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Esophageal Varices in Pregnancy Secondary to Hepatic Cirrhosis: A Case Report

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ABSTRACT

Introduction: Pregnancy complicated by liver disease is a rare but clinically challenging condition. Portal hypertension is one such liver disease that may occur in pregnant women, primarily caused by cirrhosis. Esophageal varices, a manifestation of portal hypertension, carry significant maternal and fetal mortality risks.

Case Description: We present the case of a 22-year-old woman referred from the Internal Medicine-Gastroenterology Department with a diagnosis of grade IV esophageal varices and grade IV gastric fundal varices. At the initial referral to the Obstetrics and Gynecology clinic, her pregnancy was estimated at 5–6 weeks gestation. Despite the high morbidity and rarity of this case, the patient maintained the pregnancy until 34–35 weeks of gestation. She had a two-year history of esophageal varices, with previous hospitalizations for melena and hematemesis. Fetomaternal ultrasound revealed a singleton fetus in cephalic presentation, consistent with 34–35 weeks of gestation, with suspected intrauterine growth restriction (IUGR). Abdominal ultrasound suggested hepatic cirrhosis, and endoscopic evaluation confirmed grade IV esophageal and gastric fundal varices. Termination of pregnancy was performed via abdominal delivery.

Conclusion: Preventing pregnancy complications, accurate diagnosis, and meticulous management that balances maternal and fetal risks are crucial in such cases to improve outcomes.

Keywords

Cirrhosis in pregnancy, Esophageal varices, Maternal outcomes, Portal hypertension, Pregnancy complications.

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INTRODUCTION

Pregnancy-associated liver disease is a rare condition, as these patients typically experience impaired fertility due to disruptions in the hypothalamic-pituitary axis combined with disturbances in hepatic sex hormone metabolism [1,2]. However, when it occurs, it can lead to complex clinical situations that require careful management. Portal hypertension is a liver-related complication that may develop during pregnancy and arises from various underlying causes. In Western countries, cirrhosis is the most common cause of portal hypertension, although non-cirrhotic etiologies, such as porto-sinusoidal vascular disease, acute fatty liver of pregnancy, and Budd-Chiari syndrome, may also contribute [3].

The incidence of cirrhosis during pregnancy has been reported to be approximately 1 in 5,950 pregnancies in previous epidemiological studies [4]. Cirrhosis may worsen during pregnancy due to physiological compensation, including the development of a hyperdynamic circulatory state. This progression can lead to poor maternal and fetal outcomes, particularly when complicated by gastrointestinal bleeding. In

developing countries, non-cirrhotic portal hypertension is more prevalent and tends to have a better prognosis, with reports indicating the possibility of spontaneous pregnancies and relatively preserved liver function [4].

Esophageal varices are a key manifestation of portal hypertension, resulting from increased blood flow through collateral circulation. Variceal rupture can lead to gastrointestinal bleeding in up to one-third of pregnant women with cirrhosis and poses a serious risk of mortality for both the mother and fetus [4]. Therefore, understanding the impact of pregnancy-induced physiological changes on portal hemodynamics, as well as the consequences of portal hypertension and its underlying causes on maternal and fetal health, is crucial for optimizing pregnancy outcomes. This case report discusses a case of esophageal varices during pregnancy and provides a comprehensive review of the clinical assessment and management options for pregnant women affected by this condition.

CASE DESCRIPTION

A 22-year-old woman was first referred from the Internal Medicine-Gastroenterology Department to the Obstetrics and Gynecology outpatient clinic in March 2023 with a diagnosis of grade IV esophageal varices and grade IV gastric fundal varices. At the time of referral, the patient was unaware of her pregnancy but had been referred due to amenorrhea for the preceding two months. Transvaginal ultrasonography confirmed an intrauterine pregnancy of 5–6 weeks gestation, consistent with her last menstrual period on January 23, 2023, with an estimated due date of October 30, 2023.

In February 2023, she presented to the emergency department at Dr. Zainoel Abidin Regional General Hospital with complaints of hematemesis — dark red vomiting, occurring once, approximately 1.5 glasses in volume — accompanied by melena with a frequency of once and a characteristic foul odor. On admission, the patient appeared weak, pale, and anorexic and was subsequently admitted to the internal medicine ward. Laboratory findings revealed severe microcytic hypochromic anemia (Hb 5.12 g/dL; MCV 73 fL; MCH 24 pg; MCHC 32 g/dL), hematocrit 16%, leukocytes 3.49 × 10^9/L, platelets 79 × 10^9/L, urea 15 mg/dL, creatinine 0.5 mg/dL, albumin 3.1 g/dL, random blood glucose 107 mg/dL, SGOT 22 U/L, SGPT 25 U/L, total bilirubin 0.42 mg/dL, direct bilirubin 0.12 mg/dL, indirect bilirubin 0.3 mg/dL, PT 6 seconds, and APTT 5.6 seconds. FibroScan confirmed mild hepatic fibrosis, with a liver stiffness measurement of 8.4 kPa.

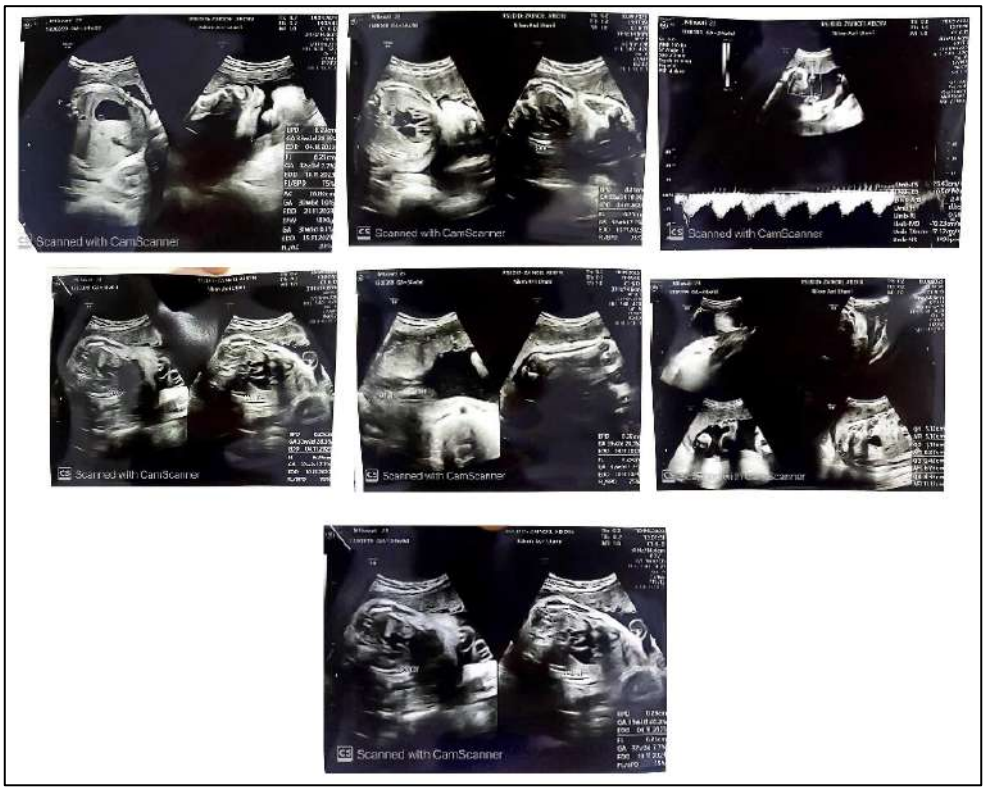

Figure 1. Fetomaternal ultrasound was performed on September 19, 2023.

Throughout her pregnancy, the patient experienced recurrent episodes of hematemesis, totaling nine episodes, up to the eighth month of gestation. She denied uterine contractions, fluid leakage, or vaginal spotting and reported no abnormal vaginal discharge. Urination remained within normal limits, and the stools were characteristically dark. She attended regular monthly antenatal care visits at our obstetric clinic.

Her medical history included a two-year history of esophageal varices. She denied the use of over-the-counter medications or alcohol consumption but reported the regular use of traditional herbal remedies aimed at weight gain. There was no history of hepatitis or other familial conditions. Her medications included propranolol, vitamin K, cefixime, ferrous sulfate, octreotide, and curcumin. The patient's menarche began at 15 years of age, with regular menstrual cycles lasting seven days and no dysmenorrhea. She married at 20 years of age and was gravida 1 para 0 abortus 0, with no history of contraceptive use. She was a housewife, and her husband was self-employed. Her pre-pregnancy BMI indicated underweight status (16 kg/m^2), with a pre-pregnancy weight of 36 kg and height of 150 cm. She gained approximately 8 kg of weight during pregnancy. Vital signs were within normal limits: blood pressure, 90/60 mmHg; pulse, 90 bpm; respiratory rate, 20 breaths/min; and body temperature, 36.5°C. General examination revealed anemic conjunctivae and icteric sclerae, with otherwise unremarkable findings on examination. Obstetric examination revealed a calm vulva and urethra. Speculum examination revealed a smooth cervix with a closed external os, no bleeding, and no abnormal discharge. A vaginal examination was not performed.

The latest laboratory tests prior to delivery revealed persistent microcytic hypochromic anemia (Hb 8.4 g/dL), improved coagulation profile (APTT 30.3 s, PT 14.4 s), decreased fibrinogen (243 mg/dL), elevated D-dimer (3010 ng/mL), and persistent thrombocytopenia (61 × 10^3/μL). Renal function tests showed low serum creatinine (0.48 mg/dL) and urea (5.0 mg/dL) levels. Electrolytes were within the normal range (Na^+ 138 mmol/L, K^+ 3.7 mmol/L). The ferritin level was 9.12 ng/mL. Fetomaternal ultrasound at 34 weeks revealed a singleton cephalic presentation fetus with a positive fetal heart rate, BPD of 8.29 cm, HC of 32.2 cm, AC of 26.80 cm, FL of 6.25 cm, and an estimated fetal weight of 1840 g, suggestive of intrauterine growth restriction (IUGR). Abdominal ultrasound performed in March 2023 indicated features consistent with hepatic cirrhosis, portal vein thrombosis, splenomegaly, and a gravid uterus.

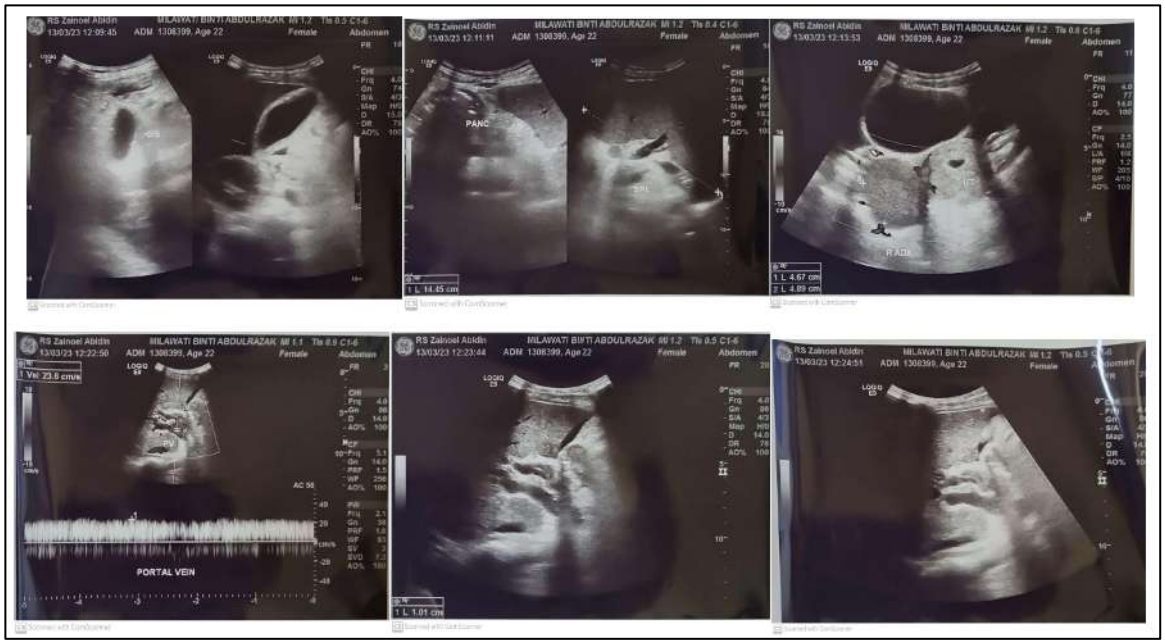

Figure 2. Abdominal ultrasound was performed on March 13, 2023.

The diagnosis was established as G1P0A0, 34 weeks and 1 d of gestation, cephalic presentation, suspected fetal IUGR, and maternal complications of grade III–IV esophageal varices, grade IV gastric fundal varices, and microcytic hypochromic anemia secondary to iron deficiency. Gastroscopy findings in March and September 2023 confirmed the progression of esophageal and gastric varices.

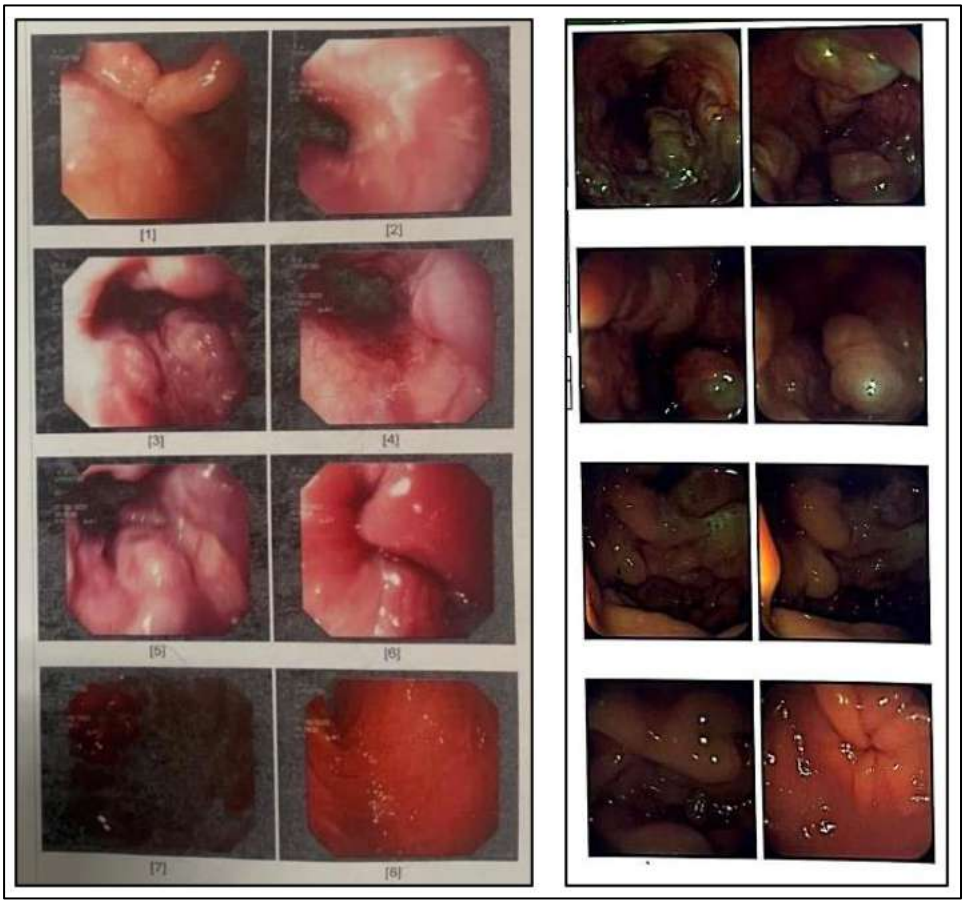

Figure 3. Gastroscopy images from March [left] and September [right] 2023.

The patient received antenatal corticosteroids for fetal lung maturation and was referred to the maternal-fetal medicine, pediatrics, internal medicine-gastroenterology, and anesthesiology departments for multidisciplinary planning. Maternal-fetal medicine recommended elective delivery prior to 37 weeks, as the fetus was deemed viable (>34 weeks) following lung maturation therapy. The pediatric team agreed to early delivery, anticipating level IIB NICU care. The gastroenterology team concurred, emphasizing the need to optimize beta-blocker therapy and provide future contraceptive counseling due to the high risk of hemorrhage in subsequent pregnancies. The anesthesiology team approved spinal anesthesia for the delivery.

An elective cesarean section was performed, delivering a female infant weighing 1900 g, length 43 cm, Apgar scores of 8/9, and a Ballard score consistent with 34–36 weeks. The amniotic fluid was clear and of adequate volume, and the placenta was delivered intact. The neonate was admitted to a level 2A NICU with diagnoses of respiratory distress syndrome, very low birth weight, and preterm small-for-gestational-age status.

DISCUSSION

Gastrointestinal bleeding, ascites, hepatic encephalopathy, and hepatorenal syndrome are potential complications associated with portal hypertension. The primary manifestations of portal hypertension include esophageal varices, splenomegaly, and hypersplenism [5]. Herein, we report a case of a pregnant woman at 34 weeks of gestation who presented with grade IV esophageal varices and grade IV gastric fundal varices, as confirmed by gastroscopy. The presence of splenomegaly, confirmed by abdominal ultrasound, further indicated portal hypertension in this patient. Portal hypertension represents a hyperdynamic circulatory state characterized by decreased peripheral vascular resistance and elevated portal vein pressure (>10 mmHg). This condition can progress significantly during pregnancy due to the physiological changes associated with gestation [5]

Numerous hemodynamic and physiological changes occur during pregnancy to accommodate a growing fetus. These changes begin around six weeks of gestation and peak at approximately 32 weeks of gestation. One of the earliest changes is an increase in plasma volume by approximately 40–50%, accompanied by a rise in maternal cardiac output by 30–50%, driven by increased stroke volume and heart rate. Systemic vascular resistance also decreases due to the effects of progesterone and placental vasculature development. Collectively, these changes contribute to a hyperdynamic state in the heart. While these adaptations support fetal growth, they can exacerbate portal hypertension in pregnant women and elevate the risk of variceal bleeding [6,-8].

The etiology of portal hypertension is generally divided into two categories: cirrhotic and noncirrhotic portal hypertension. Cirrhosis-related portal hypertension is more common and strongly associated with complications, whereas non-cirrhotic portal hypertension can result from conditions such as portal vein obstruction, malignancies, or Budd-Chiari syndrome [9]. Hepatotoxic substances and alcoholism are known triggers of cirrhosis. Certain medications (such as amiodarone, methotrexate, and steroids) and chemicals can cause acute or chronic liver injury. Acute liver damage results in necrosis or steatosis, whereas chronic injury leads to cirrhosis. Compared with non-cirrhotic portal hypertension, patients with cirrhosis tend to experience reduced fertility due to hypothalamic-pituitary axis disruption, hepatic metabolism impairment of sex hormones, portosystemic shunting of weak androgens, and peripheral aromatization of androgens. Consequently, menstrual irregularities are common in patients with cirrhosis [10]. In this case, the patient's abdominal ultrasound confirmed cirrhosis; however, she displayed atypical characteristics, such as a regular menstrual cycle, possibly due to the relatively recent onset of cirrhosis (within two years) or her young age.

Gastrointestinal bleeding is a major complication in pregnant women with portal hypertension (PH). Previous literature reports an incidence of 18–32% among pregnant patients with cirrhosis and approximately 75% among those with esophageal varices. This is due to the increased flow and pressure within the collateral circulation in the hyperdynamic state of pregnancy. Bleeding from esophageal varices can occur at any stage of pregnancy, although the highest risk periods are during the second and third trimesters and the second stage of labor. Major predictors of variceal bleeding in pregnancy include large variceal size, the presence of high-risk endoscopic signs, and a history of gastrointestinal bleeding prior to conception [4,5]

In the present case, the patient experienced gastrointestinal bleeding during the first trimester (10–11 weeks gestation). Her principal risk factors were portal hypertension secondary to cirrhosis and large varices classified as grade III–IV on gastroscopy. The management of portal hypertension during pregnancy encompasses emergency management (for acute gastrointestinal bleeding), peripartum management, and post-partum care. In the event of active gastrointestinal bleeding, immediate maternal resuscitation and stabilization are critical, accompanied by intensive monitoring and emergency variceal management. Upper gastrointestinal endoscopy is considered safe during pregnancy, with minimal risk of fetal hypoxia due to sedation and appropriate maternal positioning. The primary treatment modality is endoscopic variceal ligation (EVL), which is preferred over sclerotherapy to avoid the potential placental transfer of sclerosant toxins [5].

Pharmacological therapy with vasopressors plays a role in managing acute variceal bleeding. Terlipressin, a category D drug during pregnancy, should be avoided. Octreotide, a category B medication, may be considered, although its safety profile during pregnancy has not been fully established. Third-generation cephalosporins, such as cefazolin, can be used as prophylaxis against spontaneous bacterial peritonitis (SBP) in variceal bleeding, whereas fluoroquinolones should be avoided [4,5].

Pregnant patients at risk of variceal bleeding should receive primary prophylaxis, either through EVL or beta-blocker therapy. Beta-blockers are generally considered safe during pregnancy, although propranolol and nadolol (both category C drugs) carry the risk of fetal bradycardia, growth retardation, and neonatal hypoglycemia. Ascites is a relatively uncommon finding in pregnant women with portal hypertension (7–11%), and its management with diuretics and paracentesis should be cautiously considered based on a risk-benefit analysis[11]. In the present case, the patient was previously hospitalized for gastrointestinal bleeding and treated with octreotide. Subsequently, she received propranolol as prophylactic therapy. The occurrence

of intrauterine growth restriction (IUGR) in this case was likely associated with propranolol use, a category C medication known to cause fetal IUGR, bradycardia, and hypoglycemia.

The choice of delivery method remains controversial in pregnant women with portal hypertension. A major concern with vaginal delivery is the risk of excessive straining, which may elevate intra-abdominal and portal pressure, potentially triggering variceal rupture. To date, no definitive studies have evaluated the impact of vaginal delivery on the risk of variceal bleeding. Consequently, many experts recommend elective cesarean delivery for these patients. The cesarean section rates among pregnant women with cirrhosis vary widely (12–81%) and are influenced by patient and institutional preferences and evolving obstetric practices. Cesarean delivery also carries risks in this population, including poor wound healing and infection. Some institutions advocate for cesarean sections only for obstetric indications, while others favor vaginal delivery with a shortened second stage of labor to minimize increases in intra-abdominal pressure [5,6,13]. In this case, elective cesarean delivery was performed based on multiple considerations: gestational age, large variceal size with a high bleeding risk, and asymmetric IUGR. Asymmetric IUGR, often caused by maternal anemia, hypertension, cardiac disease, or bleeding, typically arises between 28 and 40 weeks of gestation due to impaired cellular hypertrophy. The prognosis for asymmetrical IUGR is generally favorable [13].

Postpartum management aims to detect and treat postpartum hemorrhage, which occurs in 5–45% of women with cirrhosis. This complication can result from factors such as thrombocytopenia, coagulopathy, and aberrant variceal formation. Management strategies include the administration of coagulation factors, uterotonic agents, vascular ligation, and, if necessary, hysterectomy. Antibiotics should be administered postpartum to prevent SBP, particularly in the presence of ascites [4,14].

Postpartum fever must be promptly investigated and treated with appropriate antibiotic therapy. In cases of cirrhosis related to infectious etiologies such as chronic hepatitis B, vertical transmission should be prevented by administering immunoglobulin and hepatitis B vaccination to neonates at birth. Breastfeeding is generally not contraindicated unless the mother is using Food and FDA category D or X medications. The American College of Obstetricians and Gynecologists (ACOG) recommends breastfeeding for mothers with hepatitis C or B, provided that breastfeeding is initiated after immunoglobulin administration to the newborn for hepatitis B [4].

Contraception, including barrier methods, intrauterine devices (IUDs), and permanent sterilization, should be advised for this patient population. However, permanent sterilization may be contraindicated due to coagulopathy, and hormonal contraceptives are avoided because of the risk of cholestasis [4]. The patient was using an IUD for contraception. The live birth rates in pregnant women with cirrhosis range from 58-100%, with a neonatal mortality of 0-8.3%, which is higher than that in the general population. Neonatal deaths are linked to prematurity, low birth weight, and congenital malformations. Stillbirth rates are 1-8%, and congenital anomalies occur in 0.4-2%, similar to general population rates [6-9]. Among women with cirrhosis, the first-trimester spontaneous abortion rate is 20%, while that in non-cirrhotic portal hypertension patients is 3-6%. Prematurity rates range from 19-67%. The infant was delivered at 1900 g, classified as very low birth weight and preterm small-for-gestational age, with NICU admission for respiratory distress. The patient had grade IV esophageal varices. Gastric varices are classified by Mathur: type I involves esophageal varices with lesser curvature varices; type II includes esophageal varices with fundal varices (2a subcardial, 2b diffuse fundal); type III refers to isolated fundal varices (3a from splenic vein thrombosis, 3b from portal hypertension); type IV involves lesser curvature gastric varices with esophageal and fundal varices; type V includes antral varices [15]

DECLARATIONS

None

CONSENT FOR PUBLICATION

The Authors agree to be published in the Journal of Society Medicine.

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The authors declare no conflicts of interest in this case report.

AUTHORS' CONTRIBUTIONS
All authors have made substantial contributions to this case report. SRD was responsible for patient management, data collection, and initial drafting of the manuscript. All authors reviewed and approved the final version of the manuscript, ensuring its accuracy and integrity and being accountable for all aspects of the study.

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