MHA MEDIHEALTH ACADEMY

Eurasian Journal of
Anesthesiology & Intensive Care

Case Report

doi
10.51271/EAJAIC-0064

Anesthetic management of major multivisceral surgery in an anuric hemodialysis-dependent patient: a case report

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Cite this article: Diker MS, Türkay Yılmaz AS, Akbaba Genç B. Anesthetic management of major multivisceral surgery in an anuric hemodialysis-dependent patient: a case report. *Eur J Anesthesiol Intens Care*. 2026;3(4):112-115. doi:10.51271/EAJAIC-0064

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Received: 06/09/2026 ◆ **Accepted**: 21/11/2026 ◆ **Published**: 23/11/2026

ABSTRACT

Patients receiving maintenance hemodialysis for end-stage kidney disease present substantial anesthetic challenges during extensive surgery, particularly when residual urine output is negligible. A 45-year-old female with bilateral atrophic kidneys, dependent on hemodialysis and producing approximately 3–5 ml of urine daily, underwent bilateral nephrectomy, radical cystectomy with urethral resection, total abdominal hysterectomy with bilateral salpingo-oophorectomy, anterior vaginal wall resection, and lymph node dissection. Combined general and epidural anesthesia, invasive hemodynamic monitoring, serial arterial blood gas analyses, individualized crystalloid administration, and titrated norepinephrine support were used. Total surgical blood loss was recorded as 800 ml, while approximately 1,000 ml of 0.9% isotonic crystalloid was administered. Serial intraoperative assessments showed low lactate concentrations and potassium levels within acceptable limits. Norepinephrine, titrated during surgery, was discontinued before extubation; the patient was extubated in the operating room and transferred to the intensive care unit. Maintenance hemodialysis was resumed on postoperative day (POD) 2, and the patient was transferred from intensive care to the surgical ward on POD 3. This case illustrates the importance of physiology-guided, individualized fluid and vasopressor management aimed at maintaining adequate tissue perfusion while avoiding unnecessary fluid loading in an anuric, dialysis-dependent patient.

Keywords: Anuria, renal dialysis, fluid therapy, norepinephrine, analgesia; epidural, nephrectomy

INTRODUCTION

Patients receiving maintenance dialysis have increased perioperative morbidity, and particular attention should be paid to volume status, electrolyte balance, anemia, cardiovascular risk, and altered drug pharmacokinetics.[1,2] Perioperative fluid management is especially challenging in functionally anuric patients because residual renal fluid excretion is absent. At the same time, extensive abdominal and pelvic procedures may be associated with blood loss, vasodilation, and prolonged hemodynamic stress.

Contemporary approaches to perioperative fluid management recommend individualizing treatment according to intravascular volume status, tissue perfusion, comorbidities, and surgical characteristics rather than applying fixed restrictive or liberal fluid regimens.[3,4]

We report the anesthetic management of a functionally anuric patient receiving maintenance hemodialysis who underwent extensive multivisceral surgery, focusing on the balance between limited crystalloid administration and titrated vasopressor support.

CASE REPORT

A 45-year-old female, 160 cm tall, with a recorded body weight of 75 kg and a documented dialysis dry weight of 73 kg, was scheduled for bilateral nephrectomy, radical cystectomy with urethral resection, total abdominal hysterectomy and bilateral salpingo-oophorectomy, anterior vaginal wall resection, and lymph node dissection because of bilateral atrophic kidneys and bladder malignancy.

The patient received maintenance hemodialysis three times weekly. Daily urine output was approximately 3–5 ml, and she was considered functionally anuric. Her medical history included hyperparathyroidism, gout, surgically

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treated spina bifida with residual neurological deficits, and neurogenic bladder. She was classified as American Society of Anesthesiologists (ASA) physical status III.

On the day before surgery, she underwent four hours of heparin-free hemodialysis with a documented ultrafiltration volume of 2,300 ml. Preoperative hemoglobin was 7.8 g/dl, and potassium, sodium, and calcium concentrations were considered acceptable for surgery.

For perioperative monitoring, an arterial catheter was inserted into the left radial artery and a central venous catheter into the right internal jugular vein. A thoracic epidural catheter was placed at the T10 level for perioperative analgesia, and blood products were made available in case transfusion was required.

General anesthesia was induced with intravenous midazolam 1 mg, fentanyl 50 µg, propofol 170 mg, and rocuronium 50 mg. Epidural analgesia was provided using a solution prepared in 100 ml of 0.9% saline containing 0.125% bupivacaine and a total of 200 µg of fentanyl, administered as an initial 3-ml bolus followed by an infusion at 3 ml/h with a 20-minute lockout interval.

Initial arterial blood gas analysis showed pH 7.509, $PaCO_2$ 25.8 mmHg, HCO_3^- 20.1 mmol/L, base excess −2.4 mmol/L, lactate 0.88 mmol/L, potassium 3.49 mmol/L, and ionized calcium 1.18 mmol/L. Minute ventilation was reduced because of respiratory alkalosis.

Serial intraoperative arterial blood gas analyses were performed. At 10:40, pH was 7.44, $PaCO_2$ 28.9 mmHg, HCO_3^- 19.2 mmol/L, potassium 3.95 mmol/L, and lactate 0.80 mmol/L. Thus, the initially marked alkalemia had diminished following adjustment of ventilation. No clinically significant hyperkalemia or rise in lactate was detected on serial measurements.

Bilateral nephrectomy was performed first. Pelvic surgery then proceeded with removal of the bladder and uterus, urethral and anterior vaginal wall resection, and lymph node dissection.

The operation lasted from 08:30 to 14:15 (5 hours 45 minutes). Arterial pressure was monitored continuously and invasively. Hypotension was considered potentially multifactorial, reflecting anesthetic- and epidural-related vasodilation as well as surgical blood loss; the relative contributions could not be objectively distinguished. Rather than administering routine fluid boluses to this functionally anuric patient, the team assessed blood loss, arterial pressure trends, and serial laboratory findings. Norepinephrine was titrated within approximately 0.05–0.1 µg/kg/min according to mean arterial pressure (MAP) and the overall hemodynamic response. The dose was increased toward 0.1 µg/kg/min when MAP fell below approximately 60 mmHg and reduced toward 0.05 µg/kg/min when MAP recovered above this level. This describes the clinical titration approach, not a reconstructed minute-by-minute MAP series.

Approximately 1,000 ml of 0.9% isotonic crystalloid was administered during surgery. Total surgical blood loss was recorded as 800 ml, with the most pronounced bleeding observed during the hysterectomy phase. No packed red blood cells were transfused intraoperatively. Perioperative hemoglobin measurements were 9.5 g/dl at 08:13 (before the recorded surgical start), 9.2 g/dl at 10:41, 9.5 g/dl at 11:26, 9.5 g/dl at 13:21, 8.7 g/dl at 13:52, and 8.4 g/dl at 15:12 (after the recorded surgical end). The discrepancy between the preoperative hemoglobin of 7.8 g/dl and the first perioperative measurement may reflect changes in plasma volume around dialysis and ultrafiltration, although the cause cannot be confirmed from the available records. Transfusion was deferred following repeated assessment of hemoglobin, surgical bleeding, hemodynamic response, and lactate, rather than on the basis of a single hemoglobin threshold.

Toward the end of surgery, arterial blood pressure was 90/52 mmHg while norepinephrine was running at 0.05 µg/kg/min; the corresponding calculated MAP was approximately 65 mmHg. Final arterial blood gas analysis showed pH 7.391, $PaCO_2$ 31.1 mmHg, HCO_3^- 18.4 mmol/L, base excess −5.7 mmol/L, lactate 1.13 mmol/L, potassium 4.06 mmol/L, and ionized calcium 1.11 mmol/L. Persistently low lactate concentrations and acceptable potassium levels on serial intraoperative measurements supported the absence of marked global hypoperfusion or clinically important hyperkalemia.

Norepinephrine was discontinued before emergence. The patient was extubated in the operating room and transferred to the intensive care unit without a requirement for vasopressor support.

The postoperative course was clinically stable without a persistent vasopressor requirement. The early postoperative hemoglobin measurement at 15:12 was 8.4 g/dl. Maintenance hemodialysis was resumed on postoperative day (POD) 2, with an ultrafiltration volume of 2,800 ml. The patient was transferred from the intensive care unit to the surgical ward on POD 3. At that time, lactate was 1.22 mmol/L and potassium was 3.99 mmol/L. Reduced bicarbonate and increased chloride concentrations in the presence of normal lactate were consistent with mild hyperchloremic metabolic acidosis.

The patient subsequently continued her regular three-times-weekly hemodialysis schedule. During the pre-discharge hemodialysis session on POD 11, 3,500 ml was removed by ultrafiltration. She was discharged in clinically stable condition on POD 12.

DISCUSSION

The principal anesthetic challenge in this case was balancing the maintenance of adequate perfusion during extensive surgery against the avoidance of preventable fluid loading in a patient with virtually absent renal fluid clearance.

Although patients with dialysis-dependent kidney failure face increased perioperative risk, evidence specific to many perioperative treatment decisions in this population remains limited.[1,2] Contemporary reviews emphasize individualized assessment of volume status, electrolytes, anemia,

cardiovascular risk, and dialysis timing in patients receiving maintenance hemodialysis.[3]

The 2024 PeriOperative Quality Initiative (POQI) consensus recommends individualizing perioperative fluid therapy according to surgical characteristics, intravascular volume status, and comorbidities.[4] Similarly, the POQI consensus on arterial pressure management recommends treating hypotension according to its likely underlying cause rather than adopting a uniform approach.[5]

This distinction was particularly important in our patient. General anesthesia and epidural sympathetic blockade may contribute to vasodilation, whereas repeated crystalloid boluses in a functionally anuric patient could create a fluid burden that cannot subsequently be eliminated through the kidneys. Accordingly, hypotension was not automatically interpreted as hypovolemia: both vasodilation and blood loss were plausible contributors, and no advanced measure was available to quantify their respective roles. Fluid administration and vasopressor requirements were evaluated in conjunction with clinical and hemodynamic findings.

Nevertheless, the intention was not fluid restriction per se. In the RELIEF trial, a restrictive fluid regimen in patients at increased risk of complications undergoing major abdominal surgery did not improve disability-free survival and was associated with a higher incidence of acute kidney injury.[6] These findings cannot be directly extrapolated to our patient, who was already dialysis-dependent and underwent bilateral nephrectomy. We cite RELIEF only to support the broader principle that arbitrary fluid restriction may be harmful and that maintaining adequate circulating volume and tissue perfusion remains essential.

In our case, approximately 1,000 ml of isotonic crystalloid was administered while norepinephrine was titrated according to hemodynamic response. This should be regarded as an individualized fluid–vasopressor strategy rather than a fixed restrictive fluid protocol.

Serial metabolic measurements were reassuring with regard to global tissue perfusion. Intraoperative lactate remained between 0.80 and 1.13 mmol/L, and no clinically significant hyperkalemia developed. Norepinephrine could be discontinued before emergence. Lactate and potassium levels also remained acceptable on POD 3, when the patient was transferred from intensive care to the ward. Mild metabolic acidosis with hyperchloremia and normal lactate at that time suggested a hyperchloremic component rather than ongoing global hypoperfusion. The influence of crystalloid composition on acid–base balance has also been examined in nonrenal surgical populations.[7] However, causality cannot be established from a single case.

Preoperative hemodialysis was performed one day before surgery. The documented dry weight was 73 kg, and 2,300 ml was removed during the four-hour heparin-free session. These data provide an objective context for the cautious intraoperative fluid strategy, although ultrafiltration volume alone does not establish that the patient was euvolemic at the start of surgery.

Maintenance hemodialysis was resumed on POD 2 with 2,800 ml of ultrafiltration, followed by continuation of the regular dialysis schedule. A large observational cohort study reported a stepwise association between longer intervals from the last hemodialysis session to surgery and increased postoperative mortality.[8] Although the observational design precludes causal inference, these findings support scheduling dialysis close to elective surgery when appropriate.

Preoperative anemia was another important challenge. The 2023 international guidelines from the American Association of Blood Banks (AABB) support restrictive red blood cell transfusion strategies in most hemodynamically stable adults, while emphasizing that transfusion decisions should account for the overall clinical condition rather than hemoglobin concentration alone.[9]

In this case, despite the preoperative hemoglobin of 7.8 g/dl, serial perioperative values ranged from 8.4 to 9.5 g/dl, including an early postoperative value of 8.4 g/dl. The higher initial perioperative concentration could have reflected dialysis-related plasma-volume changes, but this remains unverified. The decision not to administer an intraoperative red blood cell transfusion was individualized on the basis of serial hemoglobin measurements, recorded blood loss, hemodynamic response, low lactate concentrations, and the absence of documented signs of inadequate global perfusion. These observations do not exclude occult hypoperfusion or establish the absence of hypovolemia.

Current POQI guidance describes goal-directed hemodynamic therapy as a protocolized approach involving fluids, vasopressors, and inotropes guided by monitoring and physiological targets.[10] Therefore, it would not be appropriate to describe our management as a formal goal-directed hemodynamic therapy protocol. Instead, we used a pragmatic, patient-specific, physiology-guided approach based on invasive arterial pressure monitoring, serial blood gas measurements, assessment of surgical losses, cautious fluid administration, and titrated norepinephrine.

This report has important limitations inherent to a single case. Advanced cardiac output monitoring or dynamic indices of fluid responsiveness were not used, and perioperative volume status was not quantitatively determined using a dedicated objective method. The timing of the preoperative hemoglobin sample relative to dialysis was not independently verified; therefore, the proposed plasma-volume explanation remains speculative. Although dry weight and ultrafiltration volumes were obtained from dialysis records, these measurements alone cannot establish intraoperative euvolemia. Therefore, the favorable clinical course does not demonstrate superiority of the described fluid–vasopressor strategy or support a fixed fluid or vasopressor regimen for other dialysis-dependent patients.

CONCLUSION

This case demonstrates the feasibility of individualized hemodynamic management in an anuric, hemodialysis-dependent patient undergoing extensive multivisceral surgery. Cautious crystalloid administration, titrated

norepinephrine support, invasive arterial monitoring, and serial metabolic assessments were associated with a stable perioperative course without a marked lactate rise, severe hyperkalemia, or persistent vasopressor requirement.

The key principle is not fluid restriction itself but preservation of adequate tissue perfusion while avoiding unnecessary volume loading. Observations from this single case should not be interpreted as evidence supporting any fixed fluid or vasopressor regimen.

ETHICAL DECLARATIONS

Informed Consent

Written informed consent was obtained from the patient included in this report. Signed consent forms are retained by the authors and are available upon request.

Peer Review Process

This report underwent external peer review.

Conflict of Interest

The authors declare no conflicts of interest.

Financial Disclosure

This case report did not receive any financial support.

Author Contributions

Concept: MSD, ASTY, BAG; Design: MSD, ASTY, BAG; Control/Supervision: MSD, ASTY, BAG; Resources: MSD, ASTY, BAG; Materials/Clinical Case Management: MSD, ASTY, BAG; Data Collection and/or Processing: MSD, ASTY, BAG; Analysis and/or Interpretation: MSD, ASTY, BAG; Literature Review: MSD, ASTY, BAG; Article Writing: MSD, ASTY, BAG; Critical Review: All Authors.

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