SYSTEMATIC REVIEW

Diabetes Insipidus after Vasopressin Withdrawal: A Scoping Review

Raquel S Pérsico[1], Marina V Viana[2], Luciana V Viana[3]

Abstract

Objectives: The objective of this study is to synthesize the current literature about the relationship between the occurrence of diabetes insipidus (DI), its diagnosis criteria, and management after withdrawal of vasopressin (VP) in critically ill.

Data sources: This scoping review followed the recommendations of Preferred Reporting Items for Systematic Review and Meta-Analyses for Scoping Review (PRISMA-ScR). The search literature was conducted in MEDLINE and EMBASE databases, until March 2022. A manual search was also conducted in order to include articles that were not identified in the initial search performed in the databases.

Study selection and data extraction: The selection of studies and extraction of data were carried out in a paired and independent manner. There was no restriction regarding the language of publication of the included manuscripts.

Data synthesis: The analysis included 17 studies (16 case reports and one retrospective cohort). All studies used VP, with a median time of drug infusion of 48 hours (IQR: 16–72) and DI incidence of 1.53%. The diagnosis of DI was based on diuresis output and concomitant hypernatremia or changes in serum sodium concentration, with median time to symptoms onset after discontinuation of VP of 5 hours (IQR: 3–10). The treatment of DI consisted mainly of fluid management and the use of desmopressin.

Conclusions: DI after VP withdrawal was present in 51 patients described in 17 studies, but diagnosis and management varied among each report. Using the available data, we propose a diagnosis suggestion and a flowchart for managing patients with DI after withdrawal of VP in the Intensive Care Unit. Multicentric collaborative research is urgently needed to obtain more quality data on this topic.

Keywords: Arginine vasopressin, Critical care, Diabetes insipidus, Hypernatremia, Intensive care units.

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Highlights

- Diabetes insipidus is a life-threatening and self-limited complication of vasopressin withdrawal that can lead to major changes in sodium and water.
- It is important to recognize this adverse effect of vasopressin to diagnose and treat it promptly.
- Treatment options include fluid management, desmopressin, and vasopressin resumption.

Introduction

Vasopressin, also called antidiuretic hormone (ADH), is a hormone released from the posterior pituitary gland that has numerous metabolic effects, such as hemodynamic and osmoregulatory responses.[1–3] Changes in the regulation of the metabolic pathways of VP can lead to DI, a water balance disorder that results in high hypotonic urine excretion, leading to plasma hyperosmolality and polydipsia.[4]

DI can be classified as central, nephrogenic, or gestational: (a) central or neurogenic DI is due to impaired synthesis, secretion or storage of VP by the hypothalamus and neurohypophysis; (b) nephrogenic DI occurs when the kidneys respond poorly to VP and is associated with alterations in receptors and aquaporins; and (c) gestational DI occurs only during pregnancy, when the placenta produces high amounts of vasopressinase.[4,5]

Its diagnosis in the intensive care unit (ICU) can be challenging, since DI shares symptoms that are common among many other conditions and thirst is impaired.[5,6] Some studies have reported the development of DI after discontinuation of VP in ICU patients.[7,8] Ferenchick et al. found the incidence of DI after cessation of VP in patients with shock was low (1.53%) but not negligible.[7]

In the early stages of shock, VP increases to supraphysiologic levels, decreasing rapidly thereafter.[1] VP plays an important role in maintaining body homeostasis by contributing to vasoconstriction and can be used to treat hypotension in both vasodilatory and hemorrhagic shock.[1,9–11] Its use has increased over time, and approximately one-fifth of patients with septic shock have received VP.[9] Early withdrawal of VP infusion in septic shock can lead to a rapid drop in VP levels, which may explain the development of DI after cessation of VP.[10,11]

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Clinical management of DI consists of replacing body water deficits and stopping losses, which can be done by administering hypotonic fluid and exogenous VP or an analog, such as D-arginine-vasopressin (desmopressin, DDAVP).[1,4] DDAVP has become the first choice of treatment to manage central DI, due to its less pronounced vasoconstriction effects and better antidiuretic results as compared to VP.[5] Managing nephrogenic DI includes treating the underlying cause, and prescription of hydrochlorothiazide, amiloride, and indomethacin being reported to reduce water diuresis.[4] If left untreated in critically ill patients, both nephrogenic and central DI can lead to life-threatening hypernatremia and hypovolemia.[4] Thus, it is paramount that physicians are aware of the risk of the development of DI after VP discontinuation, although it is unclear how to best manage this adverse effect of VP.

In summary, diagnosing DI in the critical care setting is challenging, but its early identification and correct management are crucial. The use of VP as a powerful vasoconstrictor agent in managing shock and the development of DI after its withdrawal as a side effect call for a broad understanding of the relationship between VP and DI. Thus, this scoping review aims to explore the scientific evidence available in the literature regarding the relationship between DI and VP, its diagnosis, and clinical management.

Materials and Methods

This scoping review was designed according to the Joanna Briggs Institute's manual for scoping review and described following criteria predefined by the PRISMA-ScR. The study protocol was registered in the Open Science Framework scoping review registry (https://osf.io/p76rn).

Research Question

"What is known in the literature about vasopressin use and the development of DI in intensive care unit patients?"

Eligibility Criteria

Inclusion criteria were as follows: (a) patients who developed DI after receiving continuous VP or an analog in the critical care setting; (b) diagnosis of DI defined by serum sodium, urine output, urine specific gravity, and/or urine and serum osmolality; (c) observational or experimental studies with prospective or retrospective design, such as cross-sectional, case-control, cohort, clinical trials (randomized or not), case series, and individual case reports; (d) gray literature, such as conference abstracts, theses, and expert opinions; and (e) published in any language.

Exclusion criteria were as follows: literature reviews of all categories; studies with patients presenting with other conditions that could induce DI, such as pituitary dysfunction due to tumor or surgery, presence of hyperparathyroidism, use of VP or an analogue in bolus instead of continuously; or studies with animal models. Also, we excluded studies in which patients did not have serum sodium higher than 145 mEq/L.

Literature Search and Study Selection

A literature search was conducted in two electronic databases, MEDLINE and EMBASE, until February 2020 (updated on March 8, 2022), being included manuscripts published until this date. The first author outlined the search strategy according to PubMed Medical Subject Headings (MESH) terms, being then adjusted to the specific terms of the other database. Search terms included "vasopressin," "diabetes insipidus," "polyuria," "hypernatremia," "sodium," and its corresponding derivatives. Supplementary Table 1 summarizes the final search strategies and search terms used in both databases, as well as the number of studies found.

After transferring the data collected to Abstrackr® software, the researchers conducted a first study selection by reading titles, abstracts, and keywords; then, the selected manuscripts were read in full. Manual searches were also carried out in the references of the studies selected. These two selection processes were conducted in a duplicate and independent manner (MVV and RSP), with a third reviewer (LVV) solving disagreements when necessary.

Data Extraction and Results Synthesis

Data extraction was conducted by two independent reviewers (MVV and RSP) and based on a standardized extraction form developed by the authors. After a pilot test with three articles, the extraction tool was adjusted to improve data collection. Any disagreements were resolved by a third reviewer.

The data extracted included the following: authors, year, country, study design, sample size, information on population demographics (age and gender), clinical characteristics (use of VP or an analog), development of DI (serum sodium, serum osmolality, urine output, urine osmolality, and specific gravity), and its clinical management (use of DDAVP). Diuresis was described in mL/day and mL/hour; when the mL/hour measurement was not available, the total diuresis was divided by total hours. Response to DDAVP was assessed by reduced urinary output or serum sodium, followed by classification as complete (improvement in both parameters) or partial (improvement in one parameter).

In case of unavailable data, the authors were contacted to request additional information. Graphs with clinical information regarding the development of DI (serum sodium, urine output, urine specific gravity, and VP dose) were read by Web Plot Digitizer Software v4.3 (Pacifica, California, USA). Medians and ranges were calculated by summarizing data and using R® software (version 3.5.1, The R Foundation). No critical appraisal of the studies was performed. Key findings were described in tables and in a narrative summary according to the objectives and study design.

Results

Description of the Articles and Patient Demographics

The search strategy returned 1,792 titles in the databases. After removing 29 duplicate articles and identifying four studies by manual search, a total of 1,767 papers remained, of which the authors selected 50 to be read in full (Flowchart 1). Development of DI without the use of VP and use of VP for clinical reasons unrelated to DI accounted for most exclusions. In all, the review included 17 manuscripts with a total of 51 individuals.[7,8,12–26]

Except for one retrospective cohort study that included 29 patients, all other papers were case reports.[7] Two studies reported the development of DI after interruption of VP in two children, aged 1 year and 4 months and 3 years, respectively.[12,13]

Of 51 patients, 34 (66.7%) patients were male, and the mean adult age was 52 ± 20 years. Reasons for ICU admission were sepsis (n = 12, 23.5%), followed by cardiovascular (n = 10, 20.4%), respiratory (n = 10, 20.4%), surgery (n = 9, 17.6%), and neurologic (n = 8, 16.3%) events and one patient with a right ventricular thrombus and one with a gastrointestinal diagnosis. Eleven (22.4%) individuals were classified as neurocritical, 11 (22.4%) individuals

Flowchart 1: Flowchart of the proposed treatment of diabetes insipidus after withdrawal of vasopressin

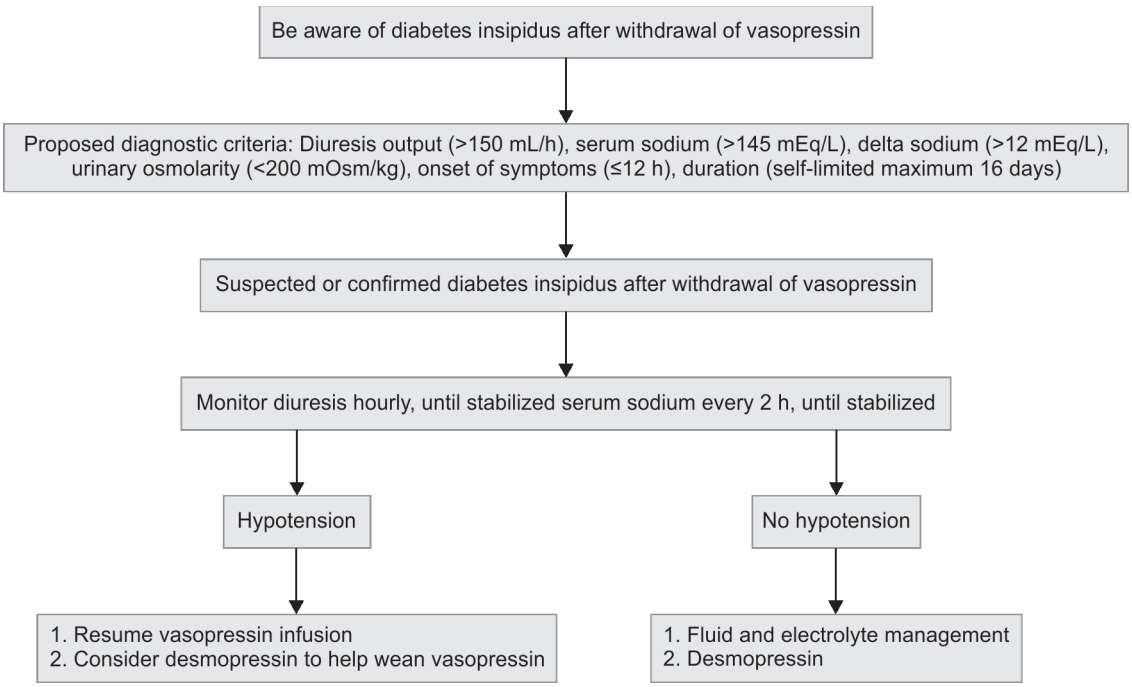


Table 1: Methodological characteristics of the manuscripts included (*n* = 17)

Authors	*Year*	*Country*	*Design*	*n*	*Drug*	*Dose*	*Reason for VP use*
Kristeller et al.	2004	USA	Case report	1	VP	0.1 U/min	Septic shock
Ramers et al.	2005	USA	Case report	1	VP	NA	Septic shock
Peskey et al.	2009	USA	Case report	1	VP	0.08 U/min	Cardiothoracic surgery
Hayes-Bradley et al.	2011	UK	Case report	1	VP	0.03 U/min	Shock CABG
Shah et al.	2011	USA	Case report	1	VP	0.06 UI/kg/hour	Septic shock
Bhaskar et al.	2014	Qatar	Case report	1	VP	0.0003 U/kg/min	Cardiothoracic surgery
Katayama et al.	2014	Japan	Case report	1	VP	NA	Septic shock
Shiber et al.	2015	USA	Case reports	2	VP	0.04 U/min	Polytraumatized patient
Bohl et al.	2016	USA	Case reports	6	VP	NA	Neurological ICU*
Sundar et al.	2016	—	Case report	1	VP	NA	Septic shock
Rana et al.	2017	USA	Case report	1	VP	0.04 UI/hour	Septic shock
Morkos et al.	2018	USA	Case report	1	VP	0.03 U/min	Septic shock
Carman et al.	2019	USA	Case report	1	VP	0.04 U/min	Septic shock
Ferenchick et al.	2019	USA	Retrospective cohort	29**	VP	NA	Multiple reasons***
Kim et al.	2020	USA	Case report	1	VP	0.04 U/min	Septic shock
Cobb et al.	2021	USA	Case report	1	VP	NA	Septic shock
Cristiano et al.	2022	USA	Case report	1	VP	NA	Septic and hypovolemic shock

CABG, coronary artery bypass graft; ICU, intensive care unit; NA, not available; VP, vasopressin. *Treatment of vasospasm, septic shock, hypotension, and increased mean arterial pressure in a setting of intracranial hypertension. **Patients that developed diabetes insipidus. ***Mainly: Cardiothoracic interventions, septic shock, and hemorrhagic shock

had already used diuretics, and seven individuals (14.3%) received corticosteroids prior to the diagnosis of DI.

No study has used VP analogs, such as ornipressin or terlipressin. The median time of VP infusion was 48 hours (IQR: 16–72). Table 1 summarizes the main reasons for VP use and other characteristics of the articles.

In the retrospective cohort study with 1,896 individuals who received VP infusion to treat shock, the incidence of DI was 1.53%, and half of these patients underwent a cardiothoracic intervention.[7] When comparing patients who received VP with those who received norepinephrine to treat shock, the incidence of DI in the norepinephrine group (*n* = 1,320) was 0.15%.[7]

DI Diagnostic Criteria

The diagnosis of DI was based primarily on diuresis output and concomitant hypernatremia or changes in serum sodium

Table 2: Diagnostic criteria used by authors to diagnose DI after VP withdrawal

Authors	*Diuresis output*	*Serum sodium*	*Plasmatic osmolality*	*Urinary osmolality*	*Urinary density*
Kristeller et al.	X	X			
Ramers et al.	X	X		X	
Peskey et al.	X	X	X	X	
Hayes-Bradley et al.	X	X	X	X	
Shah et al.	X	X			
Bhaskar et al.	X	X	X	X	
Katayama et al.	X	X			
Shiber et al.	X	X		X	
Bohl et al.	X	X			X
Sundar et al.	X	X	X	X	X
Rana et al.	X	X	X	X	
Morkos et al.	X	X		X	
Carman et al.	X	X	X	X	
Ferenchick et al.	X	X			
Kim et al.	X	X	X		
Cobb et al.	X	X		X	
Cristiano et al.	X	X		X	

DI, diabetes insipidus; VP, vasopressin

concentration, with urinary specific gravity being the least common criterion used. Only one study considered polydipsia as a diagnostic criterion for DI.[14] Table 2 summarizes the diagnostic criteria adopted by the studies.

The highest serum sodium and sodium delta found were 180 mEq/L and 46 mEq/L, respectively, for a 48-year-old man with a subarachnoid hemorrhage who received VP for 72 hours to treat vasospasm and presented with initial symptoms 9 hours after cessation of VP.[8] Regarding urine output, the highest value observed was 12 L in 8 hours, in a 34-year-old man admitted to the ICU for septic shock who received VP to treat hypotension (dose and infusion time not described).[15]

The median time for symptom onset after VP discontinuation was 5 hours (IQR: 3–10), with one patient presenting with symptoms 1 hours after cessation. Mean DI duration was 7 days (IQR: 2–12); however, a 51-year-old woman with septic shock who received VP at a rate of 0.03 U/minute for 30 hours presented with symptoms for 16 days.[16] This was a self-limiting adverse effect in all studies, lasting no longer than 16 days after interruption of VP.

Managing DI after Interruption of VP

The treatment of DI included fluid management in 10 (19.6%) patients and use of DDAVP in 16 (31.4%) patients.[8,13–23,26] Among those who received DDAVP, nine (56.3%) patients had a complete resolution, and seven (50.0%) patients showed a partial response. Eight (16.3%) patients resumed VP due to disease severity, showing improvement in clinical symptoms after restarting VP.[8,12,19,24,25] In children, DI management included fluids and electrolytes in one case, and VP resumption in the other case.[12,13]

Discussion

Vasopressin is frequently used to treat vasoplegic shock, with research showing that withdrawal of VP can lead to the development of DI with potential severe consequences, such as hypovolemia and hypernatremia. Our study found 17 articles, mainly case reports, addressing this issue. Diagnostic criteria and management varied among these reports, thus impeding physicians from recognizing and treating this condition.

Vasodilatory shock management requires fluid resuscitation and vasopressors. Norepinephrine is the first-line vasopressor, but some situations call for other vasopressors, such as VP or epinephrine.[1,27] A retrospective cohort study with 584,421 critical care patients showed that VP alone or combined with other vasopressors was commonly prescribed in the ICU to treat septic shock, being used in 17.2% of individuals.[9] Twelve studies included in this scoping review had patients with some type of shock, especially septic shock; this may contribute to the development of VP deficiency, since serum VP levels can drop in the late stages of shock.[1]

A randomized controlled trial and meta-analysis of patients with septic shock showed that, when compared with norepinephrine, VP had no difference with regard to 28-day mortality and serious adverse events, not including DI.[28,29] Similarly, another meta-analysis evaluating the use of VP and an analog (terlipressin) in relation to renal outcomes (renal replacement therapy and the incidence of acute kidney injury) showed favorable results for VP when compared to any other vasopressor during distributive shock.[30] In addition, polyuria with increase in serum sodium and low urinary osmolality can also occur after coronary artery bypass surgery, due to variations in extracellular fluid that can be detected by mechanoreceptors located in left atrium, aortic arch, or carotid artery, and affect VP release.[31]

The presence of acute kidney injury can cause impairment on urinary output and urine osmolality, as well as the use of diuretics and other drugs which affects water metabolism, masking a possible diagnosis of DI.[5] Septic shock is a common indication of VP use and it also can lead to acute kidney injury increasing the difficulty identifying DI in ICU patients.[32]

Diabetes insipidus is a disorder of water and sodium homeostasis that involves the hormone VP, which has a powerful vasoconstrictor (V2-receptors) and antidiuretic (V1-receptors) effects. The main difference between central and nephrogenic DI is that central DI responds to infusion of VP or an analog.[4,5] DI can be diagnosed by clinical and biochemical manifestations, such as

hypotonic polyuria, hypernatremia, high plasma osmolality, low urinary gravity, and polydipsia, which can lead to hemodynamic, neurologic, and renal impairments.[4,5] Plasma sodium and diuresis output were the most used criteria to diagnose DI, which can be considered good criteria as both are easily and frequently measured in the ICU.

Since critical care patients are prone to develop electrolyte imbalances, and hypernatremia can lead to irreversible neurologic damage, contributing to mortality. As a result, it is important to monitor serum sodium levels.[33] Only one study used polydipsia to diagnose DI, which is understandable, since many ICU patients cannot report thirst due to an impaired level of consciousness and the use of mechanical ventilation, conditions that can contribute to a more significant increase in serum sodium levels.[5] Serum copeptin levels, a peptide derived from VP that is more stable and easier to measure, can also be used to support the diagnoses of DI.[34] Unfortunately, copeptin dosages were not available in the reviewed articles.

Ferenchick et al. found a 1.53% prevalence of DI after withdrawal of VP, a number that might be underestimated due to some limitations of the study, especially its retrospective design.[7,34] Lack of medical knowledge on DI after interruption of VP and nonexistence of established criteria for its diagnosis hinders estimating the prevalence of DI. As such, we propose diagnostic criteria for this condition to help doctors identify this complication of treatment with VP (Table 3). This proposal is based on the summarized data available in the literature and further prospective studies are needed to validate these diagnostic criteria.

Although hypotension is frequent after withdrawal of VP, polyuria is not described as frequently. In our review, however, polyuria was a common feature, with values as high as 1500 mL/hour. This volume of diuresis output can lead to hypovolemia, hypotension, hypernatremia, or changes in serum sodium. Of note, the highest serum sodium found was 180 mEq/L, and the highest difference in serum sodium was 46 mEq/L.[8] As hyponatremia may occur during VP infusion, physicians must evaluate changes in serum sodium. These findings reinforce the importance of recognizing DI after withdrawal of VP and managing it promptly.

Even the dose and time of VP infusion can induce the development of DI; however, not all studies described the VP dose used, and for those who did, the units of measurement among them were divergent. The median time of VP infusion in this scoping review was 48 hours (IQR: 16–72), which is similar to a study that reported a median infusion time of 1 day (IQR: 1–2).[9]

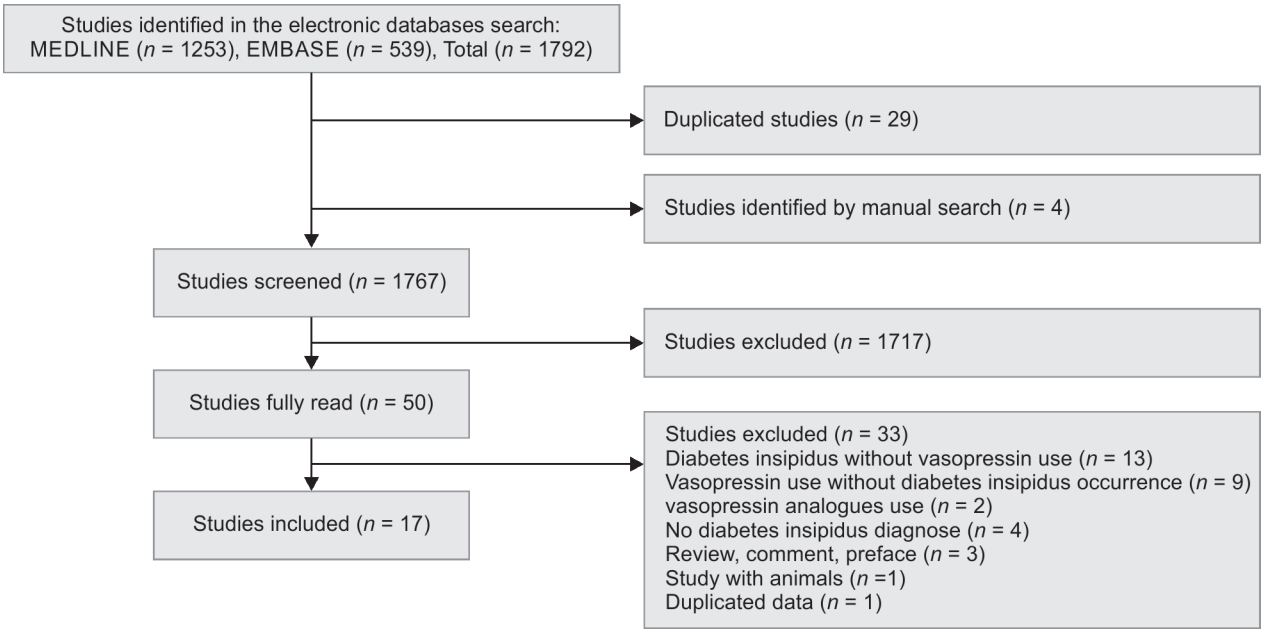
Management of DI must include replacement of VP and fluid resuscitation due to water loss to avoid or correct hypernatremia.[4,5] VP acts on the kidneys, stimulating the expression of aquaporin channels, and on vascular smooth muscle, promoting vasoconstriction.[2] However, DDAVP has fewer side effects regarding vasoconstriction and a better antidiuretic response than VP, being indicated as a first-line treatment.[5] Fluid resuscitation can be performed by replacing free water or with crystalloid solution, but care should be taken in correcting hypernatremia due to the risk of

Table 3: Clinical data of the studies included and proposed diagnostic criteria for DI after withdrawal of VP

Parameters	*Summarized data (n = 51)*	*Proposed diagnostic criteria*
Diuresis, mL/hour (*n* = 48)	222 (156–767)	>150 mL/hour
Highest serum sodium, mEq/L (*n* = 50)	152 (148–159)	>145 mEq/L
Delta sodium, mEq/L (*n* = 24)*	23 (12–30)	>12 mEq/L
Urinary osmolality (*n* = 12)	164 (111–240)	<200 mOsm/kg
Onset of symptoms, hour (*n* = 14)**	5 (3–10)	≤12 hours
Duration, days (*n* = 15)	7 (2–12)	Self-limited (maximum 16 days)

Data reported as median (IQR). DI, diabetes insipidus; VP, vasopressin. *Sodium after interruption of VP—sodium before interruption of VP. **After withdrawal of VP

Flowchart 2: Flowchart of Preferred Reporting Items for Systematic Review and Meta-Analyses (PRISMA)



cerebral injury.[5] Management of DI after withdrawal of VP varied between studies, with some patients restarting VP infusion while others managed symptoms conservatively. Flowchart 2 proposes an algorithm for DI management after withdrawal of VP according to the presence or absence of hypotension. This algorithm is based on the results of this scoping review. The intent is that it can serve as a tuition for clinical care of patients in ICU with DI after VP withdraw.

To our knowledge, this is the first study to review the relationship regarding the occurrence of DI after vasopressin withdrawal in critically ill patients. The main limitations of this study are most of the manuscripts included were case reports and case series, and a critical appraisal of the studies included was not performed.

In this review, all patients with DI after withdrawal of VP and that resumed VP infusion or were administered DDVP for DI management showed improvement in clinical symptoms, suggesting that DI is possibly of neurogenic origin. The mechanisms underlying DI after the discontinuation of a continuous VP infusion remain unclear. We hypothesize that administration of VP in supraphysiological doses can lead to a decrease in its vasoconstrictor effect, which is related to downregulation of the V2-receptor. Also, the development of DI after VP discontinuation may have a nephrogenic origin due to the downregulation of V1-receptors or aquaporin-2 channels when VP is at supraphysiological levels.[8]

Conclusion

Given the increased use of VP and its analogs to treat shock, and the research available on the development of DI after withdrawal of VP, physicians must be aware of the possibility of patients developing this condition. The diagnosis of DI in the critical care setting can be challenging to the medical staff, taking into account the difficulty in distinguishing the causes of hypotonic polyuria and electrolyte imbalances. Considering this syndrome can lead to severe consequences for ICU patients, early diagnosis with the aid of serum markers, such as copeptin, can contribute to a better clinical management. Multicentric collaborative research is urgently required to obtain more quality data on this topic.

Supplementary Materials

The Supplementary Table 1 is available online on the website of www.IJCCM.org.

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