Aluminum Urinary Excretion in Patients with Chronic Renal Failure in Treatment with Conservative Conventional Therapy and with Ketoanalogues: Research on Fasting Patients and after a Pharmacological Load

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Dear Sir,

We have already demonstrated [1] that initial CRF causes an increased urine excretion of Al even after an interruption of months of aluminum (Al) pharmacological intake.

We studied 79 patients (36 M, 43 F), mean ($\pm$ SD) age 37.66 ± 15.15 . They were distributed as follows:

Group A: controls (15 M, 15 F);

Group B: initial CRF, CrCl between 70 and 30 ml/min (10 M, 18 F);

Group C: advanced CRF, CrCl between 30 and 5 ml/min (6 M, 7 F);

Group D: most advanced CRF, CrCl between 12 and 5 ml/min in treatment on an artificial diet of essential amino acids and ketoanalogues (5 M, 3 F).

All the patients interrupted their treatment to take the Al hydroxide for at least 3 months.

The results (mean $\pm$ SD) are as follows:

Al-s, μ g/l:

Group A: 5.65 ± 2.51 ;

Group B: 6.01 ± 3.80 ;

Group C: 5.78 ± 4.04 ;

Group D: 5.67 ± 1.97 .

Al-s group A does not demonstrate statistical differences with Al-s of group B, C and D.

Al-u, μ g/die:

Group A: 13.09 ± 10.07 ;

Group B: 22.12 ± 17.25 ;

Group C: 22.87 ± 24.46 ;

Group D: 4.81 ± 1.49 .

Al-u group A $p < 0.01$ vs. group B, C and D.

Urinary ratio Al/Cr * 10^{-3} mg was:

Group A: 0.0105 ± 0.0096 ;

Group B: 0.0301 ± 0.0367 ;

Group C: 0.0351 ± 0.0427 ;

Group D: 0.0060 ± 0.0023 .

Al/Cr group A $p < 0.001$ vs. B and C; NS vs. D.

Figure 1 shows a straight line of linear regression of Al/Cr vs. Al-u:

$$y = 8.25x + 7.0$$

$$r = 0.41$$

$$p < 0.001.$$

The data of our research show that the initial renal insufficiency is a cause for Al accumulation if there is an overexposure. The increased urinary excretion of Al persists do be high even months after interrupting Al pharmacological administration (indirect sign of stores that still release Al? or increased gastroduodenal absorption for alteration of the gastrointestinal barrier? or substances occurring in the diet, promoting Al gastroduodenal absorption? or PTH? or acidemia?) [2-4].

In addition, the urinary Al/Cr ratio is shown to be a valid indicator of increased Al urinary excretion for values > 0.02 (n.v. appears to be < 0.01). Even in initial renal failure, the Al/Cr ratio increases to 341% as compared to controls, and in advanced renal failure it increases to 398%.

Finally, conservative therapy with ketoanalog normalizes both Al urinary excretion (even the Al-u values are reduced compared to controls) and the Al/Cr urinary ratio in uremic patients.

Thirty-six patients ($n = 10$ in group A, B and C; $n = 6$ in group D) were given an oral load of Al hydroxide (4 g/die for 7 days).

The results are as follows:

Al clearance, ml/min:

Group A: 4.08 ± 1.84 (+148%);

Group B: 2.82 ± 0.77 (-61%);

Group C: 1.56 ± 0.85 (-54%);

Group D: 0.975 ± 0.07 (+5%).

Al Fe, %:

Group A: 2.88 ± 1.22 (+ 168%);

Group B: 9.26 ± 4.3 (-77%);

Group C: 11.45 ± 8.66 (-49%);

Group D: 30.3 ± 3.25 (-71 %).

The data show that the functional reserve of residual nephrons in eliminating Al after a pharmacological load is already reduced in initial CRF. This same functional reserve is not modified by an artificial diet.

Regression of Al/Cr on Al_{ur}

0.3

Fig. 1. Al/Cr vs. A-u simple regression straight line, $y = 8.25x + 7.0$, $r = 0.41$, $p < 0.001$.

0.2

0.1 -

0 -

S

40

100

120

20

80

60 Alur

References

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