Corrigendum

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Corrigendum to "Hwang KS, Kim GH: Thiazide-induced Hyponatremia. Electrolyte Blood Press 8:51-57, 2010"

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In several sections of our review paper, cited in the title, we have found some errors in quotation of sentences from the Dr. Aaron Spital's review article entitled "Diuretic-induced hyponatremia" published in American Journal of Nephrology 19:447-452, 1999. Quotation marks were missed, and we should have specifically acknowledged the source of our statements.

In Introduction (p. 51), "The first detailed description of diuretic-induced hyponatremia was published over 35 years ago[4]. Since that time, numerous additional cases have been reported" 14. Spital A: Diuretic-induced hyponatremia. Am J Nephrol 19:447-452, 1999

In Clinical characteristics of TIH (p. 53), "One of the most remarkable features of TIH is the rapidity with which it can occur. In susceptible individuals, the serum sodium may fall within hours of administration[8], and severe hyponatremia can develop within less than 2 days[7,14]. In most reported cases (50% to 90%) the duration of thiazide use was less than 2 weeks[7,8,14,22]" 14. Spital A: Diuretic-induced hyponatremia. Am J Nephrol 19:447-452, 1999

In Pathogenesis of TIH (p. 54), "Friedman et al.[8] showed that within 6 h of ingesting a single hydrochlorothiazide- amiloride tablet, previously affected patients had a small rise in urine osmolality and a fall in serum sodium of 5.5 mmol/L in association with a small gain in weight; controls had only a slight fall in serum sodium, and their mean weight fell. Although water intake was not measured, the authors suggested that thiazides might cause polydipsia which, when combined with the renal effects, results in expansion of total body water and development of hyponatremia" 14. Spital A: Diuretic-induced hyponatremia. Am J Nephrol 19:447-452, 1999

In Pathogenesis of TIH (p. 54), "While thiazide diuretics do not inhibit concentrating ability, they do impair diluting ability in several ways[15,26,34]. As mentioned above, they inhibit electrolyte transport at the cortical diluting sites, thereby raising the minimum urinary osmolality[34-36]. Diuretics may also reduce glomerular filtration rate and enhance reabsorption of Na^+ and water in the proximal nephron, diminishing fluid delivery to the distal diluting sites[35]." 14. Spital A: Diureticinduced hyponatremia. Am J Nephrol 19:447-452, 1999

In Pathogenesis of TIH (p. 54), "There is much evidence that patients with TIH are electrolyte-deficient. First, virtually all relevant studies have found that during the development of TIH, sodium balance is negative[4,6,10] Second, once diuretics are withdrawn, urinary sodium excretion falls to very low levels[4,10]. Third, many of these patients are hypokalemic[4,6, 10]. Fichman et al.[10] emphasized the importance of potassium depletion in TIH. The great majority of their 25 patients were hypokalemic, and hyponatremia was corrected in 4 of them by potassium repletion despite continued diuretic use and sodium restriction. These investigators argued that potassium depletion predisposes the patients to hyponatremia because the serum sodium concentration is dependent upon the ratio of the sum of exchangeable sodium and potassium

to total body water. They also speculated that potassium depletion might cause a shift of sodium into the intracellular space, thereby further compromising the extracellular volume and stimulating vasopressin release" 14. Spital A: Diuretic-induced hyponatremia. Am J Nephrol 19: 447-452, 1999