over 18 years of age of both sexes, of any etiology, treated at Centro Médico Nacional 20 de Noviembre between January 2013 and June 2023, who underwent large volume paracentesis, were selected and matched 2:1 with controls who did not require high volume paracentesis, adjusted for disease severity, age, sex, and Child-Pugh stage. Exclusion criteria were pregnancy or lactation, under 18 years of age, and ascites of a different origin than chronic liver disease. The data was extracted from clinical records.

Results: A total of 226 patients were analyzed, 61.9% women (n=140) and 38.1% men (n=86). The average age was 64.28 years (SD=13.33). The minimum age was 19 years and maximum was 91 years. The most frequent etiology was hepatic steatosis in 34.07% (n=77), followed by hepatitis C in 19.91% (n=45), alcoholism in 12.38% (n=28), autoimmune hepatitis in 10.17% (n=23). The distribution of patients by Child-Pugh classification was B in 69% (n=156) and C in 31% (n=70). The average MELD-NA score was 16.93 (SD=7.10). The main comorbidities were 36.7% (n=83) type 2 diabetes mellitus, 24.8% (n=56) systemic arterial hypertension, 15% (n=34) chronic kidney disease, and 16.4% (n=37) obesity.

Out the 226 patients with liver cirrhosis with ascites, 33.2% (n=75) underwent large volume paracentesis while 66.8% (n=151) underwent paracentesis less than 5 liters. The mortality of patients undergoing large volume paracentesis was 32% compared to 20.5% RR 1.55, IC 95% (0.98-2.45) of patients who did not. In bivariate analysis by sex, there were no statistically significant differences in mortality. Stratified analysis by nutritional status with body mass index did not show differences in mortality in patients undergoing large volume paracentesis.

Conclusions: No statistically significant differences in mortality were observed between patients undergoing large volume paracentesis and those who did not. It is important to consider that factors other than paracentesis volume may influence patient survival.

Ethical statement: This study adheres to ethical principles in clinical research involving human subjects and presents no risk to the population under investigation as only information obtained from clinical records will be evaluated.

Declaration of interests: None.

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Table 1
Demographic Characteristics

	LARGE VOLUME PARACENTESIS (n = 75)	NO LARGE VOLUME PARACENTESIS (n = 151)	P VALUE
AGE, MEAN	63.15	64.85	0.35
FEMALE (%)	41 (54.66)	99 (65.56)	0.11
MALE (%)	34 (45.33)	52 (33.77)	0.11
CHILD-PUGH (%)			
B	44 (58.66)	112 (74.17)	0.01
C	31 (41.33)	39 (25.82)	
MELD-NA, MEAN	19.92	15.45	0.00
HEPATOPATHY ETIOLOGY			
HEPATIC STEATOSIS (%)	24 (32)	53 (35.09)	0.04
HEPATITIS C INFECTION (%)	9 (12)	36 (23.84)	0.04
ALCOHOL (%)	11 (14.66)	17 (11.25)	0.04
HEPATITIS B INFECTION (%)	0 (0)	2 (1.32)	0.04
AUTOIMMUNE HEPATITIS (%)	9 (12)	14 (9.27)	0.04
CBP (%)	6 (8)	15 (9.93)	0.04
CEP (%)	0 (0)	2 (1.32)	0.04
IDIOPATHIC (%)	11 (14.66)	10 (6.62)	0.04
OVERLAP HAI AND CBP (%)	5 (6.66)	1 (0.66)	0.04
OVERLAP CBP AND CEP (%)	0 (0)	1 (0.66)	0.04

CBP: Chronic Biliary Pancreatitis, CEP: Chronic Extrahepatic Pancreatitis, HAI: Hepatic Acute Inflammation, MELD-Na: Model for End-Stage Liver Disease with Sodium.

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HGF decreases ANIT-induced liver damage through modulation of redox status.

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Introduction and Objectives: Intrahepatic cholestasis is the partial/total obstruction of bile flow, with inflammation and increased reactive oxygen species (ROS). Previous studies indicate that hepatocyte growth factor (HGF) generates hepatoprotective effects in alpha-naphthylisothiocyanate (ANIT)-induced cholestasis. We focused on characterizing the mechanisms of HGF-induced protection in cholestasis.

Materials and Methods: Male CD1 mice aged 8-10 weeks were randomly divided into 4 experimental groups: 1) untreated control group (NT), 2) ANIT-treated group via intragastric administration at a dose of 60 mg/kg, 3) ANIT+HGF-treated group, where HGF will be administered at a dose of 10 μg/kg intravenously 24 hours after ANIT administration, and 4) control group treated only with HGF. Mice were sacrificed at 30 h, 36 h, and 48 h post-treatment initiation for liver tissue and serum collection. The collected samples were used for biochemical assays, Western Blot, TBARS, and H&E staining.

Results: The histological results suggest that HGF can reverse the cholestatic damage observed in time-independent H&E stains, which impacts the architecture of the liver parenchyma, through the decrease in inflammatory infiltrate corroborated with the reversal of the size of the sinusoid area. It was also observed that pyknotic nuclei decrease, which suggests a decrease in cell death as well as an increase in proliferation. These results at the cellular level also impact the decrease in markers of damage at the serum level, such as transaminases, and the decrease in liver size to normal levels. It was also observed that HGF modulates the production of ROS through decreased lipoperoxidation over time, which may be one of the main causes of its hepatoprotective effect in experimental cholestasis. That is why we evaluated the effect of N-acelticistein (NAC) as a therapeutic proposal for cholestasis. The proteomic results indicated that NAC increases the protein content of the glutathione system to decrease damage.

Conclusions: HGF regulates a hepatoprotective response by modulating ROS, which favors the reduction of tissue damage reduction and the antioxidant response through the glutathione system. On the other hand, NAC could be suggested as a therapeutic option in cholestatic disease.

Ethics statement: The care and management of the experimental subjects were carried out following the ethical guidelines established by the Universidad Autónoma Metropolitana (UAM) and the National

Institutes of Health of the United States (NIH) and in accordance with the Official Mexican Standard (NOM-062 -ZOO-1999).

Declaration of interests: None.

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Simplification of the diagnostic approach and treatment of Hepatitis C Virus.

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Introduction and Objectives: Twenty-five years after the discovery of the hepatitis C virus (HCV), it is the chronic infection with the greatest impact on diagnosis and treatment. The objective of this study is to describe the simplification of HCV management for a cohort of 270 patients evaluated from 2018 to 2023.

Materials and Patients: A prospective cohort study was carried out to evaluate patients with HCV (Ac vs HCV +). In 2018 we had direct-acting antivirals (DAA) to treat HCV genotype 1; in addition to the HCV viral load (HCV RT-PCR), we required the viral genotype and liver elastography, 2019 we already had pan-fibrotic pangenotypic schemes, so genotype and liver elastography were excluded; however, due to the capacity of HCV to infect lymphocytes, screening prior to treatment with DAAs for diabetes, kidney disease, thyroid disease, rheumatic musculoskeletal disease, and associated proliferative disorders continues to be necessary. To B lymphocytes and in patients with cirrhosis determination of alpha fetoprotein (AFP) and liver ultrasound. In the clinical presence of cutaneous purpura, determination of cryoglobulins, rheumatoid factor and complement fractions, in addition to excluding coinfections with the Hepatitis B Virus and the human immunodeficiency virus. In the initial assessment, the risk factors for HCV were obtained by questioning. Patients who received treatment were evaluated every month during the months of treatment and the sustained viral response 12 weeks after completing treatment (SVR12) and every 6 months thereafter.

Results: 269 patients with chronic HCV infection were included, sent from 11 first-level medical units and 3 second-level hospitals in Northeast Mexico. 53% were women with an average age of 54 years. The main risk factor identified was blood transfusion followed by intravenous drug use (IVDU). 28% had previous treatment with pegylated interferon and ribavirin. 30% had compensated cirrhosis. Fibrosis was calculated using the APRI algorithm, finding 53/130 with >1.5 and 60/130 with >3.25, which predicted F3-4. Liver elastography was performed in 55/130 patients, with 37 at F3-4. Among the diseases possibly related to chronic HCV infection we found 29 diabetes, 21 hypothyroidism, 9 cutaneous vasculitis with cryoglobulins, 1 diffuse large cell non-Hodgkin lymphoma, 1 monoclonal gammopathy of uncertain origin, 1 chronic lymphocytic leukemia and 3 cases of hepatocellular carcinoma., a patient with HCV relapse in a transplanted liver. Of these, 155 (58%) presented positive HCV RT-PCR with genotype 1 in 80% of the patients. 130 (84%) received treatment, the most used regimens were those based on sofosbuvir with SVR12 in 97% (Table 1).

Conclusions: The diagnostic approach and treatment of chronic HCV infection has been simplified with the rapid test for detection and mainly due to the safety of the new treatments, DAAs, since these have proven to be safe and highly effective in the heterogeneous population that suffers from this infection.

Ethical statement: Approval was obtained from the ethics and research committee of our hospital.

Declaration of interests: None.

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Table 1
Baseline characteristics of patients included

N=269	
Males/Females %Ages, years, mean, (range)	128/141 (48/52) 54 (15-85)
Risk Factors, n (%)	
Transfusion of blood and derivatives	58 (45)
IVDU	33 (25)
Promiscuity	15 (11.5)
Punctures, contaminated surgical material	4 (3)
Tattoos	3 (2.5)
Unidentified	17 (13)
Viral Factors	
Positive Viral load	155
Negative viral load	114
Genotype n (%)	122 (79)
1	98*(80)
1a	58
1b	33
2	16 (13)
3	8 (6.5)
4	1
Others	Two coinfections: 1a + 2, 1a + 3
Stage of liver disease, n (%)	
APRI/ FIB-4 F0-1	80 (61.5)
APRI/ FIB4 F3-4	50 (38.5)
Child Turcotte Pugh A	118 (91)
Child Turcotte Pugh B	9 (7)
Child Turcotte Pugh C	3 (2)
HCV Syndrome, n (%)	
Diabetes	29 (22)
Hyphotiroidism	21 (17)
Cutaneous vasculitis	9 (7)
Other proliferative disorders	3 (2)
Liver carcinoma	3 (2)
Comorbidities n (%)	
Obesity	34 (26)
Hypertension	32 (25)
Coinfection with HIV	10 (8)
Previous therapy n (%)	
Naïve	94 (72)
Experienced	36 (28)
DAA Regimens, n (%)	
Sofosbuvir/Ledipasvir, 12 weeks	9 (7)
Sofosbuvir/Ledipasvir + ribavirin, 12 weeks	1 (0.7)
Ombitasvir, dasabuvir, paritaprevir + ritonavir, 12 weeks	9 (7)
Ombitasvir, dasabuvir, paritaprevir + ritonavir + ribavirin, 12 weeks	4 (3)
Sofosbuvir/Velpatasvir, 12 weeks	91 (70)
Sofosbuvir/Velpatasvir + ribavirin 12 weeks	2 (1.5)
Glecaprevir/Pibrentasvir 8-12 weeks	14 (11)
Virological response (%)	
RVS12	97

APRI: Aspartate Aminotransferase to Platelet Ratio Index, FIB-4: Fibrosis-4 Index, HCV: Hepatitis C Virus, DAA: Direct-Acting Antiviral, RVS12: Sustained Virological Response at 12 weeks, IVDU: Intravenous Drug Use.

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