Journal of Communicable Diseases
Volume 58, Issue 1 - 2026, Pg. No. 47-54
Peer Reviewed & Open Access Journal

Research Article

Efficacy and Safety of Tenofovir in Preventing Vertical Transmission of Hepatitis B Virus

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DOI:https://doi.org/ 10.24321/0019.5138.202606

I N F O

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How to cite this article:
Abdikerimova M, Suranbaeva G, Kanatbekova A, Abdikerimov M, Ysmaiylova B, Djumagulova A, Mambetova M, Zholdoshev S, Aitkuluev N, Tagaev T. Efficacy and Safety of Tenofovir in Preventing Vertical Transmission of Hepatitis B Virus. J Commun Dis. 2026;58(1):47-54.

Date of Submission: 2025-12-05
Date of Acceptance: 2026-02-25

A B S T R A C T

Introduction: Chronic hepatitis B remains a major global health challenge, with mother-to-child transmission substantially contributing to the hepatitis B virus (HBV) burden in endemic settings such as the Kyrgyz Republic. This study evaluated the efficacy and safety of tenofovir in pregnant women with high viral loads for preventing vertical HBV transmission.

Methods: Twenty-three women received tenofovir 300 mg/day from 28–30 weeks' gestation through 4 weeks postpartum, while 35 women served as controls. HBV DNA levels were quantified every 4 weeks during pregnancy and postpartum. Newborns received hepatitis B vaccination within 12 h, and HB immune globulin was administered when available.

Results: Tenofovir rapidly suppressed viremia, with lower HBV DNA levels at 4 weeks than those in controls (3.20×10^6 vs. 8.43×10^6 copies/mL; $p<0.001$). By delivery, 95.6% of patients achieved HBV DNA levels <3,000 copies/mL, and 26.1% had undetectable HBV DNA, with 13.1% showing HBeAg/anti-HBe. Biochemical improvement was greater with tenofovir, with alanine aminotransferase and bilirubin normalization in 82.6% of patients versus 14.3% in controls ($p<0.05$). No vertical transmission occurred in the tenofovir group, compared with 8.5% in the control group.

Conclusion: The maternal and neonatal safety profiles were favorable, with no increase in complications or adverse events and no post-cessation exacerbations.

Keywords: Hepatitis B Virus, Vertical Transmission, Tenofovir, Viral Load, Pregnancy, Immunoprophylaxis, Antiviral Therapy

Introduction

Chronic hepatitis B (CHB) is a major global health challenge. The World Health Organization (WHO) estimates that 350 million individuals worldwide are infected with the hepatitis B virus (HBV), with 70% residing in Asia. Annually, 650,000 people succumb to HBV infection.[1,2] The Kyrgyz Republic is among the regions where HBV is an endemic disease, with the virus transmitted vertically from mother to child.[3,4] Pregnant women with HBV face a heightened risk of passing the infection to their newborns.[5,6]

Before implementing combined passive-active immunoprophylaxis (administering HBV immunoglobulin (HBIG) and HBV vaccination within the first hours of life), chronic hepatitis B rates in children born to mothers positive for Hepatitis B Surface Antigen (HBsAg) and Hepatitis B e-Antigen (HBeAg) were 70-90%.[7,8] This immunoprophylaxis reduces the rate to 5-10%, if the mother has a high viral load, the risk of newborn infection remains 8-30%.[9-12]

Studies have shown that maternal HBV viral load primarily influences immunoprophylaxis failure, with a higher risk of mother-to-child transmission in women with elevated HBV DNA levels, despite neonatal vaccination and hepatitis B immunoglobulin. Cohort studies have shown that infants born to mothers with HBV DNA levels ≥200,000 IU/mL are at risk of developing chronic HBV infection despite standard immunoprophylaxis, necessitating maternal antiviral treatment to prevent vertical transmission.[6,8,9]

Tenofovir disoproxil fumarate, a nucleotide analog with a high barrier to resistance, has become the preferred antiviral during pregnancy because of its efficacy and safety for both the mother and the child. Studies have shown that tenofovir treatment in late pregnancy reduces maternal HBV DNA levels and lowers neonatal HBsAg and HBV DNA positivity without increasing adverse outcomes.[7,10,13] These findings are supported by randomised clinical trials and cost-effective analyses, recommending tenofovir prophylaxis for pregnant women with high viral loads.[14,15]

According to the EASL guidelines, women who test positive for HBsAg and have viral loads exceeding 10^6-10^7 IU/ml during the third trimester should be prescribed antivirals, such as lamivudine, telbivudine, or tenofovir, to prevent intrauterine and perinatal HBV transmission.[16-18]

Consequently, antiviral treatment during pregnancy has become crucial in the global effort to eradicate HBV infection by minimizing vertical virus transmission.

This study assessed the clinical effectiveness and safety of tenofovir in pregnant women with high viral loads to prevent vertical HBV transmission.

Methods

This prospective study included 58 HBsAg-positive pregnant women with a high HBV DNA viral load (exceeding 10^6 copies/ml). Among them, 23 women received tenofovir (Hetero Labs, Ltd, Hyderabad, India) at 300 mg daily (tenofovir group), while 35 women who declined treatment formed the control group. The participants' ages ranged from 18 to 39 years. The mean age in the study group was 23.0 ± 4.2 years, compared to 24.9 ± 5.6 years in the control group. Those with a high viral load received tenofovir at 300 mg daily from the 28th-30th week of pregnancy until the 4th week postpartum.

Before antiviral treatment, all pregnant women with hepatitis C virus underwent clinical and laboratory evaluations, including clinical examination, blood and urine tests, assessment of alanine aminotransferase (ALT), aspartate aminotransferase (AST), bilirubin, thymol test, total protein and fractions, coagulogram, and HBV markers (HBsAg, HBeAg, anti-HBe, Immunoglobulin M and Immunoglobulin G) via enzyme-linked immunosorbent assay, polymerase chain reaction for HBV DNA, followed by viral load measurement using quantitative fluorescent polymerase chain reaction. The women were screened for hepatitis D virus infection, which was ruled out in all the participants. No significant differences were observed between the groups ($p>0.05$) in terms of age, gestational age, treatment duration, initial HBV DNA levels, or alanine aminotransferase activity.

The primary criterion for prescribing tenofovir to pregnant women with HBV was an initial viral load exceeding 10^6–10^7 copies/ml, as shown in Table 1. Participants were excluded if they had co-infections with hepatitis C, D, A, E, or human immunodeficiency viruses, other liver conditions (medicinal hepatitis, non-alcoholic steatohepatitis, autoimmune hepatitis, intrahepatic cholestasis of pregnancy), or fetal dysplasia.

To evaluate the impact of tenofovir on viral replication, HBV DNA levels in the blood serum were measured every four weeks during pregnancy and until the 28th week postpartum.

Patients with pregnancy complications or fetal malformations, as shown by ultrasound, were not administered tenofovir. All newborns received immunoglobulin and their first hepatitis B vaccine dose within 12 h of birth.

Antiviral therapy effectiveness was assessed by vertical HBV transmission rate (determined by HBsAg and HBV DNA in infant blood serum at 28 weeks), reduction in HBV DNA levels at delivery, clearance and seroconversion of HBsAg and/or HBeAg, normalization of ALT activity, and adverse events in mothers and newborns.

Adverse outcomes in mothers with HBV infection were monitored during pregnancy and childbirth. Newborns had

ISSN: 0019-5138
DOI: https://doi.org/10.24321/0019.5138.202606

anthropometric data recorded at birth and follow-up (height and weight), Apgar score, birth defects, breastfeeding characteristics, and cognitive development (IQ score).

Pregnant women with HBV were treated following the "Clinical Protocol of the Ministry of Health of the Kyrgyz Republic for diagnosing and treating adult patients with hepatitis B" and the EASL (2020-2023) guidelines.

Statistical analysis was performed using Statistica 6.0 (StatSoft), and descriptive statistics, including the mean, standard deviation, standard error of the mean, and frequency percentages, were used to summarize the data. The statistical reliability criterion (p) was considered the standard value in medicine. Written informed consent was obtained from the parents of all patients. The study was approved by the Bioethics Committee of the Kyrgyz State Medical Academy named after I.K. Akhunbaev (Protocol No. 17, April 13, 2019).

Results

In a study of 58 pregnant women with HBV and high viral loads, 21.8% (5 out of 23) of the study group and 20.0% (7 out of 35) in the control group were diagnosed with HBV before their current pregnancy, with a history of acute hepatitis. Both groups had 41.6% of patients with HBsAg history of 5-10 years, while 58.4% exceeded 10 years. HBsAg was first identified during clinical examination in most women, indicating primary chronic hepatitis B, with 78.2% and 80.0% in the study and control groups, respectively. HBeAg was present in 13.1% (3 out of 23) of pregnant women receiving tenofovir and 14.3% (5 out of 35) of the controls. In the study group, 95.6% of the participants and all controls had not received HBV vaccination.

Among those receiving tenofovir, 39.2% (9 out of 23) were in their first pregnancy, 69.5% (16 out of 23) were first-time mothers, and 30.5% (7 out of 23) were in their second pregnancy/motherhood. In the control group, 37.2% (13 out of 35) were first-time pregnant, 65.7% (23 out of 35) were first-time mothers, and 34.3 % (12 out of 35) were in their second pregnancy. The mean age was 23.0 ± 4.2 years in the study group and 24.9 ± 5.6 years in the control group.

The primary requirement for inclusion was an initial HBV DNA viral load exceeding 10^6 copies/ml. Baseline HBV DNA levels in blood serum were similar: $3.78 \times 10^6 – 1.73 \times 10^7$ copies/ml for the study group and $3.67 \times 10^6 – 1.71 \times 10^7$ copies/ml for the control group (Table 1). Four weeks after starting tenofovir treatment, HBV DNA levels were reduced to 3.20×10^6 copies/ml, while the control group maintained a high viral load of 8.43×10^6 copies/ml ($p < 0.001$). By delivery, 95.6% of women receiving tenofovir had reduced their viral load to less than 3,000 copies/ml (average 2.83×10^3 copies/ml), with 21.8% of those starting therapy at week 28 showing undetectable HBV DNA levels. The control group consistently maintained high viral loads ($p < 0.001$). Additionally, 13.1% of patients experienced HBeAg/anti-HBe seroconversion. No virological breakthrough occurred during treatment or at 28 weeks postpartum.

Blood test results for pregnant women with HBV showed no notable differences between the study and control groups at delivery (Table 2).

In the tenofovir group, 86.9% of women and 82.8% of controls showed minimal ALT activity and slight bilirubin elevation before therapy (Table 3). By delivery, ALT and bilirubin levels were normalized in 82.6% of the study group, compared with 14.3% in the control group ($p < 0.05$). Coagulogram and blood glucose levels showed no significant changes between groups before childbirth.

Table 3 outlines the pregnancy and childbirth characteristics of women treated with tenofovir and the controls. As shown in Table 4, there were no significant differences in pregnancy or childbirth outcomes between the groups.

No congenital anomalies or severe side effects were observed in children born to women administered tenofovir, suggesting the absence of mutagenic or carcinogenic effects. ALT levels in the newborns' blood serum were similar in both groups.

Newborns from tenofovir-treated mothers and controls showed no significant differences (Table 5): body weight averaged 3321 ± 316 g and 3313 ± 372 g, respectively, and height was 51.9 ± 1.8 cm and 51.3 ± 1.9 cm, respectively. A reduced Apgar score occurred in 17.4% (4/23) of infants born to mothers receiving tenofovir, compared with 22.8% (8/35) in infants born to untreated mothers ($p = 0.279$). During follow-up, the children developed in line with age norms.

All infants born to mothers with HBV infection received hepatitis B vaccination at the hospital. Additionally, seven of 23 children whose mothers received tenofovir and eight of 35 children whose mothers did not receive the medication were administered hepatitis B immunoglobulin intramuscularly at 100 IU within 12 h of birth. Immunoglobulin administration was not feasible in all cases because of unavailability.

We assessed the impact of tenofovir on HBV vertical transmission. Infection in children was identified using HBsAg and HBV DNA at birth and 6 months of age. Our study found no cases (0/23) of vertical transmission among newborns whose mothers received tenofovir during pregnancy. In the untreated group, HBV infection occurred in 8.5% of the infants (3/35). By week 28, these infants had not undergone HBsAg/anti-HBs and HBeAg/anti-HBe seroconversion, and HBV DNA levels remained high (>6 log copies/ml). The remaining children (91.5%) in the control group responded well to immunoprophylaxis treatment.

ISSN: 0019-5138
DOI: https://doi.org/10.24321/0019.5138.202606

Administering tenofovir early (at 28-30 weeks of pregnancy) to women with high HBV DNA viral loads reduces the risk of vertical transmission, which immunoprophylaxis alone cannot prevent.

Tenofovir was well tolerated, with no serious side effects, premature birth, or postpartum bleeding.

Antiviral therapy was stopped at week 4 postpartum per guidelines, allowing breastfeeding. Blood biochemical parameters were checked 2 weeks after delivery and then monthly. HBV DNA levels were measured 3 months post-treatment. No clinical, biochemical, or virological exacerbations occurred after treatment cessation.

During follow-up, ALT and AST levels remained stable, and the viral load increased by no more than 1-2 log in 78.3% of patients; in 21.7%, it remained below 300 copies/ml for 2-3 months. In the controls, HBV DNA levels remained consistently high before and after childbirth.

Table 1.Viral load indicators in pregnant women with HBV on the background of antiviral therapy with nucleoside analogues

Study period	Tenofovir group (n=23)	Control group (n=35)	P
	HBV DNA (copies/ml)	HBV DNA (copies/ml)	
Before treatment	8.65×10^6 ($3.78 \times 10^6 – 1.73 \times 10^7$)	8.55×10^6 ($3.67 \times 10^6 – 1.71 \times 10^7$)	<0.001
38-40 weeks	$0-2.75 \times 10^3$	$2.43 \times 10^6 – 1.53 \times 10^7$	<0.001

Table 2.Characteristics of the general blood test in pregnant women with HBV who received tenofovir and women from the control group

Indicators M±m		Tenofovir group (n=23)		Control group (n=35)		P
		95% CI	M±m	95% CI		
RBCs, $\times 10^{12}$/l	Before therapy	3.9±0.2	3.5–4.3	3.8±0.14	3.52–4.08	>0.05
	38-39 weeks	3.8±0.16	3.48–4.12	3.7±0.1	3.5–3.9	>0.05
Hemoglobin, g/l	Before therapy	109.6±3.8	102.0–117.2	110.0±3.6	102.8–117.2	>0.05
	38-39 weeks	109.9±3.3	103.3–116.5	111.9±3.4	105.1–118.7	>0.05
Platelets, $\times 10^9$/l	Before therapy	223.0±11.1	200.8–245.2	228.4±20.7	187.0–269.8	>0.05
	38-39 weeks	228.1±14.0	200.1–256.1	225.8±19.8	186.2–265.4	>0.05
WBCs, $\times 10^9$/l	Before therapy	7.9±0.8	6.3–9.5	8.5±0.6	7.3–9.7	>0.05
	38-39 weeks	8.43±0.6	7.23–9.63	9.4±0.6	8.2–10.6	>0.05
Eosinophils, %	Before therapy	2.0±0.5	1–3	1.9±0.4	1.1–2.7	>0.05
	38-39 weeks	2.1±0.4	1.3–2.9	1.6±0.5	0.6–2.6	>0.05
Neutrophils, %	Before therapy	62.2±3.3	55.6–68.8	61.1 ±4.1	52.9–69.3	>0.05
	38-39 weeks	61.2±4.2	52.8–69.6	62.4±3.9	54.6–70.2	>0.05
Lymphocytes, %	Before therapy	32.3±3.8	24.7–39.9	33.9±4.2	25.5–42.3	>0.05
	38-39 weeks	31.9±3.2	25.5–38.3	30.2±3.7	22.8–37.6	>0.05
Monocyte, %	Before therapy	5.7±0.8	4.1–7.3	5.5±0.7	4.1–6.9	>0.05
	38-39 weeks	5.5±0.8	3.9–7.1	5.2±0.8	3.6–6.8	>0.05
ESR, mm/h	Before therapy	23.3±4.2	14.9–31.7	22.4±2.8	16.8–28.0	>0.05
	38-39 weeks	27.7±3.6	20.5–34.9	25.6±2.4	20.8–30.4	>0.05

Values are expressed as the M ± m = Mean ± Standard deviation. RBCs – Red Blood Cells, WBCs – White Blood Cells, ESR – Erythrocyte Sedimentation Rate. 95% CI – 95% confidence interval. *P<0.05.

ISSN: 0019-5138
DOI: https://doi.org/10.24321/0019.5138.202606

Table 3.Characteristics of the biochemical blood test index and coagulogram in pregnant women with HBV who received tenofovir and the control group

Indicators M±m		Tenofovir group (n=18)		Control group (n=17)		P
		95% CI	M±m	95% CI		
ALT, IU/L	Before therapy	92.5±4.8	82.9–102.1	95.3±5.3	84.7–105.9	>0.05
	38-39 weeks	33.6±3.7	26.2–41.0	92.3±4.6	83.1–101.5	<0.05*
AST, IU/l	Before therapy	65.7±4.5	56.7–74.7	68.9±7.1	54.7–83.1	>0.05
	38-39 weeks	25.5±1.9	21.7–29.3	62.5±5.4	51.7–73.3	<0.05*
Total bilirubin, mmol/l	Before therapy	23.6±5.1	13.4–33.8	22.3±4.8	12.7–31.9	>0.05
	38-39 weeks	18.3±0.8	16.7–19.9	22.1±5.7	10.7–33.5	>0.05
Total protein, g/l	Before therapy	64.7±1.6	61.5–67.9	64.8±1.2	62.4–67.2	>0.05
	38-39 weeks	64.2±2.2	59.8–68.6	62.5±0.3	61.9–63.1	>0.05
Glucose, g/l	Before therapy	4.6±0.2	4.2–5.0	4.5±0.1	4.3–4.7	>0.05
	38-39 weeks	4.5±0.2	4.1–4.9	4.8±0.1	4.6–5.0	>0.05
Fibrinogen, g/l	Before therapy	3.5±0.3	2.9–4.1	3.2±0.4	2.4–4.0	>0.05
	38-39 weeks	4.8 ±0.2	4.4–5.2	4.3±0.6	3.1–5.5	>0.05
APTT, s	Before therapy	34.3±1.2	31.9–36.7	35.5±0.7	34.1–36.9	>0.05
	38-39 weeks	32.4±1.3	29.8–35.0	33.7±0.6	32.5–34.9	>0.05
PI	Before therapy	81.3±1.2	78.9–83.7	82.5±1.4	79.7–85.3	>0.05
	38-39 weeks	76.2±1.8	72.6–79.8	77.7±1.7	74.3–81.1	>0.05

Values are expressed as the M ± m = Mean ± Standard deviation. ALT – Alanine aminotransferase, AST – Aspartate aminotransferase, APTT – Activated Partial Thromboplastin Time, PI – Prothrombin Index. 95% CI – 95% confidence interval. *P <0.05.

Table 4.Features of the course of pregnancy and childbirth in women with HBV treated with tenofovir and in the control group

The course of pregnancy and childbirth	Tenofovir group (n=23)	Control group (n=35)	P
Anemia	5 (21.8%)	9 (25.7%)	>0.05
Threat of interruption	4 (17.4%)	7 (20.0%)	>0.05
Edema of pregnant women	3 (13.1%)	5 (14.3%)	>0.05
Fetal growth retardation	2 (8.6%)	3 (8.5%)	>0.05
Placental insufficiency	4 (17.4%)	8 (22.8%)	>0.05
Intrauterine fetal hypoxia	2 (8.6%)	4 (11.4%)	>0.05
Polyhydramnios	3 (13.1%)	6 (17.2%)	>0.05
Hypotrophy	2 (8.6%)	4 (11.4%)	>0.05
Premature discharge of amniotic fluid	4 (17.4%)	7 (20.0%)	>0.05
Premature birth	2 (8.6%)	4 (11.4%)	>0.05
Weakness of labor activity	3 (13.1%)	5 (14.3%)	>0.05

ISSN: 0019-5138
DOI: https://doi.org/10.24321/0019.5138.202606

Table 5.Physical parameters and markers of HBV infection in newborns from mothers with CHB and high viral load

Newborn's health status		Tenofovir group (n=23)	Control group (n=35)	P
Physical parameters				
On the Apgar scale	8-10	82.6%	77.2%	>0.05
	4-7	17.4%	22.8%	>0.05
	0-3	0	0	
Weight (g)		3321±316	3313±372	>0.05
Height (cm)		51.9±1.8 cm	51.3±1.9 cm	>0.05
Markers of HBV infection				
HBsAg-positive		0	8.5%	<0.05*
HBeAg-positive		0	8.5%	<0.05*
HBV DNA-positive		0	8.5%	<0.05*

Values are expressed as the % = percentage of patients. HBV – Hepatitis B virus, HBsAg – Hepatitis B Surface Antigen, HBeAg – Hepatitis B e-Antigen, *P <0.05.

Discussion

Perinatal transmission of HBV remains a significant public health issue, particularly in areas with high endemic rates, such as Kyrgyz Republic.[19] In endemic countries, up to half of chronic HBV infections stem from perinatal transmission.[20] Even with passive-active immunoprophylaxis, combining vaccination with immunoglobulin administration, the risk of vertical transmission persists in women with high viral loads, ranging from 8-30%.

This study validates the effectiveness and safety of tenofovir in pregnant women with high viremia. After 4 weeks of treatment, HBV DNA levels decreased significantly, and by delivery, most patients' viral loads had fallen below 3,000 copies/mL. In 25% of women, the viral load was undetectable. These results align with those of Pan et al. and Greenup et al., who showed similar rapid HBV suppression with tenofovir during the third trimester of pregnancy.[10,18]

Notably, no neonatal infections occurred in the tenofovir group, even without consistent availability of HBV immunoglobulin. This highlights the crucial role of tenofovir in prevention, especially in resource-constrained settings. Our findings align with the EASL (2017, 2023) and WHO (2020) guidelines, which state that antiviral therapy in women with HBV DNA levels exceeding 200,000 IU/ml significantly reduces the risk of vertical transmission.[16,17]

Therefore, it is essential to verify the safety of tenofovir. The study found no differences in obstetric complications, birth defects, or physical development in newborns between groups. There were no cases of liver toxicity or virological failure in the treated women. These findings align with reviews suggesting a favorable safety profile for tenofovir during pregnancy.

Compared to nucleoside analogs such as lamivudine and telbivudine, tenofovir has several advantages. Lamivudine has a high resistance risk and limited safety (Food and Drug Administration category C), whereas telbivudine shows less antiviral activity. Tenofovir, rated category B by the Food and Drug Administration, offers a high resistance barrier and proven safety, making it the preferred first-line treatment per international guidelines.[16,21,22]

Stopping therapy four weeks postpartum proved clinically appropriate, allowing breastfeeding without the risk of virological relapse. This aligns with the current international management strategies.

In addition to achieving viral suppression at delivery, evidence shows that maternal tenofovir treatment may decrease postpartum liver flares during increased immune activity. Postpartum ALT flares and HBV reactivation occur in 25–44% of untreated women with chronic hepatitis B, particularly in those with high viral loads. Studies have shown that maintaining antiviral prophylaxis until delivery, with planned discontinuation, reduces postpartum hepatitis flares while ensuring maternal and neonatal safety.[21,23] These align with international liver societies and WHO recommendations for personalized antiviral discontinuation with postpartum monitoring during the first six months after childbirth.[23,24]

Real-world data from low- and middle-income countries demonstrate the benefits of tenofovir-based prophylaxis during pregnancy. Studies from Asia and Africa have shown

ISSN: 0019-5138
DOI: https://doi.org/10.24321/0019.5138.202606

that antenatal antiviral therapy reduces mother-to-child transmission, where timely vaccination and immunoglobulin administration are inconsistent.[23,25] Analyses indicate that maternal HBV DNA screening with third-trimester tenofovir is cost-effective for preventing new chronic HBV infections.[24,25] These findings support the incorporation of maternal antiviral therapy into national antenatal care programs alongside neonatal immunization, particularly in high-endemicity regions, targeting the 2030 hepatitis B elimination goals.

This study emphasizes the importance of determining the viral load in HBsAg-positive pregnant women. Initiating tenofovir from the 28th week of pregnancy is recommended to prevent vertical HBV transmission, particularly in areas where immunoglobulin access is limited.

Conclusions

Tenofovir administration during the third trimester in pregnant women with chronic hepatitis B and high viral loads reduces HBV DNA levels, with 26.1% achieving undetectable levels by delivery. Tenofovir effectively prevents vertical HBV transmission, as no infections occurred in newborns of treated mothers compared to an 8.5% infection rate in controls. Tenofovir treatment is safe for both the mother and fetus, without negative impacts on pregnancy, childbirth, or newborn development, or significant side effects. In areas with limited immunoglobulin access, initiating tenofovir treatment from week 28 of pregnancy is crucial for preventing vertical HBV transmission in endemic regions.

Source of Funding: None

Conflict of Interest: None

Authors Contribution: Conceptualization, Myskal Abdikerimova, and Gulmira Suranbaeva; methodology, Altynai Kanatbekova, and Mamazia Abdikerimov; data collection, Madina Mambetova; writing—original draft preparation, Begaiym Ysmaiylova, and Anara Djumagulova; writing—review and editing, Madina Mambetova, Saparbay Zholdoshev, and Narynbek Aitkuluev. All authors have read and agreed to the published version of the manuscript.

Declaration of Generative AI and AI-Assisted Technologies in the Writing Process: None

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ISSN: 0019-5138
DOI: https://doi.org/10.24321/0019.5138.202606