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A Case Series

Syndrome of Inappropriate Secretion of Antidiuretic Hormone Associated with H1N1 Infection

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Abstract:
The syndrome of inappropriate secretion of antidiuretic hormone (SIADH) is related to several conditions. However, there are few reports associated with H1N1 virus infection. We describe a 36 year old patient with SIADH admitted to our department if internal medicine with fever, cough and myalgias. The labooratory analysis showed serum hypo-osmolar hyponatremia and high level of urinary sodium concentration. All other biological analysis were within normal range. Moreover, All bacterial and fungal cultures, blood and urine cultures were all negative for any microbial pathogen. The thoraco-abdominal CT scan was normal. Rapid flu test was positive for influenza type A 15 days after. The patient recovered with intravenous infusion of hypertonic saline and fluid restriction and he did not received any anti-viral drug. Despite a strong relationship betwwen H1N1 virus infection and SIADH needs to be established, physicians should be aware of this potential seroius effect and should monitor the serum sodium closely.
Keywords : H1N1 infection, inappropriate secretion of antidiuretic hormone, hyponatremia

Introduction

The syndrome of inappropriate secretion of antidiuretic hormone (SIADH) is the most common cause of hyponatremia (1,2). The underlying diseases associated with SIADH are mainly malignancies, pulmonary diseases and central nervous system diseases (1,2,3). We describe a patient with SIADH during the course of H1N1 virus infection and we reviewed the other cases reported in literature. To the best of our knowledge, this is the 9th report of SIADH associated with H1N1 virus infection.

Case report :

A 36-year-old man with no medical history was admitted to our department of internal medicine for persisting fever, non productive cough and myalgias for 10 days. He did not received any medication at home. Physical examination at the time of admission revealed a well-orientated man and headache. His temperature was 39.2°C, blood pressure 140/80 mmHg, pulse 102 beats per minute and respiration rythm 18/minute. No other abnormal findings were noted.

Laboratory data showed elevated markers of inflammation (Table 1). Hyponatremia 121 mmol/l was noticed, kalemia 3.87 mmol/l and serum osmolality was 257 mOsm/kg (normal range: 280-300 mOsm/kg). However, urinary sodium concentration and osmolality were

high: 78 mmol/24h, 3174.8 mOsm/kg respectively (Table 1). A lumbar puncture demonstrated clear cerebrospinal fluid (CSF) with 2 cells/mm^3, glucose : 0.7 g/l and protein 0.65 g/l. All bacterial and fungal cultures, blood and urine cultures were all negative for any microbial pathogen. Screening of mycobacterium tuberculosis in the respiratory tract was negative. Thyroid function and adrenal steroid hormones were normal. A chest x-ray did not show abnormalities. Abdominal ultrasound, echocardiography and thoraco-abdominal CT scan were unremarkable.

Rapid flu test was positive for influenza type A 15 days after. Intravenous infusion of hypertonic saline and fluid restriction were started and serum sodium concentration inceased to 135 mmol/l in the 7^{th} day.

Since pulmonary and central nervous system disorders remained negative, the patient was diagnosed for SIADH due to H1N1 (Influenza A) virus infection. Our patient did not received any anti-viral treatment and he recovered within 1 month with no recurrence of fever and normal sodium level.

Discussion:

SIADH was first described in 1957 by Schwartzaelt (). SIADH is associated with a large number of diseases such malignancy, pulmonary conditions, central nervous system disorders and medications in clinical practice (1,2,3).

Our patient met the SIADH criteria : hypo-osmolar hyponatremia, urine osmolality >100 mOsm/kg, urine sodium concentration >40 mmol/l, euvolemic state with normal renal, thyroid, and adrenal functions. Since the present case had no previous described conditions of SIADH, we considered that the hyponatremia and SIADH resulted from H1N1 virus infection. In fact, our patient had no evidence of malignant tumor in CT scan and no malignant cells were found in the CSF. Infections and other disorders of the central nervous system and respiratary tract have also been reported to be the underlying diseases causing SIADH (1,2). Nevertheless, our patient had no neurological abnormalities , no signs of meningial irritation and no documented pulmonary infection. Furthermore, throat and nasal specimen to the H1N1 virus was positive.

Few cases of SIADH during the course of viral infections were reported (2,3,4,5,7). Far as we know, only 9 other cases, 5 women and 4 men, of SIADH have been described in association with H1N1virus infection (4-11) (Table 2). The most frequent ical manifestations included neurological disorders, dyspnae, fever and cough (Table 2). The diagnosis of H1N1 infection was often established after a mean duration of 8 days (extremes : 2-17 days) (4,5,9,10). All patients had a laboratory-confirmed H1N1 virus infection. The delay to diagnosis of SIADH due to H1N1 virus infection may in part be due to the lack of awareness of this condition amongst physicians. Otherwise, the clinical presentation is miscellaneous and often misleading.

Fluid restriction should be the mainstay therapy and 0.9% saline solution is a safer alternative according to recommendations allowing for an increase in serum sodium of 10-12 mmol/24 hours (11,12). Our patient recovered within 1 month with no recurrence.

Only 4 patients were treated with Oseltamivir. There was a complete recovery in 5 patients (4, 6, 8, 9, 11) and neurological sequelae in one patient (5).

Conclusion:

Considering the SIADH with severe hyponatremia as an adversely clinical outcome in increased mortality, and extended hospitalisation, physicians should then be aware of this potential side effect and should monitor the serum sodium closely.

Table 1: Laboratory data :

	Results
Hemogram	
WBC	**9480/mm^3**
Hb	**16 g/dl**
Platlet count	**167000/mm^3**
Blood chemistry	
Na+	**121.9mmol/l**
K+	**3.67mmol/l**
Creatinine	**82.1µmol/l**
Glucose	**6.22 mmol/l**
Ca2+	**2.2mmol/l**
SGOT/SGPT	**69/105 U/l**
LDH	**172 U/l**
C reactiv protein	**105.19 mg/l**
Blood osmolality	**253.3 mOsm/kg**
Urinalysis	
Na+	**78 mmol/24h**
K+	**29 mmol/24h**
Cl-	**71 mmol/24h**
Urea	**182.7 g/l**
Creatinine	**3197.7 µmol/l**
Urin osmolality	**3174.8 mmol/24h**
Cortisol	**370 .74 ng/ml**
TSH	**1.23 U/ml**
T4	**22.7 pmol/l**

Table 2: Cases of SIADH associated with H1N1 virus infection

Cases	Age/sex	History	Na+/osmolality	Duration (days)	Antiviral therapy	Follow-up
1 (4)	3/female	Rhabdomyolysis	121mmol/l/272mmol/kg	11	None	Recovery
2 (5)	68/female	Neurological disorder	108mmol/l/214mmol/kg	17	None	Neurological sequelae
3 (6)	7/male	Delirium	SIADH	Unknown	Oseltamivir	Recovery
4 (7)	?/female	?	SIADH	Unknown	?	Unknown
5 (8)	75/male	Cough and chest pain	130mmol/l/261mmol/kg	Unknown	Oseltamivir	Recovery
6 (9)	7/male	Seizures	131mmol/l/ ?	2	Oseltamivir/ Rimantadine	Recovery
7 (10)	26/female 29/male	Dyspnae Cough, chest pain and dyspnae	126mmol/l/ ? 123mmol/l/ ?	2 5	None None	Died Died
8 (11)	65/male	Fever, productive cough and dyspnae	122mmol/l/276mmol/kg	Unknown	Oseltamivir	Recovery
9 (our case)	36/male	Cough, dyspnae	121.9mmol/l/253.3mmol/kg	15	None	Recovery

Ethics approval and consent to participate

The authors obtained the approval of research ethics committee of the regional hospital of Ben Arous to carry out this study.

Consent for publication

A written consent was obtained from the described subject to publish his data.

No identifying information of the patient appears in writing or within image.

Availability of data

Not applicable.

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