LETTER TO THE EDITOR

Severe and symptomatic hyponatraemia after moxifloxacin intake

Hyponatraemia, i.e., serum sodium levels below 134 mmol/l, is one of the most common electrolyte disorders, affecting up to 1% of all hospitalised patients and as many as 18% of nursing home patients. Despite the relatively large number of potential causes of hyponatraemia, antidiuretic hormone (ADH) dysregulation appears to be the most common. In the syndrome of inappropriate antidiuretic hormone secretion (SIADH), which consists of hyponatraemia, inappropriately elevated urine osmolality, excessive urine sodium excretion, and decreased serum osmolality, hyponatraemia is a result of excess water accumulation in the body.[1] Causes of SIADH can be divided into central nervous system diseases, pulmonary disorders, malignant diseases, drugs, surgery, and idiopathic. SIADH is a well-established side effect of certain psychoactive drugs, comprising antidepressants as well as antipsychotic and antiepileptic drugs, and anticancer agents.[2] In contrast, antibiotics are rare causes of drug-induced SIADH;[3] however, they require clinical attention. Therefore, we wish to add a case of moxifloxacin-associated SIADH.

A 73-year-old woman was started on antibiotic treatment with moxifloxacin 400 mg daily for acute febrile upper airway infection. The patient has been on steady antihypertensive medication, comprising hydrochlorothiazide 12.5 mg, amlodipine 5 mg, ramipril 10 mg, bisoprolol 5 mg, and olmesartan 20 mg once daily, for several years. In the following days, nausea, tiredness, agitation/anxiety, disorientation, dysarthria, and muscle cramps occurred. After an incidental fall on day 3, the patient was referred to our emergency room. Laboratory studies showed marked hyponatraemia (108 mmol/l, normal: 136 to 148) with corresponding serum hypo-osmolality (230 mOsm/kg, normal: 275 to 300) and urine osmolality (525 mOsm/kg, normal: 50 to 1400) exceeding serum osmolality, indicating SIADH in the absence of renal, adrenal, and thyroid failure. Moxifloxacin was discontinued and intravenous saline treatment was initiated. Cranial computed-tomography and chest X-ray proved negative. Serum sodium concentration normalised within eight days with the antihypertensive medication continued and the patient recovered fully.

A score of 7 out of a possible 12 on the Naranjo Adverse Drug Reaction Probability Scale indicated a probable relationship between hyponatraemia and moxifloxacin. However, it is worth noting that some of the antihypertensive drugs taken by the patient may have contributed to or aggravated the moxifloxacin-associated hyponatraemia. While diuretics, including thiazides, are common causes of drug-induced hyponatraemia, angiotensin-converting enzyme inhibitors and amlodipine are rare causes.[2]

Clinicians should be alert to hyponatraemia as a rare potential adverse effect of moxifloxacin, or possibly quinolones as a class, in addition to their well-known central nervous system adverse effects. Concomitant use of diuretics, advanced age, and female gender are risk factors for drug-induced hyponatraemia.

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