CASE STUDY

CONTENTS

An anesthesiological complication related to bone cement use during reconstructive osteoplasty using the Masquelet technique in a pediatric patient: A case report

Volodymyr Mishchuk
DANYLO HALYTSKY LVIV NATIONAL MEDICAL UNIVERSITY, LVIV, UKRAINE

ABSTRACT

A 16-year-old male (ASA II, BMI 31) underwent reconstructive osteoplasty with insertion of a polymethylmethacrylate (PMMA) spacer. Shortly after cement application, he developed sudden hypotension, tachycardia, and decreased end-tidal CO_2, consistent with Grade II BCIS. Supportive management with fluids, ketamine, and norepinephrine stabilized the patient. Postoperatively, he experienced recurrent hypotension, acute kidney injury, and elevated liver enzymes and creatine kinase. Intensive care treatment resulted in recovery, and he was discharged from the ICU on day five.

Bone cement implantation syndrome, though exceptionally uncommon in pediatric patients, may develop during orthopedic procedures involving polymethylmethacrylate. Early recognition, continuous anesthetic vigilance, and timely multidisciplinary intervention are crucial to reduce morbidity and improve perioperative outcomes.

KEY WORDS: bone cements, anesthesia, hypotension, acute kidney injury, vasopressors, creatine kinase

Wiad Lek. 2026;79(7):1709-1714. doi: 10.36740/WLek/222248 DOI

INTRODUCTION

The use of bone cement (BC), based on polymethyl methacrylate, is widely adopted in orthopedic practice for procedures such as fracture repair and joint arthroplasty. Bone cement facilitates the stabilization and reinforcement of the fracture site, thereby promoting bone healing and functional recovery [1]. Despite the favorable physical properties of BC and its undeniable benefits in orthopedic reconstructive surgery, its use has been associated with a number of complications that may lead to sudden clinical deterioration and even death [2-5].

One of the most severe complications related to bone cement use is Bone Cement Implantation Syndrome — a critical and potentially life-threatening condition that typically occurs during cementation and is characterized by a constellation of clinical features, including hypotension, hypoxemia, cardiac arrhythmias, elevated pulmonary vascular resistance, and, in some cases, cardiac arrest. BCIS usually manifests shortly after cement injection but may also occur in the early postoperative period. The pathophysiology is primarily attributed to the exothermic nature of the cementing process, which leads to local tissue injury, histamine and prostaglandin release, and the formation of microemboli. These events trigger a systemic inflammatory response that may culminate in respiratory and cardiovascular failure [1,2,6].

BCIS most frequently occurs during hip arthroplasty and is closely associated with increased intramedullary pressure following cement injection. However, cases have also been reported during knee and shoulder arthroplasty [7,8].

To assess the severity of BCIS, the grading system proposed by Donaldson et al. is commonly used, which is based on hemodynamic and oxygenation parameters [2]:

Grade 1: Oxygen saturation (SpO_2) < 94% or >20% decrease in systolic blood pressure (SBP);

Grade 2: SpO_2 < 88%, >40% decrease in SBP, or sudden loss of consciousness;

Grade 3: Cardiovascular collapse requiring cardiopulmonary resuscitation.

As for the incidence of BCIS in pediatric patients, only a few isolated reports have been published to date regarding this serious complication [9,10]. We report a clinical case of BCIS in a 16-year-old male patient that occurred intraoperatively and continued into the early postoperative period following orthopedic surgery involving the use of bone cement.

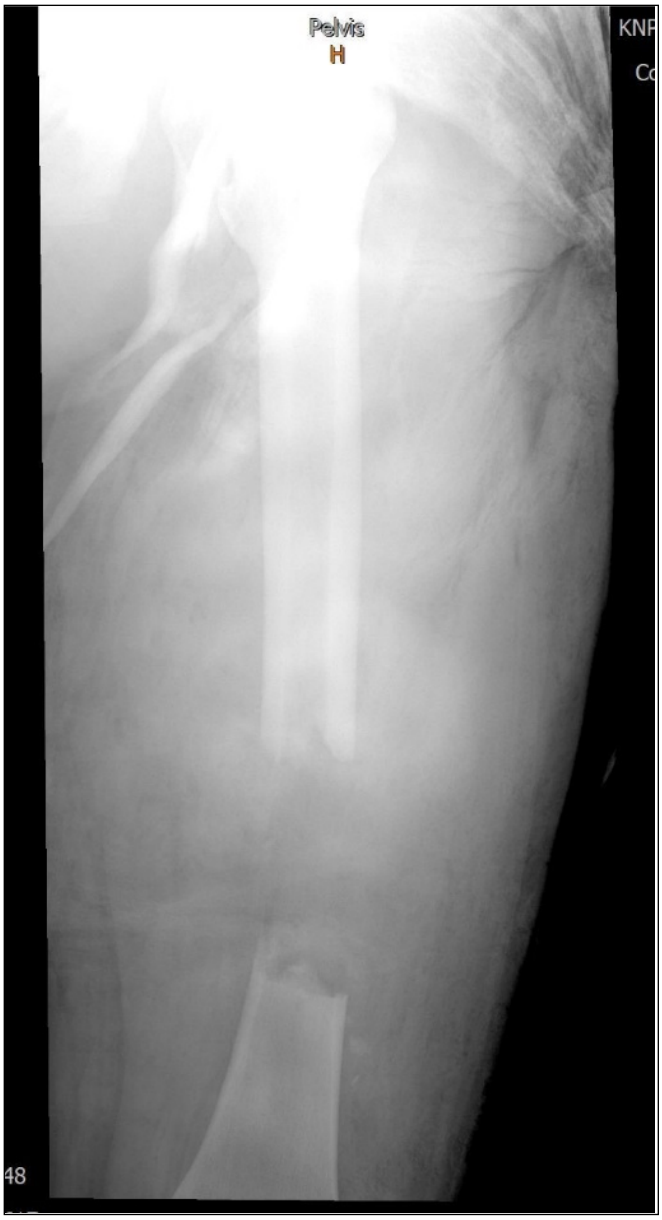

Fig. 1. Initial segmental femoral bone defect following trauma
Picture taken by the authors

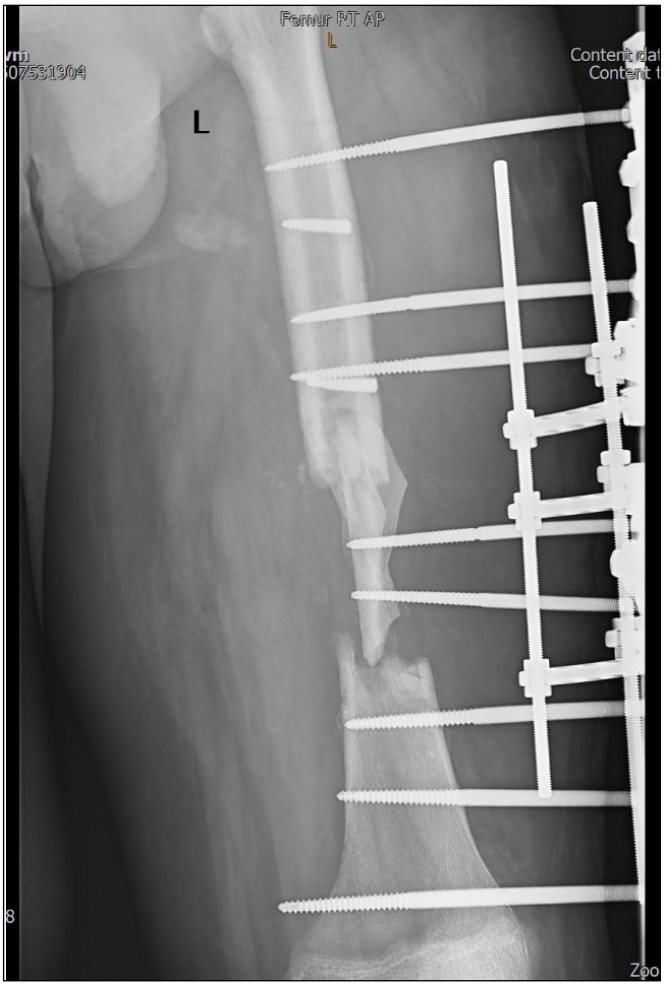

Fig. 2. External fixation and metal osteosynthesis of the femoral fragments
Picture taken by the authors

CASE REPORT

A 16-year-old male pediatric patient with a body mass index (BMI) of 31 presented without significant comorbidities. He was classified as American Society of Anesthesiologists (ASA) Physical Status II. The patient was scheduled for the first stage of surgical treatment using the induced membrane technique (Masquelet technique) to manage a large segmental defect of the femoral shaft.

The patient's history was notable for a high-energy road traffic accident that resulted in an open fracture of the femur with substantial bone loss (Fig. 1). Emergency surgical management was performed, consisting of metal osteosynthesis of the femoral fragments and application of an external fixation device (Fig. 2). Due to delayed bone healing and insufficient incorporation of the autologous bone graft, the first stage of the Masquelet technique was carried out as part of a staged reconstructive approach (Fig. 3).

Course of Anesthesia: anesthesia was induced with intravenous propofol, fentanyl, and atracurium. Endotracheal intubation was performed, followed by mechanical ventilation in pressure-controlled mode (PCV) with the following parameters: FiO_2 50%, peak inspiratory pressure (PIP) 12 cm H_2O, positive end-expiratory pressure (PEEP) 4 cm H_2O, respiratory rate (RR) 15 breaths/min, inspiratory-to-expiratory ratio (I:E) 1:2, and tidal volume (TV) 570 mL. An epidural catheter was inserted at the L2–L3 level. Epidural anesthesia was achieved with 20 mL of 0.25% bupivacaine. Anesthesia was maintained with a continuous propofol infusion at 6 mg/kg/h in combination with epidural anesthesia and intermittent boluses of fentanyl for analgesic augmentation. Tranexamic acid (1 g) was administered intraoperatively to reduce blood loss. Antibiotic prophylaxis was given to prevent

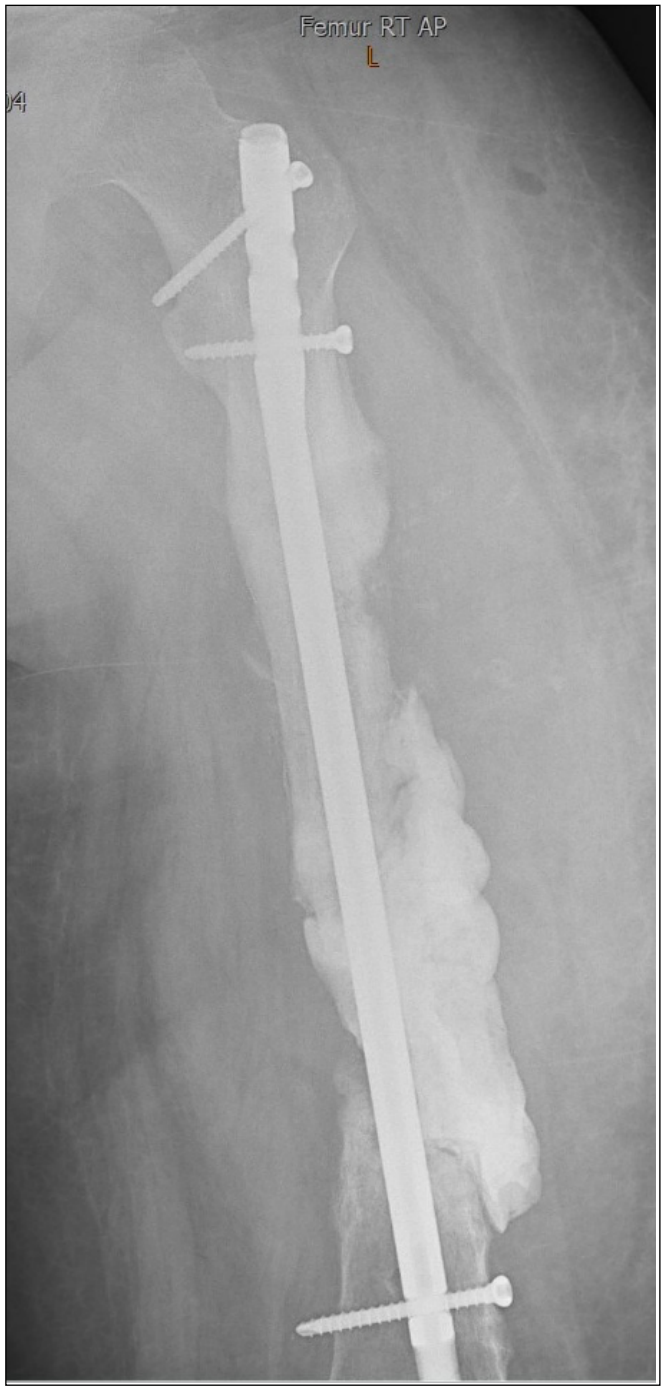

Fig. 3. First stage of reconstruction using the Masquelet technique
Picture taken by the authors

surgical site infections. Intravenous fluid therapy was performed using balanced crystalloids, titrated according to physiological requirements and estimated intraoperative fluid and blood loss.

Intraoperative Monitoring and Complication: vital signs remained stable until the use of bone cement. Baseline monitoring parameters included: SpO_2 97–98%, end-tidal CO_2 ($EtCO_2$) 40–44 mmHg, heart rate (HR) 68–77 bpm, blood pressure (BP) 135/78 mmHg, mean arterial pressure (MAP) 95 mmHg, and core temperature 36.3°C. At the third hour of surgery, shortly after bone cement application, the patient experienced an abrupt hemodynamic deterioration consistent with bone cement implantation syndrome: BP dropped to 62/34 mmHg (MAP 43 mmHg), HR increased to 115 bpm, and $EtCO_2$ decreased to 26 mmHg. A 500 mL bolus of Ringer's lactate was administered, resulting in partial hemodynamic improvement: BP 90/55 mmHg, MAP 66 mmHg. However, hypotension persisted (BP 84/52 mmHg, MAP 62 mmHg). The propofol infusion was reduced to 4 mg/kg/h, and intermittent ketamine boluses (100 mg every 30 minutes) were initiated. A norepinephrine infusion was started at 0.1 µg/kg/min. Blood pressure stabilized in the range of 92/63 to 98/65 mmHg. The total duration of MAP < 60 mmHg was approximately 27 minutes.

By the end of the surgery, the patient was hemodynamically stable with the following parameters: SpO_2 97–98%, $EtCO_2$ 40 mmHg, HR 87 bpm, BP 105/68 mmHg, MAP 79 mmHg, and temperature 36.1°C. The patient was extubated in the operating room and transferred to the pediatric intensive care unit (PICU). Total intraoperative fluid administered: 4300 mL over 7 hours. Estimated blood loss: ~1600 mL. Packed red blood cell (PRBC) transfusion: 1300 mL

Postoperative Period: upon admission to the PICU, the patient developed recurrent hypotension: BP 55/35 mmHg, MAP 39 mmHg, SpO_2 dropped to 88–90%, HR increased to 115 bpm. Norepinephrine was increased to 0.15 µg/kg/min, and supplemental oxygen (10 L/min) was administered via face mask. Hemodynamic stabilization occurred within 2 hours. Norepinephrine infusion was continued for a total duration of 14 hours and then discontinued. Oxygen therapy was ceased 12 hours postoperatively. On postoperative day 1, the patient exhibited oliguria (urine output 0.36 mL/kg/h) and laboratory findings consistent with acute kidney injury (AKI). Additionally, there were significantly elevated levels of AST, ALT, and creatine kinase (CK). Postoperative laboratory results are summarized in Table 1.

Over the following three days, the patient maintained stable hemodynamic and respiratory parameters, and urine output returned to normal. A regression of renal insufficiency was observed, along with a decline in serum levels of creatine kinase, AST, and ALT. On postoperative day five, the patient was transferred from the intensive care unit to the general ward in a satisfactory condition.

To date, complications associated with the use of bone cement are well-documented in the literature. Although the first report of bone cement implantation

Table 1. Laboratory values during the postoperative period

Parameter (Reference range)	Postoperative time (h)							
	12	24	36	48	60	72	96	120
Urea (2.5–8 mmol/L)		9	8.37		7.8		7.1	7.5
Cr (62–115 μmol/L)		227	243		146		127	115
K^+ (3.5–5.0 mmol/L)		5.2	4.3	4.5	4.8		3.8	4.2
AST (0–47 U/L)			599	507		234		144
ALT (0–37 U/L)			135	132		123		115
CK (<270 U/L)				32628				12838
CRP (<5 mg/L)		145		150				28
Ph (7,35-7,45)	7.24	7.33	7.4					
PaO_2 (80–100 mmHg)	59	88	101					
$PaCO_2$ (35–45 mmHg)	34	37	37					
BE N (±2 mmol/L)	-11	-5	-3					
APTT (25–35 s)	47	50	46		45		39	41
INR (0.8–1.2)	1.4	1.5	1.3		1.3		1.0	1.1

Source: compiled by the authors of this study

syndrome (BCIS) was published by Powell J.N et al. in 1970, there remains considerable variation in the reported incidence and associated mortality. This is primarily due to the lack of a standardized diagnostic approach and limited clinical awareness. Furthermore, in earlier studies, BCIS was often misclassified as pulmonary embolism [1,11].

The wide variability in BCIS incidence also stems from the heterogeneity of clinical presentations, which depends on the severity of the syndrome, as described by Donaldson A,J et al. [2]. Reported incidence rates of BCIS in the literature range from 28% to 61.5%, largely due to the absence of a universally accepted definition [12,13]. In a study by Weingärtner K involving 208 patients, 77 (37.0%) developed BCIS. Of these, 53 (25.5%) had Grade I, 17 (8.2%) had Grade II, and 7 (3.4%) had Grade III BCIS [6].

Several theories have been proposed to explain the pathogenesis of BCIS, including embolic, allergic, and toxic responses to polymethylmethacrylate. However, none of these hypotheses fully accounts for the complex and varied clinical manifestations of BCIS, which are more likely to arise from a multifactorial interaction of these mechanisms [1].

BCIS is most commonly associated with cemented arthroplasty procedures of the hip and knee joints, and less frequently with shoulder arthroplasty. Many authors attribute this to the elevated intramedullary pressure that occurs during cement insertion [1,6]. In contrast, the Masquelet technique does not involve increased intramedullary pressure, as the cement spacer is implanted into a bone defect. Its primary purpose is to induce and maintain a biological membrane that facilitates subsequent bone regeneration [14,15].

In the presented case, the acute clinical deterioration following cement application was likely triggered by the exothermic reaction of PMMA affecting adjacent tissues, resulting in the release of histamine, prostaglandins, and other systemic inflammatory mediators, which led to hypotension. Of particular concern was the recurrent episode of hypotension in the postoperative period, suggestive of a prolonged systemic inflammatory response to PMMA.

Also noteworthy was the marked elevation in creatine kinase levels, which may indicate secondary skeletal muscle injury due to the thermal effect of PMMA and cytokine cascade activation. Several clinical and experimental studies have reported similar elevations in CK, ALT, and AST following bone cement use [16,17]. Alternatively, these laboratory abnormalities may have been partially attributable to surgical trauma and transient hypoperfusion of muscle tissue during the intraoperative period [18,19].

The development of acute kidney injury (AKI) in the early postoperative period was likely a consequence

of transient hemodynamic instability. Fortunately, it resolved rapidly following the restoration of effective circulation.

This case of BCIS in a pediatric patient undergoing orthopedic surgery using the Masquelet technique is exceedingly rare in our experience. This may be explained by the generally lower prevalence of comorbid risk factors and the superior hemodynamic compensatory capacity in children compared to adult patients.

CONCLUSIONS

Bone cement implantation syndrome is a multifactorial complication most frequently encountered in elderly patients undergoing cemented hip arthroplasty. Its occurrence in pediatric patients is exceptional. Nevertheless, even rare complications such as BCIS in children warrant further investigation and the development of safety guidelines to improve perioperative outcomes.

REFERENCES

1. Yang Y, Meng X, Huang Y. Study of the cement implantation syndrome: A review. Medicine (Baltimore). 2024;103(24):e38624. doi: 10.1097/MD.0000000000038624. DOI
2. Donaldson AJ, Thomson HE, Harper NJ et al. Bone cement implantation syndrome. BJA. 2009;102:12–22. Br J Anaesth. 2009;102(1):12-22. doi: 10.1093/bja/aen328. DOI
3. Zhang M, Liu P. Progress of research on pathogenesis and diagnosis and treatment of bone cement implantation syndrome. Shandong Med. 2020;60:4.
4. Al-Husinat L, Jouryyeh B, Al Sharie S et al. Bone Cement and Its Anesthetic Complications: A Narrative Review. J Clin Med. 2023;12(6):2105. doi: 10.3390/jcm12062105. DOI
5. Mudgalkar N, Ramesh KV. Bone cement implantation syndrome: A rare catastrophe. Anesth Essays Res. 2011;5(2):240-2. doi: 10.4103/0259-1162.94796. DOI
6. Weingärtner K, Störmann P, Schramm D et al. Bone cement implantation syndrome in cemented hip hemiarthroplasty - a persistent risk. Eur J Trauma Emerg Surg. 2022;48(2):721-729. doi: 10.1007/s00068-020-01587-8. DOI
7. Rassir R, Schuiling M, Sierevelt IN et al. What Are the Frequency, Related Mortality, and Factors Associated with Bone Cement Implantation Syndrome in Arthroplasty Surgery? Clin Orthop Relat Res. 2021;479(4):755-763. doi: 10.1097/CORR.0000000000001541. DOI
8. Humez M, Kötter K, Skripitz R, Kühn KD. Evidence for cemented TKA and THA based on a comparison of international register data. Orthopadie (Heidelb). 2024;53(8):597-607. doi: 10.1007/s00132-024-04489-4. DOI
9. Tansey PJ, Wainwright JD, Johnson BA, Montgomery NI. Intraoperative Bone Cement Implantation Syndrome in a Pediatric Patient: A Case Report. JBJS Case Connect. 2024;14(4). doi: 10.2106/JBJS.CC.24.00390. DOI
10. Ramanathan S, Vora T, Gulia A et al. Pulmonary cement embolism in a child following total elbow replacement for primitive neuroectodermal tumour (PNET) of the humerus. Skeletal Radiol. 2017;46(5):715-718. doi: 10.1007/s00256-017-2602-0. DOI
11. Powell JN, McGrath PJ, Lahiri SK et al. Cardiac arrest associated with bone cement. Br Med J. 1970;3(5718):326. doi: 10.1136/bmj.3.5718.326. DOI
12. Hagio K, Sugano N, Takashina M et al. Embolic events during total hip arthroplasty. An echocardiographic study. J Arthroplasty. 2003;18(2):186–92. doi: 10.1054/arth.2003.50027. DOI
13. Olsen F, Kotyra M, Houltz E, Ricksten S-E. Bone cement implantation syndrome in cemented hemiarthroplasty for femoral neck fracture, incidence, risk factors, and effect on outcome. Br J Anaesth. 2014;113(5):800–6. doi: 10.1093/bja/aeu226. DOI
14. Cho JW, Kent WT, Kim JK et al. Outcome of multi-staged induced membrane technique based on post-debridement cultures for the management of critical-sized bone defect following fracture-related infection. Sci Rep. 2022;12(1):22637. doi: 10.1038/s41598-022-26746-2. DOI
15. Lu Y, Lai CY, Lai PJ, Yu YH. Induced Membrane Technique for the Management of Segmental Femoral Defects: A Systematic Review and Meta-Analysis of Individual Participant Data. Orthop Surg. 2023;15(1):28-37. doi: 10.1111/os.13604. DOI
16. Matuszewski L, Matuszewska A, Jaszek M et al. The presence of pamidronate in bone cement affects serum biochemical markers in the rat. Bull Vet Inst Pulawy. 2014;58(2):295-299. doi:10.2478/bvip-2014-0045. DOI
17. Mouzopoulos G, Kouvaris C, Antonopoulos D et al. Perioperative creatine phosphokinase (CPK) and troponin I trends after elective hip surgery. J Trauma. 2007;63(2):388-93. doi: 10.1097/01.ta.0000241236.54304.a3. DOI
18. Tonotsuka H, Sugiyama H, Tanaka D et al. Postoperative creatine kinase elevation following hip arthroscopy and associated risk factors. Acta Orthop Traumatol Turc. 2019;53(6):397-401. doi: 10.1016/j.aott.2019.08.011. DOI
19. Cohen RG, Katz JA, Skrepnik NV. The relationship between skeletal muscle serum markers and primary THA: a pilot study. Clin Orthop Relat Res. 2009;467(7):1747-52. doi: 10.1007/s11999-009-0809-7. DOI

CONFLICT OF INTEREST
The Author declare no conflict of interest

CORRESPONDING AUTHOR
Volodymyr Mishchuk
Danylo Halytsky Lviv National Medical University
7 Chernihivska St., 79010, Lviv, Ukraine
e-mail: mishchukdoc@gmail.com

ORCID AND CONTRIBUTIONSHIP
Volodymyr Mishchuk: 0000-0003-0542-0891 A B D E F

A – Work concept and design, B – Data collection and analysis, C – Responsibility for statistical analysis, D – Writing the article, E – Critical review, F – Final approval of the article

RECEIVED: 18.08.2025
ACCEPTED: 20.05.2026