JOURNAL OF THE Selva Andina Research Society
Official Journal of the Selva Andina Research Society.

Case Report

ISSN 2072-9294 (print edition)
JSARS
ISSN 2072-9308 (online edition)

Primary biliary cholangitis in pregnancy associated with vitiligo

Colangitis biliar primaria en embarazo asociada a vitíligo

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Keywords:

Hepatic autoimmune disease,
pregnancy,
ursodeoxycholic acid,
vitiligo,
maternal-fetal complications.

J. Selva Andina Res. Soc.
2026; 17(2):162-171.
Article ID: 196/JSARS/2025

Article History

Received April 2025.
Returned September 2025.
Accepted May 2026.
Available online, August 2026.

Edited by:
Selva Andina
Research Society

Abstract

Primary biliary cholangitis, known before 2014 as primary biliary cirrhosis, is an autoimmune cholestatic liver disease, rare and progressive, which can evolve into cirrhosis and liver failure. Its presentation in women of childbearing age is rare and is characterized by the presence of antimitochondrial antibodies in the blood. We describe the case of a 25-year-old patient, referred from a second-level hospital, with a history of unstudied chronic thrombocytopenia and vitiligo since the age of 21. Gynecological-obstetric history: gestation 1, delivery 1, interreproductive period of 3 years. She was 24 weeks pregnant by the date of last menstrual period and presented a clinical picture of three days of evolution with general malaise, myalgia, arthralgias, headache, unquantified fever and chills. The studies showed positive antimitochondrial antibodies (12.50 U mL^{-1}), elevated lupus anticoagulant (1.4; range 0.8-1.3), negative antinuclear antibodies (2.24), negative anticardiolipin (24.50 U mL^{-1}) and hypoalbuminemia (2.00 g dL^{-1}). Ursodeoxycholic acid, a naturally occurring component of human bile that accounts for 1-3 % of bile acids in healthy individuals, is the treatment of choice for primary biliary cholangitis, cholestasis gravidarum, and cholesterol gallstones. In pregnancy it can coexist with other autoimmune diseases such as vitiligo, so its timely detection is essential to prevent miscarriages, premature births and maternal-fetal complications. In this case, the diagnosis was established at 25 weeks and treatment with ursodeoxycholic acid was initiated, achieving a favorable evolution and eutocic delivery at 36 weeks of gestation.

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Resumen

La colangitis biliar primaria, denominada antes de 2014 cirrosis biliar primaria, es una enfermedad hepática colestásica autoinmune, infrecuente y progresiva, que puede evolucionar hacia cirrosis e insuficiencia hepática. Su presentación en mujeres en edad fértil es rara y se caracteriza por la presencia de anticuerpos antimitocondriales en sangre. Se describe el caso de una paciente de 25 años, referida desde un hospital de segundo nivel, con antecedentes de trombocitopenia crónica no estudiada y vitíligo desde los 21 años. Antecedentes gineco-obstétricos: gesta 1, parto 1, periodo intergenésico de 3 años. Cursaba embarazo de 24 semanas por fecha de última regla y presentó cuadro clínico de tres días de evolución con malestar general, mialgias, artralgias, cefalea, fiebre no cuantificada y escalofríos. Los estudios manifestaron anticuerpos antimitocondriales positivos (12.50 U mL^{-1}), anticoagulante lúpico elevado (1.4; rango 0.8-1.3), anticuerpos antinucleares negativos (2.24), anticardiolipina negativos (24.50 U mL^{-1}) e hipoalbuminemia (2.00 g dL^{-1}). El ácido ursodesoxicólico, componente natural de la bilis humana que representa entre 1-3 % de los ácidos biliares en individuos sanos, constituye el tratamiento de elección para la colangitis biliar primaria, la colestasis gravídica y los cálculos biliares de colesterol. En el embarazo puede coexistir con otras enfermedades autoinmunes como el vitíligo, por lo que su detección oportuna es fundamental para prevenir abortos, partos prematuros y complicaciones materno-fetales. En este caso, el diagnóstico se estableció a las 25 semanas y se inició tratamiento con ácido ursodesoxicólico, logrando evolución favorable y parto eutócico a las 36 semanas de gestación.

Palabras clave:

Enfermedad autoinmune hepática,
embarazo,
ácido ursodesoxicólico,
vitíligo,
complicaciones materno-fetales.

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Introduction

Primary biliary cholangitis, known before 2014 as primary biliary cirrhosis, is an autoimmune cholestatic liver disease, rare and progressive, which can evolve into cirrhosis and liver failure[1]. Its occurrence in women of childbearing age is rare and is characterized by the presence of antimitochondrial antibodies in the blood[2]. Primary biliary cholangitis most frequently affects postmenopausal women in northern European and American countries; however, thanks to new diagnostic tests, about 25 % of cases are detected during the fertile period[3,4]. Risk factors described include family history, coexistence of other autoimmune diseases, recurrent urinary or vaginal infections, smoking, and exposure to nail polishes and hair dyes[5].

During pregnancy, physiological changes in biochemical parameters occur; transaminases (alanine aminotransferase and aspartate aminotransferase) and γ-glutamyl transferase are usually within normal values or slightly decreased by hemodilution; total and indirect bilirubin tend to decrease slightly, while direct bilirubin decreases in the second and third trimesters. In contrast, alkaline phosphatase can increase two- to four-fold during the third trimester[6].

Histopathologically, primary biliary cholangitis is characterized by senescence of biliary epithelial cells, apoptosis, and progressive loss of ducts[7]. The diagnosis is based on three criteria; presence of antimitochondrial antibodies type M2, persistent elevation of serum alkaline phosphatase (ALP) and compatible liver histology. In addition, several subtypes of antinuclear antibodies, such as anti-Sp100, anti-PML, and anti-gp210, are considered specific markers. The disease may begin subclinically with elevated cholestatic enzymes and slowly progress to portal hypertension and advanced liver disease[8].

Primary biliary cholangitis has a chronic course and can evolve into end-stage liver failure, requiring liver transplantation[9]. Clinically, it manifests with fatigue, pruritus, malabsorption syndrome, osteoporosis, hyperbilirubinemia, hypercholesterolemia, and positive antimitochondrial antibodies and antinuclear antibodies[9]. Diagnosis includes medical history, physical examination, liver function tests, viral markers, autoimmunity studies, and abdominal ultrasound[9]. Differential diagnoses include primary sclerosing cholangitis, autoimmune hepatitis, biliary obstructions, drug-induced cholestasis, cholestasis of pregnancy, cholangiocarcinoma, and other liver pathologies[9].

Since 1987, ursodeoxycholic acid (UDCA) has been established as a standard treatment, improving prognosis, delaying progression and reducing complications[10].

Vitiligo, on the other hand, is an autoimmune depigmenting skin disorder with a worldwide prevalence of 0.5-2 %, characterized by selective loss of melanocytes and the appearance of whitish non-squamous macules, with a significant psychological impact[11]. Its treatment includes phototherapy, topical and systemic immunosuppressants, as well as surgical tech-

niques, which together can stop progression, stabilize lesions and promote repigmentation[12].

The objective of this study was to describe the clinical case of a young patient of childbearing age diagnosed with primary biliary cholangitis during pregnancy, associated with vitiligo and chronic thrombocytopenia, emphasizing the importance of timely diagnosis and treatment with ursodeoxycholic acid to prevent maternal-fetal complications and achieve a favorable obstetric outcome.

Case Presentation

A 25-year-old patient with a history of chronic thrombocytopenia that has not been studied, vitiligo since the age of 21, with a history of obstetric gynecology; 1 pregnancy, childbirth 1, interreproductive period 3 years, menarche at 18 years, regular menstrual cycles, sexually transmitted diseases not reported, cervical Pap smear has never been performed, prenatal controls 6, ultrasounds 4, iron and folic acid received from the first trimester, tetanus vaccine received two doses, urinary tract infections do not refer, vaginal infection does not refer, HIV and non-reactive VDRL, pregnancy was not planned, she received two vaccines for COVID 19, in times of pandemic.

At 24 weeks of pregnancy, she presented a clinical picture of 3 days of evolution with general malaise, muscle pain, joint pain, headache, unquantified thermal rise, chills, self-medicated with paracetamol 1 g orally three times a day. *Lab report*; leukocytes 8290 µL, neutrophils: 91.6 %, hematocrit 29.3 %, hemoglobin 10.4 mg dL^{-1}, platelets 50 000, creatinine 0.55, glucose: 105, TGO: 36, TGP: 33, PTR: 15.3, TTP: 38, INT: 1.3, syphilis negative, non-reactive HIV, decreased protein and albumin, negative urine test, procalcitonin 1.74. She is transferred to the maternal intensive care unit; febrile fever, it occurs with a tendency to hypotension with oliguria, capillary filling in 3 seconds, so vasopressor support and nasal cannula at 3 L were placed, hematology consultation and antimitochondrial antibodies were requested. *Vital signs*; blood pressure 96/39 mmHg. Heart rate 89 bpm, Respiratory rate 20 rpm, SpO_2 100 %, temperature 38.8º C. In the intensive care area, antibiotic treatment was started with clinical improvement. After 3 days, she was already afebrile with vital signs: blood pressure: 104/61 mmHg, heart rate 76 bpm, respiratory rate 18 per minute, temperature: 36.1º C, SpO_2 100%, alert state of consciousness, proteinuria: negative, MAMA Score: 00, obstetric risk: very high. A new laboratory control was carried out: hematocrit 18.8 %, hemoglobin: 6.6 mg dL^{-1}, platelets 46 000, sodium: 131, chlorine: 109, potassium 3.6, as they decided to transfuse one unit of concentrated red blood cells. Urine culture without development, direct/indirect Coombs: negative, Dengue: negative. Results of antimitochondrial antibodies 12.50 µ mL^{-1} (positive), lupus anticoagulant 1.4 (increased, reference range 0.8-1.3), antinuclear antibodies: 2.24 (negative), anticardiolipin 24.50 µ mL^{-1} (negative), hypoalbuminemia (2.00), albumin replacement was decided, abdominal ultrasound showed 1300 mL of ascitic fluid.

Abdominal ultrasound at 24 weeks gestation: chronic liver disease with signs of portal hypertension. *Obstetric ultrasound*: 24 weeks, 0 days pregnant. Weight: 690 g. Amniotic fluid index 13.1 right anterolateral placenta, grade 1.

Diagnostic impression: primary biliary cholangitis, portal hypertension, sepsis, unspecified, high-risk pregnancy monitoring.

At 26 weeks of gestation: blood smear: red blood: hypochromia anisocytosis, normal white series,

platelets 155 000, negative Coombs IgG test, negative C3. FT4 0.86, TSH 2.96, vitamin D 24.59 (optimal level greater than 30).

Blood count: white blood cells 5.74, neutrophils 77.1 %, lymphocytes 13.8, platelets 155 000 manual, hemoglobin 9 mg dL^{-1}, hematocrit 25.5 %, total bilirubin 0.96, direct bilirubin 0.71 (elevated), indirect bilirubin 0.24 (low), calcium 8.2, alkaline phosphatase 192, GGT 52.3 (elevated), quantitative CRP 1.66, TGO 23, TGP 25, reticulocytes percentage 3.34 (decreased) reticulocytes 93.50, fraction of immature reticulocytes 7.

Abdominal echo of the upper abdomen at 26 SG: ultrasound signs corresponding to splenomegaly liver disease and perisplenic lamellar fluid. *Obstetric ultrasound*: gravid uterus is explored, single product longitudinal breech heartbeat positive 153 per minute fetal movements present, ILA 11, fundic placenta adequate thickness measures 2.8 cm, estimated weight 1019 g. Pregnancy of 26.4 weeks.

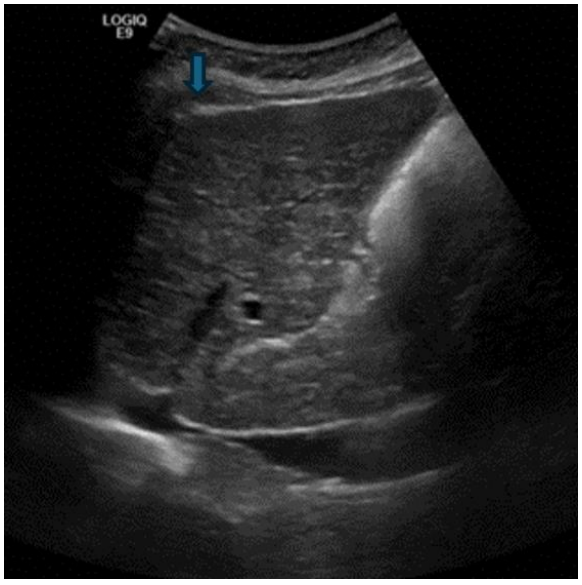


Figure 1 Liver of increased size at the expense of the left lobe measuring 65 mm, right lobe measuring 124 mm, irregular contours, heterogeneous parenchymal structure of micronodular appearance, no focal or diffuse lesions, portal vein and suprahepatic of normal characteristics. Ultrasound signs corresponding to liver disease and perihepatic laminar fluid

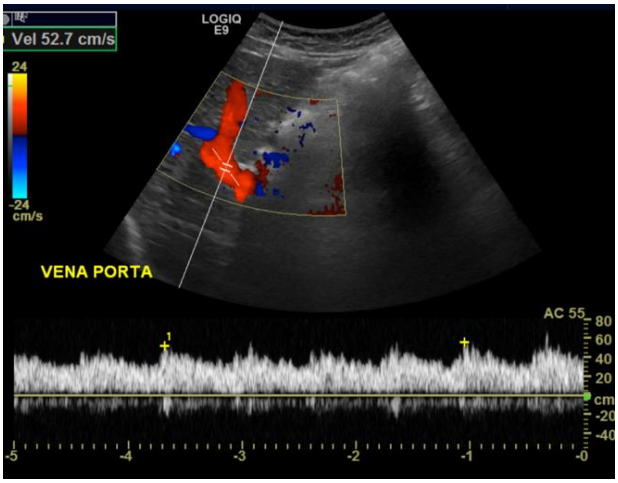


Figure 2 Color and spectral Doppler mode portal vein measures 13 mm permeable, increased caliber, change in diameter with the respiratory cycle, hepatopetal flow, velocity 53 cm/s, non-visible splenic vein, suprahepatic veins and inferior vena cava of normal caliber and preserved phaszicity, with no evidence of signs of hepatic artery thrombosis, of normal caliber, laminar flow, resistance index within normal limits. Portal vein without signs of thrombosis. The Doppler study of the splenoportal axis does not meet criteria for portal hypertension. Ultrasound signs of liver disease and splenomegaly

From week 26 to 35, he was treated with ursodeoxycholic acid 250 mg orally every 8 h, iron + folic acid orally one tablet daily, calcium 1 g orally daily.

At 36 weeks of gestation, she presented clinical symptoms characterized by abdominopelvic pain of moderate intensity that radiated to the hips, fetal movements present, fetal heart rate 150 bpm. On vaginal examination: central cervix dilated 10 cm effaceted 100 % intact membranes, plane III, she went to the delivery room where she obtained a single live product sex woman, crying present, weighing 2295 g, size 43 cm, Apgar 7-9-9, subsequently performing joint accommodation with her mother.

Results

In contrast to the case presented, during the research, the literature found the case of a 36-year-old Nepalese woman, without a significant pathological personal history, who was treated for pruritus and jaundice at 17 weeks of gestation. Serial blood tests

showed direct hyperbilirubinemia and elevated serum levels of liver enzymes, with a predominance of cholestatic diseases. Bile acids at 21 weeks and were elevated (91.1 μmol L^{-1}). They also identified fasting hyperglycemia, compatible with gestational diabetes. They performed an abdominal ultrasound that revealed no signs of extrahepatic cholestasis or liver abnormalities. Acute viral hepatitis was ruled out by serological tests, but he did present weakly positive fine mottled antinuclear antibodies, increased IgM and strongly positive antimitochondrial antibodies, compatible with primary biliary cholangitis[13].

Table 1 Results of laboratory tests before and after delivery of a patient with primary biliary cholangitis

Laboratories	Prenatal	Postpartum
Leukocytes	8.27	7.3
Neutrophils	86.1	83.3
Hemoglobin	12.3	12.8
Hematocrit	35	37.1
Platelets	86 000	100 000
Urea	12	25
Creatinine	0.35	0.30
Uric acid	3.40	
TGO	393	336
TGP	400	373
Total, bilirubin	8.54	6.32
Direct bilirubin	5.54	4.32
Indirect bilirubin	3.00	2
Alkaline phosphatase	248	
Sodium		137
Potassium		3.8
Chlorine		107

Decreased liver enzymes in postpartum control

We found the case of a 37-year-old Portuguese Caucasian patient who consulted for generalized pruritus and alteration of liver function tests with cholestatic pattern. They suspected intrahepatic cholestasis of pregnancy, so they started treatment with ursodeoxycholic acid, with slight improvement of cholestasis. The pregnancy was complicated by occlusive hemorrhagic placenta, and at 30 weeks they performed an emergency cesarean section due to significant blood loss. After periodic evaluation and postpartum care, they concluded that it was primary biliary cholangitis[14].

A meta-analysis of eleven studies involving 2179 female patients with primary biliary cholangitis showed that pregnant women with primary biliary cholangitis have significantly higher rates of miscarriage, threatened miscarriage, and lifetime smoking, compared to matched controls from national health care registries. The probabilities of gestational diabetes and preterm birth were also high, without reaching statistical significance[15].

Pregnancy in patients with squamous cell cholangitis and primary sclerosing cholangitis is well tolerated, but women should be informed of the increased risk of preterm birth[16].

In another study, they presented the case of 3 patients of First Nations ancestry who developed primary biliary cholangitis after postpartum resolution, with case 1: A 27-year-old woman who presented with pruritus and jaundice at 20 weeks of her fourth pregnancy. She previously developed intrahepatic cholestasis of pregnancy (ICD) at 31 weeks during her third pregnancy. At 20 weeks, bilirubin was 103 μmol L^{-1}, ALP 371 IU L^{-1}, GGT 14 IU L^{-1}, ALT 34 IU L^{-1}; antinuclear antibodies and antimitochondrial antibodies negative. He responded to ursodiol (UDCA) and cholestyramine. At 30 weeks, bilirubin was 9 μmol L^{-1}, ALP 216 IU L^{-1}, GGT 17 IU L^{-1}, ALT 13 IU L^{-1}. She gave birth at 33 weeks, stopping the medication shortly thereafter because she was asymptomatic at that time[17].

Case 2: A 30-year-old woman who presented with pruritus at 20 weeks of her second pregnancy. She had developed ICD at 20 weeks after her first pregnancy, with postpartum resolution. During her second pregnancy, bilirubin was 17 μmol L^{-1}, FA 324 IU L^{-1}, GGT 28 IU L^{-1}, ALT 26 IU L^{-1}, bile acids 71 μmol L^{-1}. He was diagnosed with ICD and responded

to the UDCA. She gave birth at 35 weeks and discontinued the UDCA shortly thereafter as she was asymptomatic at the time. She presented again 2 months later with pruritus. Restarting treatment with UDCA improved symptoms, but cholestasis was maintained at follow-up 4 months later[17].

Case 3 A 30-year-old woman with a history of primary biliary cholangitis in her mother presented with pruritus and jaundice at 20 weeks after her fourth pregnancy. ICD was diagnosed and responded to the UDCA. She gave birth at 37 weeks and discontinued UDCA treatment shortly thereafter, as she was asymptomatic at the time[17].



Figure 3 Patient in the puerperal stage, in joint accommodation with her baby, breastfeeding the newborn, hypochromic gaits compatible with vitiligo are observed, patient with a diagnosis of primary biliary cholangitis

Discussion

During pregnancy, physiological changes occur in biochemical parameters. Transaminases (alanine aminotransferase and aspartate aminotransferase) and γ-glutamyl transferase are usually within normal values or slightly decreased by hemodilution. Total and indirect bilirubin tend to decrease slightly, while direct bilirubin decreases in the second and third trimesters. In contrast, alkaline phosphatase can increase two- to four-fold during the third trimester. In the case presented, a progressive increase in liver enzymes was evidenced throughout pregnancy[14,18].

The patient, young and of childbearing age, with a history of vitiligo, an autoimmune disease that has been little studied and without previous treatment, presented with an infectious condition that responded to intravenous antibiotic therapy. He required transfusion of concentrated red blood cells and platelets due to severe anemia and thrombocytopenia. Ultrasound signs of chronic liver disease and splenomegaly were documented, along with progressive elevation of liver enzymes and bilirubin's with direct predominance, as well as hypoalbuminemia that favored the formation of ascites. Glucose, amylase, and lipase values remained within normal limits, and the test for dengue was negative. Immunological studies detected positive antimitochondrial antibodies, while antinuclear, anticardiolipin, and C3 antibodies were negative. Throughout pregnancy, prolonged coagulation times, elevated direct bilirubin from the beginning and an increase in liver enzymes greater than ten times their normal value in the third trimester, accompanied by a significant increase in alkaline phosphatase, were observed. At the end of pregnancy, hemoglobin, hematocrit, and platelets were normalized. The delivery was performed vaginally without complications, obtaining a single, live female newborn. The patient, diagnosed with primary biliary cholangitis and vitiligo, had a favorable evolution under treatment with UDCA at a dose of 250 mg orally every 8 hours, established from the 26th week of pregnancy.

UDCA is a naturally occurring component of human bile, accounting for 1-3 % of bile acids in healthy individuals. It is currently approved for the treatment of primary biliary cholangitis, cholesterol gallstones and the prevention of their formation in obese patients undergoing rapid weight reduction, such as after bariatric surgery. Stimulation of hepatocellular secretion induced by UDCA could be key to rapid relief of pruritus and improvement of serum liver tests. Regarding diagnosis, the presence of antimitochondrial antibodies in titers ≥1:140 together with alterations in liver tests, particularly an alkaline phosphatase elevated more than 1.5 times the upper limit of normal, allows the diagnosis of primary biliary cholangitis to be established without the need for liver biopsy. Histologically, intrahepatic bile duct injury is the first evidence of primary biliary cholangitis and is present in all stages of the disease[19,20].

In pregnant women, vaginal delivery is not contraindicated unless there are other obstetric conditions. Close follow-up is required, and it is important to note that no side effects or teratogenicity have been reported in children of mothers with chronic cholestasis treated with UDCA during pregnancy[21].

Primary biliary cholangitis has been associated with a higher frequency of induced abortions and fewer full-term deliveries. Likewise, mild to moderate alcohol consumption is negatively related to the disease. Since biliary lesions are limited to small intrahepatic ducts, imaging findings are usually normal in these patients. Magnetic resonance cholangiopancreatography and endoscopic retrograde cholangiopancreatography are indicated in patients who are negative for antimitochondrial antibodies and antinuclear antibodies specific to primary biliary cholangitis, but with rapid elevation of serum bilirubin and ultrasound suspicion of bile duct stenosis or dilation. Transient or magnetic resonance imaging elastography is a reliable noninvasive method for evaluating fibrosis and can be used in the staging of primary biliary cholangitis[22,23].

Pruritus, a particularly relevant symptom in pregnant women, can significantly affect quality of life. There are currently treatment options for pruritus in fibrosing cholangiopathies, such as primary biliary cholangitis and primary sclerosing cholangitis, including PPAR receptor agonist (bezafibrate) and pregnane X receptor agonist (rifampicin). In intrahepatic cholestasis of pregnancy, the treatment of choice is UDCA, and rifampicin may be supplemented in the third trimester if pruritus persists[24].

Conclusion

Primary biliary cholangitis is a rare autoimmune disorder during pregnancy, which can manifest in patients with other underlying autoimmune diseases, such as vitiligo. Its timely detection is essential to prevent obstetric and perinatal complications, including miscarriages, premature births and maternal-fetal morbidity.

In the case presented, the diagnosis was established at 25 weeks of gestation and treatment with UDCA was instituted, achieving a favorable evolution and culminating in a eutocic delivery at 36 weeks. This outcome shows that, with early recognition and adequate management, pregnancy can reach the third trimester with a good perinatal prognosis.

On the contrary, in untreated cases, primary biliary cholangitis may be associated with adverse outcomes such as fetal death secondary to fetal distress derived from maternal metabolic alterations, in addition to the clinical decompensation of the mother. In such circumstances, care in high-risk units and a multidisciplinary approach are required to optimize maternal-fetal outcomes.

Funding Source

Without external sources of financing, authors' own funds.

Conflicts of interest

The authors declare that they have no conflict of interest.

Acknowledgements

We thank the University Hospital for allowing us to have access to the medical history and the patient to develop this research.

Ethical considerations

For this publication, authorization was requested from the patient by signing an informed consent.

Limitations in research

Patient dropout, she could not have a good follow-up by outpatient consultation.

Publishing permissions

Se adjunta documento firmado por paciente donde autoriza la publicación del caso clínico.

Identification of the responsibility and contribution of the authors in the article

The authors declare to have contributed to the original idea (*María Calderón*), research design (*Cinthya Ulloa*), data collection (*Monica Moran, María Acosta, Doris Domo, Angel Chango*), data analysis (*María Calderón, Katryn Nagua, Cinthya Cecilia Ulloa*), draft writing and article writing (*María Calderón, Cinthya Ulloa, Mónica Moran, María José Acosta, Doris Domo, Ángel Chango, Katryn Nagua*.

Use of artificial intelligence

No artificial intelligence was used in the development of the manuscript.

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