Anesthetic Considerations for Patients with Protein-Losing Nephropathy

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Pathophysiology

Protein-losing nephropathy (PLN) in dogs and cats is characterized by the abnormal urinary loss of plasma proteins, primarily albumin, due to glomerular damage. The glomerular filtration barrier consists of three components: the endothelial cells, the glomerular basement membrane, and the podocytes. In PLN, this barrier becomes compromised, allowing plasma proteins to leak into the urine.

The underlying causes of PLN vary widely, including immune-mediated glomerulonephritis, amyloidosis, infectious diseases (Lyme disease, leptospirosis), neoplasia, and certain drugs. Regardless of etiology, the common pathway leads to proteinuria, hypoalbuminemia, loss of antithrombin III, and subsequent complications that significantly impact anesthetic management.

As protein loss progresses, the decrease in plasma oncotic pressure leads to fluid redistribution from the intravascular to interstitial space, resulting in edema formation and potential hypovolemia. Concurrently, the body attempts to compensate by increasing hepatic protein synthesis, which can deplete other essential nutrients and energy reserves. The protein loss can also affect drug-protein binding, altering pharmacokinetics of anesthetic agents, although this is generally a minor issue.

Kidney Function Concerns

Patients with PLN often develop varying degrees of renal insufficiency or failure. The primary concerns related to kidney function during anesthesia include:

Reduced glomerular filtration rate (GFR) affects drug clearance, potentially prolonging the effects of anesthetic agents that rely on renal excretion. This altered pharmacokinetics necessitates careful dose adjustments of renally-cleared drugs.

Azotemia results in accumulation of uremic toxins, which can affect the blood-brain barrier and increase sensitivity to central nervous system depressants, including most anesthetic agents.

Compromised renal autoregulation makes these patients particularly vulnerable to perioperative hypotension, which can further exacerbate renal injury and decrease GFR.

Coagulation Concerns

Coagulation abnormalities in PLN patients stem from multiple mechanisms:

Antithrombin III (ATIII) loss: Being a relatively small protein, ATIII is readily lost in the urine, predisposing patients to a hypercoagulable state and increasing the risk of thromboembolic complications during the perioperative period.

Platelet hyperaggregability: Uremic toxins can paradoxically enhance platelet aggregation despite concurrent platelet dysfunction, creating a complex hemostatic picture.

Altered fibrinolytic activity: Imbalances between procoagulant and anticoagulant factors can lead to abnormal clot formation or premature breakdown.

Preoperative Assessment and Preparation

Complete evaluation of renal function through blood urea nitrogen, creatinine, electrolytes, and urinalysis is essential. Quantification of proteinuria using urine protein:creatinine ratio helps assess disease severity.

Thorough assessment of hydration status and intravascular volume is critical, as these patients may appear edematous despite being intravascularly depleted.

Coagulation profile including prothrombin time, activated partial thromboplastin time, and fibrinogen levels should be evaluated if surgery with blood loss is anticipated. Consider thromboelastography if available for comprehensive hemostatic assessment.

Albumin levels should be measured, with severe hypoalbuminemia (<2.0 g/dL) potentially warranting preoperative colloid administration.

Blood pressure measurement and echocardiography may be indicated to assess cardiovascular status, particularly in patients with suspected hypertension or volume abnormalities.

Stabilization of electrolyte abnormalities, particularly potassium and sodium imbalances, before anesthetic induction is crucial.

Fluid Therapy Management

Crystalloids should be administered judiciously, typically at maintenance rates (2-3 mL/kg/hr) to prevent volume overload, dilution of albumin, and coagulopathy due to further disruption of the balance of the coagulation factors.

Colloids (synthetic or natural) may be necessary to maintain intravascular volume and oncotic pressure. Synthetic colloids should be used cautiously due to potential adverse renal effects

Blood pressure should be maintained above 60-65 mmHg mean arterial pressure to ensure adequate renal perfusion. Hypotension should be addressed promptly with reduced anesthetic depth, appropriate fluid boluses, inotropes, and vasopressors if necessary.

Diuretics should generally be avoided unless specifically indicated for management of pulmonary edema or refractory ascites.

Perioperative Monitoring

Continuous ECG monitoring is essential due to increased risk of arrhythmias from electrolyte disturbances.

Blood pressure measurement at frequent intervals (ideally continuous, invasive monitoring) helps detect and address hypotension promptly.

Temperature monitoring prevents hypothermia, which can worsen coagulation abnormalities and delay drug metabolism.

Postoperative Care

Continued fluid therapy management with careful monitoring of input/output balance and adjustment based on clinical parameters.

Thromboprophylaxis with low-dose heparin or other anticoagulants may be warranted in high-risk patients.

Aggressive multimodal analgesia using opioids, NSAIDs (if renal function permits), and adjunctive analgesics helps control pain while minimizing opioid requirements.

Close monitoring for signs of uremic crisis, fluid overload, or thromboembolic complications is essential during the postoperative period.

Regular assessment of kidney function parameters helps guide ongoing management and detect potential deterioration early.