Conservative Treatment of Huge Hepatic Subcapsular Hematoma Complicated with Hepatic Infarction after Cesarean Section Caused by HELLP Syndrome – a Case Report and Literature Review

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ABSTRACT

Hepatic subcapsular hematoma and hepatic infarction in labor are mostly secondary to HELLP syndrome and preeclampsia. There are few reported cases with a complicated diagnosis and treatment and high mortality. Here, we present a case of a huge hepatic subcapsular hematoma complicated with hepatic infarction after cesarean section that was secondary to HELLP syndrome and the patient was treated conservatively. Further, we have discussed the diagnosis and treatment of hepatic subcapsular hematoma and hepatic infarction caused by HELLP syndrome.

Background

Hypertensive disorders in pregnancy are among the leading causes of maternal and perinatal mortality, with a global prevalence of 2–8 %. However, maternal deaths caused due to hypertensive disorders is nearly 26 % in Latin America and the Caribbean, and 9 % in Africa and Asia [1]. HELLP syndrome is considered to be a severe complication of hypertensive disorders in pregnancy [2, 3]. The "HELLP" acronym was introduced in 1982 by Weinstein to describe the hemolytic syndrome, elevated liver function tests and low platelet levels that occur during late pregnancy [4]. In recent years, with the gradual increase in the number of cases, there have been standardized criteria for the diagnosis and treatment of HELLP syndrome, which is still challenging to treat due to its complex pathophysiological mechanisms and the multiple organ damage it causes, especially for rare complications such as hepatic hematoma and hepatic infarction. The incidence is approximately in the presence of preeclampsia and HELLP syndrome, which increases to one case in every 200 to 2000 cases [5].

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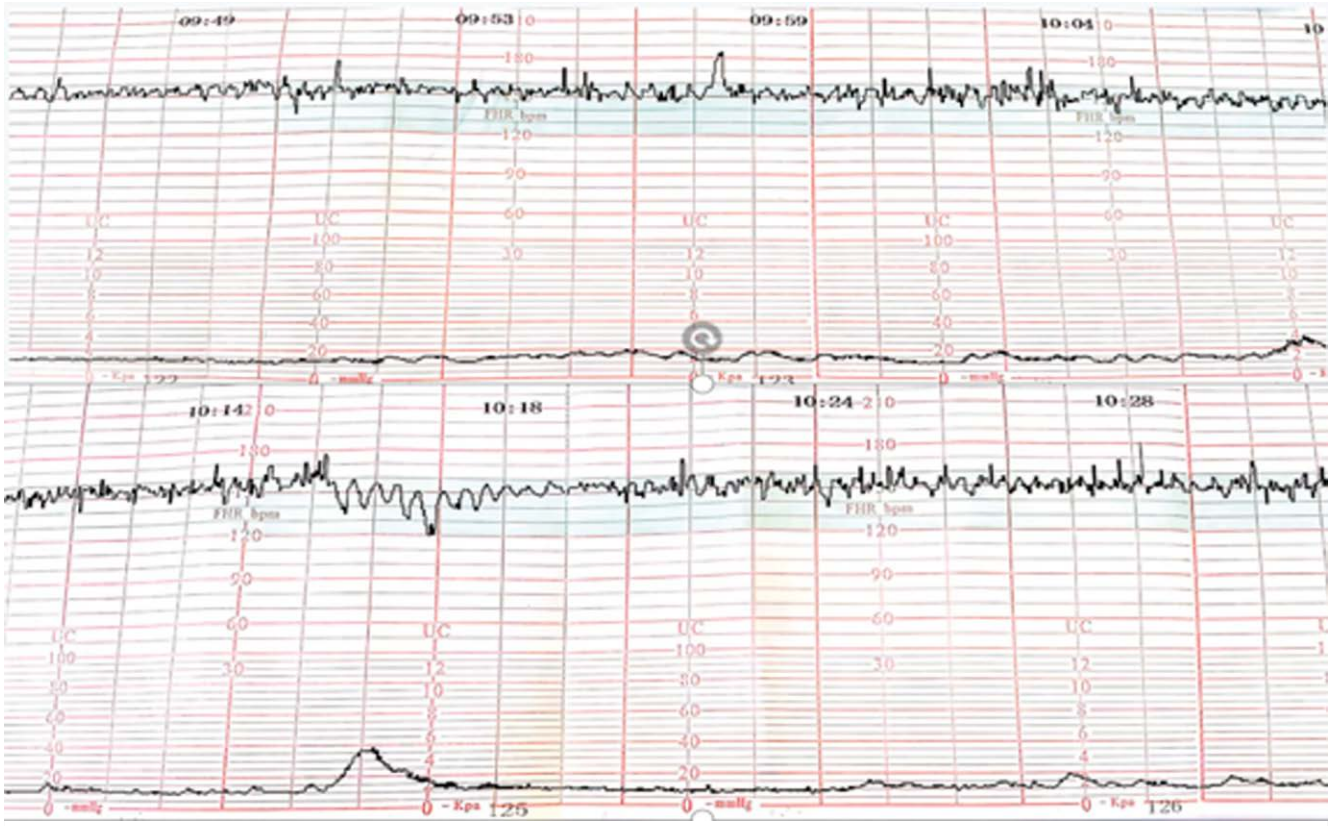
Case report

A 31-year-old pregnant woman with regular menstruation who denied a history of hypertension and abnormal pregnancy had an elevated blood pressure of 160/100 mmHg at the first obstetric examination in 1 + months of menopause. Further, it was reported that the pregnant woman did not receive antihypertensive treatment and confirmed that her blood pressure could return to normal without any medication. However, from 29 weeks onwards, the highest blood pressure reached 171/118 mmHg and urinary protein levels were 2 + . It was considered a case of preeclampsia complicated with chronic hypertension. Thus, antihypertensive and fetal lung maturation treatments were given. It was observed that the patient's blood pressure was stable at 130–145/85–93 mmHg until 35 weeks of pregnancy. However, she developed upper abdominal discomfort from 35 + weeks onwards and had a maximum blood pressure of 212/123 mmHg. The blood test reports revealed the following: white blood cell (WBC) 13.65×10^9/L, hemoglobin (Hb) 134 g/L, hematocrit (HCT) 35 %, platelet (PLT) 88×10^9/L, alanine aminotransferase (ALT) 92 U/L, aspartate aminotransferase (AST) 129 U/L, uric acid 557 µmol/L, and urine protein 2 + . Fetal ultrasound was performed and indicated elevated fetal umbilical artery flow spectrum values and decreased pulsatility index (PI) values in the middle cerebral artery (RI: 0.69, PI: 1.21). The estimated fetal weight was 1600 g. The non-stress test (NST) was a suspicious reactive type (▶ **Fig. 1**). The reports indicated severe preeclampsia with chronic hypertension, HELLP syndrome, intrauterine distress, and intrauterine growth restriction (IUGR). Therefore, a cesarean section was performed the same day at the obstetric hospital, and the operation went well. A baby boy was delivered weighing 1650 g with an APGAR score ranging from 8–9 and the pH value of the cord blood was 7.25. The premature infant was transferred to the neonatology department for further treatment. In addition, the delivered placenta was sent for pathological examination (▶ **Fig. 2**). After the operation, the patient was transferred to the intensive care unit (ICU) and continued receiving symptomatic and supportive care such as antihypertensive, antispasmodic, analgesic, and sedative treatment to prevent infection and thrombosis and to strengthen uterine contractions. However, on the postoperative and the first day thereafter, the patient suddenly developed pain in the right shoulder, abdominal distension, weakness, cold sweat, and other discomforts with a heart rate of 110–136 beats/min, and her blood pressure dropped to 50–62/40–45 mmHg. The emergency blood test revealed WBC 17.13×10^9/L, Hb 105 g/L, HCT 28.3 %, PLT 52×10^9/L, blood potassium 7.1 mmol/L, ALT 797 U/L, AST 1128 U/L, PT 17.5 s, APTT 26.6 s, FBG 4.2 g/L, INR 1.55, and D-dimer 12 228 ng/mL. Further, the ultrasound suggested a mixed echogenic sonogram between the subdiaphragm and liver, whose nature was to be investigated. Furthermore, the computerized tomography angiography (CTA) suggested that a range of mass-like high and low mixed-density foci were seen under the envelope of the right lobe of the liver ranging from about 18.5 × 4.3 cm, while the right lobe of the liver was reduced in volume and hypodensity. These reports indicated the possibility of HELLP syndrome with hepatic injury changes (huge hematoma under the liver envelope and infarction of the right lobe of the liver). Thus, the patient was diagnosed with severe preeclampsia with chronic hypertension, and HELLP syndrome, leading to huge hepatic hematoma and hepatic infarction after cesarean section. Next, she was transferred to the ICU of the Third Hospital of Guangzhou Medical University on the third postoperative day after shock correction.

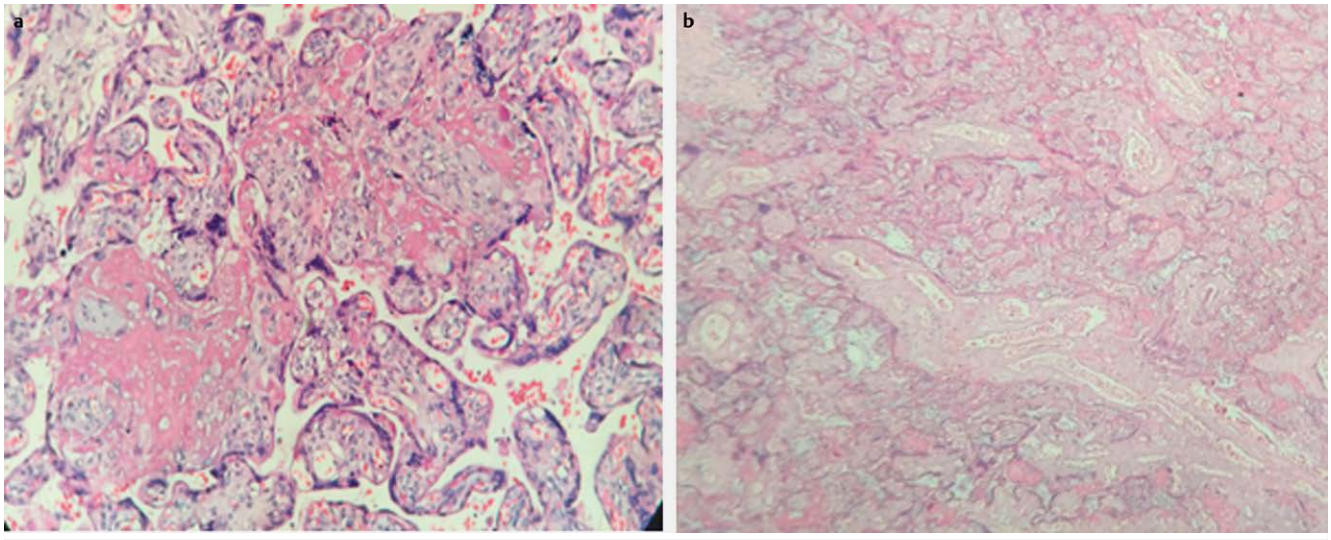
On admission, the patient presented with an acute appearance of illness, blood loss, decreased breath sounds, and epigastric pain. Laboratory findings suggested that ALT had risen to 7056 U/L, AST 14064 U/L, total bilirubin 24.0 (µmol/L), and platelets decreased to 46.00 (10^9/L). In addition, the right liver had a mixed perihepatic echogenic area (129 × 46 mm) with hematoma. Furthermore, computerized tomography (CT) suggested hepatic parenchymal echogenic heterogeneity and multiple hypoechoic areas in the right liver (▶ **Fig. 3a**). After admission, a multidisciplinary consultation was held immediately, and the patient's critical condition was discussed. The treatment plan was conservative, including artificial liver enzyme reduction to restore liver cell function, plasma replacement, anti-infection, transfusion of red blood cells, plasma, platelet concentrate, blood volume supplementation, and prevention of lower limb venous thrombosis. During this period, there were changes in the patient's condition, such as infection, decreased hemoglobin, gastrointestinal dysfunction, increased peritoneal fluid, and blood coagulation function disorders. After several multidisciplinary discussions, a conservative treatment strategy was followed as dynamic CT monitoring of the size of hematoma and infarction showed no significant increase (▶ **Fig. 3b–f**). With changes in the antibiotic regimen, blood transfusion, and peritoneal puncture and drainage, the patient's condition gradually improved, and her blood pressure stabilized in the normal range. Values were PLT 120.00 (10^9/L), urea 12.78 (mmol/L) ↑, creatinine 79 (µmol/L) ↑, total bilirubin 21.2 (µmol/L) ↑, albumin 32.1 (g/L), ALT and AST were normal, and D-dimer 2595 ng/mL. Further, the right hepatic perihepatic mixed echogenic area was 108 × 80 mm and the peritoneal fluid had disappeared. Thus, the patient was transferred to the general ward for further observation and treatment, discharged on the 37th postoperative day, and continued to be followed up in the outpatient clinic. The blood test results during hospitalization are listed in ▶ **Table 1**. At the time of publication, the patient was in better condition and continued to treat for hypertension and proteinuria in an outpatient clinic, and the size of hepatic hematoma was monitored until it disappeared completely.

Summary of published cases

We conducted a literature survey and looked for similar cases [6–9] to identify the special features of our case as well as the characteristics of HELLP syndrome that led to hepatic subcapsular hematoma. The summary of these cases is shown in ▶ **Table 2**. The available literature suggested that all eight patients were diagnosed with hepatic subcapsular hematoma secondary to HELLP or preeclampsia, and imaging examinations were favorable for diagnosing liver hematoma. Spontaneous hepatic rupture occurred in five cases, including four cases of shock, which were accompanied by decreased hemoglobin and platelets. The characteristics of the elevated liver enzymes, including the case of maternal hepatic hematoma removal, but died of multiple organ failure caused by loss of blood and hepatic failure, the case of maternal liver transplan-



▶ **Fig. 1** Non-stress test (NST) before the operation, baseline fetal heart rate is 140 beats/min, short variation exists, no significant acceleration, and fetal heart rate monitoring suspicious response type.

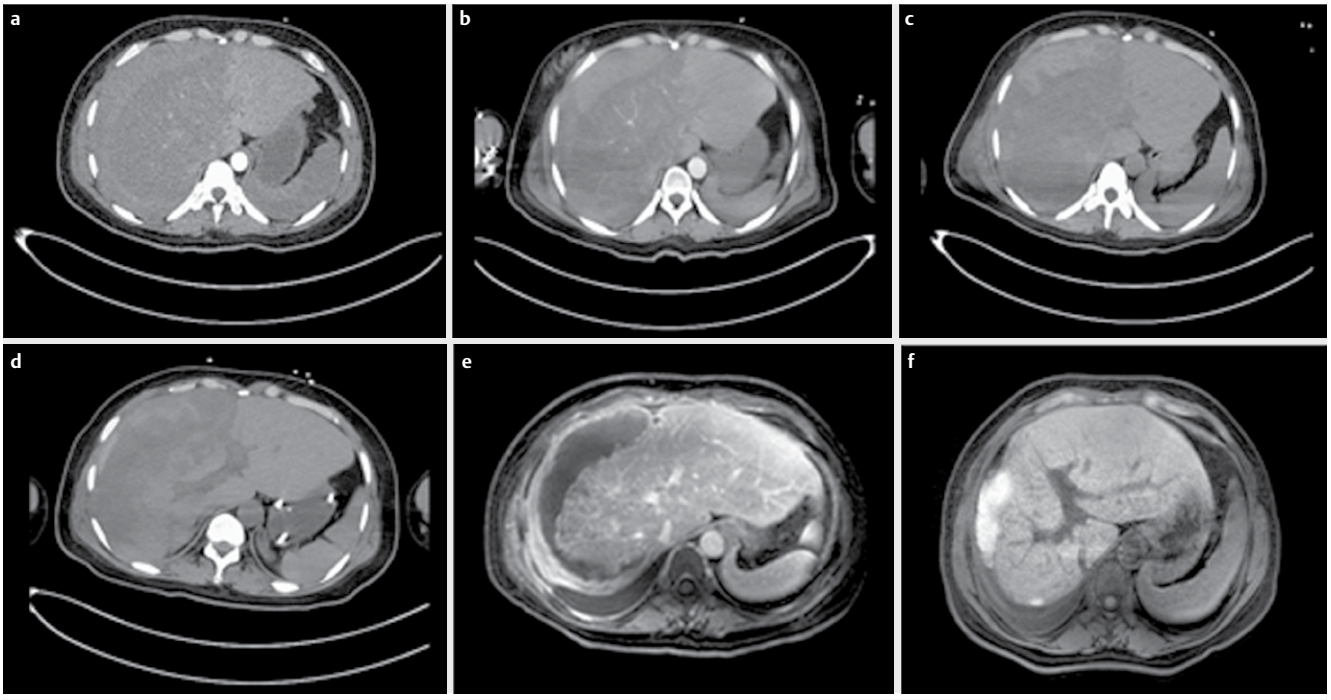


▶ **Fig. 2** **a** The placental villi narrow space and cellulose deposits. **b** The placental villi infarction.

tation and rescued, the two cases of liver tamponade hemostasis and open operation for many times.The four cases without rupture were treated conservatively and successfully. Preeclampsia and gestational age determined the outcome of the baby.

Pathogenesis and pathology

The pathogenesis of subperitoneal hematoma and hepatic infarction associated with HELLP syndrome and preeclampsia remains unclear. However, the pathogenesis involves mechanisms such as increased inflammatory factors for activation of the complemen-



▶ **Fig. 3** **a** Computed tomography (CT) of the hepatic subcapsular hematoma and infarction after the 3rd day after cesarian section (CS). **b** CT of the hematoma and infarction after the 5th day of CS. **c** CT of the hematoma and infarction after the 8th day of CS. **d** CT of the hematoma and infarction after the 16th day of CS. **e** CT of the hematoma and infarction after the 35th day of CS. **f** CT of the liver after the 77th day of CS.

▶ **Table 1** Blood and related test results.

Discharge	Day of surgery	Day of transfer	At
		(3 days post-op)	(37days post-op)
Leukocytes (10^9/L)	13.65	35.75	5.43
Hemoglobin (g/L)	134	83	66
HCT (%)	35%	28.3%	25.2%
Platelets (10^9/L)	88	46	291
INR	1.48	1.55	1.23
D dimer (ng/ml)	12228	15012	1828
Fibrinogen (g/l)	2.65	4.2	3.78
ALT (u/l)	92	7056	10.9
AST (u/l)	129	14064	28
Pseudo-Cholinesterase (U/L)	3566	3628	3458
Albumin (g/L)	32.8	19.2	27.2
Calcitoninogen (ng/ml)	9.23	4.2	1.3
Creatinine (umol/L)	161.2	162	53
Urea (mmol/L)	16.8	5.58	2.6
Uric acid (umol/L)	557	1054	462
Urine protein	2+	2+	2+
Hematoma size on ultrasound (mm*mm)	–	129*46	108*80

tary system, thrombotic microangiopathy, hemolytic microangiopathic anemia [10], microangiopathy leading to ischemic lesions in the liver [11], microthrombosis and endocytic injury causing hepatic sinusoidal obstruction [12], and further development of hepatocellular necrosis and hemorrhage, which are still not fully understood. Moreover, neovascularization occurs in the involved paren-

▶ **Table 2** Summary of published cases.

	Case1	Case2	Case3	Case4	Case5	Case6	Case7	Case8
	hepatic hematoma gram	(Surviving)	(Ruptured)	(Subcapsular hematoma)	(Subcapsular hematoma)	(Subcapsular hematoma)	(Subcapsular hematoma)	(Subcapsular hematoma)
Age	30	38	27	33	26	26	30	29
Gravida/parity	1/0	2/0	–	2/1	–	–	2/1	1/0
Gestational age at presentation	Full-term pregnancy	29 + 5	34 + 4	37	36	22	40	34
Presenting symptoms	Lethargy	Sudden-onset right upper quadrant abdominal pain, visual disturbance and frontal headache	Mild left shoulder and back pain	Right upper quadrant pain	Right upper quadrant pain and tachycardia	Right upper quadrant abdominal pain	Oliguria, hypotension, and an acute abdomen	Nausea, vomiting, and diarrhea
Initial blood pressure	–	140/80 mmHg	–	–	–	–	–	Normal
Peak blood pressure	–	170/90 mmHg	142/88 mmHg	–	–	180/95 mmHg	–	–
Shock	Yes	Yes	No	No	No	No	Yes	Yes
Minimum blood pressure	65/25 mmHg	85/35 mmHg	–	–	–	–	–	–
Hematocrit nadir	–		–	–	23 %	15.3 %	22 %	
Hemoglobin nadir	–	71 g/L	–	–	–	–	–	–
Platelets at presentation	–	–	143	33.000/µL	143.000/µL	–	–	–
Platelet nadir	–	70	80	–	–	35.000/µL	25.000/µL	44.000/µL
Alanine aminotrans-ferase (ALT) at presentation	–	271 U/L	–	–	–	–	–	–
Aspartate aminotrans-ferase (AST) at presentation	–	853 U/L	208 U/L	–	–	–	–	–
AST peak	–	–	258 U/L	116 U/L	573 U/L	2030 U/L	990 U/L	730 U/L
Creatinine at presentation	–	–	79,6 µmol/L	–	–	–	–	–
Imaging diagnosis of liver hematoma	Abdominal ultrasound	–	Computed tomography (CT) scan	Computed tomography (CT) scan	Computed tomography (CT) scan	Computed tomography (CT) scan	Computed tomography (CT) scan	–
Mode of delivery	Emergency cesarean section	Emergency cesarean section	Emergency cesarean section	Emergency cesarean section	Emergency cesarean section	Induction of labor prostaglandin	Cesarean section	Emergency cesarean section
Hepatic rupture	Yes	Yes	Yes	No	No	No	Yes	Yes
Birth weight	–	–	–	–	–	–	–	–

▲ **Table 2** Continued.

	Case1	Case2	Case3	Case4	Case5	Case6	Case7	Case8
	hepatic hematoma gram	**(Surviving)**	**(Ruptured)**	**(Subcapsular hematoma)**	**(Subcapsular hematoma)**	**(Subcapsular hematoma)**	**(Subcapsular hematoma)**	**(Subcapsular hematoma)**
Operative or non-operative management	Evacuation of the hepatic hematoma	Full laparotomy and tamponade with gauze to stop bleeding	Non-operative manage-ment	Non-operative management	Non-operative management	Non-operative management. Thoracic and abdominal puncture drainage	Full laparotomy and tamponade with gauze to stop bleeding	Liver transplant
Estimated blood loss	–	2300 ml	1000 ml	–	–	–	–	3000 ml
Other complications	–	Supraven-tricular tachycardia, persistent pyrexia, Type II respiratory failure	–	–	–	–	–	Acute renal failure and pleural effusions develop
Maternal death	Yes	No	No	No	No	No	No	No
Fetal death	No	One twin survived	No	No	No	Yes	No	Yes

chyma, and the newly formed vessels are more likely to bleed during hypertensive episodes. Further, intrahepatic hemorrhage forms a subperitoneal hematoma that ruptures into the peritoneal cavity as it expands or is due to elevated blood pressure or minor trauma [10]. The liver tissue biopsies obtained in some cases revealed pathological features of periportal fibrin deposition, periportal hemorrhage, hepatic lobular necrosis and hepatocellular steatosis [13]. However, there was no statistically significant correlation between the severity of histological findings of periportal hemorrhage and fibrin deposition and clinical laboratory findings [9].

Clinical presentation

The clinical presentation of HELLP syndrome often includes right upper or epigastric pain (approximately 65 % of cases), nausea and vomiting (35 % of cases), and headache (30 % of cases) [14–16]. Moreover, headache, vision changes, and symptoms associated with thrombocytopenia such as mucosal bleeding, hematuria, petechial hemorrhage, or petechiae have also been reported [17]. However, hypertension is present in most patients but may be absent in 12–18 % of cases. Similarly, proteinuria may be found in most cases but may not be present in 13 % of patients [10].

Hepatic subcapsular hematoma and rupture are most commonly seen in the right lobe of the liver [9, 10, 18]. Henny et al. (1982) reported that the right lobe of the liver was involved 75 % of the time, both lobes were involved 14 % of the time, and only 11 % of the time was the left lobe involved [3, 6]. Therefore, the right upper abdominal or epigastric pain is the most common symptom of hepatic subcapsular hematoma. This pain may radiate to the right shoulder and is thought to be caused by distension of the pericardium or possible hepatic dilatation due to obstruction of blood flow in the hepatic sinusoids, resulting in right upper abdominal or epigastric pain [9]. It may also include severe shoulder pain, nausea, vomiting, and abdominal distention. However, the incidence of pericardium rupture will lead to severe bleeding, showing signs of hypovolemic shock, such as severe hypotension, anemia, and a large accumulation of fluid in the abdominal cavity [19].

Examination and imaging

Patients with hepatic subcapsular hematoma are required to reduce activity to avoid increased bleeding. It is recommended that bedside imaging be performed as much as possible, and bedside ultrasound is more convenient. The common ultrasound presentation of an acute hematoma is a crescent-shaped hypoechoic area beneath the liver envelope with clear borders to the liver parenchyma. Gradually, the hematoma becomes more hypoechoic and cystic. Segregation and echogenic fragments may also be observed. In cases of periportal rupture, the echogenic material will extend into the subdiaphragm and perihepatic space. However, CT has been reported to be more sensitive and accurate in determining the extent of the hematoma. The CT morphology of a hepatic subcapsular hematoma is similar to the ultrasound presentation. The density of the hematoma correlates with the time of hematoma onset. The collection will be dense relative to normal liver parenchyma on non-contrast-enhanced or contrast-enhanced scans, with a gradual decrease in density with time in case of acute hemorrhage [15]. In contrast, liver infarction shows wedge-shaped, inhomogeneous

areas of low attenuation at the periphery on CT with enhanced vessels crossing these areas [20].

Treatment

The first step in treatment is close ICU monitoring, including continuous monitoring of vital signs, regular measurement of abdominal circumference, changes in abdominal signs, and active review of blood tests to keep a dynamic picture of the condition. In addition, transfusion of large amounts of blood products, prevention of infection, and artificial liver therapy are the basic life support treatments.

Treatment of HELLP syndrome depends on the gestational age and the maternal and fetal status at the time of diagnosis. In addition, women with HELLP syndrome require prompt termination of pregnancy after maternal stabilization in the presence of one or more of the following conditions: fetal death, placental abruption, pulmonary edema, eclampsia, hepatic hemorrhage, or stroke [10].

The literature has reported the use of therapeutic plasma exchange (TPE) within 24 hours postpartum as an effective and life-saving treatment option for HELLP syndrome. The exact benefit of TPE in patients with HELLP syndrome is unknown. However, HELLP syndrome is certainly associated with severe endothelial dysfunction, and TPE may involve the removal of aggregation and procoagulation factors released by activated platelets and endothelial cells. Thus, the main goal of the TPE is to remove some plasma components, such as antibodies, immune complexes, and endogenous and exogenous toxins as well as to replace some plasma proteins and coagulation factors [21].

There are no clear guidelines for the treatment of hepatic hematomas, and they mainly include conservative and surgical treatment [17]. In the case of hemodynamic stability, conservative treatment can be considered [22, 23]. However, in the presence of hemodynamic instability, urgent angiography and hepatic artery embolization are required [18, 24] with/without surgical intervention. Surgical options range from liver tamponade to hepatic artery ligation and partial hepatectomy [24]. Furthermore, liver transplantation is also considered to be a life-saving method for patients with ruptured liver hematomas. It should be considered especially in cases of failed conservative treatment or acute liver failure. However, timely identification and transfer to a hospital with appropriate qualifications are required [15, 22, 23]. The drawback is that a transplant is governed by the urgency of the patient's condition, and the availability of liver sources and transplantation centers.

Prognosis and repeat pregnancy

The overall incidence of adverse maternal outcomes in HELLP is 38 %, and hepatic hematoma and liver infarction secondary to HELLP syndrome are severe complications. This can cause hematoma rupture, hypovolemic shock, and acute liver failure to multiorgan failure with a high maternal mortality rate ranging from 17–59 %, which usually depends on the hematoma rupture, the speed of diagnosis, and the availability of treatment options. Fetal mortality is usually associated with preterm delivery and hypoxia, ranging from 38–62 % [10]. Death is mainly caused by complications such as disseminated intravascular coagulation, pulmonary edema, or acute renal insufficiency. In contrast, HELLP syndrome cases causing hepatic hematoma and hepatic infarction are less common, and re-pregnancy is less frequently reported. Therefore, a similar treatment plan is followed for re-pregnancy and future births.

Discussion

In the present case, the diagnosis of HELLP syndrome was made promptly. The pregnancy was diagnosed and terminated early with aggressive hypotension given that the gestational week had reached 35 weeks. The postoperative blood pressure was also maintained at a normal level, but the presence of hepatic hematoma and liver infarction was unexpected. The reported cases of massive hepatic hematoma combined with liver infarction are rare in China. The patient had typical symptoms and an uncomplicated diagnosis that lacked treatment guidelines. In recent years, many cases reported in the literature have been treated by surgery, but surgery is not the only treatment route. Therefore, the specific treatment strategy needs to be evaluated in the context of the patient's hemodynamic changes, changes in the size and coagulation of the hepatic hematoma, the medical level of the local hospital, and even the attitude of the family. Further, the role of active multidisciplinary consultation should not be overlooked as a combined multidisciplinary approach can bridge the gaps between disciplines at different stages of the patient's condition and allow for a more comprehensive assessment of the patient's condition to make the most appropriate decisions. The present treatment strategy of a large hematoma combined with hepatic infarction demonstrated that conservative treatment is still possible with multidisciplinary cooperation and close monitoring of hemodynamics and could be used as a case study for conservative treatment.

Authors' Contributions

Lifeng Yao conceived, designed, and supervised the study. Jun Liu and Li Liu performed data analysis and drafted the manuscript. Guangyuan Liao collected the data. All authors reviewed and approved the final manuscript.

Conflict of Interest

The authors declare that they have no conflict of interest.

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