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Case Report

Anesthesia Management on Acquired Prothrombin Complex Deficiency

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Abstract

Acquired prothrombin complex deficiency (APCD), often resulting from vitamin K deficiency, is a rare but potentially life-threatening hemorrhagic disorder in neonates and infants. This case report details the anesthesia management of a 36-day-old infant presenting with subdural hematoma and severe coagulopathy due to APCD. The patient underwent successful surgical intervention following correction of coagulopathy with vitamin K, fresh frozen plasma, and packed red cell transfusions. The report emphasizes the importance of comprehensive preoperative assessment, individualized coagulation therapy, intraoperative hemodynamic stability, and multidisciplinary collaboration to optimize outcomes in APCD cases. Advanced diagnostic tools and personalized treatment strategies are crucial for reducing perioperative complications and improving patient prognosis.

Keywords: acquired prothrombin complex deficiency, anesthesia management, coagulopathy, vitamin K deficiency

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All relevant data are within the paper and its Supporting Information files.

Introduction

It is very important for pregnant women to maintain their health and nutritional intake during pregnancy because improper diets issues can endanger their babies, among them bleeding. One of the potential impacts is vitamin K deficiency, which can occur because the baby does not receive enough vitamin K from the mother during pregnancy. Vitamin K deficiency can disrupt the blood coagulation process, which can lead to bleeding, either spontaneously or after invasive procedure. Vitamin K Deficiency Bleeding (VKDB), which is also known as Acquired Prothrombin Complex Deficiency (APCD).[1] APCD occurs when bleeding happens, either spontaneously or during procedures such as blood draws or surgeries, due to a lack of blood coagulation factors related to vitamin K (factor II, factor III, and factor IV).[2,3]

APCD can occur in all newborns, especially those who do not receive vitamin K prophylaxis immediately after birth. However, since the administration of vitamin K prophylaxis to neonates, the incidence of APCD has decreased. Recent research shows that the incidence of classic type APCD is 0.01–0.44% of all births worldwide, and slow type APCD is 1 in 15,000–120,000 live births. Babies with cholestasis, babies who are exclusively breastfed, and babies with malabsorption often experience APCD. Spontaneous intracranial hemorrhage in newborns is often marked by the finding of APCD. This bleeding is responsible for 20% to 50% of neonatal deaths and disabilities. In Southeast Asia, the mortality rate of APCD in infants ranges from 1:1200 to 1:1500 live births, and it can be higher in regions that do not provide vitamin K prophylaxis to newborns. Until

now, there is no national data on APCD cases in Indonesia.[4]

Case

A male, 36-day old infant, weighing 4,4 kg patient was admitted from the emergency department with chief complain of seizure. Initially, 5 days ago, the patient's head was hit by a rubber ball accidentally kicked by his brother. 1 day later, the patient had 2 times full-body seizure and vomited 5 times. The Patient doesn't want to drink. Seizure <5 minutes. Since being treated, the patient has not had any seizures or vomiting. The patient was born via C-section due to placenta previa with birth weight is 3000 grams. In full term birth, with an Apgar score of 8/9. There is no history of cyanosis and get injection vitamin K on thigh.

During hospitalization, the patient was given injections of 2mg vitamin K1 for 3 days, 40 cc fresh frozen plasma (FFP) transfusion, and 3 times of 40 cc packed red cell (PRC) transfusion. VACTERL history was denied. There is no family history with similar complaints.

Physical examination reveals normal limit for vital signs and other primary surveys except PGCS Score was E3M5V4 while the patient is on 1 lpm of nasal cannula, pallor and bulging fontanel. The Baby Gram examination was conducted and there are no abnormal findings. Head CT scan also conducted and reveal Subdural hematoma right frontotemporoparieto occipital region with cerebral edema and midline shift >15 mm without ventriculomegaly.

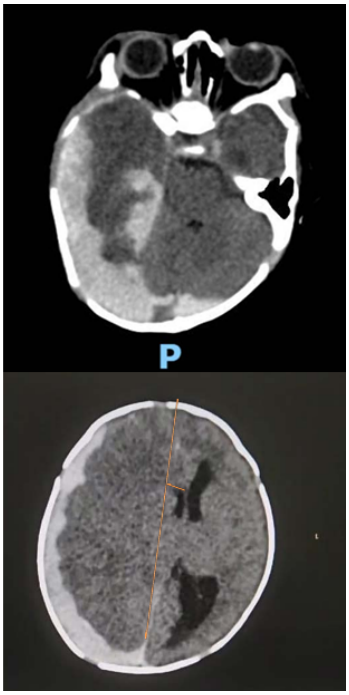

Figure 1. Head CT-Scan

Laboratory examination conducted when admitted in the emergency department reveals:

Table 1. Preoperative Blood Test

Parameter	Value	
	Day 1	Day 5
Hemoglobin	6,6	11
Leukocyte	10.730	15.390
Thrombocyte	571.000	805.000
Hematocrit	17	31,4
Prothrombin Time	>90	14,6
Activated Partial Thromboplastin Time	83	31,6
International Normalized Ratio	-	1,03
Sodium	120	132
Potassium	5,8	3,7
Chloride	91	-

Hemoglobin 6,6 mg/dl and prolonged PT/APTT, >90/83,5 respectively while other parameters within normal limit. On the 5th day of hospitalization, blood test was conducted again to review the administered treatment, the test reveals Hemoglobin has risen to 11 mg/dl and PT/APTT/INR improved to 14,6/31,6/1,03 respectively. This patient showed no signs of neurological abnormalities.

Based on the above examination we diagnosed the patient with Subdural Hematoma at right frontotemporoparietal region with 10,3mm thickness. The Patient has planned undergo Evacuation Craniotomy with pre-operative preparation 4 hours preoperative fasting, 3 PRC 40cc, 1 FFP 40cc, and post operative PICU care.

We performed anesthesia induction on patient with fentanyl 10 mcg, propofol 10 mg, and rocuronium 4mg. While maintenance using sevoflurane 3,2 vol% and O2: Air ratio is 50:50. Patient vital signs was monitored during the surgery as shown in **Figure 2**.

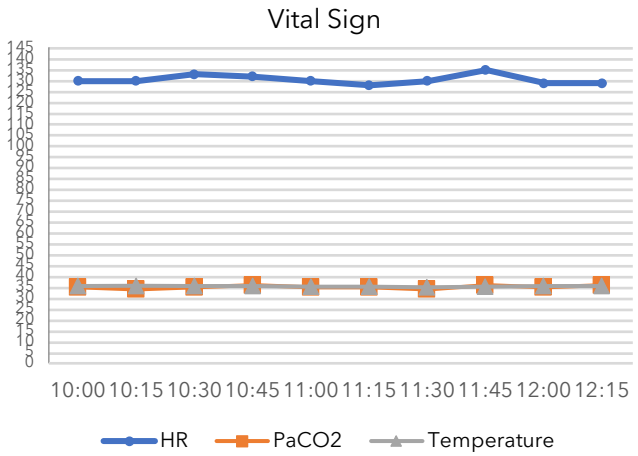

Figure 2. Vital Sign Monitoring During Surgery

The Evacuation Craniotomy was undergo as planned with administered 3x40 cc PRC, NaCl 0,9% 20 cc, Mannitol 100cc, Tranexamic Acid 50mg, Continuous Propofol 50 µg/kg body weight/min for maintaining brain reflex and Paracetamol 8,8 cc while surgery. During surgery there is blood lost around 120 cc and urine production of 2,2 ml/kg body weight. The Patient was admitted to PICU after surgery and the vital signs of HR, RR, and SpO_2 are 138 times/minute, 25 times/minute, and 99% (AC VC VT 30 RR PEEP 5 FiO_2 60% respectively. After the surgery, patients administered paracetamol 60mg every 8 hours as analgetic. We also conduct post-operative laboratory testing as follows:

Table 2. Postoperative Blood Test

Parameter	Value
Hemoglobin	11,1
Leukocyte	14.460
Thrombocyte	571.000
Hematocrit	33,6
Prothrombin Time	17,1
Activated Partial Thromboplastin Time	27,4
International Normalized Ratio	1,22
Albumin	2,6
AST	44
ALT	32
Urea N	23
Creatinine	0,31

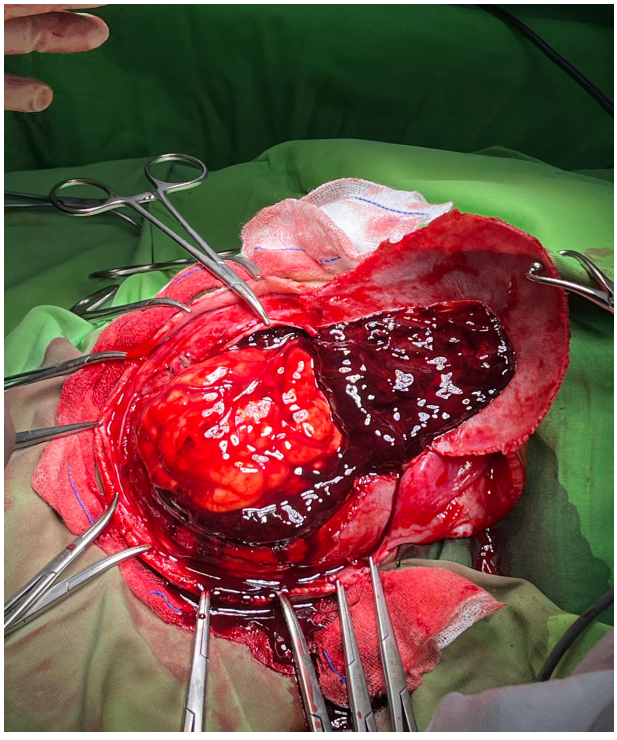

Figure 3. Intraoperative finding of subdural hematoma

Discussion

Acquired prothrombin complex deficiency (APCD) is an uncommon yet severe hemorrhagic condition that presents considerable difficulties in anesthesia management. This syndrome is marked by a lack of vitamin K-dependent coagulation factors, resulting in extended bleeding durations and an elevated risk of hemorrhagic consequences.[5] Based on onset of disease, APCD classified into 3 groups, early (0-24 hours), classic (1-7 days), late (≥2 weeks). Incidence rate of late onset APCD as happens in this case varies in several countries as shown in **Table 3**.[4]

Table 3. Incidence Rate Late Onset APCD[4]

Country	Incidence Rate Late Onset APCD (per 100.000 birth)
United Kingdom	0,1
Canada	0,37
New Zealand	0,16
Netherland	1,2
Germany	0,44
Australia	1,5
Switzerland	0,87
Sweden	6
Thailand	2,8
Japan	1,9

In early-onset APCD, there is often a transfer of maternal components that disrupts the metabolism and function of vitamin K through the placenta; this is related to the consumption of certain medications by the mother. The occurrence of early-onset APCD is also associated with newborns who do not receive vitamin K prophylaxis, with an incidence rate of 6-12%. The cause of classical APCD in the first week of life is generally idiopathic. Classic APCD is associated with physiological vitamin K deficiency at birth, accompanied by relatively low levels of vitamin K in breast milk. Bleeding due to late-onset vitamin K deficiency generally occurs between 2 weeks and 6 months of life, although it can happen at any age; usually due to other conditions (secondary) that cause decreased absorption or production of vitamin K, such as celiac disease, ulcerative colitis, and gastrocolic fistula leading to vitamin K malabsorption, or due to the use of broad-spectrum antibiotics or long-term sulfonamides that alter gut flora, resulting in decreased vitamin K production. Bleeding in APCD can be spontaneous or due to trauma, such as cephalhematoma during the birth process. Massive bleeding can cause hemodynamic disturbances, shock, and even anemia. If intracranial bleeding occurs, the baby may experience decreased consciousness or

other neurological disturbances depending on the location of the bleeding in the brain.[4] The management of APCD requires a multidisciplinary approach, with anesthesia playing a critical role in both surgical and nonsurgical interventions.

Preoperative Assessment and Preparation

Diagnosis and Risk Stratification The diagnosis of APCD is based on laboratory findings, including prolonged prothrombin time (PT) and international normalized ratio (INR), as well as clinical evidence of bleeding. In cases of intracranial hemorrhage, imaging studies such as CT scans are essential for assessing the severity of bleeding and guiding management. Preoperative assessment should also include a thorough evaluation of the patient's coagulation profile, including fibrinogen levels, platelet count, and markers of fibrinolysis.[6–8]

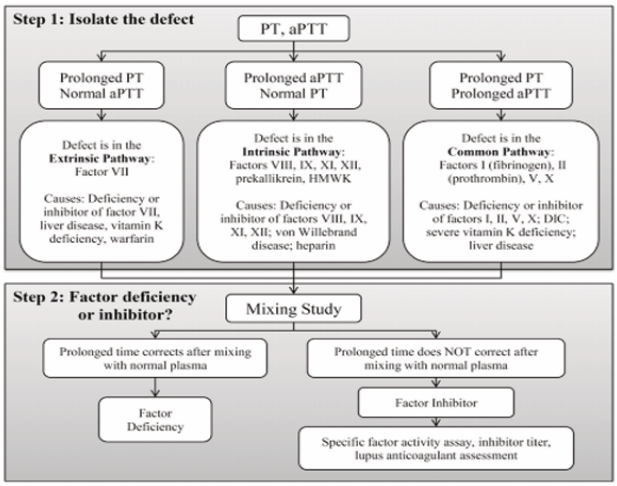

Figure 3. Coagulopathy evaluation.[9]

The first step: isolating the coagulation pathway. Elongated prothrombin time (PT) indicates a defect in the extrinsic pathway, and elongated activated partial prothrombin time (aPTT) indicates a defect in the intrinsic pathway. When PT and aPTT are prolonged, they indicate defects in both pathways. The second step: determine the deficiency factor or inhibitor factor. If the elongation time is corrected by mixing with normal plasma, then there is a deficiency factor. If the elongation of time cannot be corrected by normal plasma mixing, it means there are inhibitor factors and specific tests must be done.[9]

The cornerstone of preoperative management in APCD is the correction of coagulopathy. Vitamin K administration is the primary treatment, as it reverses the deficiency of vitamin K-dependent clotting factors. However, the response to vitamin K may take several days, and in cases of severe bleeding, additional interventions such as fresh frozen plasma (FFP) or clotting factor concentrates may be required.[6,7,10]

FFP should be administered within dose of 10–15 ml per kg in infants with extensive bleeding while increasing vitamin K dependent coagulation factor by 0.1–0.2 unit per ml. The treatment outcome is expected within 4 – 6 hours after the injection, which is marked by the cessation of bleeding and improved coagulation parameter. If no improvement happens within 24 hours, other causes should be suspected.[11]

Point-of-Care Coagulation Monitoring Conventional coagulation tests such as PT and INR have limitations in assessing the complexity of coagulopathy in APCD. Viscoelastic hemostatic assays, such as rotational thromboelastometry (ROTEM) and thromboelastography (TEG), provide more detailed information on clot formation, strength, and fibrinolysis. These point-of-care tests are particularly useful in guiding goal-directed therapy and reducing the need for allogeneic blood product transfusions.[7,8,10]

Intraoperative Anesthesia Management Choice of Anesthetic Technique

The choice of anesthetic technique depends on the patient's clinical condition, the nature of the surgical procedure, and the severity of coagulopathy. General anesthesia is often preferred in cases requiring surgical intervention, as it provides optimal control of the airway and hemodynamic stability. However, regional anesthesia may be considered in selected cases where the risk of bleeding is minimized.[6,12]

Sevoflurane, propofol, and sodium thiopental, favored for induction, are commonly used drugs in pediatric neuroanesthesia. The administration of these medications in our study was structured according to the patient's age and surgical characteristics. In accordance with the literature, sevoflurane was favored for the induction of patients lacking vascular access, especially within the neonatal cohort, while propofol was preferred for patients whose airway management utilized a laryngeal mask airway (LMA), as it more effectively inhibited upper airway reflexes compared to alternative anesthetic agents. Sevoflurane has been shown to have little impact on cerebral blood flow in young patients, akin to adults, making it the optimal inhalation anesthetic for neuroanesthesia. In elective craniotomies, propofol was shown to reduce intracranial pressure while simultaneously elevating cerebral perfusion pressure in comparison to inhalation anesthesia. Consequently, the injection of propofol would be advantageous, especially in

instances of elevated intracranial pressure and midline shift.[13]

Propofol have a rapid onset and are short acting, which allows for rapid recovery post-surgery and evaluation of neurological function. It also decreases intracranial pressure, cerebral blood flow, cerebral metabolism, and edema while supporting cerebral perfusion pressure and mean arterial pressure. This aggregate of effects is neuroprotective during cerebral ischemia. Due to propofol's rapid onset and short duration, infusion rates can be adjusted to allow for patient cooperation when necessary, during a procedure.[14]

Hemodynamic Stability and Blood Loss Management Maintaining hemodynamic stability is critical during surgery. Anesthesiologists should monitor for signs of hypovolemia and ensure adequate fluid resuscitation. In cases of significant blood loss, the use of blood product transfusions, including red blood cells, platelets, and clotting factor concentrates, may be necessary. The administration of antifibrinolytic agents such as tranexamic acid may also be beneficial in reducing bleeding.[7,8,10]

Hyperosmotic fluids are used as resuscitation fluids in hemorrhagic hypovolemia and as osmotherapeutic agents to reduce brain edema or elevated ICP. It has more rapid resuscitation with smaller infused volume, improving cardiac output, decreasing peripheral resistance, and lowering the ICP.[15] A prospective multi center cohort study of children with moderate to severe TBI reveals that the use of hypertonic saline was not associated with increased survival or improved functional outcomes compared with mannitol. Therefore, both agents could be equally effectively used singly in the management of elevated ICP.[16]

Temperature and Acid-Base Balance Hypothermia and acidosis can exacerbate coagulopathy and increase the risk of bleeding. Active measures to maintain normothermia and correct acidosis are essential during surgery. The use of warming blankets and the administration of bicarbonate may be required in cases of severe acidosis.[8]

Postoperative Care and Monitoring

Pediatric Intensive Care Unit (PICU) Admission Patients with severe APCD or significant intraoperative bleeding should be admitted to PICU for close monitoring. ICU care includes continuous monitoring of vital signs, coagulation parameters, and neurological status in cases of intracranial hemorrhage.[6,17]

Coagulation Profile Monitoring Postoperative monitoring of the coagulation profile is essential to ensure that coagulopathy has been adequately corrected. Regular measurements of PT, INR, fibrinogen, and platelet count are recommended. Viscoelastic hemostatic assays may also be used to guide further transfusion or therapeutic interventions.[7,10]

Effective pain management is critical in the postoperative period to minimize stress and prevent bleeding complications. A multimodal approach, including the use of nonsteroidal anti-inflammatory drugs (NSAIDs) and opioids, may be employed. However, the use of NSAIDs should be avoided in patients with platelet dysfunction or ongoing bleeding.[8]

Special Considerations in Neurosurgical Cases

Intracranial Hemorrhage Management In cases of intracranial hemorrhage, surgical intervention may be required to evacuate the hematoma and relieve intracranial pressure. The decision to operate depends on the size and location of the hematoma, as well as the patient's neurological status.[6,18]

Neuroprotective Strategies Anesthetic management in neurosurgical cases should include neuroprotective strategies to minimize brain injury. These strategies include maintaining optimal cerebral perfusion pressure, avoiding hyperglycemia, and providing adequate oxygenation.[8]

Coagulation Management in Neurosurgery The management of coagulation in neurosurgical patients requires a balanced approach to prevent both bleeding and thromboembolic complications. The use of viscoelastic hemostatic assays can help guide the administration of blood products and clotting factors concentrate.[10,18]

Personalized Approach to Anesthesia Management

Individualized Coagulation Therapy A personalized approach to coagulation management is essential in APCD. This involves tailoring therapy to the patient's specific coagulation profile and clinical condition. The use of point-of-care testing and viscoelastic hemostatic assays can help guide therapy and reduce complications.[7,19] Patient Blood Management Patient blood management strategies, including the use of blood product transfusions and clotting factor concentrates, should be based on the results of coagulation testing. This approach can help reduce the risk of allogeneic blood product transfusions and improve clinical outcomes.[7,10]

Multidisciplinary collaboration effective anesthesia management in APCD requires collaboration between anesthesiologists, surgeons, hematologists, and other healthcare professionals. A multidisciplinary approach

ensures that all aspects of the patient's care are coordinated and optimized.[17,18]

Conclusion

Anesthesia management in acquired prothrombin complex deficiency is a complex and challenging task that requires a comprehensive understanding of the underlying coagulopathy and its implications for surgical and anesthetic care. By leveraging advanced diagnostic tools, personalized treatment strategies, and multidisciplinary collaboration, anesthesiologists can improve clinical outcomes and reduce the risk of complications in patients with APCD

Ethics approval

The Ethical approval is not required.

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All authors equally contributed to case identification, manuscript drafting, and revision.

Competing interests

All the authors declare that there are no conflicts of interest.

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Underlying data

Derived data supporting the findings of this study are available from the corresponding author on request.

Declaration of artificial intelligence use

We hereby confirm that no artificial intelligence (AI) tools or methodologies were utilized at any stage of this study, including during data collection, analysis, visualization, or manuscript preparation. All work presented in this study was conducted manually by the authors without the assistance of AI-based tools or systems.

References

1. Tertia C, Yoesdyanto K, Irfani I, Sembiring H. Laporan kasus: Perdarahan intraserebral pada acquired prothrombin complex deficiency onset lambat. *Callosum Neurology*. 2018;1(2):67-72.
2. Hidayat I. Acquired Prothrombin Complex Deficiency. *International Journal of Clinical Pediatrics and Child Health*. 2019;1(1):28-33.
3. 3.Kher P, Verma RP. Hemorrhagic disease of newborn. Published online 2020.
4. Linardi JI. Perdarahan Neonatus Akibat Defisiensi Vitamin K: Diagnosis, Tata Laksana, dan Pencegahan. *Cermin Dunia Kedokteran*. 2022;49(1):10-13.
5. Utomo A, Parenrengi M, Bintoro S. A case report of Acquired Prothrombin Complex Deficiency in Infant. *International Journal of Research Publications*. 2023;117. doi:10.47119/IJRP1001171120234418
6. Dewi LP, Nurfitri E, Evodia E, Romli MT. Good outcomes in operative management of acquired prothrombin complex deficiency: a serial case report. *Paediatr Indones*. 2011;51(5):298-302.
7. Biancofiore G. Perioperative coagulation management and point-of-care monitoring in anesthesia. *Practical Trends in Anesthesia and Intensive Care 2018*. Published online 2019:3-19.
8. Kwon MA, Ji SM. Revolutionizing trauma care: advancing coagulation management and damage control anesthesia. *Anesth Pain Med (Seoul)*. 2024;19(2):73-84.
9. Hidayat I. Acquired Prothrombin Complex Deficiency. *International Journal of Clinical Pediatrics and Child Health*. 2019;1(1):28-33.
10. Bolliger D, Tanaka KA. Point-of-care coagulation testing in cardiac surgery. In: *Seminars in Thrombosis and Hemostasis*. Vol 43. Thieme Medical Publishers; 2017:386-396.
11. Widodo HP, Adji NK. Acquired prothrombin complex deficiency. *Indonesian Journal of Neurosurgery*. 2021;4(2).
12. Cherenfant C, Umeh U. Coagulation and Regional Anesthesia. *Essentials of Blood Product Management in Anesthesia Practice*. Published online 2021:423-431.
13. Emmez G, İnan G, Pampal HK, et al. Pediatric neuroanesthesia experiences: A single center retrospective cohort study: Neuroanesthesia in pediatric patients. *Journal of Surgery and Medicine*. 2023;7(3):214-219.
14. Nguyen A, Mandavalli A, Diaz MJ, et al. Neurosurgical anesthesia: optimizing outcomes with agent selection. *Biomedicines*. 2023;11(2):372.
15. Cottrell JE, Patel P. *Cottrell and Patel's Neuroanesthesia E-Book*. Elsevier; 2023. https://books.google.co.id/books?id=k5rxEAAAQBAJ
16. Chong SL, Zhu Y, Wang Q, et al. Clinical outcomes of hypertonic saline vs mannitol treatment among children with traumatic brain injury. *JAMA Netw Open*. 2025;8(3):e250438-e250438.
17. Levi M, Sivapalaratnam S. Hemostatic abnormalities in critically ill patients. *Intern Emerg Med*. 2015;10:287-296.
18. Germans MR, Rohr J, Globas C, et al. Challenges in Coagulation Management in Neurosurgical Diseases: A Scoping Review, Development, and Implementation of Coagulation Management Strategies. *J Clin Med*. 2023;12(20):6637.
19. Dabbagh A. Personalized Anesthesia and Perioperative Coagulation Management: a Main Step Forward. *Journal of Cellular & Molecular Anesthesia*. 2019;4(4):105-106.