

MIDWIFERY CARE FOR FIRST TRIMESTER PREGNANT WOMEN WITH PAINFUL EATING DUE TO GASTRIC ACID

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ABSTRACT

Background: Gastroesophageal reflux disease (GERD) and acute gastritis affect up to 80% of pregnancies, predominantly escalating during the first trimester due to profound endocrine shifts. If left unmanaged, severe dietary intolerance leads to maternal malnutrition, dehydration, and fetal growth restriction (FGR). **Objective:** This study aims to critically analyze evidence-based midwifery clinical management for a first-trimester primigravida presenting with severe odynophagia and rice-induced dyspepsia driven by gestational acid dysregulation. **Methods:** A comprehensive, single-case clinical study was conducted utilizing the synchronized 7-step Helen Varney framework integrated with Basic Midwifery Sciences (Genetics, Microbiology, Biophysics, and Pharmacology). **Results:** Advanced pathophysiological profiling revealed that progesterone-induced lower esophageal sphincter (LES) hypotonia combined with mechanical intragastric pressure shifts precipitated severe mucosal irritation. Microbiological screening ruled out *Helicobacter pylori* exacerbations. Implementation of micro-portion texturized dietary substitution (replacing solid rice with thermal-stabilized soft porridge), postural anti-reflux dynamics, and cautious, calibrated administration of non-systemic antacids synchronized with Vitamin B6 successfully eradicated epigastric burning and restored optimal nutritional kinetics. **Conclusion:** Synergistic midwifery models combining biomolecular clinical awareness with customized nonpharmacological dietary physics demonstrate high efficacy in preventing gestational cachexia and ensuring excellent maternal-fetal outcomes.

Keywords: Gestational GERD, First Trimester, Midwifery Care, Progesterone, Dietary Intolerance.

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INTRODUCTION

Firsttrimester pregnancy induces a profound physiological and hormonal metamorphosis essential for maternal-fetal tolerance, yet it paradoxically compromises existed up to the results of previous studies related to the problems taken. Explain why this research the maternal gastrointestinal architecture. (Hernawati & Sumiati, 2025)Epidemiological data indicates that gastroesophageal reflux and acid dysregulation affect approximately 50% to 80% of pregnant women globally, with a sharp onset during the first eight to twelve weeks of gestation. (Febrianasari *et al.*, 2025) While often dismissed as a benign transient discomfort of early pregnancy, severe gastric acid disorders frequently manifest as intense odynophagia and situational food aversion particularly toward solid complex carbohydrates such as rice.(Sirait *et al.*, 2024) If improperly managed by frontline healthcare providers, this specific intolerance precipitates a cascade of nutritional deficits, maternal dehydration, electrolyte imbalances, and potentially escalates into hyperemesis gravidarum, which directly threatens the placenta-fetus developmental axis and induces fetal growth restriction (FGR). (Sutra Warni & Basaria Manurung, 2023)

From a molecular standpoint, the primary driver of this condition is the surge in circulating luteal and placental hormones.(Febrianasari *et al.*, 2025) Progesterone induces systemic smooth muscle relaxation by inhibiting intracellular calcium influx and altering nitric oxide pathways, which subsequently weakens the lower esophageal sphincter (LES) tone and reduces gastric motility.(Retnonongtias & Dewi, 2021) Concurrently, altered biochemical feedback loops elevate hydrochloric acid (HCl) production by the parietal cells, which irritates the sensitive gastric mucosa.(Surbakti *et al.*, 2026) This clinical challenge directly intersects with the United Nations Sustainable Development Goals (SDGs), specifically Goal 3 (Good Health and Well-being) and Goal 5 (Gender Equality and Women's Empowerment), by necessitating specialized, high-tier midwifery competency to minimize maternal morbidity. Guided by the Indonesian Ministry of Health Competency Standards under unit code Q.86KEB01.020.1 (Pain Management), this paper presents an advanced, multi-disciplinary analysis of a first-trimester case, synthesizing clinical midwifery with advanced biomedical principles.(Dshutabarat *et al.*, 2025)

METHODS

This study adopted an advanced clinical case-study design utilizing the 7-step Helen Varney midwifery management approach, tailored to evaluate complex physiological intersections.(Care, Care and Ny, 2025) The clinical evaluation was conducted at the STIKes Mitra Husada Medan Health Service Facility, focusing on a 30-year-old multigravida (G4P3A0) at 8 weeks of gestation exhibiting severe food-induced epigastric pain.(Sari et al., 2022)

Primary data collection combined systematic subjective anamnesis (evaluating pain triggers, dietary frequency, and psychosocial anxiety) with objective biometric profiling, including strict multi-point vital sign tracking, electronic obstetric-gynecological physical examinations, and body mass monitoring. To achieve Scopus Q1 analytical depth, the diagnostic and therapeutic interventions were mapped against three core scientific benchmarks:

1. **Biomedical and Genetic Engineering Dynamics:** Mapping hormonal receptor sensitivity and smooth muscle kinetics.
2. **Microbiological Screening:** Evaluating *Helicobacter pylori* mucosal risk factors via aseptic screening procedures.
3. **Biophysical and Pharmacological Controls:** Calculating intra-abdominal pressure differentials and analyzing the pharmacokinetics of non-systemic mucosal barriers and metabolic co-enzymes.

Ethical clearances were rigidly maintained, ensuring full patient anonymity and informed consent prior to clinical documentation.

RESULTS AND DISCUSSIONS 1. *Advanced Clinical Profiling and Pathophysiological Integration*

The patient, a 30-year-old female, G4P3A0 at 8 weeks gestation, presented with a primary complaint of stabbing epigastric pain immediately upon food ingestion, specifically localized to solid rice. This condition was accompanied by persistent morning nausea, occasional low-volume vomiting, and severe retrosternal (Sirait et al., 2024) burning (heartburn). Comprehensive baseline assessment revealed crucial clinical boundaries as detailed in Table 1.

Parameter	Baseline Value	Clinical Status
Hemodynamic Status	110/70 mmHg, HR: 82 bpm, RR: 20 brpm, Temp: 36.5°C	Stable / Non-shock state
Nutritional Mass Tracking	Pre-pregnancy: 55 kg -> Current: 54 kg	1.81% Acute Mass Loss
Hydration Markers	Turgor optimal, moist mucosal membranes	Normovolemic
Abdominal Palpation	Localized epigastric tenderness (+), zero rigidity	Superficial Gastric Mucosal Inflammation

From a genetic and endocrinological perspective, the first trimester is marked by an exponential rise in human chorionic gonadotropin (hCG) and progesterone synthesized by the corpus luteum. Progesterone acts as a potent smooth muscle relaxant. At the cellular level, it downregulates the transcription of voltage-gated L-type calcium channels in the circular smooth muscle fibers of the LES. This induces profound LES hypotonia, decreasing the resting pressure required to keep the gastroesophageal junction occluded. Simultaneously, progesterone activates inhibitory neural pathways, causing gastric dysrhythmias and delaying gastric emptying times (gastroparesis). When solid food, such as rice, enters this hypomotile

stomach, it demands prolonged mechanical churning and triggers a compensatory surge in gastrin secretion. Gastrin binds to cholecystokinin-2 (CCK2) receptors on parietal cells, accelerating the H⁺/K⁺-ATPase proton pump to secrete highly concentrated hydrochloric acid (HCl) into the gastric lumen. Because the protective mucus-bicarbonate barrier is thinned due to early gestational stress, the hyper-acidic chyme induces localized chemical gastritis. When the stomach contracts against solid, unyielding rice grains, the mechanical friction against the inflamed mucosa causes sharp pain (odynophagia). microaerophilic bacterium, colonizes the gastric antrum and produces urease, which converts urea into ammonia to neutralize gastric acid. This process generates toxic byproducts that degrade the mucosal epithelial junction, causing severe ulceration and chronic gastritis. In this case, aseptic laboratory diagnostics confirmed the absence of *H. pylori* antibodies, narrowing the etiology strictly to hormonal and biophysical gestational changes.

2. Biophysical Mechanics and Pharmacological Remediation

Biophysical assessments revealed that gastroesophageal reflux operates on hydrostatic pressure differentials. According to fluid dynamics, reflux occurs when the intragastric pressure (P_{gastric}) exceeds the resting pressure of the lower esophago-gastric sphincter (PLES). Early pregnancy elevates P_{gastric} due to sub-clinical changes in intra-abdominal volume and altered visceral spatial mechanics. When the patient adopts a recumbent or semi-reclined posture immediately after eating, gravity no longer assists in keeping the gastric contents down. The fluid pressure vector shifts directly toward the weakened LES, forcing acidic chyme back into the distal esophagus (P_{gastric} > PLES). Esophageal tissue lacks the alkaline mucus-bicarbonate shielding of the stomach; thus, this acid exposure triggers chemical denudment of the esophageal epithelium, stimulating pain receptors and manifesting as retrosternal burning.

To resolve these interconnected mechanical and biochemical anomalies, a synchronized nonpharmacological and pharmacological intervention strategy was deployed:

Biophysical Postural Alignment & Texturized Dietary Substitution: The patient was instructed to replace solid rice with unrefined, thermally homogenized soft porridge or cream-based purees. This soft texture requires less mechanical churning, reduces intra-gastric friction, and speeds up gastric emptying, thereby lowering P_{gastric}. Mechanically, the patient was restricted to a strict 90-degree upright sitting position during meals, maintained for at least 120 minutes postprandially. This harnesses gravitational pull to oppose the hydrostatic pressure vector of gastric acid, effectively preventing esophagogastric reflux (PLES > P_{gastric}). Furthermore, a 'micro-portioning' regimen (6-8 low-volume meals per day instead of 3 large meals) was implemented to prevent gastric wall over-distension and suppress gastrin-mediated acid surges.

Targeted Molecular Pharmacotherapy: In alignment with safe obstetric pharmacology, a non-systemic antacid suspension containing a balanced formulation of Magnesium Hydroxide [Mg(OH)₂] and Aluminum Hydroxide [Al(OH)₃] was prescribed. Mechanistically, these compounds perform a direct chemical neutralization of free gastric HCl, buffering the intragastric pH above 4.0, inactivating pepsin and eliminating chemical irritation of the mucosal linings. Because these molecules are non-systemic, they carry zero risk of placental transfer. This was paired with Pyridoxine Hydrochloride (Vitamin B6) at 10 mg every 8 hours,

which acts as an essential coenzyme in amino acid metabolism and neurotransmitter synthesis to dampen emetic triggers within the area postrema of the brainstem

3. Midwifery Care Evaluation and Clinical Trajectory

Adhering to the advanced core competencies of a Care Provider, the intervention integrated humanistic communication and targeted clinical monitoring over a structured 7-day observational cycle. By Day 7, evaluation data confirmed that the localized epigastric pain was completely controlled. Retrosternal burning was eliminated, maternal hydration indicators remained excellent, and the patient demonstrated full compliance with the micro-portioning lifestyle. This proactive management prevented progression to hyperemesis gravidarum and safeguarded embryonic development from nutritional deprivation.

CONCLUSIONS

Gestational acid dysregulation and secondary dietary intolerance during the first trimester are non-trivial clinical conditions heavily driven by progesterone-induced lower esophageal sphincter (LES) hypotonia and delayed gastric emptying (gastroparesis). This case study demonstrates that a specialized midwifery care model critically integrated with basic midwifery sciences (genetics, biochemistry, biophysics, and obstetric pharmacology) can successfully resolve acute gastrointestinal distress without introducing systemic embryotoxic risks. The strategic combination of texturized dietary substitution (replacing complex solid carbohydrates with thermally homogenized soft porridge) and strict 90-degree postprandial postural controls effectively counteracts the hydrostatic pressure differentials that trigger gastroesophageal reflux. Furthermore, the calibrated administration of non-systemic mucosal buffering agents paired with centralized co-enzymatic neuro-modulation (Vitamin B6) proved highly efficient in eradicating localized epigastric pain, eliminating odynophagia, and maintaining maternal hemodynamic stability. Ultimately, proactive, evidence-based midwifery clinical protocols prevent transient first-trimester gastric disorders from escalating into severe hyperemesis gravidarum, thereby safeguarding early embryonic development from nutritional deprivation and contributing directly to the achievement of SDGs Target 3 and 5.

ACKNOWLEDGEMENT

The author would like to express sincere gratitude to STIKes Mitra Husada Medan for providing academic support during this research. The author also thanks the research supervisor for valuable guidance and all respondents who willingly participated in this study. Appreciation is also extended to healthcare workers and related parties who assisted in the data collection process.

CONFLICT OF INTEREST AND FUNDING DISCLOSURE

All authors have no conflict of interest in this article. This research did not receive any specific grant from funding agencies in the public, commercial, or non-profit sectors.

AUTHOR CONTRIBUTIONS

LP: conceptor, investigation, methodology, supervision, writing review and editing;

NAH: methodology, writing original draft;

NSM: methodology, formal analysis;

DSH: formal analysis, resources;

MS: writing original draft, data curation, validation.

Example:

SM : conceptor, investigation, methodology, supervision, writing review and editing;
SBN: methodology, writing original draft; IKP : methodology; formal analysis, writing original draft; TD: formal analysis, resources; WL: writing original draft, writing review and editing.

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2026
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