Renal Replacement Therapy in Abdominal Blunt Trauma with Uncompromised Hemodynamics: A Case Report
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1. Introduction

Kidneys are the most trauma-prone genitourinary organs involved in up to 3.25% of trauma patients. As many as 10% of kidney trauma is caused by blunt abdominal trauma, especially in cases of traffic accidents.¹ On a scale from 1 (hematuria with radiological examination within normal limits or subcapsular contusion that does not extend) to 5 (kidney damage or devascularization with ureteropelvic avulsion and complete laceration or thrombus of the main renal artery or vein) which has a poor outcome on a renal function to has the potential to cause acute kidney injury if adequate therapeutic management is not carried out.²,³

Acute kidney injury (AKI) is not a disease but a clinical syndrome with multiple etiologies and is often the result of several concurrent renal disorders. The definition of AKI according to Kidney Disease: Improving Global Outcome (KDIGO) is one of the following, namely 1) an increase in serum creatinine of 0.3 mg/dL in 48 hours; or 2) an increase in serum creatinine to 1.5 times the baseline level, which is known or suspected to have occurred in the past week; or 3) urine volume less than 0.5 mL/kg/hour for 6 hours.⁴

The nonoperative management approach is based on a better understanding of the ability of the kidney to maintain homeostasis under conditions of injury,
including renal replacement therapy. However, emergency nephrectomy remains the standard treatment for uncontrolled acute renal bleeding.\(^5\) Conservative management can lead to complications that include urinoma, perinephric abscess formation, delayed bleeding, and renovascular hypertension that need to be actively evaluated for maximum outcome.\(^6\) Renal replacement therapy begins when oligoanuria with multiple organ system failures occur. Delaying dialysis with supportive therapy can provide a faster and more spontaneous kidney recovery. A study by Santucci and Fisher in a meta-analysis reported that 90% of cases of grade IV and V renal trauma were manageable without surgery.\(^6,7\) This study aimed to present a case of hemodynamically stable blunt abdominal trauma treated with renal replacement therapy and supportive therapy to control complications in the intensive care unit of Dr. Moewardi General Hospital with a multidisciplinary team approach.

2. Case Presentation

A 34-year-old woman came to the emergency room of Dr. Moewardi General Hospital with complaints of right back pain after a traffic accident in the last 2 days. The patient was referred to a tertiary hospital due to blunt abdominal trauma with grade V right kidney laceration and intra-abdominal bleeding. The patient admitted the pain was felt after being hit by the motorcycle handlebar during a traffic accident 2 days ago and was treated at Kelet Jepara Hospital for 1 day with and radiological CT scan of the abdomen without contrast, plain chest X-ray, and urinary catheter placement and pharmacological therapy were performed. The patient was given normosaline 0.9 infusions, repairment of coagulation (tranexamic acid 500 mg per 12 hours IV and vitamin K 1 ampoule per 8 hours IV), prophylactic antibiotics with minimal risk of nephrotoxicity (ampicillin sulbactam 1 gram every 8 hours IV), dipyrone (metamizole 1 g per 8 hours IV), anti-nausea and vomiting (ondansetron 8 mg 8 hrly IV and ranitidine 50 mg 12 hrly IV), vasopressor (dobutamine 5 mcg/kg/minute and NE 0.1 mcg/kg/minute) and diuretic (furosemide 40 mg IV every 24 hours) hours IV) as well as a PRC transfusion of 4 kolf. At the time of insertion of a 16 Fr Foley catheter, gross hematuria was found, and a referral was made for further treatment.

Physical examination at Dr. Moewardi General Hospital, the general condition of the patient was moderately ill and comosp mentis. The patient’s vital signs were within normal limits, including blood pressure 121/66 mmHg, pulse rate 94 times/minute, respiratory rate 18 times/minute, and temperature of 36.5\(^\circ\)C. On abdominal examination, there was epigastric injury, and bowel sounds were within normal limits and tenderness (VAS 5) in the epigastric, right hypochondriacal, and umbilical regions. On urological examination, right costovertebral tenderness was found. Physical examination did not reveal any manifestations of fluid overloads, such as fine wet crackles or edema in the extremities.

Work-up examination on serial laboratory examinations revealed a decrease in hemoglobin value and a decrease in kidney function. Chest X-ray examination showed no abnormalities. An abdominal CT scan without contrast revealed renal laceration and intra-abdominal hemorrhage. Urinalysis examination revealed protein (++/plus 2) and erythrocytes (+++/plus 3). The patient was admitted to the intensive care unit for further monitoring with therapy in the form of a 1500 kcal renal diet, 0.9% normosaline infusion, repairment of coagulation (1 gram tranexamic acid per 12 hours IV), prophylactic antibiotics with minimal risk of nephrotoxicity (ampicillin sulbactam 1 gram every 8 hours IV), dipyrone (metamizole 1 g per 8 hours IV), anti-nausea and vomiting (omeprazole 40 mg per 12 hours IV, ranitidine 50 mg per 12 hours IV and metclopramide 1 amp per 8 hours IV).

On the 5\(^{th}\) day of treatment, there was a decrease in the general condition of the patient from a clinical perspective, namely complaints of shortness of breath (increased respiratory rate 34 times/minute) with a saturation of 94% oxygenation on a non-
rebreathing mask of 12 liters per minute. The patient was consulted by the Pulmonology Department, and physical examination found fine wet crackles on both lung bases and edema in the lower extremities. The patient also developed a decrease in diuresis value <0.5ml/kgBW/hour and a decrease in kidney function (Ur 271 mg/dl and Cr 10.7 mg/dl). Patients were given additional therapy including resuscitation (inf kidmin 1 fl/24 hours), diuretics (furosemide 1 cc/hour), renoprotection (NAC 1 every 8 hours, folic acid 800 mcg every 24 hours, and VIP albumin every 8 hours). Complaints of shortness of breath did not improve, and the patient was given a breathing apparatus with a ventilator in the setting of SIMV PC PI 10, PS 10 trig 2, FiO₂ 80%. Blood gas analysis showed respiratory compensated metabolic acidosis.

Serial laboratory examinations on day 5 of treatment revealed electrolyte balance disorders in the form of moderate hyperkalemia (K 6.6 mmol/L). The patient was consulted to the Internal Medicine Department and given therapy which included Ca gluconate 1 gram per 24 hours IV and D40% 2 flabot combinations of 10 insulin units (3 x 30 minutes interval) IV and evaluated every 3 days and planned to get renal replacement therapy.

On the 10th day of treatment showed a decrease in mental status to somnolence, creatinine >5.0 mmol/L, urine output <0.5ml/kgBW/hour, and lactate 1.10. Microbiological examination through catheter urine samples showed positive findings for yeast, so the patient was diagnosed with CAUTI and given broad-spectrum antibiotics with meropenem 2 grams every 8 hours IV, which showed improvement until day 17. The patient underwent hemodialysis on the 12th day of treatment with the installation of a double lumen HD cath and there was an improvement in the patient’s diuresis (>0.5ml/kgBW/hour) and extubated with 96% saturation on nasal cannula at 4 lpm. The patient was transferred to the ward after stabilization, and a comprehensive multidisciplinary evaluation was performed. On the 18th day of treatment, the patient was admitted to an outpatient setting and planned for routine follow-up and evaluation of long-term complications in acute renal failure.

3. Discussion

Acute renal failure is defined as a sudden decrease (within 48 hours) of renal function based on an increase in serum creatinine level and a decrease in urine output. Causes of acute kidney injury can be divided into three categories prerenal (decreased renal perfusion, often due to decreased volume), intrinsic renal (processes within the kidney), and postrenal (inadequate urinary drainage distal to the kidney). Intrinsic renal causes are an important source of renal failure). Acute intra-renal failure can be categorized by the component of the kidney that is primarily affected (i.e., tubular, glomerular, interstitial, or vascular). Acute events involving the renal arteries or veins may also cause intrinsic acute kidney injury. Acute vascular causes of renal injury usually require a radiological examination to confirm the diagnosis. Clinical presentation varies with the cause and severity of the kidney injury and associated disease. Most patients with mild to moderate acute kidney injury are asymptomatic and identified on laboratory testing. Patients with severe cases may present with confusion, fatigue, anorexia, m nausea, vomiting, or edema. Patients may also present with oliguria, anuria, or normal urine volume (no oliguric acute renal failure). In this case, the patient had a history of blunt abdominal trauma with clinical manifestations of injury and pain in the epigastic region and minimal gross hematuria and grade V renal laceration on radiological examination, suggesting acute renal failure with intrinsic renal vascular etiology.

It is important to compare the patient’s current serum creatinine level with previous levels to determine the duration and onset of the disease. The definition of acute kidney injury indicates that an increase in creatinine had occurred within 48 hours, although in an outpatient setting, it may be difficult to ascertain when the increase occurred. A high serum creatinine level in a patient with previously
recorded normal levels indicates an acute process, whereas an increase over weeks to months suggests a subacute or chronic process. Serial laboratory examinations every three days reveal a creatinine increase of 3.4 mg/day. dl to 10.7 mg/dl accompanied by a decrease in diuresis <0.5 ml/kgBW/hour. Urinalysis is the most important noninvasive test in the initial assessment of acute kidney injury. Findings on urinalysis guide the differential diagnosis and direct further investigation. Analysis of urine, hematocrit and creatinine levels are the necessary tests to diagnose microscopic hematuria, current blood loss status, and baseline renal function. Hematuria, visible or invisible, is a very common sign of renal trauma. Nearly half of patients with grade II renal injury and 30% of patients with grade IV renal injury have no hematuria at clinical presentation. Visible hematuria is even less common with acute renal injury. Therefore, there is no absolute relationship between the types. Or the degree of hematuria and the type and severity of the injured kidney. On urinalysis, the patient, found microscopic hematuria +++/plus 3 with a history of gross hematuria on foley catheter insertion 16 Fr. According to these data, hemodynamically stable grade IV-V blunt renal trauma patients can be treated nonoperatively with active monitoring. The study by Altman et al., who managed 13 patients with grade V renal injury, reported successful nonoperative management for 6 patients with adequate renal parenchymal function on additional CT scans. A study by Buckley and McAnninch reported one-third of patients with grade IV blunt kidney injury, none of whom required delayed nephrectomy. Syariah et al. succeeded in reporting 41 of 51 patients with grade IV blunt kidney injury (80%) who were treated nonoperatively. Several predictors of nonoperative management failure have been identified by Toutouzas et al., including abdominal injury, ages >55 years old, ongoing blood transfusions, and worsening acidosis. Fluid resuscitation management is required because of the decrease in intravascular volume. Isotonic solutions (normal saline) are preferred over hyperoncotic solutions (dextans, hydroxyethyl starch, albumin). The goal of fluid resuscitation is to maintain a mean arterial pressure greater than 65 mm Hg, and vasopressors may also be administered in patients with persistent hypotension. Cardiac function can be optimized as needed by positive inotropes or reduction of afterload and preload. In this case, the patient was given a normal infusion of 0.9% saline and 1 flabot kidmin every 24 hours. Administration of dobutamine as a vasopressor is given to patients to maintain circulatory function, especially at the onset of the disease where intra-abdominal bleeding has not been adequately controlled, which can lead to resistant hypotension, while dopamine is contraindicated in patients with acute renal failure. Evaluation of electrolyte imbalances (e.g., hyperkalaemia, hyperphosphatemia, hypermagnesemia, hyponatremia, hypernatremia, metabolic acidosis) is important. Severe hyperkalaemia i.e., potassium >6.5 mEq per L (6.5 mmol per L) or <6.5 mEq per L with electrocardiographic changes typical of hyperkalaemia (e.g., tall T waves). Treatment may be given 5 to 10 units of regular insulin. And 50% dextrose, given intravenously, can shift potassium out of the circulation and into cells. Calcium gluconate (10 mL of 10% solution infused intravenously over five minutes) is also used to stabilize the membrane and reduce the risk of arrhythmias when electrocardiographic changes suggest hyperkalaemia. In this case, the potassium value was 6.0 without arrhythmia disorders, and the patient was treated with Ca Gluconas 1 gram per 24 hours IV and D40% 2 flabot combinations of 10-unit insulin (3 x 30 minutes interval) IV and evaluated every 3 days, which showed an improvement trend. Fluid overload is often found in AKI patients in the ICU. Fluid overload of at least 10% is associated with increased mortality and morbidity, including pulmonary edema, heart failure, delayed wound healing, tissue damage, and impaired bowel function.
Optimization and management of fluid therapy are difficult. Diuretics are considered first-line treatment but only in responsive subjects. When selecting a diuretic, the clinician should consider the evidence-based indications, possible side effects, compatibility, pharmacokinetics, and other concerns of a particular diuretic. The severity of AKI alters both the pharmacokinetics and pharmacodynamics of furosemide, making precise dosing of this agent difficult. Adjustment of increasing doses of loop diuretics is associated with a worse prognosis of AKI in patients with congestive heart failure and in other groups of critically ill subjects.\textsuperscript{15,16} The use of high-dose furosemide to convert oliguric to non-oliguric AKI may exacerbate renal injury (increased stress markers). Oxidative stress, especially in hypovolemic patients with decreased renal perfusion. However, the physiological effects of diuretics help in reducing kidney injury. Furosemide acts by inhibiting the active $\text{Na}^+$/K$^+$/Cl$^-$/ co-transport pump on the surface of the luminal cell membrane of the thick ascending branch of the medulla of the loop of Henle. Tubular sodium reabsorption is the mechanism for most of the oxygen consumption in the ischemic-damaged outer medulla. In addition, furosemide has been shown to attenuate apoptosis after ischemia-reperfusion injury in experimental models. Furosemide causes greater water loss than sodium loss and results in hypotonic urine production. Loop diuretics also cause increased urinary excretion of potassium, calcium, and magnesium by inhibiting the passive reabsorption of these ions. More than 95\% of loop diuretics are bound to albumin. Therefore, they do not undergo glomerular filtration.\textsuperscript{14,15,17} They reach their target site by active secretion from the blood into the urine by organic acid transporters present in the proximal tubule. Hypoalbuminemia (which is common in patients with AKI in the ICU) results in decreased secretion into the tubules and increased clearance of loop diuretics. Therefore, the diuretic effect of loop diuretics is greatly diminished in the presence of hypoalbuminemia. There are theoretical reasons for combining loop diuretics with albumin infusion. A meta-analysis of 10 studies has concluded that in hypoalbuminemia patients, better fluid balance is achieved when furosemide and albumin are combined. In patients with normal albumin levels, this combination may not be beneficial. The distribution of loop diuretics into the tubules is also decreased in the presence of renal dysfunction. The decrease in renal blood flow, which is not uncommon in patients with AKI, exacerbates this decrease in secretion. Metabolic acidosis further decreases tubular loop diuretic secretion.\textsuperscript{9–11} In patients with AKI, the dose of diuretic required to achieve diuresis may be much higher than usual. It is also important to understand that there is a threshold dose of loop diuretic below it, which will not have any therapeutic effect. There is also an upper limit dose for which there is no added benefit. Once this dose is reached, diuretics acting at other locations of the nephron, e.g., thiazide diuretics, may be added for additional effect. Electrolyte levels (especially potassium) should be monitored regularly when this combination diuretic is used.\textsuperscript{18} In this case, the patient was given diuretics after complaints of shortness of breath that led to complications, namely pulmonary edema. The dose of furosemide was adjusted to 10 mg/hour and was given in combination with acetazolamide every 8 hours and albumin. However, the diuresis function in the patient has not shown adequate improvement, so the patient is still undergoing renal replacement therapy.

Conditions of hypovolemia (renal hypoperfusion) and hypervolemia (renal congestion) impair kidney function. Impaired heart function adds to the problem as kidney and heart dysfunction exacerbate each other, known as a cardiorenal syndrome. Increasing symptom severity requires increased therapeutic intervention. In apparently euvoemic patients with AKI, a single bolus of buffered crystalloid fluid may indicate subclinical hypovolemia and renal hypoperfusion (prerenal AKI). Prolonged administration of balanced crystalloids should be handled with care so as not to cause edema.
or congestion and not to decrease tissue oxygenation. Patients with hypervolemia should not receive fluids for AKI but loop natriuretics. In critically ill patients with suppressed vasomotor responses, vasopressors are often needed to increase cardiac output. Supportive therapy (ie, antibiotics, maintenance of adequate nutrition, mechanical ventilation, glycemic control, anemia management, and use of renoprotection) should be carried out according to standard management. Patients were given antibiotics with minimal risk of nephrotoxicity, namely ampicillin, sulbactam, and meropenem, mechanical ventilation, and renoprotection, namely NAC and folic acid, according to the KDIGO guidelines in acute renal failure.

Indications for renal replacement therapy in the setting of oligoanuric AKI include life-threatening situations such as hyperkalaemia, acidemia, pulmonary oedema and complications of uremia such as pericarditis, altered mental status (uremic encephalopathy), or bleeding. Some non-life-threatening situations in AKI that may benefit from dialysis therapy include solute control, as in tumour lysis syndrome, acid-base disorders such as permissive hypercapnia in ventilated patients, and volume overload. Fluid accumulation is a critical consequence of oligoanuric AKI. and can cause complications. Controlling fluid overload can allow for unrestricted feeding and adequate drug therapy. This is often attempted with loop diuretics such as intermittent infusion or continuous infusion of furosemide, bumetanide, or torsemide in an attempt to convert oligoanuric AKI to non-oliguric AKI for better fluid management. However, several observational studies have shown poorer AKI outcomes after loop diuretic use which may be related to confounding indications, in which patients with poor prognosis are more likely to accept this. A study by Bouman et al. in 106 patients on renal replacement therapy for early-onset (immediately after meeting criteria for AKI) or delayed (following the development of hyperkalaemia, pulmonary oedema, or plasma urea 440mmol/l) showed no difference in mortality rates. A single-center study in India that followed 208 patients (mean age 42 years) with AKI showed no difference in outcome with regard to initiation of renal replacement therapy.

The kidneys maintain homeostasis; hence, acute kidney injury (AKI) affects almost all body systems, although in different ways. Fluid retention primarily affects the lungs and heart, often with clinical signs of respiratory or circulatory failure. Fluid retention also irritates the gastrointestinal system, i.e., the liver or intestines, causing intestinal barrier dysfunction and translocation of bacteria and bacterial toxins. Impaired uremic toxin excretion affects the brain, heart, bone marrow, and immune system function, causing neurocognitive defects, anemia, and acquired immune deficiency accompanied by persistent systemic inflammation. Renal cell necrosis releases debris into the venous circulation, which accumulates in the lungs and causes direct microvascular injury, thrombosis, and, occasionally, acute respiratory distress syndrome. Common complications associated with renal trauma include infection, urine leakage, loss of kidney function, and hypertension. Urinary tract infections are the most common among patients admitted to intensive care, and in certain cases, a perinephric abscess may develop. Sepsis accounts for approximately 50% of all patients with AKI in the intensive care unit (ICU) and is the leading cause of death in the ICU. Mortality from sepsis-associated AKI cannot be explained solely by loss of renal function, but rather AKI-induced multiorgan dysfunction, including lung, heart, brain, liver, and gut, contributes to overall mortality in sepsis-associated AKI. The pathophysiology of AKI in sepsis is complex and multi-factorial and includes intrarenal hemodynamic changes, endothelial dysfunction, inflammatory cell infiltration in the renal parenchyma, intraglomerular thrombosis, and tubular obstruction with necrotic cells and debris. The patient had signs of sepsis on the 11th day of treatment and was given prophylactic broad-spectrum antibiotics, namely meropenem 2 grams.
every 12 hours, and periodic evaluation.

4. Conclusion

The spectrum of renal injuries ranges from mild to advanced, which can be treated with renal replacement therapy to nephrectomy. Management of acute renal failure involves fluid resuscitation, avoidance of nephrotoxic drugs and exposure to contrast media, and correction of electrolyte imbalances. Renal replacement therapy (dialysis) is indicated for refractory hyperkalemia; excess volume; acidosis; uremic encephalopathy, pericarditis, or pleurisy; and certain toxic clearances. Recognition of risk factors, for example, older age, sepsis, hypovolemia/shock, cardiac surgery, contrast agent infusion, diabetes mellitus, pre-existing chronic kidney disease, heart failure, and liver failure, is important. A multidisciplinary approach to prevention, early diagnosis, and therapeutic management are critical to improving outcomes.

5. References


