J Anesth (1995) 9:354–356

Journal of
Anesthesia

Fat embolism syndrome due to femoral shaft fracture during pregnancy

TAE-YOOL CHUN, MUNETAKA HIROSE, and MASAO MIYAZAKI

Department of Anesthesia, National Maizuru Hospital, 2410 Yukinaga, Maizuru, Kyoto, 625 Japan

Key words: Fat embolism syndrome, Pregnancy, Bone fracture

Introduction

We report herein a rare case of fat embolism syndrome during pregnancy and discuss the effect of the pregnant state on this syndrome.

Case report

A 31-year-old female, 30 weeks' gestation, para 1, was scheduled to undergo an operation for left femoral shaft fracture sustained in a traffic accident. At admission, her consciousness was clear, and no other significant wound was recognized. She had no medical history and the course of pregnancy had been fair until the accident. Ritodrine (100 μg·min^{-1}) had been continuously administered for uterine tocolysis because of an increase in uterine tone since admission. On the second day after admission, profound respiratory failure developed suddenly in association with severe hypoxia (PaO_2 28 mmHg, $PaCO_2$ 28 mmHg at room air). Her consciousness level decreased, and she showed delirium. Bloody sputum, tachypnea (respiratory rate [RR] 45 breaths·min^{-1}), tachycardia ([HR] 146 beats·min^{-1}), and diffuse infiltrates demonstrated by chest radiogram indicated that the patient's condition was critical. We suspected that our patient had fat embolism syndrome (FES) because she exhibited many features of Guard's diagnostic criteria [1], including two of the major features (diffuse pulmonary infiltrates and loss of consciousness with deepening coma), one of the intermediate features (severe hypoxemia), and three of the minor features (tachycardia, elevation of body temperature and elevated erythrocyte sedimentation rate). We decided to perform an emergency cesarean section. The maternal vital signs at this time were blood pressure (BP) 120/50 mmHg, HR 160 beats·min^{-1}, and body temperature (BT) 38.0°C, although the basal fetal heart rate was almost normal (fetal heart rate: 160 beats·min^{-1}). The patient underwent a rapid-sequence induction with 250 mg thiopental and 7 mg vecuronium bromide intravenously. After tracheal intubation, anesthesia was maintained with isoflurane (0.6%) in oxygen under 8 cmH_2O positive end-expiratory pressure (PEEP) throughout the operation. Pulse oximetry showed 48% of SpO_2 just after induction. The premature baby (1790 g, Apgar score 4/7) was delivered, but died during severe idiopathic respiratory distress syndrome 12 h after the cesarean section, despite all attempts at treatment. The obstetricians found abruptio placenta during the operation. The induction–delivery time was about 3 min. SpO_2 increased gradually to 70%–80% soon after the delivery, and to 96% by the end of the operation. The data acquired via a Swan–Ganz catheter, which had been inserted during the operation, were 37 mmHg mean pulmonary artery pressure (MPAP) and 9 mmHg mean pulmonary capillary wedge pressure (PCWP), and arterial blood gas analysis revealed pH 7.12, $PaCO_2$ 62.2 mmHg, PaO_2 60.5 mmHg, BE −10, and SaO_2 79.2%. Medication after the delivery consisted of dobutamine (3 μg·kg^{-1}·min^{-1}), prostaglandin E_1 (0.05 μg·kg^{-1}·min^{-1}), and 1 g methylpredonisolone sodium succinate. The acidosis was partially compensated by injection of 60 ml of 7% sodium bicarbonate and hyperventilation. At the end of the operation, arterial blood gas analysis data were improved to pH 7.29, $PaCO_2$ 54.2 mmHg, PaO_2 69.4 mmHg, BE −1.1, and SaO_2 90.7%. The final operating room vital sign values

Address correspondence to: T. Chun, Department of Anesthesia, Shakaihoken Kobe Central Hospital, 2-1-1 Soyama-cho, Kita-ku, Kobe, Hyogo, 651–11 Japan
Received for publication on November 21, 1994; accepted on April 27, 1995

were BP 100/60 mmHg, HR 163 beats·min^{-1}, and BT 37.7°C. S–T segment depression, shown on the electrocardiogram, had continued since the operation. Administration of diltiazem (1.5 μg·kg^{-1}·min^{-1}) and digoxin (0.25 mg·day^{-1}) was started to improve myocardial ischemia and sustained tachycardia, respectively. On postoperation day (POD) 10, echocardiogram and radionuclide studies (^{99m}Tc and ^{201}Tl) were performed. Hypokinesis of the left ventricle wall and 55% ejection fraction were noted, and the findings of the radionuclide studies were consistent with typical myocardial ischemia. The patient was also under mechanical ventilation (SIMV, FIO_2 1.0–0.6; PEEP 8–5 cmH_2O) until extubation on POD 4. On POD 13, her general condition was improved with fairly reversed respiratory state and ischemic heart, and the operation for left femoral bone fracture was performed under general anesthesia (isoflurane with nitrous oxide and 200 μg fentanyl). The anesthetic course was uneventful, and the intraoperative blood gas analysis findings were fair.

Discussion

Although the mechanism of FES has not yet been clarified, two hypotheses regarding this mechanism have been presented. Fat emboli may mechanically block capillaries in the lungs. However, the medullary bone content is of insufficient volume to produce widespread pulmonary microvascular embolization. An alternate hypothesis is that fatty acids, especially oleic acid, derived from fat emboli are toxic to the parenchyma of the lung and may lead to disruption of its capillary network [1–3].

In pregnancy, we believe, two factors could worsen the respiratory state of a patient with FES. One is the maternal hormonal and metabolic state. It is known that human placental lactogen (hPL), which is produced at the syncytium of the placenta, enhances lipolysis and increases the free fatty acid level in the maternal blood. The hPL concentration in the blood increases in proportion to the enlargement of the placenta [4], and is highest at term. At that time the serum total lipid level is elevated by as much as 50% and the serum level of free fatty acids by 60% compared with those in the non-pregnant state [5]. The hPL level declines rapidly after the birth and is not detectable 7 h after the end of labor. Furthermore, lipoprotein lipase activity, which transfers free fatty acids in the bloodstream into the tissues, is lower in pregnant women. The hormones and enzymes released from the placenta (hPL, insulinase and progesterone) also have an antagonistic effect on insulin, and this decreases the rate of removal of fatty acids into the extrahepatic tissues [6,7]. Also, blood in a parturient is apt to coagulate and lead to disseminated intravascular coagulation. Therefore once FES was established a parturient could deteriorate very rapidly. A pregnant woman is also disadvantaged by the effect of pregnancy on her lungs. It is generally recognized that in a parturient, functional residual capacity (FRC), expiratory reserve volume, and residual volume are decreased. These changes are related to the cephalad displacement of the diaphragm by the large gravid uterus. The respiratory mucous membrane also becomes more congested, edematous, and friable. These changes in the lungs of a pregnant woman will tend to weaken her respiratory system, and under the above-mentioned conditions she might be at high risk of respiratory failure.

In our case, the parturient's respiratory state had deteriorated suddenly at midnight. We tried to improve here condition by the cesarean section prior to the orthopedic operation. Delivery would reduce her enlarged uterus and stop the release of placental substances. Her fractured left leg was thought not to be contributing to her worsening condition because FES was almost established. Therefore, repairing the fracture seemed to be of secondary importance in her critical condition. In general, a fractured shaft should be set very exactly, and an emergency orthopedic repair is controversial [8–10]. Repair surgery may release a large quantity of fatty acids all at once on deflation of the tourniquet. Case reports concerning FES during pregnancy are rare [3]. The relation between FES and pregnancy should be examined in detail.

FES must also have had some influence on the infant's respiratory distress. The mother had had an uneventful pregnancy, but the obstetricians found abruptio placenta after the delivery. One-third of the placenta on the mother's side showed infarction or necrosis. The infant failed to respond to any treatment, including surfactant therapy. We suspected that FES, not transient hypoxia, was responsible. In studies on normal fetal heart rate just before cesarean section, it has been shown that a fetus occasionally shows a normal fetal heart rate pattern when it has adapted itself to hypoxia.

We administered dobutamine and prostaglandin E_1, both to reduce pulmonary hypertension, and methylpredonisolone sodium succinate to improve the ventilation–perfusion ratio, decrease shunt, and increase PO_2 [11].

In summary, we report a rare case of FES in a pregnant woman involved in a traffic accident. FES has graver consequences in a pregnant patient than in a non-pregnant patient. It is not clear why the incidence of FES is rare in pregnant women. Detailed examination of parturients with a long bone fracture is needed. However, prompt and careful therapy, including emergency delivery, is required for pregnant patients

with FES because of their hormonal and respiratory state.

References

1. Guard AR, Wilson RI (1974) The fat embolism syndrome. J Bone Joint Surg 56B:408–416
2. Peltier LF (1988) Fat embolism. Clin Orthop 232:263–270
3. Moore P, James O, Saltos N (1981) Fat embolism syndrome. Aust N Z J Surg 6:546–551
4. Singer W, Desjardins P, Friesen HG (1970) Human placental lactogen. Obstet Gynecol 36:222–232
5. Potter JM, Nestel PJ (1979) The hyperlipemia of pregnancy in normal and complicated pregnancies. Am J Obstet Gynecol 133:165–170
6. Fabian E, Stork A, Kucerova L, Sponarova J (1968) Plasma levels of free fatty acids, lipoprotein lipase, and postheparin esterase in pregnancy. Am J Obstet Gynecol 7:904–907
7. Biale Y (1985) Lipolytic activity in the placentas of chronically deprived fetuses. Acta Obstet Gynecol Scand 64:111–114
8. Sugai K, Sugai Y, Aoki H, Honjo M (1991) A case of fat embolism syndrome after femoral shaft fracture (in Japanese). Seikeigeka (Orthop Surg) 42:391–394
9. Shibata S, Yoshioka T (1992) Fat embolism syndrome after the fracture of the femoral neck (in Japanese). Seikeigeka (Orthop Surg) 43:532–534
10. Talucci RC (1983) Early intramedullary nailing of femoral shaft fracture; a cause of fat embolism syndrome. Am J Surg 146:107–111
11. Fischer JE, Turner RH, Herndon JH, Riseborough EJ (1971) Massive steroid therapy in severe fat embolism. Surg Gynecol Obstet:667–672