

EFFECT OF SUCCINYLCHOLINE ON PATIENTS USING STATINS

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Introduction: Over the past 2 decades, statin use has grown to more than 100 million prescriptions per year. Statins cause breakdown of the T-tubular system and subsarcolemal rupture (1), impair sarcoplasmic reticulum calcium cycling in mitochondria, and provoke myotoxicity and myalgias (2). Patients taking statins may thus be especially susceptible to succinylcholine-induced muscle injury. We therefore tested the hypothesis that succinylcholine administration increases postoperative muscle pain, as well as plasma myoglobin, potassium, and creatine kinase (CK) concentrations more in patients who take statins than in those who do not.

Method: With IRB approval, we screened patients 40-80 years of age scheduled for elective surgery not involving bone or muscle. Patients who took statins for at least three months and those who had never used statins were enrolled. Potential confounding factors were accounted for by stratification (every 10 years of age from 40-80 and gender) and statistical adjustment. General anesthesia was induced with fentanyl IV, 1-3 $\mu\text{g}/\text{kg}$, and propofol IV, 2-3 mg/kg . Thereafter, the succinylcholine 1.5 mg/kg was given. The incidence and degree of fasciculation after succinylcholine administration was recorded. Each patient was interviewed 2 and 24 hours after surgery by a blinded investigator to determine the degree of myalgia. Blood samples were obtained before induction of anesthesia, and then 5 minutes, 20 minutes, and 24 hours after succinylcholine administration for myoglobin, potassium, and CK concentrations. The primary outcome – myoglobin concentration 20 minutes after succinylcholine administration – was compared (after logarithmic transformation) in statin and non-statin users using analysis of covariance with adjustment for baseline confounders and baseline myoglobin concentration. To assess the difference between statin users and non-users in the intensity of muscle pain, we used proportional odds logistic regression with adjustment for baseline potential confounders and random intercept to account for within-subject correlation.

Results: We enrolled 20 patients who used statins and 14 who did not. Gender, age, ASA status, and medical history – including neck pain and hypercholesterolemia – differed significantly in patients who did and did not take statins. These factors were therefore included in a multivariable analysis. Myoglobin concentrations 20 minutes after succinylcholine administration were similar in patients who did and did not use statins ($P=0.29$), after adjusting for baseline myoglobin and baseline confounders. The ratio of geometric mean (95% interim-adjusted CI) myoglobin concentration at 20 minutes was 1.28 (0.67, 2.42) for statin users versus non statin users. Furthermore, we did not find a significant difference between the groups in myoglobin over time (Figure 1, $P=0.45$). No group differences were found in any secondary outcomes, including the duration of succinylcholine ($P=0.86$), serum potassium concentration ($P=0.33$), change in CK concentration ($P=0.33$), pain scores at 2 and 24 hrs score ($P = 0.63$) or the incidence of any side effect ($P = 0.97$).

Conclusion: Although statins and succinylcholine both cause muscle injury, patients taking statins do not appear to be at special risk from succinylcholine administration.

Myoglobin

