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Cavernous transformation of the portal vein complicating pregnancy

Portal venin kavernöz transformasyonu ve gebelik

To the Editor

Cavernous transformation of the portal vein (CTPV) is a very rare digestive tract disorder resulting from extrahepatic portal vein thrombosis and development of collateral venous circulation1. Tuncer et al. reported on a 45-year-old parturient with two giant hemangiomas of the liver that caused CTPV (2). The patient described by the authors had no complaints other than a mass and slight pain in the right upper quadrant. There were no abnormalities in laboratory values. Delivery was accomplished without complication.

As CTPV may pose a number of other rare complications, which might be of interest to gastroenterologists, I would like to comment on our recent experience regarding a pregnant patient with an underlying congenital CTPV.

At the University of California, San Diego, we encountered a 26-year-old pregnant patient with congenital CTPV associated with persistent consumptive coagulopathy and chronic thrombocytopenia who was admitted to obstetric service at 37 weeks' gestation for evaluation of worsening pregnancy-induced hypertension (PIH) (3).

At the age of 17 months, the patient was diagnosed with splenomegaly and subsequently underwent exploratory laparotomy with portal venogram. This diagnostic procedure demonstrated portal hypertension secondary to CTPV due to portal thrombosis of unknown etiology, presumably congenital in nature.

The consultation with the gastroenterologist and the hematologist revealed ongoing chronic consumption of coagulation factors and platelets, resulting in generalized mild decrease in activity of the coagulation factors (levels of 68%, 42%, 84%, 40%, 52% and 39% for factors 2, 5, 7, 8, 9 and 12, respectively) and mild thrombocytopenia (average platelet level of 60,000/mm^3).

Her admission blood pressure was 160/105 mmHg, pulse rate 65 beats/minute and respiratory rate 17 breath/minute. An ultrasound revealed a single fetus in cephalic presentation without any gross abnormalities. Fetal heart rate was 140 beats/min and reactive. The laboratory findings (normal values at our hospital are in parenthesis) were as follows: hemoglobin level 13.3 g/dl (12-17 g/dl), platelet count 68,000/mm^3 (130,000–400,000/mm^3), prothrombin time 14 sec (9-12 sec), partial thromboplastin time 45 sec (25-33 sec), and bleeding time as conducted in our laboratory of 5 min. (up to 7 min.). The liver enzymes were all normal and included alanine aminotransferase (ALAT) 45 units/L (30-130 units/L), aspartate aminotransferase (ASPAT) 15 units/L (10-45 units/L), total bilirubin level 1.0 mg/dl (<1.2 mg/dl), albumin 4.5 g/dl (3.3-5.0 g/dl), and total protein 7.6 g/dl (6.8-8.9 g/dl).

After five days of hospitalization, the decision was made to proceed with cesarean section for worsening PIH. On the day of cesarean section, platelet count was 67,000/mm^3. Her physical examination conducted prior to surgery and the administration of anesthesia was negative for any evidence of abnormal hemostasis. Single dose spinal anesthesia was safely conducted for an uneventful cesarean delivery. The postoperative course including neurological evaluation was uneventful.

The minimum platelet count recommended to safely conduct regional anesthesia in pregnant women is unknown. Bromage advised against regional anesthesia in any patient whose platelet count was below 100,000/mm^3; however, this recommendation may be "opinion" rather than based on factual data. Others have remained similarly cautious

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(4, 5). Although the coexistence of two disorders affecting platelet count in our patient, CTPV with persistent consumption of coagulation factors and platelets and PIH, posed an initial dilemma regarding the safety of spinal anesthesia, the additional platelet count obtained in the immediate period prior to delivery showed no decrease in platelet level, and her coagulation status as judged by a hematologist was adequate to safely undergo both surgery and neuraxial anesthesia. It is also important to emphasize that the thrombocytopenia in this patient could be simply due to hypersplenism caused by CTPV–induced splenomegaly. As both PIH and CTPV (hypersplenism) may lead to thrombocytopenia, it is important to exercise caution with neuraxial blocks (e.g., spinal and/or epidural anesthesia) in these patients.

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