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Case Report

Glimepiride induced Syndrome of Inappropriate Antidiuretic Hormone Secretion (SIADH): A case report

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Abstract

The Syndrome of Inappropriate Antidiuretic Hormone Secretion (SIADH) is an important cause of hyponatremia in hospitalized patients and can be caused by a variety of drugs. SIADH is a recognized side effect of 1st generation sulfonylurea (Chlorpropamide) due to its effect on vasopressin secretion. Here we described a 55-year-old woman with uncontrolled type 2 diabetes on glimepiride with SIADH presenting with anorexia, asthenia, and altered mental status. After the withdrawal of glimepiride, these symptoms were improved and sodium level returned to a normal value which demand monitoring of serum sodium level during treatment with glimepiride.

Introduction

The SIAD is produced when plasma levels of vasopressin are elevated at times when the physiologic secretion of vasopressin from the posterior pituitary would normally be osmotically suppressed. The clinical abnormality is a decrease in the osmotic pressure of body fluids, so the hallmark of SIAD is hypoosmolality. This led to the identification of the first well-described cases of this disorder in 1957 [1] and the subsequent clinical investigations that resulted in the delineation of the essential characteristics of the syndrome [2]. Although initially called the syndrome of inappropriate antidiuretic hormone (SIADH) secretion, the term SIAD is more appropriate in view of the inability to measure elevated vasopressin levels in some patients with this disorder.

Patients with SIADH exhibit hyponatremia (<130mmol/L), serum hypo-osmolality (<275 mOsmol/kg), urine osmolality not minimally low (typically >100mOsmol/kg), urine sodium concentration not minimally low (>30mmol/L), low-normal plasma urea, creatinine, uric acid, clinical euvolemia, absence

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of adrenal, thyroid, pituitary, renal insufficiency, no recent use of diuretics, exclusion of other causes of hyponatremia [3].

SIAD is the most common cause of euvolemic hypoosmolality and it is also the single most common cause of hypoosmolality of all etiologies encountered in clinical practice with prevalence rates from 20% to 40% among all hypoosmolar patients [4,5]. Many different types of tumors have been associated with SIAD, but bronchogenic carcinoma of the lung has been uniquely associated with SIAD since the first description of this disorder in 1957 [1]. Other common causes are pulmonary disorders (as tuberculosis, pneumonia, COPD, etc), disorders of the central nervous system (as for mass lesion, inflammatory disease, degenerative disease, etc), drugs, AIDS, senile atrophy, idiopathic [2]. It can be caused by a variety of drugs, mainly anticonvulsants, psychotropics, antidepressants, cytotoxics, oral hypoglycaemic agents, and opiates [3]. Glimepiride also has been reported to be associated with the SIADH [7,8].

Hyponatremia is often asymptomatic but can also be associated with a profound disturbance of cerebral function, manifesting as anorexia, nausea, vomiting, delirium, lethargy, seizures, coma related to the rate of onset rather than the severity of hyponatremia [3]. Recent studies have found that hyponatremia is independently associated with greater cognitive impairment and cognitive decline in an older population [9], and a retrospective study found that the hyponatremic patients had a 2.36-old higher hazard ratio of developing dementia. Thus the major clinical significance of chronic hyponatremia may lie in the increased morbidity and mortality associated with falls, fractures, neurocognitive impairments, and dementias in our older population as well as potential adverse effects not yet studied in humans [10].

Case summary

A 55-year-old woman attended our endocrinology outpatient clinic with generalized weakness, anorexia, nausea, and altered mental status. She was hypertensive on an amlodipine and olmesartan combination (5/20). History of diabetes for 10 years which was not well controlled with glimepiride 2mg tablet for last 3 months before which she was on linagliptine 5 mg tablet without any significant complaint. Fifteen days after starting glimepiride her symptoms started and patiently gave a history of recurrent hospital admission for recurrent hyponatremia for the last two and half months and she was treated accordingly. On examination, her blood pressure was 130/80mm of hg (no postural drop), pulse was 76 beats/minute. No clinical evidence of adrenal insufficiency, or hypothyroidism. There was no hypervolemic (subcutaneous edema) or hypovolemic (orthostatic hypotension, tachycardia, dehydration) features. There were no pointers of malignancy or chest disease. On admission serum sodium was 127mmol/L(corrected), serum potassium 4.0, FBS 18mmol/l, HbA1c 10.1%, serum osmolarity 263mOsm/kg, urine osmolarity 460mOsm/kg, urine sodium 322mmol/24 hrs, 24 hrs urine volume 2.8L, serum creatinine 0.5mg/dl, serum urea 2.1mmol/L, SGPT 13U/L, total cholesterol 131mg/dl, HDL 34mg/dl, LDL 66mg/dl, TG 153mg/dl, FT4 1.21ng/dl, TSH 4.87µIU/ml, plasma ACTH 42.1pg/ml, basal cortisol 237.0ng/ml. Chest X-ray and USG abdomen reports were normal. CT scan of the head shows mild cortical atrophy. The clinical, biochemical, and radiological data denied any central nervous system disorder, a pulmonary disorder, and renal insufficiency. Insulin was started by stopping glimepiride and fluid was restricted. The clinical symptoms and the serum sodium level improved after stopping glimepiride. She was diagnosed with SIADH induced by glimepiride. On the day of discharge, her serum sodium was 140.2mmol/L, serum potassium 3.22mmol/L, Fasting blood sugar was 8.7mmol/L, 2 hrs after breakfast blood sugar was 12.2mmol/L.

Discussion

The syndrome of inappropriate antidiuretic hormone secretion (SIADH) is a difficult clinical situation to conclude and a common cause of which comes to be drug-induced [11]. SIADH is a recognized side effect of 1st generation sulfonylurea (Chlorpropamide) due to its effect on vasopressin release [12-14].

Drug-induced SIADH is always a diagnosis of exclusion. The temporal relation of onset of SIADH and starting drug subsequently drug withdrawal and improvement of the condition suggests drug-induced SIADH. However, pituitary, adrenal, thyroid, CNS, chest diseases, and hypovolaemic and hypervolemic disorders should be excluded based on clinical features and laboratory investigations.

Hagen et al. reported in 1970 the first case in which sulphonylurea compounds (chlorpropamide and tolbutamide) induced SIADH [13]. Since then, other cases of first-generation sulphonylureas (chlorpropamide and tolbutamide) induced SIADH have been reported. The development of Chlorpropamide induced SIADH was considered due to potentiation of the action of ADH on renal tubules by increasing the sensitivity of adenylate cyclase to ADH [14]. Second-generation sulphonylurea (glimepiride) is reported to induce SIADH in many cases [7]. Interestingly, glibenclamide (a second-generation sulphonylurea) can be used to treat SIADH because this compound causes diuresis due to antagonism to the action of ADH [15]. The underlying mechanism of the glimepiride-induced SIADH observed in our present case may be glimepiride augments the peripheral (antidiuretic) action of ADH and/or increase the release of ADH [16].

Conclusion

Glimepiride is a widely used oral diabetes medicine that is used together with diet and exercise to improve blood sugar control in people with type 2 diabetes. The serum sodium levels of patients receiving glimepiride should be monitored and clinicians need to consider hyponatremia observed in patients treated with this sulphonylurea as a possible case of glimepiride-induced SIADH.

Ethical consideration

Written informed consent was taken from the patient for publishing the history and pictures. Utmost respect and sympathy were shown to the patient during treatment.

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