How is the hyponatremia management in hospitalized patients? A cross-sectional study

Jorge Gabriel Ruiz Sánchez (gajo_Saru@hotmail.com)
Hospital Clínico San Carlos https://orcid.org/0000-0003-1305-9354
Yvonne Fernández-Cagigao
Fundación Jiménez Díaz: Hospital Universitario Fundacion Jimenez Diaz
Beatriz De León Fuentes
La Paz University Hospital: Hospital Universitario La Paz
Paola Parra-Ramírez
La Paz University Hospital: Hospital Universitario La Paz
Patrica Martín Rojas-Marcos
La Paz University Hospital: Hospital Universitario La Paz
Cristina Álvarez-Escolá
La Paz University Hospital: Hospital Universitario La Paz

Research Article

Keywords: hyponatremia, hypovolemic hyponatremia, hyponatremia approach, hyponatremia management

Posted Date: May 21st, 2021

DOI: https://doi.org/10.21203/rs.3.rs-344105/v1

Abstract

Background: Hyponatremia is the most frequent hydroelectrolytic disorder in hospitalized patients and is associated with mortality. A lack in the diagnostic and therapeutic management of the hyponatremic hospitalized patient has been reported. Hyponatremia in hospitalized patients is usually managed by specialties other than nephrology and endocrinology. The aim of our study is to describe the diagnostic and therapeutic management of hyponatremia by clinicians from services other than endocrinology and nephrology in hospitalized patients.

Methods: Cross-sectional, descriptive study of a consecutive case series of adult patients hospitalized with hyponatremia, in a tertiary hospital of Madrid, during January 1st-May 30th 2019, handled by non-endocrinologist nor nephrologist clinicians. We analyzed data regarding the details of the diagnostic assessment and the treatment established by the clinicians of the respective hospitalization service.

Results: 32 cases studied, all of them with hypovolemic hyponatremia, 50% women, 50% managed in the emergency room. Volemic classification of hyponatremia was described in only 18.8%. The height of the internal jugular venous pulse was not measured in any case. Urine studies were developed in below 70% of cases. 50% of patients had a congruent treatment with the previous described volemic status. Only one case had a complete and successful diagnostic and therapeutic approach in accordance to clinical guidelines.

Conclusion: In our series there was a lack in the diagnostic and therapeutic management of hyponatremia of hospitalized patients by non-endocrinologists and non-nephrologists.

1. Introduction

Hyponatremia , defined as serum sodium (SNa) below 135 mmol/L, is the most frequent hydro-electrolytic disorder in hospitalized patients [1,2] with a prevalence of 19.7% in these cases [3]. Hyponatremia has been associated with negative endpoints during hospitalization, such as increased mortality and a high length of stay [3,4].

Patients with hyponatremia may be hypovolemic, euvolemic or hypervolemic. The determination of the hypervolemic status is usually easy. However, differentiate between hypovolemic and euvolemic status can represent a challenge for the clinicians, leading to diagnostic errors [5–8] and therefore improper treatment [2,5,9–11]. Furthermore, the mortality associated with hyponatremia varies according to volemic status. Patients with hypervolemic hyponatremia had a higher mortality rate than those with hypovolemic or euvolemic hyponatremia [4]. Therefore, a correct volemic classification of hyponatremia is crucial for a subsequent treatment.

Many studies have found that there is a lack in the diagnostic and therapeutic management of the hyponatremia in hospitalized patients [3,12–15]. Usually, in the 74 – 89 % of the cases, hyponatremia in hospitalized patients is handled by non-endocrinologist nor nephrologist clinicians [13–15]. We don´t

know how is the management of hyponatremia by non-endocrinologist nor nephrologist clinicians of our hospital environment. Our aim is to describe the diagnostic and therapeutic approach of hyponatremia in hospitalized patients made by non-endocrinologist nor nephrologist clinicians.

2. Materials And Methods

This is a descriptive study of a consecutive case-series of patients over 18 years, with hyponatremia, hospitalized in Emergency Room (ER) or In-patient Floor (IF) of medical services other than Endocrinology and Nephrology, from La Paz Hospital of Madrid, Spain, during January 1st – May 30th 2019. These patients were initially handled by clinicians from the Emergency services or in-patient Service (IS) and later by the clinicians of the Endocrinology service (ES). We only included patients with hypovolemic Hyponatremia (HH) with a complete assessment made by the endocrinologists, admitted at least 24 hours (24h) before the endocrinologists' evaluation. We did not include other types of hyponatremia due to unavailability of initial hormone studies (e.g. thyroid-stimulating hormone and cortisol) in the Emergency room, and therefore avoid collection biases.

Hyponatremia was defined as SNa < 135 mmol/L. Hyponatremia was corrected by serum glucose according to prior descripted formula [16] if glycemia was > 150 mg/dL. Although the patients were diagnosed of HH by ES physicians, we additionally considered that hypovolemic status was present if the following conditions were fulfilled: height of the internal jugular venous pulse (HIJBP) < 1 cm at 30° inclination of the sternal angle on physical examination, as well as at least two of the following: thirst, orthostatic symptoms or signs, blood pressure ≤ 90/60 mmHg, tachycardia (≥ 90 beats per minute), urinary sodium (UNa) <30 mmol/L in case of extrarenal losses [5,9], increase of serum creatinine (SC) or urea (SU) related with SNa decreasing [17,18]]. When the HIJBP was not measured, HH was considered as present if at least three of the clinical data previously described were present. Furthermore, in order to corroborate the volemic status, we reviewed that after therapy with isotonic saline solution prescribed by the ES physicians, no worsening of the SNa was observed.

Clinical data were collected from the 24-h prior to the endocrinologists´ assessment. The only data collected from the ES assessment was the volemic classification made by the endocrinologists, which was used to compare it with that performed by the IS clinicians. Therefore, collected variables corresponded with those registered by the practitioners of the ER or IS. The collected variables were age, sex, location of the management (ER or IF), serum glucose mg/dL, serum potassium (SK) mmol/L, SC mg/dL, SU mg/dL, SNa mmol/L categorized as severe if SNa ≤ 120 mmol/L, hyponatremia temporality (acute if < 48 hours, or chronic if ≥ 48 hours), risk factors for osmotic demyelination syndrome (ODS: SNa < 105 mmol/L, malnutrition, hypokalemia, alcoholism, chronic liver disease) [2], measurements of: blood pressure (BP), heart rate (HR) and maximum HIJBP; description/registration of orthostatic symptoms/signs; blood gas assessment, urine ions measurement (sodium, potassium and chlorine), plasma and urinary osmolarity (UOsm) assessment, volemic classification registered by the clinicians, description of the target of 24-h correction of the SNa (TCNa) by the IS doctors, type of treatment prescribed by the IS doctors [water restriction, furosemide, intravenous saline therapy, serum therapy

tonicity: hypotonic, isotonic - solutions containing from 131 to 154 mmol/L of sodium [19,20] -or hypertonic; use of hypertonic saline 3% (HSS) if NaS ≤ 120 mmol/L or severe hyponatremia defined by symptoms], congruence between the volemic classification and the treatment indicated by the IS clinicians (it was considered congruent treatment if: hypovolemic classification was treated with isotonic saline therapy and/or other types of volemia replacement; hypervolemia was treated with diuretics and/or water restriction; and euvolemia was treated with water restriction, furosemide or tolvaptan, but isotonic saline fluid-therapy was not prescribed) and time elapsed from the start of treatment indicated by IS physicians to the first control of SNa (< 6 hours, 6 - 12 hours,> 24h). No data were collected regarding the measurement of serum cortisol or thyrotropin because both of them are not frequently analyzed in the emergency laboratory of the hospital.

The descriptive analyses of the quantitative variables were performed with measures of central tendency according to whether they were parametric (mean and standard deviation) or non-parametric (median and interquartile range). The comparative analysis was performed with the Chi square test and Fisher's exact test for the categorical variables and with the Mann – Whitney, Kruskal – Wallis, T-Student or ANOVA tests for the quantitative variables. For inferential analysis, statistical significance was considered if $p < 0.05$ in the two-tailed test, and 95% confidence intervals were created if necessary. The statistical package SPSS version 25 (IBM Corp., Armonk, N.Y., USA) was used for the analyses.

The study was performed according the ethical principles of the Declaration of Helsinki and Good Clinical Practice Guidelines, Spanish regulation and local Research committee. Data registry of patients and practitioners was anonymized. Written informed consent was waived because of the retrospective design of the study.

3. Results

Thirty-two clinical records of patients with confirmed HH with a complete study were included in final analysis. Median age of patients was 72 years [61 - 83], 16 (50%) were female and 5 (15.6%) patients presented SNa ≤ 120 mmol/L. Anyone did have a serum glucose ≥ 150 mg/dL in the blood samples, so, SNa did not need to be corrected by glycemia. The characteristics of all cases 24h before of the ES assessment are described in Table 1.

Figure 1 shows the frequency of each clinical or laboratorial test developed by IS physicians as a diagnosis support. During the management of the IS physicians, the volemic classification of the hyponatremia was described only in 6/32 patients (18.8%), 2 of those patients were assessed at IF and coincided with that described by ES clinicians. One (25%) of the other 4 remaining cases had a volemic classification equal to that of ES and were evaluated in the ER.

Details about therapeutic approach made by IS clinicians are described in Table 1. Thirty of the 32 cases (93.8%) received isotonic saline fluid therapy. Congruence between type of volemic status and treatment indicated by IS physicians was present in 3/6 (50%) cases in which volemia was described. The IS clinicians prescribed HSS in 2/5 (40%) cases with NaS ≤ 120 mmol/L or severe symptoms.

The TCNa was described in 5/32 (15.6%) cases. The first SNa measurement after the start of treatment in patients with descripted TCNa was developed at < 6 hours only in 1 of the 5 case, between 6 – 12 hours in 3 cases, and ≥ 24 hours in the remaining case.

The diagnostic and therapeutic approach made by the IS clinicians according to the place of patient´s stay is described in the Table 2. In ER, compared with IF, a higher rate of blood gas assessments (93.8% vs 50%, *p=0.018*) as well as of SNa measurements before 6 hours post treatment (50% vs 6.3%, *p=0.018*) were observed.

Only in 1 of the 32 cases (3%), all diagnosis-support parameters were developed by IS physicians and volemic status was described, which was congruent with the treatment indicated by them, and coincided with that made by ES practitioners. Furthermore, the first control of SNa was developed at < 6 hours since the treatment was initiated.

4. Discussion

Our results indicate the existence of a deficiency in the diagnostic and therapeutic approach of the hyponatremia made by non-endocrinologist nor nephrologist clinicians in hospitalized patients.

The lack of description/identification of the volemic state made by IS physicians, observed in almost 82% of our cases, is very striking. We observed that the measurement of HIJBP in the physical examination was apparently not carried out by any IS clinician, which guides us to hypothesize the existence of a scarce knowledge of this exploration or an underestimation of its usefulness. Our results coincide with others described in previous retrospective [12–14] and prospective [3,15] studies, which have shown the poor performance of a complete and accurate diagnostic study of hyponatraemia, including volemia assessment.

Because the clinical classification of volemia could be difficult [5–8], other test have been recommend to assay for this purpose. A value of UNa < 30 mmol/L has been found as a useful indicator of hypovolemia [5,7,21,22]; nevertheless, disorders that cause urinary sodium losses or states of urinary dilution will hinder its interpretation for this purpose. Some authors have proposed using UOsm as an unique criterion for classifying hyponatremia and to direct the initial treatment [23,24]. Our results regarding the UOsm and UNa measurements are similar to those observed in the Hyponatremia Registry, where only in 68% and 63% of the patients diagnosed with syndrome of inappropriate secretion of antidiuretic hormone, the UOsm and UNa measurements were performed , respectively [15]. These results suggest that many clinicians might not know the recommendations about using urine tests for initial diagnostic or therapeutic support in the hyponatremia management.

Although the main clinical guidelines not include a routine acid-base status assessment in their recommendations [1,2], there is a close pathophysiological relationship between acid-base status, sodium homeostasis and effective circulating volume (ECV) [25]. Sahay M. and Sahay R. included the blood gas assessment within the diagnostic algorithm of hyponatremia [24]. Low ECV, when the integrity of the

renin-angiotensin-aldosterone system is maintained, is usually accompanied by metabolic alkalosis as a consequence of hyperaldosteronism secondary to low ECV [26]. On the other hand, when hyponatremia appears in context of hypoaldosteronism, hyperchloremic metabolic acidosis could be observed [27,28]. Therefore, the study of the acid-base status would help in the diagnosis of hyponatremia. In our study, patients handled in ER had a higher rate of blood gas measurements than those in IF. It is probably explained by the accessibility for performing this test in the ER of the hospital. Another explanation might be that this test is included in a “basic” profile assessment of the patients admitted to the ER. Anyway, this is a good practice that should be enhanced.

In the current study there was an incongruity between the volemic classification - when it was developed - and the treatment prescribed by the IS physicians. Furthermore, the most patients received treatment with isotonic saline infusion despite the lack of volemic status description in the clinical records. Although that treatment was correct, as all patients of this series were hypovolemic, we have to hypothesize that it was a coincidence. Additionally, we observed that IS clinicians treated severe hyponatremia with HSS, according to the guidelines [29], in only 40% of patients. Whether clinicians truly know the importance of the relation between a correct initial classification of the hyponatremia and its treatment, remains to elucidate.

Monitoring of SNa every 4-8 hours during the first 48 hours after starting treatment is a recommended approach to avoid overcorrection of SNa [1,2,30,31]. In the most patients of our series, the first SNa control was carried out after 6 hours post-treatment. But we observed a significantly higher percentage of SNa controls before 6 hours in patients placed in the ER (Table 2). It might be explained by the perennial presence of physicians in the ER, a rapid access to a blood-gas machine, or the use of HSS in 2 of the 8 patients of this group. Both human and logistical resources as well as the use of HSS could be factors associated with the frequency and intervals of the post-treatment SNa controls.

Our results suggest us that the training and knowledge about Hyponatremia approach should probably be improved in non-endocrinologist nor nephrologist clinicians. It has been found that when hyponatremia in hospitalized patients is managed from the beginning by well-trained practitioners, hospitalization time decrease [32] and approximately 80% of patients could receive a complete diagnostic assessment, as compared to 5% when the physicians are not well-trained [14]. Furthermore, a complete and correct diagnostic of hyponatremia from the beginning allows a better treatment [15], with up to 8-fold more probabilities of achieving a NaS > 130 mmol/L at discharge [32], as well as a reduction of the hospital mortality [33]. Therefore, we believe that, in level-3 hospitals, there should be multidisciplinary teams for the management of hyponatremia, and thus guarantee the best care for these patients in all services.

Our study has some limitations. Its retrospective design leads to the existence of biases despite our attempt to avoid them. The results come from a small number of cases and from a unique hospital center. The medical specialty and the degree of training/experience of the IS physicians were not collected, which could be a factor that interfered with the results.

The main strength of our research is the selectivity and correct volemic classification of patients, based on various known scientific parameters [2,11,17,18] and a concise methodology. The exposed results are relevant and show the probable reality about hyponatremia approach by the physicians of a high-resolution hospital. Nevertheless, this situation is likely to occur in other hospitals in our region. Therefore, more studies, preferably multicenter and prospective, should be carried out.

In conclusion, we evidenced the existence of deficiencies in the management of patients with hyponatremia by non-endocrinologists nor nephrologists in our series, a situation similarly observed in other studies. The likelihood of this happening in other hospitals could be high. Therefore, we believe it is prudent to reinforce training in matters of hyponatremia already from medical schools, as well as during formation in medical specialties.

Declarations

Funding:

JGRS has a contract as a researcher with the Foundation for Biomedical Research at the Hospital Clínico San Carlos [reference: INV-15-2019]. But this research did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit sectors, for its development.

Conflicts of Interest:

The authors declare that they have no conflicts of interest.

Available of data and material:

No applicable

Ethics approval and consent to participate:

The study was performed according the ethical principles of the Declaration of Helsinki and Good Clinical Practice Guidelines and local committee. Data registry of patients was anonymized. Written informed consent was waived because of the retrospective design of the study.

Authors' contributions:

Conceptualization: JGRS; Methodology: JGRS; Formal analysis: JGRS Resources: JGRS, YFG, BLF Writing original draft: JGRS; Writing-review and editing: JGRS PPR, PMRM and CAE

Supervision: PPR, PMRM, CAE. All authors reviewed and corrected the draft and approved the final manuscript

Acknowledgment:

Authors thank the colleagues of the Endocrinology Department the La Paz University Hospital and the San Carlos Clinical Hospital, who constantly offered their support and scientific advice for the development of the current study. A special thanks to Dr. Isabelle Runkle for the scientific teachings.

Bibliography

1. G. Spasovski, R. Vanholder, B. Allolio, D. Annane, S. Ball, D. Bichet, G. Decaux, W. Fenske, E. J. Hoorn, C. Ichai, M. Joannidis, A. Soupart, R. Zietse, M. Haller, S. van der Veer, W. Van Biesen, and E. Nagler, Nephrol. Dial. Transplant. **29**, i1 (2014).
2. J. G. Verbalis, S. R. Goldsmith, A. Greenberg, C. Korzelius, R. W. Schrier, R. H. Sterns, and C. J. Thompson, Am. J. Med. **126**, (2013).
3. S. S. Waikar, D. B. Mount, and G. C. Curhan, Am. J. Med. **122**, 857 (2009).
4. M. Cuesta, A. Garrahy, D. Slattery, S. Gupta, A. M. Hannon, K. McGurren, M. Sherlock, W. Tormey, and C. J. Thompson, Clin. Endocrinol. (Oxf). **87**, 400 (2017).
5. H.-M. Chung, R. Kluge, R. W. Schrier, and R. J. Anderson, Am. J. Med. **83**, 905 (1987).
6. S. McGee, W. B. Abernethy, and D. L. Simel, JAMA **281**, 1022 (1999).
7. W. Musch, J. Thimpont, D. Vandervelde, I. Verhaeverbeke, T. Berghmans, and G. Decaux, Am. J. Med. **99**, 348 (1995).
8. E. J. Hoorn, M. L. Halperin, and R. Zietse, QJM An Int. J. Med. **98**, 529 (2005).
9. E. J. Hoorn and R. Zietse, J. Am. Soc. Nephrol. **28**, 1340 (2017).
10. K. A. Gharaibeh, J. M. Brewer, M. Agarwal, and T. Fülöp, Am. J. Med. Sci. **349**, 170 (2015).
11. P. Tzoulis and I. Runkle-De la Vega, in *Front. Horm. Res. Disord. Fluid Electrolyte Metab. Focus Hyponatremia*, edited by A. Peri, C. J. Thompson, and J. G. Verbalis (KARGER, 2019), pp. 190–199.
12. T. Krummel, E. Prinz, M.-A. Metten, C. Borni-Duval, D. Bazin-Kara, E. Charlin, J.-M. Lessinger, and T. Hannedouche, BMC Nephrol. **17**, 159 (2016).
13. P. Tzoulis, R. Evans, A. Falinska, M. Barnard, T. Tan, E. Woolman, R. Leyland, N. Martin, R. Edwards, R. Scott, K. Gurazada, M. Parsons, D. Nair, B. Khoo, and P. M. Bouloux, Postgrad. Med. J. **90**, 694 (2014).
14. P. Tzoulis and P. M. Bouloux, Clin. Med. (Northfield. Il). **15**, 20 (2015).
15. A. Greenberg, J. G. Verbalis, A. N. Amin, V. R. Burst, J. A. Chiodo III, J. R. Chiong, J. F. Dasta, K. E. Friend, P. J. Hauptman, A. Peri, and S. H. Sigal, Kidney Int. **88**, 167 (2015).
16. T. A. Hillier, R. D. Abbott, and E. J. Barrett, Am. J. Med. **106**, 399 (1999).
17. I. Runkle, E. Gomez-Hoyos, M. Cuesta-Hernández, J. Chafer-Vilaplana, and P. de Miguel, Rev. Clin. Gerontol. **25**, 31 (2015).
18. J. G. Ruiz Sanchez, J. Cárdenas Salas, C. López Nevado, A. Durán Rodriguez-Hervada, M. Cuesta Hernández, A. Calle Pascual, and I. Runkle de La vega, Endocrinol Diabetes Nutr **66**, 7 (2019).
19. NICE guidelines, *Intravenous Fluid Therapy in Children and Young People in Hospital* (NICE: National Institute for Health and Care Excellence, London, 2015).

20. J. Green and J. Lillie, Arch. Dis. Child. - Educ. Pract. Ed. **102**, 327 (2017).
21. W. Musch and G. Decaux, Int. Urol. Nephrol. **32**, 475 (2001).
22. W. Fenske, S. Störk, A.-C. Koschker, A. Blechschmidt, D. Lorenz, S. Wortmann, and B. Allolio, J. Clin. Endocrinol. Metab. **93**, 2991 (2008).
23. W. Fenske, S. K. G. Maier, A. Blechschmidt, B. Allolio, and S. Störk, Am. J. Med. **123**, 652 (2010).
24. M. Sahay and R. Sahay, Indian J. Endocrinol. Metab. **18**, 760 (2014).
25. R. G. Narins, E. R. Jones, M. C. Stom, M. R. Rudnick, and C. P. Bastl, Am. J. Med. **72**, 496 (1982).
26. V. Gillion, M. Jadoul, O. Devuyst, and J.-M. Pochet, Acta Clin. Belg. **74**, 34 (2019).
27. R. A. DeFronzo, Kidney Int. **17**, 118 (1980).
28. J. Yaxley and C. Pirrone, Ochsner J. **16**, 525 (2016).
29. I. Runkle, C. Villabona, A. Navarro, A. Pose, F. Formiga, A. Tejedor, and E. Poch, Nefrologia **34**, 439 (2014).
30. N. A. Pirzada and I. I. Ali, Mayo Clin. Proc. **76**, 559 (2001).
31. A. Peri, C. J. Thompson, and J. G. Verbalis, editors , *Disorders of Fluid and Electrolyte Metabolism* (S. Karger AG, 2019).
32. P. Tzoulis, H. Carr, E. Bagkeris, and P. M. Bouloux, Endocrine **55**, 539 (2017).
33. M. Whyte, C. Down, J. Miell, and M. Crook, Int. J. Clin. Pract. **63**, 1451 (2009).

Tables

Table 1. Basal Characteristics of the studied patients and therapeutic approach performed by the clinicians before endocrinologists´ assessment	
N = 32	
Age, years	72 [61 - 83]
Sex M/F, n (%)	16 (50) /16 (50)
Serum Biochemical parameters	
SC mg/dL	1.2 ± 0.9
SU mg/dL	43 [19.4 – 66.6]
SK mmol/L	4 ± 1.9
SNa mmol/L	126.3 ± 4.9
Hypovolemic hyponatremia, n (%)	**100 (100)**
Causes	
Gastrointestinal losses, n (%)	10 (31)
Urinary losses, n (%)	14 (44)
Haemorrhage, n (%)	1 (3)
Characteristics of hyponatremia	
Acute Hyponatremia, n (%)	7 (21.9)
Chronic Hyponatremia, n (%)	25 (78.1)
SNa < 120 mmol/L, n (%)	5 (15.6)
Risk factors for ODS, n (%)	25 (78.1)
Location of the patients´s management	
In-patient floor, n (%)	16 (50)
Emergency room, n (%)	16 (50)
Treatment	
Water restriction, n (%)	3 (9.4)
Furosemide, n (%)	3 (9.4)
Fluid-therapy, n (%)	32 (100)
Hypotonic, n (%)	0
Isotonic, n (%)	30 (93.8)
Hypertonic, n (%)	2 (6.2)

Control and follow-up		
Description of TCNa, n (%)	5 (15.6)	
Time to first SNa control		
< 6h, n (%)	9 (28.1)	
6 - 12h, n (%)	10 (31.3)	
> 24h, n (%)	13 (40.6)	

Age and SU are described as median and [interquartile range]. SC, SK and SNa are described as mean and +/- standard deviation.
SC: serum creatinine, SU: serum urea, SK: serum potassium, SNa, serum sodium, ODS: osmotic demyelination syndrome, TCNa: target of correction of the SNa, SNa: serum sodium

Table 2. Hyponatremia approach according place of management			
	In-patient floor (N=16)	**Emergency room (N= 16)**	***P****
Diagnostic support assessments			
HIJP exploration, n (%)	0	0	-
Orthostatism inquiry, n (%)	10 (62.5)	12 (75)	0.7
Blood Gases, n (%)	8 (50)	15 (93.8)	0.018 *
Urine ions, n (%)	11 (68.8)	11 (68.8)	0.7
Urine Osmolarity, n (%)	9 (56.3)	10 (62.5)	1
Volemia description, n (%)	2 (12.5)	4 (25)	0.6
Details of the treatment			
Congruence with descripted volemia, n (%)	2/2 (100)	1/4 (25)	0.38
Water restriction, n (%)	1 (6.3)	2 (12.5)	1
Furosemide, n (%)	2 (12.5)	1 (6.3)	1
Fluid therapy, n (%)	16 (100)	16 (100)	-
Isotonic, n (%)	16 (100)	14 (87.5)	-
Hypertonic, n (%)	0	2 (12.5)	-
SNa $\leq$ 120 mmo/L treated with HSS, n (%)	0	2/5 (40)	-
Control and follow-up			
Description of TCNa, n (%)	0	5 (31.3)	0.051
Time to firs SNa control			
< 6h, n (%)	1 (6.3)	8 (50)	0.018*
6 - 12h, n (%)	6 (37.5)	4 (25)	0.7
> 24h, n (%)	9 (56.3)	4 (25)	0.14
HIJP: height of the internal jugular pulse, TCNa: target of correction of the serum sodium * p < 0.05			

Figures

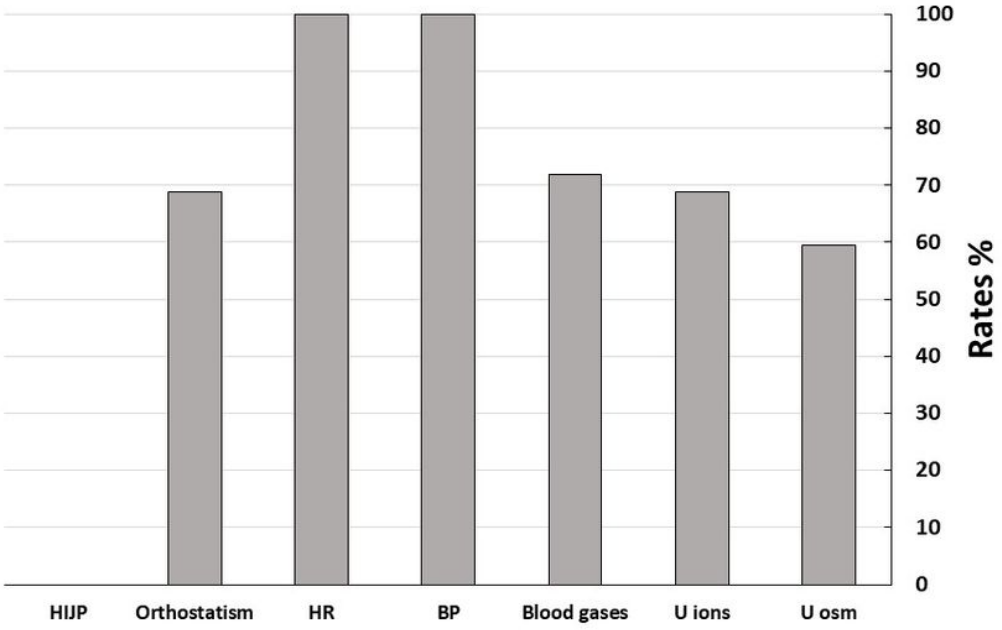

Figure 1

The frequency of each clinical or laboratorial test developed by IS physicians as a diagnosis support