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Feasibility and Utility of Infrared Thermography for Intraoperative Temperature Monitoring in Cold Agglutinin Disease during Cardiopulmonary Bypass

Original Article

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ABSTRACT

Purpose: Cold agglutinin disease (CAD) is a rare autoimmune hemolytic disorder that poses significant challenges during cardiovascular surgery requiring cardiopulmonary bypass (CPB), owing to the risk of cold-induced red blood cell agglutination.

Methods: This study evaluated the utility of a noninvasive and easy-to-use infrared thermography device for intraoperative temperature assessment to address the risk of hypothermia-associated complications in CAD. Among 416 open-heart surgeries performed at our institution between September 2013 and December 2023, 5 cases (1.2%) involved CAD.

Results: Temperature control was tailored to each patient's thermal amplitude (19°C–26°C). CPB was conducted at systemic temperatures of 34°C–36°C, and cardioplegia was administered at 28°C–32°C, both above the agglutinin threshold. All 5 patients successfully underwent surgery and were discharged. Temperature management was particularly critical during sternotomy, CPB initiation, and cardioplegia delivery. In 4 cases, infrared thermography was used for monitoring of epicardial and cardioplegic temperatures, which was associated with an uneventful postoperative course. In contrast, 1 case without thermographic monitoring developed postoperative myocardial infarction and decreased cardiac function.

Conclusion: These findings suggest that noninvasive thermography may serve as a useful adjunct for real-time temperature surveillance during cardiac surgery in patients with CAD, providing information that is not captured by systemic temperature monitoring alone.

Keywords: cold agglutinin disease, cardiac surgery, infrared thermography, cardiopulmonary bypass

Introduction

Cold agglutinin disease (CAD) is a rare form of autoimmune hemolytic anemia mediated by cold-reactive immunoglobulin M (IgM) autoantibodies that bind to red blood cell (RBC) surface antigens at low temperatures, resulting in agglutination and complement-mediated hemolysis.[1)] Although CAD is uncommon in the general population, its clinical relevance becomes pronounced in the setting of cardiovascular surgery requiring cardiopulmonary bypass (CPB), where systemic hypothermia, cold cardioplegia, and extracorporeal circulation may precipitate catastrophic hematologic and microcirculatory complications.[1,2)]

During CPB, exposure of blood to nonphysiological surfaces, dilutional effects, and temperature manipulation collectively create an environment that can exacerbate cold agglutinin–mediated RBC aggregation. Even mild hypothermia may induce intravascular agglutination in

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susceptible patients, leading to hemolysis, impaired microcirculatory perfusion, myocardial ischemia, renal dysfunction, and neurologic injury.[3] Of particular concern is myocardial protection, as cardioplegia administration may induce localized myocardial cooling that is not adequately reflected by systemic core temperature monitoring.

The clinical impact of CAD in cardiac surgery is heterogeneous and depends largely on the cold agglutinin titer and, more importantly, the thermal amplitude—the highest temperature at which agglutination occurs.[2,3] Patients with low titers but high thermal amplitudes may still be at substantial risk during surgery. Consequently, perioperative management strategies have shifted away from reliance on antibody titers alone toward individualized thermal management based on thermal amplitude assessment and clinical history.[2]

Several strategies have been proposed to mitigate CAD-related risks during cardiac surgery, including avoidance of hypothermia, use of warm or tepid blood cardioplegia, perioperative plasma exchange, immunosuppressive therapy, and meticulous warming of all fluids and blood products.[3] However, despite these precautions, CAD-related complications continue to be reported, underscoring the difficulty of ensuring uniform temperature control at the tissue level—particularly at the myocardial surface and within the cardioplegia delivery system.

Traditional intraoperative temperature monitoring relies primarily on core measurements, such as nasopharyngeal, bladder, or pulmonary artery temperatures. While these measurements are useful for systemic management, they fail to capture spatial temperature heterogeneity, especially at exposed organs. The myocardial epicardial surface, which is directly exposed during sternotomy, may experience significant cooling despite normothermic core temperatures. Similarly, the temperature of the cardioplegia solution may decrease substantially between the heat exchanger and the point of coronary infusion, potentially dropping below the patient's thermal amplitude.[4,5]

Infrared thermography has emerged as a noninvasive imaging modality capable of providing real-time, two-dimensional visualization of surface temperature distribution. In cardiovascular surgery, thermography has been investigated primarily as a research tool for evaluating myocardial cooling and rewarming during cardioplegia.[6] However, its role as a practical intraoperative adjunct for real-time temperature surveillance—particularly in high-risk populations such as patients with CAD—remains underexplored.

At our institution, we encountered several cases of CAD in patients undergoing open-heart surgery requiring CPB. Based on a critical adverse event in an early case without thermographic monitoring, we implemented routine intraoperative infrared thermography to monitor myocardial and cardioplegia temperatures in subsequent CAD cases. The present study reports our institutional experience with 5 patients with CAD, focusing on the feasibility, clinical utility, and potential protective role of infrared thermography in optimizing intraoperative temperature management and preventing cold-induced myocardial injury.

Materials and Methods

This retrospective case series included all patients with confirmed CAD who underwent open-heart surgery requiring CPB at our institution between September 2013 and December 2023. Patients without CAD or with incomplete perioperative data were excluded. This retrospective study was approved by the Institutional Review Board of our institution, and written informed consent was obtained from all participants.

All 5 cases in this series underwent elective surgery for valvular heart disease, and all patients were evaluated preoperatively by the hematology department for cold agglutinin titer and thermal amplitude. Because the thermal amplitudes were relatively high (15°C–26°C) and none of the procedures required deep hypothermic circulatory arrest, we judged that setting the CPB and cardioplegia temperatures sufficiently above these thresholds would be adequate; therefore, plasma exchange or immunosuppressive therapy was not administered, and a temperature-based management strategy was selected instead.

During surgery, the operating room temperature was maintained at an elevated range of 24°C–28°C. Intravenous medications and blood products were prewarmed and administered via a hotline infusion system. To avoid cold-induced agglutination, the systemic temperature during CPB was increased from the usual 32°C to 34°C–36°C, and the temperature of the cardioplegic solution was increased from the conventional 20°C to 28°C–32°C. Core body temperature was monitored via tympanic temperature (our institution's standard site for temperature monitoring during CPB).

Myocardial protection was achieved using a blood cardioplegia protocol (microplegia) delivered via the MPS2 system (Quest Medical, Inc., Allen, Texas, USA). This system utilizes a 50-mL Arrest Agent Cassette (ARR) for the potassium solution and an Additive Cassette (ADD) for other adjunctive agents, which are mixed with oxygenated blood to formulate the cardioplegic solution.

The ARR was loaded with 2 mol/L potassium chloride (80 mEq/40 mL), whereas the ADD contained magnesium sulfate (40 mEq/40 mL), 2% lidocaine (100 mg/5 mL), nicorandil (5 mg/5 mL), and short-acting insulin (10 units). To induce cardioplegic arrest, potassium and additives were administered at concentrations of 22 and 18 mL/L, respectively (target potassium concentration: 26 mEq/L). After cardiac arrest, maintenance cardioplegia was administered at an ARR of 12 mEq/L and an ADD of 9 mL/L for 5 min (target potassium: 16 mEq/L). The infusion temperature was maintained at 28°C–32°C and

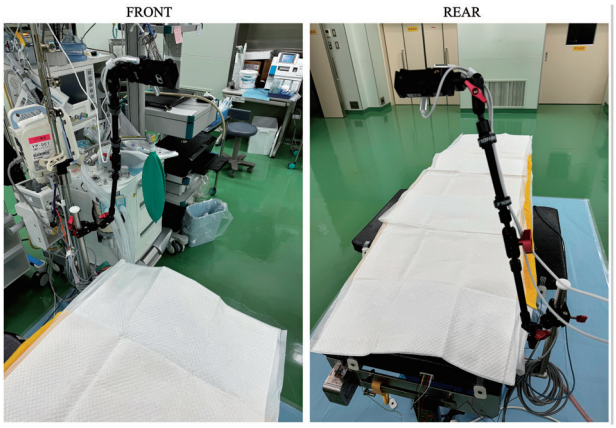


Fig. 1 Thermography camera setup.

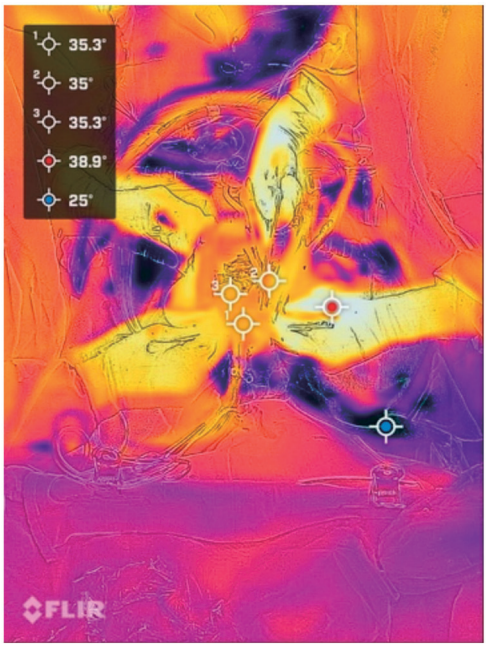


Fig. 2 Epicardial temperature.

delivered antegrade at a circuit pressure of <300 mmHg and a flow rate of 250–350 mL/min. For standard valve procedures (e.g., aortic valve replacement [AVR], mitral valve replacement [MVR], etc.), antegrade delivery alone is generally sufficient. Case 4, however, was a combined mitral valve replacement and tricuspid annuloplasty anticipated to require a relatively longer cross-clamp time; therefore retrograde delivery was added to achieve more uniform myocardial protection.

In Case 1, infrared thermography was not performed. In Cases 2–5, real-time temperature monitoring was performed using a noninvasive, compact infrared thermographic camera (FLIR ONE Pro; Teledyne FLIR LLC, Wilsonville, OR, USA) (**Fig. 1**). The device was positioned approximately 80 cm above the surgical field on the cranial side to evaluate the epicardial temperature (**Fig. 2**). In the initial version of the software, it was necessary to click on a specific area of the heart to display the temperature each time. For the purpose of objective evaluation, temperatures at 3 points were measured and averaged.

The thermography-guided intervention protocol was as follows: When the epicardial surface temperature (which was, on average, 3.1°C lower than the target systemic temperature after sternotomy, as shown by thermography) approached the patient's individual thermal amplitude, the epicardial surface was irrigated with warm saline, and the temperature rise was confirmed with thermography before proceeding with further surgical steps (e.g., initiation of CPB or cross-clamping). Because the cardioplegia line temperature immediately before administration was, on average, 11.4°C below the target systemic temperature due to residual priming solution in the circuit, the priming solution was continuously drained until thermography confirmed that the line/epicardial temperature had reached the target range (28°C–32°C)—only then was cardioplegia actually administered. In addition, the recalibration of the thermography device against the actual cardioplegia-line thermometer was performed whenever a discrepancy was detected.

Results

Between September 2013 and December 2023, 416 open-heart surgeries were performed at our institution. Among them, 5 patients (1.2%) were diagnosed with CAD. The average age was 74.6 years (range: 67–81 years), and 4 patients were male. The thermal amplitude of cold agglutinins in these cases ranged from 15°C to 26°C (**Table 1**).

All 5 patients underwent surgery for valvular heart disease. In all cases, systemic temperatures during CPB were maintained at 34°C–36°C. Similarly, the cardioplegia temperature was adjusted to 28°C–32°C, exceeding the thermal amplitude by >3°C in all cases (**Table 2**).

Among Cases 2–5, where infrared thermography was employed, the observed epicardial temperature was, on average, 3.1°C (range: 1.7°C–4.1°C) below the target systemic temperature, indicating that sternotomy alone resulted in notable surface cooling of the heart.

Furthermore, the actual temperature of the cardioplegia within the infusion line immediately before administration averaged 23.3°C (range: 21.5°C–25.9°C), approximately 11.4°C (range: 10.1°C–13.5°C) below the target systemic temperature, and significantly below the desired range of 28°C–32°C in all cases.

Postoperative outcomes were favorable in the 4 patients (Cases 2–5) whose epicardial temperatures were monitored using thermography, with no complications and successful discharge. In contrast, Case 1 (without thermographic monitoring) experienced a marked elevation in creatine kinase (CK 3025 U/L, creatine kinase-myocardial band [CK-MB] 288 U/L), and postoperative echocardiography revealed diffuse left ventricular dysfunction (decreased ejection

Table 1 Preoperative characteristics

Case	Diagnosis	Age	Gender	EF (%)	AR	Etiology of CAD	CA titer	CA temperature	Urgency of surgery	Plasma exchange	Thermography
1	AR, MR	77	Male	69	III	Idiopathic	256	25	Elective	No	Unused
2	AS, HOCM	71	Male	78	I	Idiopathic	512	26	Elective	No	Used
3	MR, TR, Paf	77	Female	61	—	Idiopathic	512	15	Elective	No	Used
4	IMR, TR	81	Male	36	—	Idiopathic	256	19	Elective	No	Used
5	AS	67	Male	62	—	Infection (*Mycoplasma pneumonia*)	512	19	Elective	No	Used

AR, aortic regurgitation; MR, mitral regurgitation; AS, aortic stenosis; HOCM, hypertrophic obstructive cardiomyopathy; TR, tricuspid valve regurgitation; Paf, paroxysmal atrial fibrillation; IMR, ischemic mitral regurgitation; EF, ejection fraction; CAD, cold agglutinin disease; CA, cold agglutinin

Table 2 Operative data

Case	Procedure	OT (min)	CCT (min)	Myocardial protection method	① Cardiopulmonary temperature (°C)	② Epicardial temperature (①–②)	③ Pre cardioplegia temperature (①–③)	④ Post cardioplegia temperature	Lowest temperature (tympanic)	CK (IU/L)	Catecholamine (γ)	EF (%)	Complication
1	DVR	383	169	Selective	34	?	?	?	34	3025	3	39	Pacemaker
2	AVR, myocardial resection	276	93	Ante	36	31.9 (−4.1)	25.9 (−10.1)	32	36	1031	—	73	—
3	MVR, TAP maze	325	136	Ante	34	29.9 (−4.1)	22.4 (−11.6)	28	34	1127	—	63	—
4	MVR, TAP	312	98	Ante, retro	35	33.3 (−1.7)	21.5 (−13.5)	28	35	766	3	33	—
5	AVR	403	120	Ante	34	31.6 (−2.4)	23.5 (−10.5)	28	34	440	—	62	—

①: blood delivery temperature, ②: heart surface temperature, ③: pre-drainage circuit temperature (including residual priming solution), ④: confirmed temperature immediately before administration (after priming-solution drainage and thermographic confirmation).

DVR, double-valve replacement; AVR, aortic valve replacement; MVR, mitral valve replacement; TAP, tricuspid annuloplasty; OT, operative time; CCT, cross-clamp time; CK, creatine kinase; EF, ejection fraction

fraction from 69% preoperatively to 39% postoperatively). Additionally, a 6.21-second pause was observed during sinus rhythm conversion following postoperative atrial fibrillation, necessitating the implantation of a DDD pacemaker. This finding does not directly confirm the microcirculatory injury hypothesis but is presented as part of the clinical picture reflecting the severity of myocardial injury.

Coronary angiography in this case revealed no new obstructive lesions compared with the preoperative study, suggesting that myocardial injury may have resulted from microcirculatory ischemia due to cold-induced agglutination of RBCs during cardioplegia administration. All 5 patients were ultimately discharged from ambulatory care.

Discussion

The present study highlights the critical importance of precise intraoperative temperature management in patients with CAD undergoing cardiac surgery with CPB. Our findings suggest that noninvasive infrared thermography provides valuable real-time information on epicardial and cardioplegia temperatures that cannot be captured by conventional systemic temperature monitoring alone.

A key consideration in cardiac surgery with CPB in patients with CAD is temperature management during both bypass and myocardial protection. Previous reports from Japan have indicated cold agglutinin reactivity at 22°C–25°C, CPB temperatures of 34°C–36°C, and cardioplegia temperatures of 32°C–36°C. Despite controversy regarding the safety of mild hypothermia and tepid cardioplegia, cardioplegia remains effective even at 32°C—the point at which myocardial oxygen consumption is approximately double that at 15°C.[4,5] Several Japanese studies have consistently reported maintaining cardioplegia temperatures within 32°C–36°C.[7–9] However, in cases requiring deep hypothermic circulatory arrest below the cold agglutinin thermal amplitude, temperature management alone may be insufficient, and preoperative plasma exchange should be considered.[9]

Historically, myocardial temperature has been measured using needle-type thermocouple probes inserted directly into the myocardium. Compared with this invasive technique, infrared thermography offers several advantages: (1) it is entirely noninvasive, avoiding the risk of myocardial trauma, local bleeding, or arrhythmia associated with needle insertion into a beating or recently arrested heart; (2) it provides two-dimensional, real-time spatial temperature mapping over the entire visible epicardial surface, rather than a single-point measurement, allowing detection of regional temperature heterogeneity (e.g., localized cold spots near the cardioplegia infusion site, as illustrated in **Fig. 2**) that a single needle probe would miss; (3) it requires no additional insertion time and carries no risk of introducing infection via a penetrating device, and the same camera can be freely repositioned to examine different anatomical regions or the cardioplegia delivery site as needed; and (4) it is comparatively inexpensive and technically simple to deploy and interpret intraoperatively.

Since May 2020, we have used a noninvasive infrared thermography device to intraoperatively monitor epicardial temperature.[10] This approach enables the real-time assessment of temperature changes, which is particularly critical in cases of CAD because of the risk of cold-induced myocardial infarction or hemolysis. Our findings underscore the importance of temperature monitoring at 3 key time points: sternotomy, CPB initiation, and the start of cardioplegia.

Infrared thermography, although easy to use, is susceptible to external interferences such as reflection and ambient radiation.[11] Therefore, the thermographic measurements were compared with the actual temperature of the cardioplegic circuit in the present study. When discrepancies were detected, recalibration was performed to ensure accuracy.

In Cases 2–5, which utilized thermography, the average actual epicardial temperature was 3.1°C lower than the target systemic temperature (**Table 2**, ①–②). These findings align with those of previous studies reporting that skin surface temperatures tend to drop below core temperatures, especially under ambient room temperatures of 25°C–34°C.[12] This result emphasizes the risk of cooling the myocardial surface simply through exposure during sternotomy, highlighting the need to prevent temperatures from falling below thermal amplitude thresholds. We addressed this issue by irrigating the heart surface with warm saline and monitoring it using thermography.

In addition, the temperature of the cardioplegia in the delivery line just before infusion (**Table 2**, ③) was, on average, 11.4°C lower than the target systemic temperature (**Table 2**, ①–③), and was significantly below the target cardioplegia range of 28°C–32°C. We ensured that the priming volume was drained until the cardioplegic solution reached the desired temperature (**Table 2**, ④), as confirmed by thermography before administration.

In contrast, Case 1 (without thermography) showed no new coronary artery disease on postoperative angiography but had elevated creatine kinase levels and a marked reduction in ejection fraction (69% preoperatively to 39% postoperatively). Since the standard cardioplegia technique and dosage were used without obvious technical errors, and because postoperative regional wall motion abnormalities did not improve despite resolution of mitral regurgitation, insufficient myocardial protection seemed unlikely. The patient's cold agglutinin thermal amplitude was 25°C, while the target CPB and cardioplegia temperatures were 34°C and 28°C, respectively. However, based on the thermographic trends observed in the other cases, the actual temperature of the cardioplegia before

injection might have dropped by 11.4°C—possibly to around 19.6°C—which is below the target, due to insufficient warming of the priming solution. This suggests that without thermographic confirmation, the operator may have judged the temperature subjectively (e.g., by touch), leading to the premature administration of cardioplegia at a temperature below the thermal threshold. Based on the clinical course and the absence of new epicardial coronary lesions, we hypothesize that cold-induced microcirculatory injury and myocardial infarction can occur. However, no direct perfusion imaging (myocardial scintigraphy or cardiac MRI) was performed; therefore, the microcirculatory-ischemia hypothesis cannot be directly confirmed. Alternative mechanisms of myocardial injury are considered, including: (a) myocardial stunning related to the relatively long CPB and cross-clamp times (cross-clamp time [CCT] 169 min), (b) heterogeneous cardioplegia distribution given the multivalve procedure requiring selective coronary perfusion, (c) air or particulate microembolism during CPB, (d) ischemia–reperfusion injury, (e) demand ischemia related to postoperative atrial-fibrillation-associated tachyarrhythmia, and (f) inadequate confirmation of the cardioplegia delivery temperature due to reliance on subjective (e.g., tactile) assessment in the absence of thermography.[13–15]

Thermography is 1 component of a comprehensive, multimodal management strategy encompassing perioperative warming, CPB management, cardioplegia strategy, and hematologic management, and it should not be regarded as the sole determinant of surgical outcome. Furthermore, infrared thermography measures radiant temperature from the epicardial surface and cannot directly assess transmural myocardial temperature gradients or temperature within the coronary microcirculation. A discrepancy may therefore exist between the epicardial surface temperature and the temperature of the deeper myocardium or microcirculation, and the present findings do not directly confirm the proposed microcirculatory injury hypothesis.

Conclusions

The results of this study suggest that noninvasive infrared thermography may serve as a useful adjunctive tool, not only for evaluating the efficacy of myocardial protection but also for real-time intraoperative monitoring of critical temperature changes in patients with CAD, particularly during sternotomy, CPB initiation, and cardioplegia administration, where strict thermal control is essential.

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Declarations

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Authors' contributions

YN and GK designed the study and collected the data; YN performed the data analysis and thermographic evaluation; GK and HW drafted the manuscript. All authors read and approved the final manuscript.

Availability of data and materials

No datasets were generated or analyzed during the current study.

Ethics approval and consent to participate

This study was approved by the Institutional Review board of Fukuoka University (Approval No. U24-11-005), and written informed consent was obtained from all patients prior to data collection and analysis.

Consent for publication

Not applicable.

Competing interests

The authors declare that they have no competing interests.

References

1) Berentsen S. Diagnosis and management of cold agglutinin disease. Hematology (Am Soc Hematol Educ Program) 2025; 2025: 295–304.
2) Sayed NI, Basantwani S, Bhalerao C, et al. Cardiopulmonary bypass surgery-cold alert! Ann Card Anaesth 2023; 26: 223–6.
3) Barbara DW, Mauermann WJ, Neal JR, et al. Cold agglutinins in patients undergoing cardiac surgery requiring cardiopulmonary bypass. J Thorac Cardiovasc Surg 2013; 146: 668–80.
4) Fan Y, Zhang AM, Xiao YB, et al. Warm versus cold cardioplegia for heart surgery: a meta-analysis. Eur J Cardiothorac Surg 2010; 37: 912–9.
5) Abah U, Garfjeld Roberts P, Ishaq M, et al. Is cold or warm blood cardioplegia superior for myocardial protection? Interact Cardiovasc Thorac Surg 2012; 14: 848–55.
6) Nishimura Y, Kuwahara G, Wada H. Comparison of myocardial cooling effects between antegrade and retrograde cardioplegia: a retrospective study using thermography. J Cardiothorac Surg 2025; 21: 52.
7) Kawashima T, Shimada Y, Okubo N, et al. Aortic valve replacement in a patient with cold agglutinins: Report of a case. Kyobu Geka 2026; 79: 287–9. (in Japanese)
8) Yu Z, Minakawa M, Aoki T, et al. Aortic stenosis combined with cold agglutinin disease. Kyobu Geka 2017; 70: 490–2. (in Japanese)

9) Kondo Y, Shiraishi S, Takashima N, et al. Ascending aortic replacement and aortic valve replacement in a patient with cold agglutinins. Kyobu Geka 2023; 76: 447–9. (in Japanese)
10) Kakimoto M, Sasayama K, Nishimura Y, et al. Study on myocardial temperature cooling during myocardial protection using thermography. Jpn J Cardiovasc Surg 2023; 52: 381–6. (in Japanese)
11) Fujimasa I. Samogurafi no genri oyobi kiso. Jpn J Med Instrum 1969; 39: 296–302. (in Japanese)
12) Tamura T. A note on estimating of whole and regional mean skin temperature by using thermography. J Home Econom Jpn 1980; 31: 461–3. (in Japanese)
13) den Os MM, van den Brom CE, van Leeuwen ALI, et al. Microcirculatory perfusion disturbances following cardiopulmonary bypass: a systematic review. Crit Care 2020; 24: 218.
14) Kanuparthy M, Manthana R, Kaushik H, et al. Microvascular dysfunction following cardioplegic arrest and cardiopulmonary bypass: impacts of diabetes and hypertension. Biomedicines 2025; 13: 409.
15) Aykut G, Ulugöl H, Aksu U, et al. Microcirculatory response to blood vs. crystalloid cardioplegia during coronary artery bypass grafting with cardiopulmonary bypass. Front Med (Lausanne) 2022; 8: 736214.