

CLINICAL VIGNETTE

Obstetric Anesthesia Considerations in Chorioamnionitis, Hemorrhage and Disseminated Intravascular Coagulation (DIC)

Leo Varzi, DO

Case

The patient is a 33-year-old female, G2P1 at 39 weeks gestational age with past medical history of anxiety and previous cesarean section for placenta previa. She presented to the obstetric department with uterine contractions and was admitted for a trial of labor after cesarean section. The patient had initial hemoglobin of 14.1 g/dL and platelets of 178 K/cumm. A well-functioning epidural was established for pain control and as a source of anesthesia in case the patient needed a cesarean section. During labor she developed category 2 fetal heart tracings. She also had a temperature of 38.2 degrees Celsius and suspected chorioamnionitis, for which she received ampicillin and gentamicin. Her fetal heart tracings were unresponsive to interventions and the patient developed constant abdominal pain in between contractions despite having a well-functioning labor epidural. The decision was made to proceed with emergent cesarean section with concern for uterine rupture.

In the operating room the patient's well-functioning epidural was used for the cesarean section with an epidural bolus of 2% lidocaine with 1:200,000 epinephrine, fentanyl and sodium bicarbonate. A phenylephrine infusion was started at the beginning of the case to maintain appropriate blood pressures and her vital signs were stable with blood pressure readings in the 120/80. The surgeon noted uterine rupture intraoperatively at the patient's prior hysterotomy site. An infant was delivered without difficulty with 1, 5 and 10 minute APGAR scores of 4, 7 and 7. After delivery of the infant, an oxytocin infusion was started, however the surgeon noted continued uterine atony.

At this time the patient was requiring increased phenylephrine and crystalloids to maintain appropriate blood pressures. The surgeon reported no visible bleeding and 500ml of estimated blood loss so far. Evaluation under the drapes also showed no visible signs of vaginal or other sources of bleeding at this time. Methylergonovine and carboprost were given to help with uterine tone. The patient continued requiring increased phenylephrine and fluid resuscitation. Reevaluation under the drapes the second time showed notable vaginal bleeding. Complete blood count (CBC), prothrombin time (PT), international normalized ratio (INR), Partial thromboplastin time (PTT) were drawn and sent by the nurse. The patient was given tranexamic acid were given. Packed red blood cells (PRBC) were requested to the operating room and 750ml of 5% albumin was given, 3 units of PRBC were transfused in the operating room immedi-

ately as they were received. The initial CBC, PT/INR, PTT was marked invalid by the laboratory due to dilutional error. Repeat CBC, PT/INR, PTT and BMP after 3 units of PRBC, 7 liters of crystalloid, 750ml of 5% albumin, showed a hemoglobin of 10.1 g/dL, platelets of 102 K/cumm, PT 44.3 sec, INR 4.74, PTT 112.6 sec, fibrinogen less than 60mg/dL and calcium 8.1mg/dL. Intraoperative lab work was concerning for Disseminated Intravascular Coagulation (DIC) and the patient was additionally transfused 2 units of Fresh Frozen Plasma (FFP) and given intravenous calcium chloride.

Postoperatively in the Post Anesthesia Care Unit (PACU) the patient received an additional 2 units of PRBC, 2 units of FFP and 1 unit of platelets. Estimated blood loss was 2,500ml. Her temperature was 36.7 degrees Celsius. Given the coagulopathy, the epidural was left in place until the coagulation status improved in order to avoid risk of epidural hematoma. With coagulopathy epidural catheter placement and removal are both associated with increased risk of developing epidural hematomas. Laboratory values were carefully monitored postpartum, with stable hemoglobin and platelets on postoperative day one. The epidural was removed on postoperative day two, with a platelet count of 95 K/cumm and INR of 1.19. Her hemoglobin continued to be stable and ranged between 8.5 – 8.9 g/dL throughout her admission and her platelets were 150 K/cumm at discharge. She was continued on antibiotics postpartum for 24 hours and was afebrile for the remainder of her admission. Her white blood cell count had improved at discharge.

Discussion

Chorioamnionitis is an infection which can include the placenta, amniotic fluid, fetus/fetal membranes or decidua, and is usually due to a polymicrobial infection after membrane rupture.¹ It is estimated that between 2-5 percent of term deliveries are affected by an intra-amniotic infection.^{2,3} Chorioamnionitis can lead to acute neonatal morbidity with meningitis, sepsis, pneumonia.² It can also lead to maternal morbidity with endometritis, as well as postpartum uterine atony and hemorrhage as seen in our patient.⁴ Suspected chorioamnionitis is based on clinical criteria including maternal intrapartum fever along with at least one of the following: fetal tachycardia, maternal leukocytosis or purulent cervical discharge. Con-

firmed chorioamnionitis is based on positive amniotic fluid test results or placental pathology.¹ Management of chorioamnionitis consists of antipyretics, antibiotics and ensuring proper labor progression given its association with dysfunctional labor.^{3,5} Intrapartum antibiotics decreases rates of neonatal bacteremia, pneumonia and sepsis along with decreasing maternal febrile morbidity and hospital length of stay.⁵ Chorioamnionitis by itself is not an indication for immediate delivery or an indication for cesarean delivery. The route of delivery should be based on standard obstetric indications and clinical judgement.⁶

It is important for anesthesiologists to be aware of the possible complications that can arise from chorioamnionitis and be prepared to treat them. As in our case, the patient was suspected to have chorioamnionitis and later developed uterine atony with hemorrhage. Intraoperatively the patient was requiring increased phenylephrine and crystalloids to maintain appropriate blood pressures. The surgeon initially stated no visible bleeding and 500ml of estimated blood loss and initial evaluation under the drapes showed no visible signs of vaginal or other sources of bleeding. However, the patient continued requiring increased phenylephrine and fluid resuscitation. Reevaluation under the drapes the second time showed notable vaginal bleeding. Therefore, it is very important to beware of the possibility of uterine atony and occult uterine hemorrhage in patients with chorioamnionitis in order to diagnose and treat the patient in a timely manner.

Disseminated Intravascular Coagulation (DIC) is often described as a thrombo-hemorrhagic disorder, where patients can present with thrombosis or bleeding, either simultaneously or at different times. The pathophysiology may be explained by the two different ways of excess thrombin generation, one which is rapid and another which is slower. An extremely rapid burst of excess thrombin production is believed to lead to a hyperfibrinolytic form of DIC, as seen in obstetrical DIC. A more gradual increase in the amounts of thrombin is believed to lead to a procoagulant form of DIC, as seen in septic DIC. This can lead to simultaneous hemorrhage from distant sites and thrombosis in the microcirculation.^{7,8}

Although DIC can have multiple causes, an important cause to be aware of in a parturient is from hemorrhage, as seen in our case. Bleeding in Hemorrhagic DIC can be secondary to thrombocytopenia, platelet dysfunction, endothelial damage, interference of the fibrin degradation products with the clot structure and consumption of clotting factors.^{7,9} DIC is a clinical and laboratory diagnosis and the diagnosis is only made in patients with an underlying disorder known to be associated with DIC in conjunction with laboratory abnormalities in platelet count, PT/PTT, serum fibrinogen and fibrin degradation markers. DIC is always secondary to an underlying process, so it is important to treat the underlying cause in order to limit the excess thrombin generation.^{7,10-12} In our patient it was important to identify and stop the source of bleeding and to treat the hemorrhage with the appropriate blood products. DIC also has implications in a parturient with a labor epidural when deciding

the appropriate time to remove the epidural catheter to minimize risk of developing an epidural hematoma. If DIC develops in a patient with a labor epidural, the catheter should be removed once normal clotting criteria have been established and there are no clinical signs of DIC.^{13,14}

REFERENCES

1. **Higgins RD, Saade G, Polin RA, Grobman WA, Buhimschi IA, Watterberg K, Silver RM, Raju TNK; Chorioamnionitis Workshop Participants.** Evaluation and Management of Women and Newborns With a Maternal Diagnosis of Chorioamnionitis: Summary of a Workshop. *Obstet Gynecol.* 2016 Mar;127(3):426-436. doi: 10.1097/AOG.0000000000001246. PMID: 26855098; PMCID: PMC4764452.
2. **Newton ER.** Chorioamnionitis and intraamniotic infection. *Clin Obstet Gynecol.* 1993 Dec;36(4):795-808. doi: 10.1097/00003081-199312000-00004. PMID: 8293582.
3. **Kim CJ, Romero R, Chaemsaitong P, Chaiyasit N, Yoon BH, Kim YM.** Acute chorioamnionitis and funisitis: definition, pathologic features, and clinical significance. *Am J Obstet Gynecol.* 2015 Oct;213(4 Suppl):S29-52. doi: 10.1016/j.ajog.2015.08.040. PMID: 26428501; PMCID: PMC4774647.
4. **Hauth JC, Gilstrap LC 3rd, Hankins GD, Connor KD.** Term maternal and neonatal complications of acute chorioamnionitis. *Obstet Gynecol.* 1985 Jul;66(1):59-62. PMID: 4011072.
5. **Gibbs RS, Dinsmoor MJ, Newton ER, Ramamurthy RS.** A randomized trial of intrapartum versus immediate postpartum treatment of women with intra-amniotic infection. *Obstet Gynecol.* 1988 Dec;72(6):823-8. doi: 10.1097/00006250-198812000-00001. PMID: 3186087.
6. Committee Opinion No. 712: Intrapartum Management of Intraamniotic Infection. *Obstet Gynecol.* 2017 Aug;130(2):e95-e101. doi: 10.1097/AOG.0000000000002236. PMID: 28742677.
7. **Thachil J.** Disseminated Intravascular Coagulation: A Practical Approach. *Anesthesiology.* 2016 Jul;125(1):230-6. doi: 10.1097/ALN.0000000000001123. PMID: 27031011.
8. **Montagnana M, Franchi M, Danese E, Gotsch F, Guidi GC.** Disseminated intravascular coagulation in obstetric and gynecologic disorders. *Semin Thromb Hemost.* 2010 Jun;36(4):404-18. doi: 10.1055/s-0030-1254049. Epub 2010 Jul 7. PMID: 20614392.
9. **Yaguchi A, Lobo FL, Vincent JL, Pradier O.** Platelet function in sepsis. *J Thromb Haemost.* 2004 Dec;2(12):2096-102. doi: 10.1111/j.1538-7836.2004.01009.x. PMID: 15613012.
10. **Miller RD.** *Miller's anesthesia.* 7th ed. Philadelphia, PA: Churchill Livingstone/Elsevier; 2015.
11. **Mackey DC, Butterworth JF, Mikhail MS, Morgan GE, Wasnick JD.** *Morgan & Mikhail's clinical anesthesia.* 5th ed. New York, NY:McGraw-Hill Education LLC; 2013.

12. **Levi M, Toh CH, Thachil J, Watson HG.** Guidelines for the diagnosis and management of disseminated intravascular coagulation. British Committee for Standards in Haematology. *Br J Haematol.* 2009 Apr;145(1):24-33. doi: 10.1111/j.1365-2141.2009.07600.x. Epub 2009 Feb 12. PMID: 19222477.
13. **Horlocker TT, Wedel DJ, Benzon H, Brown DL, Enneking FK, Heit JA, Mulroy MF, Rosenquist RW, Rowlingson J, Tryba M, Yuan CS.** Regional anesthesia in the anticoagulated patient: defining the risks (the second ASRA Consensus Conference on Neuraxial Anesthesia and Anticoagulation). *Reg Anesth Pain Med.* 2003 May-Jun;28(3):172-97. doi: 10.1053/rapm.2003.50046. PMID: 12772135.
14. **Bauer ME, Arendt K, Beilin Y, Gernsheimer T, Perez Botero J, James AH, Yaghmour E, Toledano RD, Turrentine M, Houle T, MacEachern M, Madden H, Rajasekhar A, Segal S, Wu C, Cooper JP, Landau R, Leffert L.** The Society for Obstetric Anesthesia and Perinatology Interdisciplinary Consensus Statement on Neuraxial Procedures in Obstetric Patients With Thrombocytopenia. *Anesth Analg.* 2021 Jun 1;132(6):1531-1544. doi: 10.1213/ANE.0000000000005355. PMID: 33861047.