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Intranasal vasopressin (DDAVP) and intra-dialysis hypotension incidence in end-stage renal disease

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Интраназальный вазопрессин (DDAVP) и частота внутридиализной гипотензии при терминальной стадии почечной недостаточности

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Key words: *Hemodialysis; hypotension; vasopressin; intranasal*

Abstract

Introduction: **intradialytic hypotension (IDH) remains one of the most common and potentially lethal acute complications encountered by patients under hemodialysis. To determine the effect of intranasal DDAVP (vasopressin) on IDH incidence and the volume of normal saline required to manage hypotension in hypotension-prone patients with chronic kidney disease stage 5 receiving hemodialysis treatment (CKD 5D).**

Material and Methods: **ten hypotension-prone CKD 5D patients were included in the study. They had experienced IDH for at least 30% of hemodialysis treatments in the preceding month. For 30 days, they received a placebo intranasal spray. For the next month, they received 2 puffs (each containing 10 μg) of vasopressin (DDAVP) 30 minutes before hemodialysis. IDH was defined as a symptomatic decrease in systolic blood pressure (BP) by more than 20 mmHg or a drop in mean arterial pressure (MAP) by more than 10 mmHg.**

Results: **IDH was observed in 68 hemodialysis sessions in the placebo group (63.6%) and 53 sessions in the vasopressin group (49.5%) with a marginally significant difference (P=0.07). A significant difference (P=0.04) was found between the two groups regarding the decrease in systolic BP that was more pronounced in the placebo group. Mean (±SD) normal saline volume administered intravenously was significantly lower in the vasopressin group (34±67.6 mL) compared to the placebo group (77.1±89.8 mL); P<0.001. Hypertonic saline was not required in either group.**

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Conclusion: **Although no statistically significant difference was observed between the placebo and intranasal vasopressin groups in terms of IDH incidence, mean systolic BP and MAP measurements were significantly higher in the vasopressin group. Also, the vasopressin group required significantly lower volumes of intravenous normal saline to manage IDH. Although we suggest intranasal vasopressin as a possible pharmacologic treatment to prevent IDH, it must be considered that the low sample size and non-significant difference in IDH frequency made our results non-generalizable. Further studies with a larger number of observations are required to make this statement convincing.**

Резюме

Введение. **Интрадиализная гипотензия (ИДГ) остается одним из наиболее частых и потенциально смертельных острых осложнений, с которыми сталкиваются пациенты, находящиеся на гемодиализе. Цель исследования – определить влияние интраназального DDAVP (вазопрессина) на частоту ИДГ и объем физиологического раствора, необходимого для лечения гипотонии у склонных к гипотензии пациентов с хронической болезнью почек 5 стадии, получающих лечение гемодиализом (ХБП 5D).**

Методы. **Включено десять пациентов с ХБП 5D, предрасположенных к гипотензии. ИДГ фиксировалась как минимум в 30% случаев гемодиализа в предыдущем месяце. В течение 30 дней они получали интраназальный спрей плацебо. В течение следующего месяца они получали 2 впрыскивания (каждое содержало 10 мкг) вазопрессина (DDAVP) за 30 минут до начала гемодиализа. ИДГ определяли как симптоматическое снижение систолического артериального давления (АД) более чем на 20 мм рт.ст. или падение среднего артериального давления (САД) более чем на 10 мм рт.**

Результаты. **ИДГ была зарегистрирована в 68 сеансах гемодиализа в группе плацебо (63,6%) и в 53 сеансах в группе вазопрессина (49,5%) со статистически незначимой разницей (P=0,07). Между двумя группами существовали достоверные различия (P=0,04) в отношении снижения систолического АД, и оно было более выраженным в группе плацебо. Средний (±SD) объем физиологического раствора, вводимого внутривенно, был значительно ниже в группе вазопрессина (34±68 мл) по сравнению с группой плацебо (77±90 мл); P<0,001. Гипертонический раствор не требовался ни в одной из групп.**

Заключение. **Хотя статистически значимых различий в частоте ИДГ между группой плацебо и группой интраназального введения вазопрессина не наблюдалось, средние значения систолического АД и САД были значительно выше в группе вазопрессина. Кроме того, группе вазопрессина требовались значительно меньшие объемы внутривенного введения физиологического раствора для лечения ИДГ. Хотя мы предлагаем интраназальный вазопрессин в качестве возможного фармакологического лечения для предотвращения ИДГ, необходимо учитывать, что небольшой размер выборки и статистически незначимая разница в частоте ИДГ сделали наши результаты не обобщаемыми. Чтобы сделать это утверждение убедительным, необходимы дальнейшие исследования с большим числом наблюдений.**

Ключевые слова: *гемодиализ; гипотония; вазопрессин; интраназальный*

Introduction

Intradialytic hypotension (IDH) or dialysis-induced hypotension remains one of the most common and life-threatening acute complications encountered by patients undergoing hemodialysis [1, 2]. It is estimated that IDH occurs in 20-30% of hemodialysis treatments [3, 4], but this important clinical condition may be underestimated [5]. For instance, in a recently published study on 1,137 patients, 75% of patients had at least one episode of IDH [6]. Although there is no uniform consensus among experts about the precise definition of IDH [7], according to the European Best Practice Guidelines, a decrease in systolic blood pressure (BP) of more than 20 mmHg or a decrease in mean arterial pressure (MAP) of 10 mmHg accompanied by complaints such as abdominal discomfort, nausea and vomiting, dizziness or fainting, and muscle cramps is used to define IDH [5, 8].

IDH sometimes necessitates decreasing blood flow rate in hemodialysis apparatus or even termination of ultrafiltration and dialysis. If done, the patient does not receive adequate hemodialysis. Also IDH, in long term, results in the development of cardiovascular complications, a more frequent need for hospital admissions, and even higher mortality [5].

In most patients who experience IDH, this complication occurs in the absence of serious conditions such as infections, arrhythmias, or pericardial tamponade [9]. Since plasma water is removed during hemodialysis, it causes a reduction in blood volume. When this process (i.e., ultrafiltration) is rapid or excessive, the resultant intravascular volume depletion and hyperosmolality are so significant that are beyond the capacity of the cardiovascular compensatory system. Among various possible mechanisms proposed for IDH, the mentioned mechanism is a major contributor to IDH occurrence [10].

Vasopressin (anti-diuretic hormone) is synthesized in the hypothalamus and stored in the posterior pituitary gland. Stimuli that cause a release of this hormone include volume depletion, hypotension, hyperosmolality, etc. By acting on distal convoluted tubules it increases water reabsorption. It also has vasoconstrictor effects. These effects along with observed evidence that vasopressin release is suboptimal in hemodialysis patients, even though the serum vasopressin level may be higher in hemodialysis patients than in healthy subjects [11-13] who develop IDH have all contributed to studying the effects of administering vasopressin (intranasally or intravenously) for prevention of IDH [14, 15]. However, the studies considering the effects of administered vasopressin (DDAVP) for preventing IDH are not enough. Hence, we intended to study the effect of intranasal vasopressin (DDAVP) on IDH occurrence, the total volume of normal saline infused to manage hypotension, systolic BP as well as MAP changes throughout the hemodialysis.

Methods

The study population consisted of CKD 5D patients who were referred to our academic hemodialysis center. Inclusion criteria were experiencing symptomatic IDH in at least 30% of hemodialysis episodes which resulted in discontinuation of hemodialysis during the preceding month. Exclusion criteria were serum sodium levels lower than 130 mg/dL, anemia (hemoglobin level lower than 10 g/L), ischemic heart disease, previous history of cardiovascular or cerebrovascular events such as myocardial infarction or cerebrovascular accidents, and simultaneous use of other medications to prevent IDH.

Our sampling was based on a conventional method. Considering the prevalence of IDH which was about 20-30% [3, 4], the sample size was calculated. Ten patients were sampled. First, the patients received a placebo nasal spray for one month. Then, all patients received vasopressin spray (Minirin®, Ferring GmbH, Kiel, Germany) for the next month. The sprays were administered 30 minutes before hemodialysis session initiation in a dosage of one puff in each nasal nostril (10 mcg in each puff, a total of 20 mcg for each patient).

The systolic BP measurements were done at several time points including at the initiation of the hemodialysis, during the dialysis (1, 2, and 3 hours after starting hemodialysis), and finally at the end of the dialysis session. We used the definition suggested by the European Best Practice Guidelines to define IDH. It was referred to as a decrease in systolic BP of more than 20 mmHg or more than 10 mmHg decrease in MAP along with symptoms such as headache or restlessness, abdominal discomfort, nausea/vomiting, fainting or dizziness, sighing or yawning, anxiety, and muscle cramps [8].

MAP was calculated by dividing the sum of doubled diastolic BP and systolic BP, by 3. To prevent hypotension during dialysis we 1) set the dialysis solution temperature at 0.5°C below the patient's average pre-dialysis tympanic membrane temperature; 2) balanced the solution sodium and potassium level based on the patient's sodium level; 3) gave oral food and glucose to the patients during and after the dialysis process; 4) made sure that the patient took his/her daily dose of antihypertensive medication after dialysis; 5) made sure that the pre-dialysis hemoglobin level was not lower than 10 g/dL; 6) considered diuretics in cases of residual kidney function and monitored the blood volume. Midodrine was not used in our study since it is not part of the routine protocol in our center. In case of IDH development, the patient's position was changed to the Trendelenburg position and a bolus of 0.9% saline (100 mL or more, as necessary) was rapidly administered through the bloodline. The ultrafiltration rate was reduced to as near zero as possible. The patient was then observed carefully

Statistical analyses

To describe the data, we used frequency or percentage for categorical data. For continuous data, the mean and its standard deviation (SD) were used. For analytical statistics, the categorical data were compared between the two groups using the Chi-squared test. For comparing continuous data, independent sample t-test and mixed ANOVA (analysis of variance) were used. The significance level was set at 0.05. All data analyses were done by the SPSS software for Windows (ver. 19.0).

Ethical considerations

At the outset of the study, the objectives of the protocol of the study were explained to the patients. If agreed, written informed consent was obtained from them. Also, the study protocol was reviewed by the Ethics Committee of our medical school and its contents were approved.

Results

Ten patients (* five males and five females) were enrolled here with a mean (±SD) age of 40.7 (±21.99) years. The etiology of CKD 5D was diabetic nephropathy in 4 patients, hypertension in one patient, urinary obstruction in one patient, and unknown in 4 patients. In the placebo group, a total of 107 hemodialysis sessions were done. In the vasopressin group, 105 hemodialysis sessions were done.

IDH was recorded in 68 hemodialysis sessions in the placebo group (63.6%) and 53 sessions in the vasopressin group (49.5%). Although this difference was not significant, it was relatively close to a significant difference ($P=0.07$). Table 1 presents systolic BP measurements at various times recorded. We used the mixed ANOVA test to compare the decrease in systolic

Table 1 | **Таблица 1**

Mean (SD) systolic blood pressure measurements at various times and its comparison between the two studied groups

Среднее (SD) измерение систолического артериального давления в разное время и его сравнение между двумя исследуемыми группами

group	Initiation of hemodialysis		One hour		Two hours		Three hours		End of hemodialysis	
	Mean	P	Mean	P	Mean	P	Mean	P	Mean	P
Vasopressin	127 (±13.5)	P<0.05	122.5 (±12.1)	P<0.05	120.4 (±10.9)	P<0.05	119.7 (±10.5)	P>0.05	119.8 (±10.3)	P>0.05
Placebo	122.5 (±14.5)		117 (±12.2)		113.6 (±12.9)		112.1 (±11.5)		110.8 (±9)	

Table 2 | **Таблица 2**

Mean (SD) mean arterial pressure (MAP) measurements at various times and its comparison between the two studied groups

Среднее значение (SD) среднего артериального давления (САД) в разное время и его сравнение между двумя исследуемыми группами

group	Initiation of hemodialysis		One hour		Two hours		Three hours		End of hemodialysis	
	Mean	P	Mean	P	Mean	P	Mean	P	Mean	P
Vasopressin	100 (±11.5)	P<0.05	96.1 (±9.6)	P<0.05	92.5 (±9.1)	P<0.05	91.4 (±8.2)	P<0.05	91.2 (±8.1)	P>0.05
Placebo	96.8 (±11.7)		90.1 (±9.3)		87.8 (±9.7)		86.7 (±8.8)		86.2 (±7.3)	

BP values between the groups and revealed a significant difference between the two groups (P=0.04), and the overall mean systolic BP decrease in the placebo group was larger. The mean systolic BP was significantly higher in the vasopressin group compared to the placebo group during hemodialysis (P<0.001).

Using the Bonferroni test, we revealed that systolic BP decreased significantly at the initiation of hemodialysis, hour 1 and 2 of dialysis (P<0.05), however after this time the changes were not significant (P>0.05).

Table 2 presents MAP measurements at various times recorded. Using the mixed ANOVA test to compare the decrease in systolic BP between groups, it was found that a significant difference existed between the two groups (P=0.01). In two-by-two comparisons between the two groups, MAP was significantly higher in the vasopressin group than in the placebo group during hemodialysis (P<0.001). MAP was decreased significantly in the placebo group at the initiation of hemodialysis, hours 1, 2, and 3 of dialysis (P<0.05), however after this time the changes were not significant (P>0.05). But in the vasopressin group, the changes were not significant after hour 2 of the hemodialysis.

In patients that developed IDH, a change in body position (i.e., Trendelenburg position) was done and normal saline was administered. With these measures, IDH improved. Hypertonic saline was not required in either group. The mean (±SD) volume of normal saline administered was lower in the vasopressin group (34±67.6 mL) compared to the placebo group (77.1±89.8 mL; P<0.001).

Discussion

There are different definitions for IDH based on the different blood pressure parameters (decrease in SBP, nadir SBP, or MAP), parameters for BP cut-off value, and the presence or non-presence of symptoms. However, the National Kidney Foundation's Kidney Disease Outcomes Quality Initiative guidelines defined it as a decrease ≥20 mmHg in SBP or ≥10 mmHg in MAP which leads to symptoms [16, 17]. In a recent review manuscript, it was given no exact definition for IDH, however, it was emphasized to manage any symptomatic decrease in blood pressure or a nadir intradialytic SBP <90 mmHg [18]. According to the obtained results, a clinically (but not statistically) significant difference was observed regarding the frequency of IDH between vasopressin and placebo groups. Administration of vasopressin resulted in fewer IDH occurrences, a lower total volume of infused normal saline, and significantly higher systolic BP and MAP. These findings are to some degree consistent with previous studies. The previous study [14], with a relatively similar design, reported that IDH occurred more significantly in the placebo group (125 times, 61.2%) than in the vasopressin one (DDAVP) group receiving 2 puffs, 30 minutes before hemodialysis (18 times, 8.8%). When compared to our results, this difference is more pronounced. Although we observed a lower rate of IDH in the vasopressin group (49.5% vs. 63.6%), this difference was not statistically significant. The reason for this discrepancy between studies [14] is noticeable. One possible cause seems to be related to the definition used to describe IDH. In the mentioned study [14], IDH was defined as a decrease in systolic BP of at least 10 mmHg, 2 hours after starting hemodialysis or at the end of the hemodialysis. However, we used a definition of a decrease in systolic BP of at least 20 mmHg after starting hemodialysis to spot patients with IDH. Also, the mean age of our patients (about 40 years) was lower than the mentioned study (about 47 years). The mentioned study did not report

systolic BP at baseline before starting hemodialysis, so we were not able to compare this item between the two studies. Also, because anti-diuretic hormone release is not normal in CKD 5D patients, this could be a contributor to these differences as its level was not measured in either study. In another study, Lindberg et al. [15] studied the role of lysine vasopressin on six patients with refractory hemodialysis-induced hypotension. They reported that the mean number of hypotensive episodes was significantly lower in the vasopressin group (0.9 episodes) than in the placebo group (1.5 episodes). Similar to what we observed here, the total volume of IV fluid administered was lower in the vasopressin group (155 mL) in comparison to the placebo group (280 mL). They concluded that lysine vasopressin is an effective pharmacologic tool to manage refractory dialysis-induced hypotension. There are several suggested approaches to prevent IDH which include optimizing the dialysis prescription (UF rate, cool dialysate, high-flux haemofiltration, and sodium profiling), as well as interventions during (midodrine, food intake, fluid administration) and between the dialysis sessions (lower drugs which decrease BP and lower intra-dialytic weight gain) [16]. Some other studies suggested inadequate increase levels of Arginine-Vasopressin (AVP) during hemodialysis as possible pathogenesis of IDH. These studies reported that the administration of exogenous AVP can have a positive role in preventing IDH. The reason that vasopressin can have a role in the management of IDH is justified by several factors. Firstly, there are studies consistently supporting the fact that vasopressin release is not sufficient in CKD 5D patients [13-22]. Hence, the experts have postulated that exogenous administration of vasopressin may facilitate ultrafiltration during hemodialysis by providing more stable arterial pressure. The second issue relates to the vasoconstrictive effect of vasopressin. As mentioned earlier, insufficient cardiovascular compensation is recognized as one of the main factors responsible for IDH. So, it is possible that with concerning the vasoconstrictive effect of vasopressin, this insufficient mechanism can somehow be compensated. Van der Zee et al. [14] studied the effect of constant infusion of a non-pressor dose of vasopressin in 22 CKD 5D patients who had hypertension. This resulted in a more stable arterial pressure and better excess extracellular fluid removal during ultrafiltration (250 mL) than in the placebo group (64 mL). As mentioned previously, IDH is a multi-factorial condition. Here, we face some limitations as we were not able to include all possible causes of IDH. It is recommended that in future studies, the researchers consider as many contributing factors to IDH as possible in an attempt to better illustrate the beneficial role of intranasal vasopressin in specific groups. Also, it is suggested to compare the efficacy of intranasal vasopressin to other pharmacologic treatments proposed for the prevention of IDH such as sertraline [22].

Conclusions

Although no statistically significant difference was observed between the placebo and the intranasal vasopressin group in terms of IDH incidence, mean systolic BP and MAP measurements were significantly higher in the vasopressin group. Also, the vasopressin group required significantly lower total volumes of intravenous normal saline to manage IDH. Although we suggest intranasal vasopressin as a possible pharmacologic treatment to prevent IDH, it must be considered that the low sample size and non-significant difference in IDH frequency made our results non-generalizable. Further studies with a larger number of observations are required to make this statement convincing.

Ethics approval and consent to participate

At the outset of the study, the objectives of the protocol of the study were explained to the patients. If agreed, written informed consent was obtained from them. Also, the study protocol was reviewed by the Ethics Committee of our medical school and its contents were approved.

The authors declare no conflict of interests.

Авторы заявляют об отсутствии конфликта интересов.

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Author's contribution: *AA participated in Conception and design of the study, library searches and assembling relevant literature, critical review of the paper, supervising writing of the paper, Database management. TM participated in Data collection, library searches and assembling relevant literature, writing the paper, and critical review of the paper. Both authors read and approved the final manuscript.*

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