**The Effectiveness of Intravenous Vitamin K in Correcting Shock Liver-Associated Coagulopathy**

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**Background:** Shock liver is diffuse hepatic injury resulting from acute hypoperfusion, congestion, or hypoxia.1 Causes of shock (i.e., septic shock or hypoxic respiratory failure) and blockage of blood supply (i.e., hepatic sickle cell crisis or hepatic artery thrombosis) can cause ischemic liver injury.2 Patients with severe liver disease may be at higher risk of bleeding due to reduced synthesis of the procoagulant proteins.3 However, recent research in hemostasis has revealed patients with liver disease have compensatory factor production to counteract bleeding.4 Saja MF, et al found no significant improvement in the majority of coagulation parameters after vitamin K 10 mg subcutaneous administration.5 Per the 2019 American Gastroenterological Association guidelines, absent an improvement in INR after a 10 mg dose of vitamin K, repeated administration is unlikely to have benefits.6 The purpose of this project is to evaluate the effects of total dose of IV vitamin K in patients with shock liver coagulopathy in preventing major bleeding events.

**Objective**: The primary objective of this study is to compare incidence of major bleeding occurrences during hospitalization between the two groups: total dose of IV vitamin K 10 mg (control) vs. total dose of IV vitamin K > 10 mg (intervention). The secondary objective is to assess the percent change in INR from baseline post IV vitamin K administration.

**Methods**: This is a retrospective case study evaluating patients from January 2015 to May 2021 diagnosed with shock liver coagulopathy defined by ICD code K72.00. Patients were included if at least 18 years old, admitted to one of the intensive care units at UNC Rex, with a baseline INR of > 1.5, received at least one dose of IV vitamin K, and received at least one INR within 24 hours post IV vitamin K administration. Patients who received a total dose of IV vitamin K 10 mg (control) were compared to patients who received total dose of IV vitamin K >10 mg (intervention)**.**  Descriptive statistics (mean, standard deviation. minimum, maximum) on all variables were estimated initially. Treatment group differences on independent variables were estimated using t-tests, chi-square tests, or Fisher exact tests according to the metrics and distributions on relevant variables. Logistic regression was used to estimate the effects of dosage and other predictors on binary outcome.

# **Preliminary Results**: Total of 17 patients were included in this study—5 patients in the control group and 12 patients in the intervention group. Eighty-five percent of all doses (33/39) were 10 mg, and every respondent receiving a smaller dose (< 10 mg) received a 10 mg dose as well. Median total dose was 21 mg in the intervention group. Total of 15 patients experienced a major bleeding event post IV vitamin K administration, 4 patients (80%) of the control group and 11 patients (91.6%) of the intervention group. Following an initial dose of IV vitamin K, major bleeding event occurred within 24 hours—a median of 20 hours and 16 hours in the control and intervention group, respectively. Median baseline INR was 4.7 in the control group and 3.8 in the intervention group. Median time to first INR post IV vitamin K dose was 2.7 hours in the control group with a median INR percent change of -27% from baseline. Median time to first INR post IV vitamin K dose was 21.4 hours in the intervention group with an INR percent change of -31% from baseline.

**Conclusion**: Due to the lack of statistical significance, the results of this case study is hypothesis generating. The results of this case study shows that total dose of IV vitamin K > 10 mg did not reduce the incidence of major bleeding events in patients with shock liver coagulopathy compared to total dose of IV vitamin K 10 mg .

**References:**

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