

Most Common Complications in Patients Post Liver Transplantation at a Hospital in Mexico.

Saúl A. Vera-Nungaray¹, Diego F. Abendaño-Rivera¹, Karina Cazarín-Chavez¹, María F. Higuera-De la Tijera¹, Viridiana López-Ladrón de Guevara¹, Isidoro A. Sanchez-Cedillo², Erika F. Rodríguez-Aguilar², Victor M. Paez-Zayas²

¹ Department of Gastroenterology and Hepatology, General Hospital of Mexico “Dr. Eduardo Liceaga”, Mexico
² Department of Transplantation, General Hospital of Mexico

Introduction and Objectives: Liver transplantation worldwide presents a series of complications that need to be identified to adequately treat and prevent them; however, information about these in Mexico is limited. The objective is to report the epidemiology of these complications in a third-level hospital transplant center in Mexico

Materials and Patients: A cohort study was conducted, including post-liver transplant patients from 2019 to 2023. The presence of infectious and postsurgical complications after the event was determined, as well as the rate of both acute and chronic rejection. Data are expressed using descriptive statistics with proportions and percentages for qualitative variables and mean and standard deviation or median and range for quantitative variables.

Results: 110 patients were included, 46 women and 64 men with a mean age of [insert average age]. Complications were classified according to type and timing of presentation after surgery. Regarding rejection rates, a 10.00% acute rejection was observed with a mean presentation of 7.6±6.6 months, and 2.8% chronic rejection (mean time: 17.7±8.1 months). Results are summarized in Table 1 and Figure 1.

Conclusions: Complications in liver transplant recipients are common and jeopardize both patient life and graft function. Although rejection rates remain high, our center presents a favorable epidemiology compared to reported literature, below global rates for acute rejection (15–25%) and chronic rejection (3–17%).

Ethical statement: The research was conducted following the Helsinki Declaration of the World Assembly 2013.

Declaration of interests: None.

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Table 1
Postoperative, Early, and Late Complications Following Liver Transplantation

Liver Rejection	Post operative		Early		Late	
	n	Average	n	Average	n	Average
Acute Rejection	0	0	6	5.454545455	5	4.545454545
Chronic Rejection	0	0	0	0	3	2.727272727
Infections	n	Average	n	Average	n	Average
Pneumonia	0	0	17	15.45454545	0	0
Infectious Diarrhea	0	0	14	12.72727273	0	0
Urinary Tract Infection (UTI)	1	0.90909091	9	8.181818182	0	0
Spontaneous Bacterial Peritonitis (PBE)	0	0	1	0.909090909	0	0
Upper Respiratory Tract Infection (URTI)	0	0	5	4.545454545	0	0
Cytomegalovirus (CMV)	0	0	4	3.636363636	0	0
Cholangitis	0	0	3	2.727272727	1	0.909090909
Abdominal Abscess	0	0	3	2.727272727	0	0
Oropharyngeal/Esophageal Candidiasis	0	0	3	2.727272727	0	0
Soft Tissue Infection	0	0	1	0.909090909	0	0
Osteomyelitis	0	0	1	0.909090909	0	0
Bacteremia	0	0	1	0.909090909	0	0
Postsurgical Complications	n	Average	n	Average	n	Average
Biliary Stricture	0	0	5	4.545454545	2	1.818181818
Hemothorax	1	0.90909091	0	0	0	0
Pneumothorax	1	0.90909091	0	0	0	0
Abdominal Wall Hematoma	1	0.90909091	0	0	0	0
Retropertitoneal Hematoma	1	0.90909091	0	0	0	0
Hemoperitoneum	2	1.81818182	0	0	0	0
Cavo-Suprahepatic Stenosis	0	0	0	0	1	0.909090909

(continued)

Table 1 (Continued)

Liver Rejection	Post operative		Early		Late	
	n	Average	n	Average	n	Average
Abdominal Adhesions	1	0.90909091	0	0	0	0
Miscellaneous	n	Average	n	Average	n	Average
Non-inflammatory Diarrhea	2	1.81818182	7	6.363636364	11	10
Acute Kidney Injury (AKI)	11	10	13	11.81818182	11	10
Chronic Kidney Disease (CKD)	0	0	8	7.272727273	5	4.545454545
De novo Primary Biliary Cholangitis	0	0	0	0	1	0.909090909
Portal Vein Thrombosis	0	0	1	0.909090909	0	0
Pleural Effusion	2	1.81818182	2	1.818181818	0	0
Seizures	4	3.63636364	1	0.909090909	0	0
Cancer Relapse	0	0	0	0	1	0.909090909
Non-variceal Upper Gastrointestinal Bleeding	0	0	1	0.909090909	0	0
Anal Fistula	0	0	0	0	1	0.909090909
Pathological Fractures	0	0	2	1.818181818	0	0
Lymphoid Hyperplasia	0	0	1	0.909090909	0	0
Unstable Angina	1	0.90909091	0	0	0	0
Superficial Venous System Thrombosis	0	0	0	0	1	0.909090909

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Peripheral cellular immune alterations during excessive alcohol consumption

Karla Z. Medina-Avila¹, Abigail Hernandez-Barragan¹, Marisela Hernández-Santillan¹, Isabel Villagómez-López¹, Adrián Flores-Sanchez¹, Melanie Acosta-Gutiérrez¹, Viridiana Bautista-Nava¹, Isaac B Flores-Islas¹, Jesús A Dorantes-Álvarez¹, Moisés Martínez-Castillo¹, Fátima Higuera-de la Tijera², José L Pérez-Hernández², Gabriela Gutiérrez-Reyes¹

¹ Liver, Pancreas, and Motility Laboratory (HIPAM), Experimental Medicine Research Unit, Faculty of Medicine, UNAM, General Hospital of Mexico, Dr. Eduardo Liceaga, Mexico

² Gastroenterology and Hepatology Department, General Hospital of Mexico “Dr. Eduardo Liceaga”, México

Introduction and Objectives: The mechanisms that participate in the pathophysiology of chronic alcohol consumption and Alcoholic Liver Disease (ALD) include alterations of the innate and adaptive immune system, until now there is little information about the damage inducing mechanisms and their participation in the development of the disease. The objective was to determine the imbalance of peripheral cellular immunity according to the pattern of alcohol consumption.

Materials and Patients: Cross-sectional study included 5 groups of subjects with different patterns of alcohol consumption using AUDIT and DSM-IV. G1: Control with OH consumption<10g/day (CT); G2: Risk (Ri) and G3: Abuse (A) with AUDIT>8; G4: Alcoholism, without clinical or biochemical stigmata of damage (OH); G5: Patients with alcoholic liver cirrhosis (CiOH). T cells, T-CD4+, T-CD8+, B cells, NK and NKT were determined in peripheral blood by flow cytometry. For statistical analysis we performed U-Mann Whitney, considering p<0.05 significant.

Results: In the study, 589 subjects were included, average age 32±11, 30±11, 23±3, 31.5±13 and 47.5±7.7 years CT, Ri, A, OH y CiOH respectively (p<0.001). Alcohol consumption (g/day) was higher in OH 158(210,107), and CiOH 293(340,246) (p<0.001,both). Cellular determination of NK we found elevated (15.4 and 13.3vs11.1) and NKT (3.7 and 2.5vs1.7) in groups A, OH and CiOH (p<0.001), (p<0.001), (p<0.05) only in NK, during CiOH NKT decrease (1.4vs1.7)(p<0.001). In T cells we observed a decrease in OH (62.3vs66.5) and CiOH (56.8vs66.5) (p<0.001), the percentage of CD4 + cells decreased from the Ri group (35.7vs38.8)(p<0.01) until OH