Postpartum Hemolytic Uremic Syndrome Following Abruptio Placenta: Report of a Case

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Abstract: Postpartum hemolytic uremic syndrome (HUS) is an unusual complication that presents with microangiopathic hemolytic anemia, thrombocytopenia, and acute renal failure after delivery. In this report, we describe a 32-year-old patient (gravida 3, para 1, artificial abortion 1) who developed postpartum HUS following abruptio placenta. After cesarean delivery due to abruptio placenta, the patient developed acute renal failure, microangiopathic hemolytic anemia, and thrombocytopenia followed by hypertension. Plasma exchange led to recovery from thrombocytopenia and improvement in renal function. This case highlights the importance of observation of peripheral blood smears in patients with abruptio placenta who develop thrombocytopenia after delivery.

Case Report

A 32-year-old woman (gravida 3, para 1, artificial abortion 1) was admitted because of massive vaginal bleeding at 34 weeks' gestation. No hypertension, proteinuria, edema or sudden increase in weight was noted during serial antepartum examinations. The patient's blood pressure was 86/50 mmHg at admission. Clinical findings included external uterine bleeding, uterine hypertonus and hyperactivity, and fetal death. Laboratory studies disclosed a hemoglobin (Hb) level of 12.4 g/dL and a platelet count of 53,000/µL. The patient underwent cesarean section because of abruptio placenta with fetal death in utero. The body weight of the fetus was 2.1 kg. Sections of the placenta revealed intense hemorrhage with blood clot formation and hyaline necrosis, which was consistent with abruptio placenta. The patient's blood pressure returned to 100/76 mmHg 6 hours postoperatively.

Twenty-four hours after cesarean section, the patient developed anemia (Hb, 6.8 g/dL) and thrombocytopenia (platelet count, 22,000/µL). Packed red blood cells 4 U and platelets 18 U were given, after which the patient's blood pressure rose to 150/72 mmHg. She was then transferred to National Taiwan University Hospital for further evaluation and treatment on the third postpartum day. There was no more vaginal bleeding after arrival. No purpura was found on physical examination. Repeated blood examination revealed anemia (Hb, 6.0 g/dL) with normal reticulocytes (2.44%), thrombocytopenia (platelet count, 16,000/µL), normal prothrombin time (13.7 sec) and activated partial thromboplastin time (44.9 sec), negative plasma, protamine, paracoagulation.
Hemolytic Uremic Syndrome Following Abruptio Placenta

A 28-year-old, gravida 3, para 2, 34-week gestation patient presented in labor with a history of abdominal pain and vaginal bleeding. She was transferred to our hospital on the 37th week of pregnancy due to severe anemia and coagulopathy. The patient was admitted in preeclampsia with preterm labor. The diagnosis of abruptio placenta was confirmed by ultrasound imaging.

(3p) test, mild elevation of fibrin-degradation products (10–20 µg/mL), and negative direct and indirect Coombs' tests. Blood chemistry revealed marked elevation of serum lactate dehydrogenase (LDH) level (5,830 U/L), mild elevation of serum glutamate-oxaloacetate transaminase (73 IU/L), but normal serum glutamate-pyruvate transaminase level (GPT; 2 IU/L). The blood urea nitrogen level was elevated to 68.3 mg/dL and serum creatinine level was 6.0 mg/dL, with daily urine output of 3,850 mL. Serum bilirubin was mildly elevated (total bilirubin, 1.35 mg/dL).

Fragmented red blood cells (schistocytes and helmet forms) and poikilocytosis were found in the blood smears (Fig. 1). Urinalysis showed microscopic hematuria and proteinuria. Marked hypertension of up to 209/103 mmHg was noted, and was controlled with labetalol from the third to the sixth postpartum days. Hb and platelet levels remained low, even though no abnormal vaginal bleeding was noted after admission, and packed red blood cells were repeatedly transfused.

Because HUS was highly suspected, the patient received six courses of plasma exchange starting on the fifth postpartum day. Hemodialysis was also performed once on the fifth postpartum day. Dramatic improvements in renal function and the hemogram were noted after plasma exchange (Fig. 2). On the 10th postoperative day, creatinine level was 4.33 mg/dL, platelet count was 427,000/µL, Hb was 10.6 g/dL, and LDH was 1,188 U/L. The creatinine level dropped further to 2.32 mg/dL 4 days later. Renal biopsy on the 12th postpartum day showed focal increase in mesangial cellularity and matrix, with swollen endothelium and focal obliteration of capillary lumina. Periodic acid-Schiff stain revealed focal, double-contour appearance of glomerular basement membrane. Electron microscopy showed endothelial change with sub-endothelial loosening and an appearance compatible with thrombotic microangiopathy (Fig. 3). No neurologic symptoms or signs were noted during the hospitalization. The patient was discharged in a stable condition on the 19th postpartum day. Serum creatinine level was 0.67 mg/dL 1 month after discharge and on follow-up 6 months later. The patient remained normotensive with no signs of impaired renal function.

Discussion

HUS seems to occur under certain stresses, including infections, autoimmune diseases, intake of certain medicines,
pregnancy, or even idiomatically. In pregnant women with thrombocytopenia, it is not easy to distinguish HUS from thrombotic thrombocytopenic purpura (TTP) and preeclampsia, specifically the variant called HELLP syndrome (hemolysis, elevated liver enzymes and low platelet count). HUS and TTP have similar clinical pictures of hypertension, hemolysis, elevated liver enzymes, low platelet counts, and renal failure [3]. However, TTP usually occurs in the first trimester, while HUS is typically seen in the peripartum or postpartum period [8]. Unlike TTP, HUS has few neurologic deficits but usually shows severe renal involvement. The absence of impaired consciousness and the presence of acute renal failure in our patient favored the diagnosis of HUS rather than TTP.

This patient lacked clinical and laboratory evidence of coagulopathy during progressive anemia and thrombocytopenia after delivery. Furthermore, the findings of normal GPT level and the development of progressive renal dysfunction and thrombocytopenia, as well as hemolysis after cesarean section, made the diagnosis of HELLP unlikely. On the other hand, persistent renal failure, thrombocytopenia, microangiopathic hemolytic anemia with fragmented red blood cells in peripheral blood smear after delivery, and the histopathologic findings of the renal biopsy, supported the diagnosis of HUS. Also, an accurate diagnosis to differentiate HUS from preeclampsia is required because early, intensive treatment can reduce the associated morbidity and mortality of HUS by as much as 90% [7].

Remuzzi et al first demonstrated how plasma exchange could successfully correct thrombocytopenia in adult patients with HUS [9]. The patient in our report also showed dramatic improvement after plasma exchange. Pajor et al reported a similar case of postpartum HUS following abruptio placenta and proposed that placental substances released into the maternal circulation during and after separation of the placenta may be a factor in triggering HUS; this patient also responded well to plasma exchange, with rapid improvement of clinical and laboratory profile [10]. Dashe et al reported the long-term consequences in patients with thrombotic microangiopathy in pregnancy [2]. In their review of 13 cases of TTP or HUS associated with pregnancy, at least one recurrence of thrombotic microangiopathy appeared in half of the patients. Three women suffered from multiple relapses, and one died. In our patient, there was no residual renal insufficiency or hypertension. Sibai et al reported that the prognosis was most favorable in patients with reversible acute tubular necrosis without preexisting chronic hypertension [11].

In summary, a high index of suspicion is needed to recognize thrombotic microangiopathy in pregnancy, as many women with this disorder present initially with symptoms that are non-specific or may be confused with preeclampsia. If delivery does not ameliorate the condition after 72 hours, the diagnosis of TTP or HUS must be considered. Once the diagnosis is made, prompt and aggressive therapy with plasma exchange is indicated.

References