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ACUTE RENAL FAILURE WITH NORMAL PLASMA UREA LEVEL SECONDARY TO ACUTE PYELONEPHITIS IN A SINGLE KIDNEY PATIENT

Musso CG^{1, 2}, Vilas M^{1, 2}, Fernandez Otero L², Jauregui R², Imperiali N¹, Algranati L¹

¹ Nephrology Department¹ and Centro Medico Agustin Rocca². Hospital Italiano. Buenos Aires. Argentina

carlos.musso@hospitalitaliano.org.ar

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[Comment of the Reviewer Jesús Garrido MD.](#) Serviço de Nefrologia e Diálise, Hospital São Teotónio. Viseu. Portugal.

[Comment of the Reviewer Jeyaraj Balasubramaniam, MD. PhD.](#) Resident Director. Kidney Care Centre. Tirunelveli. Tamilnadu, India.

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SUMMARY

Acute renal failure is a syndrome that usually runs with an increase in creatinine and urea plasma levels. However, there are clinical situations in which this syndrome may run with an increase in plasma creatinine keeping normal the urea one.

In this report we present a case of acute renal failure with normal plasma urea level secondary to an acute pyelonephritis in a single kidney patient. The patient had an increased fractional excretion of urea which could explain the normal plasma urea levels found despite of his reduced glomerular filtration. This increased urea excretion state was interpreted as a consequence of the nephrogenic diabetes insipidus and alteration of the intra-renal urea recycling process that the acute pyelonephritis induced. In conclusion: Acute pyelonephritis in a single kidney patient can appear as a pattern of acute renal failure with normal plasma urea levels.

Key words: acute renal failure, normal uremia, acute pyelonephritis

RESUMEN:

La insuficiencia renal aguda es un síndrome que característicamente cursa con niveles plasmáticos elevados de urea y creatinina. Sin embargo, hay situaciones clínicas en las cuales este síndrome puede cursar con un incremento de la creatininemia sin presentar elevación de la uremia.

En este reporte presentamos un caso clínico de una insuficiencia renal aguda con uremia normal secundaria a una pielonefritis aguda en un paciente con riñón único. El paciente presentaba una elevada excreción fraccional de urea lo cual podía explicar su uremia normal pese a estar cursando una caída del filtrado gomerular. Dicha excreción de urea elevada fue interpretada como secundaria a una diabetes insípida nefrogénica y una alteración en el recirculado intra-renal de la urea ambos producto de la pielonefritis aguda. Concluimos que la pielonefritis aguda en un paciente mono-reno puede presentarse con un patrón de insuficiencia renal aguda con uremia normal.

Palabras Clave: insuficiencia renal aguda, uremia normal, pielonefritis aguda

INTRODUCTION

Acute renal failure is a syndrome that usually runs with an increase in creatinine and urea plasma levels since in both substances the glomerular filtration plays an important role in their excretion ¹.

However, there are clinical situations in which an acute renal failure may run with an increase in plasma creatinine keeping normal the urea level. Examples of the afore mentioned clinical situations are those patients who suffer from acute renal failure in the context of low protein intake, hepatic insufficiency, or/and diabetes insipidus ²⁻⁴.

In the following report we present a case of acute renal failure with normal plasma urea level secondary to an acute pyelonephritis in a single kidney patient.

CASE REPORT

Male patient, sixty-two years old who suffered from the following diseases:

- Diabetes mellitus (type II) treated with diet and 4 mg/day of glymepiride
- Right nephrectomy performed ten years before due to uropyonephrosis.
- Urolithiasis (in the past)
- Hypercholesterolemia treated with sinvastatin (10 mg/day) and ezetimibe (10 mg/day)

He was admitted in our hospital presenting two day of evolution of a left lumbar pain, dysuria, and fever. Blood and urine laboratories were performed. Abundant leucocytes and pyocytes were documented in the urinalysis, while reduced creatinine clearance (35 ml/minute), normal plasma urea (29 mg/dl)and increased plasma creatinine (2,1 mg/dl) and fractional excretion of urea (80%) were detected.

The patient presented water polyuria (urine volume: 3000 cc with urine osmolality: 176 mOsm/l), slightly increased plasma glucose (130 mg/dl) and normal plasma sodium (135 mmol/l) levels. (Table 1)

The patient was not under any nephrotoxic drug and he had no clinical or laboratory evidence of rhabdomyolysis. He did not suffer from any disease that could reduce his plasma urea level such as malnutrition, hepatic disease or Fanconi syndrome.

The case was interpreted as an acute renal failure secondary to acute pyelonephritis in a single kidney patient. After blood and urine cultures were obtained intravenous ceftriaxone (2 gr/day) was initiated. Urine culture was positive to *Escherichia coli* sensitive to the prescribed antibiotic.

After the infection was cured the fractional excretion of urea (48%), plasma creatinine (1.3 mg/dl), creatinine clearance 110 ml/minute and plasma urea (39) levels reached their usual values.

TABLE 1: Laboratory values before and after the acute pyelonephritis treatment

	Al Ingreso	Post- tratamiento antibiótico	Rango Normal
Hto	45 %		40-53 %
Hb	15 g/dl		13-17 g/dl
WC	17,0000 / mm ³	8000 / mm ³	5,000 – 10,000 /mm ³
PU	29 mg/dl	39 mg/dl	20 – 50 mg/dl
PCr	2.1 mg/dl	1.3 mg/dl	0.5 – 1.3 mg/dl
Pna	135 mmol/l		135 – 145 mmol/l
PK	4.6 mmol/l		3.5 – 5.5 mmol/l
PG	140 mg/dl		70 – 110 mg/dl
GOT	16 UI/l		10 – 42 UI/L
GPT	14 UI/l		10 – 40 UI/L
PCa	9 mg/dl		8.5 – 10.5 mg/dl
UO	176 mOsm/l		100 – 1400 mOsm/l
FEU	80%	48.00%	

Hto: hematocrit, Hb: hemoglobin, WC: white cells, PU: plasma urea, PCr: plasma creatinine, PNa: plasma sodium, PK: plasma potassium, PG: plasma glucosa, GOT: glutamic oxalacetic transaminases, GPT: glutamic piruvic transaminases, PCa: plasma calcium, UO: urine osmolality, FEU: fraccional excretion of urea.

DISCUSSION

Urea is the major end product of protein catabolism in mammals. It is synthesized in the liver and excreted mainly by the kidney. Under basal conditions, this substance has a glomerular filtration of 100% although its final excretion is around 50%. This lower excreted amount respect to the filtrated one is a consequence of its reabsorption in the proximal tubules and in the very late part of the collecting ducts, close to the papillary tip. Moreover, since urea is also secreted in the S3 segment of proximal tubules, this substance suffers an intra-renal recycling process which contributes to reduce its excretion^{5, 6}.

Acute renal failure syndrome usually runs with an increase in creatinine and urea plasma levels since in both substances glomerular filtration plays an important role in their excretion¹.

However, there are situations of acute renal failure with increased plasma creatinine levels but normal urea ones. This phenomenon can be justified mainly by two physio-pathologic mechanisms:

- low body urea production, as is the case of malnutrition, hepatic insufficiency, etc
- Increased excretion of urea, as is the case of proximal tubular dysfunction (Fanconi syndrome), osmotic diuresis (glucosuria, etc), water diuresis (diabetes insipidus), urea tubular secretion (syndrome of inappropriate antidiuretic hormone secretion), etc⁶

Regarding acute pyelonephritis, this infection can induce nephrogenic diabetes insipidus since it generates an inflammation of the renal interstitium damaging the medullary tonicity⁷. Besides, acute pyelonephritis can alter the intra-renal urea recycling process⁸, and also induce an acute renal failure in a single kidney or chronic renal disease patient².

In our reported case it can delineate three syndromes:

- A non-oliguric acute renal failure secondary to a pyelonephritic process. This situation could be justified since this infection took place in an aged single kidney patient.
- Nephrogenic diabetes insipidus (water diuresis) secondary to the renal interstitium alteration induced by the acute pyelonephritis.
- An elevated urea excretion secondary to a reduced water reabsorption capability and intra-

renal urea recycling process. Both disorders could be induced by the acute pyelonephritis. This increase in the fractional excretion of urea can justify the patient's normal plasma urea levels despite of his reduced glomerular filtration rate.

CONCLUSION:

Acute pyelonephritis in a single kidney patient can appear as a pattern of acute renal failure with normal plasma urea levels.

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Comment of the reviewer Jesús Garrido MD. Serviço de Nefrologia e Diálise, Hospital São Teotónio. Viseu. Portugal.

Musso et al. present a clinical case, in which the interest relies not only in the fact of being a normo-uremic renal failure but also in the mechanisms that are used to explain it. Since 1956, when Ullrich and Jarausch¹ published the role of urea as principal factor of medullary tonicity, there have been many works that have help to understand the concentration and dilution mechanisms. However, it is interesting to see how the aggressions against this media can produce, not only tubular-interstitial damage with renal failure, but also medullary disequilibrium (urea concentration, recycling tubular urea pathways, vasopressin sensibility.) that produce a normal uremia because of an increasing of compensatory excretion. It is known that urea, usually used as a renal function marker, it is not a reliable indicator². Its variability in production, reabsortion and excretion depending on different factors (dietay, hepathopathy, cardiopathy, drugs.) affects plasmatic levels through a different way then the glomerularly filtration rate. This work indicates perfectly one of these situations, showing one more example of this variability.

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Comment of the reviewer Jeyaraj Balasubramaniam, MD. PhD. Resident Director. Kidney Care Centre. Tirunelveli. Tamilnadu, India.

Dr.Musso, by intuitive observation of single case study , has brought to our notice more than one phenomenon.

1. Interstitial nephritis due to sepsis can cause proximal tubulopathy and so can present with features of tubular dysfunction.
2. Normal blood urea in the face of elevated creatinine is a marker of tubulopathy.
3. Fractional Excretion of Urea (FEUrea) is a useful index in diagnosing and assessing Acute Renal Failure.

Though these are not new, the way this simple presentation drives home these phenomena to a reader is greatly commendable. It is notable that Dr.Musso has already presented an illustrative case to show that tubulotoxic drugs can cause similar phenomena(Electron J Biomed 2004;2:1-78)

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