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Persistent Lymphadenopathy and Mesenteric Lymphadenitis After Influenza and Ebv Infection in an 8-Year-Old Child: Limits of Interpretation of Laboratory and Genetic Markers

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Case Report

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Abstract

Purpose: Persistent lymphadenopathy is among the most frequent reasons for referral of children to an immunologist, and the increasing availability of laboratory and genetic panels creates a new problem, namely over-interpretation of the results obtained.

This case report aims to examine, using a clinical case, the diagnostic approach to persistent post-infectious lymphadenopathy in a child and to discuss the limits of clinical interpretation of selected laboratory and molecular genetic markers.

Methods: A single pediatric case was reviewed in accordance with the CARE case-report reporting guideline. Clinical, ultrasound, immunological, etiological, laboratory, and molecular genetic findings were reassessed against current reference values and clinical practice guidelines.

Results: An 8-year-old boy was examined for persistent enlargement of submandibular and cervical lymph nodes (up to 32 mm on ultrasound) and mesenteric lymphadenitis, which developed after influenza and a recurrent febrile episode. Cellular and humoral immunity corresponded to age norms and primary immunodeficiency was excluded. Latent tuberculosis was excluded, and the Epstein-Barr virus load corresponded to the latent stage. The complete blood count showed an age-appropriate differential with no reactive lymphocytes or immature cells and an erythrocyte sedimentation rate of 2 mm per hour; eosinophils accounted for 5% with a calculated absolute count of approximately 0.40 × 10^9 per L, that is, without eosinophilia. An isolated increase in eosinophil cationic protein up to 102 ng per mL with a normal total immunoglobulin E and an isolated decrease in parathyroid hormone to 8.74 pg per mL with normocalcaemia were detected. Genotyping results led to diagnoses that require revision.

Conclusion: Elevated eosinophil cationic protein with normal immunoglobulin E requires mandatory exclusion of parasitic infestation and eosinophilic disorders. Isolated low parathyroid hormone with normocalcaemia requires exclusion of hypomagnesaemia first. The LCT -13910 CT genotype is not a basis for diagnosing primary hypolactasia, and vitamin D receptor gene polymorphisms cannot serve as a basis for adjusting vitamin D dosage.

WHAT IS KNOWN – WHAT IS NEW

• What is Known: Elevated eosinophil cationic protein and low parathyroid hormone are often left without clinical interpretation; single-gene results are increasingly over-interpreted.

• What is New: These findings require structured, evidence-based interpretation rather than routine diagnostic labels, elimination diets, or vitamin D dose adjustments.

INTRODUCTION

Enlargement of peripheral lymph nodes is one of the most common reasons parents seek consultation with a pediatrician, immunologist, or hematologist. In the vast majority of pediatric cases it is reactive in

nature and related to a prior infection; however, it is precisely the prolonged persistence of the process that creates diagnostic tension and prompts an expansion of the diagnostic work-up [1, 2].

Current clinical guidelines provide benchmarks for triaging such patients. Isolated lymphadenopathy smaller than 20 mm with no other alarm features that fails to regress within 4–6 weeks warrants elective referral to a specialist; nodes larger than 20 mm require early referral, within 2–3 weeks [3]. Alarm features include firm consistency, fixation of the node, its relentless enlargement, and the presence of systemic symptoms [1, 2]. Surgical evaluation with consideration of excisional biopsy is indicated when alarm features are present or when persistence exceeds 4–6 weeks [3, 4].

In parallel with the growing availability of laboratory and molecular genetic panels, a new problem has emerged: over-interpretation of the results obtained. Indicators designed for risk assessment in population studies are increasingly being used to formulate individual clinical diagnoses and to prescribe prolonged restrictive regimens. This is particularly sensitive in pediatrics, where restrictive diets carry an independent risk of nutritional deficiency.

The aim of this publication is to describe a case of persistent post-infectious lymphadenopathy in an 8-year-old child and to discuss two groups of issues: laboratory findings that were left without clinical interpretation, and genetic results whose interpretation went beyond the available evidence base.

CASE REPORT

An 8-year-old boy was examined on an outpatient basis by a pediatric immunologist; the initial consultation took place in May 2026, with a follow-up review based on laboratory monitoring results in June 2026.

Complaints and history. Persistent enlargement of submandibular and cervical lymph nodes, episodes of nocturnal cough, snoring during sleep, and recurrent nosebleeds. The frequency of acute respiratory infections was moderate, 2–4 times per year. In March 2026 the child had influenza, confirmed by rapid antigen test, treated with oseltamivir. In April 2026 a repeat episode of high-grade fever without clear catarrhal symptoms was recorded, accompanied by an increase in C-reactive protein to 39.91 mg/L. At the time of examination, C-reactive protein had fully normalized to 0.6 mg/L.

Status praesens objectivus. Height: 128 cm, Body weight: 26 kg, BMI: 16.0 kg/m² (calculated);

Peripheral lymph node groups accessible to palpation (submandibular, cervical, occipital, axillary, inguinal) are enlarged: single, up to 2.0–4.0 cm in size, consistency soft-elastic, mobility preserved (not matted to one another, to underlying tissue, or to the skin), non-tender on palpation, with unchanged overlying skin and no local hyperthermia. The liver edge is palpable at the costal margin — no more than 1–2 cm below the costal margin along the right midclavicular line, with a soft, smooth, non-tender edge. The spleen is not palpable. Hepatosplenomegaly is absent.

Instrumental investigations. Ultrasound findings showed reactive lymphadenopathy with the largest node clusters measuring up to 32 mm, In the right cervical region, there is an enlarged lymph node measuring 13 × 6 mm, with a homogeneous structure and reduced echogenicity. On the left, in the submandibular region, there is a lymph node measuring 11 × 7 mm, with a homogeneous structure. Mesenteric lymphadenitis with enlargement of the abdominal lymph nodes. Pronounced hypertrophy of the adenoid vegetations was detected. Chest radiography was performed; findings were normal.

Immunological work-up. Cellular immunity indices — total T-lymphocytes CD3+, T-helper cells CD4+, cytotoxic T-cells CD8+, B-lymphocytes CD19+, and natural killer cells CD3-CD56+ — were within age-appropriate reference values. The immunoregulatory index was 1.52. Serum immunoglobulin A, M, and G levels were balanced. Circulating immune complexes were 66 units. Primary immunodeficiency and severe secondary immunodeficiency were excluded.

Etiological work-up. Polymerase chain reaction testing of the blood did not detect DNA of cytomegalovirus, herpes simplex virus types 1 and 2, or human herpesvirus types 6 and 7. The Epstein-Barr virus load was 2.7×10^2 IU/mL, which, together with a previously documented high titer of IgG antibodies to the nuclear antigen (over 100), is consistent with a latent stage following a past infection. The QuantiFERON test was negative.

Laboratory findings. The main biochemical and immunochemical results are presented in Table 1, and the complete blood count parameters in Table 2. Indicators traditionally used to assess the risk of a lymphoproliferative process — lactate dehydrogenase, beta-2-microglobulin, and erythrocyte sedimentation rate — were within normal limits. At the same time, two findings outside the reference range were identified: elevated eosinophil cationic protein and decreased parathyroid hormone.

Table 1. Main laboratory parameters

Complete blood count. The test was performed on June 2, 2026. The white blood cell differential corresponded to age reference values: neutrophils 42.2% (band forms 4%, segmented forms 41%), lymphocytes 44.5% with an absolute count of 3.57×10^9/L, monocytes 6.8% with an absolute count of 0.55×10^9/L. No reactive (atypical) lymphocytes were detected, the immature granulocyte fraction was 0.1% against a reference value of less than 2.0%, and nucleated red blood cells were absent. The erythrocyte sedimentation rate by Westergren was 2 mm/h. Hemoglobin was 131 g/L.

Eosinophils accounted for 5%, against the laboratory's reference interval of 1–5%, and basophils for 1%, against an interval of 0–1%; that is, both parameters were at the upper limit of normal.

Table 2. Complete blood count parameters

Molecular genetic testing. Genotyping was performed for two polymorphisms of the vitamin D receptor gene — VDR rs2228570 (genotype A/G, heterozygous) and VDR rs1544410 (genotype A/A, homozygous) — as well as for the LCT − 13910 rs4988235 polymorphism (genotype CT, heterozygous). Parasitological testing was negative.

Diagnoses established. Generalized reactive persistent lymphadenopathy; nonspecific mesenteric lymphadenitis; unspecified disease of the parathyroid glands; vitamin D insufficiency; unspecified iron-deficiency anemia at the pre-latent iron deficiency stage; primary adult-type hypolactasia. The previously established working diagnosis of hypoparathyroidism was withdrawn based on the results of repeat testing, given the persistent normocalcemia. The diagnoses listed above were recorded by the treating physician at the time of the outpatient visits; several of them — vitamin D insufficiency, iron-deficiency anemia at the pre-latent stage, unspecified disease of the parathyroid glands, and primary hypolactasia — are critically re-examined in the Discussion in light of the laboratory data reported in Tables 1–2 and are not endorsed by the authors as definitive.

Treatment prescribed. An elimination diet for 6 months was recommended, excluding sweets, sweet fruits, refined baked goods, and milk, with maximal restriction of gluten; 4–5 meals per day without snacking. Medication prescribed included a combined calcium and vitamin D preparation for 2 months, a probiotic for 3 months, ursodeoxycholic acid for 3 months, zinc citrate for 1 month, and an iron preparation.

Planned further work-up. Ionized calcium, densitometry, assessment of magnesium status (repeat serum magnesium together with urinary magnesium excretion), and IgA anti-tissue transglutaminase antibodies together with total IgA to exclude celiac disease.

During post-treatment follow-up, the size of the lymph node decreased, and the level of eosinophil cationic protein did not rise after the completion of therapy. A pediatric hematologist was consulted, and acute and chronic conditions were ruled out.

Patient perspective. A 6-week diet is recommended that excludes foods containing lactose, casein, and sugary foods

DISCUSSION

Assessment of lymphadenopathy according to alarm criteria. The size of the largest lymph node cluster in the patient — 32 mm — exceeds the generally accepted threshold of 20 mm, at which early referral to a specialist within 2–3 weeks is recommended [3]. Combined with the persistent nature of the process, this formally corresponds to a situation in which the guidelines call for considering excisional biopsy [3, 4]. At the same time, among the etiological causes of lymphadenopathy for which children undergo biopsy, reactive changes remain the most common finding [4], and lymphadenopathy that persists for more than 6 months without a change in size or character and without systemic symptoms has a low probability of neoplastic origin [2].

It should be emphasized that the decision in this situation is determined not by the absolute size of the node but by the totality of ultrasound characteristics — the shape of the node and the ratio of its longitudinal to transverse dimensions, preservation of the hilum, the type of vascularization — as well as its dynamics over time [1, 2]. Normal values of lactate dehydrogenase and beta-2-microglobulin are

indirect markers with limited sensitivity and do not, on their own, exclude a lymphoproliferative process. An additional argument in favor of a reactive process is the peripheral blood picture: a preserved ratio of cell populations without a shift in the white blood cell differential, the absence of blast cells, reactive lymphocytes, and nucleated red blood cells, an immature granulocyte fraction of 0.1%, and an erythrocyte sedimentation rate of 2 mm/h. None of these features has independent exclusionary value on its own, but taken together with normal lactate dehydrogenase and beta-2-microglobulin values, they lower the pretest probability of a lymphoproliferative disease. In the case presented, it is precisely the ultrasound characteristics of the nodes that require documentation.

Post-infectious origin. The chronology in this case is consistent: lymphadenopathy developed after influenza, followed by a repeat episode of fever and a transient rise in C-reactive protein, against a background of a previously documented Epstein-Barr virus infection with a high antibody titer to the nuclear antigen. The detected viral load of 2.7×10^2 IU/mL is residual and corresponds to the latent stage rather than active replication, which is consistent with the absence of reactive (atypical) lymphocytes in the peripheral blood. Adenoid hypertrophy and mesenteric lymphadenitis in this context are regarded as manifestations of systemic hyperplasia of lymphoid tissue in response to repeated antigenic stimulation. However, in persistent lymphadenopathy it is advisable to assess viral load over time together with a complete antibody profile, since chronic active Epstein-Barr virus infection is a rare but significant differential diagnosis.

Elevated eosinophil cationic protein with normal immunoglobulin E. The most important, and at the same time the least utilized, finding in this case is the elevation of eosinophil cationic protein to 102 ng/mL against the laboratory's upper reference limit of 24 ng/mL — that is, a 4.25-fold increase — with a normal total immunoglobulin E level (41.7 IU/mL). Such a deviation is substantial and cannot be regarded as incidental.

It is fundamentally important that eosinophil cationic protein and immunoglobulin E are independent markers: elevation of the former reflects eosinophil activation and can occur outside an IgE-mediated mechanism — in nonallergic asthma, respiratory infections, sinonasal polyposis, idiopathic hypereosinophilia, and also in neurological conditions unrelated to allergy, although in the latter case this concerned expression of the protein's messenger RNA rather than its serum level [5]. Of particular importance is the role of this protein in antiparasitic defense: it is secreted locally in the presence of a helminth, and its serum level is associated with the presence and intensity of helminthic infestation [6]. Elevated eosinophil cationic protein has also been described in eosinophilic lesions of the gastrointestinal tract — esophagitis, gastroenteritis, colitis — and in intestinal parasitic infestations.

This finding is given particular weight by a quantitative benchmark. In a study conducted in communities in Ghana endemic for hookworm infection, an eosinophil cationic protein threshold of 84.98 ng/mL was associated with a positive predictive value of 98% (95% CI 92–100) and a negative predictive value of 76% (95% CI 62–87) for classifying helminthic infestation status, with an area under the ROC curve of 96.3% [6]. Our patient's value — 102 ng/mL — exceeds this threshold. It should be emphasized that the

study was conducted in an endemic region with a high pretest probability of infestation, so direct extrapolation of this threshold to the Ukrainian population is not appropriate, and the comparison presented here is only indicative. Nevertheless, it is sufficiently compelling to make parasitological testing a mandatory step rather than an optional one.

A notable feature of this observation is the dissociation between the eosinophil cationic protein level and the peripheral blood picture: a 4.25-fold elevation of the protein was not accompanied by eosinophilia — the relative eosinophil count was 5%, at the upper limit of the laboratory's reference interval of 5%, and the calculated absolute count of approximately 0.40 × 10^9/L did not reach the threshold of 0.5 × 10^9/L. This circumstance does not diminish the significance of the finding. Eosinophil cationic protein reflects the activation and degranulation of eosinophils rather than their number, and is released predominantly in tissues; therefore a normal or borderline blood cell count does not exclude local eosinophilic inflammation, in particular in the intestinal or airway mucosa.

At the same time, the absence of marked eosinophilia makes massive helminthic infestation and primary hypereosinophilic syndromes less likely and shifts the differential diagnosis toward localized eosinophilic lesions and limited parasitic infestation. A pre-analytical factor should also be taken into account: the serum concentration of eosinophil cationic protein depends on the time and temperature of sample clotting, since eosinophil degranulation continues in vitro; therefore, in the absence of eosinophilia, it is advisable to repeat the test under standardized collection conditions. Thus, the complete blood count data refine, but do not eliminate, the indications for targeted parasitological testing.

In the case presented, this finding potentially explains several clinical manifestations at once: the mesenteric lymphadenitis and abdominal component may correspond to parasitic infestation or eosinophilic bowel disease, while the nocturnal cough and adenoiditis may correspond to eosinophilic inflammation of the airways. Given the eosinophil count already available, the mandatory minimum in this situation is a three-fold stool examination for helminth eggs and serological testing for toxocariasis, ascariasis, and giardiasis; cases have been described in which precisely this approach made it possible to identify toxocariasis as the cause of eosinophil activation, albeit in an adult patient [7]. Evaluation of bronchial hyperreactivity is warranted given the nocturnal cough.

Isolated decrease in parathyroid hormone with normocalcemia. A parathyroid hormone level of 8.74 pg/mL, below the lower limit of normal of 14.9 pg/mL, requires an explanation consistent with physiology. An explanation invoking a vitamin D receptor gene polymorphism with the development of vitamin D resistance does not withstand scrutiny: in vitamin D deficiency and in tissue resistance to it, the expected response is a compensatory rise in parathyroid hormone, that is, secondary hyperparathyroidism, not its decrease.

The differential diagnosis of isolated low parathyroid hormone includes hypomagnesemia, hypercalcemia, activating mutations of the calcium-sensing receptor, autoimmune polyglandular syndrome, and damage to the parathyroid glands [8]. The first and most important step is to exclude

hypomagnesemia: impaired parathyroid hormone secretion due to magnesium deficiency is referred to as paradoxical hypoparathyroidism, and even moderate depletion of magnesium stores can cause a clinically significant decrease [9]. This condition is fully reversible with correction of magnesium levels, and the accompanying hypocalcemia does not respond to calcium supplementation. Only after hypomagnesemia has been excluded is a diagnostic work-up directed toward hypoparathyroidism warranted [8, 10]. The minimum set of investigations includes ionized and total calcium, phosphorus, magnesium, creatinine, and repeat measurement of parathyroid hormone. In this patient, total serum calcium and magnesium were already within reference limits (Table 1); however, a single normal total serum magnesium value does not fully exclude clinically relevant magnesium depletion, since even moderate reductions in body magnesium stores can impair parathyroid hormone secretion without lowering serum magnesium below the laboratory reference range [9]. Assessment of urinary magnesium excretion is therefore planned to further characterize magnesium status before attributing the low parathyroid hormone level to another cause.

Limits of interpretation of molecular genetic results. The LCT − 13910 (rs4988235) polymorphism is a marker of European-type lactase persistence. The C allele is associated with lactase non-persistence, whereas the T variant disrupts a transcription factor binding site and ensures continued expression of the enzyme [11, 12]. The diagnostic marker of primary adult-type hypolactasia is the homozygous CC genotype; carriers of the TT and CT genotypes are classified as lactase-persistent [11, 13]. In heterozygotes, lactase levels — both enzymatic activity and messenger RNA level — are intermediate, but sufficient to remain asymptomatic [12]. Therefore, establishing a diagnosis of primary hypolactasia based on the CT genotype in the absence of clinical manifestations and without a hydrogen breath test has no genetic basis.

A similar caveat applies to polymorphisms of the vitamin D receptor gene. Associations between the rs2228570 and rs1544410 variants and 25(OH)D levels and clinical outcomes remain weak and inconsistent [14, 15, 18]. Approaches to individualizing dose based on genetic variants are considered investigational and are recommended with caution — mainly within the scope of research studies or when standard strategies have proven ineffective [14]. Current clinical guidelines on vitamin D prescribing, including the Endocrine Society's recommendations, do not provide for dose adjustment based on vitamin D receptor genotype [16, 17]. A statement about genetically verified resistance necessitating higher therapeutic doses is an extrapolation beyond the available evidence base.

The wording used in the laboratory report deserves separate attention. The interpretation provided in the accompanying text of the LCT genotyping result indicated reduced enzyme activity in the heterozygous state, which most likely became the source of the subsequent clinical conclusion. This illustrates a more general problem: a clinical decision is often shaped by the laboratory's wording rather than by the primary genetic data.

Justification of the dietary and medication prescriptions. A 6-month elimination diet excluding milk, restricting gluten and carbohydrates, was prescribed before the results of serological testing for celiac

disease were obtained and without clinical or functional confirmation of lactose intolerance. In an 8-year-old child, prolonged dietary restrictions carry an independent risk of nutritional deficiency, effects on growth, and the development of disordered eating patterns; their prescription therefore requires verified indications. Likewise, the prescription of ursodeoxycholic acid, calcium, zinc, and iron preparations in this observation was not accompanied by documented indications: liver function tests, zinc level, transferrin saturation, and total iron-binding capacity of the serum are not reported, and the ferritin level was within the reference interval for boys of this age group.

The unresolved question of recurrent nosebleeds. Epistaxis is among the leading complaints, yet no coagulation work-up was performed. In a child with recurrent nosebleeds, it is mandatory to exclude von Willebrand disease and platelet function disorders, which requires a coagulogram, determination of von Willebrand factor, and platelet aggregation studies. Attributing epistaxis to subclinical vascular changes without such testing remains a mere assumption.

STRENGTHS OF THE DIAGNOSTIC APPROACH

The observation presented also demonstrates a number of methodologically sound decisions. The immunological work-up was performed as a full panel with assessment of lymphocyte subpopulations and serum immunoglobulins, which made it possible to reasonably exclude primary and severe secondary immunodeficiency. The herpesvirus panel was tested by polymerase chain reaction rather than serology alone, which is the correct approach for distinguishing latency from active replication. Latent tuberculosis was excluded using the QuantiFERON test — a mandatory step in persistent lymphadenopathy. Finally, the working diagnosis of hypoparathyroidism was reviewed and withdrawn based on the results of repeat testing, illustrating good practice in the dynamic management of the patient.

CONCLUSIONS

1. Persistent lymphadenopathy with node size exceeding 20 mm in a child requires not only laboratory testing but also a structured ultrasound description of the nodes over time; normal values of lactate dehydrogenase and beta-2-microglobulin are indirect markers and do not, on their own, exclude a lymphoproliferative process.
2. Elevated eosinophil cationic protein with a normal immunoglobulin E level is not a diagnostically neutral finding; a normal absolute eosinophil count in peripheral blood does not negate this finding, since the marker reflects activation rather than cell number, and it requires targeted exclusion of parasitic infestation and eosinophilic diseases of the gastrointestinal tract and airways, as well as repeat measurement under standardized pre-analytical conditions.

3. Isolated decreased parathyroid hormone with normocalcemia requires, above all, the exclusion of hypomagnesemia as a fully reversible cause; attributing this phenomenon to vitamin D resistance contradicts the physiology of calcium metabolism regulation.
4. The LCT – 13910 CT genotype corresponds to lactase persistence and is not a basis for a diagnosis of primary hypolactasia; polymorphisms of the vitamin D receptor gene cannot serve as a basis for adjusting the vitamin D dose, since current clinical guidelines do not provide for such an approach.
5. Prolonged elimination diets in pediatrics should be prescribed only for verified indications, since they themselves carry a risk of nutritional deficiency.

DECLARATIONS

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Author contributions: Both authors contributed to the study conception and clinical management. S.O.N. examined the patient, conceived the analysis, and drafted the manuscript. I.M.T. contributed clinical data from the referring hospital and critically revised the manuscript. All authors read and approved the final manuscript.

Ethics approval: This study was performed in line with the principles of the Declaration of Helsinki. Approval was granted by the Ethics Committee of I. Horbachevsky Ternopil National Medical University (No. 86 , July 01, 2026).

Consent to participate: As the patient is a minor (aged 8 years at the time of examination), written informed consent to participate was obtained from the patient's parents (legal guardians).

Consent to publish: As the patient is a minor (under 18 years of age), written informed consent for publication was obtained from the patient's parents (legal guardians). The consent covers publication of the clinical details of this case, including the history, physical examination, ultrasound, laboratory, and molecular genetic findings presented in this article. No clinical images of the patient are included in the article. All personal data have been de-identified so that the patient cannot be identified.

Data availability: The datasets analysed during this case report are not publicly available due to patient privacy but are available from the corresponding author on reasonable request.

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TABLES

Table 1. Main laboratory parameters

Parameter	Result	Reference values	Assessment
C-reactive protein, mg/L	39.91 → 0.6	< 5	normalization
Lactate dehydrogenase, U/L	294	age norm	normal
Beta-2-microglobulin, ng/mL	1729	604–2157	normal
Ferritin, ng/mL	22.3	18.2–90	normal, lower third
Total IgE, IU/mL	41.7	< 52	normal
Eosinophil cationic protein, ng/mL	102	< 24.0	elevated 4.25-fold
25(OH)D, ng/mL	31.1	30.0–50.0	lower limit of normal
Parathyroid hormone, pg/mL	8.74	14.9–56.9	decreased
Ionized Ca mmol/L	1.2	1.15–1.32	normal
Mgmmol/L	0.8	0.70–0.95	normal
ALT U/L	32	30–35	normal
AST U/L	38	32–48	normal

Table 2. Complete blood count parameters

Parameter	Result	Reference values	Assessment
Hemoglobin, g/L	131	age norm	normal
Neutrophils, %	42.2	35–65 (6–12 years)	normal
Band neutrophils, %	4	1–5	normal
Segmented neutrophils, %	41	35–65 (6–12 years)	normal
Lymphocytes, %	44.5	24–54 (6–12 years)	normal
Lymphocytes, abs., ×10⁹/L	3.57	age norm	normal
Monocytes, %	6.8	2–10 (1–16 years)	normal
Monocytes, abs., ×10⁹/L	0.55	0.30–0.82	normal
Eosinophils, %	5	1–5	upper limit of normal
Eosinophils, abs., ×10⁹/L	≈ 0.40 (calculated)	< 0.50	no eosinophilia
Basophils, %	1	0–1	upper limit of normal
Reactive lymphocytes, %	0	0	absent
Immature granulocytes (IG), %	0.1	< 2.0	normal
Nucleated red blood cells (NRBC), %	0.0	0.0	absent
ESR (Westergren), mm/h	2	< 10 (up to 17 years)	normal