Biochemistry & Physiology: Open Access

ISSN: 2168-9652

Toledo, Biochem Physiol 2023, 12:4

Mini Review Open Access

Urinary Biochemistry is Used in the Diagnosis of Acute Kidney Injury

Jennifer Toledo*

Department of Molecular and Clinical Medicine, School of Medicine and Psychology, Australia

Abstract

Acute kidney damage is a clinical illness that complicates and affects the course of a large number of hospitalized patients. Recent breakthroughs in clinical and basic research will aid in the characterization of this condition and the understanding of its pathophysiology. With this information, we will be able to perform more reliable epidemiologic studies in order to acquire a better understanding of the syndrome's impact. AKI is a condition with no single and distinct pathogenesis. Despite evidence that questions the use of biochemical indicators in clinical practise, they continue to be used. A better marker would include more particular information about the type, severity, and location of the injury. In this overview, we'll look at the factors that influence urea and salt fractional excretion. We believe that routinely examining the microscopy and biochemistry of urine can help determine whether or not AKI is reversible. Early injury biomarkers could help direct clinical therapy.

Keywords: Acute kidney injury; Ischemia; Creatinine

Introduction

In recent years, the idea of Acute Renal Failure has been extensively re-examined. Traditionally, the most severe acute loss in kidney function, as shown by severe azotemia and, in some cases, oliguria or anuria, was prioritised. Recent research suggests, however, that even very modest kidney injury or impairment, reflected by small changes in blood keratinize and/or urine output is a predictor of substantial clinical effects [1].

The term Acute Kidney Injury has recently superseded the term ARF. AKI is described as a sudden decline in kidney function that includes both damage and impairment. It is a syndrome with no single and identifiable pathophysiology. Many patients with AKI have a mixed aetiology, which complicates diagnosis and therapy because sepsis, ischemia, and nephrotoxicity frequently co-exist. Furthermore, the syndrome is quite common in individuals who do not have a critical illness, thus it is crucial that health care workers, particularly those who do not specialize in renal disorders, can quickly diagnose it. Clinicians often monitor daily serum creatinine concentrations to establish the glomerular filtration rate since serum creatinine is easily filtered in the glomerulus and only a little quantity is normally released along the tubule. Age, gender, muscle mass, muscle metabolism, medicines, and state of hydration all have an effect on blood creatinine levels that are unrelated to GFR. Furthermore, because the balance between production and elimination takes days, acute changes in GFR are seldom accompanied by concomitant increases in serum creatinine. As a result, serum creatinine considerably underestimates the extent of renal function loss, particularly in the first 48 hours after injury. Another common limitation of creatinine is the cumulative fluid balance, which is common in patients with severe sickness. Because of an increase in the volume of distribution of serum creatinine, the creatinine concentration may be overestimated, further delaying the diagnosis. Furthermore, sepsis, the most common cause of AKI in hospitalised patients, reduces serum creatinine production. Sepsis has been shown to decrease serum creatinine production even in the absence of muscle mass loss, limiting its utility as a marker of AKI [2-5].

Urine production has its own set of constraints. To begin with, not all patients' urine volumes can be quantified. Diapers are difficult to correctly weigh, making determining urine production in children and babies problematic. Because of the risk of nosocomial urinary tract infection, urinary catheters are only used in the most seriously ill patients. Most importantly, 25% to 80% of all AKI cases and 33% of patients at the time of diagnosis are nonoliguric. Nonoliguric AKI can occur in any form of AKI, including those caused by surgery, trauma, hypotension, nephrotoxins, and rhabdomyolysis. Some of the potential causes of nonoliguric AKI include volume expansion, extremely powerful diuretics, and renal vasodilators. Aggressive fluid resuscitation and improved supportive care for critically ill patients are other significant. Even while the residual level of GFR is the key determinant influencing urine volume in patients with AKI, there is a difference between spontaneous and induced urine flow, and urine flow does not correspond with the severity of renal impairment [6-7].

In some cases, a decrease in urine volume may not reflect impairment in renal function but rather an expected response to decreased renal perfusion. Even if blood pressure is normal, renal hypoperfusion can occur due to dehydration in the presence of vomiting or diarrhoea. In persons with hepatorenal syndrome type 1, increased levels of nitric oxide and endothelium-derived relaxing factor result in a decrease in splanchnic and total vascular resistance, which is assumed to be reversible. Any further harm caused by gastrointestinal losses, bleeding, or therapy with a diuretic or NSAID causes a further drop in GFR. In this case, it is expected that an increase in renal perfusion will be able to quickly reverse the prerenal disease or condition. Despite this, not all hypo perfusion situations will respond to fluid expansion. Because decreased cardiac output lowers glomerular perfusion pressure and raises venous pressure, acute heart failure inhibits glomerular filtration. Medicines that further lower effective volume, such as diuretics, or medicines that interfere with glomerular perfusion pressure, such as NSAIDs or angiotensin blockers, can have an impact on the renal auto regulatory response, resulting in prerenal conditions [8-10].

***Corresponding author:** Jennifer Toledo, Department of Molecular and Clinical Medicine, School of Medicine and Psychology Australia, E-mail: jennifertoledo@gamil.com

Received: 01-May-2023, Manuscript No: bcp-23-99426, **Editor Assigned:** 04-May-2023, pre QC No: bcp-23-99426 (PQ), **Reviewed:** 18-May-2023, QC No: bcp-bcp-23-99426, **Revised:** 22-May-2023, Manuscript No: bcp-23-99426 (R), **Published:** 29-May-2023, DOI: 10.4182/2168-9652.1000418

Citation: Toledo J (2023) Urinary Biochemistry is Used in the Diagnosis of Acute Kidney Injury. Biochem Physiol 12: 418.

Renal ultrasound: Renal ultrasonography is effective for evaluating existing structural renal illness and identifying urine collecting system blockage. Reduced corticomedullary differentiation and decreased kidney size, in particular, are indicators of underlying CKD. In patients with abdominal distension, ultrasonography might be technically demanding, necessitating the use of alternative imaging investigations [11].

Renal Doppler ultrasonography and contrast-enhanced ultrasound are two relatively new techniques for estimating renal perfusion and renal cortical microcirculation at the bedside, respectively. Although the non-invasiveness, reproducibility, and accessibility of these approaches sound promising, their widespread clinical application is still hampered by training requirements as well as confusion about how to interpret the data obtained. Finally, while Doppler scans can detect the existence of decreased renal blood flow, they are ineffective in determining the cause of AKI [12].

Renal biopsy: Renal biopsies are infrequently conducted in critically ill patients, owing to the perceived risk of bleeding problems and the lack of therapeutic implications. A renal biopsy, on the other hand, may provide information not available through other procedures and should be considered if underlying parenchymal or glomerular renal illness is suspected. Interestingly, it was shown that diffuse histological alterations of AKI could exist in the absence of a significant decrease in serum creatinine. Only 198 patients satisfied the KDIGO creatinine or urine criteria for AKI among 303 patients with biopsy-proven acute parenchymal renal abnormalities, including crescentic glomerulonephritis and acute thrombotic microangiopathy. In a second French study, over 50% of AKI patients who underwent renal biopsy had a diagnosis other than acute tubular necrosis, which frequently led in a change in treatment plan. According to recent research, Tran's jugular renal biopsies may be safer than percutaneous or open procedures [13, 14].

Discussion

The findings of this study are expected to provide recommendations for the best course of action to address the weaknesses of present biochemistry and genetics teaching techniques, as well as to better inform medical educators and health policymakers about these deficiencies. It was notable that more than half of the practitioners were aware of current advancements in biochemistry and genetics and how these have impacted modern medicine. Pharmacogenetics, whole genome association studies, and personalised medicine were all topics that doctors were familiar with. Our findings also show that most medical professionals recognise the importance of biochemistry and genetics in modern clinical practise, such as disease diagnosis and focused treatment. Despite the generally favourable outlook, medical personnel were hesitant to hire a clinical biochemist to assist with diagnosis and/or monitor the development of the disease. The low percentage of Jordanians with professional board certification in clinical biochemistry, as well as the lack of national clinical biochemistry board certification programmes and fellowships, could explain this. As a result, clinical biochemists are not commonly found on the medical teams of Jordanian doctors [15, 16].

In any case, given how quickly these fields are developing and how they are changing medical practise and research, if this finding is confirmed by larger studies, health policymakers will have a compelling reason to target public sector physicians with educational programmes to change their perspective on biochemistry and genetics [17].

This study has various shortcomings, including: Despite the fact that the survey's total response rate was higher than that of most comparable research, the bulk of nonresponders were doctors working in hospitals in southern Jordan. The opinions of the aforementioned doctors were not ultimately considered. As a result, the study does not accurately reflect the perspectives of all Jordanian physicians [18].

To overcome this constraint, a more extensive national-scale study is required. Furthermore, rather than providing an objective rating, the questionnaire asked clinicians to grade their own grasp of genetics and biochemistry. For example, the phrasing of several of the sentences supports affirmative responses. Despite these limitations, this is Jordan's first survey of its kind, and given its design and sample size, medical educators should find it useful [19].

This finding is consistent with a number of previous studies conducted in other countries, and it may be explained by the fact that many Jordanian medical schools did not change the way basic medical science courses were taught at the same time as the syllabus's content changed. In fact, these courses continue to rely heavily on didactic lectures and do not encourage active student participation in the classroom [20].

Conclusion

We believe that by monitoring UM and urine biochemistry indicators, we can predict the likelihood of early AKI recovery. The combined examination of the urine biochemistries of FeNa, FeU, and UM can help with the differential diagnosis of AKI. To aid in the detection of reversibility, urinary biochemistry/microscopy and early biomarkers of injury should be associated in large trials with an early diagnosis of AKI.

Conflicts of Interest

None

Acknowledgment

None

References

1. Semba RD, Nicklett EJ, Ferrucci L (2010) Doe's accumulation of advanced glycation end products contribute to the aging phenotype. J Gerontol: Biol 65: 963-975.
2. Uribarri J, Cai W, Peppa M (2007) Circulating glycotoxins and dietary advanced glycation endproductstwo links to inflammatory response, oxidative stress, and aging. J Gerontol: Biol 62: 427- 433.
3. Shafee M, Abbas F, Ashraf M (2014) Hematological profile and risk factors associated with pulmonary tuberculosis patients in Quetta, Pakistan. Pak J Med Sci 30: 36-40.
4. Jiménez-Corona ME, Cruz-Hervert LP, García-García L (2013) Association of diabetes and tuberculosis: impact on treatment and post-treatment outcomes. Thorax 68: 214-220.
5. Mimoun S, Andriamihaja M, Chaumontet C (2012) Detoxification of H2S by differentiated colonic epithelial cells: implication of the sulfide oxidizing unit and of the cell respiratory capacity. Antioxidants & Redox Signaling17: 1-10.
6. Matallo J, Vogt J, McCook O (2014) Sulfide-inhibition of mitochondrial respiration at very low oxygen concentrations. NOx 41: 79-84.
7. Helmy N, Prip-Buus C, Vons C (2014) Oxidation of hydrogen sulfide by human liver mitochondria. NOx 41: 105-112.
8. Wang SN, Liu Z, Tang HZ, Meng J,Xu P,et al. (2007) Characterization of environmentally friendly nicotine degradation by Pseudomonas putida biotype a strain S16. J Microbiol 153: 1556-1565.
9. Yuan YJ, Lu ZX, Huang LJ, Bie XM, Lü FX, et al. (2006) Optimization of a medium for enhancing nicotine biodegradation by Ochrobactrum intermedium. J Microbiol, 101: 691-697.

10. Ruan A, Min H, Peng X, Z. Huang Z (2005) Isolation and characterization of Pseudomonas strain HF-1, capable of degrading nicotine. Microbiol. Res 156: 700-706.

11. Wada E and Yamasaki K (1953) Mechanism of microbial degradation of nicotine. Sci 117: 152-153.

12. Zhao L, Zhu C, Gao Y (2012) Nicotine degradation enhancement by Pseudomonas stutzeri ZCJ during aging process of tobacco leaves. World J Microbiol. Biotechnol 28: 2077-2086.

13. Schmit CG, Jahan K, Schmit KH, Debik E, Mahendraker V, et al. (2009) Activated sludge and other aerobic suspended culture processes. WER 81: 1127-1193.

14. Nauck MA, Homberger E, and Siegel EG (1986) Incretin effects of increasing glucose loads in man calculated from venous insulin and C-peptide responses. J Clin Endocr 63: 492-498.

15. Elrick H, Stimmler L, Hlad CJ, and ARAI Y (1964) Plasma insulin response to oral and intravenous glucose administration. J Clin Endocr 24: 1076-1082.

16. Kipnis (1967) Plasma insulin responses to oral and intravenous glucose studies in normal and diabetic subjects. JCI 46: 1954-1962.

17. Elliott RM, Morgan LM, Tredger JA, Deacon S, Wright J, et al. (1993) Glucagon-like peptide-1 amide and glucose-dependent insulinotropic polypeptide secretion in response to nutrient ingestion in man: acute post-prandial and 24−h secretion patterns. J Endocrinol 138: 159-166.

18. Falko JM, Crockett SE, Cataland S, and Mazzaferri EL (1975) Gastric inhibitory polypeptide stimulated by fat ingestion in man. J Clin Endocr 41: 260-265.

19. Kauth T, Metz J (1987) Immunohistochemical localization of glucagon-like peptide 1. Use of poly- and monoclonal antibodies. Histochem 86: 509-515.

20. Dollet L, Magré J, Joubert M (2016) Seipin deficiency alters brown adipose tissue thermogenesis and insulin sensitivity in a non-cell autonomous mode. Sci Rep 6.