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Case Report

A Rare Case of Drug-Induced Allergy in Childhood: Acute Generalized Exanthematous Pustulosis (AGEP) Case Report

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Abstract

Background and Clinical Significance: Acute generalized exanthematous pustulosis (AGEP) is a rare, usually drug-induced severe cutaneous reaction characterized by the sudden appearance of numerous sterile non-follicular pustules on erythematous skin. This case highlights the diagnostic challenges of AGEP in paediatric patients. **Case presentation:** We report a case of a 13-year-old boy who developed a rapidly progressive pustular eruption predominantly involving intertriginous areas. The patient had a history of epilepsy treated with long-term antiepileptic therapy, with levetiracetam introduced two weeks before the onset of the pustular eruption and prior exposure to cefadroxil. The diagnosis was confirmed using the European Study of Severe Cutaneous Adverse Reactions (EuroSCAR) scoring system. Histopathological examination revealed subcorneal neutrophilic pustules, dermal oedema and a mixed perivascular infiltrate including eosinophils, favouring AGEP. The skin lesions improved after withdrawal of suspected medications, topical treatment, systemic corticosteroids and methotrexate, which was prescribed while pustular psoriasis remained a concern. Patch testing performed five months later was positive to cefadroxil, supporting delayed sensitization without establishing it as the cause of this episode. Levetiracetam remained a competing suspect. **Conclusions:** This case highlights the need to distinguish evidence of sensitization from certainty about drug causality.

Keywords: acute generalized exanthematous pustulosis; delayed-type hypersensitivity; drug allergy; children; case report

1. Introduction and Clinical Significance

Acute generalized exanthematous pustulosis (AGEP) is a rare, usually drug-induced severe cutaneous adverse reaction characterized by the abrupt onset of erythema with numerous small, non-follicular sterile pustules, often accompanied by fever [1]. The estimated annual incidence is approximately 1–5 cases per million people, and it occurs predominantly in adults, while paediatric cases are considerably rarer [1,2]. Although the clinical course is typically self-limited after withdrawal of the causative drug, the morphology of AGEP may overlap with other pustular dermatoses, which can make diagnosis challenging [1].

AGEP represents a delayed hypersensitivity reaction involving drug-specific T cells and neutrophil recruitment. Interleukin (IL)-8 promotes neutrophil accumulation, while IL-17 and IL-36 pathways may amplify the inflammatory response. These mechanisms help explain the formation of sterile pustules and the clinical overlap with generalized pustular psoriasis [1,2].

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The evidence base is largely derived from adults. In a multicentre United States series of 340 adults, medications were the suspected trigger in 85.6% of cases, with antimicrobial exposure particularly prominent [3]. Paediatric presentations require a broader assessment: recent reports describe both drug-associated and infection-associated episodes, sometimes in the same child [4]. A detailed chronology of new prescriptions, intermittent medications and preceding illness is therefore particularly important when several potential triggers coexist.

Diagnosis relies on the combined assessment of morphology, histopathology and clinical evolution rather than on a single laboratory finding. The European Study of Severe Cutaneous Adverse Reactions (EuroSCAR) validation score provides a structured diagnostic framework, whereas subsequent allergy investigations address a different question: identification of the responsible drug. Distinguishing diagnostic certainty from certainty about the trigger is essential in patients receiving several medications [1,5].

Case reports help improve recognition of this rare condition in children [1,6–9]. Here, we present a paediatric case of suspected AGEP treated at the Lithuanian University of Health Sciences Hospital, where only two cases have been documented over the past 30 years. This reflects institutional experience rather than a systematically ascertained incidence estimate.

2. Case Presentation

A 13-year-old boy was admitted to the Paediatric Department of the Lithuanian University of Health Sciences Hospital because of a rapidly progressive pustular skin eruption.

Since the age of 6 years, the patient had experienced recurrent inflammatory skin changes during cold seasons, mainly affecting the axillae, groins, and intergluteal area, clinically resembling intertrigo (Figure 1). These episodes were successfully managed with emollients and topical corticosteroids. There was no personal or family history of psoriasis, atopic disease, or severe drug allergy. The relevant medical history also included epilepsy, as detailed below.

At age 13, at the beginning of the cold season, the patient again developed erythematous, oedematous lesions affecting the neck folds, axillae, groin, and intergluteal region. The flexural eruption began approximately two months before the acute pustular rash and progressed despite topical treatment. During this period, a culture was obtained from the skin lesions and yielded *Staphylococcus aureus*. The patient was subsequently prescribed a 10-day course of oral cefadroxil (500 mg twice daily), initiated four weeks before the onset of the pustular eruption.

The patient had a history of epilepsy diagnosed at 1.5 years of age. Since approximately 3 years of age, he had been treated with a stable combination of antiepileptic drugs: topiramate (75 mg in the morning and 100 mg in the evening) and lamotrigine (100 mg in the morning and 125 mg in the evening). Due to recurrent seizures, levetiracetam (250 mg in the morning and 375 mg in the evening after gradual titration) was added to the treatment regimen two weeks before the onset of the pustular rash.

Because the eruption continued to spread, the patient was referred to a paediatric allergologist and hospitalized. The chronology of the reported episode and relevant medication exposures is summarized in Table 1.



Figure 1. Recurrent flexural dermatitis preceding the acute pustular episode. Erythematous patches and plaques, with weeping in some areas, involve the skin folds.

Table 1. Reported chronology and relevant medication exposures.

Period	Clinical Event or Medication	Dose and Indication
From the age of 3	Long-term treatment with topiramate and lamotrigine for epilepsy	At age 13, the patient was receiving topiramate (75/100 mg morning/evening) and lamotrigine (100/125 mg morning/evening)
From age 6 to 13	Recurrent cold-season flexural dermatitis	Emollients and topical corticosteroids; earlier episodes not established as acute generalized exanthematous pustulosis (AGEP)
Two months before acute pustular rash	Flexural erythematous, oedematous eruption	Progression despite topical treatment
Four weeks before acute pustular rash	Cefadroxil: oral 10-day course for suspected bacterial superinfection	500 mg twice daily for 10 days, prescribed after *Staphylococcus aureus* was isolated from a culture from skin lesions

Table 1. *Cont.*

Period	Clinical Event or Medication	Dose and Indication
Two weeks before acute pustular rash	Levetiracetam added for recurrent seizures	250/375 mg morning/evening at admission
Acute pustular episode and admission to the hospital		
Day 1 of hospitalization	Confluent non-follicular pustules; no mucosal involvement or fever	Cefazolin 1.5 g intravenously four times daily introduced for suspected infection after pustulation began
Day 4 of hospitalization	Progression; cefazolin and levetiracetam withdrawn	Following dermatological assessment, cefazolin and levetiracetam were discontinued as suspected causative agents
Days 6–15 of hospitalization	Lesional and perilesional chest biopsies obtained during day 6 of inpatient treatment; histology favoured AGEP and direct immunofluorescence was negative	Inpatient treatment included systemic methylprednisolone (60 mg for 2 days, 50 mg for 2 days, 40 mg for 5 days, 30 mg for 4 days, 20 mg for 1 day, 10 mg for 1 day) and topical dexamethasone; oral methotrexate 5 mg once weekly started while psoriasis remained a concern
Day 16 of hospitalization	Substantial improvement with residual erythema and desquamation	Discharge: - Methotrexate 10 mg once weekly; topiramate 100 mg twice daily; lamotrigine 100/125 mg morning/evening; - Recommendation to avoid all beta-lactam antibiotics and levetiracetam until testing
Three months after onset of the pustular rash	No skin rash; dermatology consultation	Methotrexate discontinued at the three-month follow-up because no skin lesions were observed
Five months after onset of the pustular rash	Patch testing	Cefadroxil positive at 48 and 72 h; four other drugs negative

On day 1 of hospitalization, physical examination revealed erythematous plaques with overlying numerous confluent, non-follicular pustules involving the lower neck, axillary, inguinal, and intergluteal regions. The lesions were tender and indurated. No mucosal involvement was noted (Figure 2).

The patient was afebrile, with mildly elevated inflammatory markers (C-reactive protein 20 mg/L) and no clinical evidence of mucosal or internal organ involvement. During the acute episode, the white blood cell count was 10.7×10^9/L, with neutrophils 7.5×10^9/L, lymphocytes 2.2×10^9/L, monocytes 0.9×10^9/L, eosinophils 0.0×10^9/L and basophils 0.0×10^9/L; haemoglobin was 141 g/L and platelets 285×10^9/L. This sample was obtained during systemic corticosteroid treatment. Creatinine was 57 µmol/L, alanine aminotransferase (ALT) 8 IU/L and aspartate aminotransferase (AST) 22 IU/L. Chest radiography and abdominal and urinary tract ultrasonography showed no relevant abnormalities. Suspecting intertriginous dermatitis complicated by secondary infection, treatment with emollients, topical antiseptics, systemic corticosteroids, and intravenous cefazolin (1.5 g four times daily) was started on day 1 of hospitalization, after the pustular eruption had begun. Superficial wound cultures obtained at admission, before the initiation of inpatient antibiotic therapy, showed no bacterial or fungal growth. Despite the treatment, the rash progressed over the first days of hospitalization, spreading to the upper chest, lower abdomen, arms, and thighs. The lesions consisted of violaceous, indurated erythematous

plaques with annular groups of pustules and superficial detachment on gentle pressure (a pseudo-Nikolsky sign). No fever or mucosal involvement developed (Figure 3).



Figure 2. Admission findings: erythematous, oedematous plaques with confluent non-follicular pustules, predominantly affecting the lower neck and axillary folds, with inguinal and intergluteal involvement on clinical examination.

AGEP was suspected based on the presence of sterile pustules and worsening of the rash following initiation of antimicrobial treatment. Cefazolin and levetiracetam were discontinued on day 4 of hospitalization. Generalized pustular psoriasis and immunoglobulin A (IgA) pemphigus were considered. Lesional and perilesional chest biopsies were obtained during day 6 of hospitalization. The lesional biopsy showed subcorneal neutrophilic pustules, dermal oedema and perivascular lymphocytes, neutrophils and eosinophils, favouring AGEP (Figure 4). Direct immunofluorescence of adjacent, clinically uninvolved chest skin showed no fluorescence. Serum enzyme-linked immunosorbent assay (ELISA) for 180-kDa bullous pemphigoid antigen (BP180), 230-kDa bullous pemphigoid antigen (BP230), desmogleins 1 and 3, envoplakin and type VII collagen, and indirect immunofluorescence for autoimmune blistering disease were negative. The diagnosis was confirmed using the EuroSCAR scoring system (Table 2).



Figure 3. Progression during the first days of hospitalization. Violaceous, indurated plaques involve the neck, upper chest, axillae, groin and intergluteal region. Yellowish pustules form annular groups along the advancing margins, with superficial detachment on gentle pressure (a pseudo-Nikolsky sign).

Table 2. European study of Severe Cutaneous Adverse Reactions (EuroSCAR) assessment based on available case documentation [1,7].

EuroSCAR Item	Supporting Patient Findings	Points
Pustules	Numerous confluent non-follicular pustules; typical morphology.	+2
Erythema	Erythematous, oedematous plaques; typical morphology.	+2
Distribution/pattern	Initial flexural predominance, followed by spread to trunk and limbs.	+2
Post-pustular desquamation	Documented during improvement and at discharge.	+1
Mucosal involvement	Not observed.	0
Acute onset $\leq$ 10 days	The acute pustular episode is distinct from the previous intertriginous dermatitis	0
Resolution $\leq$ 15 days	Improvement was observed 11 days after discontinuation of the suspected causative drugs (cefazolin and levetiracetam)	0
Fever $\geq$ 38 °C	Patient was afebrile.	0

Table 2. *Cont.*

EuroSCAR Item	Supporting Patient Findings	Points
Neutrophils $\geq 7.0 \times 10^9$/L	7.5×10^9/L during the acute episode, after systemic corticosteroids had been started.	+1
Histology	Subcorneal neutrophilic pustules, dermal oedema and a mixed perivascular infiltrate containing lymphocytes, neutrophils and eosinophils, favouring AGEP.	+2
Total score		10

EuroSCAR categories: $\leq$0, no acute generalized exanthematous pustulosis (AGEP); 1–4, possible; 5–7, probable; 8–12, definite.

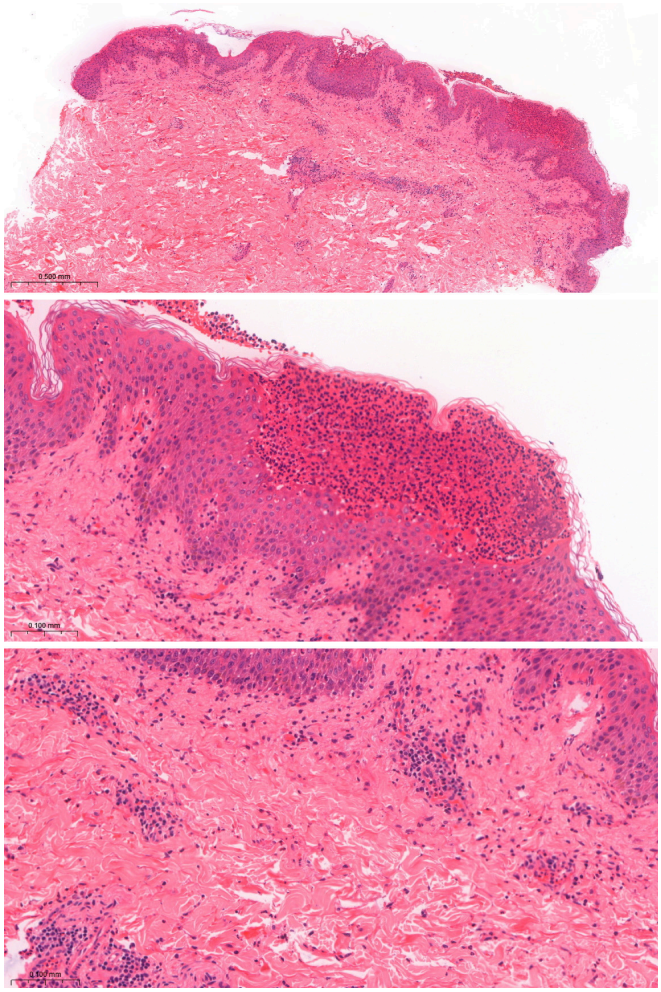


Figure 4. Histopathological features favouring acute generalized exanthematous pustulosis (AGEP) (haematoxylin and eosin). **Upper** panel: overview of the lesional biopsy. **Middle** panel: subcorneal neutrophilic pustules. **Lower** panel: dermal inflammatory infiltrate, with perivascular neutrophils and eosinophils. Scale bars: 500 µm (**upper** panel) and 100 µm (**middle** and **lower** panels).

Treatment included topical corticosteroids (dexamethasone), bilastine and emollients. Because pustular psoriasis remained a diagnostic possibility in view of the preceding intertriginous dermatitis and spreading pustular eruption, systemic corticosteroid treatment (methylprednisolone 60 mg for 2 days, 50 mg for 2 days, 40 mg for 5 days, 30 mg for 4 days, 20 mg for 1 day, 10 mg for 1 day) was prescribed. Methotrexate was subsequently introduced (initially 5 mg once weekly after dermatological assessment, increased to 10 mg once weekly at discharge and continued until the three-month follow-up, with oral folic acid 5 mg the following day). Topiramate and lamotrigine were continued under paedi-

atric neurological supervision after levetiracetam withdrawal. Seizures recurred during hospitalization, prompting an increase in topiramate to 100 mg twice daily; lamotrigine remained at 100 mg in the morning and 125 mg in the evening.

The skin condition had improved substantially by discharge on day 16 of hospitalization. Pustules began to resolve with subsequent desquamation. At discharge, erythema and induration had significantly regressed, oedema had resolved, and residual desquamation was present (Figure 5). The family was advised to avoid levetiracetam and beta-lactam antibiotics pending specialist allergy assessment. At the dermatology follow-up three months after the onset of the pustular rash, no skin lesions were observed. Methotrexate was therefore discontinued. Five months after the onset of the pustular rash, patch testing was performed with topiramate, lamotrigine, levetiracetam, cefadroxil, and cefazolin, each at a recorded concentration of 10% in petrolatum. The patch-test preparations were made in-house because commercially manufactured preparations were unavailable at the time. Cefadroxil elicited a positive reaction (+) at 48 h and a strongly positive reaction (++) at 72 h, whereas the other four drugs were negative at both readings (Table 3). The increasing cefadroxil reaction was consistent with delayed sensitization. Although the in-house patch-test preparations were prepared in accordance with published guidelines and with consideration of the solubility of each drug, the use of non-standardized preparations limits the interpretation of the results. An irritant contribution to the positive reaction cannot be excluded, and negative results do not rule out drug hypersensitivity. The findings should therefore be interpreted cautiously and in conjunction with the clinical history.



Figure 5. Clinical improvement after inpatient treatment, with reduced erythema and induration, resolution of oedema and residual desquamation.

Table 3. Patch-test results for medications discussed in the case.

Medication	Recorded Concentration	Vehicle	48 h	72 h
Cefadroxil	10%	Petrolatum	+	++
Cefazolin	10%	Petrolatum	−	−
Topiramate	10%	Petrolatum	−	−
Lamotrigine	10%	Petrolatum	−	−
Levetiracetam	10%	Petrolatum	−	−

Grades are reproduced from the testing record: +, positive; ++, strongly positive; −, negative.

3. Discussion

Acute generalized exanthematous pustulosis (AGEP) is a rare, usually drug-induced, acute pustular rash. AGEP is characterized by the acute onset of erythema predominantly in the large skinfolds with multiple pinpoint non-follicular sterile pustules, usually associated with fever, and eventually leads to peeling of the skin [1]. About 20% of patients develop mild mucosal involvement. In some severe cases, systemic involvement such as liver, kidney and pulmonary involvement, can occur [1,5]. Our patient developed a typical AGEP eruption without mucosal involvement or clinical evidence of systemic involvement. The absence of fever in our patient does not exclude AGEP. In the adult series by Creadore et al., fever of at least 38 °C was documented in 154 of 310 patients with available temperature data (49.7%) [3]. Although these figures cannot be directly extrapolated to children, they demonstrate that fever is not universal. In the present case, the pustular morphology, biopsy findings and subsequent desquamation were more informative than the absence of a febrile response.

The estimated annual incidence of AGEP is 1–5 cases per million people [1]. The incidence of AGEP in the paediatric population remains poorly defined, with published evidence consisting largely of individual case reports and small case series [2,8]. In the Paediatrics Department of the Lithuanian University of Health Sciences (LSMU) Hospital, we treat approximately 4500 inpatients and 4000 outpatients for various diseases every year. Over the past 30 years, we have had only two documented cases of AGEP, one of which is presented in this article. These two documented cases illustrate our limited institutional experience and do not provide a population-based incidence estimate.

Recently reported paediatric cases show both clinical similarities to and differences from our case. Leung et al. described an 11-year-old girl with four AGEP episodes attributed to drug and infectious precipitants over two years. As in our patient, the eruption consisted of non-follicular pustules on erythematous skin without mucosal involvement. All four episodes improved with topical corticosteroids and emollients, with desquamation by 14 days [4]. Our patient required 16 days of inpatient care and received systemic treatment while the differential diagnosis remained unresolved. His earlier intertriginous episodes were not documented as pustular, consequently, they cannot retrospectively be classified as recurrent AGEP. Furuta et al. reported a 3-year-old boy with suspected carbocysteine-associated AGEP, initially showing flexural pustules and no mucosal lesions. Unlike our patient, he subsequently developed recurrent fever, conjunctival hyperaemia and other findings leading to a diagnosis of Kawasaki disease [10]. This comparison emphasizes the need to reassess an evolving eruption when new systemic or mucosal features emerge.

Medications are the most frequent AGEP triggers, although their relative contribution differs between adult and paediatric reports [3–5]. Commonly implicated medications include beta-lactam and other antibiotics, iodinated contrast agents and corticosteroids [5,11]. AGEP typically presents within 24–48 h of ingestion of the offending drug, with a median

time of 24 h for antibiotics. However, some drugs have been shown to have a lag time of up to 10–22 days [1]. Improvement after withdrawal of cefazolin and levetiracetam to our patient supports a drug-related reaction but does not identify a single culprit, particularly because anti-inflammatory treatment was also administered. Cefazolin was introduced after the pustular eruption had begun and therefore cannot explain its initial onset, although a contribution to subsequent worsening remains possible. The positive cefadroxil patch test supports delayed sensitization, but the reported four-week interval between initiation of the 10-day cefadroxil course and the onset of pustulation is longer than the usual antibiotic-associated latency and limits causal certainty. Allergic cross-reactivity between cefadroxil and cefazolin in unlikely, but it cannot be completely ruled out. Although both are first-generation cephalosporins, their R1 side chains—the structures most relevant to allergic cross-reactivity—differ. Cross-reactivity is primarily determined by side-chain similarity, rather than cephalosporin generation or route of administration [12]. However, R1 is not the only possible recognition site, so comparing molecular structures cannot guarantee tolerance. Levetiracetam remains another temporally plausible suspect.

In children, both drug-related and infectious triggers warrant assessment. A systematic review of 47 paediatric cases found medications to be the most common trigger, with a greater contribution from infections than in adult reports [13]. A preceding positive skin culture may indicate colonization or superinfection rather than an infectious cause of AGEP. Negative inpatient cultures also do not exclude untested viral triggers [13].

The morphology of AGEP overlaps with other pustular dermatoses. Therefore, differential diagnosis is very important [5]. In this patient, the acute pustular eruption superimposed on preceding flexural dermatitis, flexural predominance, post-pustular desquamation and an eosinophil-containing dermal infiltrate favoured AGEP. Subcorneal neutrophilic pustules alone are not specific. The pathology report did not describe regular psoriasiform hyperplasia, parakeratosis or other epidermal architectural features that would allow a more definitive distinction from pustular psoriasis. The negative perilesional direct immunofluorescence reduced support for IgA pemphigus. Overall, AGEP was favoured clinically and histologically. The pre-existing dermatitis remains incompletely characterized and is not proof of psoriasis or earlier AGEP episodes.

Although patch testing in AGEP is more sensitive than in Stevens–Johnson syndrome (SJS) or toxic epidermal necrolysis (TEN), it is not fully reliable. Negative patch tests do not reliably exclude a culprit drug; sensitivity in AGEP remains incompletely defined and has been estimated at approximately 50%, with variation between medications [11]. Testing at five months is within the 6-week to 6-month interval proposed by the European consensus [1]. However, in our patient, patch testing was performed after a three-month course of methotrexate, raising the possibility that prior immunosuppressive treatment may have influenced test sensitivity and contributed to the negative results for cefazolin and levetiracetam. Although the extent of this potential effect cannot be established, the negative results should be interpreted with caution.

AGEP management centres on immediate withdrawal of suspected drugs, supportive care and topical corticosteroids. Severe or refractory cases may require systemic corticosteroids, but evidence for their benefit is limited. Other systemic agents, including cyclosporin and biologics such as secukinumab or infliximab, are not routine treatments [1,5]. Methotrexate in this case reflected the initial concern for pustular psoriasis and should not be interpreted as standard AGEP therapy. Because drug withdrawal and several treatments occurred together, their individual contributions to recovery cannot be separated.

The main practical lessons are early recognition of a sterile pustular drug eruption, careful evaluation of competing diagnoses, and clear documentation of confirmed sensitization alongside unresolved drug suspicions. This case illustrates why suspected-drug

avoidance and coordinated dermatology, allergy and neurology follow-up are especially important when seizure control requires ongoing medication.

4. Conclusions

This case highlights the diagnostic challenges of acute generalized exanthematous pustulosis in children and the importance of integrating clinical findings, histopathology and medication chronology. A positive cefadroxil patch test supported delayed sensitization but did not establish cefadroxil as the cause of this episode; levetiracetam remained a competing suspect. Clear documentation of confirmed sensitization and unresolved drug causality is essential to guide future prescribing and coordinated specialist follow-up.

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Institutional Review Board Statement: Ethical review and approval were not required for this case report under the Law on Ethics of Biomedical Research of the Republic of Lithuania (No. VIII-1679, Article 1 and Article 2(7); https://www.e-tar.lt/portal/lt/legalAct/TAR.234B15954C2F/asr, accessed on 9 October 2026), as it describes a single case managed in routine clinical practice and does not involve the testing of a biomedical hypothesis using scientific research methods. No additional investigations or interventions were performed for research purposes.

Informed Consent Statement: Written informed consent for publication was obtained from the patient's mother and legal guardian.

Data Availability Statement: The raw data supporting the conclusions of this article will be made available by the authors on request.

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

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