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Transient Cortical Blindness Following Amniotic Fluid Embolism

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Abstract

Postpartum cortical blindness is a rare complication. It may be due to the hypercoagulable state associated with pregnancy, eclampsia, and severe blood loss due to either intra or postpartum hemorrhage. A 42-year-old woman underwent a hysterectomy for disseminated intravascular coagulation (DIC) and massive hemorrhage in amniotic fluid during labor. Blindness occurred 8 hours after surgery. Fundus examination was normal. Magnetic resonance imaging (MRI) showed abnormal signals in bilateral ventricles and mild edema of the pituitary gland. After treatment with vitamin B1, mecobalamin, and methylprednisolone, light perception was restored on day 2, and visual acuity returned to normal on day 5. After 2 years of follow-up evaluation, both the patient and the newborn were normal.

Subject Areas

Gynecology & Obstetrics

Keywords

Vision Loss, Cortical Blindness, Amniotic Fluid Embolism

1. Introduction

Transient visual loss (cortical blindness) in the postoperative period is an uncommon complication with an incidence varying between 0.05% and 0.1% [1]. Although the exact cause is yet speculative, the most likely underlying reasons include middle-age pregnancy, diabetes mellitus, hypertension, smoking, cerebral hypoxia, and procedure duration [2]. The symptoms of blurring of vision and blindness may begin and develop during and after the procedure respectively, with a completely normal neurological examination [3]. The outcome is favorable, with

the return of sight within 24 - 48 hours [4]. We report a case of a patient who underwent a hysterectomy due to uncontrolled postpartum bleeding caused by an amniotic fluid embolism during delivery, but developed bilateral transient vision loss 8 hours after surgery. After 5 days of hormone therapy, vision was completely restored without any sequelae. We conclude that hormone therapy for transient vision loss after childbirth may help restore vision in patients.

2. Case Report

A 42-year-old woman (gravida 4, para 1-0-2-1) with an uneventful prenatal course arrived at the hospital after she felt pain for 1 hour at home at 40 weeks of pregnancy. A vaginal examination revealed that it was dilated 3 cm with an intact fetal membrane. She was well-oriented and her vital signs were stable (blood pressure 122/82 mmHg and pulse 84 beats/minute (bpm)). Fetal heartbeat at the time of presentation was 145 bpm. Vaginal examination after 0.5 h revealed 8 cm dilation. Suddenly, with the rupture of the fetal membrane, the parturient felt restlessness, dyspnea, and the sense of being close to death. The vital signs of the patient were 110/62 mmHg blood pressure, 85 bpm heart rate, and 100% oxygen saturation on high ventilatory parameters. Simultaneously, on abdominal examination, sustained uterine contraction and fetal heartbeat displayed bradycardia of 90 bpm. After 5 minutes, a baby girl, weighing 3.4 kg was born with an Apgar score of 3/1 min, 5/5 min, and 7/10 min. The baby was handed over to the pediatrician for resuscitation. Following the placenta delivery, a large amount of vaginal bleeding of about 800 mL was non-coagulated blood. The laboratory tests could not detect the prothrombin time (PT), The red blood cell (RBC) count dropped from 117×10^{12}/L to 71×10^{12}/L, and the platelet count dropped from 246×10^{9}/L to 196×10^{9}/L. The level of HCT declined from 0.375 to 0.220. The contractile agents we used included oxytocin 20 U, Capabectin 100 mg, and Carboprost Tromethamine 250 mg. A massive transfusion protocol was initiated with a total of 6 units of red blood cells (RBCs) 6 fresh frozen plasma (FFP), 20 cryoprecipitates, fibrinogen 3 g, and 1 treatment platelet volume. Further laboratory tests showed the presence of severe coagulopathy with values of PT > 200 (normal: 10.5 - 13.5) seconds, activated partial thromboplastic time (APTT) > 180 (normal: 21 - 36) seconds, International Normalized Ratio (INR) > 9.0, dimer 3.3 mg/L (normal < 0.2 mg/L), and fibrinogen < 0.6 (normal: 1.5 - 4.0) g/dL, and fibrinogen degeneration product (FDP) > 20 (normal: 0 - 4) μg/L. Since vaginal bleeding did not stop and hemoglobin decreased continuously, we inserted a Bakri postpartum balloon with 400 mL distilled water to compress the uterine cavity.

However, the uterine bleeding did not stop and a diagnosis of amniotic fluid embolism was considered. A hysterectomy was performed. After extensive transfusion and antiallergic therapy, the patient's vital signs were stable and 5680 ml of blood was lost during delivery. As the patient had lost 5680 ml of blood during delivery, a massive transfusion protocol was used, which included 6 units of red cell suspension, 600ml of fresh frozen plasma, 20 units of cryoprecipitate, 3 g of

fibrinogen, and a therapeutic amount of platelets. The patient was transferred to the intensive care unit (ICU) after 8 h, postoperative patients with bilateral vision disappeared. Ophthalmic examination showed normal body fundus and retina examination showed normal nervous system. As shown in **Figure 1** below, MRI inspection showed abnormal signals of the cerebral ventricles, so considering the ischemic lesions, cortical blindness was diagnosed after neurological and ophthalmic evaluation. Therefore, treatment included vitamin B1 100 mg IM QD, mecobalamin 1000 mg, and methylprednisolone 80 mg IV QD. Shadow tremors and light sensations were felt 18 hours after surgery, and full vision was restored within five days. After two days of observation, the patient regained full vision and other physical functions. She was discharged seven days after surgery and followed up for two years. Both maternal and newborn babies were normal.

3. Discussion

Postpartum cortical blindness is a rare complication [5]. It is thought to be caused by a hypercoagulable state associated with severe blood loss due to pregnancy, eclampsia, and postpartum bleeding [5]. A common pathological component is occipital cortical ischemia, which can be the result of a local event (bleeding or embolism) or a holistic process such as pre-eclampsia or eclampsia [6]. Pituitary apoplexy is not infrequently seen postpartum in patients with severe postpartum haemorrhage. Postpartum bilateral blindness can be due to vascular insult involving both the bilateral middle and posterior cerebral arteries (Gerstmann's syndrome) [7]. Amniotic fluid embolism is a very rare complication of pregnancy/puerperium that can vary from cardiac arrest and circulatory failure to death

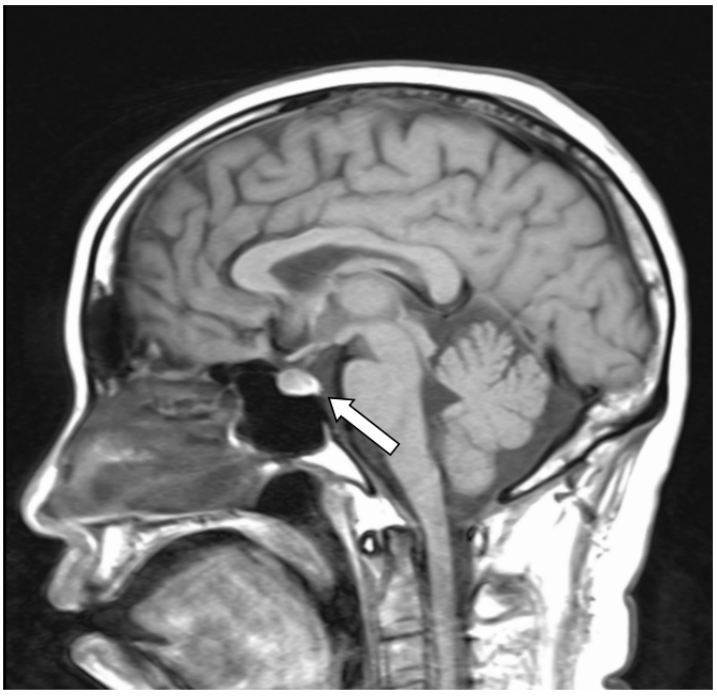

Figure 1. A representative view of MRI: enlarged pituitary gland.

to mild organ system dysfunction with or without a clotting disorder [8]. The perinatal mortality rate for AFE is approximately 25%, and 50% of surviving newborns are neurologically intact. Many survivors do show some degree of neurological damage [9]. This case is typical of AFE and cortical blindness etiology and clinical symptoms. The clinical manifestations of uneasiness, dyspnea, near-death, acute fetal distress, severe neonatal asphyxia after delivery, and postpartum hemorrhage with coagulation dysfunction suggest the diagnosis of amniotic fluid embolism. The patient was completely blind 8 hours after surgery, and there was no obvious retinal arteriovenous obstruction or organic change in the optic nerve. The three diagnostic criteria for cortical blindness include decreased vision, normal pupil response, and normal fundus [6]. Four hours (18 hours after surgery) after the use of hormones (vitamin B1, mecobalamine, and methylprednisolone) under the guidance of the neurosurgeon, the patient can feel shadow shaking and light, and complete vision is restored within 5 days without any sequelae. The mother suffered from the most serious and rare complications of obstetrics, amniotic fluid embolism and cortical blindness, but fortunately, both she and the newborn made a full recovery.

4. Conclusion

In analyzing this case, we learned that: 1) Immediate resuscitation of amniotic fluid embolism and an interdisciplinary approach, including active cardiopulmonary stabilization, hemodynamic correction, early use of vasopressants, correction of hemostatic disturbance, and undelayed cesarean section, played an important role in the outcome [8]. 2) Acute massive blood loss due to amniotic fluid embolism may lead to transient blindness due to pituitary apoplexy. 3) Hypovolemic shock leads to hypoxia and ischemia in the occipital cortex, resulting in postoperative blindness. Although previous studies have reported that cortical blindness does not require treatment, it can recover on its own in a short time or within a year [4]. Due to our lack of experience, under the guidance of the neurosurgeon, hormone therapy was actively used to restore the patient's vision. At present, there is no evidence that hormone therapy is a strong predictor of disease recovery, but the patient regained vision four hours after medication and did not experience any hormonal side effects at a 2-year follow-up. Therefore, we recommend that in the event of transient blindness caused by low volume perfusion shock due to postpartum hemorrhage or other bleeding, early use of hormone therapy is positive and effective in restoring vision.

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Conflicts of Interest

The authors declare no conflicts of interest.

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