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CYSTATIN C VERSUS UREA AND CREATININE IN EARLY DETECTION OF ACUTE KIDNEY INJURY AFTER LAPAROSCOPIC PROSTATECTOMY

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ABSTRACT

Introduction: Acute kidney injury (AKI) is a potentially serious complication subsequent to laparoscopic radical prostatectomy (LRP). Serum creatinine, the prevailing standard biomarker, has demonstrated inadequacy for the early identification of AKI due to its delayed elevation in the initial stages. There is a necessity for innovative biomarkers that facilitate early detection and prompt intervention, hence enhancing patient outcomes.

Objectives: To assess and compare the values of biochemical markers of renal function (serum urea, creatinine, and cystatin C) in the preoperative and postoperative periods in patients undergoing LRP.

Materials and Methods: This prospective, comparative study included 30 patients who underwent LRP. The investigated serum parameters—urea, creatinine, and cystatin C—were assessed at three time points: preoperatively (T1), immediately after LRP (T2), and 12 hours after the start of LRP (T3).

Results: All patients exhibited normal preoperative biochemical indicators of renal function. Postoperatively, AKI was identified in three patients. Out of a total of 30 patients who underwent LRP, only three patients (10%) met the criteria for AKI diagnosis. One patient (3%) developed AKI based on increased serum creatinine levels, while three patients (6%), including the one who met the creatinine-based criteria, were diagnosed with AKI based on elevated serum cystatin C levels. Serum cystatin C levels were significantly elevated in patients with AKI at T2 and remained elevated at T3 in comparison to T1.

Conclusion: The results indicate that serum cystatin C has greater sensitivity for identifying AKI than serum urea and creatinine in the initial 12 hours post- LRP.

Keywords: acute kidney injury, serum creatinine, serum cystatin C

INTRODUCTION

Laparoscopy is becoming prevalent in contemporary surgery and is swiftly supplanting the open technique in numerous surgical disciplines. Despite some benefits, such as expedited surgical recovery and improved esthetic outcomes of the wound, it undoubtedly entails certain hazards for individuals. The elevated intra-abdominal pressure resulting from pneumoperitoneum during laparoscopy might impact the cardiovascular, pulmonary, and renal systems [1]. The renal system is affected by compression of the renal veins and parenchyma, elevated total renal vascular resistance, increased secretion of antidiuretic hormone, and subsequent consequences of diminished cardiac output [1, 2]. In patients with minimal comorbidities, alterations in renal function due to pneumoperitoneum are often temporary and of negligible clinical importance. In patients with pre-existing renal impairment, multiple comorbidities, or extended laparoscopy, pneumoperitoneum may exacerbate renal function deterioration, leading to reperfusion-related ischemic hypoxemia, reduced glomerular filtration rate, oliguria, and ultimately acute kidney injury [2, 3].

Acute kidney injury (AKI) is characterized by a rapid deterioration in renal function, manifested by reduced urine output and diminished elimination of the waste products urea and creatinine, resulting in increased blood levels of urea and creatinine [4]. The widely recognized categorization of AKI is predicated on blood urea and creatinine concentrations, as well as urine output, and encompasses three stages [5]. Recent classifications include additional sensitive indicators, such as serum cystatin C, with serum urea and creatinine [6]. AKI markedly elevates morbidity and death rates, prolongs hospital stays, and escalates expenses [7, 8]. Postoperative AKI elevates the likelihood of chronic renal failure and end-stage renal disease [9]. The prevalence of AKI is 5–10% among all hospitalized patients, 4–13.4% in those receiving major urological and abdominal procedures, 3–12% following laparoscopic surgeries, and as high as 60% in intensive care units [8,9]. Risk factors for AKI may be categorized as preoperative, operative, and postoperative. Preoperative risk factors encompass age, sex, obesity, patient classification per the American Society of Anesthesiologists (ASA) 4 and 5, elevated preoperative serum urea and creatinine levels, use of nonsteroidal anti-inflammatory drugs, contrast media, anemia, blood transfusion, and hyperglycemia. Operative risk factors encompass hypoperfusion, hypotension (mean arterial pressure MAP $\leq$ 60 mmHg), and venous congestion. Postoperative risk factors encompass hypovolemia, hemorrhage, and the use of aminoglycosides and diuretics [10].

The identification of AKI using serum urea and creatinine levels has proven to be inadequately sensitive for early detection, particularly in stage 1, when intervention can most effectively alter the patient's illness trajectory. Blood cystatin C, with normal values ranging from 0.62 to 1.15 mg/L, serves as a biomarker for the early identification of AKI, potentially detecting it up to 24 hours prior to increases in blood creatinine levels. Studies indicate that up to 56% of patients exhibit normal serum creatinine levels in stage 1 of AKI [11]. This paper aimed to ascertain the biochemical parameters of renal function—serum urea, creatinine, and cystatin C—before, during, and 12 hours post-laparoscopic radical prostatectomy (LRP) and to compare the predictive efficacy of serum cystatin C against serum urea and creatinine in the early diagnosis of acute renal failure.

MATERIAL AND METHODS

Study Design

This study was designed as a prospective comparative clinical evaluation conducted at the University Clinic of Urology, Skopje, and the University Clinic for Anesthesiology, Reanimation, and Intensive Care Medicine Faculty of Medicine, Ss. Cyril and Methodius University, Skopje, RN Macedonia. The evaluation included a total of 30 male patients scheduled for LPR between January and December 2024. All patients scheduled for LPR were consecutively enrolled in the study, provided they had no prior history of renal disease and their preoperative laboratory assessments indicated normal kidney function. The study protocol received ethical approval from the Institutional Review Board (IRB) and ethical committee from the medical faculty, and written informed consent was obtained from all participants before inclusion in the investigation.

Patient Selection Criteria

Inclusion criteria encompassed male patients aged 50–75 years with histologically confirmed prostate carcinoma requiring LPR. Patients with pre-existing chronic kidney disease (CKD, stages 3–5 as per KDIGO criteria), uncontrolled hypertension, severe cardiovascular comorbidities (NYHA class III-IV heart failure), and known hypersensitivity to nephrotoxic agents were excluded to minimize confounding factors affecting renal function outcomes.

Examined Parameters

Renal function was assessed through serial biochemical analyses of serum urea, creatinine, and cystatin C. These markers were measured at three predefined time points:

- T1—One hour prior to surgery (baseline measurement)
- T2—Immediately post-surgery
- T3—Twelve hours after the initiation of the surgical procedure

Blood samples were collected under sterile conditions and processed within one hour of collection. Serum marker concentrations were determined using nephelometry on a BN PrOsPEC II platform (Siemens Healthcare Diagnostics, Marburg, Germany) following the manufacturer's guidelines. All laboratory analyses were performed by blinded laboratory personnel to ensure objectivity.

Assessment of AKI

Diagnosis of AKI was established using the most recent 2024 criteria from the Acute Kidney Injury Network (AKIN) and the Kidney Disease Improving Global Outcomes (KDIGO) [6]. The AKI stages were defined as follows:

- Stage 1: Increase in serum creatinine or cystatin C by 1.5 to 1.9 times the baseline value or by ≥0.3 mg/dL, with urine output reduced to ≤0.5 mL/kg/h for 6 hours.
- Stage 2: Increase in serum creatinine or cystatin C by 2 to 2.9 times, with urine output remaining ≤0.5 mL/kg/h for 12 hours.
- Stage 3: Increase in serum creatinine or cystatin C by 3 times or ≥4 mg/dL, with urine output reduced to ≤0.3 mL/kg/h for 24 hours or complete anuria for 12 hours (5).

Anesthesia Protocol

According to the protocol, all patients underwent standard preoperative preparation, including a minimum of six hours of fasting and maintaining normothermia. Two hours before surgery, patients received 5 mg of oral diazepam as premedication. Upon entering the operating room, patients were placed under continuous hemodynamic monitoring using Datex-Ohmeda S/5 Avance (Helsinki, Finland), tracking the following parameters: electrocardiography (ECG), heart rate (HR), non-invasive blood pressure (NIBP) and invasive mean arterial pressure (MAP) every 5 minutes, oxygen saturation (SpO2), capnography (end-tidal CO2, EtCO2 fraction of inspired oxygen (FiO2) and intra-abdominal pressure (9–12 mmHg) via the laparoscopic insufflation system. An intravenous cannula was placed in each patient, and a crystalloid infusion was administered at a rate of 6–12 ml/kg/hr during anesthesia. Prior to induction, patients were preoxygenated with 100% oxygen at 6 L/min for three minutes. General endotracheal anesthesia was induced with 0.04 mg/kg midazolam, 0.002 mg/kg fentanyl, 1–2 mg/kg propofol, and 0.6 mg/kg rocuronium. Following loss of consciousness and cessation of spontaneous respiration, patients were manually ventilated, and two minutes after rocuronium administration, endotracheal intubation was performed. Mechanical ventilation was initiated using Datex-Ohmeda S/5 Avance, in Pressure-Controlled Ventilation-Volume Guarantee (PCV-VG) mode with small lung protective volumes of 6–8 ml/kg, a gas mixture of 50% oxygen and 50% air, an inspiratory-to-expiratory ratio (I:E) of 1:2, respiratory rate adjusted to maintain EtCO2 between 35–45 mmHg, and positive end-expiratory pressure (PEEP) of 5 cm H2O. Anesthesia was maintained using a balanced anesthesia technique with remifentanil (0.05–1 μg/kg/min) and sevoflurane at 1 MAC, ensuring MAP was maintained within ±20% of baseline values. During the procedure, a nasogastric tube was placed for decompression, intraoperative normothermia was maintained using forced-air warming blankets, and anti-embolism pumps were applied to all patients to prevent thromboembolic complications. Postoperative care included standardized

pain management, fluid resuscitation as per institutional protocol (2024 NICE guidelines 25-30ml/kg/day water and 1mmol/kg/day sodium, potassium and chloride and according to the British Consensus Guidelines on IV Fluid for Adults Surgical Patients GIFTASUP recommended low volume maintenance fluid of 1-1.5 ml/kg/hr, with 0.5ml/kg/hr fluid boluses for postoperative oliguria resuscitation need) [12], and serial assessments of renal function up to 24 hours postoperatively. Patients experiencing AKI were managed according to the latest KDIGO recommendations, with nephrology consultation as required.

Statistical Analysis

The data was analyzed using SPSS software (version 27.0, IBM Corp.). Continuous variables were tested for normality using the Shapiro-Wilk test. Parametric data were expressed as mean ± standard deviation (SD) and analyzed using the paired t-test, while non-parametric data were presented as median with interquartile range (IQR) and compared using the Mann-Whitney U test. A p-value <0.05 was considered statistically significant.

RESULTS

Out of a total of 30 patients who underwent LRP, only three patients (10%) met the criteria for AKI diagnosis. One patient (3%) developed AKI based on increased serum creatinine levels, while three patients (6%), including the one who met the creatinine-based criteria, were diagnosed with AKI based on elevated serum cystatin C levels.

Table 1 presents the demographic characteristics of the study population and biochemical parameters of kidney function, including serum creatinine, urea, and cystatin C. The mean age of patients without AKI was 61±11 years, while the mean age of patients with AKI was 65±10 years. The average duration of LRP was 125.4±52.9 minutes in patients who did not develop AKI, whereas the procedure lasted significantly longer (140.6±68 minutes) in those diagnosed with AKI ($p<0.05$).

Among patients who developed AKI, a history of prior surgical interventions was noted in all cases (3 patients, 10%), whereas none (0%) of the remaining 27 patients (90%) without AKI had a history of previous surgical procedures.

Table 1. *Demographic Data and Clinical Characteristics of Patients Undergoing Laparoscopic Radical Prostatectomy (LRP) Stratified by AKI Criteria*

Variable	Non-AKI (n=27) (Mean ± SD)	AKI (n=3) (Mean ± SD)
Age (years)	61 ± 11	65 ± 10
Surgery duration (minutes)	125.4 ± 52.9	140.6 ± 69.8
Previous surgeries [n (%)]	0 (0%)	3 (10%)

Table 2. *Descriptive Statistics for Cystatin C, Urea, and Creatinine Before and After Laparoscopic Prostatectomy in All Patients (Entire Group)*

Parameter	T1	T2	T3
Creatinine (0.7–1.3 mg/dl)			
Mean ± SD (mg/dl)	0.63 ± 0.12	0.79 ± 0.1	1.01 ± 0.12
Min - Max	0.48 - 0.9	0.62 - 1.04	0.89 - 1.47
Cystatin C (0.62–1.15 mg/dl)			
Mean ± SD (mg/dl)	0.66 ± 0.08	0.81 ± 0.1	1.02 ± 0.13
Min - Max	0.51 - 0.9	0.6 - 1	0.9 - 1.35
Urea (7–20 mg/dl)			
Mean ± SD (mg/dl)	10.98 ± 2.46	15.26 ± 2.46	18.62 ± 2.3
Min - Max	8.4 - 17.36	12.32 - 22.12	14 - 25.76

T1 – 1 hour before surgery; T2 – 1 hour after surgery; T3 – 12 hours after surgery.

Table 2 presents the descriptive statistics for the three biochemical markers in all patients (entire group) at three time points. The obtained results indicate that the mean values for all three serum biochemical markers—creatinine, cystatin C, and urea—remained within the reference range at all three measured time points. On time point 3, three patients exhibited values exceeding the upper limit of the normal range.

Graph 1 presents the serum creatinine, cystatin C, and urea values in patients classified as AKI and non-AKI at the three measured time points.

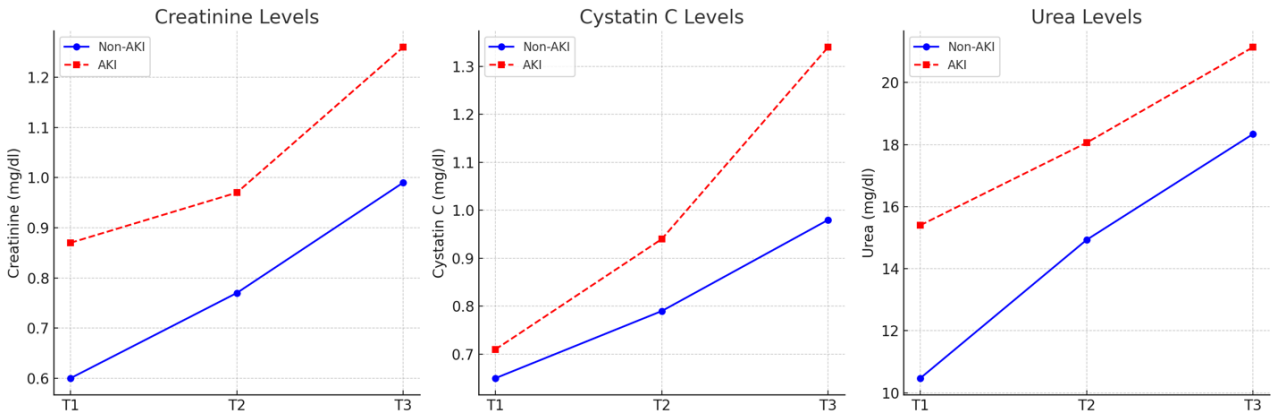
The mean creatinine values at T2 were 0.77 ± 0.13 mg/dl in non-AKI patients and 0.97 ± 0.07 mg/dl in the AKI group, both within the normal reference range for males. However, at T3, the values increased to 0.99 ± 0.13 mg/dl in the non-AKI group, while the AKI group exhibited values exceeding the reference range at 1.26 ± 0.21 mg/dl.

The reference range for serum cystatin C is 0.62 - 1.15 mg/dl. In T1, the mean value in the non-AKI group was 0.65 ± 0.25 mg/dl, and 0.71 ± 0.09 mg/dl in the AKI group. The mean value of serum cystatin C in T2 was 0.79 ± 0.11 mg/dl in the non-AKI group and 0.94 ± 0.01 mg/dl in the AKI group, both within normal reference values. However, in T3, unlike the mean values in the non-AKI group (0.98 ± 0.12 mg/dl), which are within the reference range, the values in the AKI group (1.34 ± 0.01 mg/dl) were higher than the reference range. When comparing T1 to T3, we observe an increase in serum cystatin C values of 0.33 mg/dl (33%) from the baseline value in the non-AKI group and 0.63 mg/dl (89%) in the AKI group. This indicates that only the AKI group meets the criteria according to AKIN and KDIGO for stage 1 of AKI/AKI with serum cystatin C values greater than 1.5-1.9 times the baseline reference values.

The reference range for urea in men is 7-20 mg/dl. In T1, the initial mean value in the non-AKI group was 10.47 ± 4.09 mg/dl and 15.4 ± 1.96 mg/dl in the AKI group. These values were within normal limits, but with significantly higher baseline serum urea values in the AKI group. The mean value of serum urea in T2 was 14.93 ± 1.57 mg/dl in the non-AKI group and 18.06 ± 4.01 mg/dl in the AKI group. The last measurement showed mean values of 18.34 ± 1.82 mg/dl in the non-AKI group and 21.14 ± 4.62 mg/dl in the AKI group. Only the urea concentration in T3 for AKI patients was above the upper reference limit.

DISCUSSION

After completion of intensive care, all patientsTo our knowledge, this study is the first in our country to investigate the biochemical parameter of kidney function, cystatin C, in patients after LRP, aiming to detect the early onset of AKI. The incidence of AKI in our patients was relatively low (10%) and consistent with previously published studies on similar interventions. Preoperative biochemical parameters of kidney function (creatinine and cystatin C) were within the normal range for all patients, yet those who developed AKI exhibited elevated values at the



Graph 1. *Changes in Cystatin C, Urea, and Creatinine Levels in AKI nd Non-AKI Patients Across Three Consecutive Measurements*

first measurement point. Notably, patients who developed AKI had a history of prior surgical interventions, and the duration of laparoscopic surgery in this subgroup was significantly prolonged ($p<0.05$). These findings suggest that prolonged surgical time and a history of surgical stress may contribute to renal dysfunction, warranting closer postoperative monitoring in high-risk patients. Those findings align with previous findings from the literature. Additionally, studies have shown that patients with a history of previous surgeries may have compromised renal function, making them more susceptible to AKI following subsequent procedures. Therefore, recognizing these risk factors is crucial for implementing preventive strategies and ensuring closer postoperative monitoring in high-risk patients [13-15].

There is growing evidence supporting the advantages of cystatin C above traditional renal biomarkers for early AKI detection. Unlike creatinine, which is influenced by factors such as muscle mass, age, and diet, cystatin C is produced at a constant rate by all nucleated cells and is freely filtered by the glomeruli without significant tubular secretion and reabsorption [16, 17]. As a result, it offers a more reliable assessment of glomerular filtration rate (GFR) and kidney function. A 2024 review study confirmed that cystatin C exhibits superior sensitivity and specificity in detecting both acute and chronic changes in renal function, thereby improving treatment accuracy and clinical decision-making [18]. Furthermore, a large multicenter cohort study in 2023 demonstrated that cystatin C is significantly more sensitive in detecting neonatal AKI compared to the modified KDIGO criteria, reinforcing its potential role as a preferred biomarker in various patient populations [19].

The limitations of traditional biomarkers, such as serum urea and creatinine, in detecting renal dysfunction have been well documented [20]. Serum creatinine, the most commonly used marker, does not rise until substantial renal impairment, delaying diagnosis and potential intervention. This delay can be critical in the perioperative setting, where timely recognition of AKI may improve outcomes by allowing for early therapeutic strategies. Murty and Sharma (2013) highlighted that creatinine levels do not increase until a moderate to severe decline in GFR has taken place, making it an inadequate early biomarker for AKI [21]. Given that creatinine levels are influenced by non-renal factors such as muscle mass and dietary protein intake, its diagnostic accuracy can vary significantly among individuals. In contrast, cystatin C is not affected by age, sex, or muscle mass, and it is not influenced by extrarenal factors such as inflammation or hemodynamic fluctuations, making it a superior endogenous marker of renal function.

Recent studies have further underscored the role of cystatin C in detecting early-stage AKI. In a study involving over 200 patients, 56.2% of those with AKI exhibited normal serum creatinine levels during the early stages of the disease (stage 1), whereas 100% of these patients had elevated serum cystatin C levels, confirming its superior sensitivity [22]. This aligns with our findings, where cystatin C proved to be a more effective biomarker for early AKI detection compared to serum creatinine. The rapid increase in cystatin C levels in our AKI patients at the first measurement point suggests its potential for routine use in perioperative kidney function monitoring.

The clinical importance of cystatin C has also been emphasized in various surgical and critical care settings. A systematic review by Shlipak et al. demonstrated that cystatin C is a superior biomarker for AKI prediction in critically ill patients, showing better prognostic value for short- and long-term renal outcomes compared to creatinine [23]. Moreover, a meta-analysis by Zhang et al. confirmed that cystatin C has a higher predictive accuracy for AKI in cardiac surgery patients, where kidney function is often compromised due to ischemia-reperfusion injury [24].

In the perioperative setting, several studies have reported a direct correlation between prolonged surgery duration and an increased risk of AKI, particularly in laparoscopic procedures. Park et al. demonstrated that prolonged pneumoperitoneum during laparoscopic surgery leads to intra-abdominal hypertension, reduced renal perfusion, and increased susceptibility to AKI [25]. Additionally, Cho et al. found that longer operative times in robotic-assisted prostatectomy were associated with a higher incidence of postoperative renal dysfunction. These findings are in line with our results, further reinforcing the importance of perioperative renal function monitoring, particularly in prolonged laparoscopic procedures [26].

Furthermore, the findings of our study emphasize the importance of individualized

perioperative risk assessment for AKI in patients undergoing LRP. Factors such as prolonged surgical time, history of previous surgeries, and potential hemodynamic instability should be considered when evaluating postoperative kidney function. The integration of cystatin C measurements into routine postoperative assessment protocols could lead to earlier identification of renal impairment, allowing for timely interventions such as fluid optimization, nephroprotective strategies, and avoidance of nephrotoxic agents [27].

In summary, our study supports the growing body of evidence that cystatin C is a more sensitive and specific biomarker than creatinine for early AKI detection. Its incorporation into routine clinical practice may improve early diagnosis, facilitate timely intervention, and ultimately enhance patient outcomes in surgical settings [28]. Future research with larger patient cohorts and longitudinal follow-up is warranted to further validate these findings and establish standardized cystatin C-based AKI detection protocols in perioperative care.

Despite the valuable insights gained from this study, several limitations should be acknowledged. First, the relatively small sample size may limit the generalizability of our findings, and larger multicenter studies are needed to confirm these results. Second, we relied on postoperative biochemical markers without incorporating advanced renal function tests or imaging techniques that could provide a more comprehensive evaluation of renal perfusion changes during surgery. Third, while cystatin C demonstrated higher sensitivity than creatinine in detecting early-stage AKI, our study did not evaluate long-term renal outcomes, making it unclear whether these early biochemical changes translate into clinically significant renal impairment over time.

Another limitation is that we did not account for potential confounding factors such as intraoperative hemodynamic fluctuations, fluid management strategies, and the use of nephrotoxic medications, all of which could have influenced AKI development. Additionally, while we observed a correlation between prolonged surgical duration and AKI risk, we did not investigate the specific intraoperative physiological changes that may have contributed to renal dysfunction, such as variations in intra-abdominal pressure during pneumoperitoneum. Future studies should aim to integrate hemodynamic monitoring data and perioperative fluid balance analysis to better understand these interactions.

Our study was limited by its observational design, which prevents us from establishing a direct causal relationship between elevated cystatin C levels and AKI progression. A prospective, randomized controlled trial comparing cystatin C-guided renal protection strategies versus standard care could provide more definitive evidence on its clinical utility in perioperative settings.

Finally, our study is small in number of patients, therefore the result in patients developing early stages of AKI is minor, only three patients (10%) meet the criteria for AKI stage one.

CONCLUSION

Good prediction of the onset of AKI is crucial for the timely diagnosis and treatment of this undesirable complication following laparoscopic interventions. Cystatin C has the potential to be a successful indicator for prompt and timely diagnosis of early stages of AKI.

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Резиме

РАНО ОТКРИВАЊЕ НА АКУТНА БУБРЕЖНА ПОВРЕДА КАЈ ЛАПАРОСКОПСКА ПРОСТАТЕКТОМИЈА: УЛОГАТА НА ЦИСТАТИН Ц, УРЕА И КРЕАТИНИН

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Вовед: Акутното бубрежно оштетување претставува потенцијално сериозна компликација по лапароскопската радикална простатектомија. Серумскиот креатинин, кој е стандарден биомаркер, покажува ограничена ефикасност за рана детекција на акутно бубрежно оштетување поради неговото задоцнето покачување во почетните фази. Потребни се иновативни биомаркери, кои ќе овозможат рана детекција и навремена интервенција, со цел подобрување на исходот кај пациентите.

Цели: Да се проценат и да се споредат вредностите на биохемиските маркери на бубрежната функција (серумска уреа, серумски креатинин и цистатин С) во предоперативниот и во постоперативниот период кај пациенти подложени на лапароскопска радикална простатектомија.

Материјали и методи: Оваа проспективна компаративна студија опфати 30 пациенти подложени на лапароскопска радикална простатектомија. Истражуваните серумски параметри – уреа, креатинин и цистатин С – беа евалуирани во три временски точки: предоперативно (Т1), веднаш по лапароскопската радикална простатектомија (Т2) и 12 часа по почетокот на хируршката интервенција (Т3).

Резултати: Сите пациенти покажаа нормални предоперативни биохемиски индикатори на бубрежната функција. Постоперативно, акутна бубрежна инсуфициенција беше идентификувана кај тројца пациенти. Од вкупно 30 пациенти што биле подложени на лапароскопска простатектомија, само тројца (10 %) ги исполнија критериумите за дијагноза на акутна бубрежна инсуфициенција (АКИ). Еден пациент (3 %) разви АКИ врз основа на зголемени нивоа на серумски креатинин, додека на тројца пациенти (6 %), вклучувајќи го и оној што ги исполни критериумите базирани на креатинин, им беше дијагностициран АКИ врз основа на покачени нивоа на серумски цистатин Ц. Серумските нивоа на цистатин Ц беа значително покачени кај пациентите со акутна бубрежна повреда на Т2 и останаа покачени на Т3 во споредба со Т1.

Заклучок: Резултатите покажуваат дека серумскиот цистатин С има поголема сензитивност за идентификација на акутно бубрежно оштетување во првите 12 часа по лапароскопската радикална простатектомија во споредба со серумската уреа и со креатининот.

Клучни зборови: акутно бубрежно оштетување, серумски креатинин, серумски цистатин С