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ORIGINAL ARTICLE

Hyponatremia in spinal cord injury patients: new insight into differentiating between the dilution and depletion forms

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Study design: This is a retrospective study.
Objectives: The objectives of this study were to present a new model for differentiating between the dilution and depletion forms of hyponatremia in patients in the postacute phase after spinal cord injury (SCI), and to identify possible etiological factors contributing to hyponatremia in these patients.
Setting: University Hospital Motol, Prague, Czech Republic.
Methods: Eighty-seven of 352 patients hospitalized in 2008–2012 in the Spinal Cord Unit were hyponatremic. Seventy-four patients had $S_{Na+} = 130–135$ mmol l^{-1} and 13 patients had S_{Na+} below 130 mmol l^{-1}. We propose a simple model of an electrolyte solution in which the Na^+ concentration is higher than the Cl^- concentration, making it possible to compare the effects of dilution and depletion of Na^+ and Cl^- on the Na^+ concentration. The depletion of Na^+ and Cl^- leads to a significant increase in the Na^+/Cl^- ratio, with the $Na^+–Cl^-$ value remaining unchanged. Dilution with water results in a decrease of $Na^+–Cl^-$ with the Na^+/Cl^- ratio remaining unchanged.
Results: In patients with S_{Na+} below 130 mmol l^{-1}, hyponatremia was consistent with the depletion model in 46% and with the dilution model in 32%. In patients with S_{Na+} ranging between 130 and 135 mmol l^{-1}, the respective rates were 34 and 12%.
Conclusion: Examination of $S_{Na+}–S_{Cl-}$ and S_{Na+}/S_{Cl-} in patients with SCI could be helpful in considering whether hyponatremia is consistent either with the NaCl dilution model or with the NaCl depletion model. Further studies are needed for more accurate interpretation of the results, particularly with respect to volume and acid–base disorders.
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INTRODUCTION

Spinal cord injury (SCI) results in impaired function of the somatic and autonomic nervous systems below the level of injury. One of the consequences of SCI is the presence of metabolic abnormalities. The prevalence of hyponatremia is significantly higher in SCI patients compared with other diseases or postsurgical conditions.[1] Hyponatremia is most often reported within the first 2 weeks after SCI,[2] but it can be observed even later.[3] The cause is multifactorial; however, etiological factors involved in the development of hyponatremia in SCI patients differ, in part, from those in the general population.[3,4]

When considering hyponatremia, it is necessary to differentiate whether the cause is water retention (dilution hyponatremia) or loss of Na^+ and Cl^- (depletion hyponatremia). Dilution hyponatremia is mainly caused by inappropriate secretion of antidiuretic hormone (SIADH), whereas depletion hyponatremia results from an increase of renal or extrarenal natrium wasting. If the renal wasting is related to brain injury, it is called cerebral salt wasting syndrome. Differentiation between these two forms is difficult, and primarily it is diagnosed on the basis of volume change observation.[5,6]

Differential diagnosis between dilution and depletion hyponatremia is essential for appropriate therapy. Patients with sodium loss would benefit only from sodium provision, whereas those with fluid overload would benefit from fluid restriction or water diuresis. Decrease in the serum sodium concentration (S_{Na+}) needs to go along with a reduced anion concentration. From the quantitative perspective, changes in the concentrations of chlorides (S_{Cl-}) and bicarbonates (S_{HCO3-}) are likely to occur. The changes to the S_{Cl-} to S_{HCO3-} concentration ratio in the presence of hyponatremia may vary widely, and therefore the development of hyponatremia can be associated with changes to the acid–base balance. The S_{Na+}/S_{Cl-} ratio and $S_{Na+}–S_{Cl-}$ difference have been used successfully in the differential diagnosis of acid–base balance disorders.[7–11] These findings challenged us to investigate whether the above-mentioned values vary concordantly or independently in dilution and depletion hyponatremia and whether their calculation could be helpful in the differential diagnosis between dilution and depletion hyponatremia. We designed a simple model of an electrolyte solution in which the Na^+ concentration was higher than the Cl^- concentration, similarly as is the case in the extracellular fluid, to see how dilution with water and depletion of NaCl influence the $Na^+–Cl^-$ and Na^+/Cl^- values. On the basis of these findings, we want to specify whether the diagnosis of hyponatremia in SCI patients is consistent either with the dilution model or the NaCl depletion model.

PATIENTS AND METHODS

Laboratory data of patients hospitalized in the Spinal Cord Unit (SCU) were analyzed retrospectively for the 5-year period 2008–2012. The hospitalized cohort included 352 patients with acute SCI. Eighty-seven patients had, at least once, the serum Na^+ level below the limit of 135 mmol l^{-1}. The mean age of these 87 patients at the time of SCI was 51.1 ± 17.5 years. They were admitted to the SCU with an average interval of 44.3 days after SCI. The patients were

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grouped according to the severity of hyponatremia.[12] Seventy-four patients were assigned to class I (S_{Na+} = 130–135 mmol l^{-1}), six patients to class II (S_{Na+} = 125–129 mmol l^{-1}) and seven patients to class III (S_{Na+} 115–124 mmol l^{-1}) (Table 1). None of the patients in the study met the class IV criteria (S_{Na+} < 115 mmol l^{-1}). All study patients were also checked for levels of other ions, creatinine, plasma osmolality and CRP and most patients assigned to classes II and III also checked for urine Na^+ and Cl^- and urine osmolality.

The main goal of the differential diagnosis is to determine whether hyponatremia is due to dilution or depletion of Na^+ and Cl^-. Differentiating between these two forms is of high importance, as it influences the choice of therapeutic regimen. The potential of the S_{Na+}–S_{Cl-} and S_{Na+}/S_{Cl-} for use in the differential diagnosis between the two types of hyponatremia was supported by the following generally accepted hypotheses. When considering changes in the Na^+ concentration, it is assumed that the distribution volume of Na^+ correlates with total body water.[13] The simple mathematical model proposed by us is a single-compartment system, as the body maintains isotonicity of the extracellular fluid with the intracellular fluid and the shifts of water between these two compartments influence accordingly the Na^+ and Cl^- concentrations. Figure 1 shows a compartment with 20 l of electrolyte solution containing Na^+ (140 mmol l^{-1}), Cl^- (100 mmol l^{-1}) and HCO_3^- (40 mmol l^{-1}). On the upper panel of the figure, two liters of water are added to model the dilution. Consequently, the Na^+–Cl^- decreases from 40 to 36 mmol l^{-1}. On the other hand, the Na^+/Cl^- remains unchanged (1.40). The lower panel of the figure illustrates the loss of 200 mmol of Na^+ and 200 mmol of Cl^-, with the solution volume remaining unchanged. Under such conditions, the Na^+–Cl^- remains unchanged, but the Na^+/Cl^- increases to 1.44. This serves as a model of the Na^+ depletion. If the solution volume decreased, for example, by 1 l, the Na^+/Cl^- would remain unchanged, but the Na^+–Cl^- would increase. Renal sodium wasting is associated with the loss of both ions. If more Cl^- than Na^+ were lost, both the Na^+/Cl^- and Na^+–Cl^- would increase.

To enable the use of the above-mentioned simple model in clinical practice, the normal ranges of the S_{Na+}–S_{Cl-} and S_{Na+}/S_{Cl-} need to be established in healthy individuals. For these reasons, 33 healthy adults, health-care professionals, with average eating habits were investigated. The following means and s.d. were obtained for the parameters under study: S_{Na+}–S_{Cl-} = 35.0 ± 1.6 mmol l^{-1} and S_{Na+}/S_{Cl-} = 1.33 ± 0.02. The normal ranges calculated as mean ± 2 s.d. were 31.8–38.3 mmol l^{-1} and 1.29–1.37 mmol l^{-1}, respectively.

Table 1 Characteristics of the study population

Class (mmol l^{-1})	*I. (130–135)*		*II. (125–129)*		*III. (115–124)*	
	N	%	N	%	N	%
Sex						
Male	56	75.7	3	50.0	6	85.7
Female	18	24.3	3	50.0	1	14.3
NLI						
C	31	41.9	5	83.3	5	71.4
T	27	36.5	1	16.7	2	28.6
L	16	21.6	0	0.0	0	0.0
AIS						
A	32	43.2	3	50.0	2	28.6
B–D	42	56.8	3	50.0	5	71.4
Cause						
Traumatic	55	74.3	3	50.0	5	71.4
Nontraumatic	19	25.7	3	50.0	2	28.6
Total	74	100.0	6	100.0	7	100.0

Abbreviations: AIS, ASIA Impairment Scale; NLI, neurological level of injury.

To what extent the above-mentioned model is consistent with the known clinical findings of changes in S_{Na+}–S_{Cl-} and S_{Na+}/S_{Cl-} in altered acid–base balance associated with volume changes is commented on in the Discussion section.

The following step was the identification of all factors possibly involved in the development of hyponatremia in each individual patient. We considered factors responsible for water retention and those responsible for increased loss of Na^+ and Cl^-.

RESULTS

Classes II and III (S_{Na+} = 115–129 mmol l^{-1})

Hyponatremia in the range shown above was found in 13 SCI patients. Their basic laboratory test results are summarized in Table 2. The level of hyponatremia below 135 mmol l^{-1} was detected with a mean of 13.6 times in this group. From the table, it is evident that in all these patients hyponatremia was associated with a decrease in S_{Cl-} (< 97 mmol l^{-1}) and S_{osm} (< 295 mmol kg^{-1}). The S_{Na+}–S_{Cl-} was below the lower limit of the normal range (< 32 mmol l^{-1}) in four patients, and in one patient it was close to the limit value. The upper limit of the normal range (> 37 mmol/) was exceeded in four patients, whereas in the other patients under study the values were in the normal range. In five patients, the S_{Na+}/S_{Cl-} was above the upper limit of the normal range (> 1.37), in two patients this ratio was close to the limit and in the remaining patients this ratio was in the normal range.

When the S_{Na+}–S_{Cl-} and the S_{Na+}/S_{Cl-} were considered together (Table 3), the dilution model was applicable to four patients (decreased S_{Na+}–S_{Cl-} and the S_{Na+}/S_{Cl-} in the normal range). The depletion model was applicable to seven patients (increased S_{Na+}/S_{Cl-} and the S_{Na+}–S_{Cl-} in the normal range or increased).

The U_{osm} value was above 100 mmol kg^{-1} in all patients, being higher than S_{osm} in six of them. The urinary concentration of Na^+ was above 30 mmol l^{-1} in all but one patients. The urinary Na^+/Cl^- (U_{Na+}/U_{Cl-}) was close to 1.0 (with a mean of 0.994). From Table 2, it is evident that the serum creatinine concentrations were very low (29–99 μmol l^{-1}), 50 μmol l^{-1} on average.

Of four patients assigned to the dilution hyponatremia group, two had acute urinary infection, and in the three patients the cause of hyponatremia may have been the synthetic antidiuretic hormone (ADH) given to treat polyuria. Moreover, all these patients took antidepressants, one of them in combination with antiepileptics, and two patients had pronounced arterial hypotension. In six patients with depletion hyponatremia, the cause may have been impaired autonomic regulation of kidney function.

Class I (S_{Na+} = 130–135 mmol l^{-1})

Seventy-four (85.1%) patients had hyponatremia, with the S_{Na+} serum levels between 130 and 135 mmol l^{-1} and S_{Cl-} serum levels between 89 and 106 mmol l^{-1}. Hyponatremia was detected most often only once, with a mean of 3.2 times. In these mildly hyponatremic patients, serum electrolytes were only checked and the S_{Na+}–S_{Cl-} and S_{Na+}/S_{Cl-} were calculated. The values of the S_{Na+}–S_{Cl-} and S_{Na+}/S_{Cl-} established for individual patients are shown in Figure 2. The results are summarized in Table 3, from which it is evident that the dilution model was applicable to four patients (decrease in the S_{Na+}–S_{Cl-} and the S_{Na+}/S_{Cl-} in the normal range), and five patients had lower values of S_{Na+}–S_{Cl-} and at the same time lower S_{Na+}/S_{Cl-}. The depletion model was applicable to 29 patients (increase in the S_{Na+}/S_{Cl-} and the S_{Na+}–S_{Cl-} in the normal range or elevated).

Of nine patients with dilution hyponatremia, six had acute infection and five of these had urinary infection at presentation. The others had

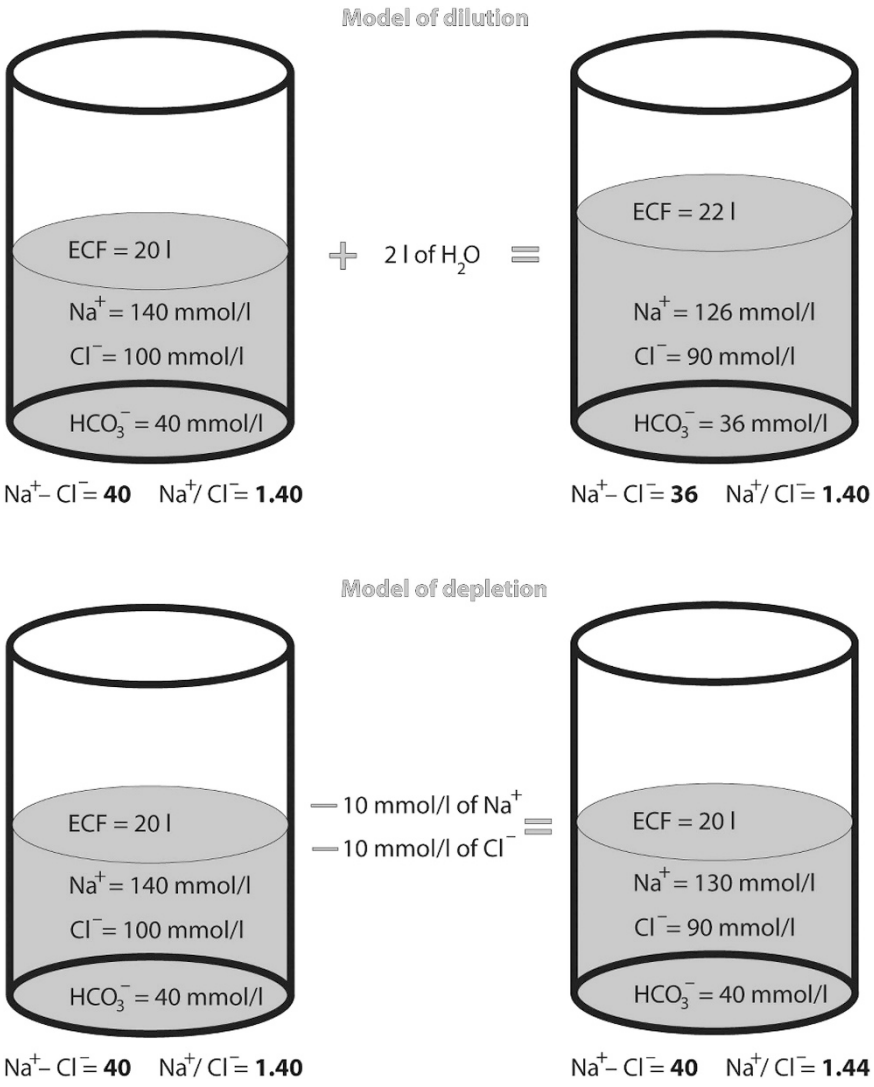

Figure 1 Mathematical model to illustrate the Na^+–Cl^- and Na^+/Cl^- changes after water retention or Na^+ and Cl^- depletion. Abbreviations: ECF, extracellular fluid; Na^+, sodium; Cl^-, chloride; HCO_3^- bicarbonate; H_2O, water; Na^+–Cl^-, sodium–chloride difference; Na^+/Cl^-, sodium–chloride ratio.

pronounced arterial hypotension. All patients were treated by antidepressants and two of them in combination with antiepileptics. In 29 patients, depletion hyponatremia might be caused by impaired autonomic regulation of kidney function. Other factors that may have had a role in hyponatremia were significantly low blood pressure (systolic blood pressure below 100) in 14 patients, diuretics given to control chronic arterial hypertension in four patients, diarrhea in one patient and vomiting in one patient.

Practically in all study patients, multiple factors possibly involved in the development of hyponatremia were identified. These factors are listed and their distribution is presented in Table 4.

DISCUSSION

In the literature, higher prevalence of hyponatremia has been generally reported in SCI or surgery patients. The rates of hyponatremia reported in SCI patients range from 25 to 80%.[1,2,4] Nevertheless, these rates relate to the patients in the acute phase or in the first month after SCI. In our cohort of 352 patients admitted to the SCU on day 44 following SCI on average, 24.7% had hyponatremia.

In general, it is difficult to differentiate between the two types of hyponatremia and thereby select the appropriate treatment. After establishing S_{Na+}–S_{Cl-} and S_{Na+}/S_{Cl-}, it is possible to consider which of the models is applicable to hyponatremia diagnosed in a particular patient. Verifying whether the conclusion is correct would require simultaneous measurement of volume changes. These data are not available to our study cohort, but there is indirect evidence for differentiating between dilution and depletion hyponatremia on the basis of clinical findings in altered acid–base balance associated with volume changes. Dilution owing to water retention results in dilutional acidosis, characterized by a decrease in strong ion difference (SID).[14] Reduced SID correlates significantly with a decrease in S_{Na+}–S_{Cl-} (i.e., with the same changes as seen in the dilution model).[7] Large renal losses of Na^+, Cl^- and water resulting from intensive therapy with diuretics can lead to hypovolemia and hypochloremic metabolic alkalosis.[15,16] Such an alteration of electrolyte and volume homeostasis is associated with increased S_{Na+}–S_{Cl-} and S_{Na+}/S_{Cl-} (or decreased S_{Cl-}/S_{Na+})[7] (with the same changes as observed in the depletion model).

Table 2 Demographic, clinical and laboratory profile of patients in class II and III hyponatremia

N	Sex	Age	NLI	AIS	Cause	Day of adm.	S_{Na+} (mmol l^{-1})	S_{K+} (mmol l^{-1})	S_{Cl-} (mmol l^{-1})	S_{osm} (mmol kg^{-1})	S_{cr} (μmol l^{-1})	CRP (mg l^{-1})	S_{Na+}–S_{Cl-}	S_{Na+}/S_{Cl-}	U_{Na+} (mmol l^{-1})	U_{Cl-} (mmol l^{-1})	U_{osm} (mmol kg^{-1})	U_{Na+}/U_{Cl-}
1	M	71.3	C3	D	Fall	4	128	4.4	90	270	61	2.1	38	1.42	—	—	—	—
2	M	64.2	C4	A	Fall	43	126	4.9	92	275	54	9.5	34	1.37	37	37	165	1.00
3	F	61.0	C4	A	Ischemia	12	128	3.9	93	259	43	55.7	34	1.38	25	25	188	1.00
4	M	71.6	C4	C	Fall	35	115	4.4	84	241	33	6.1	31	1.37	97	106	297	0.92
5	M	53.8	C4	B	Myelitis	77	120	4.4	80	252	32	31.7	40	1.50	46	51	197	0.90
6	M	46.6	C4	C	Skiing	12	115	4.2	87	241	52	52.9	28	1.32	104	82	521	1.27
7	M	59.6	C4	C	Fall	8	122	4.3	82	258	37	44	40	1.49	66		385	
8	M	35.3	C5	A	Jump to water	16	126	4.3	93	253	57	6.6	33	1.35	52	57	252	0.91
9	F	66.2	C5	C	Discitis	21	126	4.2	91	275	69	57.2	35	1.38	—	—	—	—
10	M	42.2	C5	A	Car accident	64	123	4.1	91	254	31	2	32	1.35	187	200	628	0.94
11	M	48.2	T6	A	Car accident	30	124	3.9	94	275	29	109	30	1.32	94	89	—	1.06
12	F	78.2	T11	D	Ischemia	26	127	4.5	88	263	54	2	39	1.44	59	63	420	0.94
13	F	77.1	T12	C	Fall	11	124	4.4	94	267	99	1	30	1.32	67	67	377	1.00

Abbreviations: adm., admission; AIS, ASIA Impairment Scale; CRP, C-reactive protein; NLI, neurological level of injury; S_{Cl-}, serum chloride; S_{cr}, serum creatinine; S_{K+}, serum potassium; S_{Na+}, serum sodium; S_{Na+}–S_{Cl-}, serum sodium–serum chloride difference; S_{Na+}/S_{Cl-}, serum sodium–serum chloride ratio; S_{osm}, serum osmolality; U_{Cl-}, urinary chloride; U_{Na+}, urinary sodium; U_{Na+}/U_{Cl-}, urinary sodium–urinary chloride ratio; U_{osm}, urinary osmolality.

All patients whose urine Na^+ and Cl^- concentrations and osmolality were also analyzed had U_{OSM} levels above 100 mmol kg^{-1}, and all but one had U_{Na+} levels above 30 mmol l^{-1}. These results, in the presence of hyponatremia, are in accordance with the most used laboratory criteria, of SIADH and, consequently, of dilution hyponatremia.[6] We assume that the discrepancy between the commonly used approaches in the assessment of laboratory data indicative of SIADH and differentiation between the two types of hyponatremia based on changes in S_{Na+}–S_{Cl-} and S_{Na+}/S_{Cl-} could be explained as follows. In hyponatremia due to renal depletion of Na^+ and Cl^- (renal salt wasting syndrome), it is understandable that the urine Na concentrations exceed the criterion of 20 mmol l^{-1} and are often even higher than 30 mmol l^{-1}. Depletion of Na^+ and Cl^- is associated with hypovolemia, which induces nonosmotic activation of ADH secretion. An increased ADH level enhances tubular reabsorption of water, and thus the U_{OSM} level tends toward an increase and can exceed not only 100 mmol kg^{-1} but also the S_{OSM} level. It means that in depletion hyponatremia laboratory symptoms considered typical of SIADH may develop. Under such conditions, ADH secretion is stimulated. It is not inappropriate as is the case in SIADH, but it occurs as a physiological response to hyponatremia owing to extracellular fluid volume reduction, resulting mainly from the decrease in effective intravascular volume. Nevertheless, in patients with SIADH, ADH secretion is not stimulated as a result of regulation of volume homeostasis of the internal environment, but it is triggered by various pathogenetic or pharmacological stimuli.

In the light of the above-mentioned pathophysiological phenomena, it is evident that the laboratory criteria used for the urinary Na^+ concentrations and osmolality may not differentiate between SIADH and renal salt wasting in the presence of hyponatremia. In our opinion, apart from the criteria used, it would be helpful, for example, to differentiate between increased ADH production as a physiological response to hypovolemia (even in the presence of hyponatremia and reduction of extracellular fluid osmolality) and a pathological response resulting in inappropriate ADH secretion. The results of the present study and the presented differential diagnostic hypothesis for dilution and depletion hyponatremia suggest that in addition to the criteria used the S_{Na+}–S_{Cl-} and S_{Na+}/S_{Cl-} values would be useful to consider. However, further study of the relationship between the above-mentioned values and volume changes will be needed to test this assumption.

Differentiating between dilution and depletion hyponatremia also provides a crucial sign as to the use of vaptans (drugs that increase solute-free water excretion). In accordance with the current pathophysiological knowledge, vaptans are expected to be effective in dilution hyponatremia where water excretion needs to be enhanced (without influencing Na^+ excretion). In patients with depletion hyponatremia showing signs of hypovolemia, pharmacological reduction of tubular water resorption could deepen the water deficit in the body and aggravate the hemodynamic manifestations of hypovolemia. Current practice has been considered in the US evidence based and expert panel reviewed guidelines[17] and in European practice guideline on diagnosis and treatment of hyponatremia.[18]

Future research will also require a thorough clinical evaluation of the potential therapeutic benefit of the proposed approach for the treatment of hyponatremia in SCI patients.

The reason behind the higher incidence of hyponatremia in SCI patients is most likely because of the impairment of the autonomic nervous system. The loss of peripheral vascular resistance in patients with impairment of sympathetic vascular innervation after cervical and upper thoracic cord injuries results in chronic arterial hypotension and

Table 3 Various combinations of changes in serum sodium–serum chloride difference and serum sodium–serum chloride ratio

N	S_{Na+} mmol l^{-1}	S_{Cl-} mmol l^{-1}	S_{Na+}–S_{Cl-} ↘ S_{Na+}/S_{Cl-} →	S_{Na+}–S_{Cl-} ↘ S_{Na+}/S_{Cl-} ↘	S_{Na+}–S_{Cl-} → S_{Na+}/S_{Cl-} ↗	S_{Na+}–S_{Cl-} ↗ S_{Na+}/S_{Cl-} ↗	S_{Na+}–S_{Cl-} → S_{Na+}/S_{Cl-} →
13	115–129	80–94	4 (30.8%)	0 (0%)	3 (23.1%)	3 (23.1%)	3 (23.1%)
74	130–135	91–106	4 (5.4%)	5 (6.8%)	17 (23.0%)	12 (16.2%)	36 (48.6%)

Abbreviations: S_{Na+}–S_{Cl-}, serum sodium–serum chloride difference; S_{Na+}/S_{Cl-}, serum sodium–serum chloride ratio; ↘, decreased; →, in range; ↗, increased.

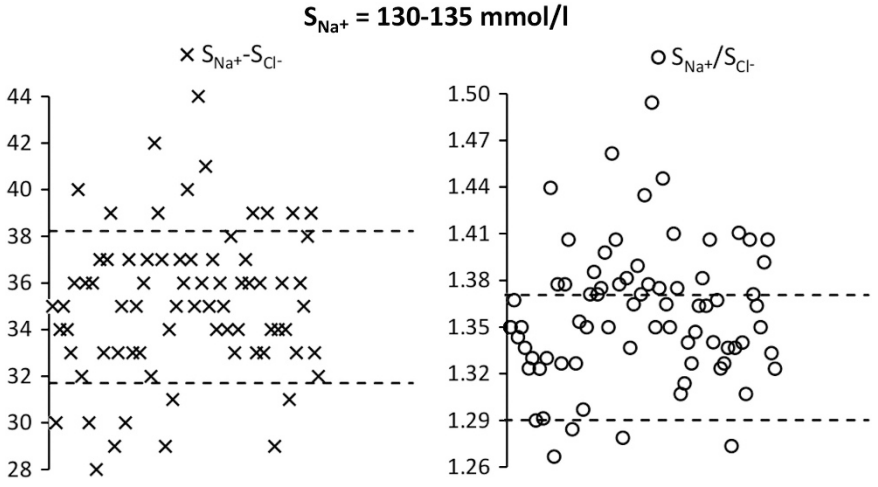

Figure 2 The values of S_{Na+}–S_{Cl-} and S_{Na+}/S_{Cl-} in class I (S_{Na+} = 130–135 mmol l^{-1}). Borderlines denote the normal range of S_{Na+}–S_{Cl-} and S_{Na+}/S_{Cl-}.

Table 4 Etiological factors considering to cause hyponatremia in SCI patients

	N	%
Total no. of hyponatremic patients	87	100.0
Renal sympathetic disruption	87	100.0
Diarrhea	4	4.6
Vomiting	2	2.3
Tumor	3	3.4
Diuretics	7	8.0
Significant hypotension	33	37.9
Urinary tract infection	30	34.5
Other infection or sepsis	10	11.5
Antidepressant		
SSRIs	67	77.0
SNRIs	2	2.3
Others	10	11.5
Antiepileptics	17	19.5
Desmopressin	5	5.7

Abbreviations: SSRIs, selective serotonin reuptake inhibitors; SNRIs, serotonin–norepinephrine reuptake inhibitors.

orthostatic hypotension. As a nonosmotic stimulus, hypotension exerts an effect on the secretion of ADH. The stimulating effect of hypotension overcomes the inhibitory effect of hypotonicity. Thus, water is retained in the body despite serum hyponatremia.[2,19,20] Damage to the autonomic conduction paths also alters kidney function. The impairment of the renal sympathetic innervation manifests itself in reduced renal blood flow, lower tubular secretion of angiotensin II and renin production by the juxtaglomerular granular cells and decreased tubular reabsorption of Na^+ and water.[4] As the renal sympathetic innervation arises from spinal cord levels T11-L3,[21] changes in renal function occur in patients with SCI at any level. Therefore, damage to the autonomic nervous system may enhance both water retention and Na^+ excretion. Consequently, a combination of dilution and depletion hyponatremia develops. Apart from small changes in the S_{Na+}–S_{Cl-} and S_{Na+}/S_{Cl-}, this may be another reason for failure to classify the condition in nearly half of the study patients.

Another often reported cause of nonosmotic stimulation of inappropriate secretion of ADH is stress associated with a severe infectious insult. This may be an acute chest infection, urinary infection or some other septic condition.[3,22] Nonosmotic stimulation of ADH secretion may also be caused by various types of drugs such as antidepressants, specifically selective serotonin reuptake inhibitors, antipsychotics or antiepileptics, which are being reported most often in this context.[23] Atsariyasing and Goldman also recorded antipsychotics-induced hyponatremia. They used the urine osmolality to distinguish it from psychosis-induced hyponatremia.[24] In both cases, the dilution form was presented. In our study, the evaluation of urine osmolality was not sufficient to distinguish between the dilution and depletion forms.

Altered osmotic regulation of ADH secretion, a reset osmostat, resulting in ADH release at a lower osmotic threshold has been discussed as a possible cause of hyponatremia in SCI patients.[2] The osmostat set point can also be shifted iatrogenically owing to excessive volume therapy, possibly inappropriately indicated to control the hemodynamic response to neurogenic shock following SCI. However, neurogenic shock develops as a result of the loss of autonomic nervous system function below the level of the lesion and needs to be treated differently from hypovolemic shock.[25]

CONCLUSION

Considering the S_{Na+}–S_{Cl-} and S_{Na+}/S_{Cl-} values in patients after SCI associated with hyponatremia may be helpful in differentiating whether the decreased S_{Na+} is consistent with the proposed dilution model (SIADH) or the NaCl depletion model (renal salt wasting). Further research is needed to evaluate the relationship between the above-mentioned values and volume changes.

Limits of the study

In this retrospective study, acid–base balance was not systematically analyzed. An assessment of the clinical outcome of therapeutic approach used could not be made and related to the assumed type of hyponatremia.

DATA ARCHIVING

There were no data to deposit.

CONFLICT OF INTEREST

The authors declare no conflict of interest.

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