LETTER

Hyponatraemia related to hypopituitarism

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I read with interest the two cases of hyponatraemia secondary to hypopituitarism by Van Tienhoven et al.[1] The authors remind us that an endocrine cause of the syndrome of inappropriate antidiuretic hormone secretion (SIADH) must always be excluded. Glucocorticoid deficiency in hypopituitarism leads to inappropriately elevated antidiuretic hormone levels and mimics SIADH. The authors did not mention the bicarbonate level of their two patients, the addition of which would have been relevant because these concentrations can help to identify the true cause of hyponatraemia. We have shown that a low bicarbonate level is frequently seen in hyponatraemia related to adrenocorticotropin deficiency (TCO_2 20.5 ± 3 mmol/l and HCO_3- 20 ± 2 mmol/l)[2] while bicarbonate is normal in non-endocrine SIADH (TCO_2 25.5 ± 2.4 mmol/l and HCO_3- 25 ± 1.7 mmol/l). In subjects with a non-endocrine cause of acute hyponatraemia, a normal bicarbonate and blood acid-base equilibrium is observed, whereas during chronic hyponatraemia (> 24 h) bicarbonate is still normal but the blood acid-base equilibrium shows a mixed respiratory and metabolic alkalosis.[2,3] In hyponatraemia related to SIADH mean aldosterone levels are usually normal despite mild volume expansion. This relative hyperaldosteronism has been well documented in animals[4] and humans.[5] However, the relative hyperaldosteronism which is typically seen in SIADH and causes the aforementioned metabolic alkalosis is only present when there is adequate availability of corticosteroids.[6] In adrenocorticotropin deficiency with hyponatraemia, the relative hypoaldosteronism explains why a metabolic alkalosis does not develop and only respiratory alkalosis is observed, which explains their lower serum bicarbonate levels. Similarly, it has been shown that plasma renin activity and aldosterone are normal in nonhyponatraemic hypopituitarism patients (reflecting euvolaemia) but that cortisol plays a permissive role in the glomerulosa response to a potassium load. Under potassium chloride stimulus the aldosterone response in hypopituitarism patients was only observed when cortisol was given.[7]

This observation (a low TCO_2 level < 22 mmol/l) could be helpful as a diagnostic tool for patients with adrenocorticotropin deficiency presenting with hyponatraemia.[2]

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