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INTERMEDIATE SYNDROME: A TYPICAL PATTERN OF PRE-RENAL ACUTE RENAL FAILURE IN THE ELDERLY

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SUMMARY:

Acute renal failure is a frequent entity in the elderly. This is due on one hand to the structural and physiological changes of the aged kidney, and on the other hand to the exposure of this population to polypharmacy and their reduced capability to metabolize drugs.

In the present report we present a case of a seventy year-old woman who developed acute renal failure secondary to severe dehydration with a clinical and laboratory pattern of intermediate syndrome: laboratory results compatible with parenchymal renal insufficiency (elevated urinary sodium, plasma urea and creatinine), but with a positive response to hydration. The main characteristics of the aged kidney that predispose to the development of an intermediate syndrome are: the vascular dysautonomy and reduced capability of sodium and water reabsorption. The intermediate syndrome is a typical pattern of pre-renal insufficiency in the elderly.

RESUMEN:

La insuficiencia renal aguda es frecuente en el anciano. Esto se debe por un lado a los cambios estructurales y funcionales propios del riñón senil, y por otro a la gran exposición que esta población tiene a la polifarmacia, y su reducida capacidad para metabolizar los medicamentos.

En este reporte presentamos el caso de una mujer de 70 años que desarrolló una insuficiencia renal aguda secundaria a severa deshidratación, mostrando un patrón clínico y de laboratorio propio de un síndrome intermedio: laboratorios compatibles con una insuficiencia renal parenquimatosa (sodio urinario, uremia y creatinemia elevadas), pero con una respuesta favorable a la hidratación. Las principales características del riñón senil que predisponen al desarrollo del síndrome intermedio: son la disautonomía vascular y la reducida capacidad en la recuperación de sodio y agua. El síndrome intermedio es un patrón típico de insuficiencia prerrenal en el anciano.

INTRODUCCIÓN

Acute renal failure is a frequent renal syndrome in the elderly. This phenomenon is due to the morphological and functional renal changes that occur with aging, the exposure of the aged population to polypharmacy and their reduced capability to metabolize drugs ¹. In the following case report a typical pattern of pre-renal acute renal failure in the aging population is presented

CASE REPORT:

A 70 year-old woman was admitted to our hospital presenting a confusional syndrome in the context of severe hypernatremia and acute renal failure. On examination, oral and axillary dryness, orthostatism, oliguria, hypotension (blood pressure: 90-50 mmHg) and slight hypothermia (rectal temperature: 36.8°C) were documented.

She was a self-sufficient person, she took no medications and she suffered no diseases.

A week before admission she had started with watery diarrhea that progressively led her to severe dehydration. At the time of her hospital admission she had no more diarrhea but she was still very dehydrated.

Plasma urea (195 mg/dl), plasma creatinine (7.3 mg/dl), and urinary sodium levels (UNa: 90 mmol/l) were high. Hyperchloremic metabolic acidosis (pH: 7.32 / bicarbonate: 11 mmol/l) was also documented. Renal ultrasonography was normal. Blood cultures were taken and water was given by nasojunal tube, at a rate of three litres per day.

Once rehydration was initiated, neurological status, diuresis, acid-base, plasma sodium, urea and creatinine values improved progressively. Blood cultures were negative.

After eight days of treatment, acute renal failure was resolved normalizing her confusional syndrome and laboratory alterations (Table I).

TABLE I: Main Analysis During Hospital Admission

	Admission	2 nd day	3 rd day	4 th day	5 th day	6 th day	8 th day	normal Values
Plasma urea (mg/dl)	195	179	199	143	90	41	35	20-40
Plasma creatinine (mg/dl)	7.3	6	4.8	3	1.9	1.3	1	0.7-1
Plasma sodium (mmol/l)	177	159	151	146	146	143	139	135-145
Plasma potassium (mmol/l)	3.4	3.4	3.5	2.9	5	4.6	3.8	3.5-5.5
Plasma chloride (mmol/l)	147	127	118	116	113	110	-	90-100
Plasma pH	7.32	7.37	7.33	7.34	-	7.38	7.40	7.40 ± 4
Plasma bicarbonate (mmol/l)	11	19	17	19	-	22	24	24 ± 2

DISCUSIÓN

Acute renal failure is usually classified in three categories depending on its pathophysiological mechanism²:

- 1) Pre-renal insufficiency, in which there is renal hypoperfusion secondary to real or effective hypovolemia: volume depletion, decreased cardiac output, peripheral vasodilatation or redistribution of extracellular fluid.
- 2) Intrinsic renal insufficiency, in which there is parenchymal damage secondary to prolonged hypoperfusion (ischemia) or renal toxicity^{2,3}.
- 3) Post-renal insufficiency, in which there is urinary tract obstruction as the pathophysiological mechanism².

In order to distinguish pre-renal insufficiency from intrinsic renal insufficiency, three parameters are usually considered: clinical examination, laboratory analyses and response to volume expansion². For instance, if a patient has low urinary sodium, higher plasma urea than plasma creatinine, signs of dehydration and a positive response to volume expansion, he will be diagnosed as having pre-renal insufficiency; while if a patient has urinary casts, high urinary sodium, high plasma urea and creatinine levels, edema, and a negative response to volume expansion, he will be considered to have intrinsic renal insufficiency^{4,5}.

However, a third situation has been described, named intermediate syndrome⁶. It consists of a patient whose blood and urinary laboratory results seem to be those of intrinsic renal insufficiency (i.e. high plasma urea, creatinine and urinary sodium), but his renal insufficiency gets better with volume expansion, behaving as a pre-renal one. However, the intermediate syndrome ameliorates more slowly than pre-renal insufficiency: its renal function recovery takes more than twenty-four hours, but less than a week. Histologically, intermediate syndrome represents a partial acute tubular necrosis, a situation between the absence of tubular necrosis (pre-renal insufficiency) and its total installation (parenchymal renal insufficiency)⁶.

Many characteristics of the aging kidney predispose aged people to suffer intermediate syndrome⁷:

- Anatomically, the aged kidney has a reduced number of glomeruli and glomerular capillaries due to the senile glomerulosclerosis process. These histological changes make the aged kidney prone to progress easily to parenchymal damage under stressful situations⁸
- A reduced renal defense against hypoperfusion due to dysautonomy, meaning that vascular autoregulation is altered making renal blood flow and the glomerular filtration rate not constant during large changes in renal perfusion pressure².
- A reduced defense against volume contraction because of the presence of primary hypodipsia, and reduced sodium and water reabsorption capability⁹. The reduced sodium reabsorption capability is secondary to an alteration in the reabsorption of this cation in the ascending loop of Henle¹⁰, and distal tubules⁸. Besides, increased plasma atrial factors contribute to urinary sodium excretion¹⁰.

The senile reduction in water reabsorption capability is due to the medullary hypotonicity and the altered response to antidiuretic hormone that it produces¹¹.

- Tubular frailty, attributed to the reduced number of mitochondria in their tubular cells. This could signify a reduction in the energetic reserve that tubular cells have in order to resist ischemia⁹.

All the above mentioned factors make the aging kidney capable of developing partial tubular necrosis, and as a consequence a pattern of intermediate syndrome.

The documented hyperchloremic metabolic acidosis was probably due to an alteration in the ammoniogenesis process in the context of tubular damage.

CONCLUSION:

Intermediate syndrome is a typical pattern of hypovolemic pre-renal insufficiency in the elderly.

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Acute renal failure is a relevant clinical problem in the elderly, since they have worse control of the internal milieu than young people. They have also a reduced functional renal reserve (secondary to a progressive nephronal loss due to the aging process and concomitant diseases such as: diabetes mellitus, hypertension, nephrotoxic drugs, etc.)

Among the main causes of acute renal failure in old ambulatory population, there are absolute hypovolemia (dehydration secondary to fever, diarrhea, etc.), non-inflammatory relative hypovolemia (cardiac failure, hypoxia, etc.) and inflammatory relative hypovolemia (sepsis, etc.).

Others causes of acute renal failure in this group are obstructive uropathy (prostatic disease) and renal parenchymal damage (nephrotoxic drugs, vasculitis, etc.).

In this report a case of an intermediate syndrome is presented. This refers to a renal alteration between a pure pre-renal insufficiency and a complete acute tubular necrosis, and is a frequent pattern especially in patients admitted to hospital, since their renal failure is caused by many variables: sepsis, nephrotoxic drugs (aminoglycoside), etc.

There are helpful markers for detecting renal damage as is urine N-acetylglucosaminidase (proximal tubules), gammaglutamyltranspeptidase (distal tubules), and doppler ultrasound resistance index (nephrotoxic damage secondary to oncologic drugs).

It is very important to point out that rehydration is always the first treatment for any acute renal failure, but one should be cautious with patients suffering from pulmonary distress or restrictive cardiopathy.

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Musso et al. describe correctly the pathophysiological changes suffered by the aged kidney that expose the elderly to develop acute renal failure. It is also important to point out the influence of senile anatomical changes (reduction in kidney size and number of glomeruli, increase in the mesangium area due to a diminution in capillary volume) and reduced metabolism of drugs, on the elderly exposed to acute renal damage.

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