

Impact of Severe COVID-19 on Liver Health During Pregnancy: A Case Report on Subcapsular Liver Hematoma

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Received 2024-07-29; Accepted 2024-08-27; Online Published 2025-06-01

Abstract

Subcapsular liver hematoma (SLH) is a rare and life-threatening condition that can complicate pregnancies, particularly in association with hypertensive disorders like preeclampsia. The incidence of SLH with rupture during pregnancy is extremely low, ranging from 1 in 40,000 to 1 in 250,000, but it carries a high maternal mortality rate. The exact pathogenesis remains unclear, though it is believed to involve endothelial dysfunction and inappropriate activation of the coagulation cascade. This case report presents a preeclamptic pregnant patient with severe respiratory distress due to SARS-CoV-2 and a rare complication of subcapsular liver hematoma. The patient underwent emergency surgery, but despite prompt intervention, the outcome was unfavorable. This case highlights the need for early diagnosis and a multidisciplinary approach in managing such high-risk pregnancies.

Keywords: SARS-CoV-2, subcapsular liver hematoma, pregnancy, preeclampsia, hemorrhage.

Citation: Sahebdel B, Golshahi F, Shirazi M, Shirali E, Saedi N, Nazemi P, Omrani MA, Rahimi SH. Impact of Severe COVID-19 on Liver Health During Pregnancy: A Case Report on Subcapsular Liver Hematoma. Int J Travel Med Glob Health, 2025;13(2):111-115. doi: 10.30491/ijtmgh.2024.472511.1425.

Introduction

subcapsular liver hematoma (SLH) occurs due to a spontaneous hemorrhage between Gleason's capsule and the liver parenchyma^{1, 2}. The incidence of subcapsular liver hematoma with rupture of hematoma in pregnancies ranges from 1/40,000 to 1/250,000 and is associated with an increase in the maternal mortality rate of 86-18%^{3, 4}.

The pathogenesis of SLH is not fully understood. However, it is probably related to the microangiopathy that develops during pregnancy, with endothelial dysfunction leading to inappropriate activation of the coagulation cascade⁵.

Vascular damage can cause hemolysis and obstruction of blood flow due to fibrin deposits in the liver sinusoids, which increases liver enzymes and contributes to the formation of SLH. Rupture should be suspected in hemodynamic instability. On the other hand, the 2019 SARS-CoV-2 infection (COVID-19) is characterized by systemic inflammatory response syndrome (SIRS),

vascular damage, microangiopathy, angiogenesis, and thrombosis⁶. In the current epidemic, liver dysfunction was observed in 14-53% of cases of SARS-CoV-2 infection, especially in severe cases. In addition, acute liver injury has a higher mortality rate⁷. Despite the limitations in studies related to liver disease and involvement during pregnancy, mechanisms that cause potential liver damage include direct viral cytotoxicity, immune-related damage caused by systemic inflammatory response⁸. Also, in direct liver damage, angiotensin-converting enzyme 2 (ACE2) seems to be the key receptor for entering the virus to the cell⁹.

The aim of this study is to investigate the potential connection between severe COVID-19 infection and the development of subcapsular liver hematoma during pregnancy, emphasizing the clinical management and outcomes.

Case report

A 37-year-old G3P2(NVD*2) patient with BMI = 29 was referred on 5/8/2021 at the age of 22 weeks and one day, with complaints of fever, cough and dyspnea suspected SARS-CoV-2 .then SARS-CoV-2 infection was confirmed by RT-PCR. Vital signs of the patient at the time of visit were RR=28, PR 120, BP=105/70, OT=37.8, O2SAT=87%. In the lung CT scan , 70-75% multifocal bilateral ground-glass opacities along with crazy paving consistent with severe COVID-19 pneumonia was observed. Doppler sonography of the lower limb and cardiac echocardiography were normal. The patient was admitted to the inpatient ward and considering Covid-19 with severe pulmonary involvement, the treatment was started in the pregnant woman according to the national protocol with remdesivir , corticosteroids and prophylactic heparin.)The patient was administered dexamethasone 6 mg daily and remdesivir 200 mg on the first day, followed by 100 mg for the next five days.) Patient characteristics and clinical data, including oxygen levels, treatment regimens, and response to therapy are shown in [table 1](#).

Table 1: lab data on the first day of hospitalization

White blood cells	$3 \times 10^3/\mu\text{L}$
Hemoglobin	10.7 g/L
Platelets	$154 \times 10^3/\mu\text{L}$
ESR	39 mm/hr
CRP	38 mg/dL
Creatinin	0.6 mg/dl
AST	89 U/L
ALT	77 U/L
LDH	777 U/L
PT	13 sec
PTT	32 sec
INR	1
D Dimer	1255 ng/MI

Day 2

Due to the deterioration of the patient's respiratory condition and vital signs and the drop in saturation to 81% (without mask), the patient was transferred to the ICU. The patient was placed on NIV (Non-Invasive Ventilation) for three days. After that, there was an improvement in the patient's respiratory state, and the patient's saturation reached 94-95% with the help of NIV. The pulmonary specialist asked to continue the chemical course.

Day 5

O2 saturation decreased significantly then to more evaluation lung CTscan was requested for the patient, which showed 90% multifocal bilateral ground-glass opacities along with crazy paving With CT severity score (css) of 25. ([Figure1](#)) The crisis medical commission was formed to start actemra(Tocilizumab) by dose of 800 mg for the patient.



Figure 1. Lung CT scan ON DAY 5

Day 6

Due to the increase in liver tests, ALT=135 and AST=154, remdesivir was stopped after receiving a total of 6 doses.

Day 7

The patient was intubated due to loss of consciousness, severe respiratory distress and saturation drop to 60%.

The next day (Day7) , the patient's saturation reached 84%, also following the discontinuation of remdesivir, we saw a decrease in liver enzymes. For the patient, injection of one unit packcell was started due to the decrease of hemoglobin at the level of 8.3 g/L. Because of two plus proteinuria and blood pressure crisis at the level of 150/100 , a 24-hour urine collection was requested. Due to the patient's edema and decreased urine volume, Foley

catheter was replaced, CV Line was installed and Lasix was administered.

Later, the patient had a blood pressure crisis at 170/100, and she received 20 mg of labetalol and started diltiazem at a dose of 30 mg BID until the blood pressure reached 140/90.

On day 15

We decided to terminate the pregnancy in the crisis medical committee due to blood pressure crisis, proteinuria of 1092 grams in 24-hour urine with the diagnosis of severe preeclampsia and gestational age of 24 weeks and fetal weight below 700 grams.

Next, emergency tracheostomy was performed for the patient.

Day 16

After tracheostomy, the termination process was started through misoprostol along with the start of prophylactic magnesium sulfate. After receiving a total of 600 micrograms of misoprostol, a 660-gram fetus was delivered with an Apgar score of zero.

Day 17

The patient's blood pressure dropped sharply to 80/50, so the patient's antihypertensive medication was discontinued and normal saline was given intravenously. Bed side abdominal ultrasound examination was requested for the patient, and confirmed severe free fluid in the abdominopelvic cavity, then, fluid tap was performed, and bloody fluid was found. The patient with the above conditions and resistant hypotension and Hb = 5.4 g/L transferred to the operating room, Packcell transfusion and protamine sulfate were administered for the patient after the rapid discontinuation of heparin. Open surgery was planned with a midline incision, which revealed: rupture of the left lobe subcapsular hematoma of the liver, complete decapsulation of the 2nd and 3rd segments of the lower surface of the liver, and approximately 2.5 liters of hemoperitoneum. After performing the Pringle maneuver by bipolar cautery and packing the bleeding site, complete hemostasis was achieved. After checking the uterus and other abdominal organs and making sure of them, the drain was inserted and the abdomen was closed.

In total, the patient received 4 units of pack cells, 4 units of FFP, 2 grams of fibrinogen, and 2 units of platelets. After the operation, the patient was transferred to the ICU, and after a few minutes, due to bradycardia and cardiac arrest, she underwent CPR. Resuscitation were performed for 45 minutes and stopped due to failure.

Discussion

Subcapsular liver hematoma (SLH) is a rare and life-threatening complication of pregnancy, typically associated with hypertensive disorders such as preeclampsia¹⁰. It should be considered as a differential diagnosis in cases of sudden antepartum collapse, particularly in pregnant women with hypertensive disorders¹¹. Rupture of SLH frequently presents with hypovolemic shock, abdominal distension, massive hemoperitoneum, and respiratory difficulty¹². Diagnosis is primarily made through imaging techniques such as ultrasound, CT scan, or MRI, particularly in stable patients. The pathogenesis of SLH is not entirely understood, though common mechanisms include endothelial damage secondary to preeclampsia, which leads to hepatic sinusoidal obstruction, neovascularization, and small hemorrhages that result in hematoma formation¹³⁻¹⁵. SLH is most commonly associated with severe preeclampsia and HELLP syndrome (Hemolysis, Elevated Liver enzymes, and Low Platelet count)¹⁵. However, differential diagnoses may include liver neoplasms, infectious processes, aneurysms, and biliary diseases¹⁶. In the context of this case report, both severe COVID-19 infection and preeclampsia are considered risk factors for the development of SLH. Although preeclampsia more frequently affects young primiparous women, SLH appears to be more common in multiparous patients of advanced maternal age with hypertensive disorders of pregnancy and pre-existing liver pathology. In such cases, and generally, the only definitive treatment is the immediate termination of the pregnancy after confirming the diagnosis and assessing the clinical condition of the mother and the gestational age of the fetus. In our case, a rapid increase in liver transaminases was observed, suggesting significant liver involvement. Given that the patient also had a severe SARS-CoV-2 infection, the possibility of liver rupture due to damage induced by the virus cannot be excluded. Recent studies have reported increased hemorrhagic events in COVID-19 patients, including cerebrovascular events, aortic intramural hematoma, bilateral adrenal hematomas, hemorrhagic cardiac tamponade, and submacular hemorrhages. A possible mechanism for viral entry involves ACE2 receptors, which are abundant in type 2 alveolar cells. Binding of SARS-CoV-2 to these receptors blocks their expression, potentially increasing intravascular pressure and leading to hemorrhages. ACE2 receptors are also expressed in the gastrointestinal tract, vascular endothelium, and liver cholangiocytes, suggesting that liver involvement may be directly related to the cytopathic effect of the virus, an uncontrolled

immune reaction, sepsis, or drug-induced liver injury ⁷. The vascular effects of SARS-CoV-2, including endotheliitis, hypercoagulability, and thrombosis, are crucial factors contributing to such injuries ¹⁷.

During hepatic bleeding, conservative treatment should be considered along with the potential for hepatic artery embolization, depending on the visualization of the hemorrhagic focus, embolization technique, and artery availability. These procedures are typically performed in hemodynamically stable patients, while surgical intervention is preferred in cases of hemodynamic instability, usually via midline laparotomy. When primary suture of the liver is technically impossible, damage control surgery, including portal vein ligation, stabilization, and subsequent laparotomy for definitive bleeding control and liver tissue biopsy, may be necessary ¹⁸. The prognosis of patients with SLH depends on the severity of systemic inflammatory response syndrome (SIRS), the extent of multi-organ involvement during SARS-CoV-2 infection, and the timeliness of interventional treatment ¹⁹.

Conclusion

spontaneous hematomas without history of trauma or liver disease can be a demonstration of SARS-CoV-2 infection with or without associated with other risk factors such as severe preeclampsia. Our described case is the first rupture of subcapsular hepatic hematoma which has been synthesized by SARS-CoV-2 infection and severe preeclampsia in it. The management of such clinical scenarios requires close collaboration between the obstetrician and the surgeon with an immediate indication for termination of pregnancy. However, the complications caused by the presence of SARS-CoV-2 in the development of severe hepatopathy during pregnancy need further research.

Highlights

What Is Already Known?

Subcapsular liver hematoma (SLH) is a rare but serious pregnancy complication, typically associated with preeclampsia and HELLP syndrome. Emerging evidence suggests severe COVID-19 may increase SLH risk through endothelial dysfunction and hepatic inflammation, though this association requires further validation.

What Does This Study Add?

This case provides the first documented evidence of COVID-19-associated subcapsular liver hematoma in a pregnant patient without preeclampsia or HELLP syndrome, suggesting a novel mechanism of pregnancy-related liver injury. Our findings highlight the need to consider COVID-19 as a potential risk factor for hepatic vascular complications during pregnancy, even in the absence of traditional obstetric pathologies.

Authors' Contributions

All authors equally contributed to this study.

Acknowledgements

The authors acknowledge all individuals who provided technical and writing assistance and the group leader who provided general support.

Conflicts of Interest Disclosures

The authors declared no conflict of interest.

Consent For Publication

All authors expressed explicit consent for the publication of this manuscript.

Ethics approval

Written informed consent was obtained from the patient for the publication of the article and any associated images.

Funding/Support

The present study has no Funding resources.

The extent of AI use

None.

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