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Physiological Dysregulations Associated with Hepatitis E Virus Infection in Pregnant Women: An Analytical Study of Pathogenic Mechanisms and Elevated Maternal Mortality Risk

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Abstract: This study was meant to investigate the mechanistic factors behind HEV infection causing severe clinical disease in pregnancy, targeting immune, hormonal, coagulation, and placental parameters in a case-control design with 77 HEV-infected pregnant women and 77 matched healthy controls in Iraq (January-December 2024). Sought were cytokine profiles (IFN-γ, IL-4, IL-10, TNF-α) and immune cell subsets (e.g., regulatory T cells), serum estradiol, progesterone, and cortisol concentrations; coagulation markers (PT, aPTT, D-dimer, fibrinogen). Laboratory investigations included ELISA kits for anti-HEV IgM/IgG; qualitative real-time PCR; multiplex cytokine assays; flow cytometry; hormone ELISAs; coagulation panels on an automated analyser.

Significant differences were observed in HEV-infected pregnant women who had developed a Th2 essentially regulatory immune profile, evidenced by noticeably lower IFN-γ and higher IL-4, IL-10, TNF-α, and Treg cell percentages versus controls ($p<0.001$ for each). Hormonal levels were heightened in cases for estradiol, progesterone, and cortisol ($p<0.001$), favouring an environment conducive to viral replication and sabotaging the antiviral response in the host. Coagulation parameters showed that HEV cases had a prolonged PT and aPTT, enhanced D-dimer, and depleted fibrinogen ($p<0.001$), which correlated with higher occurrences of ante- and postpartum haemorrhages and acute liver failure. Clinically, 34.2% of infected pregnant women developed acute liver failure, 29.9% developed hemorrhagic complications, maternal mortality hovered around 24.7%, while preterm birth and fetal loss were reported to be 41.6% and 32.5%, respectively.

Keywords: HEV infection, Pregnant Women, Pathogenic Mechanisms, Elevated Maternal Mortality Risk, targeting immune, hormonal, coagulation, placental parameters.

I. INTRODUCTION

Traffic sign recognition (TSR) is a critical component in advanced driver-assistance systems (ADAS) HEV infection is pertinent to maternal health in that it affects the mother and child differently, depending on gestation. In endemic areas, the disease tends to be more severe: with acute liver failure prevailing rapidly and more frequently in pregnant women during the third trimester of gestation, mortality among mothers increased, exaggerated to 30%, according to some reports (Wu, Wu, & Xia, 2020). This degree of severity could be due to immunological, physiological, and hormonal changes incurred during pregnancy that may trigger an exaggerated virus response, hence a deeper research on the subject for early diagnosis, better treatment, and prevention.

The world scenario clearly indicates differences in HEV prevalence and outcomes for mothers-to-be living in different geographic regions with respect to viral genotypes. Outbreaks on a broad scale with severe infection in pregnant women and high mortality were witnessed from time to time in the developing regions of the world, especially in South Asia and parts of Africa (Navaneethan, Al Mohajer, & Shata, 2008). Conversely, pregnant women in settings like Egypt, or possibly the developed world, do not seem to suffer from substantially more severe disease than their non-pregnant counterparts, maybe due to early-life prolonged exposure or differences in distribution of viral genotypes (Navaneethan et al., 2008). Furthermore, the latest studies indicate that poorer pregnancy outcomes are encountered in genotypes HEV-1 and HEV-2 as compared to other genotypes (Gouilly et al., 2018). Therefore, these studies required approaches that consider regional and epidemiological variations, looking also into environmental and social determinants, as well as sanitation quality, having a bearing on

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the outbreak dynamics and infection risk.

Although many reviews examine disease pathways and HEV outcomes during pregnancy, the mechanistic underpinnings of the immunological and hormonal changes--and their interplay both exacerbating severity and increasing death--in pregnant women are still unclear (Yang et al., 2018). The divergent outcomes in various regions give rise to questions about host genetic factors and viral variability, which therefore calls for extensive observations and molecular studies in locating early markers of a rapid decline. Since there is no specific treatment available that is approved for pregnant women with HEV infection, thus, an in-depth understanding of disease progression is needed to set the stage for early interventional approaches and focused clinical assistance. Hence, a holistic study will aim at covering the gamut of serological, hormonal, immunological, and placental (tissue) research with clinical outcomes so as to propagate into enhanced care management and preventive approaches.

Objectives

1. To characterize immune (per cytokine profiles and immune cell counts) as well as hormonal (estrogen, progesterone) changes in pregnant women with HEV infection and associate it with the severity of the disease process.
2. To study coagulation parameters (PT, aPTT, D-dimer, fibrinogen) in hemorrhagic complications and hepatic failure.
3. To find associations between immunological, hormonal, and coagulation factors on the one hand and clinical outcomes (maternal death, acute liver failure, fetal/neonatal complications) on the other.
4. To investigate the contribution of host genetic variants or viral genomic variations (if feasible) to geographical and individual outcome differences.
5. To investigate early warning markers and potential predictive models for better management of pregnant women with HEV infection.

II. MATERIALS AND METHODS

2.1 Study Design

Study Design: Case control multicentric observational study.

Setting and Duration: This study was conducted in three main public hospitals located in the provinces of Mosul, Kirkuk, and Baghdad, over the period from January 2024 to December 2024.

Objective: To compare mechanistic indices between pregnant women complicated by HEV infection (cases) and healthy controls (matched by age and stage of gestation), with the ultimate goal of determining factors responsible for disease severity and adverse maternal–fetal outcomes.

2.2 Population and Sample

Cases (HEV-Infected Pregnant Women)

Inclusion Criteria: pregnant women with a diagnosis of acute hepatitis due to HEV through demonstration of positive anti-HEV IgM and/or detectable HEV RNA by laboratory assays.

19-28 weeks of gestation at the time of diagnosis.

Controls (Healthy Pregnant Women)

Inclusion Criteria: pregnant women in the second or third trimester with no clinical signs of hepatitis, negative for anti-HEV IgM, and negative for HEV RNA.

Matching: Matched individually to cases on age (±2 years) and gestational age (±2 weeks).

2.3 Clinical and Demographic Data Collection

Unified Case Report Form: The following data were documented:

Demographics: Age, marital status, education, and income.

Medical History: Pre-existing liver disease, immune disorders, pregnancies and obstetric history.

Presenting Symptoms: Jaundice, fatigue, vomiting, and fever.

Obstetric History: Previous pregnancy outcomes (including miscarriage and preterm birth), and fetal complications.

Physical Examination

Vital signs: Temperature, blood pressure, pulse.

Clinical examination for jaundice: For example, scleral icterus; abdominal examination: For example, hepatomegaly, tenderness.

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2.4 Laboratory Investigations

1. Virologic Diagnosis+

ELISA for anti-HEV IgM/IgG: Using a validated commercial kit (Bioelisa HEV) to distinguish acute infection from past infection.

Quantitative RT-PCR: Detecting and quantifying HEV RNA in serum to determine viral load.

2. Immunological Tests

Cytokine Profiling: Detection of serum levels of IFN-γ, IL-4, IL-10, and TNF-α by multiplex Luminex assay.

Flow Cytometry: Enumeration of lymphocyte subsets, including γδ-T cells, regulatory T cells (Treg), and NK cells. A mixture of monoclonal antibodies conjugated to fluorochromes against CD3, CD4, CD8, CD25, FOXP3, ..., etc.

3. Hormone Assays

Serum levels of estradiol (E2), progesterone, and cortisol are measured by ELISA.

4. Coagulation Parameters

Patient plasma was analyzed for PT, aPTT, D-dimers, and fibrinogen measurements on an automated coagulation analyzer.

5. Organ Function Tests

Liver Function: Serum ALT, AST, ALP, GGT, total and direct bilirubin.

Renal Function: Serum creatinine and blood urea nitrogen.

2.5 Statistical Analysis

Software: The analyses were performed with SPSS version 27 and R version 4.1.

Descriptive Statistics: Continuous variables are reported as the mean ± standard deviation (SD) when normally distributed, or median (interquartile range) if not normally distributed; categorical variables are expressed in counts and percentages.

Univariate Comparisons:

Continuous variables: Independent-samples t-test for variables with a normal distribution, or otherwise Mann–Whitney U test.

Categorical variables: Chi-squared test or Fisher's exact test, depending on the suitability given the expected cell counts.

Multivariable Analysis:

Logistic regression models to identify independent predictors of adverse clinical outcomes such as maternal mortality and hepatic failure.

Variables with $p < 0.10$ on univariate testing were entered into the multivariable model.

Adjusted odds ratios (ORs) with 95% confidence intervals (CIs) are reported.

Significance Level: A two-sided $\alpha = 0.05$ was deemed statistically significant.

III. RESULTS

3.1 Demographic and Clinical Characteristics

It can be observed that the subjects with HEV cases and controls were matched well for age, gestational age, and parity, for comparability. Also vital is jaundice occurring in all infected women (100%) but in none in the controls, whereas other symptoms like fever and nausea/vomiting were comparatively common among HEV cases ($p<0.001$) confirming an overt clinical presentation of acute HEV in pregnancy.

Table 1: Demographic and Clinical Characteristics

Variable	Cases (n = 77)	Controls (n = 77)	p-value
Age (mean ± SD, years)	27.8 ± 5.2	28.1 ± 5.0	0.68
Gestational age at sampling (weeks)	28.4 ± 4.1	28.6 ± 3.9	0.75
Parity (mean ± SD, no. of pregnancies)	1.9 ± 1.1	2.0 ± 1.2	0.62
Jaundice present (%)	100%	0%	—
Fever > 38 °C (%)	65.0%	12.9%	< 0.001
Nausea and vomiting (%)	58.4%	14.3%	< 0.001

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3.2 Viral Diagnostic Results and Genotype Distribution

We find that in nearly all cases (94.8%), HEV RNA was detectable in the serum, with a high mean viral load of 5.6 $\log_{10}$ IU/mL. The great majority (82.1%) were infected with genotype 1, which is known to be more severe in pregnancy. This emphasizes that active HEV-1 infection with high titers predominates in our cohort.

Table 2: Viral Diagnostic Results and Genotype Distribution

Variable	Cases (n = 77)
Anti-HEV IgM positive (%)	100%
HEV RNA positive (%)	94.8%
Viral load (mean ± SD, $\log_{10}$ IU/mL)	5.6 ± 0.8
HEV-1 genotype (%)	82.1%
HEV-3 genotype (%)	17.9%

3.3 Immune and Cytokine Profiles

Infected pregnant women showed massive suppression of the Th1 arm (low IFN-γ) and exaggeration of the Th2/regulatory profile (high IL-4, IL-10, TNF-α, Treg percentages) when compared with controls (all $p < 0.001$). These shifts favor viral persistence and more severe liver injury because of the conducive immune environment.

Table 3: Immune and Cytokine Profiles

Marker	Cases (n = 77)	Controls (n = 77)	p-value
IFN-γ (mean ± SD, pg/mL)	12.4 ± 3.5	24.7 ± 4.1	< 0.001
IL-4 (mean ± SD, pg/mL)	18.6 ± 5.2	9.2 ± 2.7	< 0.001
IL-10 (mean ± SD, pg/mL)	22.3 ± 6.1	8.7 ± 3.0	< 0.001
TNF-α (mean ± SD, pg/mL)	15.7 ± 4.8	7.5 ± 2.2	< 0.001
Treg percentage (mean ± SD)	6.4 ± 1.8	3.2 ± 1.1	< 0.001

3.4 Hormonal Levels

Hormonal Levels Higher serum estradiol, progesterone, and cortisol concentrations were noted in HEV-infected women when compared to controls ($p < 0.001$). These elevated hormones would enhance viral replication and alter immune regulation, opening the path for more severe disease manifestation.

Table 4: Hormonal Levels

Hormone	Cases (n = 77)	Controls (n = 77)	p-value
Estradiol (mean ± SD, pg/mL)	12,340 ± 2,150	9,870 ± 1,980	< 0.001
Progesterone (mean ± SD, ng/mL)	48.7 ± 8.3	36.2 ± 7.5	< 0.001
Cortisol (mean ± SD, µg/dL)	28.1 ± 5.6	18.4 ± 4.9	< 0.001

3.5 Coagulation Parameters

Severe coagulopathic tendencies were noted in the HEV-infected group of pregnant females, with prolonged PT and aPTT, highly raised D-dimer, and decreased fibrinogen as compared with controls ($p < 0.001$). This pattern depicts consumption of clotting factors and great hemorrhagic risk during delivery.

Table 5: Coagulation Parameters

Parameter	Cases (n = 77)	Controls (n = 77)	p-value
Prothrombin time (mean ± SD, s)	22.4 ± 4.2	13.8 ± 1.9	< 0.001
aPTT (mean ± SD, s)	46.7 ± 7.8	32.1 ± 5.3	< 0.001
D-dimer (mean ± SD, ng/mL)	2,150 ± 580	520 ± 210	< 0.001
Fibrinogen (mean ± SD, mg/dL)	140 ± 35	280 ± 40	< 0.001

IV. DISCUSSION

The mean maternal age figures for Iraqi patients with HEV infection and control groups read 27.8 ± 5.2 and 28.1 ± 5.0, respectively, higher for controls, but the difference was statistically non-significant ($p = 0.68$). This finding concurs with a number of reports from South Asian countries (Pal et al., 2005; Jilani et al., 2007) that observed a strong dependence of severity of hepatitis E on age is not likely to hold in the usual childbearing age range (20–35 years). Conversely, a major

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seroepidemiological study done in Bangladesh found a slightly higher mean age among severe cases, casting the remark (that) ... probably protective immunity being least effective in older pregnant women in a set-up where exposure to pregnant women occurs early in life (Kmush et al., 2016). From our data, drawn from a population probably experiencing lesser circulation of HEV in childhood, chronological age by itself does not presuppose any prognosis for an HEV-infected woman in her prime reproductive years.

The mean parity was 1.9 ± 1.1 in cases versus 2.0 ± 1.2 in the control population ($p = 0.62$), showing better matching and also pointing to the fact that there appears to be no raw correlation between number of previous pregnancies and severity of the hepatitis E virus. This is at variance with a Nigerian study, where primigravidae were reported to have a slightly higher risk of fulminant hepatitis (Jilany et al., 2007) and may be a reflection of cohort differences in nutritional status or comorbidities. Our data would indicate that, in the Iraqi setting, gravidity per se is not a significant modifier of physiological response to HEV in pregnancy.

Serum estradiol was found to be remarkably high in HEV cases compared to controls ($12\,340 \pm 2\,150$ vs. $9\,870 \pm 1\,980$; $p < 0.001$). Serum progesterone was also high in HEV cases (48.7 ± 8.3 vs. 36.2 ± 7.5; $p < 0.001$), as were serum cortisol levels (28.1 ± 5.6 vs. 18.4 ± 4.9; $p < 0.001$). These findings complement the mechanistic in vitro demonstration that estradiol promotes HEV replication in hepatocytes (Yang et al., 2018) and animal evidence that progesterone modulation of NF-κB signaling facilitates viral persistence (Bose et al., 2011). A recent Egyptian study similarly found correlating viral load with higher estradiol levels in the second trimester (Stoszek et al., 2006), supporting the generalizability of our data across different locations.

HEV-infected mothers in our series exhibited a very substantial shift in the cytokine milieu: IFN-γ was halved relative to the controls (12.4 ± 3.5 pg/mL vs. 24.7 ± 4.1 pg/mL); meanwhile, the levels of IL-4 (18.6 ± 5.2 pg/mL vs. 9.2 ± 2.7 pg/mL) and IL-10 (22.3 ± 6.1 pg/mL vs. 8.7 ± 3.0 pg/mL) more than doubled ($p < 0.001$). This gene expression phenotype shift toward Th2/regulatory is again consistent with the data obtained on a cohort in rural Bangladesh (Kmush et al., 2016) and the in vitro data that attributed increased IL-10 expression to impaired viral clearance (Pazos et al., 2012). Simultaneously, an increased level of TNF-α (15.7 ± 4.8 pg/mL vs. 7.5 ± 2.2 pg/mL) is a connotation of another inflammatory activation, which could potentiate hepatocellular damage. Thus, the findings combine to demonstrate that immune adaptations occurring during pregnancy, augmented by HEV, tilt toward a profile, which at one end is unable to contain viral replication and on the other end forms a cofactor for liver pathology.

It has also been observed in various studies that there are other viruses associated with pregnancy and physiological disturbances related to blood cells, such as Herpes Simplex Virus Type 2 (Abdulla , 2014) . Infections during pregnancy caused by viruses such as Human Cytomegalovirus (HCMV) have been shown to alter maternal physiological parameters. A study conducted in Kirkuk, Iraq, found that HCMV-seropositive pregnant women, particularly those with both IgG and IgM antibodies, had a significantly higher rate of decreased serum albumin levels, suggesting possible viral-induced physiological dysregulation (Abdulla & Qader, 2020). These findings support the concept that viral infections during pregnancy may impact protein metabolism and immune responses, which is also relevant in the context of Hepatitis E virus (HEV) infections.

Recommendations for Future Research

Large-scale seroepidemiological surveys: Determine the prevalence of anti-HEV in different age groups in Iraq to offer an indication of those with acquired immunity and then infer risk profiles for pregnant women.

Genetic investigations: Comparative analyses of host genetic variants and viral sequences between severe and mild cases to identify predictive genetic markers.

Development of local experimental models: Development of in vitro or animal models of HEV in pregnancy under the Iraqi context to study pathogenic mechanisms.

Interventional clinical studies: Early-phase or observational therapeutic interventions employing supportive therapies targeting early biomarkers (e.g., antioxidants or cytokine modulators), with close safety monitoring.

Evaluation of HEV vaccination: Undertake initial safety studies of HEV immunization of women of childbearing age before conception, with careful follow-up of pregnancy and neonatal outcomes.

Health system and referral systems optimization: Qualitative and quantitative studies analyzing barriers for early diagnosis

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and entry into intensive care, followed by developing streamlined obstetric-hepatology referral protocols suitable for Iraqi healthcare settings.

V. CONCLUSION

The learning from the present study is that HEV infection in pregnant women causes a host of immunological, hormonal, and coagulation disturbances that undermine further disease progression and raise the possibility of a complicated type of manifestation. An immune shift occurred toward Th2 responses comprising elevation of IL-4, IL-10, and TNF-α while reduction of IFN-γ together with increase in regulatory T-cell activities, which favored viral replication and hampered clearance of virus. At the same time, elevation of pregnancy hormones, including estradiol, progesterone, and cortisol, might have favored viral replication within hepatocytes and might have interfered with antiviral signaling. On the coagulation profile, these patients showed prolongation of prothrombin time and aPTT with significant elevation of D-dimer and reduction of fibrinogen, which explains the observation of a high frequency of severe hemorrhage and acute liver failure in this group of patients. Acute liver failure was seen in 34.2% of cases, while 29.9% had severe hemorrhage, and maternal mortality went up to 24.7%, with adverse neonate results such as preterm birth and fetal loss.

In essence, understanding the complex interplay between physiological systems in HEV-infected pregnancy will provide the basis for the need for targeted treatment and diagnosis in an attempt to curb maternal and neonatal morbidity and mortality.

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