Hyponatraemia and hypothyroidism: A case report

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Abstract

In the literature, hypothyroidism-induced hyponatraemia is rare and probably occurs only in severe hypothyroidism characterised by myxoedema. An 82-year-old woman arrived at an emergency department with aphasia and a progressive cognitive impairment associated with walking problems during the last week. Examinations excluded neurological disease. The diagnosis showed a clinically relevant association between hyponatraemia and hypothyroidism disorder characterised by neurological symptoms. In conclusion, this could be one of the few cases, to our knowledge, of non-severe hypothyroidism-induced hyponatraemia.

KEYWORDS: Hyponatremia; hypothyroidism; neurology.
Riassunto


Competing interests - none declared.

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TAKE-HOME MESSAGE

Even though hypothyroidism-induced hyponatraemia is rare, our case report showed that the link between non-severe hypothyroidism and hyponatraemia should be a consideration when an old patient affected by neurological symptoms comes to an emergency department.
INTRODUCTION

Hyponatraemia is the most common electrolyte disorder, being the consequence of many different causes. For instance, research often suggests a link between hyponatraemia and hypothyroidism, even though it may not be very well defined [1]. Although previous research has traditionally implicated hypothyroidism in the development of serum hyponatraemia, more recent research has stated that this relationship might be only a coincidental association [2]. Along these lines, a 2017 review showed that hypothyroidism-induced hyponatraemia is rare and probably occurs only in severe hypothyroidism characterised by myxoedema [3]. This report presents a previously healthy patient without thyroid disorder who arrived at an emergency department with severe hyponatraemia.

CASE REPORT

The patient was an 82-year-old woman who arrived at the emergency department with aphasia. The family reported that she had a progressive cognitive impairment associated with walking problems during the last week. The physical examination showed regular heart sounds with a low heart rate (HR). Electrocardiographic (ECG) monitoring revealed a severe bradycardia (HR = 35 bpm). The blood pressure was 140/90 mm Hg. The neurological examination did not find any hemiplaegia and excluded neurological diseases such as attention deficit disorder (ADD). Computed-tomography (CT) scans of the brain were normal; the blood test showed a low platelet count (PLT = 81,000) and a low plasma sodium concentration (Na = 114 mEq/L). All other blood tests were normal. We started to treat hyponatraemia by infusing some hypertonic intravenous solutions, and to control bradycardia by administering intravenous atropine sulphate and theophylline solutions resulting in a rapid heart rate response. The patient presented a progressive clinical and laboratory improvement (Na = 120 mEq/L, HR = 60 bpm). We suspected she was affected by hypothyroidism. Subsequently, a measurement of serum thyroid hormones confirmed a thyroid disorder diagnosis (TSH = 8.24 uIU/mL, T4 = 1.04 ng/ml). Therefore, we immediately started a substitution therapy resulting in a slow improvement of the patient’s neurological symptoms.

DISCUSSION

Hyponatraemia is a common electrolyte disorder defined as a serum sodium level of less than 135 mEq per L [4]. Patients are often asymptomatic but can show irritability, nausea, weakness, altered mental status or coma [5]. Hyponatraemia occurs particularly in the elderly [6] and may result from renal or extra-renal loss of sodium, syndrome of inappropriate antidiuresis, hypocortisolism, liver cirrhosis, heart failure, renal failure or nephrotic syndrome. Another cause is hypothyroidism, which some authors have questioned because this relationship is not clear. According to some authors, hypothyroidism, even when severely undertreated, appears not to be a clinically relevant cause of hyponatraemia [7]. Indeed, the association between hypothyroidism and clinically relevant hyponatraemia may be restricted only to the status of myxoedematous coma [8]. Conversely, our report showed a clinically relevant case of probable hyponatraemia induced by mild hypothyroidism. Typical neurological manifestations such as aphasia and progressive cognitive impairment characterised this condition. Indeed, infusing some hypertonic intravenous solutions improved the patient’s condition. However, hypothyroidism disorder could also have caused the neurological symptoms. To this end, substitution therapy led to the conclusive resolution of the case. The association between hyponatraemia and hypothyroidism disorder probably caused the neurological symptoms. In conclusion, this could be one of the few cases, to our knowledge, of non-severe hypothyroidism disorder correlating with hyponatraemia.
References


