Impact of Vitamin K Administration to Critically Ill Patients with an Elevated INR

Murray M, White T

Sentara Martha Jefferson Hospital, Charlottesville, VA

Background: There have been published studies on vitamin K administration to patients with supratherapeutic INRs due to vitamin K antagonist therapy (i.e. warfarin), malnutrition, and cirrhosis. To our knowledge, no studies examining the effect of administration of vitamin K to hospitalized patients who have coagulopathy for other reasons such as shock liver or disseminated intravascular coagulation have been published. Nonetheless, the administration of vitamin K to these patients is not uncommon in the acute care setting.

Objective: To determine the effect of vitamin K administration on INR in critically ill patients with an elevated INR who are not on anticoagulation, do not have liver cirrhosis, and may have an elevated INR for other reasons.

Methods: This was a retrospective chart review of intensive care unit patients with an elevated INR (greater than or equal to 1.4), which was not the result of anticoagulant medications or liver cirrhosis, who received IV or PO vitamin K (5 mg or 10 mg). The primary outcome was the change in INR after receiving vitamin K. A subgroup analysis was performed in which the primary outcome was determined in four separate groups of patients based on the dose of vitamin K received (5 mg or 10 mg) or the administration route of vitamin K (IV or PO). Secondary outcomes included incidence of bleeding, incidence of thrombosis, hospital length of stay, in-hospital mortality, and the proportion of patients with a response of INR from vitamin K administration, with response defined as a reduction in INR of at least 0.4.

Results: A total of 215 patients were screened; 14 patients met inclusion criteria and were not excluded from the study. For the primary outcome, there was a non-statistically significant reduction in INR of 0.07 (n = 14, 95% CI -0.26 to 0.41, p = 0.65). In the subgroup analysis there was a non-statistically significant INR decrease in the 5 mg dose group (n = 6, 95% CI -0.34 to 0.51, p = 0.63), the 10 mg dose group (n = 8, 95% CI -0.53 to 0.66, p = 0.81), and the IV administration group (n = 4, 95% CI -0.34 to 0.51), while the PO administration subgroup had a non-statistically significant increase in INR (n = 10, 95% CI -0.47 to 0.23, p = 0.46). Response to vitamin K was observed in two patients (14%).

Conclusion: In this small, retrospective, non-controlled study, there was a non-statistically significant decrease in INR for patients receiving vitamin K in the ICU; the clinical relevance of this decrease is unknown. As expected, the magnitude of INR reduction appeared to be greater for IV doses, but this was also a non-statistically significant result. Further studies are warranted to examine the impact of vitamin K on INR in critically ill patients with acute coagulopathy.