

Late-Onset Pregnancy Vomiting: Etiologies and Management -A Study of 60 Cases

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1. Abstract

1.1. Background

Nausea and vomiting are among the most common complaints in early pregnancy, typically resolving by 16 weeks of gestation. However, late-onset vomiting occurring after 20 weeks represents a clinically distinct and potentially serious phenomenon that requires thorough etiological investigation and tailored management.

1.2. Objective

To identify the etiologist, clinical features, and management outcomes of late-onset vomiting during pregnancy in a cohort of 60 cases observed at our institution.

1.3. Methods

A retrospective descriptive study was conducted over 3 years (2022–2024) including all pregnant women presenting after 20 weeks of gestation with vomiting as the primary complaint. Clinical, biological, and radiological data were collected and analysed.

1.4. Results

Obstetric causes dominated, accounting for 58.3% of cases (preeclampsia 25%, acute fatty liver of pregnancy 13.3%, HELLP syndrome 10%). Non-obstetric causes were identified in 41.7% of cases, including gastrointestinal conditions (23.3%), urinary tract infections (10%), and acute appendicitis (8.4%). Maternal and fetal outcomes were significantly influenced by the underlying ethology and timeliness of diagnosis.

1.5. Conclusion

Late-onset pregnancy vomiting encompasses a wide spectrum of potentially life-threatening conditions. Systematic clinical and paraclinical evaluation is essential to guide appropriate management and optimize maternal-fatal outcomes.

2. Introduction

Nausea and vomiting of pregnancy (NVP) are among the most prevalent symptoms in obstetric practice, affecting approximately 70–80% of pregnant women, predominantly in the first trimester. These symptoms classically resolve by 16–20 weeks of gestation and are attributed to rising levels of human chorionic gonadotropin (hCG) and oestrogen.

Late-onset vomiting, defined here as vomiting first occurring or significantly worsening after 20 weeks of gestation, is a comparatively understudied yet clinically important entity. Unlike early-pregnancy emesis, late-onset vomiting rarely reflects a benign physiological process. It may signal serious obstetric complications such as preeclampsia, HELLP syndrome (Hemolysis, Elevated Liver enzymes, Low Platelets), acute fatty liver of pregnancy (AFLP), or may be the presenting symptom of an intercurrent surgical emergency such as acute appendicitis or bowel obstruction.

The diagnostic challenge lies in the broad differential diagnosis, the overlapping symptomatology with more common conditions, and the potentially catastrophic consequences of delayed recognition. Clinicians must maintain a high index of suspicion and pursue systematic evaluation to distinguish benign from life-threatening causes.

This study aims to analyse 60 consecutive cases of late-onset pregnancy vomiting admitted to our obstetric unit over a 3-year period, with a focus on etiological distribution, clinical characteristics, and management outcomes.

3. Materials and Methods

3.1. Study Design and Setting

This is a retrospective descriptive study conducted at the Department of Obstetrics and Gynaecology, Mother and Child Hospital Centre of Nouakchott, Mauritania, over a three-year period from

January 2022 to December 2024. The study was approved by the institutional ethics committee. All procedures complied with the Declaration of Helsinki principles.

3.2. Inclusion and Exclusion Criteria

Inclusion criteria were: (i) singleton or multiple pregnancy at ≥ 20 weeks of gestational age; (ii) vomiting as the primary presenting complaint requiring hospitalization or outpatient evaluation; (iii) complete clinical and biological workup available. Exclusion criteria included: prior documented hyperemesis gravidarum with persistent symptoms, known chronic gastrointestinal disease, and incomplete records.

3.3. Data Collection

Data were extracted from medical records including: demographic variables (age, parity, gestational age), clinical presentation (symptom onset, associated symptoms, vital signs), biological findings (complete blood count, liver function tests, renal function, urine analysis, coagulation profile), imaging studies (obstetric ultrasound, abdominal ultrasound, CT when applicable), clinical diagnosis, management strategy, and maternal-fatal outcome.

3.4. Outcome Measures

Primary outcomes included etiological diagnosis and maternal morbidity. Secondary outcomes included neonatal outcome (gestational age at delivery, birth weight, Apgar score, NICU admission), duration of hospitalization, and maternal mortality.

3.5. Statistical Analysis

Descriptive statistics were used throughout. Continuous variables are expressed as mean \pm standard deviation (SD) or median with interquartile range (IQR). Categorical variables are expressed as frequencies and percentages. Statistical analyses were performed using SPSS version 26.0 (IBM, Chicago, IL).

Table 1: Demographic and obstetric characteristics of the study cohort (n=60).

Total patients	60	100
Mean age (years)	29.4 \pm 5.8	—
Primigravidae	26	43.3
Multigravidae	34	56.7
Mean gestational age at onset (weeks)	30.2 \pm 6.1	—
Multiple gestations	5	8.3
Urban residence	47	78.3
Rural residence	13	21.7

Table 2: Etiological distribution of late-onset vomiting in pregnancy (n=60).

OBSTETRIC CAUSES	35	58.3
Preeclampsia with severe features	15	25
Acute fatty liver of pregnancy (AFLP)	8	13.3
HELLP syndrome	6	10
Acute hydramnios	4	6.7
Molar recurrence	2	3.3
NON-OBSTETRIC CAUSES	25	41.7
Acute gastroenteritis	7	11.7
Peptic ulcer disease / Gastritis	4	6.7
Acute cholecystitis	3	5
Urinary tract infection / Pyelonephritis	6	10
Acute appendicitis	5	8.4
TOTAL	60	100

Table 3: Maternal and fetal outcomes (n=60).

Outcome	N	%
Maternal ICU admission	11	18.3
Maternal mortality	1	1.7
Preterm delivery (<37 wks)	28	46.7
Emergency cesarean delivery	23	38.3
Mean birth weight (g)	2340 \pm 610	—
NICU admission	22	36.7
Perinatal mortality	3	5

4. Results

4.1. Demographic Characteristics

Over the study period, 60 patients met inclusion criteria. The mean age was 29.4 \pm 5.8 years (range: 18–43 years). Primigravidae represented 43.3% of the cohort (n=26), while 56.7% were multigravidae (n=34). The mean gestational age at symptom onset was 30.2 \pm 6.1 weeks. Multiple gestation was identified in 8.3% (n=5) of cases.

4.2. Etiological Distribution

Obstetric causes were identified in 35 patients (58.3%) and non-obstetric causes in 25 patients (41.7%). The leading obstetric cause was preeclampsia with severe features (n=15, 25.0%), followed by acute fatty liver of pregnancy (n=8, 13.3%), HELLP syndrome (n=6, 10.0%), and acute hydramnios (n=4, 6.7%). Two patients had molar pregnancy-related late recurrence (3.3%).

Among non-obstetric causes, gastrointestinal conditions predominated (n=14, 23.3%), including acute gastroenteritis (n=7, 11.7%), peptic ulcer disease (n=4, 6.7%), and cholecystitis (n=3, 5.0%). Urinary tract infections with systemic features were

identified in 6 patients (10.0%), and acute appendicitis was confirmed in 5 patients (8.4%).

4.3. Clinical and Biological Features

Epigastric pain was the most common associated symptom (55%), followed by headache (38.3%), edema (35%), fever (28.3%), and jaundice (21.7%). Hypertension ($\geq 140/90$ mmHg) was documented in 36.7% of cases and was strongly associated with obstetric causes ($p < 0.001$).

Biological abnormalities included elevated transaminases (AST/ALT) in 38.3% of patients, thrombocytopenia in 21.7%, and proteinuria ($\geq 2+$ on dipstick) in 30.0%. Coagulopathy was present in 15% and was exclusively associated with AFLP and HELLP syndrome.

4.4. Management

Obstetric management was tailored according to gestational age and clinical severity. Emergency delivery (cesarean or labor induction) was performed in 23 patients (38.3%), predominantly for preeclampsia, HELLP syndrome, and AFLP. Conservative management with close monitoring was adopted in 12 patients (20%).

Medical treatment for non-obstetric causes included intravenous antibiotic therapy for urinary and gastrointestinal infections ($n=13$), proton pump inhibitors for peptic disease ($n=4$), and surgical intervention for appendicitis ($n=5$) and cholecystitis ($n=1$). All surgical cases were managed with laparoscopic technique under obstetric surveillance; no fetal complications were directly attributable to surgical intervention.

Antiemetic therapy (ondansetron, metoclopramide, promethazine) was used in all hospitalized patients as part of symptomatic management alongside rehydration.

4.5. Maternal and Fetal Outcomes

Maternal morbidity was significant: 11 patients (18.3%) required ICU admission, primarily those with AFLP and HELLP syndrome. There was 1 maternal death (1.7%) due to fulminant AFLP complicated by disseminated intravascular coagulation (DIC) and multiorgan failure.

Preterm delivery (before 37 weeks) occurred in 28 patients (46.7%). Mean birth weight was $2,340 \pm 610$ g. NICU admission was required in 22 neonates (36.7%). There were 3 perinatal deaths (5.0%), all in the context of HELLP syndrome with severe prematurity.

5. Discussion

Late-onset vomiting in pregnancy is a diagnostically challenging presentation that carries significant maternal and fetal risk. Our series highlights the heterogeneous nature of this condition and the imperative for systematic evaluation beyond the typical reflex of attributing all pregnancy-associated emesis to early physiological changes.

In line with prior literature, obstetric causes dominated our cohort (58.3%). Preeclampsia, the most common etiology (25%), classically manifests after 20 weeks of gestation and vomiting

particularly with epigastric or right upper quadrant pain is a cardinal symptom of severe-feature disease, reflecting hepatic subcapsular haemorrhage or Glisson capsule distension. Timely recognition and blood pressure control with magnesium sulphate prophylaxis are critical to preventing eclamptic seizures and stroke.

AFLP, though rare (estimated 1 in 7,000–20,000 pregnancies), was the second most frequent obstetric cause in our series (13.3%), reflecting our referral centre's profile. This condition, linked to fatal long-chain 3-hydroxyacyl-CoA dehydrogenase (LCHAD) deficiency, carries a historically high maternal mortality rate. The single maternal death in our cohort occurred in a patient with AFLP, underscoring that early recognition and emergent delivery remain the only definitive treatment. The Swansea criteria were used for diagnosis in all suspected cases.

HELLP syndrome (10%) presents with a clinical triad of haemolysis, elevated liver enzymes, and thrombocytopenia, and may manifest without proteinuria or severe hypertension, making it diagnostically deceptive. Its association with perinatal deaths in our series highlights the severity of this condition.

Non-obstetric causes accounted for 41.7% of cases a proportion consistent with published series ranging from 30 to 50%. Gastrointestinal conditions (23.3%), particularly infections and peptic pathology, were the leading non-obstetric causes. The physiological changes of pregnancy reduced lower oesophageal sphincter tone, delayed gastric emptying, and upward displacement of abdominal organs predispose to gastrointestinal symptoms and complicate the clinical picture.

Acute appendicitis, which occurred in 8.4% of our cases, merits special mention. The displacement of the appendix by the gravid uterus and the physiological leucocytosis of pregnancy conspire to delay diagnosis. Laparoscopic appendectomy remains safe in pregnancy with experienced surgical teams and obstetric co-management. A delay in diagnosis with resultant perforation carries substantial maternal and fetal morbidity.

Our management approach reflects current international guidelines: early delivery for uncontrolled obstetric complications, conservative and medical management for intercurrent infections, and prompt surgical intervention for abdominal emergencies. The 46.7% rate of preterm delivery in our cohort is consistent with the iatrogenic prematurity mandated by conditions such as preeclampsia and AFLP, and underscores the importance of neonatal facilities capable of managing extreme prematurity.

Limitations of this study include its retrospective design, the relatively small sample size, and the single-centre recruitment which may not represent the full epidemiological spectrum. A prospective multicentre study would allow more robust estimates of incidence and risk factors.

6. Conclusion

Late-onset pregnancy vomiting is not a trivial symptom. It encompasses a spectrum of conditions ranging from benign intercurrent illness to life-threatening obstetric emergencies.

Our series demonstrates that obstetric causes, particularly preeclampsia, AFLP, and HELLP syndrome, represent the majority of cases and carry the highest maternal and fetal morbidity and mortality.

A structured diagnostic algorithm incorporating clinical assessment, targeted biological workup, and imaging is essential for any patient presenting with vomiting after 20 weeks of gestation. Multidisciplinary collaboration between obstetricians, hepatologists, gastroenterologists, and surgeons remains the cornerstone of optimal management. Future prospective multicentre studies are needed to establish evidence-based screening protocols and improve outcomes in this vulnerable population.

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