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Postpartum Hepatic Decompensation Revealing Type 1 Autoimmune Hepatitis: The Value of Liver Immunological Workup, Especially Anti-F-Actin Antibody Dosing

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Abstract

Autoimmune hepatitis (AIH) is an immune-mediated chronic liver disease predominantly affecting women of reproductive age. Pregnancy induces a state of immune tolerance characterized by a shift from pro-inflammatory Th1/Th17 responses to anti-inflammatory Th2/regulatory T cell profiles, often leading to remission of AIH. However, the postpartum period is marked by immune reactivation and a high risk of disease flare, occurring in up to 55% of cases. Diagnosis relies on serological markers, including anti-F-actin antibodies, which are highly specific and correlate with prognosis, alongside liver biopsy for histopathological confirmation. Management during pregnancy requires careful balancing of immunosuppressive therapy to control disease activity while minimizing maternal and fetal risks. Corticosteroids remain the first-line treatment, with azathioprine as a potential adjunct, both generally considered safe in pregnancy with appropriate monitoring. We report the case of a 44-year-old woman who developed AIH type 1 in the early postpartum period, highlighting the diagnostic challenges and therapeutic considerations. This case underscores the importance of vigilant monitoring during pregnancy and postpartum to promptly identify and manage AIH flares, thereby improving maternal and neonatal outcomes.

Keywords

Autoimmune Hepatitis Type 1, Postpartum, Anti-F-Actin Antibodies, Liver Cytolysis, Azathioprine

1. Introduction

Autoimmune hepatitis (AIH) is an immune-mediated liver disease characterized by an aberrant innate and adaptive immune response targeting hepatocyte and biliary epithelium autoantigens, resulting in hepatic inflammation. It predominantly affects women, especially of reproductive age, and can complicate pregnancy. During gestation, specific immunological mechanisms promote fetal tolerance and often lead to remission of AIH. This immunological shift is believed to result from elevated estrogen and progesterone levels, which suppress Th1 and Th17 cytokine expression, promote differentiation of Th0 lymphocytes into Th2 cells, and induce regulatory T cells (Tregs). This shift from pro-inflammatory Th1/Th17 to anti-inflammatory Th2/Treg responses favors immune tolerance during pregnancy. However, following delivery, when the immunosuppressive state resolves, 13% to 55% of patients experience disease flares [1] [2].

Anti-F-actin antibodies are more specific and sensitive than anti-smooth muscle antibodies for diagnosis. Their presence correlates with poorer prognosis. Differentiating AIH from other chronic hepatitis forms is essential, as immunosuppressive therapy is often effective. Without treatment, AIH can progress to cirrhosis with risks of decompensation, severe hepatic failure, and death. The disease carries a relatively poor prognosis, with 10-year survival rates rarely exceeding 10% without appropriate management [3].

2. Case Report

A 44-year-old woman, gravida 3, para 3, with a history of type 2 diabetes mellitus managed with insulin and a cholecystectomy five years prior, was referred five days postpartum from a peripheral maternity unit due to jaundice appearing on postpartum day three. She had delivered a healthy male infant vaginally with Apgar scores of 10/10 and a birth weight of 3500 g.

On admission, the patient was conscious but asthenic, with generalized marked jaundice and elevated blood pressure at 160/100 mmHg. Urine dipstick was negative for proteinuria, capillary blood glucose was 0.95 g/L, and abdominal examination revealed a soft abdomen with a well-contracted uterus and no organomegaly.

Laboratory investigations showed leukocytosis (21,460/mm^3), thrombocytopenia (80,000/mm^3), reduced prothrombin time (38%), activated partial thromboplastin time twice the control, factor V at 42%, and hepatic cytolysis with transaminases elevated more than tenfold (AST 584 U/L, ALT 319 U/L). Alkaline phosphatase was 174 IU/L, total bilirubin 168 µmol/L, lipase 30 U/L, creatinine 19.6 mg/L, and 24-hour proteinuria was 0.15 g. Viral serologies for hepatitis A, B, C, and E, as well as CMV and EBV, were negative. No history of hepatotoxic substance use was reported.

Abdominal ultrasound showed a homogeneous liver with regular contours and no biliary duct dilation. The portal vein was normal in caliber and patent. Kidneys were normal in size and position without dilated collecting systems. The uterus was globular and enlarged with a fine endometrial stripe and homogeneous echo-

texture. No adnexal masses were detected.

Immunological workup revealed positive anti-F-actin antibodies at a titer of 1:160 and elevated total serum immunoglobulins, more than three times the upper limit of normal. Other autoimmune markers were negative, including anti-mitochondrial type M2, anti-gp210, anti-sp100, anti-LKM1, anti-LC1, and anti-SLA/LP antibodies. Liver biopsy histopathology showed a plasma cell-rich infiltrate around portal tracts, inflammation and destruction at the portal-lobular interface, and hepatocyte rosettes, consistent with type 1 AIH. The laboratory values used are listed in Appendix **Table S1**.

Despite the definitive diagnosis and thorough counseling on the risks of hepatic insufficiency progression, the patient declined immunosuppressive therapy.

Forty-five days post-diagnosis, the patient's condition worsened, with persistent jaundice, hepatomegaly, rising transaminases, and decreasing prothrombin levels, indicating progressive hepatic dysfunction in the absence of treatment.

3. Discussion

AIH predominantly affects women (approximately 70% of cases), with about 50% of patients younger than 40 years. Disease onset typically occurs between 30 and 50 years, consistent with our patient's age [4].

Clinical presentations are heterogeneous; about one-third present with acute icteric hepatitis [5]. Other nonspecific signs include asthenia, hepatomegaly, and right upper quadrant pain. Approximately 30% of cases have an acute presentation mimicking viral hepatitis, while others have an insidious onset leading to late diagnosis at advanced stages [4]. Our patient presented with acute jaundice and asthenia, typical for severe disease onset.

Type 1 AIH (classic form) accounts for roughly 75% of cases. Antinuclear antibodies (ANA), present in over 90% of patients, are nonspecific. Anti-smooth muscle antibodies help differentiate AIH from systemic lupus erythematosus. Anti-F-actin antibodies, as detected in our patient, are more specific and sensitive and are associated with worse prognosis [6]. p-ANCA and antimitochondrial antibodies may be detected but have limited diagnostic utility. Anti-DNA native antibodies, markers for lupus, can occasionally be present in type 1 AIH [6].

Non-invasive imaging is critical to exclude biliary obstruction and thrombosis during pregnancy. Our patient's abdominal ultrasound was unremarkable. Liver biopsy is reserved for diagnostic uncertainty, especially with negative serologies or normal immunoglobulin levels. Biopsy during pregnancy has not been linked to increased risks of preterm birth or stillbirth compared to pregnancies complicated by other liver diseases [7] [8].

The course of AIH during pregnancy is unpredictable. Spontaneous remission has been reported, likely related to pregnancy-induced immune tolerance shifts. Malhotra *et al.* described four patients who improved during pregnancy without cirrhosis. However, postpartum exacerbations are common due to immune reactivation. Some cases even require early transplantation due to decompensated cir-

rhosis [9] [10].

Several series highlight that disease worsening can occur both during pregnancy and postpartum, with severe maternal complications reported, including death and transplantation [11] [12]. Postpartum flares are particularly frequent, reported in up to 52% of patients [9] [11]-[13]. Cases of initial AIH diagnosis during postpartum, especially after preeclampsia, have also been described [14]. Our patient developed type 1 AIH postpartum in the context of pregnancy-induced hypertension without proteinuria.

Postpartum immune reactivation may explain this. AIH is often stable during pregnancy but flares within 3 months after delivery. A small Australian series reported first AIH flares within 4 months postpartum in 5 women aged 27 - 38. Four had severe flares (bilirubin >80 μmol/L, ALT 297 - 1393 U/L), all ANA-positive, and responded to treatment within 4 - 12 months [15].

Corticosteroids are the most used treatment during pregnancy. However, maternal/obstetric complications include cataract, osteoporosis, gestational diabetes, preeclampsia, and premature rupture of membranes. Doses >20 mg/day or use at conception may increase prematurity risk (RR: 1.8) [13]. High-dose corticosteroids (1 - 2 mg/kg) in the first trimester may increase cleft palate risk [16].

Azathioprine appears relatively safe in pregnancy and well tolerated by mother and fetus [11]. While rare cases of intrauterine growth restriction and prematurity are reported, no major impact on neonatal immunity is noted. Schramm *et al.* found no significant difference in pregnancy outcomes between azathioprine-treated and untreated groups [12]. The placenta acts as a partial barrier, resulting in lower fetal than maternal drug levels. However, data remain limited and evidence is still low [17].

Azathioprine may be a therapeutic option during pregnancy, provided maternal-fetal monitoring is ensured. Close monitoring is essential in the third trimester and postpartum, when disease flares are most common [2]. In our case, the patient declined treatment and follow-up.

4. Conclusion

Autoimmune hepatitis presents significant challenges in the perinatal period due to the complex interplay of immunological changes during pregnancy and postpartum. While pregnancy may induce disease remission, the postpartum period is a critical time for potential severe flares. Prompt diagnosis, careful monitoring, and individualized immunosuppressive therapy are vital to optimize maternal and fetal outcomes. Patient education and adherence to therapy are crucial, as untreated AIH carries a poor prognosis with risk of liver failure. Further prospective studies are warranted to clarify optimal management strategies during pregnancy and postpartum.

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

References

[1] Sarkar, M., Brady, C.W., Fleckenstein, J., Forde, K.A., Khungar, V., Molleston, J.P., *et al.* (2021) Santé reproductive et maladies du foie: Guide pratique de l'Association américaine pour l'étude des maladies du foie. *Hépatologie*, **73**, 318-365.

[2] Pena Polanco, N.A. and Levy, C. (2024) Autoimmune Hepatitis and Pregnancy. *Clinical Liver Disease*, **23**, e0112. https://doi.org/10.1097/cld.0000000000000112

[3] Manns, M.P., Czaja, A.J., Gorham, J.D., Krawitt, E.L., Mieli-Vergani, G., Vergani, D., *et al.* (2010) Diagnosis and Management of Autoimmune Hepatitis. *Hepatology*, **51**, 2193-2213. https://doi.org/10.1002/hep.23584

[4] Trivedi, P.J. and Hirschfield, G.M. (2021) Recent Advances in Clinical Practice: Epidemiology of Autoimmune Liver Diseases. *Gut*, **70**, 1989-2003. https://doi.org/10.1136/gutjnl-2020-322362

[5] Braga, A., Vasconcelos, C. and Braga, J. (2020) Autoimmune Hepatitis and Pregnancy. *Best Practice & Research Clinical Obstetrics & Gynaecology*, **68**, 23-31. https://doi.org/10.1016/j.bpobgyn.2020.03.007

[6] Hennes, E.M., Zeniya, M., Czaja, A.J., Parés, A., Dalekos, G.N., Krawitt, E.L., *et al.* (2008) Simplified Criteria for the Diagnosis of Autoimmune Hepatitis. *Hepatology*, **48**, 169-176. https://doi.org/10.1002/hep.22322

[7] Tran, T.T., Ahn, J. and Reau, N.S. (2016) ACG Clinical Guideline: Liver Disease and Pregnancy. *American Journal of Gastroenterology*, **111**, 176-194. https://doi.org/10.1038/ajg.2015.430

[8] Ludvigsson, J.F., Marschall, H.U., Hagström, H., Höijer, J. and Stephansson, O. (2018) Issue de la grossesse chez les femmes subissant une biopsie hépatique pendant la grossesse: Une étude de cohorte nationale basée sur la population. *Hépatologie*, **68**, 625-633.

[9] Malhotra, B., Malhotra, N., Deka, D. and Takkar, D. (2002) Immunosuppressive Effect of Pregnancy on Autoimmune Hepatitis: A Case Report and Review of Literature. *European Journal of Obstetrics & Gynecology and Reproductive Biology*, **101**, 91-92. https://doi.org/10.1016/s0301-2115(01)00509-7

[10] Laifer, S.A., Abu-Elmagd, K. and Fung, J.J. (1997) Hepatic Transplantation during Pregnancy and the Puerperium. *The Journal of Maternal-Fetal Medicine*, **6**, 40-44. https://doi.org/10.1002/(sici)1520-6661(199701/02)6:1<40::aid-mfm8>3.0.co;2-s

[11] Heneghan, M.A. (2001) Management and Outcome of Pregnancy in Autoimmune Hepatitis. *Gut*, **48**, 97-102. https://doi.org/10.1136/gut.48.1.97

[12] Schramm, C., Herkel, J., Beuers, U., Kanzler, S., Galle, P.R. and Lohse, A.W. (2006) Pregnancy in Autoimmune Hepatitis: Outcome and Risk Factors. *The American Journal of Gastroenterology*, **101**, 556-560. https://doi.org/10.1111/j.1572-0241.2006.00479.x

[13] Tanaka, H., Umekawa, T., Kikukawa, T. and Toyoda, N. (2002) Autoimmune Hepatitis Complicated with Antiphospholipid Syndrome in Pregnancy. *American Journal of Reproductive Immunology*, **47**, 142-145. https://doi.org/10.1034/j.1600-0897.2002.1c082.x

[14] Carson, M.P., Smulian, J.C. and Fedorciw, B. (2003) Autoimmune Hepatitis: Diagnosis after Preeclampsia Induced Elevated Liver Enzymes Failed to Normalize Postpartum. *Obstetrics & Gynecology*, **101**, 1118-1120. https://doi.org/10.1097/00006250-200305001-00026

[15] Samuel, D., Riordan, S., Strasser, S., Kurtovic, J., Singh-grewel, I. and Koorey, D.

(2004) Severe Autoimmune Hepatitis First Presenting in the Early Post Partum Period. *Clinical Gastroenterology and Hepatology*, **2**, 622-624. https://doi.org/10.1016/s1542-3565(04)00245-9

[16] Østensen, M. (2004) Disease Specific Problems Related to Drug Therapy in Pregnancy. *Lupus*, **13**, 746-750. https://doi.org/10.1191/0961203303lu2004oa

[17] de Boer, N.K.H., Jarbandhan, S.V.A., de Graaf, P., Mulder, C.J.J., van Elburg, R.M. and van Bodegraven, A.A. (2006) Azathioprine Use during Pregnancy: Unexpected Intrauterine Exposure to Metabolites. *The American Journal of Gastroenterology*, **101**, 1390-1392. https://doi.org/10.1111/j.1572-0241.2006.00538.x

Appendix

Table S1. Index of laboratory biological values of the CHU Ibn Rochd laboratory.

Bilan	Valeur normale
ASAT	<35 UI/l
ALAT	<35 UI/l
PAL	33 - 98 UI/l
BT	3 - 12 mg/l
Lipase	67 UI/l
Créatinine	5.7 - 11.1 mg/l
Ac anti-F-actine	<1: 40
TP	70% - 140%
Protéinurie de 24H	<0.3 gr/24H
Glycémie casuelle	<1.4 mg/l
Leucocytes	4000 - 1000/µl
Facteur V	70% - 150%