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Vasopressin and Prevention of Hypotension During Hemodialysis

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Context: The occurrence of intradialytic hypotension (IDH) during hemodialysis (HD) continues to be a main problem in patients with ESRD (end-stage kidney disease). It also negatively affects health-related quality of life. We aimed to determine vasopressin effect in decreasing IDH.

Evidence Acquisition: We reviewed clinical and experimental literature in a variety of sources, including PubMed, Current Content, Scopus, Embase, and Iranmedex regarding the possible effect of vasopressin administration in prevention of hypotension during HD to clarify its mechanism, efficacy, and safety.

Results: Although arginine vasopressin is widely recognized for its anti-diuretic properties, it is also a well-recognized vasoconstrictor. It has been shown that the vasopressin release (as it would normally be expected) does not increase in the majority of HD patients with recurrent dialysis hypotension. In addition, it has also been reported that vasopressin secretion (due to the osmotic stimulation) is the most important mechanism in blood pressure control in ESRD patients receiving hypertonic solution for IDH. Therefore, it is suggested that vasopressin administration may improve hemodynamic stability among ESRD patients during HD. There are few clinical trials about this issue, suggesting that administration of exogenous vasopressin may be significantly associated with a decreased incidence of IDH as well as cardiovascular stability in ESRD patients in need of volume removal during HD.

Conclusions: Vasopressin insufficiency may have an important role in the pathogenesis of hemodynamic instability during HD and administration of exogenous vasopressin is significantly associated with a lower incidence of IDH.

*Keywords:*Arginine Vasopressin; Hemodialysis; Hypotension

1. Background

Although significant technical improvements have made since the introduction of hemodialysis (HD) in the early 1950, many complications with different underlying mechanisms still occur during HD (1-15). Intradialytic hypotension (IDH), especially during ultrafiltration dialysis (in which fluid removal is the primary goal) continues to be a main problem, especially in the elderly, diabetic, and cardiovascular compromised patients (16, 17). Development of IDH usually necessitates blood flow reduction and sometimes, discontinuation of HD. Therefore, it is an important cause of inadequate dialysis among these patients.

On the other hand, in the patients who need ultrafiltration during HD, the development of hypotension necessitates intravenous fluid replacement and therefore, it causes volume overload and some other significant complications (17). Unfortunately, the incidence of this problem is very high and ranges from 15% to 50%, especially among patients receiving ultrafiltration during HD (18). Many factors can contribute to hemodialysis hypotension, and they generally arise from these situations: fluid removal in an attempt to treat the volume overload and or to attain dry weight; reduction of plasma osmolality which causes water movement from extracellular space into the cells; use of a dialysate with low sodium concentration and or with acetate rather than bicarbonate as a dialysate buffer; reactions to the dialyzer membrane; use of antihypertensive medications and or ingestion of meal before or during HD session and cardiac arrhythmias or severe pericardial effusion with cardiac tamponade (12, 16, 19-31).

According to above etiology, different strategies with variable success rate have been offered to prevent or diminish IDH such as increased dialysate sodium concentration and sodium modeling, combination of sodium modeling and ultra-filtration, use of bicarbonate instead of acetate as a dialysis buffer, avoidance of low magnesium and low calcium dialysate, use of low dialysate temperature, avoidance of antihypertensive medicines on dialysis day, and no food intake during dialysis (32-46). In addition, a number of pharmacologic agents such as midodrine, carnitine and intravenous mannitol, have also been evaluated in the prevention of IDH; however, because of a few comparative studies, there are no generally accepted guidelines for using these agents in prevention of hypotension during hemodialysis (47-51).

Vasopressin is a well-recognized vasoconstrictor, and the role of vasopressin insufficiency has also been proposed in hypotension during HD. Thus, it is possible that vasopressin administration may improve hemodynamic stability among patients with end-stage renal disease (ESRD) during HD (29, 52-54). This article reviewed and summarized some of the observations about the possible effect of vasopressin administration to prevent IDH episodes.

2. Evidence Acquisition

We used a variety of sources to collect current data for this review article. Published articles of PubMed, Embase, Scopus, Current Content, SID, Google scholar, and Iranmedex were searched with key words of "DDAVP" or "vasopressin and Hypotension during Hemodialysis." Our review study included articles published in English language from January 1986 to July 2012, as full-text manuscripts, or abstract forms about the effect of vasopressin in prevention of hypotension during HD. Unfortunately, we did not specifically hand search conference proceedings.

3. Results

Many studies indicated the effect of vasopressin in the prevention of hypotension during dialysis. However, some studies examined other effects of vasopressin; for example, Beladi Moosavi and colleagues (3) studied its effect in relieving renal colic. Shimizu et al. (61) evaluated the mechanism of vasopressin secretion after infusion of a small amount of hypertonic saline and showed that hypertonic solutions induce a significant increase in arterial plasma osmolality without a significant effect on peripheral venous plasma osmolality. Therefore, it appears that by a mechanism similar to cardiopulmonary recirculation, increment in arterial plasma osmolality enhances vasopressin secretion and raises mean arterial pressure. Mechanism of vasopressin action was examined and concluded that its compensatory mechanism of hypotension and hypovolemia maintains and controls blood pressure, especially in inefficient cases. Mechanism of vasopressin secretion in a small amount of hypertonic saline infusion was studied, and it was found that hypertonic solutions increase arterial osmolality of plasma, but no significant effect on peripheral plasma osmolality. It seems that in ESRD patients undergoing chronic hemodialysis, administration of vasopressin can significantly reduce frequency of the hypotension episodes, which affects the dialysis quality.

4. Discussion

Arginine vasopressin (AVP), also known as anti-diuretic hormone (ADH), is derived from a preprohormone precursor. It is synthesized in the hypothalamus and stored at the posterior pituitary gland. It is released in the setting of hyperosmolality or nonosmotic stimuli, including volume depletion, hypotension, and nausea or vomiting. There are three receptors for AVP, including V1a, V1b, and V2 receptors. AVP plays a key role in the regulation of sodium balance and plasma osmolality by increasing water absorption in the distal convoluted tubules and collecting ducts of the kidney via V2 receptors (55). Its antidiuretic property has been exploited clinically for the management of patients with diabetes insipidus and nocturnal enuresis. In addition, there is also some new evidence that AVP may be effective for pain relieving in patients with acute renal colic possibly by its antidiuretic effect and decreasing intraurethral pressure. For example, a study showed that administration of desmopressin 40 μg as nasal spray in patients with acute renal colic decreases the pain scores after 30 minutes in a notable percentage of patients without any apparent side effects (56).

Vasopressin is a well-recognized vasoconstrictor by increment of peripheral vascular resistance via V1a receptors activation on vascular smooth muscle, but it appears that this effect is negligible in healthy individuals. However, in the setting of hypotension and hypovolemia, the hemodynamic response to vasopressin becomes an important compensatory mechanism in maintaining arterial pressure and tissue perfusion (56-59). For instance, in patients with septic shock, release of vasopressin (together with other vasoconstrictors) usually increases systemic vascular resistance and elevates blood pressure (57). In addition, in a study conducted in animals with hemorrhagic shock demonstrated that using V1 receptor antagonist increases hypotension (58). The results of Sato et al. (59) and Cignarelli et al. studies also supported the important role of vasopressin in the maintenance of blood pressure. They showed in two separate studies that vasopressin concentrations do not increase in the setting of orthostatic hypotension among diabetic patients with severe diabetic neuropathy (60).

In recent years, it has been observed that vasopressin release (as it would normally be expected) does not increase in the majority of HD patients with recurrent dialysis hypotension (61). Therefore, it is suggested that the vasopressin insufficiency due to decreased synthesis and or secretion may have an important role in the pathogenesis of hemodynamic instability during HD. In addition, as diabetes mellitus is the most common cause of ESRD, the results of Sato et al. and Cignarelli et al. studies also supported that the inappropriately low vasopressin concentrations may be a part of the underlying mechanism for IDH in diabetic patients (59). Shimizu et al. examined whether vasopressin is a part of the mechanism of blood pressure control in ESRD patients receiving hypertonic solution for IDH (61). They evaluated the effects of intravenous infusions of hypertonic saline, glucose, or physiological doses of vasopressin among 42 patients on long-term HD therapy during IDH. The results of the study showed that hypertonic solutions increase plasma

osmolality and plasma vasopressin levels. In addition, despite hypertonic solutions have been considered to act as plasma volume expanders; however, in the study, blood pressure increased independent of hypertonic saline effect on plasma volume. Therefore, it was suggested that vasopressin secretion due to osmotic stimulation is an important mechanism of blood pressure control. Vasopressin infusion was also increased mean arterial pressure and plasma vasopressin concentrations to the levels similar to those induced by the hypertonic saline and glucose. Finally, they strongly suggested that the osmotic stimulation of vasopressin secretion by hypertonic solutions has an important role in increasing blood pressure among these patients. They also claimed that vasopressin infusion may be an effective therapy to prevent IDH episodes (61, 62).

In another study, Shimizu et al. evaluated the mechanism of vasopressin secretion after infusion of a small amount of hypertonic saline and showed that hypertonic solutions induce a significant increase in arterial plasma osmolality without a significant effect on peripheral venous plasma osmolality. Therefore, it appears that by a mechanism similar to cardiopulmonary recirculation, increment in arterial plasma osmolality enhances vasopressin secretion and raises mean arterial pressure (63). To examine the role of insufficient vasopressin secretion in the pathogenesis of IDH, Friess et al. measured plasma vasopressin level in 23 ESRD patients with recurrent symptomatic episodes of hypotension during HD. Vasopressin concentration increased largely in 6 patients with hypotension and nausea during the study, but in the remaining 17 hypotensive patients without nausea, vasopressin level did not significantly increase during HD. Thus, the results of Friess et al. study also suggested the potential role of vasopressin insufficiency in the pathogenesis of IDH (52). The results of Rho et al. study have also clearly supported the finding of the previous article. In their observational pilot study, they assessed and compared the baseline AVP level and trend of AVP with ultrafiltration in patients with and without IDH. They observed that AVP concentration did not increase as much as it would normally be expected in the IDH patients compared with patients without IDH. In their conclusion, they suggested that ESRD patients with symptomatic IDH are unable to increase appropriately AVP secretion during hypotensive episodes (53). In summary, the results of above studies and other articles support the role of vasopressin insufficiency in the pathogenesis of IDH and possibility of AVP as a mechanism of therapy for patients with hemodynamic instability during HD.

However, clinical trials on the vasopressin effect on prevention of IDH are scant. In a double-blind, crossover fashion, Lindberg et al. assessed the efficacy of intranasal lysine vasopressin and placebo in the prevention of hypotension during HD. In their study, 6 patients with refractory hemodialysis-induced hypotension and abnormal autonomic testing were evaluated. The results of the study demonstrated that after using intranasal lysine vasopressin systolic, diastolic, and mean arterial blood pressures were significantly greater at 90 min of the dialysis session. In addition, the mean number of hypotensive episodes and the total volume of intravenous fluid administered (because of hypotension) decreased (54). van der Zee et al. (29) have also shown the efficacy of vasopressin in this issue and supported the findings of Lindberg et al. study (54). In van der Zee et al. study, the plasma vasopressin concentration during HD was measured and shown that the level of plasma vasopressin did not increase during HD in spite of significant fluid removal. Then, in a randomized, double-blinded and placebo-controlled trial they examined 22 ESRD patients and showed that, compared to a standard hemodialysis, blood pressure was more stable and the incidence of symptomatic hypotension did not increase by greater fluid removal among the patients receiving constant infusion of a nonpressor dose of vasopressin during hemodialysis. Only 9% of the patients in the vasopressin group (compare with 64% of the patients receiving placebo) had symptomatic hypotensive episodes. In addition, the increased fluid removal was also achieved only in the vasopressin group during the study. Finally, van der Zee et al. concluded that inadequate vasopressin secretion during HD is a likely contributor to the hypotension that limits removal of excess extracellular fluid, and administration of exogenous vasopressin, by supporting arterial pressure, may improve cardiovascular stability and facilitates volume removal during HD (29).

The results of double-blinded and placebo-controlled clinical trial of Beladi Mousavi et al. (64) also supported the findings of van der Zee et al. study (29). They compared the effect of intranasal desmopressin, a synthetic structural analog of antidiuretic hormone and intranasal distilled water in prevention of IDH among patients received ultrafiltration during hemodialysis. In the first month of this study, 17 patients with known symptomatic IDH received nasal spray of distill water as a placebo and after a 30-day washout period, the patients received intranasal desmopressin 30 minutes before all HD sessions. When compared to the placebo group, the incidences of IIDH episodes was significantly lower and the mean arterial blood pressure was significantly higher during hemodialysis with desmopressin (64).

Hypotension during hemodialysis is the most common and important side effect of HD, especially in the elderly and cardiovascular compromised patients. It has a negative impact on health-related quality of life too (70-78). Unfortunately the incidence of this problem is very high and ranges from 15% to 50% of dialysis sessions. Many factors have been proposed causing hypotension during HD and different strategies, and a number of pharmacologic agents have been evaluated to prevent IDH. Arginine vasopressin is widely recognized for its role in the regulation of sodium balance and antidiuretic properties. Moreover, it is a well-recognized vasoconstrictor and the

role of vasopressin insufficiency in the pathogenesis of hemodynamic instability during HD has also been demonstrated in recent years by several observations.

At first Friess et al. (52) and then other researchers showed that vasopressin release (as it would normally be expected) does not increase in the majority of hemodialysis patients with recurrent dialysis hypotension. In addition, Shimizu et al. (61) strongly demonstrated that vasopressin secretion due to osmotic stimulation is an important part of the mechanism of blood pressure control among ESRD patients receiving hypertonic solution for IDH. Hence, it is suggested that intravenous vasopressin and perhaps intranasal vasopressin administration may improve hemodynamic stability among ESRD patients during HD. There are few clinical trials, including van der Zee et al. (29), Beladi Mousavi et al. (64), and Shimizu et al. (61, 63) which have carried out on the possible effect of vasopressin in prevention of IDH episodes. All of them suggested that administration of exogenous vasopressin may be significantly associated with a decreased incidence of IDH episodes and cardiovascular stability among ESRD patients in need of volume removal during HD. Although the results of these studies are interesting, the studies have some limitations such as short duration, lack of information regarding the patients, and small number of patients enrolled in the studies. Therefore, further multicenter clinical trials with longer duration and larger number of patients are needed to determine the effect of vasopressin administration for prevention of hypotension during HD. The strength point of our study was its comprehensiveness to review all studies about vasopressin without prejudice. One of the weak points of our study was the use of articles abstract in cases where their full text was not available.

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