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Staged anesthesia for cesarean delivery in a parturient with a history of Wilson disease: a case report

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Case Report

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Abstract

Background: Cesarean anesthesia in parturients with Wilson disease is rarely reported, particularly in the presence of cirrhosis, portal hypertension, thrombocytopenia, and impaired coagulation. These conditions complicate the choice between neuraxial and general anesthesia while maternal and fetal risks must be balanced.

Case presentation: A 34-year-old woman at 35 weeks and 5 days of gestation, with a history of Wilson disease diagnosed at another institution, cirrhosis, esophagogastric varices, splenomegaly, neurologic involvement, and chronic thrombocytopenia, was admitted with persistent lower abdominal pain and progressive hepatic dysfunction. Preoperative testing showed a platelet count of 58×10^9/L, prothrombin time of 16.5 s, prothrombin activity of 62%, and total bilirubin of 91.4 μmol/L. Because neuraxial anesthesia was considered unfavorable, a staged anesthetic strategy was used. Before fetal delivery, spontaneous ventilation was maintained with face-mask oxygen, intravenous esketamine, and layer-by-layer surgical infiltration with lidocaine. After delivery, anesthesia was converted to general anesthesia using a laryngeal mask airway. A 2580-g female neonate was delivered with Apgar scores of 10 at 1, 5, and 10 min. Postoperatively, the patient developed persistent anemia, bloody abdominal drainage, hypoalbuminemia, and progressive conjugated hyperbilirubinemia and received blood products and human albumin. Drainage gradually decreased, and mother and infant were discharged together on postoperative day 8.

Conclusions: This case illustrates an individualized staged anesthetic approach for cesarean delivery in a parturient with advanced liver disease, thrombocytopenia, and coagulation impairment. This strategy achieved favorable immediate maternal and neonatal clinical outcomes, although its safety and generalizability require further evaluation.

Background

Wilson disease is an autosomal recessive disorder of copper transport caused by pathogenic variants in ATP7B. Its clinical phenotype ranges from asymptomatic biochemical abnormalities to cirrhosis, acute hepatic decompensation, neurologic disease, psychiatric manifestations, and hematologic complications [1, 2]. Pregnancy is feasible for many treated patients, but anti-copper therapy should generally be continued with specialist supervision because treatment interruption may be associated with hepatic or neurologic deterioration [1–4].

Pregnancy in patients with cirrhosis and portal hypertension is a high-risk condition. Increased circulating volume and portal pressure may aggravate variceal and ascitic complications, while hypersplenism, impaired hepatic synthesis, anemia, and the altered hemostatic balance of cirrhosis complicate decisions about delivery, anesthesia, and blood-product support [5, 6]. In obstetric patients with thrombocytopenia, platelet count alone is insufficient for decision-making; the etiology of thrombocytopenia, additional coagulation abnormalities, urgency, airway risk, and the risks of general anesthesia must be considered [7].

Published anesthetic experience in pregnant patients with Wilson disease is sparse and includes both neuraxial and general anesthesia [8–13]. Most reports describe either relatively stable disease with preserved coagulation or severe hepatic failure managed with conventional general anesthesia [9–13]. We report a parturient with a history of Wilson disease, cirrhosis, portal hypertension, esophagogastric varices, neurologic involvement, splenomegaly, chronic thrombocytopenia, and progressive hepatic dysfunction. A staged strategy—esketamine sedation plus surgical local infiltration before fetal delivery, followed by conversion to general anesthesia after delivery—was selected to avoid neuraxial puncture while limiting fetal exposure to opioids, muscle relaxant, and volatile anesthetic.

Case presentation

Patient history and antenatal course

A 34-year-old woman weighing 78 kg, gravida 2 para 0 with one previous embryonic arrest treated medically, was admitted on June 15, 2026, at 35 weeks and 5 days of gestation. Her last menstrual period was October 8, 2025. She reported persistent lower abdominal pain for more than 1 day. The fetus was a live singleton in cephalic presentation. The patient had had thrombocytopenia for approximately 20 years and bilateral involuntary hand tremor for more than 10 years.

According to the patient's history, Wilson disease and cirrhosis had been diagnosed at an outside hospital approximately 20 years earlier. She had intermittently received oral D-penicillamine. During preconception assessment at another tertiary hospital on August 19, 2025, gastroscopy and brain magnetic resonance imaging were reportedly performed, and the recorded clinical diagnoses were Wilson disease with neurologic involvement, cirrhosis with esophagogastric varices, portal hypertensive gastropathy, and pancytopenia. D-penicillamine was discontinued, three cycles of sodium dimercaptopropane sulfonate were administered, and oral zinc sulfate was prescribed after discharge. She stopped zinc therapy herself during early pregnancy. She also reported receiving further copper-removal treatment in Beijing approximately 20 days before cesarean delivery, but the medication, dose, duration, and monitoring results were unavailable.

Figure 1. Abdominal ultrasonographic findings.
A, Grayscale ultrasonography showing marked splenomegaly. On the corresponding examination, the spleen measured 17.13 cm along its longitudinal axis. **B**, Color Doppler ultrasonography showing prominent vascular signals at the splenic hilum in the setting of documented splenic vein dilatation and portal hypertension.

The original external diagnostic records, gastroscopy report, brain magnetic resonance images, serum ceruloplasmin, 24-h urinary copper, Kayser–Fleischer ring examination, and ATP7B genetic results were not available at our institution. Therefore, the present report describes a history of Wilson disease diagnosed at another institution rather than independently confirming the diagnosis.

The platelet count was 85×10^9/L before conception, 75×10^9/L at approximately 9 weeks, 59×10^9/L at approximately 14 weeks, 63×10^9/L at 22 weeks and 1 day, 67×10^9/L at 27 weeks and 2 days, and 71×10^9/L on May 26, 2026. A small-volume epistaxis episode occurred at approximately 14 weeks, but no recurrent clinically evident bleeding was documented. Hemoglobin decreased from 117 g/L before pregnancy to 100–103 g/L in the second and third trimesters. Abdominal ultrasonography on April 20, 2026 showed marked splenomegaly (longitudinal diameter 17.13 cm) and a dilated splenic vein with a maximum diameter of 1.78 cm. No ascites was reported on that scan.

Hepatic biochemical indices worsened during late pregnancy. Total bilirubin increased from 30.0 μmol/L at approximately 27 weeks to 44.4 μmol/L on May 26 and 91.4 μmol/L on June 15. On admission, total protein was 41.9 g/L, aspartate aminotransferase 50 U/L, alkaline phosphatase 295 U/L, cholinesterase 2323 U/L, direct bilirubin 56.8 μmol/L, and indirect bilirubin 34.6 μmol/L. Platelet count was 58×10^9/L, prothrombin time 16.5 s, and prothrombin activity 62%. The patient also had gestational diabetes mellitus and antenatal anemia.

Delivery and anesthetic planning

Given the severe underlying liver disease, portal hypertensive complications, progressive hyperbilirubinemia, chronic thrombocytopenia, prolonged prothrombin time, persistent lower abdominal pain, and gestational age approaching 36 weeks, the clinical team recommended timely delivery. After discussion with the patient and family, an elective cesarean delivery was scheduled for June 16, 2026, at 35 weeks and 6 days.

Neuraxial anesthesia was not selected because the platelet count of 58×10^9/L occurred in the context of cirrhosis, portal hypertension, prolonged prothrombin time, and progressive hepatic dysfunction rather than isolated gestational thrombocytopenia. The team planned a staged anesthetic technique: limited intravenous sedation and extensive surgical local infiltration before fetal delivery, followed by general anesthesia after delivery. Blood products were prepared in advance.

Intraoperative anesthetic management

On arrival in the operating room, standard monitoring was established, including electrocardiography, pulse oximetry, and noninvasive blood pressure monitoring. Supplemental oxygen was administered at 6 L/min via a face mask. Penehyclidine hydrochloride 0.5 mg was administered intravenously as an antisialagogue. Under local anesthesia, a radial arterial catheter was inserted for continuous blood pressure monitoring, followed by placement of a right internal jugular central venous catheter for central venous pressure monitoring and vascular access. The initial central venous pressure was 5 cmH2O. After central venous catheterization and before surgery, one therapeutic dose of platelets was transfused.

After surgical preparation and draping, esketamine 25 mg was administered intravenously. Once the conjunctival reflex was absent, the obstetrician performed stepwise infiltration of the abdominal wall

layers with 60 mL of 0.5% lidocaine without epinephrine (300 mg; 3.85 mg/kg). The local anesthetic was administered incrementally with repeated aspiration. Concurrently, a low-dose remifentanil infusion was initiated and titrated according to respiratory pattern, purposeful movement, vocalization or moaning, and hemodynamic responses. The clinical targets during this phase were loss of the conjunctival reflex, absence of purposeful movement, preservation of spontaneous ventilation, and maintenance of heart rate and blood pressure within approximately 20% of baseline values.

Cesarean delivery commenced at 16:03 after adequate sedation and local anesthesia had been achieved. During skin incision and abdominal wall dissection, the patient maintained spontaneous ventilation without purposeful movement, moaning, or other overt signs of distress. On entering the peritoneal cavity, approximately 500 mL of pale-yellow ascitic fluid was observed. After the uterus had been fully exposed, anesthesia was deepened with propofol 150 mg, remifentanil 120 μg, and rocuronium 50 mg. After the clinical onset of neuromuscular blockade, a disposable laryngeal mask airway was inserted, and mechanical ventilation was initiated with a tidal volume of 480 mL and an inspired oxygen fraction of 0.60. The obstetrician then incised the uterus and delivered the neonate at 16:10.

A live female neonate was delivered in the right occiput transverse position with two loops of nuchal cord. Birth weight was 2580 g, and Apgar scores were 10 at 1, 5, and 10 min. The placenta and membranes were delivered intact. Oxygen saturation remained between 98% and 100%. Continuous end-tidal carbon dioxide monitoring was not performed. Anesthesia was subsequently maintained with continuous infusions of propofol and remifentanil, titrated to maintain a bispectral index of 40–60. No clinical manifestations of local anesthetic systemic toxicity were observed.

After treatment of transient intraoperative hypertension with urapidil 20 mg, invasive arterial pressure remained between 120/75 and 143/89 mmHg. No clinically significant hypotension occurred. At 16:30, arterial blood gas analysis showed a pH of 7.29, $PaCO_2$ of 39.3 mmHg, sodium of 142 mmol/L, potassium of 4.07 mmol/L, chloride of 102 mmol/L, lactate of 1.8 mmol/L, hemoglobin of 70 g/L, and glucose of 3.3 mmol/L. The patient had no clinical symptoms of hypoglycemia and received 500 mL of a glucose-containing acetate electrolyte solution. Following the blood gas analysis, 2 units of packed red blood cells and 340 mL of frozen plasma were administered.

The recorded total intraoperative crystalloid volume was 800 mL, and 500 mL of succinylated gelatin was administered. Estimated blood loss was 300 mL, and urine output was 200 mL. Central venous pressure was 6 cmH_2O at the end of surgery.

Surgery ended at 17:00. After discontinuation of the anesthetic agents, spontaneous ventilation returned. Quantitative neuromuscular monitoring was not performed; recovery was assessed clinically by a sustained head lift for more than 5 s and strong hand grip. The laryngeal mask airway was removed at 17:06. No subsequent airway intervention or respiratory complication occurred.

For multimodal postoperative analgesia, ultrasound-guided bilateral transversus abdominis plane blocks were performed using a total of 40 mL of 0.25% ropivacaine (100 mg; 1.28 mg/kg). Patient-controlled intravenous analgesia consisted of hydromorphone 12 mg diluted with normal saline to 100 mL, with a basal infusion of 2 mL/h, a bolus dose of 0.5 mL, and a lockout interval of 15 min. Postoperative follow-up was performed on postoperative days 1 and 2, and the resting Numeric Rating Scale pain score was 2/10 on both days. No excessive sedation, nausea or vomiting, pruritus, respiratory depression, or clinically apparent inadequate analgesia was documented.

Postoperative course and outcomes

After return to the ward, the patient received another therapeutic dose of platelets and 200 mL of frozen plasma. On the evening of surgery, total bilirubin increased to 171.9 μmol/L, with direct bilirubin 100.4 μmol/L and indirect bilirubin 71.5 μmol/L; total protein was 44.5 g/L, aspartate aminotransferase 49 U/L, alkaline phosphatase 266 U/L, and cholinesterase 3170 U/L.

On postoperative day 1, the patient was alert, hemodynamically stable, and had only small-volume vaginal bleeding. Over 13 h 17 min, urine output was 1000 mL. The left pelvic drain produced 80 mL and the right abdominal drain produced 760 mL of bloody fluid. Hemoglobin was 71 g/L and platelet count was 90×10^9/L. Coagulation testing showed prothrombin time 17.1 s, prothrombin activity 58%, international normalized ratio 1.42, activated partial thromboplastin time 41.6 s, fibrinogen 2.07 g/L, and D-dimer 5892 μg/L. Two units of packed red blood cells and 390 mL of frozen plasma were transfused.

On postoperative day 2, the patient had passed flatus and remained clinically stable. Urine output was 1100 mL. The left pelvic drain produced 1000 mL and the right abdominal drain produced 230 mL of bloody fluid. Total protein was 36.8 g/L and albumin 22.7 g/L; human albumin 20 g was administered. On postoperative day 3, left and right drain outputs were 200 mL and 150 mL, respectively, and another 20 g of human albumin was administered.

On postoperative day 4, hemoglobin was 69 g/L, prompting transfusion of 1.5 units of packed red blood cells and 170 mL of frozen plasma. No cryoprecipitate, fibrinogen concentrate, or prothrombin complex concentrate was administered. No transfusion reaction occurred. The cumulative documented blood-product support was 5.5 units of packed red blood cells, two therapeutic platelet doses, and 1100 mL of frozen plasma, together with 40 g of human albumin.

Bloody drainage gradually decreased. On postoperative day 5, left and right drain outputs were 140 mL and 170 mL. On postoperative day 7, outputs were 50 mL and 40 mL, and the right abdominal drain was removed. On June 24, the left pelvic drain produced 30 mL and was removed. The patient did not develop abdominal pain or distension, wound bleeding, hematemesis, melena, or increased vaginal bleeding. Drain-fluid cell counts, hemoglobin concentration, and biochemical analysis were not performed, and postoperative abdominal ultrasonography or computed tomography was not obtained. Consequently, the bloody drainage could not be quantitatively separated into blood, ascites, and postoperative exudate and was not considered equivalent to measured blood loss.

On postoperative day 6, total bilirubin was 193.5 μmol/L, direct bilirubin 134.1 μmol/L, indirect bilirubin 59.4 μmol/L, aspartate aminotransferase 31 U/L, alkaline phosphatase 139 U/L, total protein 38.9 g/L, and albumin 25.1 g/L.

The mother and neonate were discharged together on June 24, 2026, after a 9-day hospitalization. The neonate did not require neonatal intensive care and was feeding well at discharge. The mother was advised to seek further evaluation and treatment at a tertiary hepatology center. At telephone follow-up, the mother and infant were reported to be clinically well without known readmission; however, the exact follow-up interval and objective maternal laboratory data were unavailable.

The chronological clinical course is presented in Table 1, and selected laboratory trends are summarized in Table 2.

Patient timeline

Table 1. Clinical timeline from preconception to discharge

Date / gestation	Clinical findings and investigations	Management / outcome
Mar 12, 2025 (preconception)	Platelets 85×10^9/L; hemoglobin 117 g/L.	Chronic thrombocytopenia documented before pregnancy.
Aug 19, 2025 (preconception)	External evaluation reportedly identified Wilson disease with neurologic involvement, cirrhosis, esophagogastric varices, portal hypertensive gastropathy, and pancytopenia.	D-penicillamine stopped; three cycles of sodium dimercaptopropane sulfonate; zinc sulfate prescribed.
~9 weeks	Platelets 75×10^9/L; hemoglobin 111 g/L; fibrinogen 4.25 g/L; routine coagulation reportedly normal.	Continued antenatal monitoring.
~14 weeks	Platelets 59×10^9/L; minor epistaxis; cholinesterase 4381 U/L.	No specific hemostatic intervention.
22+1 to 27+2 weeks	Platelets 63–67×10^9/L; hemoglobin 103 g/L; total bilirubin 30.0 μmol/L at ~27 weeks.	Monitoring; symptomatic dizziness/anemia noted.
Apr 20, 2026	Spleen length 17.13 cm; splenic vein up to 1.78 cm.	Findings supported portal-hypertensive splenomegaly.
May 26, 2026	Platelets 71×10^9/L; hemoglobin 100 g/L; total bilirubin 44.4 μmol/L; total protein 48.3 g/L.	Patient later reported external copper-removal treatment; details unavailable.
Jun 15, 2026; 35+5 weeks	Persistent lower abdominal pain; platelets 58×10^9/L; PT 16.5 s; PTA 62%; total bilirubin 91.4 μmol/L.	Admitted; elective cesarean delivery planned after multidisciplinary assessment.
Jun 16, 2026; 35+6 weeks	Staged anesthesia; EBL 300 mL; pale-yellow ascites ~500 mL; female neonate 2580 g; Apgar 10/10/10.	Platelets 1 dose, RBC 2 U, frozen plasma 340 mL intraoperatively; further platelets and plasma on ward.
POD 1–4	Hb 71 then 69 g/L; bloody peritoneal drainage; INR 1.42; fibrinogen 2.07 g/L; albumin 22.7 g/L.	Additional RBC 3.5 U, frozen plasma 560 mL, albumin ≥40 g.
POD 6	Total bilirubin 193.5 μmol/L; direct bilirubin 134.1 μmol/L; albumin 25.1 g/L.	Continued clinical observation; no repeat coagulation testing.
Jun 24, 2026; POD 8	Drain output decreased; both drains removed; no discharge laboratory testing.	Mother and infant discharged together; tertiary hepatology follow-up advised.

Table 2. Selected laboratory trends before and after cesarean delivery

Date / timing	Hb, g/L	Platelets, ×10⁹/L	Coagulation	TP / Alb, g/L	TBil / DBil, μmol/L
12 Mar 2025, preconception	117	85	—	—	—
11 Dec 2025, 9 weeks	111	75	Reportedly normal; Fib 4.25 g/L	—	—
17–18 Apr 2026, ~27 weeks	—	67	No evident abnormality reported	48.5 / —	30.0 / 10.0
26 May 2026, ~33 weeks	100	71	—	48.3 / —	44.4 / —
15 Jun 2026, preoperative	—	58	PT 16.5 s; PTA 62%	41.9 / —	91.4 / 56.8
16 Jun 2026, intraoperative ABG	70	—	—	—	—
16 Jun 2026, postoperative evening	—	—	—	44.5 / —	171.9 / 100.4
17 Jun 2026, POD 1	71	90	PT 17.1 s; PTA 58%; INR 1.42; APTT 41.6 s; Fib 2.07 g/L; D-dimer 5892 μg/L	—	—
18 Jun 2026, POD 2	—	—	—	36.8 / 22.7	—
20 Jun 2026, POD 4	69	68	—	—	—
21 Jun 2026, POD 5	—	59	—	—	—
22 Jun 2026, POD 6	—	—	—	38.9 / 25.1	193.5 / 134.1

Abbreviations: ABG, arterial blood gas; Alb, albumin; APTT, activated partial thromboplastin time; DBil, direct bilirubin; Fib, fibrinogen; Hb, hemoglobin; INR, international normalized ratio; POD, postoperative day; PT, prothrombin time; PTA, prothrombin activity; TBil, total bilirubin; TP, total protein; —, not measured or unavailable.

Discussion and Conclusions

This case illustrates the competing anesthetic and perioperative risks in a pregnant patient with cirrhosis with portal-hypertensive complications, portal hypertension, chronic thrombocytopenia, and impaired coagulation. The principal clinical decision was not simply between “spinal” and “general” anesthesia. It required balancing the potential for neuraxial bleeding, maternal airway and aspiration risk, altered drug handling, fetal drug exposure, anticipated transfusion, and the need for postoperative surveillance.

Diagnostic context and pregnancy-associated risk

The diagnosis of Wilson disease was made at another institution and could not be independently reconstructed because the original biochemical, ophthalmologic, genetic, gastroscopic, and neurologic imaging records were unavailable. Long-term chelation and zinc treatment, tremor, cirrhosis, portal-hypertensive complications, splenomegaly, and chronic cytopenias were clinically compatible with the reported diagnosis, but they do not replace formal diagnostic evidence. We therefore deliberately use the phrase “history of Wilson disease diagnosed at another institution.” This limitation is important because the present report is intended to address perioperative management rather than diagnosis of Wilson disease.

Current guidance and observational data generally support continuation of anti-copper treatment during pregnancy under specialist supervision [1–4]. In this patient, zinc therapy was self-discontinued in early pregnancy, and details of a later external course of copper-removal treatment were unavailable. The progressive bilirubin increase began before surgery; therefore, postoperative hyperbilirubinemia cannot be attributed solely to anesthesia or surgery, and a causal relationship with treatment interruption cannot be established.

Rationale for avoiding neuraxial anesthesia

Previous reports describe successful spinal anesthesia in patients with Wilson disease when coagulation was normal or corrected and platelet counts were considered acceptable [9, 12]. In contrast, this patient had a platelet count of 58×10^9/L together with prolonged prothrombin time, portal hypertension, splenomegaly, and progressive hepatic dysfunction. The SOAP consensus statement identifies a low estimated neuraxial hematoma risk at platelet counts of at least 70×10^9/L in selected obstetric thrombocytopenia etiologies without additional risk factors [7], but its evidence cannot be directly extrapolated to cirrhosis-associated thrombocytopenia with concomitant coagulation abnormalities. We therefore considered neuraxial puncture to have an unfavorable risk profile.

General anesthesia for cesarean delivery carries its own risks, including airway difficulty, aspiration, hemodynamic instability, neonatal drug exposure, and altered pharmacokinetics in liver disease [5]. Published Wilson disease cases with hepatic failure or marked thrombocytopenia have commonly used general anesthesia and blood-component support [10, 11, 13]. Our staged strategy was designed to postpone opioid, muscle relaxant, and volatile anesthetic exposure until after neonatal delivery while avoiding neuraxial puncture.

Staged anesthesia with conversion after uterine exposure

The choice of anesthesia was challenging because thrombocytopenia and impaired coagulation increased concern regarding neuraxial anesthesia, whereas general anesthesia before delivery could

increase fetal drug exposure. An individualized staged approach was therefore used, consisting of esketamine, low-dose remifentanil, and abdominal wall infiltration while spontaneous ventilation was maintained, followed by general anesthesia after uterine exposure. The neonate had Apgar scores of 10 at 1, 5, and 10 min. Although this approach was associated with favorable immediate maternal and neonatal outcomes, a single case cannot establish its safety or superiority, and its use requires close cooperation between the obstetric and anesthesia teams.

Hemostatic support, anemia, and bloody drainage

Cirrhosis produces a complex “rebalanced” hemostatic state in which prothrombin time, international normalized ratio, platelet count, and D-dimer do not independently quantify bleeding risk [6]. In this patient, blood-component support was based on the combined clinical context of chronic thrombocytopenia, prolonged coagulation tests, anemia, surgery, and postoperative bloody drainage. However, thromboelastography or rotational thromboelastometry was not performed, and coagulation testing was not repeated after postoperative day 1. The report therefore should not be interpreted as demonstrating goal-directed coagulation management or efficacy of plasma transfusion.

The cumulative packed red blood cell requirement of 5.5 units appears disproportionate to the intraoperative estimated blood loss of 300 mL. Several factors may have contributed, including pre-existing anemia, surgical and postoperative blood loss, hemodilution, hypersplenism, and ongoing bloody peritoneal drainage. Wilson disease-associated hemolysis was not evaluated with reticulocyte count, lactate dehydrogenase, haptoglobin, or direct antiglobulin testing. The drain fluid was not analyzed, and no postoperative imaging was performed. Because approximately 500 mL of ascites was present at laparotomy, the postoperative drainage may have contained varying proportions of blood, ascites, and inflammatory exudate. It cannot be equated directly with blood loss, and the available data do not establish postpartum hemorrhage or active intra-abdominal bleeding.

Postpartum hepatic course and follow-up

Total bilirubin increased from 91.4 μmol/L before surgery to 171.9 μmol/L on the evening of surgery and 193.5 μmol/L on postoperative day 6, predominantly because of conjugated bilirubin. Transaminases remained only mildly elevated or normal. The pattern is compatible with impaired bilirubin excretion or cholestatic/hepatic decompensation rather than massive hepatocellular necrosis, but the mechanism could not be determined because gamma-glutamyl transferase, total bile acids, hemolysis markers, infection evaluation, and biliary imaging were incomplete. Importantly, no discharge-day laboratory testing was performed. Thus, “clinical stability at discharge” should not be interpreted as biochemical recovery.

The immediate clinical maternal and neonatal outcome was favorable: the patient remained hemodynamically stable, drain output decreased, no recurrent airway intervention or documented severe

neurologic deterioration occurred, and the infant was discharged with the mother. Telephone follow-up indicated that both were clinically well. Nevertheless, the absence of a defined follow-up interval and objective postpartum laboratory results prevents assessment of the long-term hepatic, hematologic, and coagulation outcomes.

Comparison with reported obstetric anesthesia cases

Published obstetric anesthetic reports in Wilson disease are heterogeneous, reflecting differences in neurologic involvement, liver function, platelet count, and urgency (Table 3) [8–13]. Relatively stable patients with preserved coagulation have undergone spinal anesthesia, whereas patients with hepatic failure, ascites, or severe thrombocytopenia have generally received general anesthesia and blood-component therapy [9–13]. The distinguishing feature of the present case is the staged transition from pre-delivery esketamine plus local infiltration to post-delivery general anesthesia in a patient with both portal-hypertensive thrombocytopenia and abnormal coagulation tests.

Table 3. Published obstetric anesthesia case reports involving Wilson disease

Report	Maternal status	Delivery / anesthesia	Principal perioperative issue	Outcome
El Dawlatly et al., 1992 [8]	Wilson disease; obstetric comorbidity	Cesarean delivery; anesthetic management reported	Early report emphasizing individualized technique	Favorable reported outcome
Chakravarthy et al., 2015 [9]	Neurologic history; coagulation suitable for neuraxial technique	Emergency cesarean; spinal anesthesia	Avoidance of general anesthesia	Favorable reported outcome
Wan et al., 2018 [10]	Relatively stable Wilson disease	Cesarean; general anesthesia	Drug selection and maternal–fetal safety	Favorable reported outcome
Saito et al., 2019 [11]	Severe liver failure, ascites, coagulopathy	Emergency preterm cesarean; general anesthesia	Platelets, cryoprecipitate, fibrinogen; intensive care	Maternal condition improved postpartum
Özmen and Aydınlı, 2021 [12]	Irregular treatment; suitable coagulation assessment	Cesarean; spinal anesthesia	Regional technique in selected patient	Favorable reported outcome
Deshpande and Jacob, 2023 [13]	Massive splenomegaly, anemia, thrombocytopenia, portal venous disease	Cesarean; general anesthesia	Complex portal-hypertensive anatomy and bronchospasm after carboprost	Recovered
Present case	Cirrhosis, esophagogastric varices, portal hypertension, splenomegaly, platelets 58×10^9/L, prolonged PT, progressive hyperbilirubinemia	Elective preterm cesarean; staged esketamine/local infiltration followed by laryngeal-mask general anesthesia	Avoidance of neuraxial puncture while limiting pre-delivery anesthetic exposure; substantial postoperative blood-product requirement and bloody drainage	Immediate maternal and neonatal clinical outcome favorable; objective long-term laboratory outcome unavailable

Abbreviations: CSEA, combined spinal–epidural anesthesia; GA, general anesthesia; NA, not available; WD, Wilson disease.

Limitations

This report has several limitations. First, the original external diagnostic evidence for Wilson disease and the external gastroscopy and brain magnetic resonance imaging reports were unavailable. Second, no thromboelastography, rotational thromboelastometry, serial postoperative coagulation testing, drain-fluid analysis, or postoperative abdominal imaging was performed. Finally, discharge and follow-up laboratory

data were absent. These limitations prevent causal inference, detailed evaluation of transfusion efficacy, and generalization of the anesthetic strategy.

Conclusions

In a parturient with cirrhosis, portal hypertension, thrombocytopenia, abnormal coagulation tests, and a history of Wilson disease, anesthetic management required individualized balancing of neuraxial bleeding risk, general-anesthesia risk, fetal drug exposure, and anticipated transfusion. A staged approach using pre-delivery esketamine plus surgical local infiltration and post-delivery conversion to general anesthesia was associated with a favorable immediate neonatal outcome and maternal clinical stability through discharge. This single case does not establish safety or superiority. Similar management should only be considered with rigorous airway and aspiration assessment, full physiologic monitoring, readiness for immediate airway control, careful cumulative local-anesthetic dosing, objective neuromuscular monitoring, and multidisciplinary hemostatic and hepatology support.

Abbreviations

BIS
bispectral index
CVP
central venous pressure
INR
international normalized ratio
LAST
local anesthetic systemic toxicity
PaCO2
arterial partial pressure of carbon dioxide
POD
postoperative day
PT
prothrombin time
PTA
prothrombin activity
RBC
red blood cell
SOAP
Society for Obstetric Anesthesia and Perinatology
TAP
transversus abdominis plane
WD

Wilson disease.

Declarations

Ethics approval and consent to participate

According to the institutional policy of the Affiliated Hospital of North China University of Science and Technology, formal ethics committee approval was not required for this single-patient case report.

Consent for publication

Written informed consent was obtained from the patient for publication of this case report and all clinical details contained herein.

Availability of data and materials

All data generated or analyzed during this case report are included in this article. Additional de-identified clinical data are available from the corresponding author on reasonable request, subject to patient privacy and institutional policy.

Competing interests

The authors declare that they have no competing interests.

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Authors’ contributions

ZP conceived the report, participated in anesthetic management, collected and interpreted perioperative data, and drafted the manuscript. YH participated in obstetric and postoperative management, collected obstetric and maternal outcome data, and critically revised the manuscript. SZ supervised anesthetic management, contributed to case interpretation and manuscript conception, and critically revised the manuscript. All authors read and approved the final manuscript.

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Authors' information

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Figures

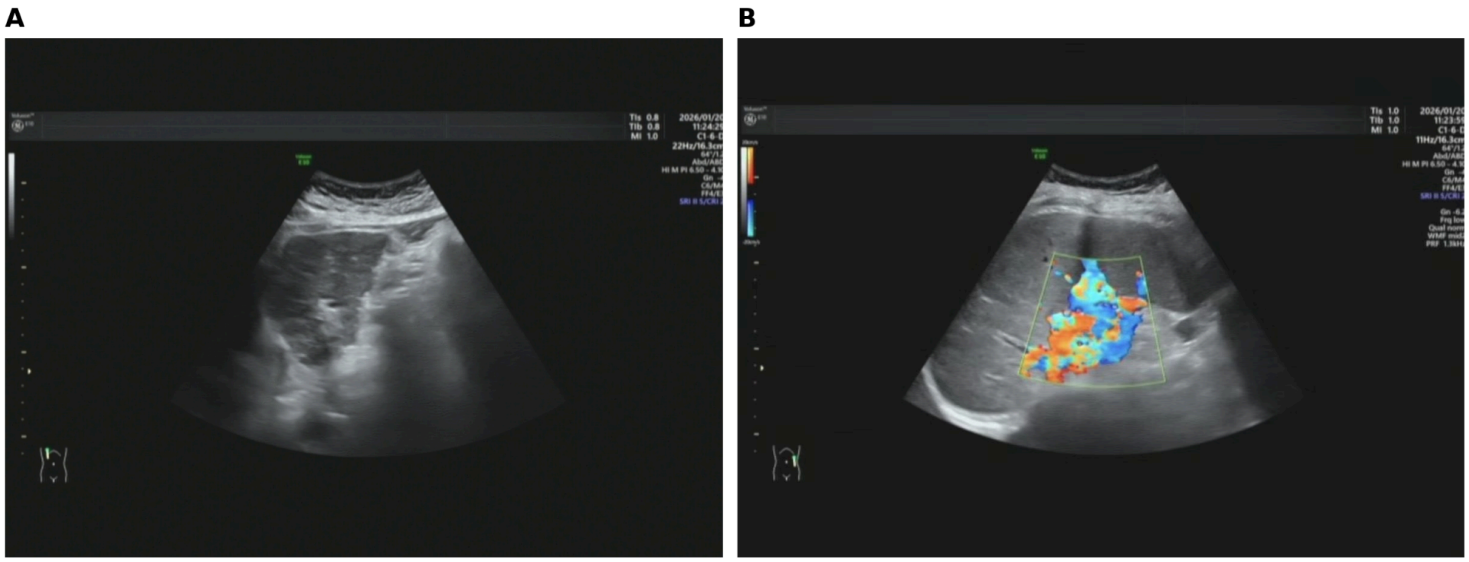


Figure 1

Abdominal ultrasonographic findings.
A, Grayscale ultrasonography showing marked splenomegaly. On the corresponding examination, the spleen measured 17.13 cm along its longitudinal axis. B, Color Doppler ultrasonography showing prominent vascular signals at the splenic hilum in the setting of documented splenic vein dilatation and portal hypertension.