Inherited Cholinesterase Deficiency in a Parturient: Prolonged Neuromuscular Block Due to Pseudocholinesterase Deficiency in a Parturient

Abstract Type: Case Report/Case Series
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Introduction: Pseudocholinesterase deficiency can be inherited or acquired, and results in the impairment of the metabolism of choline-ester drugs such as succinylcholine and chloroprocaine. We present a case in which pseudocholinesterase deficiency was detected for the first time in the immediate post partum period after emergent surgery.

Case Report: 29 yr old G5, P5 parturient with no known medical issues and no previous surgery, 6hrs post normal vaginal delivery, had profuse vaginal bleeding and was brought to OR for emergent D&E and cervical laceration repair. Rapid sequence general anesthesia was induced with standard doses of propofol and succinylcholine. Surgery lasted for 1hr and 26 min, at the end of which the patient did not return to spontaneous breathing and remained apneic, due to prolonged neuromuscular blockade (TOF revealed 0/4 twitches). The patient was left intubated with ventilator support in the PACU for 1 hr 50 min later (total of 3 hr and 40 min from succinylcholine administration), when patient was successfully extubated; following commands, breathing spontaneously with good tidal volume and TOF of 4/4 strong twitches. A diagnosis of pseudocholinesterase deficiency was suspected. Laboratory test were undertaken for dibucaine inhibition testing. Dibucaine number returned at 38% and a diagnosis of heterozygous pseudocholinesterase deficiency was made. Patient recovered well and was discharged home in good condition, while being informed that she should give this information to anesthesia providers prior for any future surgery and that family members should be tested for this condition.

Discussion: Pseudocholinesterase activity is reduced during pregnancy and early post partum period, with the highest reduction (33%) occurring 3 days post partum (1). This reduction can cause a longer lasting neuromuscular block recovery from succinylcholine, when compared to non-pregnant patients, but is usually not clinically significant (2). If there is an additional decrease in levels due to additional pathology such as impaired liver function in HELLP, then a more pronounced residual neuromuscular block from succinylcholine can occur. The prolonged neuromuscular blockade from succinylcholine seen in this case, was likely due to the combination of pregnancy and heterozygous pseudocholinesterase deficiency. Neuromuscular monitoring is essential for early detection of prolonged neuromuscular block and for clinical assessment of recovery. Pseudocholinesterase deficiency should be suspected and tested for patients who have prolonged neuromuscular block after administration of succinylcholine.

References