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REFRACTORY VASOPLEGIC SEPTIC SHOCK AFTER GALLBLADDER PERFORATION: A CASE REPORT AND FOCUSED REVIEW OF OXIRIS-ENHANCED CRRT

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ABSTRACT

Introduction: Persistent vasoplegia after source control and guideline-based care remains a major challenge in septic shock. In patients with acute kidney injury requiring continuous renal replacement therapy (CRRT), oXiris combines renal support with endotoxin and cytokine adsorption, but evidence certainty remains low.

Case description: A 70-year-old woman developed septic shock, acute respiratory distress syndrome, coagulopathy, metabolic acidosis and oliguric/anuric acute kidney injury after gallbladder perforation. Urgent surgical drainage, broad-spectrum antimicrobials, corticosteroids, mechanical ventilation, norepinephrine 20–25 µg/min and vasopressin 0.04 U/min did not reverse vasoplegia. oXiris-based continuous veno-venous haemodialysis began on intensive care unit (ICU) day 3, when the Sequential Organ Failure Assessment (SOFA) score was 18. Norepinephrine fell to 3 µg/min by treatment hour 17.75 and was discontinued on ICU day 5; vasopressin stopped on day 9. SOFA fell to 8 within 48–72 hours. CRRT ended after approximately 69 hours, mechanical ventilation was discontinued on day 8, kidney function recovered, and the patient was discharged home on hospital day 36.

Discussion: Early vasopressor de-escalation was the strongest temporally linked signal; however, source control and multimodal care prevent causal attribution. Recent oXiris meta-analyses report favourable cytokine, haemodynamic and inflammatory surrogate signals, but the 2026 Surviving Sepsis Campaign conditionally suggests against blood-purification techniques because patient-important benefits remain uncertain.

Conclusion: Time-limited oXiris-enhanced CRRT may be considered as an adjunct in carefully selected, source-controlled patients with refractory vasoplegia and an independent CRRT indication, but the observation is hypothesis-generating and does not establish efficacy; prospective phenotype-enriched trials are needed.

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KEYWORDS

Septic shock, acute kidney injury, continuous renal replacement therapy, vasoplegia, oXiris

LEARNING POINTS

- Source control, antimicrobials and guideline-based shock care remain primary; persistent vasoplegia is a clinical phenotype, not proof of endotoxaemia.
- When AKI independently requires CRRT, oXiris can add adsorption without a second circuit; benefit or futility should be reassessed within 24–72 hours using vasopressor dose, SOFA score and biomarkers.
- Evidence certainty remains low: the 2026 Surviving Sepsis Campaign conditionally suggests against blood-purification techniques because patient-important benefits remain uncertain, and resource requirements are substantial; device-specific oXiris and TIGRIS signals should therefore remain hypothesis-generating.

INTRODUCTION

Septic shock is managed with early antimicrobial therapy, source control, haemodynamic resuscitation and organ support, yet some patients remain profoundly vasoplegic despite these measures[1]. The 2026 Surviving Sepsis Campaign conditionally suggests against blood-purification techniques, including haemoperfusion, high-dose haemofiltration and plasma exchange, because patient-important benefits remain uncertain and resource requirements are substantial[1]. When severe acute kidney injury (AKI) independently requires CRRT, however, a membrane that combines kidney support with adsorption remains biologically attractive, although it is not established as routine sepsis therapy.

The oXiris set is based on an AN69 membrane with a polyethyleneimine surface and heparin graft; it provides diffusive and convective clearance while adsorbing cytokines and negatively charged endotoxin[2,3]. Evidence is derived mainly from small trials and observational studies. Meta-analyses report improvements in cytokines and short-term haemodynamic or inflammatory surrogate outcomes, but survival benefit remains unproven and certainty is low[3-6]. Sepsis is heterogeneous, and bedside phenotypes should not be equated with mechanistically proven endotypes.

We report source-controlled biliary septic shock with persistent dual-vasopressor vasoplegia and CRRT-requiring AKI in which oXiris-enhanced CRRT was followed by rapid vasopressor de-escalation. The observation is interpreted alongside current oXiris evidence, the 2025 Asia-Pacific consensus and the biomarker-enriched TIGRIS trial. The report follows CARE principles and does not infer a membrane-specific causal effect.

CASE DESCRIPTION

A 70-year-old woman with type 2 diabetes mellitus, hypertension and hypothyroidism presented after seven days of worsening abdominal pain, nausea and vomiting. Her history included endoscopic retrograde cholangiopancreatography and cholecystostomy for obstructive jaundice complicated by acute cholecystitis and pancreatitis. Computed tomography demonstrated gallbladder perforation. Urgent operative drainage was performed and she was transferred, intubated, to the intensive care unit (ICU).

She had fulminant septic shock with acute respiratory distress syndrome, coagulopathy, severe metabolic acidosis and oliguric/anuric AKI. Initial lactate was 9.3 mmol/l and pH was 7.2. Broad-spectrum antimicrobials, stress-dose corticosteroids, lung-protective ventilation, norepinephrine 20–25 µg/min and vasopressin 0.04 U/min were administered. Microbiological and antimicrobial-susceptibility details were not retrievable from the de-identified record.

Despite source control and standard support, vasoplegia and renal failure persisted. Immediately before oXiris initiation on ICU day 3, norepinephrine remained 20–25 µg/min (0.25–0.31 µg/kg/min at 80 kg), vasopressin was 0.04 U/min, creatinine was 293.8 µmol/l, procalcitonin was 40.47 ng/ml, urine output was described as oliguric/anuric and the SOFA score was 18. C-reactive protein peaked at 379.1 mg/l, and procalcitonin peaked at 141.09 ng/ml on ICU day 4. Lactate had already fallen substantially with early resuscitation, whereas vasopressor dependence persisted.

Continuous veno-venous haemodialysis was started with an oXiris haemofilter on ICU day 3. Blood flow was 150 ml/min, dialysate flow 2400 ml/h (30 ml/kg/h); no replacement fluid was used, and net ultrafiltration was 100 ml/h. Access was through a right internal jugular dialysis catheter; the first circuit used heparin. At treatment hour 17.75, high filter pressure required circuit termination. Treatment restarted 118 minutes later with a new oXiris filter and regional citrate anticoagulation. The elapsed treatment window was 69 hours 8 minutes, with approximately 67 hours 10 minutes delivered. The treatment sequence, including the circuit interruption, is shown in *Figure 1*.

Norepinephrine decreased to 3 µg/min by treatment hour 17.75 and was discontinued on ICU day 5. Vasopressin was tapered and discontinued on day 9. SOFA decreased from 18 to 8 within 48–72 hours. C-reactive protein declined to 77.4 mg/l by day 6 and 32.4 mg/l by day 10; procalcitonin declined to 2.40 ng/ml by day 10. Creatinine declined to

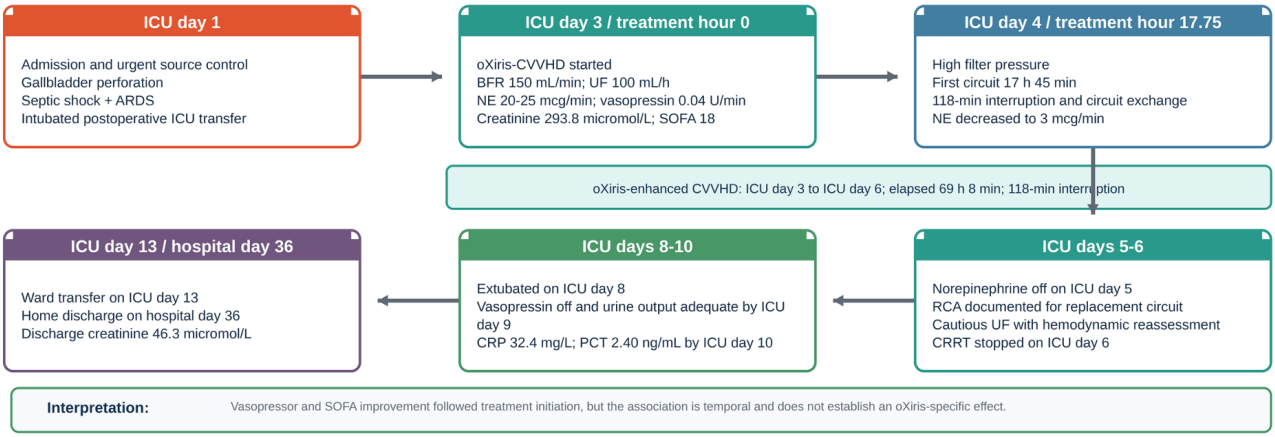


Figure 1. *ICU-day timeline of source control, oXiris-CVVHD exposure, circuit exchange, vasopressor de-escalation and recovery. The temporal sequence does not establish an oXiris-specific effect.*
Abbreviations: ARDS, acute respiratory distress syndrome; BFR, blood flow rate; CRRT, continuous renal replacement therapy; CVVHD, continuous veno-venous haemodialysis; ICU, intensive care unit; NE, norepinephrine; PCT, procalcitonin; RCA, regional citrate anticoagulation; SOFA, Sequential Organ Failure Assessment; UF, ultrafiltration.

74.5 µmol/l by day 6. CRRT was stopped on day 6, and urine output was adequate by day 9. The haemodynamic, inflammatory and renal-metabolic trajectories are shown in *Figure 2* and summarised in *Table 1*.

The patient was extubated on ICU day 8, transferred to the ward on day 13 and discharged home on hospital day 36 with creatinine 46.3 µmol/l. No adverse event directly attributable to the oXiris membrane was documented. Written informed consent for publication was obtained from the patient's legally authorised representative. *Figure 3* summarises the case pathway and observations across the clinical course, distinguishing the elapsed treatment window from delivered therapy and noting that substantial lactate improvement preceded oXiris initiation.

Variable	Pre-oXiris/early course	During oXiris-enhanced therapy	Post-therapy/follow-up	Interpretation
Timing	Admitted 30 Nov 2025; urgent operative drainage before ICU transfer	Started 2 Dec; circuit replaced 3 Dec after high filter pressure; stopped 5 Dec	Extubated 7 Dec; ward 12 Dec; home 4 Jan 2026	Elapsed exposure 69 h 8 min; approximately 67 h 10 min delivered
Haemodynamics	Norepinephrine 20–25 µg/min; vasopressin 0.04 U/min; SOFA 18	Norepinephrine 3 µg/min by treatment hour 17.75 and off ICU day 5; vasopressin tapered	Vasopressin off ICU day 9; SOFA 8 within 48–72 h	Strongest early response signal was vasopressor de-escalation
Inflammation	CRP peak 379.1 mg/l; PCT 40.47 ng/ml before initiation	PCT peak 141.09 ng/ml on ICU day 4, followed by sustained decline; CRP 77.4 mg/l by day 6	CRP 32.4 mg/l and PCT 2.40 ng/ml by day 10	Early inflammatory surge was followed by resolution
Renal/metabolic	Oliguric/anuric AKI; creatinine 293.8 µmol/l; lactate 9.3 mmol/l initially	CVVHD; creatinine 74.5 µmol/l by day 6; lactate largely improved before oXiris	Urine output adequate by day 9; discharge creatinine 46.3 µmol/l	Renal recovery continued after CRRT discontinuation
Organ support	Acute respiratory distress syndrome (ARDS) requiring invasive ventilation; dual vasopressors	oXiris-CVVHD plus standard critical care	Extubated day 8; no further renal replacement therapy; discharged home day 36	Global organ recovery followed haemodynamic stabilisation

Table 1. *Key clinical, laboratory and CRRT variables during the acute observation period.*
Abbreviations: AKI, acute kidney injury; ARDS, acute respiratory distress syndrome; CRP, C-reactive protein; CRRT, continuous renal replacement therapy; CVVHD, continuous veno-venous haemodialysis; ICU, intensive care unit; PCT, procalcitonin; SOFA, sequential organ failure assessment.

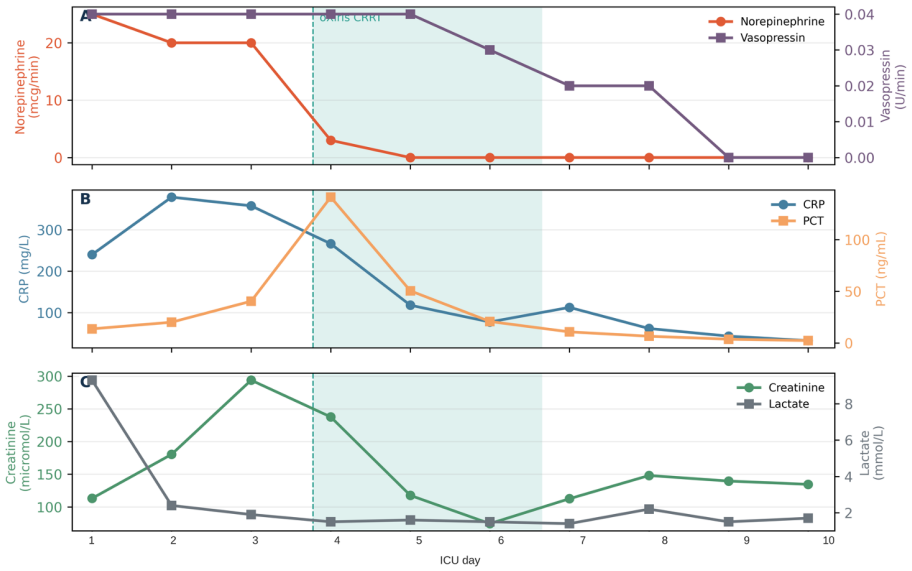


Figure 2. *Haemodynamic, inflammatory and renal-metabolic trends by ICU day. A) Vasopressor requirements; B) C-reactive protein and procalcitonin; C) creatinine and lactate. Shading marks the oXiris-CVVHD treatment window during ICU days 3–6, including a 118-minute interruption. Lactate improved before oXiris; subsequent trends are temporal observations and do not establish a membrane-specific effect.*

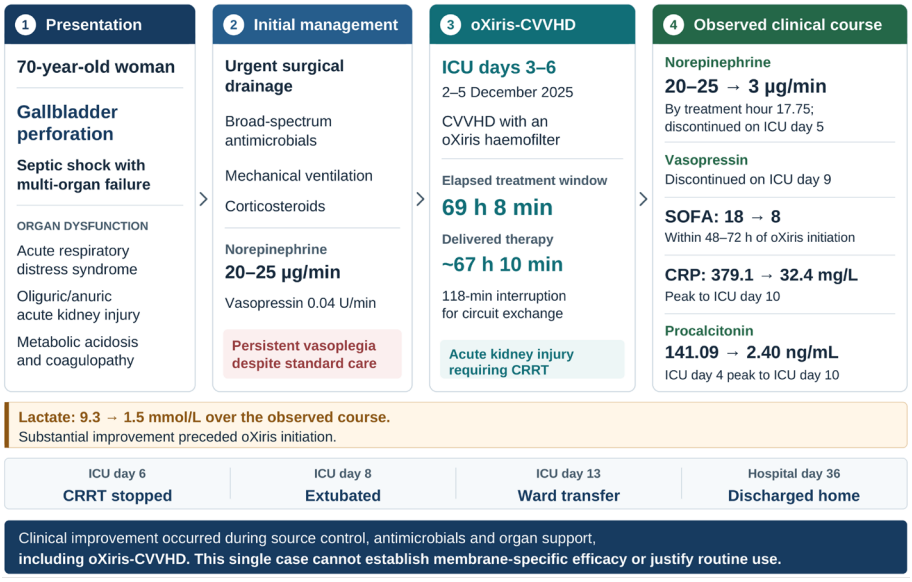


Figure 3. *Refractory vasoplegic septic shock after gallbladder perforation: response to oXiris-enhanced CRRT.*
Abbreviations: CRRT, continuous renal replacement therapy; CVVHD, continuous veno-venous haemodialysis; ICU, intensive care unit; CRP, C-reactive protein; SOFA, Sequential Organ Failure Assessment. Arrows denote clinical sequence. not causation.

DISCUSSION

The clinically important signal was the vasopressor trajectory: norepinephrine fell from 20–25 to 3 µg/min within 17.75 hours and was stopped the following day. Lactate had largely corrected before oXiris, so it should not be presented as a membrane-specific response. Source control, antimicrobials, corticosteroids, ventilation, CRRT, fluid management and time all contributed; no endotoxin activity or cytokine concentrations were measured.

The oXiris membrane integrates renal replacement with broad adsorption. In a randomised crossover study of 16 patients with endotoxin-positive septic shock and AKI, oXiris produced greater reductions in endotoxin and several cytokines than an AN69ST membrane, with a lower norepinephrine requirement[3]. A retrospective comparison reported faster improvement in the vasoactive-inotropic score, SOFA score and procalcitonin than with an ST150 membrane[4]. A 2023 meta-analysis of 14 studies and 695 patients found associations with lower 28-day mortality, norepinephrine dose, SOFA score and lactate, but rated all outcomes as low or very low certainty because the evidence was dominated by observational studies and small trials with bias concerns[5]. A subsequent systematic review, first published online in 2025, included 10 studies and meta-analysed 8 studies selected for significantly greater interleukin-6 reduction with oXiris than control; pooled analyses also favoured oXiris for vasoactive-inotropic score or norepinephrine dose, SOFA score, procalcitonin and C-reactive protein[6]. However, the enrichment strategy, heterogeneous designs and absence of robust mortality data preclude causal or survival claims. A previous case of sequential oXiris-based extracorporeal support similarly demonstrated feasibility while remaining unable to establish causality[7].

These favourable surrogate signals must be interpreted against current guideline recommendations. The 2026 Surviving Sepsis Campaign conditionally suggests against blood-purification techniques, including

haemoperfusion, high-dose haemofiltration and plasma exchange, and separately suggests against polymyxin B haemoperfusion[1]. The guideline panel judged effects on patient-important outcomes, particularly mortality, to remain uncertain; it also identified substantial resource and health-equity implications and found insufficient evidence to make separate recommendations according to purification mechanism[1]. Although oXiris is operationally distinct because adsorption is incorporated into a CRRT membrane rather than delivered through a separate circuit, that practical distinction does not establish clinical benefit or override guideline-level uncertainty.

Against this background, the 2025 Asia-Pacific consensus suggested oXiris for patients with sepsis or septic shock who require CRRT and have haemodynamic instability, hyperinflammation, severe lactic acidosis or coagulation dysfunction; the principal recommendation was explicitly graded as low certainty[8]. Practice points favour early initiation after selection, and assessment of haemodynamics, lactate and SOFA within 72 hours, and consideration of a 50% or greater vasopressor reduction by 24 hours as a response signal. Filter exchange at 12–24 hours may preserve adsorption, whereas use up to 72 hours may continue for kidney support after stabilisation; potential antibiotic adsorption should also be considered[8]. This patient matched the broader CRRT-requiring, haemodynamically unstable and hyperinflammatory phenotype, although her norepinephrine dose was below the greater-than-0.5 µg/kg/min threshold proposed as one optional initiation criterion.

TIGRIS provides an important but device-specific comparator. In 157 adults with vasopressor-dependent septic shock, multiorgan failure and endotoxin activity of 0.60–0.89 units, polymyxin B haemoadsorption was associated with 28-day mortality of 39% versus 45% with standard care, giving a 95.3% posterior probability of benefit and an adjusted odds ratio of 0.67 (95% credible interval 0.39–1.08). At 90 days, the posterior probability of benefit was 99.4% (adjusted odds ratio 0.54, 95% credible interval 0.32–0.87)[9]. Polymyxin B is a selective endotoxin adsorber delivered in separate sessions; TIGRIS therefore supports biomarker-enriched patient selection, not interchangeability or efficacy extrapolation to oXiris. Its open-label, sponsor-funded Bayesian design, including an informative prior, also warrants measured interpretation.

This case is strengthened by a clear infectious source, documented source control, an independent CRRT indication and precise treatment timing. Limitations include the absence of a comparator, endotoxin and cytokine measurements, exact concurrent mean arterial pressure and quantitative urine output, and complete microbiological and antimicrobial-exposure data. The 2026 guideline recommendation and the limitations of the available oXiris literature further preclude any inference of routine benefit. This report therefore supports only a time-limited, response-assessed and hypothesis-generating observation, not routine oXiris use or a claim of membrane-specific efficacy.

CONCLUSION

In source-controlled biliary septic shock with persistent vasoplegia and CRRT-requiring AKI, oXiris initiation was temporally followed by rapid norepinephrine de-escalation and subsequent organ recovery. The observation is compatible with recent low-certainty consensus recommendations but cannot establish causality. Future trials should enrich for defined shock, AKI and endotoxin/inflammatory phenotypes, predefine vasopressor response, and account for antimicrobial exposure.

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