World Journal of Hepatology

Submit a Manuscript: https://www.f6publishing.com

World J Hepatol 2025 November 27; 17(11): 112359

DOI: 10.4254/wjh.v17.i11.112359

ISSN 1948-5182 (online)

EDITORIAL

Impact of alcohol-associated and metabolic dysfunction-associated steatotic liver diseases upon hepatic disorder and carcinogenesis in the current era

Tomohide Hori

Specialty type: Gastroenterology and hepatology

Provenance and peer review: Invited article; Externally peer reviewed.

Peer-review model: Single blind

Peer-review report's classification
Scientific Quality: Grade C
Novelty: Grade C
Creativity or Innovation: Grade C
Scientific Significance: Grade C

P-Reviewer: Men CJ, MD, PhD, China

Received: July 24, 2025
Revised: August 18, 2025
Accepted: November 4, 2025
Published online: November 27, 2025
Processing time: 125 Days and 20.6 Hours

Tomohide Hori, Department of Hepatobiliary Pancreatic Surgery, Yokkaichi Hadu Medical Center, Yokkaichi 510-0016, Mie, Japan

Corresponding author: Tomohide Hori, MD, PhD, Professor, Department of Hepatobiliary Pancreatic Surgery, Yokkaichi Hadu Medical Center, 10-8 Haduyama-cho, Yokkaichi 510-0016, Mie, Japan. tomohidehori@yahoo.co.jp

Abstract

In this editorial, author specifically focuses upon metabolic dysfunction-associated steatotic liver disease (MASLD) and alcohol-associated liver diseases (ALD) in the current era. This editorial article is inspired by the observational study by Harris *et al* in the recent issue. Alcohol and metabolic dysfunction cause steatotic changes in the hepatic parenchyma. The ALD and MASLD are major cause of chronic liver disease. Liver cirrhosis (LC) is a result of chronic liver inflammation with many causes (*e.g.*, viral hepatitis, drug, alcohol and metabolic disorder). Metabolic dysfunction-associated steatohepatitis and alcohol-associated hepatitis can lead to liver fibrosis and LC. LC leads to hepatic dysfunction and can progress to eventual liver failure and death. Though chronic viral hepatitis is considered a main cause of LC for a long time, other etiologies (*i.e.*, ALD, MASLD) has significantly increased in the current era. From the viewpoint of carcinogenesis, LC frequently causes hepatocellular carcinoma (HCC), and HCC is the most common type of primary liver cancer worldwide. As regards major causes of HCC, chronic viral hepatitis is gradually outweighed by ALD and MASLD. Note that patients coexisting with ALD and metabolic dysfunction-associated steatohepatitis show higher occurrence of HCC. Impact of ALD and MASLD upon the development of chronic liver disease, liver fibrosis, LC, and HCC is drastically increased in the current era. Establishments of diagnostic and therapeutic strategies to overcome these hepatic disorders are still required.

Key Words: Alcohol-associated hepatitis; Alcohol-associated liver diseases; Cirrhosis; Hepatocellular carcinoma; Metabolic dysfunction-associated steatohepatitis; Metabolic dysfunction-associated steatotic liver disease

Core Tip: Alcohol and metabolic dysfunction cause steatotic changes in the hepatic parenchyma, and metabolic dysfunction-associated steatotic liver disease (MASLD) and alcohol-associated liver diseases (ALD) are currently recognized as main steatotic liver disease. The ALD and MASLD result in chronic liver disease, liver fibrosis and subsequent cirrhosis. Patients accompanied with ALD and/or MASLD are currently increasing worldwide, and therefore, this editorial focuses an impact of ALD and MASLD upon hepatic disorder and carcinogenesis. From the viewpoint of carcinogenesis (*i.e.*, hepatocellular carcinoma), chronic viral hepatitis is gradually outweighed by ALD and MASLD. Also, this editorial article mentions differences in gender and geographical region, and touches upon clinical implication for these important liver diseases.

Citation: Hori T. Impact of alcohol-associated and metabolic dysfunction-associated steatotic liver diseases upon hepatic disorder and carcinogenesis in the current era. *World J Hepatol* 2025; 17(11): 112359
URL: https://www.wjgnet.com/1948-5182/full/v17/i11/112359.htm
DOI: https://dx.doi.org/10.4254/wjh.v17.i11.112359

INTRODUCTION

Viral hepatitis transmission, direct hepatotoxicity and metabolic disorders were recognized as major causes for chronic liver disease, liver fibrosis and liver cirrhosis (LC)[1-3]. Especially in the current era, drug-induced liver injury, metabolic dysfunction-associated steatotic liver disease (MASLD) and alcohol-associated liver diseases (ALD) have significantly increased[1,2,4]. Currently, drug-induced liver injury has concern within the medical community, pharmaceutical industries and drug regulatory agencies[5]. Drug-induced liver injury is a major cause for acute liver failure, and an associated mortality rate of 60%-80% has been documented[5,6]. The ALD becomes a global concern from the viewpoint of healthcare[7]. There is increasing incidence of ALD in females[7], and the female cohort were a younger median age (45 years old)[7]. Also, there has been a significant global increase in the prevalence of MASLD[8,9]. This increase is largely driven by the worldwide increase in patients accompanied with diabetes mellitus, obesity and other metabolic disorders[8]. MASLD patients were older (50.63 ± 0.72 years old) and more likely to be female, with higher prevalence of diabetes mellitus (19.38%)[8]. Advanced portal hypertension accompanied with refractory symptoms will develop due to LC[10], and moreover, LC may result in appearance of hepatocellular carcinoma (HCC)[1,4,11]. LC is therefore considered as a major cause of morbidity and mortality[3]. Both alcohol abuse and chronic viral infections traditionally have been considered as major causes of HCC, but they are now being gradually changed by metabolic disorders (*e.g.*, diabetes mellitus, obesity and MASLD)[12,13]. Note that disease progression of metabolic dysfunction-associated fatty liver disease is strongly modified by alcoholic consumption[14].

In this editorial article, author specifically focuses upon ALD and MASLD in the current era. The ALD and MASLD are currently recognized as two main types of steatotic liver disease[12,13]. Wide spectrum of several different hepatic disorders (*i.e.*, simple steatosis, steatohepatitis, fibrosis, LC and HCC) are characterized by these important liver diseases [12,13]. This editorial article is inspired by the observational study by Harris *et al*[7] in the recent issue. Actually, ALD and MASLD are major cause of chronic liver disease and LC[2,12,13,15,16]. Metabolic dysfunction-associated steatohepatitis (MASH) and alcohol-associated hepatitis (AH) can lead to liver fibrosis and LC[2,4,17]. From the viewpoint of carcinogenesis, the impact of ALD and MASH upon etiologies for HCC is increased in the current era[15,17-21], though the global burden of chronic viral hepatitis for HCC etiologies is still important but is decreased[11,22]. Note that patients coexisting with ALD and MASH show higher occurrence of HCC[4].

VIRAL HEPATITIS

Viral hepatitis transmission, direct hepatotoxicity and metabolic disorders are recognized as major causes for chronic liver disease, liver fibrosis and LC[1-3]. LC is a major cause of morbidity and mortality[3]. Chronic viral hepatitis related with hepatitis B virus (HBV) and hepatitis C virus (HCV) was considered a main cause of LC for a long time, though other etiologies (*i.e.*, ALD, MASLD and drug-induced liver injury) has significantly increased in the current era[1,2,4,11, 17]. Liver cancer is a major cause of death worldwide, and HCC occupied approximately 80% of HCCs[1,11]. Although chronic viral hepatitis accounted for approximately 70%-80% of all HCC cases worldwide[1,11,20], this hepatitis globally represents approximately 30%-40% of primary liver cancer deaths[11,20]. Chronic viral hepatitis and alcohol abuse have been traditionally major causes of HCC. As regards major causes of HCC, chronic viral hepatitis are now being gradually outweighed by obesity, diabetes mellitus, ALD and MASLD[12,13,23]. Western dietary patterns along with a sedentary lifestyle contributed to metabolic disease spread around the world[12,13,23]. The role of dietary habits in the modulation of HCC risk has been documented[12,13,23].

ALD

Chronic excessive alcoholic consumption causes a severe liver disorder (*i.e.*, ALD). Approximately 3.5% of the global

population are affected by this liver disorder[16]. The AH is defined as an active and aggressive form of ALD[4]. Disease progression is modified by alcohol consumption in MASLD patients[14], and ALD including AH results in steatotic liver disease[12,13]. The criteria of ALD was described as follow: (1) Daily alcohol intake greater than 40 g in men and 20 g in women for over 5 years; and (2) Clinical status with liver disease, supported by blood examination. The prevalence of ALD and ALD-related HCC has been dramatically increased worldwide[4,7,11,18,19]. Incidence of ALD is currently increasing, especially in women[7]. HCC development in ALD patients can be accurately predicted by the easy-to-use model[18]. Individualized and precise treatment strategy is required for clinicians in hepatology[16,18].

The spectrum of ALD encompasses simple steatosis, AH, liver fibrosis, LC, and a potential of HCC development (*i.e.*, carcinogenesis)[4,16,18,19]. Recently, the incidence of ALD among females is increasing in United States and China[12,13]. Some studies have documented female gender as an independent predictor of ALD mortality[24,25], and have shown that female gender was associated with higher hospital readmission rates and increased the risks for acute on chronic liver failure and HCC[23,26,27].

MASLD

The MASLD is an important cause of chronic liver disease[2,9], and affects more than a quarter of the adult population worldwide[15]. The MASLD results in steatotic liver disease[12,13]. The MASH is a progressive form of MASLD[2], and can lead to fibrosis and LC[2,15]. In a word, MASLD can progress to MASH, which is associated with increased risk of progression to liver fibrosis, LC and HCC[15]. Moreover, as described above, alcohol consumption will affect disease progression in MASLD[14].

Novel concepts for therapeutic strategies for MASLD are directly or indirectly reduction of liver fibrosis[9,15,28]. Among these, the targeting of hepatocytes and metabolism have yielded fibrotic reduction in clinical trials for MASH[15,29]. However, these therapies reduce liver fibrosis only in a subset of MASLD patients, and have not yet shown benefits beyond advanced fibrotic stage[15,29]. Direct antifibrotics and macrophage-based therapies may be more suitable for advanced stages of MASH[15,29]. The arsenal of therapies for MASLD is rapidly expanding and includes macrophage transplantation, hepatocyte-specific oligonucleotides, as well as chimeric antigen receptor T cell-based therapies[15,29]. Integrating these novel therapeutic concepts into stage-specific and/or combination therapies targeting divergent pathogenic mechanisms and cell types is the focus of ongoing researches[15,28,29]. MASH patients need adequate treatments which will accomplish fibrotic reduction at high rate[15,28,29].

The extensive scientific efforts of recent decades to gain a better understanding of MASLD have led to a more solid evidence base and more detailed clinical recommendations that give clinicians more certainty in managing MASLD patients. Current status applies to the early detection and risk assessment of the MASLD with or without the liver needle biopsy and monitoring of liver disease. Optimal treatment for MASLD patients requires interdisciplinary and interprofessional co-operation[14]. In a word, outcomes of healthcare team should be more enhanced[9]. The MASLD is a prevalent liver condition linked to metabolic risk factors (*e.g.*, obesity, diabetes mellitus and dyslipidemia), and can progress to steatohepatitis (*i.e.*, MASH), fibrosis, cirrhosis, or HCC[9]. Early detection, risk stratification, and coordinated care are essential to prevent complications and improve long-term outcomes[9]. The growing prevalence of MASLD, primarily driven by rising obesity rates and sedentary lifestyles, calls for a coordinated, interprofessional approach to patient care [9]. In particular, the first specific drug authorization gives MASLD patients hope that clinicians will have a broad spectrum of specific therapeutic measures in the coming years[14]. Nevertheless, further efforts should be made to establish effective prevention programs and achieve a structural improvement in the care of complex and diverse group of affected individuals.

CARCINOGENESIS

HCC is the most common type of primary liver cancer, accounting for approximately 80% of cases around the world[1,11]. The HBV and HCV account for approximately 70%-80% of all HCC cases worldwide[1,11,20], with variation across geographic regions[11]. From the viewpoint of carcinogenesis, the global burden of viral hepatitis for HCC etiologies is decreased[11,22]. Contrastingly, the impact of ALD and MASLD upon etiologies for HCC is increased in the current era[4,7,9,11-13].

A total of 124 countries have developed national plans for eradication of viral hepatitis, and several countries have already made substantial progress for validation of viral elimination[22]. However, in the current era, the burden of other etiologies for HCC is increasing[4,11,17-19]. Chronic hepatitis related with HBV and HCV accounted for approximately 40% and 30% of global primary liver cancer deaths, respectively, followed by ALD at approximately 20% and MASLD at approximately 10%[4,11]. There is an important fact, chronic hepatitis due to HCV was associated with the lowest increase in attributable incidence of liver cancer cases, while MASH showed the fastest increase in attributable incidence of liver cancer cases and cancer deaths, respectively[11,20,21].

The ALD including AH poses a significant risk for HCC, comprising various liver conditions from steatosis to LC[16,19]. Despite accounting for a third of global HCC cases and deaths, HCCs in ALD patients lack characterization compared to HCCs in patients with viral infection[19]. Proposed mechanisms for HCCs in ALD patients include acetaldehyde toxicity, increased reactive oxygen species, and inflammation[19]. Despite advances in screening and management, HCCs in ALD patients often present at advanced stages, limiting treatment options and survival[19]. Blood examination for tumor marker (*i.e.*, alpha-fetoprotein and prothrombin induced by vitamin K antagonist-II) is recommended for

individuals with MASLD patients accompanied with LC[9,14,15,30,31].

The ALD and MASLD accompanied with steatotic changes in the hepatic parenchyma[12,13]. Chronic viral infection remains a prominent cause of HCC despite MASLD being the etiology with the highest growth rate worldwide[21]. Also, HCCs drastically increased in ALD patients[4,18,19]. The adoption of biomarkers[31] or new technologies (*e.g.*, artificial intelligence)[18] should be considered while keeping the future in mind. Patients coexisting with ALD and MASH show higher occurrence of HCC[4,9]. Although excessive alcohol consumption is presumed to be one of the most important risk factors for HCC development, alcohol, and nonalcohol metabolic risk factors act synergistically on the severity of liver fibrosis and high mortality of HCC[4,17].

Multifactorial mechanisms for HCC development in MASLD patients (*e.g.*, insulin resistance and oxidative stress) are previously documented[14,23,32,33]. Alcohol consumption an important modifier of disease progression in MASLD[14], and therefore, a coexistence with ALD and MASH results in a higher occurrence of HCC[4,9].

GENDER AND REGION

National viral hepatitis plans have been globally developed, and several countries have already made substantial progress and requested guidance from World Health Organization on criteria and processes for validation of viral elimination[22]. However, HBV and HCV account for approximately 70%-80% of all HCC cases worldwide, with variation across geographic regions[11,21]. On the other hand, the global burden of drug-induced LC has increased significantly, with notable disparities across regions and genders[1].

The prevalence of ALD is critical problem[7,11]. Females develop ALD even with less er alcoholic intake than males because of a variety of proposed mechanisms[34,35]. The increased sensitivity of women to ALD is well-established in previous studies which have investigated potential mechanisms for this finding. There is increasing incidence of ALD in females[7]. Moreover, females with AH had higher incidence of urinary tract infection, sepsis, and norepinephrine administration on univariate analysis[7]. Sepsis and mechanical ventilation were independent predictors of mortality in female ALD patients[7].

Polypharmacy is common among MASLD patients[36]. Those with LC are most affected, and women with LC are at an even greater risk[36]. Opioid, benzodiazepine, and nonsteroidal anti-inflammatory drug use is prevalent among MASLD patients with LC despite the known associated risks[36]. Frequent medication use for chronic pain in MASLD patients with LC highlights the need for management plans that optimize safety balanced against therapeutic efficacy[15,28,36]. Even the less exposure will develop ALD in woman, and several possible explanations are previously documented mechanisms[7,37-40].

CLINICAL IMPLICATIONS

LC is a result of chronic liver inflammation with many causes (*e.g.*, viral hepatitis, drug, alcohol and metabolic disorder) [1-3]. LC patients suffer from intractable symptoms (*e.g.*, ascites, encephalopathy, or variceal bleeding)[10]. LC leads to hepatic dysfunction and can progress to eventual liver failure and death[1-3].

End-stage liver diseases in ALD patients requires emergent treatment including liver transplantation[16,41]. ALD patients are the second most common population for liver transplantation, alcoholic relapse after liver transplantation will spoil their outcomes[41]. It is crucial that the risk in each ALD patients should be strictly and accurately predicted at preoperative state, and useful psychosocial evaluation criteria in order to assess the risk of alcohol use after liver transplantation has been already established[41].

The incidence and burden of MASH are increased, while diagnosis and management challenge persist[2,29]. The pathogenesis of MASLD is complicated[15,28]. Hence, fibrotic regression is a key therapeutic goal for MASH patients[15, 28,29]. Expert consensus on diagnostic strategies and treatment modalities in MASLD including MASH is required[2,29].

The MASLD represents a significant evolution in the terminology and conceptual framework surrounding hepatic steatosis[9]. Historically, liver fat accumulation without significant alcohol intake was categorized under nonalcoholic fatty liver disease[9]. However, recent shifts in nomenclature arose primarily to address two core issues. Firstly, the term 'fatty' was replaced with 'steatotic' to mitigate the stigma associated with the former term. Secondly, defining a disease by exclusion (*e.g.*, 'nonalcoholic')[9]. Mildly elevated serum aminotransferases are the primary abnormality in MASLD[9]. Several clinical findings are considered as indicative markers for hepatic synthetic dysfunction in MASLD patients[9]. Noninvasive assessment of hepatic fibrosis, serum-based indices and imaging-based elastography (including magnetic resonance image) are recommended for screening of MASLD[9]. Liver needle biopsy remains indispensable for the definitive diagnosis of MASH[9].

CONCLUSION

Alcohol and metabolic dysfunction cause steatotic changes in the hepatic parenchyma. Impact of ALD and MASLD upon the development of chronic liver disease, liver fibrosis, LC, and HCC is drastically increased in the current era. Females easily develop ALD than males, and possible causes of HCC development in MASLD patients are multifactorial. Establishments of diagnostic and therapeutic strategies to overcome these hepatic disorders including AH and MASH are

still required. The role of dietary habits is crucial for risks for liver diseases and HCC, and this point has a therapeutic potential for ALD and MASLD patients.

FOOTNOTES

Author contributions: Hori T designed the overall concept and outline of the manuscript, wrote and edited the manuscript, and reviewed the relevant literatures.

Conflict-of-interest statement: The author declare that has no conflict of interest.

Country of origin: Japan

ORCID number: Tomohide Hori 0000-0002-8282-4403.

S-Editor: Fan M
L-Editor: A
P-Editor: Wang CH

REFERENCES

1 **Chen C**, Zhou Y, Gu W, Gu Z, Jia P, Si L, Zhao S, Zhao H. Global burden of liver cancer attributable to drug use: trends from 1990 to 2021 and projections to 2040. *Discov Oncol* 2025; **16**: 1384 [*RCA*] [PMID: 40690083 DOI: 10.1007/s12672-025-03174-y] [FullText] [Full Text (PDF)]

2 **Kim Y**, Rydqvist P, Ramezani T, Haas JS, Bantel H, Buggisch P, Geier A, Hofmann WP, Mauss S, Roeb E, Schattenberg JM, Simon KG, Stefan N, Deterding K, Wiegand J, Pathil A, Tacke F. Metabolic Dysfunction-Associated Steatohepatitis Diagnosis and Management in Germany: Insights From an Expert Consensus Panel. *Liver Int* 2025; **45**: e70225 [*RCA*] [PMID: 40668568 DOI: 10.1111/liv.70225] [FullText] [Full Text(PDF)]

3 **Ching C**, Wiebe N, Zhu J. Prevalence and Predicting Factors of Caregiver Burden in Cirrhotic Patients. *Can Liver J* 2025; **8**: 355-367 [*RCA*] [PMID: 40677994 DOI: 10.3138/canlivj-2025-0013] [FullText] [Full Text(PDF)]

4 **Fu Y**, Maccioni L, Wang XW, Greten TF, Gao B. Alcohol-associated liver cancer. *Hepatology* 2024; **80**: 1462-1479 [*RCA*] [PMID: 38607725 DOI: 10.1097/HEP.0000000000000890] [FullText]

5 **Moreno-Torres M**, López-Pascual E, Rapisarda A, Quintás G, Drees A, Steffensen IL, Luechtefeld T, Serrano-Candelas E, de Lomana MG, Gadaleta D, Dirven H, Vinken M, Jover R. Novel clinical phenotypes, drug categorization, and outcome prediction in drug-induced cholestasis: Analysis of a database of 432 patients developed by literature review and machine learning support. *Biomed Pharmacother* 2024; **174**: 116530 [*RCA*] [PMID: 38574623 DOI: 10.1016/j.biopha.2024.116530] [FullText]

6 **Moreno-Torres M**, Quintás G, Castell JV. The Potential Role of Metabolomics in Drug-Induced Liver Injury (DILI) Assessment. *Metabolites* 2022; **12**: 564 [*RCA*] [PMID: 35736496 DOI: 10.3390/metabo12060564] [FullText] [Full Text(PDF)]

7 **Harris E**, Rhudy C, Roy L, Cloud A, Leyson CD. Sex differences in severity, outcomes, and healthcare utilization in alcohol-associated hepatitis. *World J Hepatol* 2025; **17**: 108063 [*RCA*] [PMID: 40901601 DOI: 10.4254/wjh.v17.i8.108063] [FullText] [Full Text(PDF)]

8 **Liang J**, Xu H, Li X, Qiao X, Ding H. Demographic and clinical characteristics of MASLD and Met-ALD patients: Insights from NHANES 2017-2020. *Ann Hepatol* 2025; **30**: 101944 [*RCA*] [PMID: 40818589 DOI: 10.1016/j.aohep.2025.101944] [FullText]

9 **Girish V**, John S. Metabolic Dysfunction-Associated Steatotic Liver Disease (MASLD). In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2025 Jan [PMID: 31082077] [FullText]

10 **Armstrong P**, Moriarty A, Dillon A, Galvin Z, Russell J, Stewart S. Transient elastography can stratify patients with Child Pugh A cirrhosis according to long-term risk of decompensation: a longitudinal cohort study. *Eur J Gastroenterol Hepatol* 2025 [*RCA*] [PMID: 40690327 DOI: 10.1097/MEG.0000000000003045] [FullText]

11 **Ivancovsky Wajcman D**, Nicolàs A, Picchio CA, van Selm L, Dusheiko G, Younossi ZM, Dillon JF, Alqahtani SA, Razavi H, Colombo MG, Kautz A, Dore GJ, Lazarus JV. Prioritising viral hepatitis elimination to prevent hepatocellular carcinoma: A public health approach for effective preventive hepatology. *JHEP Rep* 2025; **7**: 101436 [*RCA*] [PMID: 40677690 DOI: 10.1016/j.jhepr.2025.101436] [FullText] [Full Text(PDF)]

12 **Matray C**, Debras C, Chatziioannou AC, Perlemuter G, Jenab M, Voican CS. Diet Habits and Hepatocellular Carcinoma-Potential Implication for Clinical Practice. *Nutr Rev* 2025; nuaf129 [*RCA*] [PMID: 40679550 DOI: 10.1093/nutrit/nuaf129] [FullText]

13 **Qian S**, Wang X, Chen Y, Zai Q, He Y. Inflammation in Steatotic Liver Diseases: Pathogenesis and Therapeutic Targets. *Semin Liver Dis* 2024; **44**: 319-332 [*RCA*] [PMID: 38838739 DOI: 10.1055/a-2338-9261] [FullText]

14 **Horn P**, Tacke F. Key takeaways from the updated multidisciplinary European MASLD guidelines. *eGastroenterology* 2025; **3**: e100196 [*RCA*] [PMID: 40510733 DOI: 10.1136/egastro-2025-100196] [FullText] [Full Text(PDF)]

15 **Schwabe RF**, Tacke F, Sugimoto A, Friedman SL. Antifibrotic therapies for metabolic dysfunction-associated steatotic liver disease. *JHEP Rep* 2025; **7**: 101421 [*RCA*] [PMID: 40689145 DOI: 10.1016/j.jhepr.2025.101421] [FullText] [Full Text(PDF)]

16 **Zhang M**, Ji J, Song J, An C, Pei W, Fan Q, Zuo L, Wang H. Current Therapeutic Targets for Alcohol-Associated Liver Disease. *Am J Pathol* 2025; S0002-9440(25)00242 [*RCA*] [PMID: 40683560 DOI: 10.1016/j.ajpath.2025.06.009] [FullText]

17 **Huang DQ**, Singal AG, Kanwal F, Lampertico P, Buti M, Sirlin CB, Nguyen MH, Loomba R. Hepatocellular carcinoma surveillance - utilization, barriers and the impact of changing aetiology. *Nat Rev Gastroenterol Hepatol* 2023; **20**: 797-809 [*RCA*] [PMID: 37537332 DOI: 10.1038/s41575-023-00818-8] [FullText]

18 **Chang B**, Tian H, Huang A, Zhai X, Wang Q, Han L, Jin X, Gao L, Liang Q, Li B, Lu Y, Xie H, Ji D, Zou Z. Prevalence and prediction of hepatocellular carcinoma in alcohol-associated liver disease: a retrospective study of 136 571 patients with chronic liver diseases. *eGastroenterology* 2024; **2**: e100036 [*RCA*] [PMID: 39944749 DOI: 10.1136/egastro-2023-100036] [FullText] [Full Text(PDF)]

19 **Ha NB**, Yao F. Alcohol and Hepatocellular Carcinoma. *Clin Liver Dis* 2024; **28**: 633-646 [*RCA*] [PMID: 39362712 DOI: 10.1016/j.cld.2024.06.007] [FullText] [Full Text(PDF)]

20 **Rumgay H**, Ferlay J, de Martel C, Georges D, Ibrahim AS, Zheng R, Wei W, Lemmens VEPP, Soerjomataram I. Global, regional and national burden of primary liver cancer by subtype. *Eur J Cancer* 2022; **161**: 108-118 [*RCA*] [PMID: 34942552 DOI: 10.1016/j.ejca.2021.11.023] [Full Text]

21 **Kim BK**. Future Perspectives of Liver Research in the Asia-Pacific Region: Focus on Hepatitis B and C. *J Gastroenterol Hepatol* 2025; **40**: 1855-1860 [*RCA*] [PMID: 40423265 DOI: 10.1111/jgh.17028] [FullText]

22 **Easterbrook P**, Luhmann N, Newman M, Walsh N, Lesi O, Doherty M. New WHO guidance for country validation of viral hepatitis B and C elimination. *Lancet Gastroenterol Hepatol* 2021; **6**: 778-780 [*RCA*] [PMID: 34384530 DOI: 10.1016/S2468-1253(21)00267-3] [FullText]

23 **Sato-Espinoza K**, Valdivia-Herrera M, Chotiprasidhi P, Diaz-Ferrer J. Hepatocellular carcinoma in patients without cirrhosis. *World J Gastroenterol* 2025; **31**: 107100 [*RCA*] [PMID: 40575339 DOI: 10.3748/wjg.v31.i23.107100] [FullText] [Full Text(PDF)]

24 **White A**, Castle IJ, Chen CM, Shirley M, Roach D, Hingson R. Converging Patterns of Alcohol Use and Related Outcomes Among Females and Males in the United States, 2002 to 2012. *Alcohol Clin Exp Res* 2015; **39**: 1712-1726 [*RCA*] [PMID: 26331879 DOI: 10.1111/acer.12815] [FullText]

25 **Gratacós-Gines J**, Rodríguez M, Giráldez-Gallego A, Cabezas J, Vázquez IF, Cots MV, Horta D, Sánchez-Delgado J, Acosta-López S, Varasa TA, Marti-Aguado D, Monterde VB, Martin-Mateos R, Clemente A, Tejedor-Tejada J, Bandera JP, Sala M, Badia-Aranda E, Sancho VA, Tomé S, Fernández-Rodríguez C, Caballería J, Pose E. Impact of sex and recurrence in the prognosis of alcoholic hepatitis. *J Hepatol* 2022; **77**: S127 [DOI: 10.1016/s0168-8278(22)00638-9] [FullText]

26 **Pang JX**, Ross E, Borman MA, Zimmer S, Kaplan GG, Heitman SJ, Swain MG, Burak K, Quan H, Myers RP. Risk factors for mortality in patients with alcoholic hepatitis and assessment of prognostic models: A population-based study. *Can J Gastroenterol Hepatol* 2015; **29**: 131-138 [*RCA*] [PMID: 25855876 DOI: 10.1155/2015/814827] [FullText]

27 **Adejumo AC**, Cholankeril G, Iqbal U, Yoo ER, Boursiquot BC, Concepcion WC, Kim D, Ahmed A. Readmission Rates and Associated Outcomes for Alcoholic Hepatitis: A Nationwide Cohort Study. *Dig Dis Sci* 2020; **65**: 990-1002 [*RCA*] [PMID: 31372912 DOI: 10.1007/s10620-019-05759-4] [FullText]

28 **Israelsen M**, Francque S, Tsochatzis EA, Krag A. Steatotic liver disease. *Lancet* 2024; **404**: 1761-1778 [*RCA*] [PMID: 39488409 DOI: 10.1016/S0140-6736(24)01811-7] [FullText]

29 **Choe HJ**, Moon JH, Kim W, Koo BK, Cho NH. Steatotic liver disease predicts cardiovascular disease and advanced liver fibrosis: A community-dwelling cohort study with 20-year follow-up. *Metabolism* 2024; **153**: 155800 [*RCA*] [PMID: 38266957 DOI: 10.1016/j.metabol.2024.155800] [FullText]

30 **European Association for the Study of the Liver**. EASL Clinical Practice Guidelines on the management of hepatocellular carcinoma. *J Hepatol* 2025; **82**: 315-374 [*RCA*] [PMID: 39690085 DOI: 10.1016/j.jhep.2024.08.028] [FullText]

31 **Kobayashi R**, Hori T, Yamawaki M, Nakayama S, Umegae S, Iwanaga T, Nishikawa R, Shimoyama T, Suzuki S, Atsumi S, Hasegawa H, Nakashima S, Higuchi K, Onishi K, Sakaguchi R, Morita S, Miyao H, Aota S, Ohtani H, Yamamoto T. Rapid Progression of Primary Hepatic Neuroendocrine Carcinoma: A Case Report Demonstrating Drastic Oncological Behavior. *Am J Case Rep* 2025; **26**: e948500 [*RCA*] [PMID: 40853888 DOI: 10.12659/AJCR.948500] [FullText]

32 **Janku F**, Kaseb AO, Tsimberidou AM, Wolff RA, Kurzrock R. Identification of novel therapeutic targets in the PI3K/AKT/mTOR pathway in hepatocellular carcinoma using targeted next generation sequencing. *Oncotarget* 2014; **5**: 3012-3022 [*RCA*] [PMID: 24931142 DOI: 10.18632/oncotarget.1687] [FullText] [Full Text(PDF)]

33 **Kutlu O**, Kaleli HN, Ozer E. Molecular Pathogenesis of Nonalcoholic Steatohepatitis- (NASH-) Related Hepatocellular Carcinoma. *Can J Gastroenterol Hepatol* 2018; **2018**: 8543763 [*RCA*] [PMID: 30228976 DOI: 10.1155/2018/8543763] [FullText] [Full Text(PDF)]

34 **Becker U**, Deis A, Sørensen TI, Grønbaek M, Borch-Johnsen K, Müller CF, Schnohr P, Jensen G. Prediction of risk of liver disease by alcohol intake, sex, and age: a prospective population study. *Hepatology* 1996; **23**: 1025-1029 [*RCA*] [PMID: 8621128 DOI: 10.1002/hep.510230513] [FullText]

35 **Guy J**, Peters MG. Liver disease in women: the influence of gender on epidemiology, natural history, and patient outcomes. *Gastroenterol Hepatol (N Y)* 2013; **9**: 633-639 [*RCA*] [PMID: 24764777] [FullText]

36 **Neuschwander-Tetri BA**, Morris HL, Mospan AR, Yu F, Munoz B, Abraham S, Fried MW, Barritt AS 4th, Al-Sayyed L, Sanyal AJ; TARGET-NASH Investigators. The Magnitude of Polypharmacy and Role of Disease Severity and Patient Sex in Medication Use Among Patients With MASLD Enrolled in TARGET-NASH. *Aliment Pharmacol Ther* 2025; **62**: 920-938 [*RCA*] [PMID: 40665787 DOI: 10.1111/apt.70278] [FullText]

37 **Agrawal AK**, Shapiro BH. Intrinsic signals in the sexually dimorphic circulating growth hormone profiles of the rat. *Mol Cell Endocrinol* 2001; **173**: 167-181 [*RCA*] [PMID: 11223188 DOI: 10.1016/s0303-7207(00)00401-9] [FullText]

38 **Maddur H**, Shah VH. Alcohol and Liver Function in Women. *Alcohol Res* 2020; **40**: 10 [*RCA*] [PMID: 32685339 DOI: 10.35946/arcr.v40.2.10] [FullText] [Full Text(PDF)]

39 **Frezza M**, di Padova C, Pozzato G, Terpin M, Baraona E, Lieber CS. High blood alcohol levels in women. The role of decreased gastric alcohol dehydrogenase activity and first-pass metabolism. *N Engl J Med* 1990; **322**: 95-99 [*RCA*] [PMID: 2248624 DOI: 10.1056/NEJM199001113220205] [FullText]

40 **Colantoni A**, Emanuele MA, Kovacs EJ, Villa E, Van Thiel DH. Hepatic estrogen receptors and alcohol intake. *Mol Cell Endocrinol* 2002; **193**: 101-104 [*RCA*] [PMID: 12161008 DOI: 10.1016/s0303-7207(02)00102-8] [FullText]

41 **Onishi Y**, Kimura H, Hori T, Kishi S, Kamei H, Kurata N, Tsuboi C, Yamaguchi N, Takahashi M, Sunada S, Hirano M, Fujishiro H, Okada T, Ishigami M, Goto H, Ozaki N, Ogura Y. Risk of alcohol use relapse after liver transplantation for alcoholic liver disease. *World J Gastroenterol* 2017; **23**: 869-875 [*RCA*] [PMID: 28223731 DOI: 10.3748/wjg.v23.i5.869] [FullText] [Full Text(PDF)]

Published by **Baishideng Publishing Group Inc**
7041 Koll Center Parkway, Suite 160, Pleasanton, CA 94566, USA
Telephone: +1-925-3991568
E-mail: office@baishideng.com
Help Desk: https://www.f6publishing.com/helpdesk
https://www.wjgnet.com