Sodio intercambiable y sistema renina en la insuficiencia renal crónica y en la hipertensión arterial

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SODIO INTERCAMBIABLE Y SISTEMA RENINA EN LA INSUFICIENCIA RENAL CRÓNICA Y EN LA HIPERTENSIÓN ARTERIAL

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VI. CONCLUSIONES

1. – El sodio intercambiable (Na-I) expresado por kg de peso no constituye un valor indicativo sino se tiene en cuenta la composición corporal del individuo, es decir, sino se tiene en cuenta el "índice magro" (T^3/P).
2. – Los valores normales de Na-I y espacio de sodio vienen expresados para un determinado "índice magro" por las ecuaciones siguientes:

$$\underset{(mEq/kg)}{Na\text{-}I} = [282\ (T^3/P) + 18] \pm 4{,}8$$

$$\underset{(ml/kg)}{ES} = [2242\ (T^3/P) + 115] \pm 38{,}1$$

quedando comprendidos alrededor del 95 % de los valores normales.
3. – Para la comparación de distintos valores de Na-I es preciso expresar en tantos por ciento de la normalidad para un mismo índice magro.
4. – De los dos métodos descritos para determinar el Na-I, el método A (que se basa en calcular el Na-I a partir del volumen de distribución del mismo) nos parece más idóneo por:
   - ser más preciso con menos posibilidad de error;
   - permitir la determinación del espacio de sodio;
   - poder utilizarse en pacientes anúricos.
5. – La hipertensión de los pacientes con insuficiencia renal crónica terminal tratados con hemodiálisis puede ser debida a una sobrecarga hidrosalina o a un exceso de secreción de renina. La primera se acompaña de un Na-I elevado y la segunda de una actividad renina plasmática (ARP) elevada.
6. – Aún en presencia de una insuficiencia renal crónica avanzada, los riñones son capaces de segregar considerables cantidades de renina.
7. – Los niveles de ARP en estos pacientes vienen regulados por los niveles del sodio corporal.
8. – La tensión arterial de los enfermos en hemodiálisis depende del sistema renina-angiotensina y no del nivel de Na-I.
9. – El Na-I no sólo influye en la secreción de renina, sino que también aumenta su efecto presor.
10. – Los niveles de aldosterona en la insuficiencia renal crónica vendrían regulados por el sistema renina-angiotensina, tanto basales como los que resultan de la depleción hidrosalina aguda y el ortostatismo.
11. – La caliemia sería un factor de segundo orden, aunque la pérdida de potasio comportó descensos significativos de la aldosteronemia por un mecanismo directo, ya que no se observaron variaciones ni de la ARP ni del cortisol.
12. – Por el contrario, en los pacientes anéfricos el potasio juega un papel fundamental en los niveles de aldosteronemia.
13. – La heparina no tendría ningún efecto inhibitorio sobre la secreción de aldosterona.
14. – En la insuficiencia renal crónica moderada los riñones muestran una capacidad para excretar sodio disminuida, manifestada por una elevación del Na-I.
15. – En la insuficiencia renal crónica moderada los niveles de ARP dependerían del tipo de nefropatía, siendo los pacientes con nefroangiosclerosis los que mayores niveles presentan.
16. – En tales pacientes la secreción de renina no viene controlada por la retención hidrosalina ni por la filtración glomerular, lo que sugiere que hay un defecto en el control normal de la secreción de renina.
17. – En la insuficiencia renal crónica moderada o bien los niveles de renina no serían apropiados al balance hidrosalino o bien los riñones serían incapaces de compensar los efectos presores de la angiotensina II, excretando más agua y sodio.
18. – El hallazgo, en la hipertensión de la insuficiencia renal crónica moderada, de una correlación entre los niveles de ARP y la tensión arterial diastólica, sugiere que el sistema renina juega un papel importante en dicha hipertensión, igual como ocurre en los estadios terminales.

19. – La hipertensión de la poliquistosis renal incluso con funcionalismo renal conservado y en contra de lo que a primera vista pudiera parecer, sería debida a una retención hidrosalina manifestada por un Na-I y/o volumen sanguíneo elevado.
20. – Ello contrasta con el papel que desempeña el sistema renina en la hipertensión de la insuficiencia renal crónica (moderada y terminal) de otra etiología.
21. – El hallazgo en tales pacientes de niveles de ARP normales o bajos, sugiere que los riñones poliquísticos se comportarían como una estenosis bilateral de la arteria renal o como la hipertensión vasculorrenal de un monorreno.
22. – Cuando los pacientes con hipertensión esencial y funcionalismo renal normal se separan por sus niveles de Na-I bajo una dieta libre en sodio, resulta que el 65,4 % lo tienen normal y el 34,5 % tienen un Na-I elevado.
23. – Los niveles de Na-I no dependen de la ingesta de sodio, ya que la excreción urinaria del mismo era similar en ambos grupos de pacientes.
24. – Si bien existe una perfecta correlación entre los niveles porcentuales de Na-I y volumen sanguíneo, fueron mayores los aumentos del primero que los de la volemia, lo que sugiere la existencia de un fenómeno de autorregulación en tales pacientes.
25. – En estos pacientes, bajo una dieta libre en sodio, la actividad renina plasmática no viene regulada por el sodio corporal e igual como en los individuos normales, la secreción de renina disminuye con la edad.
26. – Todo ello junto con la falta de correlación de las cifras tensionales con los parámetros estudiados, sugiere que el sistema renina-angiotensina-aldosterona no constituye un mecanismo fisiopatológico primordial en la hipertensión arterial esencial benigna.
27. – La gran mayoría de pacientes con hipertensión y estenosis unilateral de la arteria renal y funcionalismo renal conservado presentan un Na-I normal, mientras que los que tienen un riñón atrófico presentan con mucha mayor frecuencia un Na-I elevado.
28. – Al contrario de lo que ocurre con el Na-I, la ARP se halló elevada con mucha mayor frecuencia entre los pacientes con estenosis de la arteria renal que entre los que presentaban riñón atrófico.
29. – La falta de correlación inversa entre el Na-I y la ARP sugiere que en condiciones basales, los niveles de renina no estarían regulados por el sodio corporal sino que también intervendría la isquemia renal.
30. – Los niveles de Na-I no dependen de la ingesta de sodio valorada por la natriuria en 24 horas, con la excepción de aquellos pacientes con estenosis unilateral y funcionalismo renal deteriorado (afectación contralateral) en que se halló una perfecta correlación directa entre el Na-I y la excreción urinaria de sodio.
31. – Como es lógico la hipertensión de los pacientes monorrenos se comportaría como una hipertensión esencial.
32. – La hipertensión de los pacientes con diabetes de origen tardío y buen funcionalismo renal se manifiesta, en su conjunto, con unos mayores niveles de Na-I y una mayor incidencia de niveles patológicos elevados que la población general e hipertensión esencial, respectivamente.
33. – A diferencia de los pacientes con hipertensión esencial, los niveles elevados de Na-I no se acompañan de estados de hipervolemia ya que el volumen sanguíneo era normal en la mayoría de los pacientes, y había una correlación inversa entre uno y otro parámetro.
34. – Los niveles de ARP en los pacientes diabéticos eran comparables a los pacientes con hipertensión esencial, a pesar de tener edades significativamente superiores a aquéllos.
35. – Los pacientes con hiperaldosteronismo primario presentaron un Na-I francamente elevado con un volumen sanguíneo dentro de la normalidad, lo que concuerda con la existencia de un fenómeno de autorregulación a nivel periférico y de escape a nivel renal.
36. – En el feocromocitoma se observó un Na-I y un volumen sanguíneo francamente disminuido, como correspondería a una hipertensión por vasoconstricción.

VII. BIBLIOGRAFÍA

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VIII. ÍNDICE DE TABLAS Y FIGURAS