St Segment Depression During Oxytocin Administration

Presenting Author: Tulsi V Akikwala M.D.
Presenting Author's Institution: Northwestern University Feinberg School of Medicine - Chicago, IL
Co-Authors: Christopher R Cambic M.D. - Northwestern University Feinberg School of Medicine - Chicago, IL
Christopher D Stevens M.D. - Northwestern University Feinberg School of Medicine - Chicago, IL

Introduction: Oxytocin is administered in the third stage of labor to prevent uterine atony. However, its administration is associated with many side effects including nausea, hypotension and tachycardia. Also, EKG changes suggestive of myocardial ischemia have been observed during oxytocin administration. We report the development of ST segment depression during oxytocin infusion in a parturient undergoing cesarean delivery.

Case Presentation: A healthy 30 yo gravida 1 para 0 parturient was scheduled to undergo a primary cesarean delivery for fetal macrosomia. Spinal anesthesia consisted of bupivicaine 0.75% 12 mg, fentanyl 15 mcg and morphine 150 mcg, resulting in a T4 surgical level bilaterally. After an uneventful delivery, an oxytocin infusion was started at 18 IU/hr. Approximately 5 minutes later, the patient became hypotensive (MAPs 52-57), and 2 mm ST segment depressions with T wave inversions were noted in leads II, III and aVF. Patient began complaining of dull, substernal, non-radiating chest pressure; her HR and SpO2 were within normal limits. Oxytocin infusion was discontinued and the patient's hemodynamics were supported with fluid and phenylephrine administration, resulting in improvement of her symptoms and MAP. She was placed on O2 6L/min via NC and stat labs (CBC, chemistry, troponins, and CK-Mb) were sent. Over the next 20 minutes, the ST segment depression gradually resolved, and oxytocin infusion was restarted at 3.6 IU/hr. A 12-lead EKG obtained in PACU was negative for ischemic changes, and all labs were within normal limits. ASA 325mg PO was given and patient remained hemodynamically stable. Follow-up CK-Mb and troponin levels were also within normal limits.

Discussion: ST segment changes following oxytocin administration have been reported, with oxytocin-induced hypotension and/or coronary vasospasm reported as the proposed mechanism. Although the exact etiology is unclear, there seems to be a dose dependent increase in the incidence of these ischemic changes, especially with bolus administration. Jonsson, et. al showed a significant difference in the occurrence of ST depressions with higher doses of oxytocin, with ST depressions occurring in 21.6% of patients receiving 10 IU of oxytocin IV bolus as opposed to 7.7% of patients who received 5 IU.1 Interestingly, our patient did not have any ST segment changes from spinal-induced hypotension, despite having a similar decrease in MAP as during the oxytocin administration. Furthermore, the ST segment changes in our patient occurred during IV infusion, as opposed to IV bolus. Although oxytocin infusion has been shown to decrease the incidence of marked hemodynamic changes when compared to IV bolus administration3, our case report suggests that the above side effects may still persist and can be deleterious.

References: