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Pregabalin-induced SIADH

Youn Joo Jung*, Beom Kim[1], Dong-Young Lee[1], Hae Won Kim[1]

[1]Nephrology, VHS Medical Center, Seoul, Korea, Republic Of

Background: The Syndrome of inappropriate antidiuretic hormone secretion (SIADH) is the most common cause of euvolemic hyponatremia. Many medications have been associated with SIADH. However, Pregabalin-induced SIADH has been rarely reported.

Methods: We report a patient with hyponatremia that was induced by pregabalin and improved after discontinuation of the drug.

Results: A 69-year-old man was presented with general weakness and fever. He was diagnosed with pneumonia and antibiotics were started to treat. He had diabetes mellitus type II and hypertension as underlying disease so he had been prescribed many drugs including angiotensin-converting enzyme inhibitor, metformin, DPP IV-inhibitor, and thioctacid for a long time. And he started to take a pregabalin 150mg two weeks before the admission to relieve the pain of right heal ulceration caused by diabetes.

On admission, his vital sign was stable and biochemical laboratory analysis was within normal range including electrolyte. One week later, he turned out to have a hyponatremia of 122 mq/L (reference range, 136 to 145) and his serum sodium level decreased further to 118 mEq/L from then on. Chest x-ray revealed an improved status of pneumonia with mild infiltrations. In an additional work-up for hyponatremia, hypothyroidism and adrenal insufficiency were excluded. Laboratory studies revealed reduced serum osmolarity of 272 mOsm/kg (reference range, 289 to 302), and urine osmolarity was 303 mOsm/kg (reference range 300 to 900). Based on the patient's clinical euvolemia and biochemical data, the SIADH was diagnosed. There was no additional medication except the antibiotics for pneumonia treatment. He was placed on a fluid restriction but it was not successful to correct the sodium level. Laboratory findings were going worse demonstrating sodium level of 116 mEq/L, serum osmolarity of 249 mOsm/kg and urine osmolarity of 451 mOsm/kg. We presumed that SIADH was secondary to a pregabalin and decided to stop the prescription. We checked serum electrolyte and urine osmolarity every 6 hours. After the pregabalin was stopped, the sodium level increased to 126 mEq/L with decreased urine osmolarity of 276 mOsm/kg. After another 2 days the patient's sodium level increased to normal level as 130mEq/L. This patient was asymptomatic even his sodium level was decreased to 116 mEq/L, and he was improved biochemically after stopping pregabalin without any side effect.

Conclusion: Hyponatremia is an uncommon side effect of pregabalin. Other 2 cases of pregabalin-induced hyponatremia have been reported and one of those cases was combined with ischemic cardiomyopathy. We speculate that pregabalin-induced SIADH occurs more frequently than reported because it is usually presented with an asymptomatic and mild hyponatremia that is not found until the laboratory studies are revealed. In

conclusion, pregabalin-induced SIADH should be included in the differential diagnosis of drug-induced SIADH if other causes of hyponatremia are excluded.

Keywords: Drug associated SIADH, Hyponatremia, Pregabalin