



Concurrent Celiac Disease and Rotavirus Infection During Pregnancy

Khushbu Bhatnagar^{1, #}, Sumaiya Khan¹, Hana Maheen Shashavali¹, Goulia Ohan², Mary Ter-Stepanyan²

¹ Faculty of General Medicine, Yerevan State Medical University after Mkhitar Heratsi, 2 Koryun, Yerevan 0025, Armenia

² Department of Epidemiology, Yerevan State Medical University after Mkhitar Heratsi, 2 Koryun, Yerevan 0025, Armenia

Abstract: Celiac Disease (CD) is an autoimmune condition marked by steatorrhea, weight loss and other signs of malnutrition due to gluten intolerance. We describe the clinical history of a 32-year-old CD patient who was pregnant and diagnosed with rotaviral (RV) enterocolitis. In resource-constrained countries, it is difficult to diagnose CD as it can be confused with infections that can cause similar symptoms such as infective gastroenteritis, ischemic enteritis and giardiasis. This case report highlights the need for a high degree of suspicion while approaching patients having poor nutritional status with little to no improvement on taking medication. Cases of RV infection in pregnancy have been poorly documented.

Keywords: Celiac disease; Rotaviral enterocolitis; Malnutrition; Gluten intolerance; Pregnancy.

Introduction

We present a rare case of celiac disease (CD) in pregnancy complicated by rotaviral enterocolitis. Pregnancy is characterized by significant changes in immune and gastrointestinal (GI) function, and these physiological shifts can amplify the complexity of managing coexisting conditions.

CD is a chronic autoimmune disorder in genetically predisposed individuals, where gluten ingestion provokes immune-mediated damage to the small intestine (SI), leading to villous atrophy, nutrient malabsorption, and a wide range of GI and extraintestinal manifestations such as diarrhea, bloating, and anemia^[1]. In women of reproductive age, CD carries additional implications, being linked to miscarriage, pregnancy-related anemia, low-birth-weight infants, and infertility. While a gluten-free diet may mitigate some risks, most patients report receiving inadequate counseling about the reproductive consequences of the disease^[2].

Diagnosis of CD often relies on GI endoscopy, which is considered a gold-standard tool^[3]. However, its use in pregnancy remains controversial due to limited safety data and concerns about teratogenic effects and the potential to precipitate preterm labor^[4]. This diagnostic uncertainty further complicates management during pregnancy, particularly when symptoms overlap with other GI infections.

One such infection is caused by rotavirus, a double-stranded RNA virus primarily spread via the fecal–oral route. Despite widespread vaccine use, rotavirus continues to be the leading cause of severe gastroenteritis (GE) and dehydration in children under five, with the greatest burden in low-income countries^[5]. In

pregnancy, any maternal infection is an area of particular concern as it can contribute to maternal morbidity and mortality while also increasing the risk of miscarriage, stillbirth, preterm labor, and low birth weight (LBW)^[6].

The coexistence of CD and RV infection therefore presents a diagnostic (See Table 1) and therapeutic challenge, as both conditions share and intensify GI symptoms (See Table 2). With limited literature addressing the interplay of CD and RV infection in pregnancy, little is known about their combined effect on maternal and fetal outcomes. This case report seeks to raise awareness among clinicians about the importance of timely recognition and management of CD in pregnancy, particularly when it may be masked or exacerbated by concurrent infections.

Case presentation

Patient Information

A 32-year-old previously healthy woman, 18 weeks pregnant with twins, was admitted to the Mother and Child Health Research Centre in Armenia. She was a multipara with a history of two normal deliveries and two miscarriages (G5P2A2).

History of Present Illness

She presented with profuse diarrhea, nausea, vomiting, low-grade fever (37.5 °C), and a sensation of heaviness in her legs. Prior to admission, she had self-medicated with over-the-counter agents including Enterol (*Saccharomyces boulardii*), Enterogermina (probiotic), Diosmectite, and Mesim Forte (pancreatic enzymes), without relief.

Vitals on Admission

- Blood pressure: 90/60 mmHg
- Pulse: 120 bpm
- Oxygen saturation: 97% on room air
- Temperature: 37.5 °C

Correspondence: Khushbu Bhatnagar. E-mail: kbhatnagar3@gmail.com. Address: Faculty of General Medicine, Yerevan State Medical University after Mkhitar Heratsi, 2 Koryun, Yerevan 0025, Armenia.

Received 10 May 2025; Revised 24 September 2025; Accepted 24 November 2025; Available Online 10 December 2025

DOI: [10.54457/DR.202504002](https://doi.org/10.54457/DR.202504002)

© The Author(s) 2026. This is an open access article under the CC BY 4.0 license (<https://creativecommons.org/licenses/by/4.0/>).

Table 1. Differential Diagnosis in a Pregnant Patient with Persistent Diarrhea.

Condition	Overlapping Features with this Case	Distinguishing Features / Key Differentiators	Confirmatory Test
Celiac Disease (CD)	Diarrhea, malabsorption, weight loss, anemia, bloating.	Triggered by gluten; extra-intestinal manifestations (e.g., dermatitis herpetiformis); family history.	Serology (anti-tTG, EMA), duodenal biopsy (gold standard).
Rotavirus Enterocolitis	Acute watery diarrhea, vomiting, fever, dehydration.	Acute onset; often self-limiting; common in children but atypical severe course in adults.	Stool PCR / Antigen test.
Giardiasis	Steatorrhea, malabsorption, nausea.	Often associated with bloating and sulfurous burping; endemic areas.	Stool ova and parasite exam.
Hyperemesis Gravidarum	Nausea, vomiting, dehydration, ketonuria.	Typically in 1st trimester; absence of infectious diarrhea.	Clinical diagnosis; exclusion of other causes.
HELLP Syndrome	Nausea, vomiting, elevated LFTs, edema.	Hypertension, proteinuria; thrombocytopenia; hemolysis.	CBC, LFTs, blood smear.

Table 2. Pathophysiological Overlap and Synergy of Celiac Disease and Rotavirus Infection.

Pathophysiological Mechanism	Effect in Celiac Disease	Effect in Rotavirus Infection	Combined Effect in this Case
Villous Atrophy	Immune-mediated, chronic flattening of intestinal villi.	Acute, virus-induced damage and shortening of villi.	Severe, compounded malabsorption of nutrients, vitamins, and electrolytes.
Malabsorption	Chronic deficiency of iron, vitamins, albumin, electrolytes.	Acute osmotic diarrhea leads to rapid fluid and electrolyte loss.	Profound dehydration, hypoalbuminemia (edema), and severe electrolyte imbalances.
Inflammatory Response	Chronic, T-cell mediated inflammation.	Acute innate immune response and cytokine release.	Enhanced systemic inflammation, potentially contributing to symptoms like fever and malaise.
Mechanism of Diarrhea	Osmotic (unabsorbed nutrients) and secretory components.	Primarily secretory (NSP4 toxin) a	

Physical Examination

- **General:** Alert, oriented, dry tongue
- **Respiratory:** Vesicular breath sounds, no added sounds
- **Abdomen:** Hepatomegaly; otherwise unremarkable
- **Neurological:** No meningeal or focal signs
- **Genitourinary:** Dark-colored urine

Investigations

Abdominal ultrasound was unremarkable.

During her hospital stay, she developed anorexia and jaundice, while diarrhea and vomiting persisted. Her stool was classified as type 7 on the Bristol Stool Scale—yellow, watery, and containing undigested food. By the third day of admission, she developed bilateral leg edema extending to the upper third of her thighs (See Fig. 1). Laboratory studies revealed ketonuria and elevated liver transaminases (ALT, AST). On the fifth day, PCR confirmed rotavirus (RV) infection, and stool culture was positive for *Candida* (See Fig. 2).

She was subsequently transferred to the Infectious Diseases Department of *Mikaelyan Hospital*, where Celiac Disease (CD) was considered as a differential diagnosis. Gastroscopy was not performed due to pregnancy being a relative contraindication. She was started on a gluten-free diet along with infusion therapy, including potassium chloride (4% 50 mL), albumin (20% 100 mL), tranexamic acid (10 mg), Chophytol (artichoke extract for hepatobiliary support), vitamin K (100 mg), and Sorbifer (ferrous sulfate with vitamin C).

Due to financial constraints, serological testing was not done to confirm the diagnosis of CD. Her condition improved, and she was transferred back to the *Mother and Child Health Research Centre* for continued symptomatic management. Multidisciplinary review of her nutritional status and laboratory results led to

the decision not to terminate the pregnancy.

She was discharged at 21 weeks of gestation in improved condition. However, a few weeks later, her health deteriorated, and she delivered vaginally at 28 weeks. Unfortunately, both neonates passed away shortly after birth in the Neonatal Intensive Care Unit (NICU) (See Table 3 for a summary).



Fig. 1. Bilateral Lower-Limb Edema in the Patient.
The figure shows pronounced bilateral pitting edema extending to the upper thighs, observed on day three of admission. This reflected severe hypoalbuminemia and fluid imbalance in the context of concurrent celiac disease and rotavirus infection.

Confirmation of Rotavirus infection in a Pregnant Patient with concurrent celiac Disease	
Day of Hospital Stay	Event/Observation
Day 1	Admission with severe diarrhea, nausea, vomiting, dehydration; empiric treatment started
Day 3	Bilateral lower limb edema up to upper thigh, elevated liver enzymes, ketonuria
Day 5	PCR test positive for Rotavirus; stool culture positive for Candida

Fig. 2. Laboratory Confirmation of Rotavirus Infection.
Stool PCR test confirming rotaviral infection in the patient, supporting the diagnosis of acute rotavirus enterocolitis complicating underlying celiac disease.

Table 3. Clinical Timeline, Diagnostic Findings, and Interventions.

Timeline	Symptoms & Signs	Key Diagnostic Findings	Interventions & Management
Pre-admission	Profuse diarrhea, nausea, vomiting, low-grade fever.	Self-medication ineffective.	Enterol, Enterogermina, Diosmectite, Mesim Forte.
On Admission (Wk 18)	BP: 90/60 mmHg, Pulse: 120 bpm, dry tongue, hepatomegaly.	Ultrasound: Unremarkable.	Supportive care (likely initial IV fluids).
During Hospital Stay	Anorexia, jaundice, persistent diarrhea (Bristol 7), bilateral leg edema.	Ketonuria, elevated ALT/AST.	Continued supportive care.
Day 5	Symptoms persist.	PCR+: Rotavirus. Stool culture: <i>Candida</i> +	Transferred to Infectious Diseases Dept.
Post-Transfer		CD suspected (clinically). Gastroscopy not performed.	Gluten-free diet initiated. Infusion therapy: KCl, Albumin, Tranexamic acid, Chophytol, Vitamin K, Sorbifer. Condition improved.
Discharge (Wk 21)	Improved condition.		Discharged for continued symptomatic management.
Outcome (Wk 28)	Health deteriorated.		Vaginal delivery at 28 weeks. Both neonates deceased.

Discussion

CD is an autoimmune disorder having a large degree of clinical polymorphism with classical, asymptomatic, oligosymptomatic and extra-intestinal forms^[7]. This makes it hard to diagnose, leading to the chance of developing serious complications in the patients^[8].

In children, CD usually manifests in its classical form, characterized by diarrhea and malabsorption, while in adults it often appears as a non-classical form, presenting with extra-intestinal signs such as anemia and fatigue. The subtle and variable symptoms in adults often make diagnosis difficult, resulting in delays or missed cases. If left undiagnosed, CD can lead to serious complications, including malignancies and infertility, and is associated with increased healthcare utilization and psychological strain^[9].

The patient consumed gluten-rich food throughout her pregnancy, potentially being the cause of her preterm labour. There are two main hypotheses for the reasons for negative pregnancy outcomes in CD. The first one states that there is an antibody-me-

diated reaction concluded by the link seen between CD activity and reproductive health. There is damage to the cytoskeleton of the endometrial endothelial cells due to the trophoblasts being attacked by anti-tTG (Anti-Tissue Transglutaminase) antibodies^[10]. The second one talks about the link between mineral and vitamin deficiencies seen in CD and increased spontaneous abortion risk^[11] (See Fig. 3).

A retrospective cohort study found that the extra-intestinal manifestations of a woman’s reproductive system in CD can include spontaneous abortions and premature deliveries^[12].

The only treatment for CD is a gluten-free diet. That is why her initial treatment with Enterol and Enterogermina, which are probiotic antidiarrheal medications, had no effect since gluten was still present in the diet. After changing hospitals, she was put on the right diet alongside infusions of Vitamin K, Albumin, Sorbifer (ferrous sulphate) and KCl to improve her electrolyte balance, protein levels and vitamin deficiencies.

RV was first discovered in 1973 as distinct viral particles found in the duodenal mucosa of children with GE^[13]. It has been one of the leading non-bacterial causes of paediatric GE.

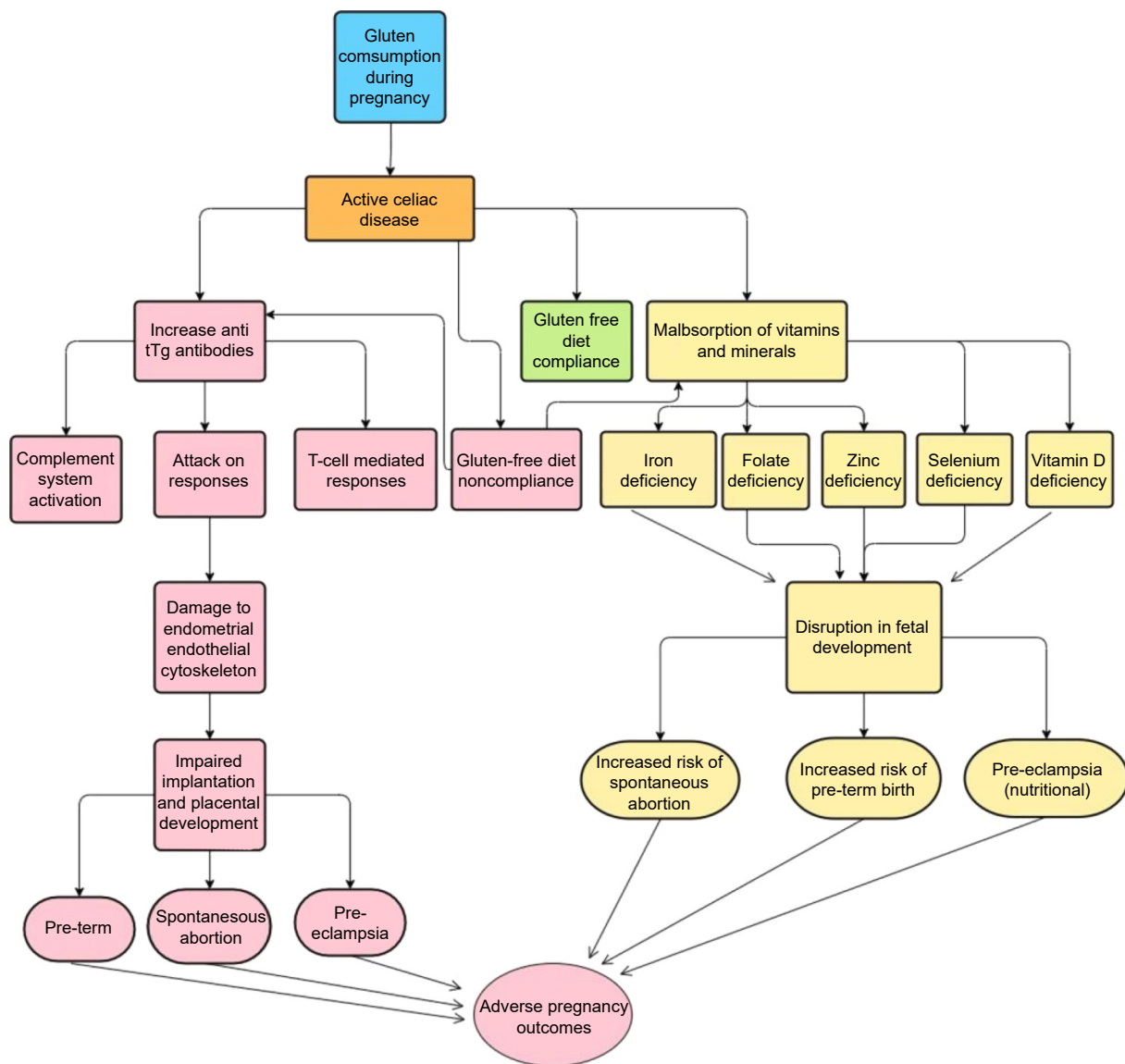


Fig. 3. Proposed Mechanisms Linking Celiac Disease to Adverse Pregnancy Outcomes.

Diagram illustrating the two major hypotheses: (1) antibody-mediated trophoblast and placental damage due to anti-tTG antibodies, and (2) maternal micronutrient deficiencies contributing to miscarriage, preterm birth, and fetal growth restriction.

However, adults show mild or even no manifestations of the disease with quick resolution of the symptoms given partial immunity from previous infections. The infection takes a severe course only when exacerbated by a concurrent GI condition like in our patient's case.

RV primarily targets the GI epithelium, leading to acute GE with prominent diarrhea and vomiting. Diarrhea results from multiple mechanisms, including NSP4-induced chloride secretion, enterocyte injury with villus atrophy and microvilli loss, and impaired nutrient absorption, producing osmotic diarrhea^[14].

Additionally, RV triggers serotonin release from enterochromaffin cells, stimulating enteric nerves and vagal pathways, which enhances intestinal motility and contributes to nausea and vomiting. In adults, particularly in high-risk groups such as pregnant women, these effects can cause severe dehydration, electrolyte disturbances, and systemic symptoms like jaundice and malaise^[14].

In this case, a 32-year-old woman carrying twins experienced persistent diarrhea, vomiting, jaundice, and lower limb edema, underscoring the potential severity of RV infection in adults and the need for timely supportive care.

According to the WHO guidelines, the treatment for RV infection is purely symptomatic including oral, intravenous or rehydration salts. Antibiotics and antivirals are ineffective. The role of RV as a pathogen in adults has long been underappreciated. Clinicians often miss out on RV in the differential diagnosis of acute GE which leads to treatment delay and complications^[15].

The purpose of this case report is to educate clinicians on including CD in the differential diagnosis of all patients with symptoms of any GI infection, especially in pregnant women. The treating doctors didn't perform a gastroscopy for the diagnosis of CD, which led to further delays in the patient receiving proper treatment and advice to avoid gluten-rich food.

The diagnosis of RV enterocolitis was also made late as it is

uncommon for an adult to have such serious manifestations of the disease. All this points to a need for better education in diagnosing atypical diseases or diseases with atypical presentations.

To our knowledge, a case of concurrent RV gastroenteritis and celiac disease has never been reported and needs to be brought to the attention of physicians.

Conclusion

This case underscores the clinical significance of RV infection in adults, particularly during pregnancy, a population in which its impact is often underrecognized. The patient's presentation with persistent diarrhea, vomiting, jaundice, and lower limb edema illustrates how even mild GI infections can lead to severe systemic complications in the context of pregnancy.

Early identification and management of both RV infection and CD could have potentially mitigated maternal morbidity and improved fetal outcomes. However, the limited data on adult RV infections, especially in pregnant women, may prevent clinicians from considering it in the differential diagnosis, leading to delays in appropriate intervention. This emphasizes the importance of heightened clinical awareness and vigilance for GI infections in high-risk adult populations.

Additionally, dietary management and patient education regarding nutrition are critical components of care, particularly when underlying conditions such as CD may exacerbate malabsorption and dehydration.

From a public health perspective, developing countries like Armenia should re-evaluate and update their standard guidelines for diagnosing and managing GI disorders in adults, with special attention to pregnant patients. Intrating such strategies can help prevent severe maternal complications, improve pregnancy outcomes, and ensure timely, evidence-based care for vulnerable populations.

Abbreviations

ALT, alanine aminotransferase; AST, aspartate aminotransferase; BP, blood pressure; bpm, beats per minute; CBC, complete blood count; CD, celiac disease; G5P2A2, gravida 5 para 2 abortus 2; GE, gastroenteritis; GI, gastrointestinal; HELLP, hemolysis, elevated liver enzymes, low platelets; KCl, potassium chloride; LBW, low birth weight; LFTs, liver function tests; NICU, neonatal intensive care unit; NSP4, nonstructural protein 4; PCR, polymerase chain reaction; RV, rotavirus; SI, small intestine; tTG, tissue transglutaminase.

Ethical approval and consent to participate

The informed consent form was obtained from the patient for publishing the case.

Authors' contributions

KB, SK and HMS: Wrote and reviewed the manuscript, analyzed patient data. KB: Prepared all figures and tables. GO and MTS: Critically reviewed the manuscript and supervised the project. All authors approved the final version.

References

- [1] Arvanitakis K, Siargkas A, Germanidis G, et al. Adverse pregnancy outcomes in women with celiac disease: a systematic review and meta-analysis. *Ann Gastroenterol*, 2022, 36(1): 12.
- [2] Arcieri M, Abrami C, Graziano A, et al. The influence of celiac disease on fertility and pregnancy: an Italian survey. *Arch Gynecol Obstet*, 2024, 310(6): 2907-2914.
- [3] Shiha MG, Penny HA, Sanders DS. Is there a need to undertake conventional gastroscopy and biopsy when making the diagnosis of coeliac disease in adults? *J Clin Gastroenterol*, 2023, 57(2): 139-142.
- [4] Savas N. Gastrointestinal endoscopy in pregnancy. *World J Gastroenterol*, 2014, 20(41): 15241-15252.
- [5] LeClair CE, McConnell KA. Rotavirus; In: StatPearls [Internet]. Available from: <https://www.ncbi.nlm.nih.gov/books/NBK558951/>. [Last accessed on 2 Jan 2023]
- [6] Kumar M, Saadaoui M, Al Khodor S. Infections and pregnancy: effects on maternal and child health. *Front Cell Infect Microbiol*, 2022, 12: 873253.
- [7] Ben Houmich T, Admou B. Celiac disease: Understandings in diagnostic, nutritional, and medicinal aspects. *Int J Immunopathol Pharmacol*, 2021, 35: 20587384211008709.
- [8] Meena DS, Kumar D, Bohra GK, et al. Hypoalbuminemia and generalized edema as an atypical presentation of celiac disease. *J Fam Med Prim Care*, 2020, 9(2): 1206-1208.
- [9] Majsiak E, Choina M, Gray AM, et al. Clinical manifestation and diagnostic process of celiac disease in Poland —comparison of pediatric and adult patients in retrospective study. *Nutrients*, 2022, 14(3): 491.
- [10] Anjum N, Baker PN, Robinson NJ, et al. Maternal celiac disease autoantibodies bind directly to syncytiotrophoblast and inhibit placental tissue transglutaminase activity. *Reprod Biol Endocrinol*, 2009, 7: 16.
- [11] Di Simone N, Silano M, Castellani R, et al. Anti-tissue transglutaminase antibodies from celiac patients are responsible for trophoblast damage via apoptosis in vitro. *Am J Gastroenterol*, 2010, 105(10): 2254-2261.
- [12] Moleski SM, Lindenmeyer CC, Veloski JJ, et al. Increased rates of pregnancy complications in women with celiac disease. *Ann Gastroenterol*, 2015, 28(2): 236-240.
- [13] Bishop RF, Davidson GP, Holmes IH, et al. Virus particles in epithelial cells of duodenal mucosa from children with acute non-bacterial gastroenteritis. *Lancet*, 1973, 2(7841): 1281-1283.
- [14] Pawluszkievicz K, Ryglowski PJ, Idzik N, et al. Rotavirus Infections: Pathophysiology, Symptoms, and Vaccination. *Pathogens*, 2025, 14(5): 480.
- [15] Guerrant RL, Van Gilder T, Steiner TS, et al. Practice guidelines for the management of infectious diarrhea. *Clin Infect Dis*, 2001, 32(3): 331-351.