

Anticoagulation with Bivalirudin in a Case of Mitral Valve Replacement and Coronary Artery Bypass Grafting Requiring Prolonged Extracorporeal Membrane Oxygenation Support: A Case of Emergency Surgery Due to Posterior Papillary Muscle Rupture

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Abstract

Extracorporeal membrane oxygenation (ECMO) is an increasingly used support method in cases of refractory cardiopulmonary failure. However, due to the non-biological surfaces of the system and the inflammatory responses it induces, coagulation disorders may develop, necessitating meticulous anticoagulation management. At present, unfractionated heparin (UFH) is used as the standard; however, in complications such as heparin-induced thrombocytopenia (HIT), alternative agents may be required. We present the management of a 59-year-old male who developed acute mitral regurgitation due to posterior papillary muscle rupture and underwent emergency mitral valve replacement and coronary artery bypass grafting. Due to post-operative right ventricular failure, venoarterial ECMO support was initiated. On the 5th day of ECMO, progressive thrombocytopenia and central catheter thrombosis occurred. With a high 4T score, HIT was suspected. UFH infusion was discontinued, and bivalirudin was initiated as an alternative anticoagulant. The anticoagulation process was monitored with activated clotting time and activated partial thromboplastin time, and therapeutic targets were successfully achieved without major bleeding or thrombotic complications. The patient was weaned off ECMO on day 15 and discharged in stable condition on post-operative day 74. This case demonstrates that bivalirudin is a safe and effective anticoagulant option in ECMO patients who develop HIT. In complex cases with heparin contraindications, bivalirudin plays an important role in individualized anticoagulation strategies.

Keywords: Bivalirudin; extracorporeal membrane oxygenation; heparin-induced thrombocytopenia; papillary muscle rupture; right ventricular failure.

Mitral Kapak Değişimi ve Koroner Arter Bypass Greft Ameliyatı Sonucunda Uzun Süreli ECMO Desteği Gerektiren Bir Vakada Bivalirudin ile Antikoagülasyon: Posterior Papiller Kas Yırtılması Nedeniyle Acil Cerrahi Vakası

Özet

Ekstrakorporeal membran oksijenasyonu (ECMO), refrakter kardiyopulmoner yetmezlik olgularında geçici do-
laşım ve oksijenasyon desteği sağlayan ileri yaşam destek yöntemlerinden biridir. Ancak ECMO devrelerinin
biyoyumumluluğunun sınırlı olması ve sistemik inflamatuvar yanıtı tetiklemesi nedeniyle koagülasyon bozuklukları
sık görülmekte ve dikkatli bir antikoagülasyon yönetimi gerekmektedir. Standart antikoagülan olarak fraksiyone
edilmemiş heparin (UFH) kullanılmakla birlikte, heparine bağlı trombositopeni (HIT) gelişen olgularda alternatif
ajanlara ihtiyaç duyulmaktadır. Posterior papiller kas rüptürüne bağlı akut mitral yetersizliği gelişen 59 yaşındaki

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erkek hastaya acil mitral kapak replasmanı ve koroner arter baypas greftleme (CABG) uygulandı. Ameliyat sonrası sağ ventrikül yetmezliği gelişmesi üzerine venoarteriyel ECMO desteği başlatıldı. ECMO'nun beşinci gününde ilerleyici trombositopeni ve santral kateter trombozu saptandı. Yüksek 4T skoru nedeniyle HIT düşünüldü ve UFH infüzyonu kesilerek alternatif antikoagülan olarak bivalirudin tedavisine başlandı. Antikoagülasyon süreci, aktive edilmiş pıhtılaşma zamanı (ACT) ve aktive parsiyel tromboplastin zamanı (aPTT) ile izlenerek terapötik hedeflere ulaşıldı. İzlem süresince majör kanama veya trombotik komplikasyon gelişmedi. Hasta, 15. günde ECMO'dan ayrıldı ve ameliyat sonrası 74. günde stabil durumda taburcu edildi. Bu olgu, ECMO desteği alan ve HIT gelişen hastalarda bivalirudin güvenli ve etkili bir antikoagülasyon seçeneği olduğunu göstermektedir. Heparinin kontrendike olduğu durumlarda bivalirudin, bireyselleştirilmiş antikoagülasyon stratejilerinde önemli bir alternatif olarak değerlendirilebilir.

Anahtar sözcükler: Bivalirudin; ECMO; heparine bağlı trombositopeni; papiller kas rüptürü; sağ ventrikül yetmezliği.

Introduction

Extracorporeal membrane oxygenation (ECMO) is a life-saving treatment method that provides temporary circulatory and oxygenation support in cases of refractory cardiac and/or pulmonary failure. With advancing technologies, the use of ECMO has expanded, and it is increasingly preferred in both pediatric and adult populations. However, due to the limited biocompatibility of ECMO circuit materials and the triggering of systemic inflammatory responses, it leads to imbalances in the coagulation system. This creates a high risk for both in-circuit thrombosis and serious hemorrhagic complications.^[1,2]

The primary goal of anticoagulation in ECMO therapy is to prevent thrombus formation within the circuit while minimizing the risk of systemic bleeding. At present, the most commonly used agent for this purpose is unfractionated heparin (UFH). Heparin is preferred due to its ease of administration and reversibility with protamine sulfate. However, long-term use of heparin can lead to serious issues such as heparin-induced thrombocytopenia (HIT), heparin resistance, and difficulties in monitoring laboratory values.^[3,4]

In such cases, direct thrombin inhibitors, particularly bivalirudin, have emerged as alternative anticoagulants. Bivalirudin's short half-life, predictable pharmacokinetics, and effectiveness on both free and fibrin-bound thrombin make it a safe and effective option in patients with HIT.^[5,6] However, the use of bivalirudin during ECMO therapy, especially in adult patients, is supported by a limited number of studies, and clinical experience remains relatively uncommon.

In this case report, we present the management of an adult patient who developed acute mitral regurgitation due to posterior papillary muscle rupture, underwent mitral valve replacement and coronary artery bypass grafting (CABG), required prolonged ECMO support, and was anticoagulated with bivalirudin following the development of HIT. Through this case, we aim to discuss the safe and effective use of bivalirudin during the ECMO process in light of clinical and laboratory findings.

Case Report

A 59-year-old male patient presented to the emergency department with sudden onset of shortness of breath. Upon evaluation, total occlusion was detected in the proximal segment of the right coronary artery (RCA), and the patient was diagnosed with an inferior myocardial infarction (MI). He underwent urgent primary percutaneous coronary intervention and was admitted to the coronary intensive care unit.

During intensive care follow-up, due to worsening dyspnea symptoms, transthoracic echocardiography (TTE) revealed severe mitral regurgitation secondary to posterior papillary muscle rupture. The patient was transferred to the cardiovascular surgery (CVS) intensive care unit, where an intra-aortic balloon pump (IABP) was inserted, and an infusion of noradrenaline (norepinephrine; steradin) at 0.5 mcg/kg/min was initiated.

Due to hemodynamic instability, the patient underwent emergency surgery. Three-vessel CABG was performed using saphenous vein grafts to the RCA, left anterior descending, and circumflex arteries, along with mechanical mitral valve replacement in the same session. Cardiopulmonary bypass (CPB) time was recorded as 302 min, and aortic cross-clamp time was 101 min. Myocardial protection was provided with antegrade delivery every 20 min and continuous retrograde cardioplegia. After bypass, high-dose inotropic support was initiated: Adrenaline at 0.15 mcg/kg/min, dobutamine at 12 mcg/kg/min, and noradrenaline (steradin) at 0.4 mcg/kg/min.

However, shortly after venous lines were removed, the patient developed right ventricular (RV) failure, resulting in hypotension and loss of contractility. CPB was reinitiated for an additional 15 min of support. Due to persistent RV dysfunction, venoarterial ECMO (VA-ECMO) support was planned. The ECMO circuit was established via bicaval venous cannulation (superior vena cava and left femoral vein) and arterial cannulation from the aorta. IABP was continued in 1:1 augmentation mode, atrial pacing was applied, and the patient was transferred to the intensive care unit with the sternum left open.

Anticoagulation with UFH was initiated during the ECMO process. The target activated clotting time range was set at 180–200 s. Intermittent blood gas monitoring showed no metabolic acidosis or elevated lactate levels, and urine output remained above 100 mL/h.

On the 3rd day, the patient was taken back to the operating room for peripheral arterial cannulation. Arterial cannulation was performed via a graft to the axillary artery, and the superior vena cava cannula was removed. ECMO support continued with a single venous cannula. Due to persistent cardiac edema, the sternum was left open; circuit pressure monitoring and oxygenator performance were regularly assessed (Fig. 1).

In the following days, inotropic support was gradually reduced. On the 5th day of ECMO, progressive thrombocytopenia was observed. The platelet count, initially 230,000/mm³, dropped to 85,000/mm³ by the 5th day. Along with this decline, thrombo-



Figure 1. Cannulation sites for extracorporeal membrane oxygenation support. (a) Arterial cannulation from the right axillary artery using a vascular graft. (b) Venous cannulation from the left femoral vein.

sis was suspected in the central catheter line, which required replacement twice. These findings raised suspicion of HIT. Clinical evaluation using the 4T score (Table 1) yielded a total of 7 points (platelet drop >50%, onset on day 5, evidence of new thrombosis, and no alternative causes), indicating a high probability of HIT.

Other potential causes of thrombocytopenia were systematically evaluated. Sepsis was excluded based on the absence of clinical signs of infection, stable inflammatory markers, and negative blood cultures. ECMO-related platelet consumption and hemodilution were considered; however, the rapid decline in platelet count (>50%) accompanied by new thrombotic events was not consistent with isolated ECMO-related thrombocytopenia.

Drug-induced thrombocytopenia was also reviewed, and no other medications known to cause thrombocytopenia were identified. Based on these findings and a high 4T score, HIT was considered the most likely diagnosis.

Due to the urgency of the clinical picture and the unavailability of confirmatory tests such as PF4/enzyme-linked immunosorbent assay in the hospital laboratory at that time, heparin infusion was immediately discontinued. Following guidelines and ELSO recommendations, bivalirudin was initiated as an alternative anticoagulant. It was administered intravenously at an initial dose of 0.15 mg/kg/h. Because the dose-response relationship is unpredictable due to the ECMO circuit's large volume of distribution and surface area, dosage titration was guided by both ACT and activated partial thromboplastin time (aPTT) values. The ACT target was 180–220 s, and the aPTT target range was 60–80 s.^[6,7]

During the treatment period, the infusion rate ranged between 0.12 and 0.22 mg/kg/h, with a mean dose of approximately 0.17 mg/kg/h. Renal function remained stable throughout the treatment (creatinine range: 1.0–1.2 mg/dL), and no dose adjustment due to renal impairment was required.

During the first 12 h of monitoring, ACT values remained subtherapeutic; therefore, the bivalirudin dose was gradually in-

Table 1. 4T score criteria for the assessment of heparin-induced thrombocytopenia

Criterion	2 Points	1 Point	0 Points
Platelet decrease	≥50% decrease, rarely <20,000/mm ³	30–50% drop or lowest level 10–19,000/mm ³	<30% drop
Timing	Onset between days 5–10 (or <1 day within the last 30 days if previous heparin exposure)	Start after 10 days or timing uncertain	<4 days, no previous heparin exposure
Thrombosis or complication	New thrombosis, skin necrosis, acute systemic reaction	Clinically suspected thrombosis, line occlusion	No thrombosis
Other reasons	There is no other possible cause	Other possible reason	Another reason is very possible

Table 2. Serial laboratory and anticoagulation parameters during extracorporeal membrane oxygenation support

Day	PLT ($\times 10^3/\mu\text{L}$)	ACT (sec)	aPTT (sec)	Anticoagulant	Dose (mg/kg/h)	Creatinine (mg/dL)
1	230	185	56	UFH	—	1.0
2	210	190	58	UFH	—	1.0
3	190	195	60	UFH	—	1.1
4	150	188	62	UFH	—	1.1
5	85	165	55	UFH → Bivalirudin	0.15	1.2
6	95	178	63	Bivalirudin	0.18	1.2
7	110	190	68	Bivalirudin	0.20	1.1
8	125	205	72	Bivalirudin	0.22	1.1
9	140	210	75	Bivalirudin	0.20	1.0
10	160	200	72	Bivalirudin	0.18	1.0
11	175	195	70	Bivalirudin	0.17	1.0
12	190	192	68	Bivalirudin	0.16	1.0
13	205	188	66	Bivalirudin	0.15	1.0
14	220	185	64	Bivalirudin	0.14	1.0
15	235	180	62	Bivalirudin	0.12	1.0

PLT: Platelet count; ACT: Activated clotting time; aPTT: Activated partial thromboplastin time.

creased by 20%. After dose adjustments, stable therapeutic levels were achieved. No significant bleeding complications were observed during this period; platelet counts stabilized, and central line thrombosis resolved. In addition, no thrombosis or need for oxygenator replacement occurred within the ECMO circuit.

Within the first 48 h after initiating bivalirudin, platelet counts stabilized, and systemic anticoagulation parameters (ACT and aPTT) reached target ranges. No thrombus formation or oxygenator dysfunction developed in the circuit. Throughout this period, there were no bleeding complications, and platelet transfusion was not required. No significant post-operative bleeding was observed, and no transfusion of blood products was required during the early post-operative period.

As shown in Table 2, the platelet count reached its nadir on day 5 and subsequently increased following the initiation of bivalirudin, in parallel with stabilization of ACT and aPTT values.

On the 10th post-operative day, clinical stability was achieved, and TTE along with invasive pressure monitoring indicated regression of intracardiac edema. Consequently, the previously left-open sternum was successfully closed.

As hemodynamic parameters improved and the need for inotropic support decreased gradually, the VA-ECMO flow was progressively reduced in accordance with daily protocols. On day 12, ECMO was transitioned to minimal flow mode, and systemic perfusion was deemed sustainable with the patient's native cardiac output. Arterial blood gas analyses, lactate levels, mixed venous oxygen saturation (SvO_2), and urine output supported adequate systemic oxygenation and organ perfusion. At the same time, a clear normalization trend was observed in parameters such as central venous pressure and pulmonary artery pressure, indicating reduced right heart strain.

On the 14th postoperative day, IABP support was discontinued in line with improved spontaneous systolic function. Cardiac out-

put adequacy was re-evaluated via TTE, revealing normalized RV function, a TAPSE >17 mm, and improved RV free wall motion.

By the 15th day of ECMO, inotropic support requirements had reached a minimum, and all vital signs and perfusion parameters had stabilized. Following advanced evaluation by the cardiology team, it was concluded that the patient's RV function and systemic perfusion could be maintained without ECMO support. Accordingly, the ECMO circuit was discontinued, and cardiopulmonary functions were monitored invasively for 12 h. No hemodynamic deterioration was observed during this period.

On the 16th day, the patient demonstrated successful performance in a spontaneous breathing trial conducted before extubation. Mechanical ventilation support was subsequently withdrawn, and the patient was extubated successfully. In the following days, respiratory physiotherapy and mobilization protocols were initiated, marking the beginning of early rehabilitation.

During the intensive care period, the patient did not develop any major hemorrhagic complications, new thrombotic events, sepsis, ventilator-associated pneumonia, renal dysfunction, or neurological deficits. Renal and hepatic functions remained within normal limits.

During ECMO support, serial laboratory parameters were closely monitored. D-dimer, lactate dehydrogenase, fibrinogen, total bilirubin, and platelet counts remained stable throughout the course of bivalirudin therapy. Oxygenator performance was assessed by monitoring the pressure gradient (DeltaP) across the oxygenator, and no significant increase was observed, indicating the absence of circuit thrombosis.

The patient was evaluated by a multidisciplinary team and was transferred to the inpatient CVS ward on post-operative day 44 (March 28, 2025). During the ward stay, no complications developed. The patient improved his physical condition and maintained adequate oral intake without difficulty.

In the final evaluation, he was found to be hemodynamically stable, afebrile, with laboratory parameters within normal limits, and classified as NYHA class I in terms of functional capacity.^[8] On post-operative day 74, the patient was enrolled in a home follow-up program and discharged in stable condition.

Detailed serial laboratory parameters including platelet count, ACT, aPTT, and other biochemical markers during ECMO support are summarized in Table 2.

Discussion

ECMO is one of the advanced life support methods that provide temporary hemodynamic and oxygenation support, particularly in cases of severe cardiac dysfunction and low-output syndrome following MI. However, the non-biocompatible surfaces of the ECMO circuit – such as the pump, oxygenator, and cannula systems – induce numerous pathophysiological mechanisms including platelet activation, coagulation cascade stimulation, and inflammatory cytokine release. These mechanisms create a fragile balance between thrombocytopenia, thrombosis, and bleeding.^[1,2]

Coagulation management in ECMO patients requires a meticulously monitored anticoagulation protocol to maintain this delicate balance. According to the current guidelines, UFH remains the first-line anticoagulant.^[6] UFH acts as a factor IIa and Xa inhibitor via antithrombin III (AT-III), and offers several advantages: it can be easily monitored using ACT, aPTT, and anti-Xa levels, it is cost-effective, and its effects can be reversed with protamine.

However, the use of heparin may lead to serious complications in some patients. The most significant of these is immune-mediated HIT, which has an incidence of 1–3% in patients undergoing CPB.^[4] HIT is caused by immunoglobulin G antibodies formed against PF4–heparin complexes, which activate platelets, leading to both thrombocytopenia and, paradoxically, venous and arterial thrombosis. Especially in ECMO patients, where the procoagulant environment is already intense, HIT must be diagnosed rapidly, and the anticoagulation strategy must be promptly revised.

In clinical practice, the 4T score is used to assess HIT suspicion. In high-risk cases, heparin should be discontinued without waiting for laboratory confirmation, and alternative anticoagulation should be initiated. Bivalirudin, a direct thrombin inhibitor, is one of the agents increasingly supported by clinical experience for such scenarios. Unlike heparin, bivalirudin does not depend on AT-III and inhibits both free and fibrin-bound thrombin, making it advantageous in patients with heparin resistance, low AT-III levels, or HIT.^[5]

The use of bivalirudin in ECMO-supported patients is supported by multiple institutional experiences, case series, and retrospective analyses in the literature. Particularly, its predictable pharmacokinetics, short half-life (~25 min), and relatively fewer reported bleeding complications make it a favorable option.^[6,9] Bivalirudin infusion is typically initiated at 0.05–0.5 mg/kg/h and titrated according to ACT or aPTT values. However, limitations include its irreversibility with protamine and higher cost. While no consensus exists in the literature regarding monitoring pa-

rameters, some centers also use thrombin–antithrombin complex, thromboelastography, and D-dimer levels for follow-up.^[10]

Several studies comparing bivalirudin and UFH in ECMO patients have demonstrated comparable or even reduced rates of bleeding and thrombotic complications with bivalirudin. Retrospective analyses suggest that bivalirudin provides a more stable anticoagulation profile and may reduce the need for transfusion.

In addition, bivalirudin has predictable pharmacokinetics and does not depend on antithrombin levels, which are often reduced in critically ill patients. This may provide a significant advantage in ECMO patients, where coagulation dynamics are highly variable and difficult to control.

In the presented case, progressive thrombocytopenia on day 5 of ECMO, thrombus formation in the central catheter line, and a high 4T score led to the discontinuation of UFH and initiation of bivalirudin under the presumptive diagnosis of HIT. Target anticoagulation levels were safely maintained with ACT and aPTT monitoring. No circuit thrombosis was observed, no platelet transfusion was required, and no systemic bleeding occurred. These findings further support the effectiveness and safety of bivalirudin in managing HIT during ECMO. Serial monitoring demonstrated a clear temporal relationship between bivalirudin initiation and improved anticoagulation control without thrombotic or hemorrhagic complications.

Unlike previously published reports focusing primarily on anticoagulation strategies in isolated ECMO or HIT settings, our case represents a complex and high-risk clinical scenario combining emergency surgery due to papillary muscle rupture, concomitant CABG and mitral valve replacement, prolonged VA-ECMO support, and suspected HIT.

The successful management of anticoagulation with bivalirudin in such a multifactorial and unstable setting, along with successful ECMO weaning without thrombotic or bleeding complications, provides practical insight for clinicians managing similarly complex patients.

Therefore, beyond confirming the known efficacy of bivalirudin, this case highlights its feasibility and safety in a challenging real-world ECMO setting requiring dynamic anticoagulation strategies.

Conclusion

HIT during ECMO support is a potentially life-threatening complication that requires urgent and effective changes in the treatment approach. As demonstrated in this case, anticoagulation with bivalirudin was safely maintained within the therapeutic range without any serious secondary complications such as systemic bleeding, circuit thrombosis, or the need for platelet replacement.

Due to its short half-life, predictable pharmacokinetics, and independence from AT-III, bivalirudin stands out as an effective and reliable alternative, particularly in ECMO patients who develop HIT or face challenges in heparin monitoring. In this context, direct thrombin inhibitors such as bivalirudin hold a significant role in individualized anticoagulation strategies based on clinical experience. However, to support its routine use with stronger evidence, large-scale randomized controlled trials are needed.

Disclosures

Ethics Committee Approval: This is a single case report, and therefore ethics committee approval was not required in accordance with institutional policies.

Informed Consent: Written informed consent was obtained from the patient for publication of this case report and accompanying images.

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