LETTERS

Hyperglycemia and Extracellular Water in Dialysis Patients

We've read with attention the article by El-Hennaury and Mahmood ''Treatment of Marked Hyperglycemia in Dialysis Patients can Lead to Significant Changes in Extracellular Water'' (Dialysis and Transplantation 34(9): 616). This is a very interesting article, but we think some points deserve further consideration.

They describe a 53-year-old end-stage renal disease diabetic women requiring hemodialysis, with no residual renal function and who had suffered from sudden acute pulmonary edema secondary to marked hyperglycemia which resolved without dialysis once her blood sugar dropped to more physiological levels. A recent workup had revealed neither significant cardiac abnormalities nor thyroid dysfunction.

Lets assume, for the sake of simplicity this woman, weighing 70 kg had a total body water (TBW) content of 42 L (ICF: 28 L and ECF: 14 L) and a body fluid osmolality of 285 mOsmol/L a figure typically found in patients similar the one discussed here. Her total osmolal content should have been 11 970 mOsmol (7980 mOsmol in the ICF and 3990 mOsmol in the ECF). Then 10 g (56 mmol or mOsmol) of glucose were added to each liter of her ECF (because, due to the lack of insulin, glucose behaved as an ''effective'' osmol or ''tonomol''). Thus, these added 784 mOsmol increased ECF osmolal content to 4774 mOsmol and consequently the ECF osmolality was 341 mOsmol/L. In order to maintain the whole body osmolality, 1.7 L of water must have shifted from the ICF to the ECF (because glucose was impeded from crossing cell membranes due to the lack of insulin) and set the new osmolality in the range of 304 mOsmol/L. Therefore the new ICF volume was 26.3L (instead of 28L) and the new ECF volume was 15.7L (instead of 14L).

It is quite probable that of these $\cong$1.8 L of water added to the ECF, two thirds (1200 mL) had remained in the interstitial space and only one third (600 mL) entered the blood volume. It seems somewhat difficult this small amount of water (600 mL) could cause severe pulmonary edema unless the patient was already fluid overloaded or had severe left ventricular dysfunction.

Furthermore, it is hard to understand why the potassium concentration in the ECF was as low as 4.0mmol/L despite the severity of the hyperglycemia displayed by this unfortunate woman. Taken into account the intracellular potassium concentration is roughly 150 mmol/L, one had expected (150mmol/L $\times$ 1.7L) 255 mmol of K^+ shifting from ICF to ECF increasing the ECF [K^+] (255 mmol/15.7 L) tremendously. This huge potassium shifting was also expected to occur in this insulin-deprived ESRD woman whose only defense mechanism to avoid it should have been the elimination of potassium through the urine or the gastrointestinal tract or the transfer of potassium back to cells. As the authors stated, the patient's residual renal function was absent, and she had neither diarrhea nor other apparent external potassium loses. The only way to avoid dangerous hyperkalemia that was left in this case was potassium transfer back to cell driven by beta adrenergic stimulation.

RICARDO M HEGUILÉN, MD
ANDRÉS LISTE, MD
AMELIA R BERNASCONI, MD
DIVISION OF NEPHROLOGY-
DEPARTMENT OF MEDICINE
HOSPITAL JUAN A FERNÁNDEZ
UNIVERSIDAD DE BUENOS AIRES
ARGENTINA