Juniper Online Journal of Case Studies
ISSN: 2476-1370

Juniper Publishers
key to the Researchers

Case Report
Volume 7 Issue 1 - May 2018
DOI: 10.19080/JOJCS.2018.07.555703

JOJ Case Stud
Copyright © All rights are reserved by Ebru Canakci

Acute Tubular Necrosis after Nephrectomy: Case Presentation

Ebru Canakci[1]*, Ahmet Karatas[2], Ahmet Gultekin[1], Zubeyir Cebeci[1], Ilker Coskun[1] and Anıl Kılınc[1]

[1]*Department of Anaesthesiology and Reanimation, Ordu University, Turkey*

[2]*Department of Internal Medicine & Nephrology, Ordu University, Turkey*

Submission: May 07, 2018; **Published:** May 18, 2018

***Corresponding author:** Ebru Canakci, Faculty of Medicine, Department of Anaesthesiology and Reanimation, Ordu University, Ordu, Turkey, Email: canakciebru@gmail.com

Abstract

Acute renal failure (ARF) has a clinical presentation with declining renal function and glomerular filtration rate within hours-days. Ischemic ATN, contrary to prerenal azotemia, is not immediately cured upon the recovery of renal perfusion. In its severe form, renal hypoperfusion leads to bilateral renal cortical necrosis and irreversible renal insufficiency. Ischemic ATN often develops as a result of major surgical intervention, trauma, severe hypovolemia, sepsis and severe burns. Acute kidney injury (AKI) is one of the frequently encountered causes of morbidity and mortality in hospitals. The aim of this study is to present the case with ATN after major surgery and subsequent permanent kidney injury in light of the information from the literature.

Keywords: Nephrectomy; Acute Tubular Necrosis; Hemodialysis

Introduction and Objective

Acute renal failure (ARF) has a clinical presentation with declining renal function and glomerular filtration rate within hours-days. Although there are several differences in the definition; it can be described as an increase in the serum creatinine level; at a rate of at least 50% of the basal level or more than 0.5mg/dL [1]. According to "Kidney Disease Improving Global Guidelines (KDIGO) 2012" AKI is diagnosed if any one of the following three conditions are present:

a) Increase in the serum creatinine levels by 0.3mg/dL or over within 48 hours

b) Known or estimated increase in the serum creatinine levels by ≥1.5 fold of the basal level within the last 7 days

c) Micturation of < 0.5ml/kg/hr within the last 6 hours.

Ischemic or nephrotoxic acute tubular necrosis (ATN) makes up of more than 90% of the renal ARF. Other than these, large vessels, glomerular microvessels and diseases of tubulointerstitium can also lead to renal ARF [2,3]. Ischemic ATN, contrary to prerenal azotemia, is not immediately cured upon the recovery of renal perfusion. In its severe form, renal hypoperfusion leads to bilateral renal cortical necrosis and irreversible renal insufficiency. Ischemic ATN often develops as a result of major surgical intervention, trauma, severe hypovolemia, sepsis and severe burns. Nephrotoxic ATN is linked to endogenous or exogenous toxins. Toxins cause intrarenal vasoconstriction, direct tubular toxicity and/or intratubular obstruction and thus lead to ARF [4]. Acute kidney injury (AKI) is one of the frequently encountered causes of morbidity and mortality in hospitals. The aim of this case presentation is to emphasize the importance of diagnosing AKI that can develop during the postoperative period of a major surgery and planning the appropriate treatment protocol.

Case Presentation

A 52-year-old male patient with no previous history of renal disease was scheduled for left-side nephrectomy with a diagnosis of renal collecting duct tumor. The patient had a history of smoking, with 20 packets/year. He was not on any continuous medication. Hb was 14gr/dL. There were no pathologies in other laboratory parameters. The patient's BMI was 30kg/m2, Mallampati score was Class II and the patient was in ASA II risk level. Consultation with a pulmonologist was requested because of his smoking history. Respiratory function test was within normal levels. The patient was taken to surgery upon requesting sufficient blood. The patient was scheduled for laparoscopic left side nephrectomy. Operation lasted 8 hours. The patient underwent intraoperative ECG monitorization and invasive blood pressure monitorization and his perioperative average arterial pressure was maintained over 60mmHg. Perioperative micturation was closely monitored, it

was maintained at or above 0.5ml/kg/hour. There was 500ml perioperative blood loss, the patient did not have intraoperative blood transfusion. Considering the major surgery and the weight of the patient, intraoperative fluid exigency was calculated and fluid requirement was met. At the end of the surgery, the patient woke up without any complications and firstly taken to recovery unit and then to surgical intensive care. At the postoperative 48th hour, the patient had oligoanuria and had respiratory failure. ATN and acute lung edema was suspected. Serum creatinine level was 7mg/dL and arterial blood gas had metabolic acidosis. Lung x-rays showed Kerley -B lines and stasis ralles were heard in both lungs. Right jugular hemodialysis catheter was placed and the patient was taken to emergency hemodialysis, 4 liters of ultrafiltration was performed. After hemodialysis (HD) the patient still had respiratory problems and thus non-invasive mechanical ventilation was performed. In the follow-up monitorization, serum creatinine levels did not decrease and the patient was taken to hemodialysis every other day. Renal Doppler USG revealed renal cortical necrosis. On his 7th day in the emergency care unit, the patient was transferred to nephrology unit. The patient was followed up for 3 more days in the nephrology unit without any complications and was scheduled for arteriovenous-fistula surgery. After recommending controls in the nephrology outpatient clinic and hemodialysis every other day, the patient was discharged.

Discussion

Clinically, ATN can be analyzed in three phases: Initiation, maintenance, and recovery.

Initiation phase

It is the period where exposure to ischemic or toxic event and damage to parenchyma occur. This phase can last hours-days and ATN can potentially be prevented in this phase.

Maintenance phase

It is the phase where parenchyma damage settles and GFR is stabilized at 5-10ml/min. It usually lasts 1-2 weeks. In this phase, micturation level is at its lowest. Uremic complications mostly occur during the maintenance phase.

Recovery phase

It is the phase of renal tissue repair and regeneration and renal function recovery. It lasts approximately 4 weeks. The initiation of recovery is understood when micturation progressively increases and serum creatinine levels start decreasing within a couple of days. Post-ATN diuresis is based on osmotic diuresis, which is related to the clearance of the accumulated water and salt and solutes. Sometimes, inappropriate and excess amounts of diuresis can be seen. At this phase, problems in fluid-electrolyte balance can be seen [5-7].

ARF treatment strategy depends on the etiology. Prerenal azotemia rapidly regresses upon the recovery of renal perfusion. Hemorrhage is replaced with erythrocyte suspension, and plasma loss (burn, pancreatitis) is replaced with isotonic fluids. Urinary and gastrointestinal losses are usually hypotonic. For this reason, the initial treatment should be with 0.45% NaCl [8].

In postrenal azotemia, the goal is to eliminate the obstruction (catheter, nephrostomy, stent). In 5% of patients, a syndrome, which causes temporary salt loss develops [9]. Renal -intrinsic-ARF treatment can be studied under four headings: precautions, specific treatments, treatment of complications and dialysis [10].

Precautions

Maintaining optimal cardiovascular function and intravascular volume are the most important actions. Diuretics, NSAID drugs, ACE inhibitors and other vasodilators should be used very carefully, especially in individuals with hypovolemic and renovascular disease [11].

Drug levels must be followed up in aminoglycoside and cyclosporine use. Diuretic stress and alkalinization of the urea are useful in urate, methotrexate nephropathy and rhabdomyolysis. N-acetyl cysteine can be preventive in radiocontrast nephrotoxicity and acetaminophen toxicity [12].

Specific treatments

While certain interventions that regulate hemodynamic status and decrease cellular damage were found useful in the experimental studies, their clinical benefits have not been shown [13].

Treatment of complications

The most important points in ARF treatment are the maintenance of fluid-electrolyte balance, improvement of acidosis, improvement of diet, and treatment of uremic signs and symptoms. Treatment approaches should be determined according to the characteristics of the patient [14].

Acute oliguric-intrinsic-renal ARF mortality is around 50% (15% in obstetric patients, 30% in toxin-associated ARFs, and 60% in ARFs after trauma-major surgery). Presence of oliguria and creatinine levels higher than 3mg/dL at the time of diagnosis are indicators of poor prognosis. In elderly patients with multiple organ failure, mortality rate is high. With the failure of more than three organ systems in ARF, mortality is almost 100% [15]. The most common cause of mortality is the infection, followed by fluid-electrolyte imbalances. Death, as a direct complication of uremia, is rare (2.3%) [16]. Renal function is recovered in most patients. In 5% of the patients, however, it is irreversible. In our case, permanent renal injury was suspected and fistula surgery was planned, and the patient was admitted to hemodialysis program (HD) every other day. Approximately 15% of the cases proceed into terminal renal insufficiency. In 50% of the cases, subclinical functional defects remain [17].

Intermittent hemodialysis is the most frequently used renal replacement therapy in ARF. Hemodialysis treatment largely depends on the rate of nitrogenous waste formation and whether

How to cite this article: Ebru Canakci, Ahmet Karatas, Ahmet Gultekin, Zubeyir Cebeci, et al. Acute Tubular Necrosıs after Nephrectomy: A Case Presentatıon. JOJ Case Stud. 2018; 7(1): 555703. DOI: 10.19080/JOJCS.2018.07.555703

fluid tolerance is good. HD treatment of 4 hours, 3 sessions per week provides sufficient replacement for oligoanuric patient. In patients with significant level of residual renal functions (as in non-oliguric patients), weekly number of HD sessions can be less. On the contrary, daily HD can be required for patients with severe hypercatabolism or cannot tolerate excessive fluid [18].

Conclusion

In conclusion, it should be kept in mind that in cases who underwent major surgery, ATN can develop during the postoperative period. Postoperative care after major surgery must be performed in the intensive care unit. Hourly follow up of micturation and hemorrhage, and close hemodynamic follow up must never be neglected. In the case of ARF, cooperation between the intensivist and the nephrologist may ensure that the patient recovers from the renal injury with minimum damage. In tertiary intensive care units, nephrologist must perform like an intensive care nephrologist and must not neglect his/her daily visit to the intensive care unit.

References

1. Lameire N, Van Biesen W, Vanholder R (2005) Acute renal failure. Lancet 365(9457): 417-430.
2. (2012) Kidney Disease Improving Global Guidelines (KDIGO).
3. Kellum JA, Bellomo R, Ronco C (2007) Classification of acute kidney injury using RIFLE: What's the purpose? Crit Care Med 35(8): 1983-1984.
4. Mehta RL, Kellum JA, Shah SV, Molitoris BA, Ronco C, et al. (2007) Acute Kidney Injury Network: report of an initiative to improve outcomes in acute kidney injury. Crit Care 11(2): R31.
5. Schrier RW, Wang W, Poole B, Mitra A (2004) Acute renal failure: definitions, diagnosis, pathogenesis, and therapy. J Clin Invest 114(1): 5-14.
6. Klingebiel T, von Gise H, Bohle A (1983) Morphometric studies on acute renal failure in humans during the oligoanuric and polyuric phases. Clin Nephrol 20(1): 1-10.
7. Mason J, Torhorst J, Welsch J (1984) Role of the medullary perfusion defect in the pathogenesis of ischemic renal failure. Kidney Int 26(3): 283-293.
8. Klingebiel T, von Gise H, Bohle A (1983) Morphometric studies on acute renal failure in humans during the oligoanuric and polyuric phases. Clin Nephrol 20(1): 1-10.
9. Tanner GA (1982) Nephron obstruction and tubuloglomerular feedback. Kidney Int Suppl 12: S213-S218.
10. Brady HR, Clarkson MR, Lieberthal W (2004) Acute renal failure. In Brenner BM (ed), Brenner and Rector's The Kidney. (7th Edn), Philadelphia, Saunders, USA, pp. 1215-1292.
11. Clive DM, Stoff JS (1984) Renal syndromes associated with nonsteroidal antiinflammatory drugs. N Engl J Med 310(9): 563-572.
12. Cooper K, Bennet WM (1987) Nephrotoxicity of common grugs used in clinical practice. Arch Intern Med 147(7): 1213-1218.
13. Lameire NH, De Vriese AS, Vanholder R (2003) Prevention and non-dialytic treatment of acute renal failure. Curr Opin Crit Care 9(6): 481-490.
14. Van Biesen W, Vanholder R, Lameire N (2003) Dialysis strategies in critically ill acute renal patients. Curr Opin Crit Care 9(6): 491-495.
15. D'Intini V, Ronco C, Bonello M, Bellomo R (2004) Renal replacement therapy in acute renal failure. Best Pract Res Clin Anaesthesiol 18(1): 145-157.
16. Jaber BL, Lau J, Schmid CH, Karsou SA, Levey AS, et al. (2002) Effect of biocompatibility of hemodialysis membranes on mortality in acute renal failure: a meta-analysis. Clin Nehrol 57(4): 274-282.
17. Bhandari S, Turney JH (1996) Survivors of acute renal failure who do not recover renal function. QJM 89(6): 415-421.
18. Kaplan AA (1992) Renal replacement therapy for acute renal failure. Current Therapy in Nephrology and Hypertension. Glassock RJ (Ed.), (3rd edn), Mosby-Year Book Inc, St. Louis, USA, pp. 264-273.

This work is licensed under Creative Commons Attribution 4.0 License
DOI: 10.19080/JOJCS.2018.07.555703

Your next submission with Juniper Publishers will reach you the below assets

- Quality Editorial service
- Swift Peer Review
- Reprints availability
- E-prints Service
- Manuscript Podcast for convenient understanding
- Global attainment for your research
- Manuscript accessibility in different formats
  (Pdf, E-pub, Full Text, Audio)
- Unceasing customer service

Track the below URL for one-step submission
https://juniperpublishers.com/online-submission.php

How to cite this article: Ebru Canakci, Ahmet Karatas, Ahmet Gultekin, Zubeyir Cebeci, et al. Acute Tubular Necrosıs after Nephrectomy: A Case Presentatıon. JOJ Case Stud. 2018; 7(1): 555703. DOI: 10.19080/JOJCS.2018.07.555703