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The Effects of Epinephrine, Norepinephrine and Dopamine on Hemogram and Coagulation Parameters

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Abstract

Introduction: Vasopressor agents stimulate endothelial cells and platelets via the sympathetic nervous system and affect the coagulation cascade through various mediators. This study aimed to investigate the effects of epinephrine, norepinephrine, and dopamine on hemogram and coagulation parameters.

Materials and Methods: A total of 28 healthy Wistar albino rats weighing 250-300 grams were used in this study. The rats were randomly divided into four groups, with 7 in each group. 0.9 % serum physiologic (group C, n=7), epinephrine (group E, n=7), norepinephrine (group NE, n= 7), dopamine (group D, n=7) were intraperitoneally perfused to the rats under sedation. Blood samples were drawn from the rats 30 minutes after perfusion. Hemogram and coagulation parameters were compared between the groups.

Results: Granulocyte values were lower in the dopamine group. Platelet cluster count was higher in the dopamine group. The highest hemoglobin and hematocrit values measured among the groups were observed in Group E, and its values were higher than those in the norepinephrine and control groups.

Conclusion: It has been determined that vasopressor drugs affect hemogram values and platelet aggregation in different ways, but do not cause any changes in coagulation parameters.

Key words: Blood coagulation factors; dopamine; epinephrine; hematocrit; norepinephrine; platelet count

Introduction

Dopamine, norepinephrine, and epinephrine are vasopressors, known as catecholamines. These agents, which are frequently used in intensive care units, increase blood pressure via vasoconstriction (1,2). In addition, they have some effects, suggesting that they change hemogram and coagulation parameters. It has been reported that catecholamines can increase platelet activation and aggregation by stimulating endothelial cells and platelets through the sympathetic system (adrenergic receptors) (3-5). These stimulated cells affect the coagulation cascade by secreting mediators, such as factor VIII, von Willebrand factor (VWF), and tissue plasminogen activator (TPA) (6,7). There are studies in the literature reporting that vasopressor agents also induce platelet adhesion and aggregation and increase the mean platelet volume (MPV) (8-9). However, the effects of these agents on hemograms and coagulation parameters have not been adequately studied. In the current experimental study, we aimed to investigate the effects of frequently used vasopressor agents on hemogram and coagulation parameters.

Materials and Methods

A total of 28 healthy Wistar albino rats weighing 250-300 grams were used in this study. Healthy rats that had not been used in any previous study and had not been exposed to any drugs were included in this study. Rats were maintained in a room with cycled light (approximately 12 hours of light on and 12 hours of light off) and a temperature of 20-24 °C. They were provided standard rat chow and access to water up to 2 hours before anesthesia and analgesia

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administration. In this study, rats were randomly divided into four groups with seven rats in each group. The subgroups were named group E (epinephrine), group NE (norepinephrine), group D (dopamine), and group C (control group; serum physiological). Ketamine (Ketalar 1 ml: 50 mg, Pfizer, Istanbul, Turkey) at 50 mg/kg intraperitoneally (i.p.) and xylazine (Xylazinbio 2%, Bioveta, Czech Republic) at 10 mg/kg (i.p.)

Table 1. Descriptive statistics and comparison results for hemogram and coagulation parameters

	Group E (n=7)	Group NE (n=7)	Group D (n=7)	Group C (n=7)	p	
	Mean ± SD Median	Mean ± SD Median	Mean ± SD Median	Mean ± SD Median		
Rat Weights (g)	281.6 ± 5.0 280.0	284.3 ± 5.8 284.0	284.3 ± 7.1 282.0	283.7 ± 5.8 286.0	0.826	K
WBC ($10^3/mm^3$)	3.3 ± 0.5 3.2	3.3 ± 0.8 3.5	3.3 ± 0.7 3.2	3.2 ± 1.0 3.2	0.999	K
LYM ($10^3/mm^3$)	2.7 ± 0.5 2.6	2.7 ± 0.7 2.7	3.0 ± 0.7 2.8	2.6 ± 0.9 2.8	0.738	K
Granulocyte ($10^3/mm^3$)	0.3 ± 0.1 [a] 0.3	0.3 ± 0.1 [a] 0.3	0.1 ± 0.0 [b] 0.1	0.3 ± 0.2 [a] 0.3	***0.014***	K
Hemoglobin (g/dL)	14.7 ± 0.5 [a] 14.7	14.1 ± 0.4 [b] 14.0	14.4 ± 0.2 [a] 14.4	14.0 ± 0.6 [b] 14.1	***0.047***	K
MCH (pg)	19.8 ± 0.3 19.6	19.8 ± 0.4 19.9	19.7 ± 0.4 19.7	19.4 ± 0.6 19.3	0.395	K
MCHC (g/dL)	33.8 ± 0.3 33.7	33.7 ± 0.6 34.0	33.7 ± 0.5 33.4	34.3 ± 0.6 34.4	0.130	K
RBC ($10^3/mm^3$)	7.4 ± 0.3 7.5	7.1 ± 0.2 7.2	7.3 ± 0.2 7.3	7.2 ± 0.3 7.3	0.141	K
MCV (μm)	58.5 ± 1.1 58.4	58.8 ± 0.9 58.9	58.6 ± 1.7 58.9	56.6 ± 1.7 55.8	0.082	K
HCT (%)	43.5 ± 1.3 [a] 43.4	41.8 ± 1.3 [b] 41.6	42.8 ± 0.5 [a] 42.6	41.0 ± 2.1 [b] 41.3	***0.030***	K
RDW (fL)	13.4 ± 0.4 13.6	13.4 ± 0.4 13.3	13.4 ± 0.4 13.4	13.3 ± 0.6 13.5	0.966	K
PLT ($10^3/mm^3$)	730.7 ± 63.6 744.0	686.7 ± 96.3 693.0	618.9 ± 233.9 701.0	596.1 ± 188.7 642.0	0.399	K
MPV (fL)	6.2 ± 0.2 6.3	6.3 ± 0.2 6.3	6.4 ± 0.3 6.4	6.4 ± 0.1 6.4	0.330	K
PT (sec)	17.5 ± 2.4 16.9	17.3±3.0 17.1	19.6 ± 11.9 16.4	19.3 ± 3.2 18.8	0.518	K
APTT (sec)	21.2 ± 1.1 21.6	20.7 ± 1.3 20.4	21.7 ± 2.9 20.7	20.9 ± 2.2 18.8	0.895	K
INR	1.7 ± 0.0 1.7	1.6 ± 0.1 1.6	1.7 ± 0.2 1.6	1.7 ± 0.1 21.3	0.530	K
Number of clusters in peripheral smear (%)	7.4 ± 1.9 8.0	5. 7 ± 2.6 7.0	17.1 ± 6.1 16.0	5.9 ± 2.1 6.0	***0.001***	K

a,b →: Different lower case in the same row represent statistically significant differences among the groups. Values are presented as Mean ± standard deviation, and median; **LYM**: Lymphocyte, **MCH**: Mean corpuscular hemoglobin; **MCHC**: Mean corpuscular hemoglobin concentration; **RBC**: Red blood cell; **MCV**: Mean corpuscular volume; **HCT**: Hematocrit; **RDW**: Red cell distribution width; **PLT**: Platelet; **MPV**: Mean platelet volume; **PT**: Prothrombin Time; **APTT**: Activated Partial Thromboplastin Time; **INR**: International Normalized Ratio; **K:** Kruskal-wallis test.

were administered to all rats for sedation and analgesia before drug administration. Anesthesia was maintained in rats with ketamine. Considering the life expectancy of rats under anesthesia, the infusion time was standardized to 30 minutes. Room temperature was maintained at 20-24 ºC, and to prevent heat loss from the anesthetized rats, air-contact surfaces were covered. The dosage of drugs and physiological serum (0.9% NaCl) administered intraperitoneally was standardized. The infusion rate for all drugs and physiological serum (0.9% NaCl) was set at 7 ml/hour using an Infusomat® Space (B Braun, Germany) for all groups. The drug doses administered to groups E, NE, and D were determined to be the maximum doses specified in their package inserts and used in clinical practice. 0.9% physiological serum (7 ml/hr), Epinephrine (0.1 mcg/kg/min; 7 ml/hr), Norepinephrine (0.1 mcg/kg/min; 7 ml/hr), and Dopamine (20 mcg/kg/min; 7 ml/hr) were administered as a 30-minute intraperitoneal infusion. Blood samples (4 ml) were drawn intracardially 30 minutes after drug administration. Subsequently, the animals were sacrificed by cervical dislocation.

Ethical approval: This study was conducted at the Van Yuzuncu Yil University Experimental Medicine Application and Research Center, following approval from the Van Yuzuncu Yil University Animal Experiments Local Ethics Committee (03.10.2019-2019/09), in accordance with the provisions of the Declaration of Helsinki.

Statistical analysis: Descriptive statistics for the continuous variables (characteristics) were presented as Mean, Standard deviation and Median, while count and percentages for categorical variables. Normality assumption of the continuous variables was tested with Kolmogov-Simirnov test. After the normality test, Kruskal-Wallis test was performed for the non-normally distributed characteristics to compare the groups. Following the analyses, Dunn multiple comparison test was used to determine different groups. Statistically significance level was considered as 5% and SPSS (ver: 26) statistical program was used for all statistical computations.

Results

A total of 28 rats were used in this study. The groups were comparable in terms of weight (p=0.826) and drug infusion time (p=1). Granulocyte counts in group D were lower than those in groups E, NE, and K (p=0.014) (Table 1). The highest hemoglobin (mean±SD: 14.7±0.5) and hematocrit (mean±SD: 43.5±1.3) values measured among the groups were observed in Group E. These values were significantly higher than those in the norepinephrine and control groups (p=0.047 and 0.030, respectively). There were no significant differences between group E, group NE, group D, and group C in terms of hematological parameters such as WBC, lymphocyte, MCH, MCHC, RBC, MCV, RDW, PLT, MPV (p=0.999, 0.130, 0.141, 0.082, 0.966, 0.399 and 0.330, respectively), and coagulation parameters such as INR, PT, and APTT values (p=0.530, 0.518 and 0.895, respectively). The platelet cluster count in the peripheral smear was significantly higher in the dopamine group than in the epinephrine, norepinephrine, and serum physiological groups (p=0.001) (Table 1).

Discussion

In the current study, we examined the effects of vasopressor agents on hemogram and coagulation parameters and found that both epinephrine and dopamine increased hemoglobin and hematocrit values compared to the groups; however, dopamine increased hemoglobin and hematocrit values less than epinephrine. In addition, we found that dopamine decreased the granulocyte count and increased platelet aggregation in the peripheral smear compared with the other three groups. We determined that a 30-minute infusion of vasopressor agents did not cause significant changes in the coagulation parameters. Vasopressor agents are mostly used in hemodynamically unstable patients such as those with sepsis or septic shock. Healthy rats were used in this study. This study aimed to determine the effects of vasopressor agents on hemogram and coagulation parameters by excluding clinical conditions such as sepsis or septic shock that affect these parameters. Burns et al. (10) investigated the effects of vasopressor agents on leukocytes in a prospective study involving six healthy male volunteers. They divided the individuals into three groups and administered 0.1 mcg/kg/min epinephrine to the first group, 5 mcg/kg/min dobutamine to the second group, and 2 mcg/kg/min dopexamine to the third group. They observed a significant increase in white cell, lymphocyte, and neutrophil counts in the epinephrine infusion group and a mild decrease in the other two groups. In the current study, while there were no significant changes in white blood cell and lymphocyte counts in any of the groups, we found a decrease in the granulocyte count in group D. However, the decrease in the granulocyte count was consistent with the results

of the previous study. In an in vitro study in which healthy adult blood samples were incubated with epinephrine, Horn et al. (11) observed increased neutrophil and thrombocyte aggregations. The increase in the aggregation of neutrophils and thrombocyte was attributed to the increase in neutrophils in the peripheral blood owing to the release of various mediators after epinephrine administration. The results of this study support the increase in white blood cell count reported by Burns et al. (10). However, this study differed from that of Burns et al. (10). There was no increase in white blood cells in the epinephrine infusion group. In the current study, we determined that both epinephrine and dopamine, especially epinephrine, increased the hemoglobin and hematocrit values. This effect was not observed in any other groups. We also determined that vasopressor agents did not affect the RBC, MCV, RDW, MCH, and MCHC values. Similar to the results of the present study, Kjeldsen et al. (8) found that in healthy volunteers, the hematocrit values increased with increasing doses of epinephrine. Another study reported a relative decrease in plasma volume as a result of vasoconstriction due to the alpha-adrenergic effects of catecholamines released via the sympathetic system; thus, hematocrit values may increase (12). However, we believe that the increase in hematocrit and hemoglobin observed with epinephrine and dopamine infusions in the current study cannot be explained by vasoconstrictive effects alone. Noradrenaline had similar vasoconstrictive effects and did not cause an increase in these parameters in the current study. Based on this, it can be said that even short-term infusions of epinephrine and dopamine can cause an increase in hemoglobin and hematocrit values. In addition, unlike previous studies, the current study was an experimental study, and the blood volume in rats was less than that in humans. This difference should be considered when interpreting changes in plasma volume. α-adrenergic receptors are present on the surface of platelets, and their activation by adrenaline and noradrenaline may cause mild aggregation (13-18). Dopamine is a known platelet co-agonist (19). Although it has recently been reported that dopamine induces platelet production from megakaryocytes through oxidative stress-mediated signaling pathways (20), this effect was not clearly detected in the current study. A 30-minute infusion of dopamine may not be sufficient to determine this effect. Therefore, further studies are warranted. A previous study (8) reported that platelet count increased with increasing epinephrine infusion doses. These changes in platelet counts were determined for all the subjects. Similarly, an increase in platelet count and volume was observed in a study in which epinephrine was administered up to 0.04 mcg/kg to healthy men, an increase in platelet count and volume was found (21). The authors suggested that catecholamines increase platelet release from the spleen. Niemann MJ. et al. (22) performed a study involving eight splenectomized subjects and eight healthy volunteers to more clearly demonstrate the effect of catecholamines. Epinephrine infusion (6 μg/kg/h) was administered to the subjects for 1 h. In this study, the increase in platelet count observed in healthy subjects was not observed in the splenectomized subjects. Although there was a mild increase in platelet count and volume in the epinephrine group in the current study, this was not statistically significant. We believe that this was due to the relatively short 30-minute infusion time, although the infusion dose was high in the current study. These findings were consistent with those of a previous study. Vasopressor agents cause the release of various coagulation factors via platelet reactivity, even at physiological doses. Activated platelets provide an important phospholipid surface for the assembly of membrane-bound procoagulant enzyme complexes such as the prothrombinase complex composed of factor Xa, factor Va, calcium ions, and a phospholipid bilayer (23). In a prospective study conducted in healthy volunteers, the blood levels of factors V, VIII, X, XI, and XII and platelet counts increased after epinephrine infusion (24). In this study, no difference was observed in INR, PT, and APTT values between subjects administered vasopressor agent infusion and those administered 0.9% serum physiological infusion. It is known that factor 7 deficiency prolongs PT value; factors 8, 9, 11, and 12 deficiency prolong APTT; and factors 2, 5, and 10 (common pathway) deficiency prolong both PT and APTT (23). In this study, although direct factor levels were not addressed, since there was no change in the APTT, INR, and PT values after vasopressor agent infusion, we can indirectly say that factor levels did not show a significant change. However, further studies are needed to investigate how vasopressor agents affect the factor levels. Factor VII deficiency is known to prolong PT, while deficiencies in factors VIII, IX, XI, and XII prolong APTT. Deficiencies in factors II, V, and X (common pathway) prolong both PT and APTT (23). In this study, although specific factor levels were not measured, the lack

of changes in APTT, INR, and PT values following vasopressor agent infusion suggests that factor levels did not significantly change. However, further studies are needed to investigate the effects of vasopressor agents on these factor levels. The frequent use of vasopressors in intensive care and surgical settings suggests that the findings of these studies could impact a large patient population. New data may enhance clinicians' decision-making processes and positively influence patient outcomes.

Study limitations: Vasopressor agents are typically used for durations exceeding 30 minutes in intensive care units. However, in this study, the infusion duration of vasopressor drugs was limited to 30 minutes, considering the survival time of rats under anesthesia. This duration was evaluated as potentially insufficient for accurately monitoring changes in hemogram and coagulation parameters induced by inotropic agents and was recognized as a limiting factor of the study. Nevertheless, demonstrating the potential of inotropic agents to alter blood parameters may provide valuable insights for future research. In this context, further studies with longer infusion durations are recommended.

Conclusion

Vasopressor agents have shown specific effects on blood cells and coagulation mechanisms. Changes in hemogram values and platelet aggregation may be clinically significant. However, the fact that coagulation parameters are not affected indicates that the general coagulation process remains stable. New studies with longer infusion durations are needed to investigate these effects.

Conflicts of interest: Competing interests: The authors declare no competing interests.

Ethics approval: The current experimental study received approval from the Local Ethics Committee on Animal Experiments of Van Yuzuncu Yil University. (03.10.2019-2019/09). All experiments were performed in accordance with relevant guidelines and regulations.

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Data availability: The datasets generated during and/or analyzed during the current study are available from the corresponding author on reasonable request.

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