bullae, atrophic flat scars covered with thin epidermis had developed (Fig. 3).



Fig. 3. 6-month-old child. ACC bullae have disappeared.

No evidence of active inflammation, ulceration, bleeding, or secondary infection was reported. The child continued to demonstrate satisfactory general health and age-appropriate neuropsychological development. No neurological complications or clinical manifestations suggesting an underlying major structural brain abnormality were observed during follow-up.

Discussion. The present case illustrates a rare bullous phenotype of ACC in a clinically stable newborn. The combination of two congenital parietal bullae, their subsequent spontaneous involution and transformation into atrophic scars, and the presence of a localized hair-collar sign represents a characteristic clinical pattern of this condition. ACC is a heterogeneous congenital disorder that most commonly affects the scalp and may range from superficial skin defects to lesions involving deeper tissues, including the skull and dura mater. Although the underlying pathogenesis remains incompletely understood, several mechanisms have been proposed, including vascular disruption during embryogenesis, genetic predisposition, intrauterine infections, teratogenic exposure, and disturbances of tissue development. In individual patients, however, a specific etiological factor frequently cannot be identified. The bullous form of ACC may create diagnostic difficulties because congenital fluid-filled

lesions can initially resemble other vesicobullous or cystic disorders. In the present case, the lesions were already evident at birth, were surrounded by clinically unchanged skin, and were not associated with signs of inflammation or systemic infection. Their gradual reduction in size and tension followed by scar formation provided additional evidence supporting the diagnosis of bullous ACC. Of particular clinical importance was the presence of a hair-collar sign adjacent to the lesions. This finding is considered a cutaneous marker that may indicate an underlying cranial dysraphic or neurovascular abnormality. Therefore, the identification of a congenital scalp lesion accompanied by abnormal hair growth should prompt careful neurological examination and consideration of neuroimaging. In our patient, neurosonography demonstrated no structural abnormalities of the brain or ventricular system, and no neurological abnormalities were identified clinically during follow-up. The differential diagnosis of congenital scalp bullae should include epidermolysis bullosa, congenital infections, traumatic lesions, vascular or lymphatic malformations, encephalocele, and other developmental anomalies. The congenital onset, characteristic localization, absence of inflammatory changes, spontaneous involution, and subsequent formation of atrophic scars favored the diagnosis of ACC in this patient. Importantly, invasive manipulation of congenital midline scalp lesions should be avoided until possible communication with intracranial structures has been excluded. The management strategy in the present case was conservative. This approach was considered appropriate because the lesions were relatively small, the infant was clinically stable, no signs of infection or bleeding were present, and neurosonography did not reveal structural abnormalities. The favorable evolution observed at 3 and 6 months, with spontaneous epithelialization and formation of flat atrophic scars, supports the effectiveness of careful observation in selected patients with superficial ACC [1, 15].

The prognosis of ACC is primarily determined by the size and depth of the lesion and the presence of associated congenital abnormalities [3]. In the present case, the absence of extracutaneous abnormalities, neurological impairment, and evidence of deep structural involvement was associated with a favorable clinical course. Nevertheless, longer-term follow-up may be useful to assess scar maturation, localized alopecia, and late manifestations of associated developmental abnormalities. The main limitation of this report is its single-case design and relatively short follow-up period. In addition, neuroimaging was limited to neurosonography; therefore, subtle cranial or cerebrovascular abnormalities that may not be detectable by this method cannot be completely excluded. Despite these limitations, the case

emphasizes the importance of recognizing ACC and, particularly, the hair-collar sign in the neonatal period. Early clinical identification followed by

appropriate imaging and individualized management may help prevent complications and ensure favorable outcomes [5].

Conclusion

Aplasia cutis congenita is an uncommon and clinically heterogeneous congenital disorder that requires careful evaluation of both the cutaneous lesion and the possibility of associated extracutaneous abnormalities. The presented case demonstrates a rare bullous form of ACC characterized by two congenital parietal lesions, a hair-collar sign, absence of systemic complications, and subsequent spontaneous transformation of the bullae into atrophic scars. The presence of a hair-collar sign in a newborn with a congenital scalp lesion should be regarded as an important clinical marker warranting evaluation for possible underlying cranial or cerebrovascular abnormalities. Neuroimaging in the present case did not reveal structural abnormalities, and the infant demonstrated normal clinical and neuropsychological development during the available follow-up period. For small and clinically uncomplicated lesions without evidence of deep tissue involvement, conservative management with careful observation may be an appropriate therapeutic strategy. However, the management of ACC should be individualized according to lesion morphology, localization, size, depth, associated abnormalities, and the risk of intracranial or vascular involvement. The case emphasizes the importance of early recognition of ACC by neonatologists, pediatricians, and dermatologists. A systematic clinical assessment combined with appropriate imaging and multidisciplinary consultation when indicated may facilitate accurate diagnosis, prevent potentially hazardous interventions, and improve the overall management and prognosis of affected children.

References

1. Begum MK, Vijayashree J, Bathina A, Gullipalli P. A Case Series of Aplasia Cutis Congenita and Its Management. *Cureus*. 2025;17(3):e80135. doi:10.7759/cureus.80135.
2. Belkhou A, François C, Bennis Y, Duquennoy-Martinot V, Guerreschi P. Aplasia cutis congenita: update and management. *Ann Chir Plast Esthet*. 2016;61(5):450-461. doi:10.1016/j.anplas.2016.07.003.
3. Bessis D, Bigorre M, Malissen N, Captier G, Chiaverini C, Abasq C, et al. The scalp hair collar and tuft signs: a retrospective multicenter study of 78 patients with a systematic review of the literature. *J Am Acad Dermatol*. 2017;76(3):478-487. doi:10.1016/j.jaad.2016.08.046.
4. Bouali S, Houimli Charfeddine S, Ghedira K, Mechergui H, Abderrahmen K, Kallel J. Large aplasia cutis congenita of the vertex: conservative management. *Childs Nerv Syst*. 2024;40(2):285-292. doi:10.1007/s00381-023-06190-x.
5. Bousquet J, Agache I, Berger U, Bergmann KC, Besancenot JP, Bousquet PJ, et al. Differences in Reporting the Ragweed Pollen Season Using Google Trends across 15 Countries. *Int Arch Allergy Immunol*. 2018;176(3-4):181-188. <https://doi.org/10.1159/000488391>.
6. Coi A, Barisic I, Garne E, Addor MC, Aizpurua Atxega A, Ballardini E et al. Epidemiology of aplasia cutis congenita: a population-based study in Europe. *J Eur Acad Dermatol Venereol*. 2023;37(3):581-589. doi:10.1111/jdv.18690.
7. Humphrey SR, Hu X, Adamson K, Schaus A, Jensen JN, Drolet B. A practical approach to the evaluation and treatment of an infant with aplasia cutis congenita. *J Perinatol*. 2018;38(2):110-117. doi:10.1038/jp.2017.142.
8. Lysanets YV, Bieliaieva OM. The use of Latin terminology in medical case reports: quantitative, structural, and thematic analysis. *J Med Case Rep*. 2018;12(1):45. <http://dx.doi.org/10.1186/s13256-018-1562-x>.
9. Marneros AG. Aplasia cutis congenita pathomechanisms reveal key regulators of skin and skin appendage morphogenesis. *J Invest Dermatol*. 2024;144(11):2399-2405. doi:10.1016/j.jid.2024.05.014.
10. Ngeow A, Hui C, Chua AWC, Koh M. Challenges in the management of extensive aplasia cutis congenita. *BMJ Case Rep*. 2022;15(2):e246627. doi:10.1136/bcr-2021-24662.
11. Neelam R, Nakamura M, Tejasvi T. Dermoscopy of aplasia cutis congenita: a case report and review of the literature. *Dermatol Pract Concept*. 2021;11(1):e2021154. doi:10.5826/dpc.1101a154.
12. Patel DP, Castelo-Soccio L, Yan AC. Aplasia cutis congenita: evaluation of signs suggesting extracutaneous involvement. *Pediatr Dermatol*. 2018;35(1):e59-e61. doi:10.1111/pde.13340.
13. Sathishkumar D, Ogboli M, Moss C. Classification of aplasia cutis congenita: a 25-year review of cases presenting to a tertiary paediatric dermatology department. *Clin Exp Dermatol*. 2020;45(8):994-1002. doi:10.1111/ced.14331.
14. Schierz IAM, Giuffrè M, Del Vecchio A, Antona V, Corsello G, Piro E. Recognizable neonatal clinical features of aplasia cutis congenita. *Ital J Pediatr*. 2020;46:103. doi:10.1186/s13052-020-0789-5.
15. Song Y, Yang R, Shi Z, Yan J, Hou S, Li X, Zhao X. Large skin defect in Type V aplasia cutis congenita treated with conservative treatment: a case report. *BMC Pediatr*. 2024;24:314. doi:10.1186/s12887-024-04777-0.
16. Yang MY, Ha DL, Kim HS, Ko HC, Kim BS, Kim MB. Aplasia cutis congenita in Korea: single center experience and literature review. *Pediatr Int*. 2020;62(7):804-809. doi:10.1111/ped.14192.
17. Zhang L, Zhang Z, Jiang HJ, Pu YJ, Shu H, Li YD. Dermoscopic characteristics of membranous aplasia cutis congenita: Report of 56 cases. *Skin Res Technol*. 2022;28(6):792-795. doi:10.1111/srt.13200

Conflict of interest. The authors have no conflicts of interest to declare.

ORCID: Tsvirenko S.M. <https://orcid.org/0009-0005-8062-057X>, Pokhylko V.I. <https://orcid.org/0000-0002-1848-0490>, Cherniavska Y.I. <https://orcid.org/0000-0002-4522-7538>, Soloviova H.O. <https://orcid.org/0000-0002-0779-0610>, Zhuk L.A. <https://orcid.org/0000-0002-5321-4630>.

Article received: 10.09.2025