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Co-infections by EBV, CMV, and *Helicobacter pylori* are highly frequent in liver transplant recipients

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ABSTRACT

The objectives of this study were to determine the frequency of multiple infections by *Helicobacter pylori*, Epstein-Barr virus (EBV), and human cytomegalovirus (HCMV) and to relate the infection by EBV and HCMV with *H. pylori cagA* genotypes in the lymph nodes in liver transplant recipients. A total of 43 HCV-positive liver-transplant patients were selected. They performed a history interview, physical, and biochemical examination. DNA was extracted from paraffin-embedded (enlarged perihepatic lymph node) tissue to detect *H. pylori* infection by polymerase chain reaction (PCR) targeting the UreA gene and the virulence gene. Additionally, Antibody screening assays on blood samples were used to look for antibodies against EBV and HCMV. In all, 53.5% patients showed *H. pylori* infection with UreA gene detection. Out of them, 8 (34.8%) were positive for the CagA virulence gene. Regarding CMV, 95.3% were CMV IgG-positive, and coinfection with *H. pylori* was detected in 56.9%. Regarding EBV, 79.7% were EBV IgG-positive, and coinfection with *H. pylori* was detected in 50.0% of cases. A triple infection of EBV, HCMV, and *H. pylori* was detected in 41.9% of cases. Significant differences were found between single infections or coinfections of *H. pylori* and its genotypes with EBV or HCMV infection. Because graft survival requires immunosuppression, co-infections with EBV, CMV, and *H. pylori* pose a serious risk to liver transplant recipients. To improve patient outcomes, it is essential to comprehend the risks and put the right management techniques into practice.

Keywords: *Helicobacter pylori*, Liver transplantation, Lymph nodes, EBV, HCMV.

INTRODUCTION

Liver transplantation (LT) can be a lifesaving treatment for patients with end-stage liver disease, with a one- and five-year survival rate of 90% and 80%, respectively (Rana *et al*., 2019; Ruijter *et al*., 2023). Infections in transplant recipients account for the main cause of mortality and morbidity, with rates of up to 80%, despite advanced surgical techniques, new immunosuppressive drugs, prophylactic antibiotics, vaccination, and infection control strategies (Pagani *et al*., 2021; Shbaklo *et al*., 2022). Bacterial infections represent up to 70% of all infections in liver transplants, followed by fungal and viral infections. The

virulence of the pathogen, along with the intensity and timing of exposure, can also impact the severity and outcome of the infection (Incicco *et al*., 2023). For these reasons, international guidelines state that active infections should be adequately treated before LT. However, the optimal timing of LT in patients surviving an episode of infection, as well as their prioritization on the LT waiting list, is still to be established (Zhang *et al*., 2020).

Infectious mononucleosis (IM) is a clinical syndrome of the reticuloendothelial and lymphatic system, mostly caused by Epstein-Barr virus (EBV) but also by other infections, including cytomegalovirus (CMV). EBV and CMV, members of the herpesvirus family, establish lifelong latent infection and can reactivate. More than 90% of adults have acquired at least EBV or CMV (Zhang *et al*., 2020). EBV can promote the replication of Hepatitis C virus (HCV) after infection, thereby leading to the conversion of HCV to liver cancer in patients (Pagani *et al*., 2021). The clinical symptoms after infection with EBV include fever, lymph node enlargement, and liver enlargement (Incicco *et al*., 2023). Studies have shown that about 85% of patients with EBV infection have liver function impairment of varying degrees, and about 6% of hepatitis cases are caused by EBV infection. EB virus infection increases the level of transaminase in the body, leading to liver function damage, which is usually characterized by swelling of liver cells (Zhang *et al*., 2020).

CMV is an enveloped DNA virus that, like other members of the herpes virus family, establishes a lifelong latency period after primary infection and becomes resident in monocytes and granulocytes. For this reason, vertical transmission can occur through primary infection, reactivation of the disease, or even contamination with another strain (Tulip *et al*., 2022). CMV continues to be the most important infectious complication following solid organ transplantation (SOT), where it may cause adverse outcomes for allograft and recipient survival due to a significant number of direct and indirect effects, including CMV disease, drug-related toxicities, bacterial and opportunistic superinfections, and graft rejection. Moreover, it may increase the cost of transplantation and negatively impact the quality of life after SOT (Pontes *et al*., 2024). Up to 20% of CMV IgG-positive liver transplant recipients develop disease in the absence of a prevention strategy (Grossi and Peghin, 2024).

Helicobacter pylori (*H. pylori*), a human pathogen, is one of the most common infections worldwide and a newly emerging bacteria with significant public health implications, affecting approximately 50% of the world's population (Horiuchi *et al*., 2021; Fernández-García *et al*., 2022). In Egypt, the prevalence was reported to be 60%-80% of the adult population, where it is the most common cause of dyspepsia (Horiuchi *et al*., 2021). Researchers from all over the world are interested in its role in the development of liver conditions, including liver cirrhosis. A curious finding is that *H. pylori* has also been linked to the aetiology of hepatic encephalopathy, a common complication that affects between 30 and 70% of patients with liver cirrhosis (Fagoonee *et al*., 2019; Rasi-Bonab *et al*., 2021). Infection with cytotoxin-associated gene A (cagA)-positive *H. pylori* is associated with disease severity. *CagA* is an oncoprotein, so earlier detection and treatment of *H. pylori,* particularly the *cagA*-positive strain, are of great importance (Flamm *et al*., 2018).

In this study, the frequency of single infections and coinfections by EBV, HCMV, and *H. pylori* in liver-transplant HCV-positive patients has been investigated. In addition, the prevalence of *H. pylori cagA* genotypes was determined, and the relationship between the viruses and *H.*

pylori-specific genotypes was evaluated. Also, the frequency of each pathogen in dual and triple infections was analyzed.

METHODOLOGY

Study design and Patient enrollment

Between April 2019 and May 2021, 43 patients participated in this retrospective cross-sectional pilot study among the liver transplantation program of the NHTMRI at the National Hepatology and Tropical Medicine Research Institute. Data on the patients' clinical, radiological, demographic, hematological, and biochemical findings were assessed both at the start of the study and over the course of the follow-up periods. This study included patients with liver cirrhosis who were above the age of 20. The study did not include patients who had a history of chronic diseases like diabetes, kidney problems, or hematologic disorders.

Tissue and Blood Samples Collection

Sections of the 4 µm-thick, paraffin-embedded lymph node tissue blocks were inserted into sterile 1.5 ml centrifuge tubes after being treated in formalin. A distinct microtome knife had been used for each sample to avoid cross-contamination. Fresh venous blood samples (10 mL) were taken from all patients after an overnight fast. Five mL were taken without any anticoagulants for serum testing, 2.5 mL were taken with EDTA for complete blood count (CBC), and 2.5 mL were taken with citrate for coagulation profile analysis. Following that, the serum samples were separated into aliquots and stored at-20°C pending further analysis.

Biochemical analysis

All serum samples used in this study were subjected to a variety of laboratory tests such as ALT, AST, T. Bilirubin, D. Bilirubin albumin, urea, creatinine, ureic acid, cholesterol, triglyceride, HDL and LDL tested using a Beckman Coulter automated biochemical analyzer, electrolytes (Na, K, T. calcium) were measured by AVL9180 Electrolyte Analyzer using the Ion-Selective Electrode (ISE) method. The coagulation profile, PT, and INR of all citrated plasma samples were measured using a cobast 511 coagulation analyzer. Additionally, the CBC of the EDTA blood samples was performed using the Sysmex Xn-550 Cell Counter and blood types. By using the Cobas 6000 (e 601 module), RT-PCR for HCV RNA and HBV DNA, and the hepatitis B surface antigen (HBsAg), anti-HCV antibodies were identified.

EBV and CMV detection

Serum samples were used to evaluate the presence of IgG antibodies against EBV and CMV. Anti-CMV and anti-EBV antibody (IgG) levels were measured with an in-house developed multiplex immunoassay (MIA) (de Melo *et al*., 2022). Individuals were CMV-seropositive with a level of more than 5 relative units (RU)/ml (Yun *et al*., 2014), whereas the threshold for EBV seropositivity was 22 RU/ml, and individuals were considered EBV-seronegative with a level $\leq$ 16 RU/ml.

DNA Extraction

Using Thermo Scientific ^{TM} K0171 following the manufacturer's instructions, DNA was extracted from Formalin-fixed paraffin-embedded (FFPE) enlarged lymph node tissues by deparaffinization, rehydration, and homogenization as described by Sasaki *et al*., (1999). Until usage, all extracted DNA was kept in a -20°C freezer. Using NanoDropTM 2000/2000c (Thermo Fisher Scientific, Waltham, MA, USA), DNA from tissues was examined for purity and concentration.

Detection of *H. pylori*

Nested PCR (n-PCR) for *H. pylori* targeting the UreA gene was performed initially for infection screening, then to confirm *H. pylori* species by detecting the virulence gene (CagA). When the amplified PCR products were electrophoresed on a 1.5% agarose gel, they revealed a 200-bp fragment of the UreA (Hirai *et al*., 2009) and a 550-bp fragment of the cagA (Tcherniaeva *et al*., 2018). The amplified DNA was stained with ethidium bromide and examined on a gel while being compared to a 100bp DNA ladder (Fermentas, Thermo Fisher Scientific Inc.).

Statistical analysis

Version 28 of the Statistical Package for Social Sciences (SPSS) was used to analyze the data. Data were presented as means ± standard deviation (SD). The Mann-Whitney test was used to assess the significance of differences in non-parametric continuous variables, and the independent t-test was performed to compare two independent groups of each infection. The significance of the difference between categorical variables was evaluated using the Chi-square test. The risk associated with each group was determined using the odds ratio (OR) and 95% confidence intervals (CI). When comparing the strength and significance of associations between parametric and non-parametric variables, Spearman's correlations were used instead of Pearson's. The correlation coefficient indicated the power of the association, and a statistically significant difference was detected when $P < 0.05$.

RESULTS

Patient's baseline characteristics

Among the 43 LT recipients, 37 (86%) were men and 6 (14%) were women, with a mean age of 46.53± 15.1 years, ranging from 20 to 85 years.

***H. pylori* prevalence and biochemical profiles**

Overall, *H. pylori* DNA was found in 23/43 (53.5%) of LT patients, and the CagA was detected in 8/23 (34.8%) of *H. pylori*-positive tissues. The comparison of sociodemographic and clinical parameters according to *H. pylori* infection is shown in Table (1). When biochemical parameters and *H. pylori* infection were analyzed, albumin and Na decreased significantly in the infected group ($P<0.05$). The baseline characteristics of age, gender, and biochemical parameters showed a significant association between infections with the CagA strain and younger age, decreased albumin, urine creatinine, HB, platelets, and total calcium. On the other hand, urea, creatinine, uric acid, INR, K, and triglyceride were significantly higher in infected than non-infected patients (Table 2).

Table 1: Comparison of sociodemographic and clinical parameters according to *H.pylori* infection.

Variables		*H. pylori* positive (n=23)	*H. pylori* Negative (n=20)	*P* value
Sociodemographic parameters	Age	48.7 ± 15.9	44.3 ± 14.3	0.352^{NS}
	Sex (M/F)	19/4	18/2	0.787^{NS}
Liver functions	ALT	49.1 ± 38.6	68.1 ± 95.8	0.388^{NS}
	AST	85.3 ± 61.6	103.1 ± 136.1	0.576^{NS}
	Total Bilirubin	4.5 ± 4.5	2.8 ± 2.3	0.122^{NS}
	Direct Bilirubin	2.3 ± 3.7	1.3 ± 1.5	0.259^{NS}
	Albumin	2.7 ± 0.5	3.4 ± 1.3	0.021^{S}
Kidney Functions	Urea	29.4 ± 9.0	29.7 ± 10.2	0.923^{NS}
	Creatinine	0.8 ± 0.2	0.9 ± 0.3	0.105^{NS}
	Uric acid	5.3 ± 1.6	5.8 ± 2.3	0.425^{NS}
Complete Blood Picture	HB	11.6 ± 2.7	12.3 ± 1.9	0.189^{NS}
	TLC	4.9 ± 2.1	5.2 ± 2.8	0.701^{NS}
	PLT	109.4 ± 78.3	129.1 ± 113.8	0.507^{NS}
Coagulation profile	PT	19.6 ± 4. 8	17.3 ± 3.4	0.082^{NS}
	INR	1.5 ± 0.4	1.5 ± 0.3	0.807^{NS}
Electrolytes	Na	133.0 ± 5.2	136.9 ± 3.0	0.005^{HS}
	K	4.0 ± 0.7	4.4 ± 0.7	0.123^{NS}
	Calcium Total	8.6 ± 0.7	8.6 ± 0. 7	0.776^{NS}
Blood glucose profile	FBS	97.1 ± 27.7	101.8 ± 41.8	0.664^{NS}
	PPBS	165.7 ± 90.3	154.1 ± 82.1	0.661^{NS}
	HbA1C	5.1 ± 1.5	5.4 ± 1.0	0.452^{NS}
Lipid profile	Cholesterol	134.0 ± 52.8	125.7 ± 37.9	0.559^{NS}
	Triglyceride	91.2 ± 56.8	92.2 ± 45.5	0.951^{NS}
	HDL	48.9 ± 55.7	37.9 ± 13.3	0.395^{NS}
	LDL	83.7 ± 48.7	70.3 ± 31.84	0.301^{NS}
	VLDL	17.3 ± 11.0	15.9 ± 7.3	0.479^{NS}

NS: Non-significant at p-value ≥0.05; S: Significant at p-value <0.05; HS: Highly significant at p-value < 0.01

Table 2: Comparison of sociodemographic and clinical parameters according to *CagA*.

Variables		*H. pylori* $CagA^{+}$ (n=8)	*H. pylori* $CagA^{-}$ (n=15)	*P* value
Sociodemographic parameters	Age	40.6 ± 12.1	53.1± 15.4	0.007^{HS}
	Sex (M/F)	8/0	11/4	0.091^{NS}
Liver functions	ALT	52.9 ± 41.7	47.1 ± 35.2	0.619^{NS}
	AST	89.0 ± 66.3	83.3 ± 56.9	0.763^{NS}
	Total Bilirubin	3.0 ± 2.0	5.33 ± 5.1	0.088^{NS}
	Direct Bilirubin	1.4 ± 1.1	2.8 ± 4.3	0.210^{NS}
	Albumin	2.5 ± 0.6	2.8 ± 0.4	0.045^{S}
Kidney Functions	Urea	35.6 ± 6.0	26.3 ± 8.1	0.0002^{HS}
	Creatinine	1.0 ± 0.2	0.7 ± 0.2	$< 0.0001^{HS}$
	Uric acid	6.3 ± 1.5	4.7 ± 1.4	0.0005^{HS}
Complete Blood Picture	HB	10.0 ± 2.8	12.1 ± 2.4	0.01^{HS}
	TLC	4.2 ± 1.3	5.3 ± 2.2	0.091^{NS}
	PLT	80.0 ± 23.7	125.1 ± 89.1	0.050^{S}
Coagulation profile	PT	20.6 ± 3.3	19.0 ± 5.2	0.264^{NS}
	INR	1. 7 ± 0.3	1.4 ± 0.4	0.028^{S}
Electrolytes	Na	133.4 ± 5.4	132.9 ± 4.9	0.748^{NS}
	K	4.3 ± 0.7	3.9 ± 0.6	0.037^{S}
	Calcium Total	8.2 ± 0.7	8.8 ± 0.6	0.002^{NS}
Blood glucose profile	FBS	91.6 ± 15.4	100.0 ± 31.2	0.319^{NS}
	PPBS	159.4 ± 67.6	169.1 ± 97.4	0.723^{NS}
	HbA1C	5.1 ± 0.5	5.2 ± 1.8	0.831^{NS}
Lipid profile	Cholesterol	141.6 ± 56.2	119.8 ± 37.9	0.171^{NS}
	Triglyceride	103.5 ± 63.8	68.1 ± 21.1	0.037^{S}
	HDL	49.0 ± 36.2	48.8 ± 62.2	0.991^{NS}
	LDL	69.5 ± 28.2	91.2 ± 53.7	0.140^{NS}
	VLDL	19.2 ± 12.5	13.8 ± 4.35	0.100^{NS}

NS: Non-significant at p-value ≥0.05; S: Significant at p-value <0.05; HS: Highly significant at p-value < 0.01

CMV prevalence, coinfection with *H. pylori,* and biochemical profiles association

Of all patients, 95.3% (41/43) were CMV IgG-positive; the sociodemographic and clinical parameters according to CMV infection (positive IgG) are shown in Table (3). All the studied biochemical parameters were statistically insignificant. Regarding coinfection with *H. pylori*, it was detected in 23/41 (56.9%), of them 8/23 (34.8%) showed coinfection with *H. pylori* CagA-positive. *H. pylori* and CMV coinfection prevalence and its association with the studied parameters are shown in Table (4).

Table 3: Comparison of sociodemographic and clinical parameters according to CMV infection status.

Variables		CMV positive IgG (n=41)	CMV Negative IgG (n=2)	*P* value
Sociodemographic parameters	Age	47.2±14.8	30.5±9.2	0.124^{NS}
	Sex (M/F)	35/6	2/0	0.985^{NS}
Liver functions	ALT	58.3 ± 72.5	49.5 ± 17.7	0.453^{NS}
	AST	94.9 ± 104.6	67.5 ± 7.8	0.731^{NS}
	Total Bilirubin	3.8 ± 3.8	2.0 ± 0.5	0.512^{NS}
	Direct Bilirubin	1.9 ± 3.0	1.0 ± 0.2	0.677^{NS}
	Albumin	3.1 ± 1.1	2.5 ± 0.1	0.438^{NS}
Kidney Functions	Urea	29.6 ± 9.6	29.5 ± 2.1	0.989^{NS}
	Creatinine	0.9 ± 0.3	0.9 ± 0.01	1.00^{NS}
	Uric acid	5.5 ± 2.0	6.0 ± 0.07	0.729^{NS}
Complete Blood Picture	HB	11.8 ± 2.5	11.5 ± 0.8	0.868^{NS}
	TLC	6.5 ± 3.4	3.6 ± 0.1	0.240^{NS}
	PLT	121.0 ± 97.4	68.5 ± 16.3	0.455^{NS}
Coagulation profile	PT	18.4 ± 4.3	20.9 ± 5.8	0.431^{NS}
	INR	1.5 ± 0.3	1.6 ± 0.2	0.646^{NS}
Electrolytes	Na	134.8 ± 4.8	135.0 ± 4.2	0.954^{NS}
	K	4.2 ± 0.7	4.3 ± 0.2	0.843^{NS}
	Calcium Total	8.6 ± 0.7	8.3 ± 0.3	0.553^{NS}
Blood glucose profile	FBS	99.6 ± 35.4	93.0 ± 12.7	0.796^{NS}
	PPBS	163.5 ± 86.4	94.5 ± 34.7	0.272^{NS}
	HbA1C	5.3 ± 1.3	5.3 ± 0.3	1.00^{NS}
Lipid profile	Cholesterol	130.6 ± 46.9	120.0 ± 35.4	0.755^{NS}
	Triglyceride	93.0 ± 52.1	64.0 ± 5.7	0.441^{NS}
	HDL	43.7 ± 42.5	45.0 ± 26.9	0.966^{NS}
	LDL	78.2 ± 42.6	62.0 ± 9.9	0.598^{NS}
	VLDL	16.8 ± 9.6	13.5 ± 0.7	0.633^{NS}

NS: Non-significant at p-value ≥0.05; S: Significant at p-value <0.05; HS: Highly significant at p-value < 0.01

Table 4: **Comparison of sociodemographic and clinical parameters according to Coinfection of CMV and *H. pylori*.**

Variables		*H. pylori* & CMV positive IgG (n=23)	*H. pylori* Negative & CMV Positive IgG (n=18)	*P* value	*H. pylori* CagA & CMV positive IgG (n=8)	*H. pylori* CagA Negative & CMV Positive IgG (n=15)	*P* value
Sociodemographic parameters	Age	48.7 ± 15.9	49.9 ± 14.7	0.806^{NS}	40.6 ± 12.1	53.1± 15.4	**0.007^{HS}**
	Sex (M/F)	19/4	16/2	0.575^{NS}	8/0	11/4	0.091^{NS}
Liver functions	ALT	49.1 ± 38.6	45.7 ± 33.5	0.769^{NS}	52.9 ± 41.7	47.1 ± 35.2	0.619^{NS}
	AST	85.3 ± 61.6	82.1 ± 54.2	0.863^{NS}	89.0 ± 66.3	83.3 ± 56.9	0.763^{NS}
	Total Bilirubin	4.5 ± 4.5	5.1 ± 5.0	0.689^{NS}	3.0 ± 2.0	5.33 ± 5.1	0.088^{NS}
	Direct Bilirubin	2.3 ± 3.7	2.7 ± 4.1	0.745^{NS}	1.4 ± 1.1	2.8 ± 4.3	0.210^{NS}
	Albumin	2.7 ± 0.5	2.8 ± 0.5	0.529^{NS}	2.5 ± 0.6	2.8 ± 0.4	**0.045^{S}**
Kidney Functions	Urea	29.4 ± 9.0	30.3 ± 9.8	0.762^{NS}	35.6 ± 6.0	26.3 ± 8.1	**0.0002^{HS}**
	Creatinine	0.8 ± 0.2	0.8 ± 0.2	1.00^{NS}	1.0 ± 0.2	0.7 ± 0.2	**001^{HS}**
	Uric acid	5.3 ± 1.6	5.5 ± 1.6	0.693^{NS}	6.3 ± 1.5	4.7 ± 1.4	**0.0005^{HS}**
Complete Blood Picture	HB	11.6 ± 2.7	11.4 ± 3.0	0.824^{NS}	10.0 ± 2.8	12.1 ± 2.4	**0.01^{HS}**
	TLC	4.9 ± 2.1	5.0 ± 2.2	0.883^{NS}	4.2 ± 1.3	5.3 ± 2.2	0.091^{NS}
	PLT	109.4 ± 78.3	114.9 ± 85.1	0.831^{NS}	80.0 ± 23.7	125.1 ± 89.1	**0.050^{S}**
Coagulation profile	PT	19.6 ± 4. 8	19.3 ± 4.7	0.842^{NS}	20.6 ± 3.3	19.0 ± 5.2	0.264^{NS}
	INR	1.5 ± 0.4	1.5 ± 0.4	1.00^{NS}	1. 7 ± 0.3	1.4 ± 0.4	**0.028^{S}**
Electrolytes	Na	133.0 ± 5.2	132.1 ± 4.8	0.573^{NS}	133.4 ± 5.4	132.9 ± 4.9	0.748^{NS}
	K	4.0 ± 0.7	3.9 ± 0.7	0.652^{NS}	4.3 ± 0.7	3. 9 ± 0.6	**0.037^{S}**
	Calcium Total	8.6 ± 0.7	8.6 ± 0.7	1.00^{NS}	8.2 ± 0.7	8.8 ± 0.6	**0.002^{NS}**
Blood glucose profile	FBS	97.1 ± 27.7	100.3 ± 30.2	0.726^{NS}	91.6 ± 15.4	100.0 ± 31.2	0.319^{NS}
	PPBS	165.7 ± 90.3	181.3 ± 93.9	0.593^{NS}	159.4 ± 67.6	169.1 ± 97.4	0.723^{NS}
	HbA1C	5.1 ± 1.5	5.1 ± 1.7	0.429^{NS}	5.1 ± 0.5	5.2 ± 1.8	0.831^{NS}
Lipid profile	Cholesterol	134.0 ± 52.8	135.8 ± 58.4	0.918^{NS}	141.6 ± 56.2	119.8 ± 37.9	0.171^{NS}
	Triglyceride	91.2 ± 56.8	99.3 ± 62.1	0.666^{NS}	103.5 ± 63.8	68.1 ± 21.1	**0.037^{S}**
	HDL	48.9 ± 55.7	51.1 ± 62.6	0.906^{NS}	49.0 ± 36.2	48.8 ± 62.2	0.991^{NS}
	LDL	83.7 ± 48.7	86.3 ± 54.2	0.873^{NS}	69.5 ± 28.2	91.2 ± 53.7	0.140^{NS}
	VLDL	17.3 ± 11.0	18.6 ± 12.2	0.722^{NS}	19.2 ± 12.5	13.8 ± 4.35	0.100^{NS}

NS: Non-significant at p-value ≥0.05; S: Significant at p-value <0.05; HS: Highly significant at p-value < 0.01

EBV prevalence, coinfection with *H. pylori*, and biochemical profiles association

Of all patients, 79.7% (34/43) were EBV IgG-positive; the sociodemographic and clinical parameters according to EBV infection (positive IgG) are shown in Table (5). All the studied biochemical parameters were statistically insignificant. Regarding coinfection with *H. pylori*, it was detected in 17/34 (50.0%), of them 6/17 (35.3%) showed coinfection with *H. pylori* CagA-positive. *H. pylori* and EBV coinfection prevalence and its association with the studied parameters are shown in Table (6).

Table 5: **Comparison of sociodemographic and clinical parameters according to EBV infection status.**

Variables		EBV positive IgG (n=34)	EBV Negative IgG (n=9)	*P* value
Sociodemographic parameters	Age	47.1 ± 15.7	45.0 ± 15.0	0.721^{NS}
	Sex (M/F)	27/7	9/0	0.275^{NS}
Liver functions	ALT	54.4 ± 43.1	37.1 ±16.8	0.248^{NS}
	AST	87.4 ± 67.1	70.3 ± 50.9	0.482^{NS}
	Total Bilirubin	3.4 ± 2.8	5.7 ± 6.5	0.115^{NS}
	Direct Bilirubin	1.5 ± 1.1	3.4 ±6.2	0.089^{NS}
	Albumin	2.9 ± 0.8	3.4 ±1.7	0.206^{NS}
Kidney Functions	Urea	28.3 ± 10.1	29.1 ± 4.3	0.819^{NS}
	Creatinine	0.9 ± 0.3	0.8 ± 0.2	0.352^{NS}
	Uric acid	5.4 ± 2.1	5.2 ± 1.5	0.791^{NS}
Complete Blood Picture	HB	11.2 ± 2.2	13.2 ± 2.5	0.023^{S}
	TLC	4.7 ± 1.7	5.0 ±2.6	0.677^{NS}
	PLT	108.7 ± 87.5	130.4 ±107.2	0.531^{NS}
Coagulation profile	PT	19.1 ± 4.6	20.3 ± 5.6	0.509^{NS}
	INR	1.6 ± 0.4	1.5 ± 0.4	0.509^{NS}
Electrolytes	Na	134.2 ± 5.1	135.3 ± 3.2	0.543^{NS}
	K	4.1 ± 0.7	4.2 ± 0.6	0.689^{NS}
	Calcium Total	8.6 ± 0.8	8.6 ± 0.5	1.00^{NS}
Blood glucose profile	FBS	103.6 ± 43.2	93.8 ± 7.3	0.505^{NS}
	PPBS	174.8 ± 106.5	129.3 ± 47.7	0.222^{NS}
	HbA1C	5.3 ± 1.8	5.2 ± 0.6	0.871^{NS}
Lipid profile	Cholesterol	128.6 ± 40.6	140.0 ± 67.9	0.523^{NS}
	Triglyceride	79.1 ± 31.7	102.9 ± 81.4	0.174^{NS}
	HDL	57.2 ± 61.8	35.0 ± 16.8	0.296^{NS}
	LDL	77.3 ± 36.1	84.5 ± 62.0	0.653^{NS}
	VLDL	14.9 ± 5.6	19.0 ± 16.6	0.226^{NS}

NS: Non-significant at p-value ≥0.05; S: Significant at p-value <0.05; HS: Highly significant at p-value < 0.01

Table 6: **Comparison of sociodemographic and clinical parameters according to Coinfection of EBV and *H.pylori* .**

Variables		*H. pylori* & EBV positive IgG (n=17)	*H. pylori* Negative & EBV Positive IgG (n=17)	*P* value	*H. pylori* CagA &EBV positive IgG (n=6)	*H. pylori* CagA Negative & EBV Positive IgG (n=11)	*P* value
Sociodemo-graphic parameters	Age	48.7 ± 16.1	45.6 ± 15.7	0.578^{NS}	44.0 ± 12.8	51.2 ± 17.7	0.472^{NS}
	Sex (M/F)	13/4	14/3	0.671^{NS}	6/0	7/4	0.091^{NS}
Liver functions	ALT	53.5 ± 43.0	55.2 ± 44.5	0.777^{NS}	54.0 ± 52.1	53.3 ± 40.0	0.894^{NS}
	AST	90.5 ± 64.5	84.3 ± 71.5	0.882^{NS}	96.2 ± 82.2	87.4 ± 56.9	0.794^{NS}
	Total Bilirubin	3.6 ± 3.0	3.2 ± 2.6	0.489^{NS}	3.4 ± 2.4	3.8 ± 3.4	0.450^{NS}
	Direct Bilirubin	1.6 ± 1.2	1.4 ± 1.1	0.492^{NS}	1.6 ± 1.3	1.6 ± 1.1	0.858^{NS}
	Albumin	2.8 ± 0.6	3.0 ± 1.1	0.190^{NS}	2.5 ± 0.8	2.9 ± 0.4	0.206^{NS}
Kidney Functions	Urea	30.2 ± 10.0	26.4 ± 10.1	0.851^{NS}	37.8 ± 5.9	26.0 ± 9.4	0.433^{NS}
	Creatinine	0.8 ± 0.3	0.9 ± 0.3	0.434^{NS}	1.0 ± 0.2	0.7 ± 0.2	0.920^{NS}
	Uric acid	5.4 ± 1.7	5.4 ± 2.5	0.110^{NS}	6.5 ± 1.8	4.8 ± 1.3	0.350^{NS}
Complete Blood Picture	HB	11.0 ± 2.8	11.4 ± 1.5	0.047^{S}	9.6 ± 3.3	11.7 ± 2.4	0.830^{NS}
	TLC	4.9 ± 1.7	4.4 ± 1.6	0.726^{NS}	4.5 ± 1.5	5.1 ± 1.8	0.471^{NS}
	PLT	113.7 ± 78.8	103.8 ± 97.7	0.983^{NS}	83.8 ± 27.8	129.9 ± 93.3	0.007^{HS}
Coagulation profile	PT	19.3 ± 4.4	18.9 ± 4.9	0.629^{NS}	20.5 ± 3.2	18.7 ± 4.9	0.383^{NS}
	INR	1.5 ± 0.4	1.6 ± 0.4	0.805^{NS}	1.7 ± 0.3	1.4 ± 0.3	0.939^{NS}
Electrolytes	Na	132.6 ± 5.9	135.9 ± 3.4	0.040^{S}	132.8 ± 6.4	132.5 ± 6.0	0.709^{NS}
	K	4.0 ± 0.7	4.2 ± 0.8	0.632^{NS}	4.3 ± 0.9	3.9 ± 0.5	0.137^{NS}
	Calcium Total	8.6 ± 0.8	8.6 ± 0.8	1.00^{NS}	8.1 ± 0.8	8.8 ± 0.6	0.356^{NS}
Blood glucose profile	FBS	97.4 ± 32.2	109.9 ± 52.2	0.090^{NS}	91.2 ± 18.6	100.7 ± 38.1	0.646^{NS}
	PPBS	172.8 ± 102.1	176.7 ± 113.8	0.813^{NS}	181.0 ± 69.5	168.4 ± 119.1	0.598^{NS}
	HbA1C	5.1 ± 1.8	5.4 ± 1.8	0.654^{NS}	5.0 ± 0.6	5.2 ± 2.2	0.208^{NS}
Lipid profile	Cholesterol	132.5 ± 43.6	124.7 ± 38.3	0.573^{NS}	119.7 ± 45.3	139.6 ± 43.2	0.717^{NS}
	Triglyceride	86.4 ± 38.4	71.8 ± 22.0	0.043^{S}	69.5 ± 26.4	95.6 ± 41.8	0.261^{NS}
	HDL	54.4 ± 63.4	59.9 ± 61.9	0.957^{NS}	50.3 ± 44.0	56.6 ± 73.8	0.687^{NS}
	LDL	83.5 ± 40.6	71.1 ± 30.9	0.557^{NS}	72.0 ± 35.0	89.8 ± 43.6	0.643^{NS}
	VLDL	16.0 ± 6.8	13.8 ± 4.0	0.044^{S}	13.8 ± 5.5	17.2 ± 7.4	0.445^{NS}

NS: Non-significant at p-value ≥0.05; S: Significant at p-value <0.05; HS: Highly significant at p-value < 0.01

Table 7: **Comparison of sociodemographic and clinical parameters according to Coinfection of EBV and *H. pylori*.**

Variables		*H. pylori* positive (n=23)	CMV positive IgG (n=41)	EBV positive IgG (n=34)	Multiple infection (triple infection (n=18)	*P* value
Socio-demographic parameters	Age	48.7 ± 15.9	47.2 ± 14.8	47.1 ± 15.7	47.8 ± 15.0	0.979^{NS}
	Sex (M/F)	19/4	35/6	27/7	15/3	
Liver functions	ALT	49.1 ± 38.6	58.3 ± 72.5	54.4 ± 43.1	69.7 ± 100.4	0.779^{NS}
	AST	85.3 ± 61.6	94.9 ± 104.6	87.4 ± 67.1	112.7 ±138.7	0.784^{NS}
	Total Bilirubin	4.5 ± 4.5	3.8 ± 3.8	3.4 ± 2.8	3.3 ± 2.9	0.652^{NS}
	Direct Bilirubin	2.3 ± 3.7	1.9 ± 3.0	1.5 ± 1.1	1.6 ± 1.6	0.685^{NS}
	Albumin	2.7 ± 0.5	3.1 ± 1.1	2.9 ± 0.8	3.0 ± 0.6	0.334^{NS}
Kidney Functions	Urea	29.4 ± 9.0	29.6 ± 9.6	28.3 ± 10.1	32.9 ± 9.7	0.440^{NS}
	Creatinine	0.8 ± 0.2	0.9 ± 0.3	0.9 ± 0.3	0.8 ±0.3	0.351^{NS}
	Uric acid	5.3 ± 1.6	5.5 ± 2.0	5.4 ± 2.1	5.8 ± 1.5	0.729^{NS}
Complete Blood Picture	HB	11.6 ± 2.7	11.8 ± 2.5	11.2 ± 2.2	11.6 ± 2.9	0.784^{NS}
	TLC	4.9 ± 2.1	6.5 ± 3.4	4.7 ± 1.7	7.2 ±1.2	0.0005^{HS}
	PLT	109.4 ± 78.3	121.0 ± 97.4	108.7 ± 87.5	127.2 ± 92.6	0.862^{NS}
Coagulation profile	PT	19.6 ± 4.8	18.4 ± 4.3	19.1 ± 4.6	17.4 ± 2.3	0.366^{NS}
	INR	1.5 ± 0.4	1.5 ± 0.3	1.6 ± 0.4	1.4 ± 0.3	0.267^{NS}
Electrolytes	Na	133.0 ± 5.2	134.8 ± 4.8	134.2 ± 5.1	133.7 ± 6.0	0.591^{NS}
	K	4.0 ± 0.7	4.2 ± 0.7	4.1 ± 0.7	4.2 ± 0.7	0.696^{NS}
	Calcium Total	8.6 ± 0.7	8.6 ± 0.7	8.6 ± 0.8	8.6 ± 0.7	1.00^{NS}
Blood glucose profile	FBS	97.1 ± 27.7	99.6 ± 35.4	103.6 ± 43.2	91.7 ± 13.6	0.681^{NS}
	PPBS	165.7 ± 90.3	163.5 ± 86.4	174.8 ± 106.5	158.6 ± 65.7	0.926^{NS}
	HbA1C	5.1 ± 1.5	5.3 ± 1.3	5.3 ± 1.8	5.1 ± 0.9	0.919^{NS}
Lipid profile	Cholesterol	134.0 ± 52.8	130.6 ± 46.9	128.6 ± 40.6	130.8 ± 43.5	0.979^{NS}
	Triglyceride	91.2 ± 56.8	93.0 ± 52.1	79.1 ± 31.7	105.3 ±51.5	0.295^{NS}
	HDL	48.9 ± 55.7	43.7 ± 42.5	57.2 ± 61.8	32.4 ±11.2	0.353^{NS}
	LDL	83.7 ± 48.7	78.2 ± 42.6	77.3 ± 36.1	80.3 ±42.0	0.947^{NS}
	VLDL	17.3 ± 11.0	16.8 ± 9.6	14.9 ± 5.6	18.2 ± 8.7	0.568^{NS}

NS: Non-significant at p-value ≥0.05; S: Significant at p-value <0.05; HS: Highly significant at p-value < 0.01

DISCUSSION

End-stage liver disease patients undergoing liver transplantation are prone to develop numerous infectious complications because of immunosuppression, surgical interventions, and malnutrition. Infections in transplant recipients account for the main cause of mortality and morbidity, with rates of up to 80% (Shbaklo *et al*., 2022). Thus, this study aimed to determine the frequency of single infection and coinfections by EBV, HCMV, and *H. pylori* in liver-transplant HCV-positive patients. In addition, we determined the prevalence of *H. pylori cagA* genotypes and evaluated the relationship between the viruses and *H. pylori*-specific genotypes. Lastly, we analyzed the frequency of each pathogen in dual and triple infections.

In the present study, the overall *H. pylori* DNA was found in 53.5% of LT patients; of them, the cagA was detected in 34.8% of *H. pylori*-positive tissues. In contrast, Rao *et al*., (2021) reported that the prevalence of H pylori infection was lower in LT recipients than in control subjects (11.8% vs. 32.0%). The present study found that the level of serum albumin and sodium (Na) in the *H.pylori* positive group was lower than that in the negative group, with a statistically significant difference. This suggests that the presence of *H. pylori* could reduce the serum albumin level. This finding is in line with Zhang *et al*., (2024), who reported that *H.*

pylori infection was strongly linked to decreased serum albumin levels. Additionally, *H. pylori* infection may be a driving factor in the development of hypernatremia in elderly people, according to Aydin *et al*., (2022).

Regarding *H. pylori CagA* status and biochemical parameters, *H. pylori* CagA-positive individuals displayed significantly decreased levels of albumin, HB, platelets, and total calcium. These study findings agree with Asiimwe *et al*. (2023), who reported an association between *H. pylori* infection and anemia. Sadia *et al*. (2022) reported that *H. pylori* infection is becoming a more common cause of immunological thrombocytopenia (ITP). In this study, CagA-positive patients had significantly high levels of urea, creatinine, uric acid, INR, K, and triglyceride. These study results are in line with Chen *et al*. (2024), who reported that *H. pylori* infection is positively associated with a higher risk of gout in the hyperuricemia population. Xie *et al*. (2023) reported that the serum triglyceride levels were significantly higher in *H. pylori*-seropositive participants than in *H. pylori*-seronegative participants.

This is the first study to document the frequency of CMV infection just before LT in patients with chronic liver disease in lymph nodes. We observed a high frequency of CMV infection in these patients; 95.3% were CMV IgG-positive, even though it had no impact on clinically significant variables. Regarding coinfection with *H. pylori*, it was detected in 56.9%, of them 34.8% showed coinfection with *H. pylori* CagA positive. The association between coinfection of *H. pylori* and CMV and biochemical parameters was statistically insignificant. On the other hand, the coinfection with *H. pylori* CagA-positive was higher in patients of a young age. Coinfection with *H. pylori* CagA-positive individuals displayed significantly decreased levels of albumin, HB, platelets, and total calcium and a significant increase in urea, creatinine, uric acid, INR, K, and triglyceride.

79.7% (18/43) of the investigated patients were EBV IgG-positive, and the sociodemographic and clinical parameters according to EBV infection (positive IgG). All the studied biochemical parameters are statistically insignificant. Regarding coinfection with *H. pylori*, it was detected in 17/34 (50.0%), of them 6/17 (35.3%) showed coinfection with *H. pylori* CagA-positive. *H. pylori* and EBV coinfection prevalence and its association with the studied parameters.

Of all patients, 41.9% (34/43) were positive for multiple infections of CMV, EBV, and *H. pylori*, and the sociodemographic and clinical parameters according to multiple infections. All the studied biochemical parameters are statistically insignificant. TLC was significantly higher in patients with multiple infections compared to patients with a single infection.

Conclusion

Multiple infections may result in a complicated clinical picture that could cause liver disorders and other health problems. *H. pylori* can encourage latent EBV and CMV infections to reactivate, which increases viral replication and raises the risk of serious complications. This study emphasizes how common multiple infections with *H. pylori*, CMV, and EBV are in liver transplant recipients.

Ethical Approval and consent to participate:

Research Involving Human Participants. The study was conducted following the Declaration of Helsinki. All participants provided written informed consent, and the Ethics Committee of the National Hepatology and Tropical Medicine Research Institute (NHTMRI) approved the study protocol.

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العدوى المشتركة بفيروس إبشتاين-بار، والفيروس المضخم للخلايا، والملوية البوابية شائعة جدًا لدى متلقي زراعة الكبد

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المستخلص

هدفت هذه الدراسة إلى تحديد معدل الإصابة بالعدوى المتعددة بالبكتيريا الملوية البوابية، وفيروس إبشتاين-بار (EBV)، والفيروس المضخم للخلايا البشرية(HCMV) ، وربط العدوى بفيروس إبشتاين-بار وفيروس تضخم الخلايا البشرية (HCMV)بالأنماط الجينية لـ *H. pylori* CagA في العقد الليمفاوية لدى متلقي زراعة الكبد. تم اختيار 43 مريضًا مصابًا بفيروس التهاب الكبد الوبائي (HCV) ممن خضعوا لزراعة كبد. وأُجريت لهم مقابلة مع تسجيل التاريخ المرضي، وفحوصات اكلينيكية، وكيمياء الوظائف الحيوية. واستُخلص الحمض النووي (DNA) من أنسجة مُغطاة بالبارافين (تضخم العقد الليمفاوية حول الكبد) للكشف عن الإصابة ببكتيريا الملوية البوابية عن طريق تفاعل البوليميراز المتسلسل (PCR) الذي يستهدف جين اليوريا (UreA) وجين الضراوة CagA. بالإضافة إلى ذلك، استُخدمت فحوصات فحص الأجسام المضادة في عينات الدم للبحث عن أجسام مضادة لفيروس إبشتاين-بار وفيروس تضخم الخلايا البشرية. إجمالاً، أظهر 53.5% من المرضى إصابةً بالبكتيريا الحلزونية البوابية عند الكشف عن جين اليوريا .(UreA) من بينهم، كانت نتائج 8 (34.8%) إيجابية لجين الضراوة .CagA أما بالنسبة لفيروس تضخم الكبد(CMV) ، فكانت نسبة 95.3% منهم إيجابية لـIgG ، بينما كُشف عن إصابة مشتركة بالبكتيريا الحلزونية البوابية لدى 56.9%. أما بالنسبة لفيروس إبشتاين بار، فكانت نسبة 79.7% إيجابية لـIgG ، وكُشف عن إصابة مشتركة بالبكتيريا الحلزونية البوابية لدى 50%. وكُشف عن إصابة ثلاثية بفيروس إبشتاين بار، وفيروس تضخم الكبد البشري (HCMV)، والبكتيريا الحلزونية البوابية لدى 41.9%. وُجدت فروق جوهرية بين الإصابة المفردة أو الإصابة المشتركة بالبكتيريا الحلزونية البوابية وأنماطها الجينية مع إصابة فيروس إبشتاين بار أو فيروس تضخم الكبد البشري. ولأن بقاء الزرع يتطلب تثبيطًا مناعيًا، فإن الإصابة المشتركة بفيروس إبشتاين بار، والفيروس المضخم للخلايا، والبكتيريا الحلزونية البوابية تُشكل خطرًا جسيمًا على متلقي زراعة الكبد. لتحسين نتائج المرضى، من الضروري فهم المخاطر وتطبيق أساليب الإدارة الصحيحة.

الكلمات المفتاحية: بكتيريا الملوية البوابية، زراعة الكبد، العقد الليمفاوية، فيروس إبشتاين-بار، الفيروس المضخم للخلايا البشرية.