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Dehydration status is associated with more severe hemispatial neglect after stroke

MN Bahouth, Z Bahrainwala, AE Hillis, and RF Gottesman

Department of Neurology, Johns Hopkins University School of Medicine

Abstract

Background—Many stroke patients are clinically dehydrated at the time of hospital presentation, which could lead to an increase in blood viscosity and alteration in cerebral perfusion. Impaired cerebral perfusion can cause hemispheric dysfunction, which can be rapidly quantified with bedside tests of hemispatial neglect. We hypothesized that hospitalized patients with laboratory markers consistent with dehydration or a volume contracted state (VCS) would have more severe cerebral dysfunction defined by greater degree of neglect.

Methods—Subjects were a consecutive series of right-handed patients with acute right hemispheric stroke admitted within the Johns Hopkins Health System. All participants had clinical syndrome and MRI consistent with acute infarction. The primary definition of a VCS was a urea/creatinine ratio greater than 15; with secondary definition including urine specific gravity over 1.010. Acute infarct volume was measured on magnetic resonance imaging. Neglect was evaluated using a standardized battery of bedside tests.

Results—Of 201 patients meeting inclusion criteria, 131/201 (65%) had elevated BUN/creatinine ratios at admission. Approximately 61% (122/201) had some degree of neglect. Elevated BUN/Creatinine ratio was associated with an increased odds of severe neglect in unadjusted models (OR 4.1, 95% CI 1.2, 14.4), with loss of significance in adjusted models (OR 4.43, (95% CI 0.99, 19.8) after adjustment for age, infarct volume, sex, and NIHSS score.

Conclusions—Our data suggest that patients who are in a VCS at the time of stroke may have more frequent and severe neglect, with attenuation of results after adjustment for factors related to stroke size and age. If proven clinically relevant, a formalized rehydration strategy based on

Correspondence: Rebecca Gottesman, MD, PhD, Department of Neurology; Johns Hopkins University School of Medicine; Phipps 446; Baltimore, Maryland 21287, rgottesm@jhmi.edu, Phone: 410-614-2381 (o); 410-614-9807 (f).

Author contributions:

1. Dr. Bahouth reports no disclosures; contributions include conceptualizing the study, designing the study; interpreting the data, and drafting the manuscript
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objective lab markers may represent an opportunity for improvement in outcome with low cost, broadly available treatment for acute stroke patients.

Keywords

Acute stroke; dehydration; hemispatial neglect; neglect; stroke severity

Background

Autoregulation is impaired at the time of acute stroke, thus leaving the brain vulnerable to fluctuations in blood pressure and blood fluidity.¹ Although prior acute stroke guidelines had recommended against the use of “excessive intravascular fluids”², recent updates in these guidelines suggest consideration of rapid repletion of intravascular volume for patients who are hypovolemic at presentation (though caution is advised for patients prone to volume overload).³ Mild hypovolemia is difficult to diagnosis; diagnosis is often subjective. Observations in the clinical arena suggest variability in practice in the area of volume resuscitation of the acute stroke patient. We hypothesize that hydration status is a potentially modifiable risk factor for poor stroke outcome. In this phase of study, we sought to understand the relationship between hydration status and stroke severity more closely.

We use the concept of hydration status as a general, oversimplified term to include volume contracted or hypernatremic states, because dehydration is a clinical diagnosis requiring physical examination and the two states can commonly overlap. This study evaluates the association between presence of dehydration and severity of cerebral dysfunction at the time of stroke onset as measured by degree of hemispatial neglect, among individuals hospitalized with acute ischemic stroke at our two-hospital hospital system. There are many indicators of severity of cerebral dysfunction in stroke (e.g. National Institutes of Health Stroke Scale (NIHSS), Magnetic resonance imaging (MRI) measured infarct size). Hemispatial neglect is used here as a measure of cerebral function because (1) it can be measured quickly with high degree of inter-rater reliability and test-retest reliability⁴; (2) we have neglect data on a large number of acute stroke patients who also had acute MRI; (3) scores on neglect tests correlate better with measures of tissue dysfunction than do scores on the NIH Stroke Scale.⁵ Finally, patients with neglect as a part of their stroke syndrome have a more challenging time adhering to the rigors of rehabilitation thus making this outcome clinically relevant⁶.

METHODS

Study population

We reviewed a consecutive series of non-demented, English-speaking, right-handed patients admitted to our two hospital system with right hemispheric stroke. Patients were included if they presented with ischemic stroke within 48 hours of symptom onset, were able to complete at least a portion of bedside neglect testing, and were able to undergo MRI. Patients were excluded if MRI did not demonstrate a stroke by diffusion weighted imaging (DWI); diffusion lesion volume 0.

Laboratory markers of dehydration

For the purpose of defining markers of a relative dehydrated state for our study population, we selected serum BUN/creatinine (blood urea nitrogen/creatinine) ratio and urine specific gravity, each evaluated on admission to the hospital. There is no gold standard diagnostic test for measuring hydration status, but these markers have been suggested in the critical care and stroke literature as potential markers of volume contraction.^{7,8} Additionally, these measures are standardized lab measures that are readily available and routinely obtained in the hospital setting. For this analysis we defined BUN/creatinine ratio > 15 and urine specific gravity > 1.010 as suggestive of dehydrated state, and evaluated each of these variables separately as well as part of a composite “hyperviscosity” variable. Blood work for BUN/creatinine ratio and urine samples for specific gravity analysis were collected only if part of routine, standard of care clinical practice at the time of hospital presentation. In general, more individuals were missing urine specific gravity measurement than BUN/creatinine levels.

Hemispatial Neglect testing

Neglect was evaluated using a standardized battery of bedside tests conducted by our stroke study group, blinded to the patients’ volume status, within the first 48 hours from stroke symptom onset including: *line cancellation*, *line bisection*, and *copying a scene*.^{9, 10} For the purpose of defining the presence of neglect and severe neglect, horizontal line bisection, line cancellation, and copy scene testing were used, with error rates as the recorded scores (and higher scores indicating worse performance).⁹ These tests have been used previously in the stroke population, have simple instructions, and can be performed even without eyeglasses for those with low vision. Agreement in scoring line cancellation and copy scene tests from 100 patients was 98% and 96%, respectively.¹¹ Line bisection scores between 2 raters did not vary by more than 0.2 inch (2% of 10 inch line). Normal control data were obtained from patients without stroke admitted with transient ischemic attack or preoperatively before cardiac bypass surgery to create all z scores. As used in previous publications, any neglect was defined as a Z-score of at least 2 on any one of the three tests.^{11,12} Severe neglect was defined as a Z-score of at least 2 on at least two of the three tests.

Imaging

Infarct volume was measured on MRI, using DWI and corresponding ADC maps. Volumetric analysis was performed manually, without knowledge of the patient’s clinical status, with the assistance of ImageJ software. Volumes of infarct or dense ischemia (bright on DWI, dark on corresponding ADC maps) were traced on individual slices and calculated based on the slice thickness, and were recorded in cubic centimeters. If subjects had multiple areas of infarct, each of these lesions was measured and volumes were summed for each patient, leading to a single total acute infarct volume per patient. Volumes were reported in cubic centimeters. A subset of individuals (n=146) had perfusion-weighted imaging (PWI) as well. Volume of hypoperfusion was similarly traced per slice of the time-to-peak sequence, with summed volumes for a total volume of hypoperfusion. For each individual, for a sensitivity analysis, a “larger” volume was calculated: either the DWI or PWI volume

of abnormality, whichever was larger, for those individuals with both measured, or the DWI volume for those without a PWI image.

Statistical analysis

Statistical analysis was conducted using Stata version 12.0 for Macintosh. Fisher's exact tests and t-tests were used for univariate analyses. Any neglect (Z-score 2 on 1 of 3 neglect tests) and severe neglect (Z-score 2 on 2 of 3 neglect tests) were each the dependent variable in separate multivariable logistic regressions evaluating the role of admission BUN/creatinine ratio and, in a smaller subset, urine specific gravity (SG), with adjustment for age, sex, DWI volume, and NIHSS score. A sensitivity analysis included adjustment for larger volume of ischemia (DWI or PWI if both available, or DWI if only DWI available) instead of DWI volume on all participants. NIHSS score was also evaluated as a dependent variable in separate linear regressions evaluating the impact of dehydration markers (as defined above) on NIHSS score on admission. For the primary analyses, we used the composite variable "hyperviscosity" (elevated BUN/creatinine > 15 *and* elevated urine specific gravity > 1.010) as a predictor of neglect presence and neglect severity. In secondary analyses, we also analyzed data separately for patients with BUN/creatinine ratios > 15 and BUN/creatinine ratios > 20.

RESULTS

We identified 369 individuals who met inclusion criteria (figure 1). Of those, 72% (266) had MRI with DWI restriction, consistent with a diagnosis of stroke. Among these, 212 (80%) completed sufficient bedside testing to score neglect. BUN/creatinine was available for 201/212 (95%). Urine specific gravity was measured in 102/212 (48%).

Patients in our study group were mean 64 ± 15 years of age, 47% female, and with a mean NIHSS score 5.7 ± 5.1 . As expected, patients with severe neglect had higher NIHSS score and DWI lesion size than patients without severe neglect (Table 1a). Demographics by hydration status were also similar (Table 1b).

A majority of our acute stroke patients were in a volume contracted state according to surrogate lab markers with elevated BUN/creatinine ratio in 65% and elevated urine specific gravity in 83%. Using our composite surrogate panel of dehydration (both BUN/creatinine ratio and urine specific gravity elevated), 57% of patients were in a volume contracted state at the time of admission. No patients had abnormalities in serum sodium levels suggestive of dehydration.

The majority, 122 (61%), of patients had "any neglect" and of those, 23 (25%) were found to have severe neglect. Average BUN for patients with severe neglect was 23 (compared with 17 in patients without severe neglect; $p = 0.0003$). BUN/creatinine ratio was 21 for those with severe neglect, on average, as compared with 17 in patients without severe neglect ($p = 0.002$) (Table 2), with an unadjusted odds ratio of 4.1 for severe neglect in individuals with an elevated BUN/creatinine ratio compared to those without an elevated ratio (95% CI 1.2,14.4).

Multivariable model

In a multivariable model, after adjustment for age, sex, NIHSS score and infarct volume, having an elevated BUN/creatinine ratio was non-significantly associated with 4.43-times higher odds of having severe neglect (95% CI 0.99,19.81) (see Table 3). Results were similar with adjustment for the larger of either DWI or PWI, instead of DWI, for all patients. Independent of creatinine, BUN level was associated with severe neglect (adjusted OR 1.12, 95% CI 1.02, 1.22, per point higher BUN). Additionally, results were consistent when analyzing data using BUN/creatinine ratio > 20 and urine specific gravity > 1.010 (adjusted OR 2.93, 95% CI 0.95, 9.02).

Analyses using urine specific gravity

All 14 individuals with severe neglect and urine measurement had an elevated urine SG ($p=0.06$). Of the 85 patients with elevated urine specific gravity, 58 (68%) also had elevated BUN/creatinine ratios. This combined variable for dehydration, defined as BUN/creatinine >15 and urine specific gravity >1.010, was significant in unadjusted models (Table 3), associated with a 5.37-fold increased odds of severe neglect (95% CI, 1.12, 25.72), and though non-significant, demonstrated a trend toward significance as a predictor for severe neglect in adjusted models (adjusted odds ratio 6.59, 95% CI 0.89, 48.95).

There was no substantive difference in the results when only the subset of patients with PWI volume data was analyzed. Similarly, no difference in results was found when recreating the definition of our composite hyperviscosity variable using BUN/creatinine ratio > 20 and urine specific gravity > 1.010.

Markers of dehydration in association with NIHSS score

An elevated urine specific gravity was significantly associated with the admission NIHSS score, independent of age, sex, and infarct volume. Having a urine specific gravity over 1.010 was associated with, on average, an NIHSS that was 2.62 points higher (adjusted; 95% CI 0.32, 4.92) than in individuals with a urine specific gravity below 1.010. An elevated BUN/creatinine ratio was not significantly associated with the NIHSS score (adjusted $p=0.41$), but when combined with elevated urine specific gravity neared significance in its association with admission NIHSS score (unadjusted Beta=1.63, 95% CI -0.27, 3.52).

DISCUSSION

Our data suggest that a majority of patients are in a volume contracted state at the time of their hospitalization for stroke, and patients who are in a volume contracted state at the time of stroke presentation have more frequent hemispatial neglect, although results did not persist with adjustment for age, sex, NIHSS score, and size of the stroke. Analysis of hemispatial neglect in stroke patients is intended as a proof-of-concept analysis; we do not expect that the associations tested are specific to hemispatial neglect, but rather that this represents one example of a cognitive deficit (a marker of hemispheric dysfunction) that might be more frequent or more severe in the setting of dehydration. Neglect was chosen as a single cortical marker that is objectively tested at the bedside. This is an important first step in determining the contribution of baseline hydration status to stroke severity and early

stroke recovery. This finding extends previously reported work summarizing a physiologic basis based on concepts in viscosity and in retrospective studies, by adding infarct volume and severity of cerebral dysfunction into multivariable models as possible confounders.^{12–19} It also further extends the suggestion that relative dehydration may be a contributing factor to dysfunction in ischemic stroke.²⁰

We proposed the combination of BUN/creatinine ratio with urine specific gravity as a surrogate marker of dehydration for a number of reasons: 1) defining a standardized algorithm of basic laboratory markers suggestive of clinical hydration will allow objective, numeric measurements in order to extrapolate hydration status and trend in future clinical trials; 2) these tests are readily available in most hospital settings and are globally available; 3) these tests are rapidly performed, crucial for assessment of patients in a time sensitive treatment period; and 4) these studies are inexpensive. Prior studies of both healthy subjects and critically ill patients used similar markers.^{21, 22}

In an ideal setting we would have a mechanism for rapid, non-invasive, objective measurement of volume status. Our bedside clinical assessments of hydration status using observations like skin turgor and jugular venous pressure in the acute period are crude at best. Therefore, combining well-validated surrogate lab markers may be one solution to quantitatively demonstrating states of dehydration at the time of acute stroke admission for the purpose of future testing and measuring the impact of selected interventions.

Our selected population of patients allows for examination of a relatively homogenous population of stroke patients, all with non-dominant hemisphere ischemic strokes, many of whom had neglect. We believe that our results are generalizable to a broader stroke population, because autoregulation may be impaired in a broad range of ischemic stroke subtypes. Our study is strengthened by the presence of MRI data on infarct size, to determine if the relationship between dehydration and cerebral dysfunction is independent of stroke volume. The timing of MRI was variable after admission which may be one limitation.

We acknowledge several additional limitations to our study common in retrospective data evaluation. First, multiple variables important to acute stroke care are not available to explore as possible confounders, most notably blood pressure, and the presence of paroxysmal atrial fibrillation. Therefore, we cannot say with certainty that there is a direct relationship between dehydration and neglect (e.g. dehydration might be a risk factor for atrial fibrillation, which increases the risk for a large artery stroke), and in general there is a risk of unmeasured confounding in the observed association. Additionally, several conditions not measured in this study could increase the BUN out of the setting of dehydration (e.g. GI bleed); however, no patients required transfusion at the time of admission, and therefore any GI bleeding if present was subclinical. We will need to collect these variables in future phases of study. Next, data were accrued for patients up to 48 hours after stroke onset; thus, we cannot say that patients were truly dehydrated at the time of their stroke versus becoming more volume contracted over time secondary to stroke related impairments. If a large stroke leads a patient to have decreased oral intake, either due to impaired mobility (and being immobilized for an extended period of time) or due to

dysphagia, the observed results might simply be due to dehydration being a marker of a large stroke, or, perhaps, being a mediator of the association between large stroke and poor outcome. Shorter time windows for enrollment from the last normal baseline will be helpful to clarify this issue in future studies. Another major potential issue is limited power; the attenuation of the association after adjustment for markers of stroke volume may be an indication of some confounding, but the lack of a statistically significant association is likely due to inadequate power. Finally, this study does not address whether or not dehydration affected eventual clinical outcome. Future studies should (1) determine whether hydration status is associated with other functional measures at onset; (2) determine whether hydration status at onset predicts functional outcome; (3) identify interventions that optimize hydration status without adverse side effects. Data of this type are needed before we can begin to determine a true association between neglect or other markers of cerebral dysfunction in stroke, with future goals of recommending a management strategy at the time of acute stroke.

Conclusions

Patients who are in a volume contracted state at the time of presentation with stroke have more severe hemispatial neglect. The next phase of study will compare the clinical status between patients arriving to the hospital for ischemic stroke who are volume contracted versus those who are normovolemic. Further study will also be needed to evaluate our current approaches to hydrating acute stroke patients with varying degrees of cardiac dysfunction in order to prescribe more precise fluid resuscitation algorithms. Future clinical trials designed to establish concrete fluid resuscitation practices at the time of ischemic stroke are needed in order to optimize the effect of this potential adjunctive treatment.

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Figure 1.
Enrollment scheme

Table 1a

Demographics by presence of neglect

	All Patients (n=201)	Any Neglect (n=122)	No Neglect (n=79)	Severe Neglect** (n=23)	No Severe Neglect (n=147)
Age (years)	63.7 ± 14.6	67.0 ± 14.9	58.8 ± 12.5	68.8 ± 11.5	62.5 ± 14.9
Sex (% female)	92 (46%)	65 (54%)	27 (35%)	8 (35%)	61 (42%)
NIH Stroke Scale Score (mean)	5.7 ± 5.1	7.2 ± 5.5	3.6 ± 3.5	11.2 ± 6.7	4.8 ± 4.2
DWI volume (cc)	25.2 ± 42.6	35.2 ± 50.7	9.7 ± 16.7	71.6 ± 70.8	18.6 ± 33.6

* All results reflect mean ± Standard Deviation unless otherwise noted

** Severe neglect requires at least two of three tests completed so could only be assessed in 170 participants.

Table 1b

Demographics by marker of dehydration

	Increased Specific Gravity (n=85)	Increased BUN/Creatinine (n=131)	Both dehydration markers elevated (n=58)	No Dehydration (n=11)
Age (years)	65.5 ± 14.2	65.2 ± 14	67.0 ± 13.6	66.5 ± 13.9
Sex (n, % female)	39 (46%)	64 (50%)	26 (46%)	6 (60%)
NIHSS Median (25–75% interquartile range)	5 (3–10)	4 (2–9)	5 (3–9)	2.5 (1–5)
DWI volume (cc)	33.6 ± 56.6	25.9 ± 42.4	34.0 ± 56.9	14.8 ± 17.7
Hemoglobin (dL; average)	12.7 ± 2.1	12.6 ± 2.0	12.2 ± 1.9	12.9 ± 2.1
Hematocrit (%; average)	38.1 ± 5.9	37.7 ± 5.9	36.9 ± 5.4	38.8 ± 6.1

Table 2

Markers of dehydration and neglect: Univariate analysis (mean values)

	With any neglect	Without any neglect	p-value	With severe neglect	No severe neglect	p-value
BUN	18.2	17.2	0.40	23.3	17.0	0.0003
Creatinine	1.10	1.10	0.91	1.17	1.14	0.89
Urine SG	1.019	1.018	0.68	1.021	1.017	0.15
Bun/Creat Ratio	18.2	16.4	0.07	21.2	16.6	0.002

Table 3

Association dehydration and severe neglect

Unadjusted and adjusted odds ratios (with 95% confidence intervals) for the association between an elevated BUN to creatinine, elevated urine specific gravity, and combined, each with severe neglect.

	Unadjusted OR	95% CI	Adjusted OR *	95% CI	Adjusted model 2 OR **	95% CI
Elevated BUN/Creatinine Ratio	4.10	1.17–14.44; p=0.028	4.43	0.99–19.81; p=0.052	4.13	0.96–17.81; p=0.057
Combined model (elevated BUN/creatinine ratio and urine SG) ***	5.37	1.12–25.72; p=0.036	6.59	0.89–48.95; p=0.065	6.22	0.85–45.51; p=0.072

* adjusted for age, sex, NIHSS, DWI volume;

** adjusted for age, sex, NIHSS, and larger volume of dysfunctional tissue (larger of either DWI or PWI, when available);

*** elevated urine specific gravity (over 1.010) and elevated BUN to creatinine (over 15)