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Diagnostic and Therapeutic Awareness of Hereditary Angioedema in Pediatrics: A Survey of Pediatricians Across Türkiye

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ABSTRACT

Objective: Hereditary angioedema (HAE) is a rare disorder characterized by C1 esterase inhibitor (C1-INH) deficiency or dysfunction, leading to recurrent and potentially life-threatening edema attacks. Despite advances in diagnostic tools and effective therapies, awareness among pediatricians remains limited, contributing to delayed diagnosis and suboptimal management.

Materials and Methods: A cross-sectional, questionnaire-based survey was administered to 460 pediatricians between May and August 2025. The survey consisted of five domains: demographics, educational exposure, clinical experience, knowledge assessment, and clinical practice scenarios. Data were analyzed using descriptive statistics, chi-square tests, and logistic regression to identify predictors of accurate diagnostic and therapeutic decision-making.

Results: Participants had a mean age of 38.8 ± 10.3 years, and 64.6% were female. Although 64.3% reported receiving HAE-related education, only 26.1% played a role in diagnosis and 26.3% in the treatment of HAE patients. Knowledge gaps were prominent: 51.7% recognized the absence of urticaria in acute HAE attacks, 45.0% identified bradykinin as the primary mediator, and 30.2% correctly identified autosomal dominant inheritance. For diagnostics, 44.3% selected C4 as the screening test and 67.2% selected C1-INH levels for confirmation. Only 40.7% chose icatibant as first-line therapy for acute attacks, while 43.3% incorrectly preferred antihistamines or corticosteroids. Receiving HAE-related education significantly predicted correct management responses (acute: OR 2.5; preoperative: OR 3.8; both $p<0.001$).

Conclusion: This survey demonstrates substantial gaps in pediatricians' knowledge and management of HAE. Formal education and prior clinical experience significantly improved diagnostic accuracy and treatment choices. Structured training programs and guideline dissemination are urgently needed to reduce diagnostic delays and improve outcomes for children with HAE.

Keywords: Awareness, diagnosis, hereditary angioedema, pediatricians, therapeutics

INTRODUCTION

Hereditary angioedema (HAE) is a rare disorder characterized by recurrent subcutaneous and submucosal edema (1,2). It results from a hereditary deficiency or dysfunction of the C1 esterase inhibitor (C1-INH), which is a key endogenous regulator of the complement and kallikrein-kinin systems (3). Insufficient C1-INH activity leads to uncontrolled bradykinin generation following contact activation; the resulting excess bradykinin increases vascular permeability, ultimately resulting in tissue edema (3,4).

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Epidemiologically, HAE is extremely rare, with an estimated prevalence of approximately 1 in 50,000 in most populations (3,5). Reported prevalence rates range from 1 in 10,000 to 1 in 150,000 across different cohorts; however, there is broad consensus that this disease is very uncommon worldwide (3). Clinically, HAE presents with sudden, painful angioedema attacks affecting the extremities, face, abdomen, and upper airway; laryngeal involvement may lead to asphyxiation and is potentially life-threatening (2, 6). Symptom onset is variable; however, the disease typically manifests during childhood or adolescence (7). Although the median age at the first attack is approximately 11 years; the reported age range is wide (8). Hormonal changes during puberty contribute to an increased frequency and severity of attacks (9).

The diagnosis of HAE is frequently delayed and challenging. Despite symptom onset often occurring in childhood, many patients experience substantial diagnostic delays, and a definitive diagnosis is often not established until adulthood (2). This delay is attributed to the rarity of the disease, overlap of clinical manifestations with more common conditions, and limited awareness among healthcare providers of the disease. In particular, HAE remains insufficiently recognized by pediatricians (2). Early diagnosis and effective treatment are crucial in pediatric patients. Prompt administration of disease-specific therapies at the onset of attacks is essential to prevent fatal outcomes, especially in cases of laryngeal edema (1,10).

The present study aimed to assess the awareness, diagnostic approaches, and treatment practices regarding hereditary angioedema among 460 pediatricians across Türkiye through a structured survey.

MATERIALS and METHODS

Study Design and Participants

This study was designed as a cross-sectional, questionnaire-based survey. A total of 460 pediatricians practicing in different regions of Türkiye were included. Participation was voluntary, and informed consent was obtained from all respondents at the beginning of the survey. The study protocol was reviewed and approved by the Institutional Ethics Committee (2025/0217). The study was conducted in accordance with the Declaration of Helsinki.

Questionnaire

The survey instrument was developed by the research team based on current international guidelines and relevant literature on HAE (9). The questionnaire content was reviewed by experienced specialists in pediatric allergy and immunology. In addition, a small pilot assessment was conducted to evaluate the clarity and comprehensibility of the questions asked. The questionnaire consisted of sections addressing (1) demographics and professional background (age, gender, years of practice, hospital type, subspecialty training), (2) educational exposure to HAE, (3) clinical experience (previous diagnosis or treatment of HAE patients), (4) knowledge assessment (pathophysiology, triggers, mediators, laboratory tests, diagnostic methods, classification), and (5) clinical practice scenarios (preferred acute treatment, preoperative prophylaxis, emergency management) (Supplementary Material 1). The educational exposure question allowed for multiple responses, as participants could select more than one source of HAE-related education. Therefore, educational categories were analyzed as multiple-response variables, and percentages were calculated using the total sample size as a denominator. Icatibant was defined as the correct answer for acute attacks; C1-INH was not included among the response options to avoid multiple correct answers and to specifically assess awareness of bradykinin-targeted therapy.

The final questionnaire was distributed online using Google Forms between May and August of 2025. The survey link was disseminated via professional social media platforms used by pediatricians across Türkiye to achieve a broad geographic reach. To minimize duplicate submissions, the Google Forms setting that restricted responses to one submission per user was applied. Responses were collected anonymously and exported to Microsoft Excel for data cleaning and analysis. Due to the open-access nature of the survey, the number of pediatricians who received the survey link, opened the questionnaire, or partially completed it could not be determined.

Statistical Analysis

All data were analyzed using the Statistical Package for the Social Sciences (SPSS) version 26.0 (IBM Corp., Armonk, NY, USA). Descriptive statistics were presented as means and standard deviations (SD) for continuous variables and frequencies and percentages for categorical variables. The normality of the data distribution was assessed using the Kolmogorov-Smirnov test. For comparisons, parametric (Student's t-test, ANOVA) and non-parametric tests (Mann-Whitney U and Kruskal-Wallis) were used as appropriate. Chi-square tests were used for categorical

variables. Multivariable logistic regression analyses were performed to identify independent factors associated with correct responses to acute treatment and perioperative prophylaxis clinical vignettes. Separate models were constructed for each of the outcomes. Variables were specified a priori based on clinical relevance and previous literature, including age (continuous), professional status (specialist/subspecialist vs. resident), receipt of HAE-related education (yes/no), previous diagnosis of a patient with HAE (yes/no), and previous experience in treating an acute HAE attack (yes/no). For categorical variables, the absence of a characteristic (e.g., no prior HAE-related education) was used as the reference category. Odds ratios (ORs) with 95% confidence intervals (CIs) were calculated for the analysis. A two-tailed p-value of <0.05 was considered statistically significant.

RESULTS

Participant Characteristics

A total of 460 pediatricians across Türkiye completed the survey. The mean age of the participants was 38.8 (SD±10.3) years, and 297 (64.6%) were female (Table I). The mean duration since medical school graduation was 20.7 (SD±13.4) years. Regarding professional status, the majority (69.3%) were pediatric specialists, followed by pediatric residents (23.4%), subspecialists (3.7%), and subspecialty residents (3.6%) excluding Pediatric Allergy and Immunology (Table I). More than half of the participants (52.6%) were employed in tertiary university or public hospitals, while 20.4% worked in secondary-level public hospitals, 19.6% in private hospitals, and 7.4% in private practices (Table I).

Table I: Demographic and professional characteristics of the participants.

Age (year) [mean (SD±)]	38.8 (±10.3)
Gender (F/M) [n (%)]	297 (64.6) / 163 (35.4)
Years since medical school graduation (years) [mean (SD±)]	20.7 (±13.4)
Professional status [n (%)]	
Specialist	319 (69.3)
Resident	108 (23.4)
Subspecialist except for PAI	17 (3.7)
Subspecialist resident except for PAI	16 (3.6)
Hospital [n (%)]	
Tertiary university/public hospital	242 (52.6)
Secondary public hospital	94 (20.4)
Private hospital	90 (19.6)
Private practice center	34 (7.4)

F: Female**, M:** Male, **PAI:** Pediatric allergy and immunology
SD: Standard deviation

Table II: Educational exposure (multiple-response) and clinical experience regarding HAE among pediatricians.

	n (%)
Attended any HAE-related lecture/seminar	296 (64.3)
Medical school course	74 (16.1)
Residency training	77 (16.7)
Both medical school + residency	52 (11.3)
Scientific congress/meeting (alone or combined)	84 (18.3)
None	164 (35.7)
Previously diagnosed an HAE patient	
+	120 (26.1)
-	340 (73.9)
Previously treated an acute HAE attack	
+	121 (26.3)
-	339 (73.7)

HAE: Hereditary angioedema
*Educational exposure items allowed multiple responses; therefore, categories are not mutually exclusive and percentages may exceed 100%. Percentages are calculated using the total number of participants

Educational Exposure and Clinical Experience

Of the 460 participants, 296 (64.3%) reported having received some form of HAE education during their careers. As multiple responses were permitted, the most frequently reported sources of education were scientific congresses (18.3%), followed by residency training (16.7%) and medical school lectures (16.1%) (Table II). Notably, 164 respondents (35.7%) stated that they had never received any structured education on the disease.

In terms of clinical exposure, only 120 pediatricians (26.1%) reported having previously diagnosed a patient with HAE, and a similar proportion (26.3%) had managed an acute HAE attack (Table II). Thus, the majority of participants had neither diagnosed nor treated patients with this rare condition.

Knowledge about Hereditary Angioedema

Overall, the pediatricians' knowledge of HAE was suboptimal. Only 238 participants (51.7%) correctly recognized that urticaria is not a feature of acute HAE attacks, whereas 319 participants (69.3%) incorrectly classified established HAE triggers as non-triggering factors (Table

III). Less than half of the respondents (45.0%) identified bradykinin as the primary mediator of HAE.

Regarding laboratory investigations, 204 pediatricians (44.3%) correctly chose serum C4 level as the appropriate screening test, and 309 (67.2%) selected C1-INH level and function for the diagnosis (Table III). Similarly, 239 participants (52.0%) correctly indicated that HAE classification is based on C1-INH level and function.

Knowledge about inheritance patterns was particularly low; only 139 respondents (30.2%) correctly recognized autosomal dominant transmission as the most common pattern, while 145 (31.5%) answered incorrectly as autosomal recessive, and 138 (30.0%) reported uncertainty. Encouragingly, 333 pediatricians (72.4%) correctly acknowledged that HAE can occur in the absence of a positive family history (Table III).

Table III: Knowledge of pediatricians regarding HAE.

Knowledge	Answer	n (%)
Symptom not observed in acute HAE	Urticaria	238 (51.7)
Non-trigger of HAE attacks	Salicylate-containing foods	141 (30.7)
Main mediator responsible	Bradykinin	207 (45.0)
Screening test	Serum C4 level	204 (44.3)
Diagnostic test	C1-INH level and function	309 (67.2)
Classification marker	C1-INH level and function	239 (52.0)
Most common inheritance pattern	AD	139 (30.2)
HAE possible without family history	Yes	333 (72.4)

AD: Autosomal dominant, **C1-INH:** C1 esterase inhibitor, **HAE:** Hereditary angioedema

Table IV: Clinical practice scenarios regarding HAE.

Clinical practice scenarios	n (%)
First-line treatment in acute pediatric HAE attack	187 (40.7)
Bradykinin receptor antagonist *(correct)*	199 (43.3)
H1-antihistamines + corticosteroids	36 (7.8)
Anti-IgE (omalizumab)	31 (6.7)
Anti-C5 (eculizumab)	7 (1.5)
NSAID (ibuprofen)	
POP in a child with HAE undergoing emergency appendectomy	
C1 esterase inhibitor concentrate *(correct)*	260 (56.5)
H1-antihistamines + corticosteroids	173 (37.6)
Beta-lactam antibiotic	14 (3.0)
Platelet transfusion	8 (1.7)
Factor VIII concentrate	5 (1.1)

HAE: Hereditary angioedema, **IgE:** Immunoglobulin E, **NSAID:** Non-steroidal anti-inflammatory drug, **POP:** Preoperative prophylaxis

*Correct responses were defined based on the WAO/EAACI 2021 guideline for hereditary angioedema (9). Icatibant was selected as the correct option for acute attacks. C1-INH was not included to preserve a single-best-answer design.

Clinical Practice Scenarios

In response to the clinical vignette regarding acute management, only 187 pediatricians (40.7%) correctly identified bradykinin receptor antagonists (e.g., icatibant) as the first-line treatment for an acute HAE attack in children (Table IV). The majority of respondents instead opted for treatments known to be ineffective for HAE attacks, including H1-antihistamines and corticosteroids (43.3%), anti-IgE monoclonal antibodies (7.8%), anti-C5 antibodies (6.7%), and nonsteroidal anti-inflammatory drugs (1.5%) (Table IV).

In the preoperative scenario of a child with HAE scheduled for emergency appendectomy, 260 (56.5 %) participants correctly recommended prophylaxis with C1 esterase inhibitor concentrate. However, a substantial proportion (37.6%) incorrectly chose unrelated treatments like H1-antihistamines and corticosteroids, antibiotics (3.0%), platelet transfusion (1.7%), or factor VIII concentrate (1.1%) (Table IV).

Factors Influencing Correct Responses

Multivariable logistic regression analyses were performed to identify the factors associated with correct responses in the two key clinical scenarios (Table V). For the acute treatment question, receiving HAE-related education was strongly associated with providing the correct answers (OR: 2.52, 95% CI: 1.46-4.01, p<0.001, Table V). Similarly, having previously diagnosed a patient with HAE was a significant predictor (OR = 1.99, 95% CI: 1.09-3.63, p=0.025, Table V). Neither age, specialist/subspecialist status, nor prior experience in treating an HAE attack was a significant factors.

In the preoperative prophylaxis scenario, both receiving HAE-related education (OR: 3.84, 95% CI: 2.43-6.09, p<0.001) and having previously treated an acute HAE

Table V: Factors associated with correct responses in acute treatment and perioperative prophylaxis scenarios.

	Acute treatment (icatibant as first-line) [OR (95% CI)]	p	Perioperative prophylaxis (C1-INH concentrate) [OR (95% CI)]	p
Age (per year)	0.99 (0.97-1.02)	0.760	1.02 (0.99-1.04)	0.200
Specialist / subspecialist	0.64 (0.38-1.08)	0.090	1.04 (0.60-1.83)	0.900
Received HAE-related education	2.52 (1.46-4.01)	<0.001	3.84 (2.43-6.09)	<0.001
Previously diagnosed HAE	1.99 (1.09-3.63)	0.025	1.99 (0.99-3.98)	0.054
Previously treated HAE attack	1.16 (0.63-2.12)	0.630	3.66 (1.57-7.58)	<0.001

C1-INH: C1 esterase inhibitor, **CI:** Confidence interval **HAE:** Hereditary angioedema, **OR:** Odds-ratio

Multivariable models adjusted for age, professional status, prior HAE-related education, previous diagnosis of HAE, and prior experience in treating an acute HAE attack.

attack (OR: 3.66, 95% CI: 1.57-7.58, p<0.001) were independent predictors of correct response (Table V). A prior diagnosis of HAE showed a trend toward significance (p=0.054). Age and professional status did not significantly influence the likelihood of correct answers in this context.

DISCUSSION

This survey demonstrates substantial knowledge and practice gaps regarding HAE among Turkish pediatricians. Despite the rarity of the disease, awareness of childhood care is essential, as delayed diagnosis and inappropriate treatment can lead to preventable morbidity and mortality.

In our cohort, only 51.7% of pediatricians recognized that urticaria is not a feature of acute HAE, and less than half identified bradykinin as the primary inflammatory mediator. These findings demonstrate a limited ability to differentiate HAE from allergic angioedema. Similar results have been reported globally. A Brazilian survey showed that general pediatricians correctly answered only approximately one-quarter of HAE knowledge questions, underscoring their limited familiarity with this condition (11). Misdiagnosis contributes directly to diagnostic delay, which averages 8-11 years in most series (11,12). Our findings further confirm that Turkish pediatricians face the same challenge, as the majority could not distinguish the key pathophysiological features of HAE. Collectively, these findings suggest that without improved awareness, children remain at risk of misclassification as having allergic or idiopathic angioedema, perpetuating delays in diagnosis.

Only 26% of our respondents had ever diagnosed or treated HAE patients. This limited clinical exposure likely reflects low clinical suspicion and poor recognition of the disease in daily practice. Previous studies have indicated that undiagnosed children often undergo unnecessary interventions, including laparotomies for presumed appendicitis or obstruction (13,14). In our study, the majority of pediatricians failed to select appropriate diagnostic tests, with only 44.3% identifying serum C4 as the initial screening marker. Comparable deficiencies have been observed internationally; in Iranian children, the median delay to diagnosis remained around three years, even with clinical suspicion (15). Therefore, our results align with global data demonstrating that limited physician experience and poor knowledge of laboratory testing prolong diagnostic journeys and expose children to avoidable procedures.

Perhaps most concerning, only 40.7% of pediatricians in our survey correctly identified icatibant as the first-line therapy for acute HAE attacks, while 43.3% selected antihistamines and corticosteroids, which are ineffective in this context. This pattern mirrors other reports showing frequent reliance on anti-allergic medications despite their lack of efficacy in bradykinin-mediated angioedema (16,17). In the United States, 82% of allergist-immunologists reported that their patients had been misdiagnosed and often treated with inappropriate medications before referral (18). The concordance between our findings and the international literature reinforces the conclusion that insufficient awareness results in persistent therapeutic errors. Notably, in our regression analysis, HAE-related education was the strongest predictor of selecting the correct acute therapy. This confirms that structured training directly enhances physicians' ability to manage HAE appropriately.

In the preoperative scenario, only 56.5% of respondents recommended C1-INH concentrate for prophylaxis before appendectomy, whereas 37.6% incorrectly

chose antihistamines or corticosteroids. These results are consistent with international experiences. An Italian observational study found that the omission of short-term prophylaxis led to severe postprocedural laryngeal attacks, whereas nearly all procedures covered by C1-INH prophylaxis were uneventful (19). Our findings suggest that many pediatricians underestimate the perioperative risk, despite guideline recommendations emphasizing prophylaxis for high-risk interventions (13). As pediatric patients have smaller airways and greater vulnerability to obstruction, the under-recognition of preoperative prophylaxis represents a significant safety concern.

Only 30.2% of pediatricians in our cohort correctly identified autosomal dominant inheritance as the most common pattern, while nearly one-third chose autosomal recessive inheritance. This mirrors the gaps reported in surveys from Brazil and North America, where many physicians demonstrated a poor understanding of the genetic aspects of HAE (11,18). The lack of awareness about inheritance not only affects patient care but also impedes family screening and early interventions. Our findings highlight the urgent need to incorporate genetic counseling principles into pediatric education on HAE, in line with international guidelines recommending systematic family evaluation (20).

The consistent theme across our study and others is that education is the most effective tool for improving awareness and its management. In our analysis, prior education increased the odds of correct responses 2.5- to 3.8-fold, a finding echoed by reports demonstrating that targeted training improves physician competence in HAE (11,18). Innovative approaches, such as electronic medical record algorithms to flag possible undiagnosed patients and partnerships with patient organizations to raise awareness, have shown measurable benefits (21,22). Our data underscore the need for similar interventions in Türkiye.

The strength of our study lies in its large sample size of pediatricians across Türkiye, providing the first comprehensive evaluation of pediatricians' awareness of HAE in the country. The alignment of our results with the international literature underscores their external validity.

Nevertheless, some limitations of this study should be acknowledged. First, response bias is possible, as pediatricians with greater interest or prior exposure to immunology and hereditary angioedema may have been more likely to participate in this voluntary online survey. Second, although the questionnaire was developed based on established international guidelines, it was not formally validated, which may have affected the generalizability of the findings. Finally, the study assessed theoretical knowledge using questionnaire items and clinical vignettes, rather than actual clinical decision-making. Therefore, participants' responses may not fully reflect real-world practice, where additional contextual and patient-specific factors influence management decisions.

In conclusion, our survey demonstrates that Turkish pediatricians, like their international counterparts, show significant gaps in knowledge and practice regarding HAE. Misdiagnosis, inappropriate use of anti-allergic medications, and insufficient awareness of preoperative prophylaxis were common. However, HAE-related education and prior clinical exposure were strong predictors of correct management decisions. These findings emphasize that structured training and dissemination of updated guidelines are essential to improve pediatricians' ability to diagnose and manage HAE in children. By bridging these knowledge gaps, early diagnosis, fewer mismanaged attacks, and better outcomes for children with HAE can be achieved.

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Conflict of Interest

The authors declare no conflicts of interest, industrial links, or relevant affiliations.

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Author Contributions

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Awareness of Pediatricians Regarding Hereditary Angioedema (HAE)

Information and Consent

1. I have read the information above and consent to participate in this study.

☐ Yes, I approve and agree to participate.

Demographic Information

2. What is your age?

…….

3. What is your gender?

☐ Female
☐ Male

4. What is your current professional position?

☐ Pediatric resident
☐ Pediatric specialist
☐ Subspecialty resident except for Pediatric Allergy and Immunology
☐ Subspecialist except for Pediatric Allergy and Immunology

5. How many years have you been practicing in the field of pediatrics?

……

6. In which type of healthcare institution do you currently work?

☐ Secondary-level public hospital
☐ Tertiary-level public/university hospital
☐ Private practice
☐ Private hospital

Educational exposure to HAE

7. Have you ever attended a lecture or seminar on hereditary angioedema (HAE)? (You may select more than one option.)

☐ No, I have not attended.
☐ Yes, during medical school.
☐ Yes, during residency training.
☐ Yes, during a scientific congress or meeting.
☐ Other: _____

Clinical experience

8. Have you ever followed a patient diagnosed with hereditary angioedema?

☐ Yes
☐ No

9. Have you ever been involved in the treatment of a patient presenting with an acute attack of hereditary angioedema?

☐ Yes
☐ No

Knowledge assessment

10. Which of the following clinical findings is not observed during an acute hereditary angioedema attack?

☐ Urticaria
☐ Angioedema
☐ Colicky abdominal pain
☐ Stridor
☐ Dyspnea

11. Which of the following is not considered a trigger or precipitating factor for hereditary angioedema attacks?

☐ Mechanical trauma
☐ Upper respiratory tract infection
☐ Stress
☐ Tooth extraction
☐ Foods containing salicylates

12. Which mediator is primarily responsible for the clinical manifestations of hereditary angioedema?

☐ Adenosine
☐ Histamine
☐ Bradykinin
☐ Tryptase
☐ Not sure

13. Which of the following is used as a screening test for hereditary angioedema? Peripheral blood eosinophil percentage and count

☐ Serum total IgE level
☐ Serum rheumatoid factor (RF) level
☐ Serum complement component C3 level
☐ Serum complement component C4 level
☐ Not sure

14. Which laboratory test is used to confirm the diagnosis of hereditary angioedema?

☐ Autologous serum test
☐ Basophil activation test
☐ Serum CH50 and AH50 levels
☐ Serum C1 esterase inhibitor level and function
☐ Serum interleukin-1 (IL-1) level
☐ Not sure

15. Which parameter is used for the classification or typing of hereditary angioedema?

☐ Serum CH50 level
☐ Serum AH50 level
☐ Serum CD203 and CD63 levels
☐ Serum C1 esterase inhibitor level and function
☐ Serum interleukin-1 (IL-1) level
☐ Not sure

Clinical practice scenarios

16. In a child presenting with an acute hereditary angioedema attack, which of the following should be the first-line treatment?

☐ H1-antihistamine (e.g., Avil) and corticosteroid (e.g., Dekort)
☐ Bradykinin receptor antagonist (e.g., Icatibant)
☐ Anti-C5 antibody (e.g., Eculizumab)
☐ Anti-IgE antibody (e.g., Omalizumab)
☐ Nonsteroidal anti-inflammatory drug (e.g., Ibuprofen)

17. A child diagnosed with hereditary angioedema is scheduled for emergency surgery due to acute appendicitis. Which of the following treatments should be preferred for prophylaxis to prevent an angioedema attack? (Select one.)

☐ H1-antihistamine and corticosteroid
☐ Platelet transfusion
☐ Beta-lactam antibiotic
☐ Factor VIII concentrate
☐ C1 esterase inhibitor concentrate

18. What is the most common mode of inheritance of hereditary angioedema? (Select one.)

☐ Autosomal recessive
☐ Autosomal dominant
☐ X-linked recessive
☐ X-linked dominant
☐ Not sure

19. Do you think hereditary angioedema can occur in an individual without a family history of the disease? (Select one.)

☐ Yes, it is possible
☐ No, it is not possible
☐ Not sure

End of Questionnaire

Thank you for your participation.

Please click the 'Submit' button to complete the survey.