

Case Report

Case Report: Opioid-Induced Movement Disorder in the Immediate Postoperative/partum Period

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Background

Opioid-induced movement disorders (OIMD) are a rare cause of dystonia in the perioperative setting. Here we present a case of OIMD, responsive to naloxone, immediately after cesarean delivery.

Objective(s): The purpose of this report is to describe the critical parts of our workup that helped determine the etiology of this patient's new movement disorder.

Study Design

Case report of a single patient seen at Tripler Army Medical Center.

Case Summary (Results): A 34-year-old G2P0 at 37+6 days' gestation with preeclampsia with severe features was delivered of a live female, vigorous infant via cesarean section due to arrest of descent. The patient's pain was managed with fentanyl-bupivacaine via epidural catheter, IV fentanyl, and IV morphine. At the time of skin closure, the patient developed episodic, dystonic neck and upper extremity movements, and confusion. She remained hemodynamically stable and responsive. The patient's symptoms resolved with IV Naloxone, with no recurrence of these episodes in the post-partum period.

Conclusion(s): To the best of our knowledge, this is the first case of OIMD in a peripartum patient. The unique physiologic conditions of pregnancy can contribute to development of movement disorders. OIMDs, although rare, are an important etiology to consider in post-operative or post-analgesic dystonia. Naloxone is the treatment of choice in OIMDs.

BACKGROUND

Opioid-induced movement disorders (OIMD) are a rare cause of dystonia in the perioperative setting. Here we present a case of OIMD, responsive to naloxone, immediately after cesarean delivery. The purpose of this report is to describe the critical parts of our workup that helped determine the etiology of this patient's new movement disorder. After a detailed literature review, we note that this is the first case of OIMD in a peripartum patient. This is an important case study the unique physiologic conditions of pregnancy can contribute to development of movement disorders as previously described (Abusrir and Bruno 2022; Cersósimo et al. 2004; Cogen and Zimmerman 1979). OIMDs, although rare, are an important etiology to consider in post-operative or post-analgesic dystonia and to treat effectively in an emergent situation.

CASE SUMMARY

A thirty-four year old gravida two, para zero at 37 weeks 6 days gestation by sure last menstrual period was admitted for induction of labor for gestational hypertension in the setting of intermittent headache. Her prenatal course was complicated by gestational diabetes mellitus managed with insulin therapy, NPH, 38 units daily, chronic headaches treated with magnesium oxide, 400 mg PO twice daily, and iron deficiency anemia. The day prior to admission she was evaluated for a unilateral, frontal headache that was refractory to acetaminophen, prochlorperazine, diphenhydramine, and bupropion/acetaminophen/caffeine. An MRI of the brain, without contrast, demonstrated no acute intracranial abnormalities, and she was discharged home with plan for a repeat blood pressure check the following day.

On the following day (day of admission), her vital signs were significant for mild range blood pressure readings in the 130-140's over 80's-90's range, meeting criteria for gestational hypertension. Her cervix was noted to be closed

on digital examination. Her induction was initiated with oral misoprostol and a Cook Cervical Ripening Balloon was placed. Her labor was augmented with IV oxytocin which resulted in regular uterine contractions. An epidural catheter was placed, and her pain was managed with titration of fentanyl-bupivacaine, 2 micrograms/ml. At time of Cook Cervical Ripening Balloon removal, her cervix was noted to be at 5 cm dilated, 60% effaced, with the vertex palpated to be at -3 station. Over the next 4 hours, her labor progressed to 6 cm dilation, 75% effacement, and -2 station and atraumatic rupture of membranes was performed productive of clear amniotic fluid. Subsequently, she developed a headache that was refractory to diphenhydramine and metoclopramide, suggestive of a diagnosis of pre-eclampsia with severe features. Intravenous magnesium sulfate was started for seizure prophylaxis. Over the next eight hours, despite adequate uterine contractions, she failed to progress in labor. The patient underwent a primary lower uterine segment transverse cesarean section because of a diagnosis of arrest of descent. The patient's magnesium sulfate infusion was paused in advance of the patient's cesarean section.

The cesarean section was productive of a vigorous, female infant with APGAR scores of 8 and 9. During closure of the hysterotomy incision, uterine atony was noted. Fundal massage was performed during which the patient expressed discomfort and fentanyl, 100 micrograms IV were administered. Despite uterine massage and closure of the hysterotomy incision, atony persisted with significant bleeding observed. The patient was given tranexamic acid, 1 gram IV, carboprost, 250 micrograms IM, methylergonovine, 0.2 micrograms IM, and misoprostol, 600 micrograms PO. Upon return of the uterus to the abdominal cavity, improved uterine tone was noted. Immediately before closing the fascial layer, uterine massage was once again performed during which the patient noted significant pain. In response, morphine, 2 mg IV was administered. The operation proceeded routinely; however, at the time of skin closure 10 minutes later, the patient was noted to be confused and expressed an inability to control her limbs. Episodes of dystonic movements of the neck and upper extremities were observed. The anesthesiology team noted associated involuntary tongue movements, superior, vertical nystagmus, and intermittent apnea. These episodes occurred approximately every two minutes and lasted several seconds.

Midazolam, 2 mg IV, was given and an emergent consultation with Neurology was requested. Throughout these events, the patient remained responsive and hemodynamically stable with O₂ saturations in the high 90's. Given the concern for a possible stroke, a non-contrast CT head was ordered, and the patient was transferred to the intensive care unit. Immediately prior to transport, the patient was given naloxone, 0.08 mg IV, and diphenhydramine, 25 mg IV. Upon reaching the ICU 15 minutes later, the patient's involuntary movements had decreased in frequency and amplitude. By the time of evaluation by the Neurology team, 50 minutes later, her dystonic episodes had ceased. Her non-contrast CT results did not demonstrate any intracranial pathology.

Intravenous magnesium sulfate was commenced, again, for seizure prophylaxis and her postpartum course proceeded without complication. The patient's qualitative blood loss was 1582 mL. Her postoperative CBC revealed a hemoglobin of 7.6 g/dL, compared to her pre-operative hemoglobin of 12.0 g/dL. The patient was discharged on postpartum day 3 in stable condition. The patient reported no recurrence of dystonic episodes at the time of her six-week post-partum appointment.

DISCUSSION

Presentation of a new movement disorder is uncommon in pregnancy. Patients more commonly experience a recurrence or exacerbation of a previously known movement disorder (Abusair and Bruno 2022; Cersósimo et al. 2004). The most frequently reported movement disorders in pregnancy include restless leg syndrome, Parkinson disease, Huntington disease, Wilson disease, and pre-pregnancy ataxia. Exacerbation of these conditions may be prompted by alterations in pharmacologic regimens because of the developing fetus. The physical and psychological stresses of pregnancy and labor may also contribute to worsening of known movement disorders (Abusair and Bruno 2022).

Coordination of movement largely originates in the basal ganglia and is controlled by the release of neurotransmitters, including dopamine, acetylcholine, gamma-aminobutyric acid, and glutamate. Dysregulation of this process can lead to exaggerated muscular contractions and abnormal posturing. Estrogen and progesterone derivatives are known to alter neurotransmitter trafficking and their concentrations throughout the cerebrum (Cogen and Zimmerman 1979). Indeed, both biochemical and behavioral research in animals has confirmed that estrogen modulates basal ganglia function (Van Hartesveldt and Joyce 1986). For example, estrogen treatment in rats increases the number of dopamine receptors in the striatum (Hruska and Silbergeld 1980). This mechanism is further supported by case reports of dystonia after initiation of combined oral contraceptives (Nausieda et al. 1979; Driesen and Wolters 1987). It follows that the elevated estrogen and progesterone levels of pregnancy may alter basal ganglia functioning and create a predisposition toward dystonia, although this has not been well studied.

Of newly presenting movement disorders in pregnancy cervical dystonia, sometimes termed dystonia gravidarum, is the best described (Lim et al. 2006; Donlon et al. 2022a, 2022b; Buccoliero et al. 2007; "Cervical Dystonia in Pregnancy in a Woman with Three Kidneys, Uterine and Ocular Anomalies (Madu?)," n.d.). Dystonia gravidarum describes a constant, involuntary spasm of the musculature of the neck, particularly the sternocleidomastoid, which develops during pregnancy without clear underlying etiology. The development of this condition has been described at varying stages of pregnancy, including the report of a 31-year-old primigravida who presented at 4 weeks gestation (Lim et al. 2006). The dystonia may wax and wane throughout pregnancy, but in all cases resolves with delivery.

In the case of our patient, her irregular movements developed after delivery of her fetus. Rather than a sustained muscular contraction, her dystonia was intermittent and rhythmic. The patient's intact level of consciousness and unresponsiveness to benzodiazepines are inconsistent with a seizure. Cerebral vascular accident was considered although less likely given that our patient lacked significant risk factors. Her rapid return to baseline and MRI quickly ruled out this diagnosis.

Given the patient's absence of a history of movement disorders, drug induced dystonia was thought to be most likely. Over the course of her induction and surgery, she received several different drug classes including NSAIDs, antihistamines, antiemetics, prostaglandins, uterotonics, and opioids. Resolution of her symptoms occurred only after administration of naloxone suggestive of an opioid-induced movement disorder (OIMD) (Iselin-Chaves et al. 2009). Indeed, similar episodic, involuntary, whole-body contractions have been described in a 22-year-old male with OIMD in the post-operative period. In that particular case, the diagnosis was suggested by symptom resolution with cessation of opioids rather than with administration of an opioid antagonist (Chino et al. 2021).

Opioids exert their effects by acting on the kappa, gamma, and mu opioid receptors. The analgesic effects of opioids originate primarily from their action on the mu opioid receptor. These opioid receptors are expressed throughout the striatum contributing to reward pathways, but are also implicated in motor control. Specifically, opioids inhibit release of acetylcholine through activation of the mu opioid receptor and by acting on cholinergic interneurons directly. In laboratory studies, a dose-dependent inhibitory effect has been demonstrated on cholinergic interneurons by mu opioid receptor agonists, giving a pathogenic mechanism for OIMD ("Enhanced Mu Opioid Receptor-Dependent Opioidergic Modulation of Striatal Cholinergic Transmission in DYT1 Dystonia???", n.d.).

In summary, we have reported a case of acute dystonia in a primiparous patient in the immediate post-partum and post-operative setting which resolved with naloxone ad-

ministration, most likely consistent with an opioid-induced movement disorder. To the best of our understanding, this is the first reported case of an opioid induced movement disorder in a peripartum patient. This is of particular interest given the unique effects of estrogen and progesterone on the basal ganglia, the primary hub of motor coordination in the brain. Opioids are a recognized, if not rare, cause of movement disorders which may be precipitated at low doses. Naloxone administration should be considered when there is clinical suspicion for OIMD.

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AUTHORS CONTRIBUTIONS

Pablo Antonio Delis: Primary Author. Chiefly responsible for concept and design of manuscript.

Eric Michael Schmitt: Made substantial contributions to analysis and interpretation of data.

DISCLOSURES

The Author(s) reports no conflicts of interest, and no funding has been received for this work.

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The views expressed herein are those of the presenter and do not reflect the official policy of the Department of the Army, Department of Defense, Defense Health Agency, or U.S. Government.

DISCLAIMER

Eric M SCHMITT, MD, Pablo A DELIS, MD are employed by the Armed Forces.

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