Pediatr Pol 2025; 100 (4): 353-358
DOI: https://doi.org/10.5114/polp.2025.156422
Submitted: 28.07.2025, Accepted: 01.09.2025, Published: 24.11.2025

PEDIATRIA Polska
PISMO POLSKIEGO TOWARZYSTWA PEDIATRYCZNEGO
Polish Journal of PAEDIATRICS
OFFICIAL JOURNAL OF POLISH SOCIETY OF PAEDIATRICS

REVIEW PAPER

Prolonged activated partial thromboplastin time in pediatric patients – an important diagnostic challenge

Aleksandra Zima[1,2], Joanna Zdziarska[3]

[1]Clinical Department of Pediatrics and Neurology, St. Louis Regional Specialized Children's Hospital, Cracow, Poland
[2]Collegium Medicum, Andrzej Frycz Modrzewski University, Cracow, Poland
[3]Department of Hematology and Internal Medicine, Jagiellonian University Medical College, Cracow, Poland

ABSTRACT

Basic hemostasis tests are among the most frequently performed laboratory investigations in pediatric practice. Therapeutic decisions regarding a child are often based on the results of screening hemostatic tests, such as the activated partial thromboplastin time (aPTT) or prothrombin time. Therefore, careful and accurate interpretation of these tests in the context of the specific patient, their clinical condition, and medical history is crucial for making the correct diagnosis. Although in most pediatric cases, aPTT prolongation is caused by pre-analytical errors, one must not overlook the possibility of underlying plasma coagulation disorders. In this article, we also highlight the relationship between the presence of mild plasma coagulation disorders and prolonged aPTT values.

KEY WORDS:
bleeding, hemostasis, child, activated partial thromboplastin time, hemorrhagic disorders.

INTRODUCTION

The activated partial thromboplastin time (aPTT), alongside the prothrombin time (PT), is one of the most commonly assessed hemostatic parameters in pediatric practice.

Diagnostic and therapeutic decisions regarding pediatric patients are often based on aPTT results. In many cases, the outcome of these coagulation tests determines the course of further medical procedures, such as surgery or other invasive interventions. For this reason, proper interpretation of this widely used hemostatic parameter in pediatrics is of paramount importance.

This article presents clinical scenarios in which prolonged aPTT occurs in pediatric patients. We will discuss both conditions of significant clinical relevance and those clinically silent and not associated with an increased bleeding risk.

HEMOSTASIS

Hemostasis is the result of the coordinated interaction between primary hemostasis and the processes governing secondary hemostasis. Immediately following injury to a blood vessel wall, multiple mechanisms are activated which contribute to primary hemostasis. Exposed, negatively charged collagen fibers in the damaged vessel wall initiate vasoconstriction, thereby reducing blood flow through the injured capillary. Simultaneously, circulating blood platelets are recruited to the site of injury. This triggers the processes of platelet adhesion, activation and then aggregation of platelets. The goal of primary hemostasis is to form a temporary platelet plug that seals the injured vessel and prevents further blood loss [1, 2].

The subsequent phase of the coagulation process is the activation of secondary hemostasis, whose purpose is

ADDRESS FOR CORRESPONDENCE:
Dr. Aleksandra Zima, Clinical Department of Pediatrics and Neurology, St. Louis Regional Specialized Children's Hospital, Cracow, Poland, e-mail: aleksandra.a.zima@gmail.com

to form a stable and durable blood clot. This is achieved through the action of numerous plasma coagulation factors [1, 2].

The traditional coagulation cascade model described the intrinsic and extrinsic pathways as separate mechanisms that independently activated plasma factor X and initiated processes within the so-called common pathway.

In 2003, a cell-based model of the coagulation cascade was introduced, indicating that cells, rather than plasma proteins, play a central and fundamental role in the formation of a stable blood clot. Modern theory emphasizes the interconnected nature of both pathways and highlights the crucial importance of cellular components in the coagulation process [2].

The cell-based coagulation model comprises the following phases:

- initiation phase – factor VII circulates in plasma predominantly in its inactive form, with only small amounts present in the active form. When the active form of factor VII encounters a sufficient quantity of tissue factor, it binds to it, simultaneously recruiting a small amount of factor X. This leads to the formation of the prothrombinase complex, which activates circulating prothrombin and generates small amounts of thrombin;
- amplification phase – the small amount of thrombin generated during the initiation phase becomes the key driver of the coagulation process. Thrombin activates platelets and, through positive feedback, activates factors V, VIII, and XI. This results in the formation of the active complex of factors IXa/VIIIa, which accumulates on the surface of platelets and generates increasing amounts of activated factor X (Xa);
- propagation phase – activated factors V and VIII (Va and VIIIa), anchored to the surface of platelets, participate in two key pathways responsible for the activation of other coagulation factors.

Factor VIII functions within the tenase complex, composed of factors IXa, VIIIa, X, and calcium ions. Interactions between factors IXa and X lead to increased generation of factor Xa. The newly formed factor Xa rapidly binds to the prothrombinase complex initiated by factor V on the surface of activated platelets.

The final step involves the conversion of factor II (prothrombin) into thrombin (active factor II).

These processes occur on all platelets that are recruited, aggregated, and activated at the site of vascular injury [2].

The fibrin mesh envelops and reinforces the platelet plug formed during the primary hemostasis phase, stabilizing it and making it more resistant to mechanical disruption [1, 2].

For educational purposes, it is generally accepted that a prolonged aPTT may reflect abnormalities within the intrinsic pathway and the common pathway [1, 3].

Thus, it may indirectly indicate deficiencies of plasma coagulation factors involved in these pathways. These include factors I, II, V, VIII, IX, X, XI, XII, as well as contact factors [1, 3].

ACTIVATED PARTIAL THROMBOPLASTIN TIME TESTING

The activated partial thromboplastin time test is performed on platelet-poor plasma after the addition of a phospholipid reagent (e.g., cephalin) and an activator of the intrinsic pathway (e.g., kaolin). This mixture is incubated for 3–5 minutes at 37°C. Calcium chloride is then added, and the time until fibrin formation is measured.

The activated partial thromboplastin time value reflects the clotting time of plasma after maximal activation of factors XI and XII in the presence of phospholipids [1, 3, 4].

It is important to note that reference ranges for aPTT may vary between diagnostic laboratories, as they depend on the specific reagents and equipment used. Therefore, each aPTT result should always be interpreted in the context of the reference ranges established and accepted by the performing laboratory [4].

ISOLATED PROLONGATION OF ACTIVATED PARTIAL THROMBOPLASTIN TIME

Activated partial thromboplastin time, particularly when prolonged, must always be interpreted in conjunction with the physical examination and clinical history obtained from the patient or their caregivers.

Isolated prolongation of aPTT refers to a clinical situation in which PT and thrombin time (TT) are within normal limits, the peripheral blood platelet count is normal, and plasma fibrinogen concentration is also within the reference range.

Thus, when a prolonged aPTT is detected in a pediatric patient, further diagnostic evaluation should include measurement of prothrombin time, thrombin time, fibrinogen concentration, and a complete blood count to assess the platelet count. Only after obtaining all of these hemostasis parameters can further diagnostic steps be properly guided.

In other words, isolated prolongation of aPTT refers to a situation in which a prolonged aPTT is the *only* abnormality detected in the hemostatic system, based on all relevant laboratory findings.

Isolated aPTT prolongation may have a variety of causes – clinically significant or clinically silent. It may indicate a deficiency of coagulation factors involved in the intrinsic or common pathway. However, in many cases, especially in pediatric patients, such isolated prolongation of aPTT is the result of pre-analytical or analytical errors rather than true hemostatic abnormality [3–5].

MOST COMMON CAUSES OF PROLONGED ACTIVATED PARTIAL THROMBOPLASTIN TIME

AGE OF THE CHILD

Before discussing clinical conditions that may significantly affect aPTT values in children, it is important to

TABLE 1. Activated partial thromboplastin time depending to age

Parameters	15 days – 4 weeks	1–5 months	6–11 months	1–5 years	6–10 years	11–17 years	Adults
PT (sec) (ratio) (%)	11.2 [9.5–12.6] 0.98 [0.83–1.13] 100.0 [83–109] (*n* = 36)	11.0 [9.7–12.8] 0.97 [0.84–1.17] 100.0 [78–108] (*n* = 320)	11.0 [9.8–13.0] 0.98 [0.85–1.13] 100.0 [81.9–108.3] (*n* = 176)	11.3 [9.9–13.4] 0.99 [0.86–1.20] 100.0 [76.0–103.8] (*n* = 507)	11.7 [10.0–14.6] 1.04 [0.90–1.30] 94.5 [66.6–111.2] (*n* = 132)	11.8 [10.0–14.1] 1.04 [0.90–1.24] 94.0 [72.0–109.0] (*n* = 262)	10.9 [9.2–12.2] 0.95 [0.81–1.07] 109.5 [90.0–148.8] (*n* = 64)
aPTT SynthASil (sec) (ratio)	35.4 [27.6–45.6] 1.16 [0.91–1.43] (*n* = 36)	33.5 [24.8–40.7] 1.10 [0.82–1.30] (*n* = 311)	32.4 [25.1–40.7] 1.07 [0.82–1.32] (*n* = 174)	31.6 [24.0–39.2] 1.06 [0.80–1.31] (*n* = 490)	31.6 [26.9–38.7] 1.04 [0.85–1.28] (*n* = 124)	31.0 [24.6–38.4] 1.02 [0.81–1.28] (*n* = 249)	31.7 [27.8–36.3] 1.04 [0.86–1.20] (*n* = 64)
aPTT SP (sec) (ratio)	39.0 [33.2–45.6] 1.28 [1.12–1.43] (*n* = 36)	33.3 [25.0–43.3] 1.10 [0.86–1.38] (*n* = 32)	34.3 [31.7–45.3] 1.12 [1.04–1.49] (*n* = 31)	32.4 [25.7–38.4] 1.07 [0.84–1.26] (*n* = 34)	32.8 [25.5–42.4] 1.08 [0.87–1.39] (*n* = 34)	32.6 [26.1–47.4] 1.06 [0.86–1.55] (*n* = 34)	32.8 [26.9–39.5] 1.08 [0.88–1.30] (*n* = 44)

aPTT – activated partial thromboplastin time, n – number of evaluated samples, PT – prothrombin time
Median values and age-specific reference ranges defined, according to the CLSI-C28-A3 guideline, as the 95% CI in the different age-groups, for routine coagulation tests in the different age-groups.
Prothrombin time was expressed as the clotting time, the patient-to-control ratio, and the percentage activity.
Activated partial thromboplastin time, evaluated using two reagents, was expressed as the clotting time and the patient-to-control ratio. The number of evaluated samples in each age group is shown in brackets.

remember that aPTT results are influenced not only by the diagnostic laboratory and the reagents used, but also by the child's age.

Immaturity of the liver in the first weeks of life is associated with reduced activity of several coagulation factors synthesized in the liver – namely, factors II, VII, IX, and X. This *physiological deficiency* may result in a prolonged aPTT [6].

Age-appropriate norms and reference ranges for pediatric patients are presented in Table 1 [7].

PRE-ANALYTICAL ERRORS

The most common clinical situations leading to isolated prolongation of aPTT in children, not related to plasma coagulation disorders, include:

- improper blood collection, traumatic venipuncture,
- hemolysis in the patient's blood sample tube,
- hyperbilirubinemia,
- prolonged storage or transport of the blood sample,
- rheumatologic diseases associated with the presence of lupus anticoagulant in the serum (e.g., systemic lupus erythematosus),
- congenital deficiencies of so-called contact factors (coagulation factor XII, prekallikrein, or high molecular weight kininogen),
- dehydration states,
- lipemic serum,
- diseases associated with increased hematocrit,
- inflammatory conditions with elevated C-reactive protein (CRP) levels,
- contamination of the tube with unfractionated heparin,
- presence of air bubbles in plasma or reagent.

Many of the above clinical situations can be avoided by proper patient preparation.

To accurately determine aPTT and minimize pre-analytical errors in pediatric patients, it is essential to collect blood for coagulation testing into tubes containing 3.2% sodium citrate solution at a volumetric ratio of 9 : 1. Even a slight deviation from this ratio can affect the accuracy of the results [1, 4, 5].

The patient should be well hydrated. In patients with elevated hematocrit (> 55%) due to an underlying condition (e.g., polycythemia), tubes with individually adjusted anticoagulant volumes prepared by the laboratory should be used [1, 4, 5].

Dehydration complicates the interpretation of aPTT also because it may be associated with traumatic venipuncture and the need for tourniquet application [4, 5].

Venous blood for coagulation studies should not be collected from central venous catheters flushed with heparin, nor from lines through which medications that may interfere with aPTT results are administered, such as anticoagulants, heparin preparations, or coagulation factor concentrates [1, 3–5, 8].

It is recommended that, due to the risk of lipemia in the serum, the patient should be fasting or have consumed a light meal prior to blood collection.

In patients with hyperbilirubinemia, whenever possible, a follow-up aPTT test should be performed after normalization of serum bilirubin levels [1, 4].

Blood samples should be stored and transported at room temperature. The time between blood collection and delivery to the analytical laboratory is critically important – samples should be delivered no later than 4 hours after collection. After this period, coagulation factor activity in the collected blood naturally decreases, which may result in falsely prolonged aPTT [4, 5].

Various inflammatory conditions and infections associated with elevated serum C-reactive protein (CRP) levels may influence prolongation of aPTT. If possible, the test should be performed (or repeated) when the patient is in full health or during recovery [1, 9, 10].

In some patients, circulating lupus anticoagulant (LA) in plasma causes prolongation of the aPTT [1, 4, 8, 11].

LA is a type of antiphospholipid antibody. It can be detected in patients with antiphospholipid syndrome, systemic lupus erythematosus, and other rheumatologic diseases. It may also be transiently and periodically detected in a small percentage of healthy individuals, including children [12].

Currently, reagents that measure aPTT and are insensitive to the presence of lupus anticoagulant are available on the market.

In patients suspected of having circulating antibodies in the serum, a mixing study with normal plasma (plasma correction test) can also be helpful [5, 8].

In the case of congenital coagulation factor deficiency, the aPTT shortens (corrects) after the addition of normal plasma. The correction test is then described as negative [5, 8].

Conversely, in the presence of circulating anticoagulants (LA) or other antibodies directed against specific coagulation factors, the addition of normal plasma does not fully correct the aPTT – the correction test is then positive [5, 8].

Significant prolongation of aPTT (often with results that are unmeasurable or in the triple digits) may be caused by deficiencies of contact factors involved in the intrinsic coagulation pathway. The most commonly observed deficiency is factor XII, while deficiencies of the other two contact factors (prekallikrein or high molecular weight kininogen) are much rarer [1, 8, 13, 14].

Clinically, deficiency of any of these factors does not cause bleeding symptoms, even with complete absence of the respective protein in plasma. Congenital deficiency of factor XII is also known as Hageman anomaly and is found in a few percent of the healthy population [13, 14].

This is clinically insignificant and considered an incidental finding, most often diagnosed due to prolonged aPTT in a patient without any bleeding symptoms. Factor XII deficiency, because of its association with prolonged aPTT, may delay necessary medical procedures, lead to disqualification of patients from surgeries or invasive procedures, and even result in unnecessary transfusions of fresh frozen plasma.

The greater the deficiency of factor XII, the more pronounced the prolongation of aPTT. In complete factor XII deficiency, the aPTT may reach values over 150–200 seconds, or even become unmeasurable [1, 13, 14].

The result may vary depending on the laboratory and, as previously mentioned, is influenced by the reagents used [4].

The most important consideration when identifying deficiency of any contact factor is for clinicians to be aware that patients with factor XII, prekallikrein, or high molecular weight kininogen deficiency do not require any special preparation for surgical procedures or invasive interventions such as lumbar puncture, bone marrow biopsy, epidural, or spinal anesthesia. This is because these conditions have no clinical significance.

At this point, it is essential to emphasize the importance of a thorough physical examination and a well-conducted/taken patient history. In cases of isolated deficiency of any contact factor, the patient shows no bleeding symptoms despite significant prolongation of aPTT. Both personal and family histories do not indicate any signs of bleeding disorders.

It should not be forgotten, however, that isolated prolongation of aPTT in a patient with factor XII deficiency (or other contact factor deficiencies) may mask other congenital or acquired deficiencies of plasma coagulation factors (e.g., factor XI, IX, VIII). For this reason, in the differential diagnosis, it is advisable to also measure other coagulation factors involved in the extrinsic pathway and the common pathway to confirm isolated deficiency of factor XII, prekallikrein, or high molecular weight kininogen – these are responsible for prolongation of aPTT without deficiency of any other plasma coagulation factor.

Undoubtedly, prolonged aPTT can also be associated with certain bleeding disorders [1, 4, 5]. However, in the course of bleeding disorder diagnostics, it is important to remember a critical clinical correlation – prolongation of aPTT occurs only when coagulation factor activity decreases below 30–40% of normal values.

Therefore, most children with mild plasma coagulation disorders will have completely normal screening hemostasis test results, including aPTT [5].

At this point, it is also important to emphasize the critical importance of both physical examination and patient history. A thorough history of bleeding symptoms (for example, excessive bleeding after injury or surgery, frequent nosebleeds, easy bruising, joint and muscle bleeds), not only in the child but also in their close relatives, is of utmost significance. A positive personal or family history should prompt pediatricians and general

practitioners to pursue more in-depth hematological diagnostics, even in patients with completely normal basic hemostasis screening tests, as they may have a mild form of a bleeding disorder without any abnormalities in laboratory tests [5].

Congenital deficiencies of factors VIII, IX, and XI cause prolongation of aPTT to a degree dependent on the severity of the deficiency and the sensitivity of the laboratory reagent used. Prolonged aPTT is also observed in all patients with severe (type 3) von Willebrand disease (VWD), as well as in some cases of type 2, and very rarely in type 1 VWD [1, 5].

This further confirms the recommendation that it is not the aPTT value but the patient's personal history and physical examination that should form the basis for further diagnostics of plasma coagulation disorders.

It is worth noting that in von Willebrand disease, prolongation of aPTT is not due to deficiency of von Willebrand factor itself, but secondary to deficiency of factor VIII [5].

In summary, regarding the variability of aPTT in plasma coagulation disorders, it can be stated that in patients with normal aPTT values, only severe and moderate forms of hemophilia, as well as severe von Willebrand disease, can be excluded.

All other plasma coagulation disorders may present with normal aPTT values [5].

When discussing prolonged aPTT, one should not forget the rare bleeding disorder known as acquired hemophilia A (AHA), which can also occur in pediatric patients.

Acquired hemophilia A is an autoimmune disease caused by the presence of antibodies directed against factor VIII.

In approximately half of the cases, it is idiopathic; however, it can be secondary to other autoimmune diseases, malignancies, or may complicate pregnancy and the postpartum period [1, 15].

In patients with AHA, the previous personal history is completely negative for bleeding disorders. Patients with AHA also have a negative family history and normal aPTT results measured in the past (prior to disease onset).

In most patients with acquired hemophilia, the first symptom of the disease is severe, often life-threatening bleeding, both spontaneous and post-traumatic.

These patients may experience not only mucosal bleeding but primarily extensive subcutaneous and intramuscular hemorrhages.

The incidence of acquired hemophilia is low (approximately 1.5 per 1 million per year), but this condition carries a high mortality rate if not diagnosed and properly treated. In the pediatric population, its occurrence is even rarer, approximately 0.045 cases per million per year [15].

However, due to the increasing prevalence of autoimmune diseases in pediatric patients, it is important to remember the possibility of acquired hemophilia occurring in this age group as well.

Isolated prolongation of aPTT is also observed in some cases of acquired von Willebrand syndrome – a rare acquired coagulation disorder seen in patients with malignancies, autoimmune diseases, or cardiovascular diseases (it is encountered in children with congenital heart defects) [1].

Finally, the authors of this paper wish to inform readers about the limitations and principles of measurement and interpretation of aPTT and aPTT-based assays in pediatric patients with hemophilia A complicated by factor VIII inhibitors, who are being treated with a non-factor replacement therapy, emicizumab (Hemlibra®) [16].

Emicizumab is a recombinant, humanized monoclonal antibody that binds activated coagulation factor IX and factor X, thereby restoring the ability to generate thrombin in factor VIII deficient plasma. In other words, emicizumab mimics activated factor VIII in the coagulation process. However, this antibody has no structural similarity to factor VIII, which translates into a significant therapeutic advantage – neutralizing antibodies against factor VIII do not interfere with emicizumab activity. The drug is administered subcutaneously, and the dosing frequency is very convenient: once weekly, every two weeks, or even every four weeks [16].

Emicizumab has a significant impact on certain coagulation assays. It shortens the aPTT and therefore interferes with the results of aPTT-based coagulation assays, including factor VIII activity testing. Even at minimal plasma concentrations of emicizumab (> 5 μg/ml), the aPTT in a child with severe hemophilia A may appear falsely normalized [16, 17].

The effect of emicizumab on PT is minimal, and no effect is observed on TT or on fibrinogen levels measured by the Clauss method.

For this reason, results of factor activity assays based on aPTT measurement are considered unreliable, whereas PT-based assays provide valid results.

In summary, it should be emphasized that in children and adolescents with severe hemophilia A complicated by inhibitors and treated with emicizumab, aPTT measurement is not suitable for monitoring therapy. In these patients, a prolonged aPTT may indicate non-adherence to therapeutic recommendations or suggest the development of anti-drug antibodies, which in turn leads to a reduction in emicizumab plasma concentration to < 5 μg/ml [16, 17].

Such patients represent a diagnostic challenge for general practitioners and pediatricians. Therefore, it is crucial that they remain under the regular care of Hemophilia Treatment Centers and Pediatric Bleeding Disorder Centers.

CONCLUSIONS

In every case of prolonged aPTT, regardless of whether the patient has current or past bleeding episodes, efforts

should be made to determine the cause of this abnormality.

An abnormal aPTT result should always be closely correlated with the patient's clinical presentation, history, and results of other screening hemostasis tests such as PT (prothrombin time), TT (thrombin time), fibrinogen concentration, and platelet count.

The fundamental question in daily clinical practice, which should be asked regardless of laboratory test results, is whether pediatric patients (and in the case of very young children – also their close relatives) have any symptoms of a bleeding disorder:

- in patients without any bleeding symptoms, and with a negative personal history, in whom aPTT was measured routinely, a repeat test should be performed with particular attention to excluding all possible pre-analytical errors. If isolated prolonged aPTT persists despite proper preparation, hematological diagnostics should be expanded/extended (possible contact factor deficiency or circulating anticoagulant);
- in children with isolated prolonged aPTT whose symptoms or personal history strongly suggest the presence of a bleeding disorder, further hematological evaluation is warranted;
- in pediatric patients with a personal and/or family history suggestive of coagulation disorders but normal laboratory hemostasis screening tests, hematological diagnostics should also be extended due to the possibility of mild plasma coagulation disorders.

DISCLOSURES

1. Institutional review board statement: Not applicable.
2. Assistance with the article: None.
3. Financial support and sponsorship: None.
4. Conflicts of interest: None.

REFERENCES

1. Santoro RC, Molinari AC, Leotta M, Martini T. Isolated prolongation of activated partial thromboplastin time: not just bleeding risk! Medicina 2023; 59: 1169.
2. Sierra C, Moreno M, García-Ruiz JC. The physiology of hemostasis. Blood Coagul Fibrinolysis 2022; 33: S1-S2.
3. Loizou E, Mayhew D, Martlew V, Murthy BVS. Implications of deranged activated partial thromboplastin time for anaesthesia and surgery. Anaesthesia 2018; 73: 1557-1563.
4. Chojnowski K, Podolak-Dawidziak M, Windyga J. Diagnosis of the prolonged activated partial thromboplastin time (aPTT). Hematologia 2010; 1: 81-86.
5. Zdziarska J, Chojnowski K, Klukowska A, Łaguna P, Łętowska M, Mital A, et al. Postępowanie w chorobie von Willebranda. Zalecenia Grupy ds. Hemostazy Polskiego Towarzystwa Hematologów i Transfuzjologów 2022. J Transfus Med 2022; 15: 100-126.
6. Blanchette VS, Kahr WHA. Work-up of a bleeding child. In: Lee CA, Berntorp EE, Hoots KW (eds.). Textbook of hemophilia. Blackwell Publishing, Oxford 2005, 112-119.
7. Toulon P, Berruyer M, Brionne-François M, Grand F, Lasne D, Telion C, et al. Age dependency for coagulation parameters in paediatric populations. Results of a multicentre study aimed at defining the age-specific reference ranges. Thromb Haemost 2016; 116: 9-16.
8. Rasmussen KL, Philips M, Tripodi A, Goetze JP. Unexpected, isolated activated partial thromboplastin time prolongation: a practical mini-review. Eur J Haematol 2020; 104: 519-525.
9. Liu J, Li F, Shu K, Chen T, Wang X, Xie Y, et al. The analysis of false prolongation of the activated partial thromboplastin time (activator: Silica): interference of C-reactive protein. J Clin Lab Anal 2018; 32: e22571.
10. Erdem-Eraslan L, Hens JJH, van Rossum AP, Frasa MAM, Keuren JFW. Inter-individual variability in phospholipid-dependent interference of C-reactive protein on activated partial thromboplastin time. Br J Haematol 2018; 183: 681-683.
11. Tripodi A. Laboratory testing for lupus anticoagulants: a review of issues affecting results. Clin Chem 2007; 53: 1629-1635.
12. Betensky M, Mosha M, Tarango C, Verma A, Bhat R, Kucine NE, Nakano T, et al. Outcomes in children with provoked venous thrombosis and antiphospholipid antibodies: findings from the Kids-DOTT trial. Blood Adv 2024; 8: 5790-5795.
13. Halbmayer WM, Haushofer A, Schon R, Mannhalter C, Strohmer E, Baumgarten K, et al. The prevalence of moderate and severe FXII (Hageman factor) deficiency among the normal population: evaluation of the incidence of FXII deficiency among 300 healthy blood donors. Thromb Haemost 1994; 71: 68-72.
14. Schmaier AH. The elusive physiologic role of Factor XII. J Clin Invest 2008; 118: 3006-3009.
15. Mouthon P, Guy A, d'Oiron R, Harroche A, Lebreton A, Gourguechon C, et al. Acquired haemophilia A in paediatric patients: a retrospective French cohort of eight cases. Br J Haematol 2024; 204: 606-611.
16. Windyga J, Chojnowski K, Klukowska A, Łaguna P, Łętowska M, Mital A, et al. Emicizumab (Hemlibra®) u chorych na hemofilię A powikłaną inhibitorem czynnika VIII – wytyczne Grupy ds. Hemostazy Polskiego Towarzystwa Hematologów i Transfuzjologów. J Transf Med Hemost 2020; 13: 153-164.
17. Feriel J, Goujon MA, Desez M, Depasse F. Impact of drugs used in intensive care on routine coagulation testing. Diagnostics (Basel) 2025; 15: 941.