Alcohol-related liver disease epidemiology in Victoria, Australia

Gaps remain in the cascade of care

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Image: Stacey Edwards (Taungurung/Boon Wurrung/Bunurong) 'Bunjil The Creator' 2021, acrylic on canvas.

Acknowledgement of Country

Burnet Institute board, staff and students recognise the Traditional Owners and Custodians of the land on which we live and work. We are proud to acknowledge the Boon Wurrung people of the Kulin Nations as the Traditional Owners and Custodians of the land on which our head office is located and recognise their strong and ongoing connection to Country. Recognising the ongoing impact of colonisation and intergenerational trauma on Aboriginal and Torres Strait Islander Peoples, our position as a leading medical research institute demands that we strengthen our commitment to close the gap in health outcomes.

About the artwork

Bunjil is the creator of the Kulin Nations. He takes the shape of a wedge-tailed eagle. Any guests of the Kulin Nations have to follow 2 rules: to obey the laws of Bunjil, and to not harm the children or land of Bunjil.

This artwork was created through The Torch, a not-for-profit organisation that provides art, cultural and arts industry support to First Nations people currently in, or recently released, from Victorian prisons.

Executive Summary

Alcohol is a significant cause of morbidity and mortality due to alcohol-related liver disease. In this report, we outline the epidemiology and time trends in three major contributors to alcohol-related death in Australia: alcohol-associated hepatitis, alcohol related liver cirrhosis and alcohol associated hepatocellular carcinoma. The findings underscore the need for comprehensive evidence-based alcohol control policies in Victoria, including measures that restrict alcohol availability and the advertising, sponsorship and promotion of alcohol.

Alcohol-associated hepatitis

- In Victoria, women account for one third of admissions for alcohol-associated hepatitis
- Compared to men, women with alcohol-associated hepatitis are:
  - Younger
  - Physically frail
  - Greater psychiatric comorbidity
  - More likely to require intensive care unit (ICU) admission
- During the COVID-19 pandemic, Victoria had a sharp increase in admissions for alcohol-associated hepatitis
- Among men, there has been a reduction in monthly admissions for alcohol associated hepatitis post COVID-19, but the monthly admission rate remains more than double the pre-COVID-19 monthly admissions rate
- Among women, there was a sharp increase in the monthly rate of alcohol-associated hepatitis admissions during COVID-19. Post-COVID-19, the monthly rate of admissions for AAH has stabilised, however the number of admissions remains higher post COVID-19 than pre-COVID-19.

Alcohol-associated cirrhosis

- Between 25-45% of people with alcohol-related liver disease have cirrhosis
- People with alcohol associated liver disease are less likely to have cirrhosis evaluation than people with hepatitis B, C and metabolic associated steatotic liver disease (MASLD)
- Alcohol associated liver disease has the fastest progression to cirrhosis
- 50% of people with alcohol-related liver disease (ALD) develop cirrhosis within 6 years

Alcohol-Associated Hepatocellular Carcinoma

- 38% of hepatocellular carcinoma (HCC) cases in Greater Melbourne between 2018 to 2022 had alcohol as a primary or contributing cause
- People with alcohol-associated HCC were:
  - Younger and male
  - More likely to have severe underlying liver disease
  - More likely to have advanced stage liver cancer stage at diagnosis due to low uptake of HCC surveillance
  - Less likely to receive curative treatment for HCC and had worse overall survival from HCC diagnosis

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1. Alcohol-associated hepatitis

1.1 Background

Alcohol-associated hepatitis (AAH) represents one of the most severe manifestations of alcohol-associated liver disease (ALD), characterized by rapid onset or worsening of jaundice, hepatic decompensation, and high short-term mortality(1). Despite advances in medical management, AAH continues to carry a 30-day mortality of 20-50% for severe presentations(2). In Australia and New Zealand, ALD has become the leading indication for liver transplantation among adults with end-stage liver disease(3).

The COVID-19 pandemic and associated public health measures have had profound impacts on mental health and substance use patterns worldwide(4). Multiple studies documented increases in alcohol consumption during the pandemic, driven by psychological and social stressors including social isolation, economic uncertainty, and reduced access to support services(5, 6). Data from Australia and international studies have reported surges in AAH hospitalizations during and after the pandemic(7-9). Victoria, Australia, experienced one of the world's longest lockdowns during COVID-19, with Melbourne enduring over 260 days of restrictions between March 2020 and October 2021. This unique context provides an important opportunity to examine the long-term impact of prolonged social restrictions on alcohol-related health outcomes. Preliminary single-center studies from Melbourne suggested increases in alcohol-related harms(10). However, comprehensive population-level data on AAH admission trends in Australia, particularly regarding differential effects on sex and persistence beyond the acute pandemic period, remain uncharacterized.

This study provides the first comprehensive population-level analysis of AAH admission trends in Victoria over a 14-year period from 2010 to 2024. Using the COVID-19 pandemic as a key temporal marker, we sought to: 1) characterize clinical and demographic features of patients admitted with AAH; 2) describe temporal trends in AAH admissions; 3) assess temporal trends in admission severity and mortality and 4) explore differential trends in AAH presentation between men and women.

1.2 Methods

Study design and setting

We conducted a retrospective, multi-center cohort study using two complementary data sources. Statewide data were obtained from the Victorian Admitted Episode Database (VAED), a comprehensive administrative database capturing all admissions and standardized clinical data for public and private hospitals in Victoria.

To explore individual-level data, we additionally used data from St Vincent's Hospital Melbourne, a major tertiary referral centre located in inner Melbourne, which serves a marginalized and culturally diverse local population including Aboriginal and Torres Strait Islander People and a sizeable regional population.

Study population and time period

From the VAED dataset, we included all patients with a principal diagnosis of AAH (ICD-10 code K70.1) from January 1st 2017 to June 30th 2024.

For the SVMH dataset, we applied the National Institute on Alcohol Abuse and Alcoholism (NIAAA) biochemical and clinical criteria for alcohol associated hepatitis and additionally used hospital ICD-10 admissions coding to identify AAH related admissions from January 1st 2010 to June 30th 2024 identified via hospital coding.

The NIAAA defines alcohol-associated hepatitis by acute-onset jaundice within the previous 8 weeks in the setting of heavy alcohol consumption (>40 g/day for women, >60 g/day for men) for at least 6 months

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with <60 days of abstinence, serum bilirubin >3 mg/dL, AST 50–400 U/L with AST/ALT ratio >1.5, and no alternative explanation for liver injury(11).

We defined three periods: **Pre-pandemic** (January 2017 [VAED] / January 2010 [SVHM] to 28 February 2020); **Pandemic** (1 March 2020 to 31 October 2021, corresponding to significant public health restrictions in Victoria, including six lockdown periods totaling over 260 days); and **Post-pandemic** (1 November 2021 to 30 June 2024). For univariate analyses of demographic and clinical characteristics, the pandemic and post-pandemic periods were combined into a single category.

Data sources and collection

We extracted demographic variables (age, sex, ethnicity, residential status, hospital location) and clinical variables from the VAED dataset. Comorbidities were evaluated using the Elixhauser, Charlson and Hospital Frailty indices(12). In the SVHM cohort, we additionally obtained detailed clinical parameters including alcohol consumption, presence of cirrhosis, portal hypertensive decompensation events, liver-related and psychiatric comorbidities, BMI, and severity scores (MELD-Na, Maddrey discriminant function, Glasgow Alcoholic Hepatitis Score, Lille, and Child-Pugh status). Metabolic-dysfunction associated steatotic liver disease (MASLD) was defined according to international criteria(13). Psychiatric comorbidities were identified from clinician-documented diagnoses recorded in the medical record at admission and coded from free-text entries into three non–mutually exclusive categories (mood, anxiety and trauma-related).

Primary and Secondary Outcomes

The primary outcomes were a) number of hospital admissions for AAH per month (VAED dataset) and b) number of individuals admitted with AAH per month (SVHM dataset), overall and stratified by gender.

Secondary outcomes included 1) in-hospital 30- and 90-day all-cause mortality, 2) liver-related mortality (days), length of stay (days), 3) ICU admission (yes/no), 4) 90-day readmission (yes/ no) per admission, overall and stratified by gender.

Ethics Approval

The study was approved by St Vincent's Hospital Melbourne Human Research Ethics Committee (HREC 2023/PID00192). Individual patient consent was waived for this retrospective analysis of de-identified data.

Details of the statistical analysis are provided in **Appendix 1.**

1.3 Results

Statewide Sociodemographic and Clinical Data

In the VAED cohort, 4111 admissions for AAH were identified across Victorian health services between January 2017 and June 2024 (pre-COVID n=1132; COVID/post-COVID n=2979; **Table 1.1**). The median age was 48 years (IQR 39–57) and did not differ between eras (p=0.55). Men comprised 62.5% of admissions overall (60.9% pre-COVID vs 63.1% COVID/post-COVID, p=0.19). The proportion of First Nations Australians was low, but significantly higher in the COVID/post-COVID period (2.9% vs 1.5%, p=0.011).

Comorbidity burden was high in both eras (**Table 1.1**). However, Hospital Frailty scores increased post-COVID, with a score >4 being more common in the COVID/post-COVID period (p<0.001). Clinical outcomes were similar across eras, with all-cause in-hospital mortality approximately 4% (4.5% vs 3.6%, p=0.191), median length of stay 6 days (IQR 4–11 vs 3–11, p=0.310), and overall ICU admissions were 13% (11.9% vs 13.3%, p=0.243).

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Table 1.1: Demographic and clinical data for patients with alcohol-related hepatitis across Victoria (VAED) stratified pre-pandemic and pandemic/recovery groups, 2017-2024.

	Overall (n=4111)	Pre-COVID Era (n=1132)	COVID and Post-COVID Era (n=2979)	P-value
Age (median, IQR)	48 (39 – 57)	48 (39-57)	47 (39-57)	0.6
Age group (n, %)				0.3
<35	575 (14.0%)	160 (14.1%)	415 (13.9%)	
36 – 45	1079 (26.2%)	276 (24.4%)	803 (27.0%)	
46 – 55	1240 (30.2%)	348 (30.7%)	892 (29.9%)	
56 – 65	840 (20.4%)	250 (22.1%)	590 (19.8%)	
66 – 76	306 (7.4%)	76 (6.7%)	230 (7.7%)	
>75	71 (1.7%)	22 (1.9%)	49 (1.6%)	
Gender				
M	2,569 (62.5%)	689 (60.9%)	1,880 (63.1%)	0.2
F	1,542 (37.5%)	443 (39.1%)	1,099 (36.9%)	
First Nations	103 (2.5%)	17 (1.5%)	86 (2.9%)	0.01
Obesity	17 (0.4%)	7 (0.6%)	10 (0.3%)	0.2
Comorbidities				
Elixhauser (median, IQR)	3 (3-4)	3 (3-4)	3 (3-4)	0.8
Elixhauser > 2	3,257 (79.2%)	901 (79.6%)	2,356 (79.1%)	0.7
Charleson (median, IQR)	1 (1-2)	1 (1-2)	1 (1-2)	0.6
Charleson > 2	1,718 (41.8%)	460 (40.6%)	1,258 (42.2%)	0.4
Frailty > 4	1578 (38.4%)	385 (34.1%)	1193 (40.0%)	<0.001
AUD	4,107 (99.9%)	1,128 (99.6%)	2,979 (100.0%)	<0.001
Severe liver disease	1,090 (26.5%)	306 (27.0%)	784 (26.3%)	0.6
Depression	273 (6.6%)	89 (7.9%)	184 (6.2%)	0.05
Psychosis	37 (0.9%)	10 (0.9%)	27 (0.9%)	1.0
All-cause mortality	159 (3.9%)	51 (4.5%)	108 (3.6%)	0.2
Admission duration (days)	6 (3-11)	6 (4-11)	6 (3-11)	0.3
ICU admission	531 (12.9%)	135 (11.9%)	396 (13.3%)	0.2

IQR, interquartile range; AUD, alcohol use disorder; ICU, intensive care unit.

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Time trends in statewide AAH admissions

Interrupted time series (ITS) analysis of the number of AAH admissions per month in the VAED dataset are shown in **Figure 1.1A**. Pre-pandemic, the monthly AAH admissions rate was modestly increasing by +0.15 admissions per month (95% CI 0.01–0.29; p=0.037). At the first interruption timepoint of March 2020, corresponding to implementation of COVID-19 restrictions, there was a sharp increase of +24.80 AAH admissions (βstep 95% CI 14.71–34.90; p<0.001).During the COVID-19 pandemic, the monthly AAH admission rate stabilized (βΔslope –0.11 admissions/month; 95% CI –0.90 to 0.69; p=0.80). Post-COVID-19 at the second interruption timepoint (November 2021), there was a non-significant decrease in the mean monthly AAH admissions (βstep −4.03, 95% CI −15.42 to 7.35; p=0.49) and no significant change in the monthly admission rates after reopening (βΔslope+0.12 admissions/month, 95% CI −0.73 to 0.98) compared to pre-pandemic mean monthly AAH admissions rate (p=0.78).

Sex-specific ITS models for monthly AAH admission rates in the VAED data showed divergent patterns (**Figures 1.1B-C**). For men, there was a small pre-pandemic increase in the mean number of AAH admissions per month of +0.15 admissions/month (βΔslope 95% CI−0.00 to 0.29; p=0.05). At the first interruption timepoint corresponding to implementation of COVID-19 restrictions, there was a step increase of +18.57 admissions (βstep 95% CI 10.75 to 26.38; p<0.001). During the COVID-19 restriction period, the mean monthly AAH admissions rate in men stabilised at this higher level, with no significant change in monthly trend (βΔslope −0.49 admissions/month; 95% CI −1.16 to 0.19; p=0.16). At the second interruption timepoint in November 2021, there was no significant level change (βstep +2.31 admissions; 95% CI −7.38 to 12.00; p=0.64) and no significant change in trend thereafter (βΔslope +0.41 admissions/month; 95% CI −0.30 to 1.12) compared with the pre-pandemic trajectory (p=0.26).

In contrast, women had no detectable pre-pandemic trend in AAH admissions prior to COVID-19 (βΔslope 0.003 admissions/month; 95% CI –0.11 to 0.11; p=0.97), but experienced a significant step increase in March 2020 of +6.24 admissions (95% CI 1.02–11.46; p=0.02), followed by a mean increase of 0.38 AAH admissions per month (95% CI 0.01–0.75; p=0.04) during the COVID-19 pandemic period. At November 2021, there was a significant reduction of -6.35 AAH admissions (95% CI –11.46 to –1.23; p=0.02), after which the mean number of AAH admissions in women per month returned to the pre-pandemic baseline (βΔslope −0.29 admissions/month; 95% CI −0.70 to 0.13; p=0.17).

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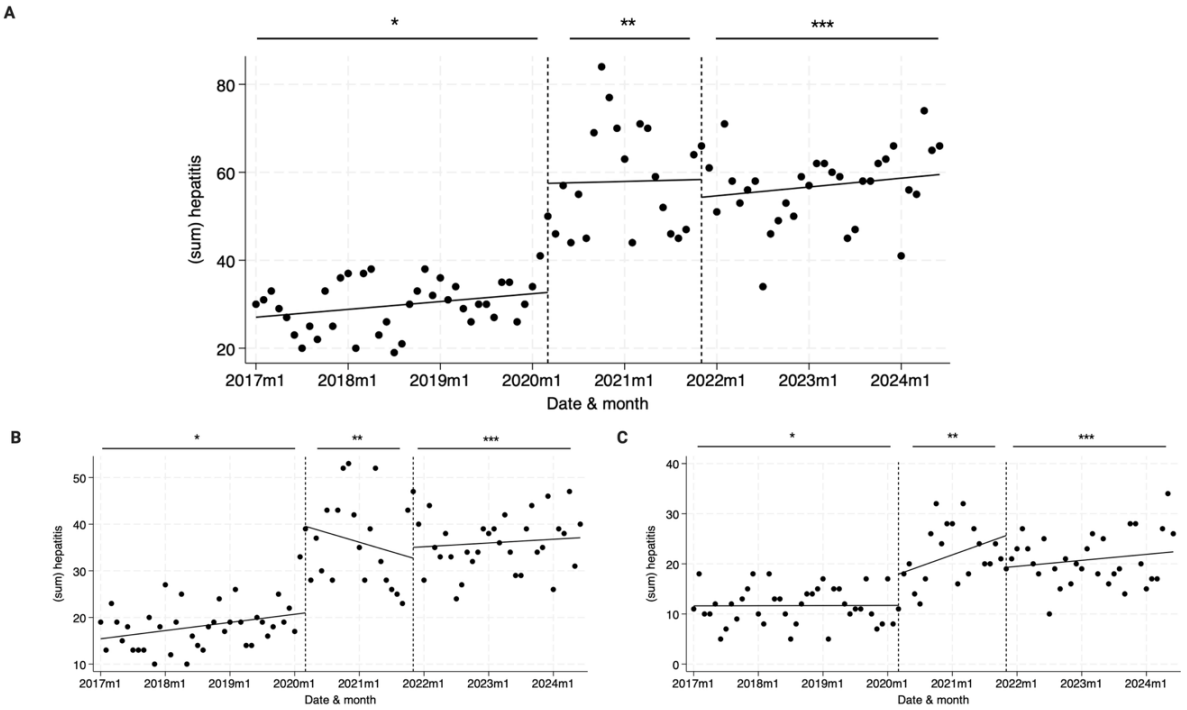


Figure 1.1. Interrupted time series analysis of mean number of alcohol-related hepatitis admissions per month across all Victorian health services (VAED) from January 2017 to June 2024. Vertical dashed lines indicate prespecified interruption timepoints: March 2020 (onset of COVID-19–related restrictions) and November 2021 (lifting of major restrictions and transition to the post-pandemic period). Solid black lines represent model-predicted values from an interrupted time series regression fitted using a generalized linear model (Gaussian family, identity link) with Newey–West standard errors. Asterisks above brackets label the segment-specific slope estimates (βslope, admissions/month) with corresponding p-values denote βslope and p-value (βslope, P-value). (A) Overall analysis (*0.2, p=0.03; **-0.1 p=0.8; *** 0.1, p=0.8); (B) Sex-specific analysis for males (*0.2, p=0.05; **-0.5 p=0.2; *** 0.4, p=0.3); (C) Sex-specific analysis for females (*0.003, p=0.09; **0.4, p=0.04; *** -0.3, p=0.3).

Patient Characteristics, Clinical and Survival Outcomes in the SVHM cohort

To understand the drivers behind the rising admission numbers, we analyzed the single-centre SVHM cohort, which comprised 340 unique individuals (pre-COVID n=147; post-COVID n=193) who accounted for 496 admissions between January 2010 and June 2024. The mean number of admissions per person was 1.45 (SD 1.01), with a median of 1.0 (range 1.0 – 7.0). Most individuals (256, 75.3%) had only one admission, while 84 individuals (24.7%) had repeat admissions.

AAH patient characteristics are shown in **Table 1.2**. The median age at index admission was

47.5 years (IQR 40.0–57.0), with a predominance of middle-aged patients (36–55 years: 203/340, 59.7%). Notably, 40/340 (11.8%) were aged <35 years. Most were male (237/340, 69.7%) and of Australian or European ethnicity (262/340, 77.1%). The proportion of people with AAH of Asian ethnicity increased from 4.8% pre-COVID to 14% post-COVID (uOR 2.97, 95% CI 1.25–7.07; p=0.014), while First Nations Australians accounted for 8.2% of

AAH admissions pre-COVID compared with 2% post-COVID (uOR 0.26, 95% CI 0.08–0.82; p=0.021). People admitted with AAH resided in areas spanning the full Socio-Economic Indexes for Areas – Index of Relative Socioeconomic Disadvantage (SEIFA IRSD) range, with 7.8% residing in suburbs within the most

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disadvantaged deciles 1–2 and 32.9% in suburbs in the least disadvantaged deciles 9–10. 16.8% of people presenting with AAH were homeless. Most people presenting with AAH lived in urban areas (85%), with no change in distribution of rural residency by era.

History of alcohol exposure was heavy and prolonged (median intake 200 g/day [IQR 100–310]; duration 20 years [IQR 10–30]), and AUD was documented in 320/340 (94.1%) of people presenting with AAH. Psychiatric comorbidity was frequent (230/340, 67.7%), with mood disorders (170/340, 50.0%) and anxiety disorders (113/340, 33.2%) the most common. Concurrent metabolic liver disease was also common (39.7% had Met-ALD).

At admission, liver disease severity was high: over half had cirrhosis (52.7%), median MELD score was 16 (IQR 11–22), median Child–Pugh score was 10 (IQR 8–12) and 55.4% (118/213) were Child-Pugh Class C. Between eras, Child–Pugh scores were modestly lower post-COVID compared with pre-COVID (median 10 [IQR 9–12] pre-COVID vs 9 [8–11] post-COVID; p=0.039). Management patterns for AAH evolved over time, with a decline in glucocorticoid use (40/147 [27.2%] to 23/193 [11.9%]; p=0.001) but an increase in addiction medicine services referrals in the post-COVID era (96/147 [65.3%] to 162/193 [83.9%]; p=0.001).

Table 1.2: Clinical characteristics for patients with alcohol-associated hepatitis at St Vincent's Hospital Melbourne, pre-pandemic and post-pandemic groups, 2010-2024.

	Overall (n = 340)	Pre-COVID (n = 147)	COVID/Post-COVID (n = 193)	uOR (median, 95% CI)	P-value
BMI (kg/m2)					
<18.5	24 (10.1%)	9 (10.0%)	15 (10.1%)	Ref	Ref
18.5 – 24.9	100 (42.1%)	43 (47.8%)	57 (38.5%)	0.8 (0.3 – 2.0)	0.6
25 – 29.9	65 (27.3%)	25 (27.8%)	40 (27.3%)	1.0 (0.4 – 2.5)	0.9
>30	49 (20.6%)	13 (14.4%)	49 (20.6%)	1.7 (0.6 – 4.7)	0.3
Alcohol					
ETOH intake (g/d)	200 (100 – 310)	168 (98 – 300)	200 (110–320)	1.0 (1.0 – 1.0)	0.6
Previous history abstinence	53 (15.5%)	29 (19.7%)	24 (12.4%)	0.6 (0.3 – 1.0)	0.07
Anti-craving medications	85 (25.0%)	42 (28.5%)	43 (22.3%)	0.7 (0.4 – 1.2)	0.2
Addiction medicine involvement	258 (75.9%)	96 (65.3%)	162 (83.9%)	2.8 (1.7 – 4.6)	**0.001**
Place of residence					
Rural	49 (15.2%)	25 (17.4%)	24 (13.5%)	0.7 (0.4-1.4)	0.3
Past history					
AUD	320 (94.1%)	133 (90.5%)	187 (96.9%)	3.3 (1.2 – 8.8)	**0.02**
Cirrhosis	213 (52.7%)	85 (57.8%)	128 (66.3%)	1.4 (0.9 – 2.2)	0.1
Psychosis	37 (0.9%)	10 (0.9%)	27 (0.9%)		1.0
Psychiatric comorbidity	230 (67.7%)	99 (67.4%)	131 (68.0%)	1.0 (0.7 – 1.6)	0.1
Mood disorders	170 (50.0%)	73 (49.7%)	97 (50.3%)	1.0 (0.7 – 1.6)	0.1

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	Overall (n = 340)	Pre-COVID (n = 147)	COVID/Post-COVID (n = 193)	uOR (median, 95% CI)	P-value
Anxiety disorders	113 (33.2%)	41 (27.9%)	72 (37.3%)	1.5 (1.0 – 2.4)	0.07
Trauma disorders	30 (8.9%)	9 (6.1%)	21 (10.9%)	1.9 (0.8 – 4.2)	0.1
Decompensation					
Ascites	135 (39.7%)	56 (38.1%)	79 (40.9%)	1.1 (0.7 – 1.8)	0.6
HE	100 (29.4%)	47 (32.0%)	53 (27.5%)	0.8 (0.5 – 1.3)	0.4
HRS-AKI	16 (4.7%)	10 (6.8%)	6 (3.1%)	0.4 (0.2 – 1.2)	0.1
HCC	10 (2.9%)	5 (3.4%)	5 (2.6%)	0.8 (0.2 – 2.7)	0.7
Variceal bleed	56 (16.5%)	20 (13.6%)	36 (18.7%)	1.5 (0.8 – 2.6)	0.2
Comorbid liver disease					
HCV	51 (15.0%)	28 (19.1%)	23 (11.9%)	0.6 (0.3 – 1.0)	0.07
HBV	9 (2.7%)	3 (2.0%)	6 (3.1%)	1.5 (0.4 – 6.3)	0.6
MASLD	135 (39.7%)	51 (34.7%)	84 (43.5%)	1.4 (0.9 – 2.3)	0.1
Prognostic scores					
Child-Pugh Score	10 (8-12)	10 (9-12)	9 (8 – 11)	0.9 (0.8 – 1.0)	**0.04**
A	28 (13.2%)	8 (9.4%)	20 (15.6%)	Ref	Ref
B	67 (31.5%)	21 (24.7%)	46 (35.9%)	0.9 (0.3 – 2.3)	0.8
C	118 (55.4%)	56 (65.9%)	62 (48.4%)	0.4 (0.2 – 1.1)	0.08
MELD	16 (11-22)	16 (11-23)	16 (11-21)	1.0 (1.0 – 1.0)	0.4
GAHS	6 (5 – 8)	6 (5 – 8)	6 (5 – 7)	0.9 (0.8 – 1.0)	0.07
Maddrey DF	24 (9 – 47)	24 (8 – 59)	24 (11 – 40)	1.0 (1.0 – 1.0)	0.1
Lille Score	0.4 (0.1 – 0.7)	0.4 (0.1 – 0.8)	0.3 (0.1 – 0.4)	0.2 (0.0 – 2.0)	0.2
Glucocorticoid therapy	64 (18.5%)	40 (27.2%)	23 (11.9%)	0.4 (0.2 – 0.6)	**0.001**
Liver-related mortality					
30 days	24 (7.0%)	14 (9.5%)	10 (5.2%)	0.5 (0.2 – 1.2)	0.1
90 days	31 (9.1%)	16 (10.9%)	15 (7.8%)	0.7 (0.3 – 1.4)	0.3
All-cause mortality					
30 days	24 (7.0%)	14 (9.5%)	10 (5.2%)	0.5 (0.2 – 1.2)	0.1
90 days	36 (10.6%)	20 (13.6%)	16 (8.3%)	0.6 (0.3 – 1.1)	0.1
Admission duration (days)	6 (3 – 11)	6 (3 – 11)	7 (4 – 12)	1.0 (1.0 – 1.0)	0.5
Intensive care admission	33 (9.7%)	15 (10.2%)	18 (18.3%)	0.9 (0.4 – 1.9)	0.8

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	Overall (n = 340)	Pre-COVID (n = 147)	COVID/Post-COVID (n = 193)	uOR (median, 95% CI)	P-value
90-day readmission	134 (39.4%)	56 (38.1%)	78 (40.4%)	1.1 (0.7 – 1.7)	0.7

**IQR, interquartile range; AUD, alcohol use disorder; CLD, chronic liver disease; HE, hepatic encephalopathy; HRS-AKI, hepatorenal syndrome acute kidney injury; HCC, hepatocellular carcinoma; HCV, hepatitis C virus; HBV, hepatitis B virus; MAFLD, metabolic associated fatty liver disease; AST, aspartate aminotransferase; ALT, alanine aminotransferase; INR, international normalised ratio; MELD-Na, Model for End-stage Liver disease including sodium; Maddrey-DF, Maddrey discriminant function.*

SVHM Single centre cohort: time trends in AAH admissions

The increase in admissions was primarily driven by a rise in the number of unique individuals presenting each year, rather than an increase in the frequency of admissions per person. A Poisson regression model showed a significant annual increase in the number of unique individuals admitted with AAH (IRR 1.11, 95% CI 1.066-1.14; p<0.001). In contrast, the increase in the number of admissions per admitted person was smaller, though remained statistically significant (IRR 1.008, 95% CI 1.002-1.014; p=0.012).

Monthly AAH admissions at SVHM increased over the study period (**Figure 1.2**). ITS analysis showed no significant pre-pandemic trend in monthly admissions (IRR per month 1.001; 95% CI 1.000 –1.006; p=0.60; Figure 2A). During the pandemic, a modest but significant increase in slope occurred (IRR 1.03 per month; 95% CI 1.00–1.06; p=0.027), and a subsequent reduction in slope post-COVID (IRR 0.97 per month relative to pre-pandemic; 95% CI 0.94–1.00; p=0.023). The modelled admission rate in in the post-COVID era remained 2.26-fold higher than the pre-COVID trend (IRR 2.26 [95% CI 1.47 – 3.47]; p<0.001), indicating a sustained elevation rather than a transient spike. Sex-specific temporal patterns were similar: in the SVHM cohort, admissions increased during the COVID-19 period in both sexes, with visually steeper increases in monthly admissions women; however, the sex–time interaction did not reach statistical significance (IRR for men vs women 0.97; 95% CI 0.93–1.01; p=0.135; Figure 2B). Absolute admission rates remained higher in men, and there was no evidence that the time trend differed by sex (p = 0.13).

Short-term outcomes and Survival analysis in the SVHM cohort

Median length of stay was 6 days (IQR 3–11), and 90-day readmission occurred in 39.4% of people with AAH, with no significant temporal variation. The 90-day all-cause mortality was 10.6%, with no evidence of a difference by era (p=0.12). Kaplan–Meier curves for 90-day survival overlapped across periods (log-rank p=0.11).

On univariable Cox analysis, higher Maddrey score was associated with increased 90-day mortality (HR 1.01 per unit, 95% CI 1.01–1.02; p<0.001). Age 40–59 years compared with under 40 years was also associated with higher mortality risk (HR 3.69, 95% CI 1.10–12.16;p=0.032). However, admission during the COVID-19/post-COVID-19 period was not associated with 90-day mortality on univariable analysis (HR 0.59, 95% CI 0.31–1.14; p=0.11). In the multivariable model (adjusted for age and sex), Maddrey score remained independently associated with increased riskof death (aHR 1.01 per unit, 95% CI 1.00–1.01; p=0.001; **Appendix 1, Supplementary Table S.1.1**).

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Sex-specific differences in AAH admission rates and presentation

Across both datasets, women with AAH presented with a distinct clinical profile compared to men. In the VAED cohort, women presenting with AAH were younger than men (median 47 [IQR 38–56] vs 48 [39–57] years; p=0.004; **Table 1.3**) and more often aged <35 years (16.0% vs 12.8%; p=0.01).Women with AAH were more frequently classified as frail (Hospital Frailty Score >4: 41.8% vs 36.4%; p<0.001). However, clinical outcomes were comparable, with similar in-hospital mortality (3.6% women vs 4.0% men; p=0.438)

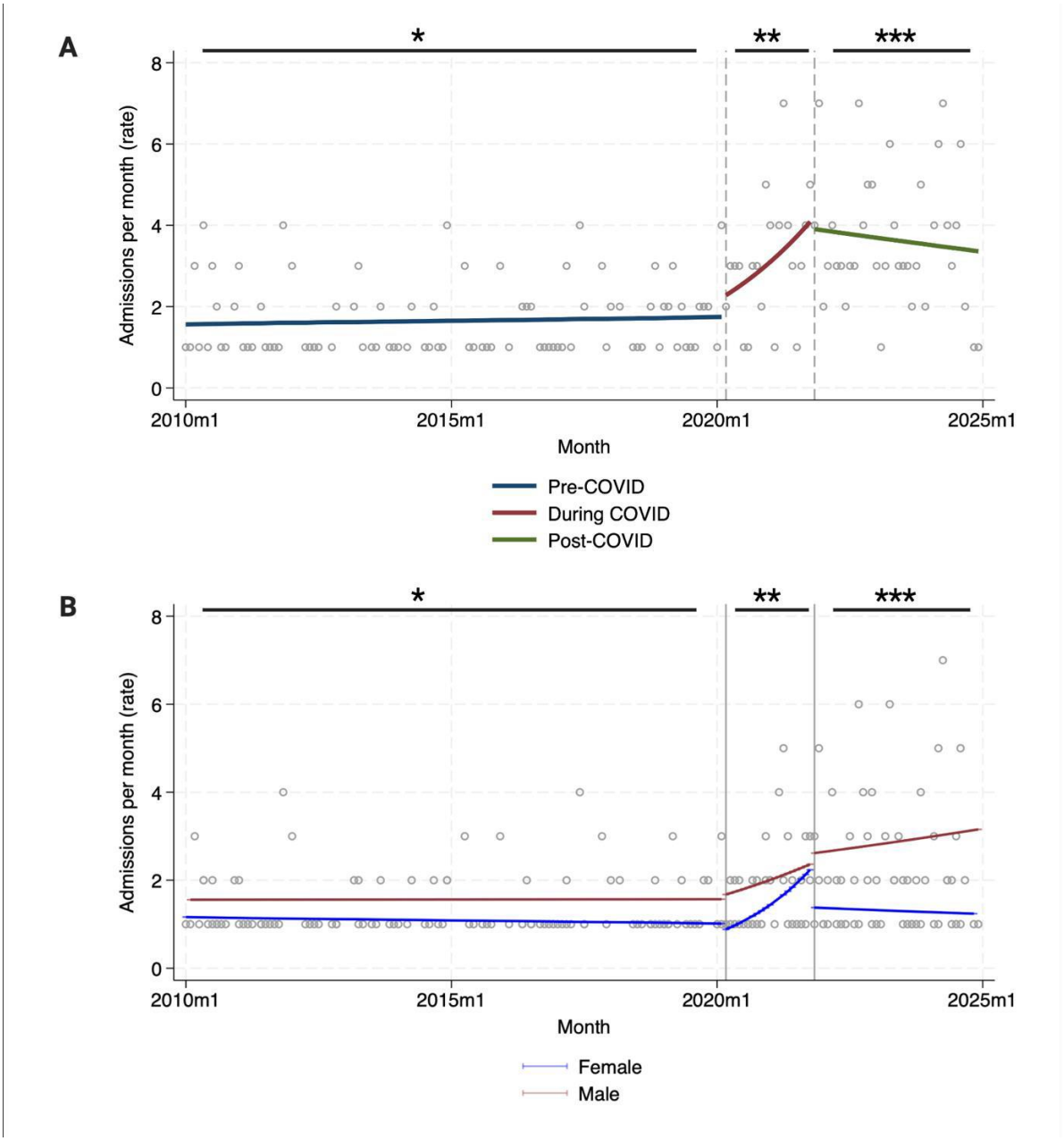


Figure 1.2: Poisson interrupted time series analysis of alcohol-associated hepatitis at St. Vincent's Hospital from January 2010 to June 2024. Vertical dashed lines indicate prespecified interruption timepoints: March 2020 (onset of COVID-19–related restrictions) and November 2021 (lifting of major restrictions and transition to the post-pandemic period). Solid lines (men, red; women, blue) represent model-predicted values from an interrupted time series poisson regression. Asterisks above brackets label the segment-specific slope estimates (IRR, admissions/month) with corresponding p-values. In

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sex-specific analysis, asterix label p-values for differences in segment-specific slope estimates. (A) Overall monthly AAH admissions (*IRR 1.0, p=0.8; **IRR 1.03, p=0.009;***IRR 01.0, p=0.04) (B) Sex-stratified analysis (*p=0.6; **p=0.1; **p=0.1).

Table 1.3: Demographic and clinical data for patients with alcohol- associated hepatitis across Victoria (VAED) stratified by sex, 2017-2024.

	Overall (n=4111)	Male (n=2569)	Female (n=1542)	P-value
Age (median, IQR)	48 (39 – 57)	48 (39-57)	47 (38-56)	0.004
Age group (n, %)				0.3
<35	575 (14.0%)	329 (12.8%)	246 (16.0%)	0.01
36 – 45	1079 (26.2%)	670 (26.1%)	409 (26.5%)	
46 – 55	1240 (30.2%)	777 (30.2%)	463 (30.0%)	
56 – 65	840 (20.4%)	546 (21.3%)	294 (19.1%)	
66 – 76	306 (7.4%)	197 (7.7%)	109 (7.1%)	
>75	71 (1.7%)	50 (1.9%)	21 (1.4%)	
First Nations	103 (2.5%)	70 (2.7%)	33 (2.1%)	0.3
Obesity	17 (0.4%)	8 (0.3%)	9 (0.6%)	0.2
Comorbidities				
Elixhauser (median, IQR)	3 (3-4)	3 (3-4)	4 (3-5)	0.08
Elixhauser > 2	3,257 (79.2%)	2015 (78.4%)	1242 (80.5%)	0.1
Charleson (median, IQR)	1 (1-2)	1 (1-2)	1 (1-2)	0.005
Charleson > 2	1,718 (41.8%)	1121 (43.6%)	597 (38.7%)	0.002
Frailty > 4	1578 (38.4%)	935 (36.4%)	643 (41.8%)	<0.001
AUD	4,107 (99.9%)	2568 (100.0%)	1539 (99.8%)	0.1
Severe liver disease	1,090 (26.5%)	674 (26.2%)	416 (27.0%)	0.6
Depression	273 (6.6%)	158 (6.2%)	115 (7.5%)	0.1
Psychosis	37 (0.9%)	28 (1.1%)	9 (0.6%)	0.1
All-cause mortality	159 (3.9%)	104 (4.0%)	55 (3.6%)	0.4
Admission duration (days)	6 (3-11)	6 (3-10)	6 (4-12)	0.003
ICU admission	531 (12.9%)	300 (11.7%)	231 (15.0%)	0.002

**IQR – interquartile range; AUD- alcohol use disorder; ICU- intensive care unit*

These findings were mirrored in the SVHM cohort (**Tables 1.4 and 1.5**), where women were also younger at presentation (median 46 vs 48 years; p=0.02). Women were also more likely to be underweight (18.3% vs 6.6%) compared to men with AAH (Table 6). In contrast to the VAED dataset, psychiatric comorbidity was more common in women than men in the SVHM cohort (80.6% vs 62.0%; OR 0.39, 95% CI 0.23–0.69; p=0.001), driven by higher rates of mood disorders (66.0% vs 43.0%; OR 0.39; p=0.0001), anxiety disorders (49.5% vs 26.1%; OR 0.36; p=0.0001), and trauma disorders (14.6% vs 6.3%; OR 0.40;

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p=0.017). Men had a higher prevalence of HBV and MASLD than women. HBV was observed in 9/237 men (3.8%) and 0/103 women (0%; p<0.001). MASLD was present in 112/237 men (47.3%) versus 23/103 women (22.3%), corresponding to higher odds in men (OR 3.10, 95% CI 1.80–5.30; p<0.001).

Overall cirrhosis prevalence and severity were similar by sex (cirrhosis 63.7% men vs 60.2% women; p=0.54). However, women were more likely to require ICU admission (15.0% vs 11.7%; p=0.002) and showed a non-significant trend toward higher 90-day readmission (45.6% vs 36.7%; OR for men vs women 0.70, 95% CI 0.43–1.10; p=0.12). 90-day mortality did not differ by sex (12.0% in men vs 9.7% in women; p=0.36.

Table 1.4: Demographic and socioeconomic data for patients with alcohol-associated hepatitis at St Vincent's Hospital Melbourne, stratified by gender, 2010-2024.

	Overall (n = 340)	Male (n = 237)	Female (n = 103)	uOR (median, 95% CI)	P-value
Age (median, IQR)	48 (40 – 57)	48 (41 – 58)	46 (39 – 55)	1.0 (1.0 – 1.1)	0.002
<35 (n, %)	40 (11.8%)	23 (9.7%)	17 (16.5%)	Ref	Ref
36 – 45	104 (30.6%)	70 (29.5%)	34 (33.0%)	1.5 (0.7 – 0.3)	0.3
46 – 55	99 (29.1%)	69 (29.1%)	30 (29.1%)	1.7 (0.8 – 0.4)	0.2
56 – 65	76 (22.4%)	58 (24.5%)	18 (17.5%)	2.3 (1.0 – 5.4)	0.004
66 – 76	16 (4.7%)	13 (5.5%)	3 (2.9%)	3.2 (0.8 – 13.0)	0.1
>75	5 (1.5%)	4 (1.7%)	1 (1.0%)	2.9 (0.3 – 28.9)	0.3
Ethnicity					
Australian/European	262 (77.1%)	175 (73.8%)	87 (84.5%)	Ref	Ref
Asian	34 (10.0%)	29 (12.2%)	5 (4.9%)	2.9 (1.1 – 7.7)	0.0
African/Middle Eastern	23 (6.8%)	16 (6.8%)	7 (6.9%)	1.1 (0.5 – 2.9)	0.8
First Nations	16 (4.7%)	13 (5.5%)	3 (2.9%)	2.0 (0.2 – 18.1)	0.5
Place of residence (urban/ regional)					
Regional	49 (15.2%)	35 (15.7%)	14 (14.1%)	1.1 (0.6-2.2)	0.720
SEIFA IRSD Decile					
1-2 (most disadvantaged)	25 (7.8%)	19 (8.5%)	6 (6.1%)	Ref	Ref
3-4	88 (27.3%)	61 (27.4%)	27 (27.3%)	0.7 (0.3 – 2.0)	0.5
5-6	44 (13.7)	31 (13.9%)	13 (13.1%)	0.8 (0.2 – 2.3)	0.6
7-8	59 (18.3%)	41 (18.4%)	18 (18.2%)	0.7 (0.2 – 2.1)	0.6
9-10 (least disadvantaged)	106 (32.9%)	71 (31.8%)	35 (35.3%)	0.6 (0.2 – 1.8)	0.4
Residence					
Community housing	42 (12.4%)	31 (13.1%)	11 (10.7%)	Ref	Ref
Home	236 (69.6%)	161 (68.2%)	75 (75.8%)	0.8 (0.4 – 1.6)	0.5

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	Overall (n = 340)	Male (n = 237)	Female (n = 103)	uOR (median, 95% CI)	P-value
Supported accommodation	11 (3.2%)	5 (2.1%)	6 (5.8%)	0.3 (0.1 – 1.2)	0.08
Homeless	50 (13.8%)	39 (16.5%)	11 (10.7%)	1.3 (0.5 – 3.3)	0.06

**IQR, interquartile range; SEIFA, Socio-Economic Indexes for Areas, IRSD: Index of Relative Socio-economic Disadvantage*

Table 1.5: Clinical characteristics for patients with alcohol-associated hepatitis at St Vincent's Hospital Melbourne, stratified by gender, 2010-2024.

	Overall (n = 340)	Male (n = 237)	Female (n = 103)	uOR (median, 95% CI)	P-value
BMI (kg/m2)					
<18.5	24 (10.1%)	11 (6.6%)	13 (18.3%)	Ref	Ref
18.5 – 24.9	100 (42.0%)	62 (37.1%)	38 (53.5%)	1.9 (0.8 – 4.7)	0.1
25 – 29.9	65 (27.3%)	52 (31.1%)	13 (18.3%)	4.7 (1.7 – 12.9)	0.003
>30	49 (20.6%)	42 (25.2%)	7 (9.9%)	7.1 (2.3 – 22.0)	0.001
Alcohol					
ETOH intake (g/d)	200 (100-310)	213 (112 – 320)	155 (80-220)	1.0 (1.0 – 1.1)	0.001
Abstinent	53 (15.6%)	43 (18.1%)	10 (9.7%)	2.0 (1.0 – 4.3)	0.05
Anti-craving medications	85 (25.0%)	55 (23.2%)	30 (29.1%)	0.7 (0.4 – 1.2)	0.2
Addiction medicine	258 (75.9%)	181 (76.4%)	77 (74.8%)	1.1 (0.6 – 1.9)	0.8
Past history					
AUD	320 (94.1%)	223 (94.1%)	97 (94.2%)	1.0 (0.4 – 2.6)	1.0
Cirrhosis	213 (62.7%)	151 (63.7%)	62 (60.2%)	1.2 (0.7 – 1.9)	0.5
Psychiatric comorbidity	230 (67.7%)	147 (62.0%)	83 (80.6%)	0.4 (0.2 – 0.7)	0.001
Mood disorders	170 (50.0%)	102 (43.0%)	68 (66.0%)	0.4 (0.2 – 0.6)	<0.001
Anxiety disorders	113 (33.2%)	62 (26.1%)	51 (49.5%)	0.4 (0.2 – 0.6)	<0.001
Trauma disorders	30 (8.9%)	15 (6.3%)	15 (14.6%)	0.4 (0.2 – 0.8)	0.02
Decompensation					
Ascites	135 (39.7%)	86 (36.3%)	49 (47.6%)	0.6 (0.4 – 1.0)	0.05
HE	100 (29.4%)	62 (26.2%)	38 (36.9%)	0.6 (0.4 – 1.0)	0.05
HRS-AKI	16 (4.7%)	11 (4.6%)	5 (4.9%)	0.9 (0.3 – 2.8)	0.9
HCC	10 (2.9%)	7 (3.0%)	3 (2.9%)	1.0 (0.3 – 3.0)	1.0
Variceal bleed	56 (16.5%)	34 (14.4%)	22 (21.4%)	0.6 (0.3 – 1.1)	0.1
Comorbid liver disease					
HCV	51 (15.0%)	43 (18.1%)	8 (7.8%)	2.6 (1.2 – 5.8)	0.02

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	Overall (n = 340)	Male (n = 237)	Female (n = 103)	uOR (median, 95% CI)	P-value
HBV	9 (2.7%)	9 (3.8%)	0 (0.0%)	Omitted	<0.001
MASLD	135 (39.7%)	112 (47.3%)	23 (22.3%)	3.1 (1.8 – 5.3)	<0.001
Prognostic scores					
Child-Pugh Score	10 (8-12)	10 (8-11)	11 (9-12)	0.9 (0.8 – 1.0)	0.004
A	28 (13.2%)	21 (13.9%)	7 (11.3%)	Ref	Ref
B	67 (31.5%)	52 (34.4%)	15 (24.2%)	1.2 (0.4 – 3.2)	0.8
C	118 (55.4%)	78 (51.6%)	40 (64.5%)	0.7 (0.2 – 1.7)	0.4
MELD	16 (11-22)	16 (11-22)	16 (11-22)	Omitted	0.8
GAHS	6 (5 – 8)	6 (5 – 8)	6 (5 – 8)	1.0 (0.8 – 1.1)	0.5
Maddrey DF	24 (9 – 47)	22 (11 – 43)	29 (7 – 56)	1.0 (1.0 – 1.0)	0.3
Lille Score	0.4 (0.1 – 0.7)	0.5 (0.1 – 0.8)	0.3 (0.1 – 0.5)	2.8 (0.3 – 25.7)	0.4
Glucocorticoids	63 (18.5%)	46 (19.4%)	17 (16.5%)	1.2 (0.7 – 2.2)	0.5
Liver-related mortality					
30 days	24 (7.1%)	18 (7.6%)	6 (5.8%)	1.3 (0.5 – 3.5)	0.6
90 days	31 (9.1%)	21 (8.9%)	10 (9.7%)	0.9 (0.4 – 2.0)	0.8
All-cause mortality					
30 days	24 (7.1%)	18 (7.6%)	6 (5.8%)	1.3 (0.5 – 3.5)	0.6
90 days	36 (10.6%)	26 (12.0%)	10 (9.7%)	1.1 (0.5 – 2.5)	0.7
Admission duration (days)	6 (3 – 11)	6 (3 – 11)	7 (4 – 12)	1.0 (1.0 – 1.0)	0.4
Intensive care admission	33 (9.7%)	17 (7.1%)	16 (15.5%)	0.4 (0.2 – 0.9)	0.02
90-day readmission	134 (39.4%)	87 (36.7%)	47 (45.6%)	0.7 (0.4 – 1.1)	0.1

**ALT, alanine aminotransferase; AST, aspartate aminotransferase; AUD, alcohol use disorder; CLD, chronic liver disease; GAHS, Glasgow alcoholic hepatitis score; HBV, hepatitis B virus; HCC, hepatocellular carcinoma; HCV, hepatitis C virus; HE, hepatic encephalopathy; HRS-AKI, hepatorenal syndrome acute kidney injury; INR, international normalised ratio; IQR, interquartile range; Maddrey-DF, Maddrey discriminant function; MAFLD, metabolic associated fatty liver disease; MELD, Model for End-stage Liver Disease.*

1.4 Conclusion

AAH admissions in Victoria rose sharply from 2020 and have remained persistently elevated, with admission rates now three-fold higher than in 2020. Although women comprise a small proportion of AAH admissions, they have a distinct, more severe clinical profile than men.

These findings underscore the need for a coordinated public health response to reduce alcohol-related harm, including evidence-based population alcohol policies, strengthened addiction treatment pathways, and targeted prevention and service models for young women, to avert downstream increases in ALD and premature mortality in Australia.

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2. Alcohol-associated cirrhosis

2.1 Background

Liver cirrhosis is a significant cause of disease burden globally, with the Asia-Pacific region accounting for 54.3% of global deaths due to cirrhosis(14) and is the leading risk factor for liver failure (decompensated cirrhosis) and the development of hepatocellular carcinoma.

From 1990 to 2017, the global estimate of compensated, asymptomatic cirrhosis prevalence increased from 66 million cases to 112 million cases (14).

The overall prevalence and burden of disease due to cirrhosis is not well characterised in Australia, with an estimated 15,786/100,000 individuals living with cirrhosis in 2019 (range:14,533-17,070/100,000) (15). While hospital admissions data for decompensated cirrhosis have contributed to current estimates (3), the proportion of the population with asymptomatic, compensated cirrhosis is not well known. This underdiagnosis of cirrhosis poses challenges to improving liver cancer surveillance and outcomes among people in the community with occult, compensated cirrhosis who are at increased risk of hepatocellular carcinoma (HCC) development and have the most to benefit from surveillance (11, 14).

Importantly, the relative contribution of alcohol-related liver disease to this burden is poorly understood, in part due to inadequate recording of alcohol intake in hospital admissions datasets and failure to capture alcohol as a cofactor when other liver diseases such as viral hepatitis are also present(3, 16, 17). Available data from a retrospective data linkage study including people with cirrhosis in Queensland between 2008-2017 recorded alcohol-related liver disease as the aetiology in 49% of people admitted to hospital with cirrhosis(18), however contemporary trends in alcohol-related cirrhosis, particularly in the post-COVID-19 era, have not been determined.

Vibration controlled transient elastography, or Fibroscan, is a non-invasive measure of liver scarring (fibrosis) and diagnostic tool for cirrhosis that captures people with both compensated and decompensated cirrhosis (17). In this study, we aimed to describe liver fibrosis testing patterns, incidence and prevalence of alcohol and non-alcohol related cirrhosis among people undergoing Fibroscan evaluation for liver fibrosis in Victoria, Australia.

2.2 Methods

Study design and setting

This retrospective observational multicentre cohort study included Fibroscans performed across Victoria within the seven major health networks (Alfred Hospital, Austin Health, Eastern Health, Monash Health, Northern Health, St Vincent's Hospital Melbourne, Western Health, Melbourne Health) between January 1, 2010 and December 31, 2022. These seven health networks include outreach and regional Fibroscan services and represent the majority of all Fibroscans performed within Victoria.

Study population and time period

We included all Fibroscans perfomed in adults over 18 years of age across participating health services between January 1, 2010 to December 31, 2022.

Data sources and collection

Raw data were collected from Fibroscan machines and also from reporting databases across all seven health networks, collected by a member of our study team. Variables collected included year of birth, date of Fibroscan and indication, Fibroscan result and quality parameters (IQR, % success rate).

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Primary and Secondary Outcomes

Primary outcomes were 1) number of Fibroscans performed per year, overall and stratified by aetiology; 2) proportion of Fibroscans which met the disease specific liver stiffness measurement cutoff for cirrhosis; and 3) number (proportion) of individuals with cirrhosis, overall and stratified by aetiology and gender.

Secondary outcomes included the number of incident cirrhosis cases over time (years) among those who had repeat Fibroscans; and median time to development of cirrhosis in individuals with repeat Fibroscans and no cirrhosis at baseline.

Ethics Approval

The study was approved by St Vincent's Hospital Melbourne Human Research Ethics Committee (HREC80482/ LRR 314/21). Individual patient consent was waived for this retrospective analysis of de-identified data.

Details of the statistical analysis are provided in **Appendix 2.**

2.3 Results

Trends in assessment of cirrhosis

A total of 78,930 Vibration Controlled Transient Elastography (VCTE, or Fibroscan), Acoustic Radiation Force Impulse (ARFI), and Shear Wave Elastography (SWE) measurements were performed in 65,132 adults across the study period. The cohort had a median age of 53 years and 47% were female. The main indications for VCTE were hepatitis B (HBV, 24%), metabolic associated steatotic liver disease (MASLD; 23%), hepatitis C (HCV, 22%) and alcohol-associated liver disease (ALD, 7.8%). VCTE utilisation peaked in 2016–2019, followed by a marked downturn in 2020 temporally associated with the COVID-19 pandemic. MASLD-related VCTE testing steadily increased over the entire study period, whereas pre-2016 HCV related VCTE testing sharply escalated preceding the introduction of universal access to direct-acting antivirals in 2016, then reduced until 2022 (**Figure 2.1**).

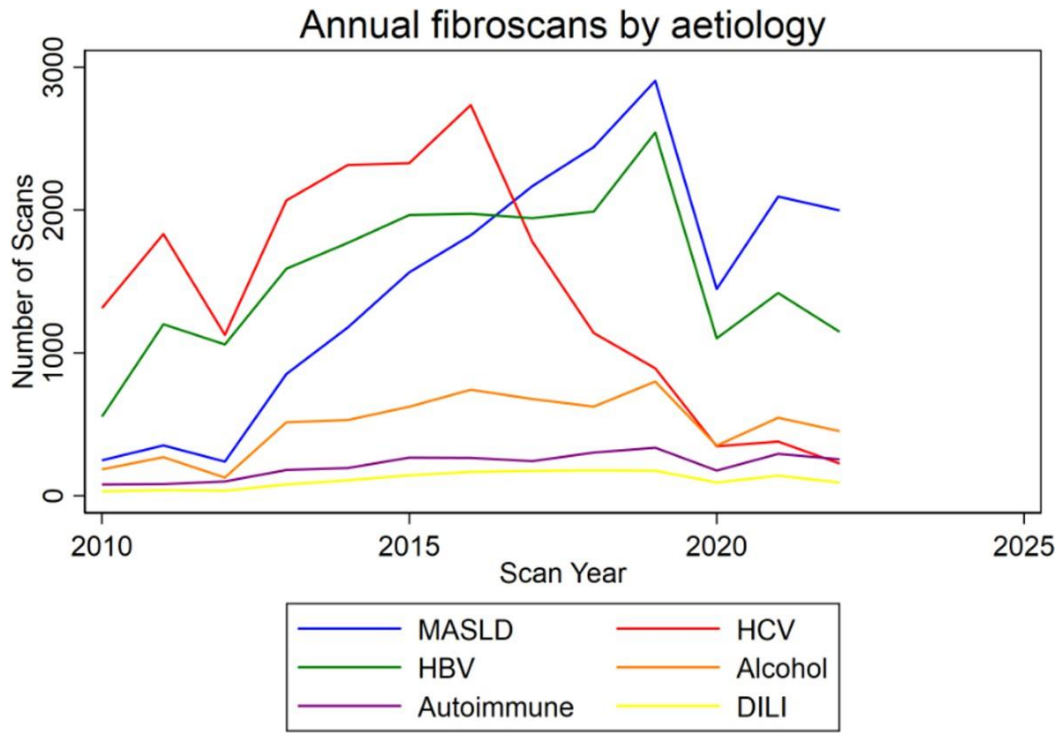


Figure 2.1. Number of Fibroscans per year, stratified by aetiology

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Cirrhosis prevalence

Overall, 13% of scans (11,052/83,038) demonstrated cirrhosis-range liver stiffness in 7,671 individuals. Among those with cirrhosis, 18.5% had ALD (N=1420). ALD-related cirrhosis was more common among men than women (20.7% compared with 13.1%; OR 1.72, 95% CI 1.48-2.01; p<0.001). The proportion of people with cirrhosis-range liver stiffness measurements declined significantly over time for HBV, ALD, autoimmune liver disease, and MASLD (all *p*<0.001), while HCV-related cirrhosis remained stable (*p*=0.15; **Figure 2.2**).

13,798 patients (21%) underwent repeat VCTE, with a median interval of 2.0 years (IQR 1.03–2.70). Among patients who had longitudinal follow-up, ALD had the fastest progression to cirrhosis on time-to-event analysis, with 50% of people developing cirrhosis within 6 years of their baseline VCTE (**Figure 2.3**). Time to progression to cirrhosis did not significantly differ by gender (p=0.346).

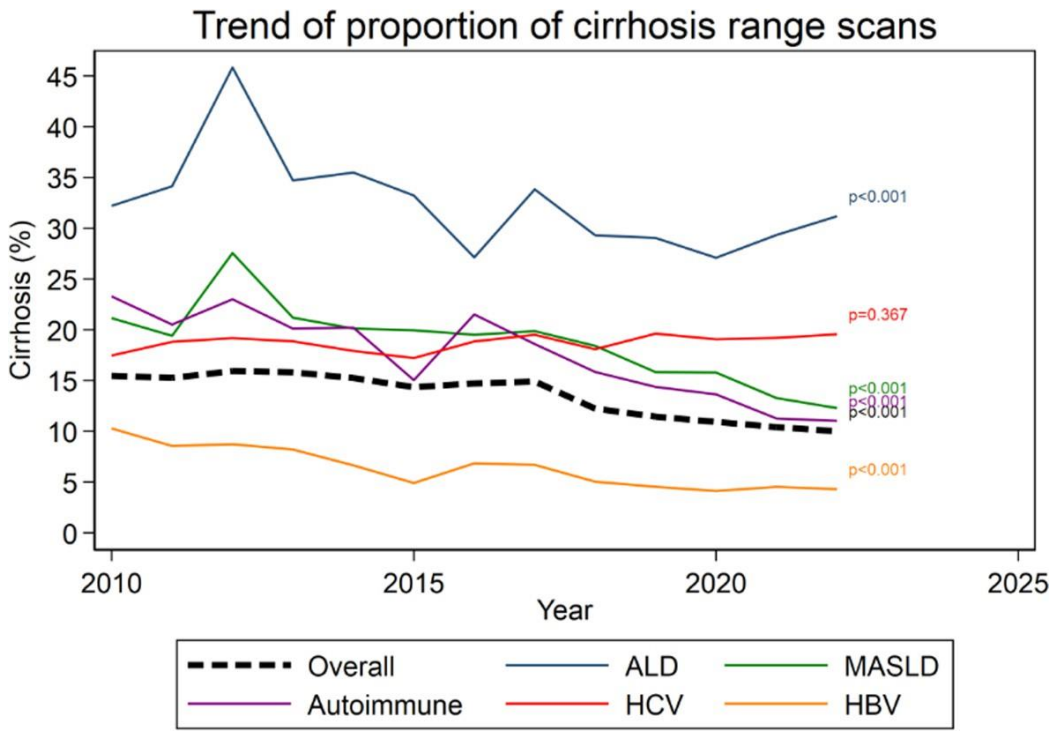


Figure 2.2. Proportion of Fibroscans with cirrhosis range liver stiffness measurement per year, stratified by aetiology

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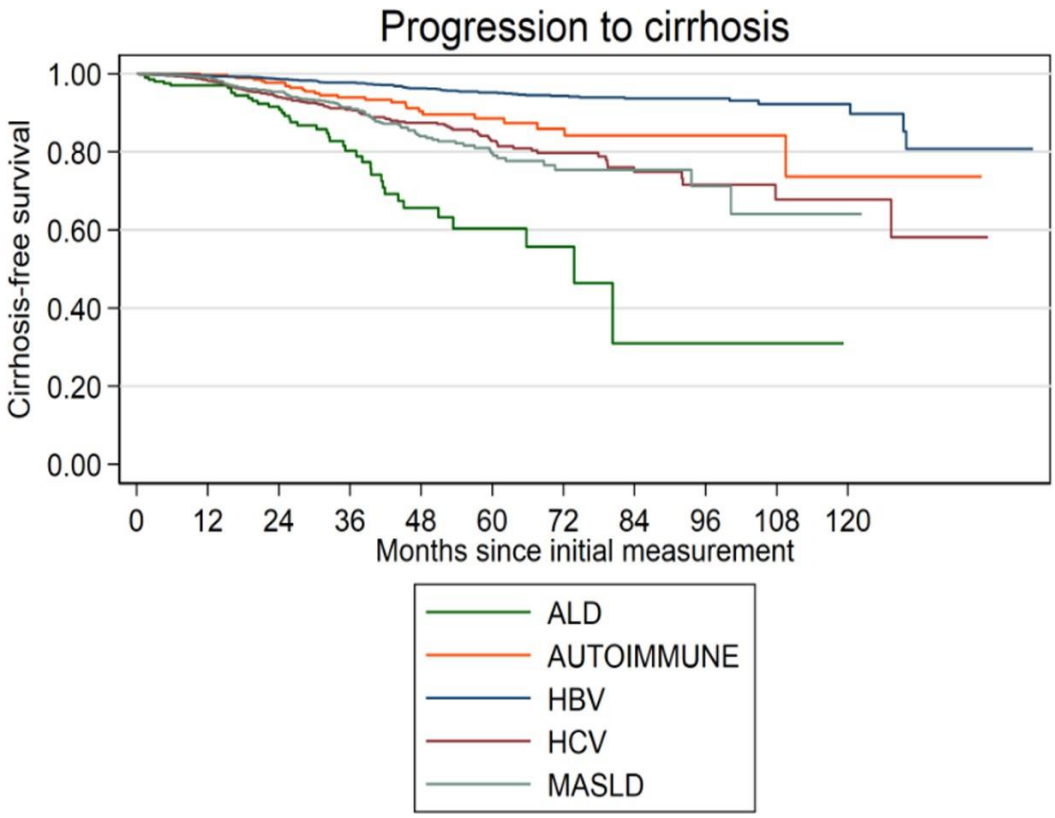


Figure 2.3. Time to progression to cirrhosis (months) among people with chronic liver disease among individuals with chronic liver disease who did not have cirrhosis on their index Fibroscan, stratified by aetiology.

2.4 Conclusion

Alcohol-related liver disease is the most common aetiology of cirrhosis in Victoria among people being assessed with VCTE. In contrast to other causes of liver disease, the proportion of people with cirrhosis due to alcohol continues to climb. Alcohol-related liver disease is associated with rapid fibrosis progression, with almost half of people with alcohol-related liver disease progressing to cirrhosis within 6 years of diagnosis. Despite this, evaluation for cirrhosis among people with alcohol-related liver disease lags behind other liver diseases, leading to lower uptake of HCC surveillance. Greater investment and education are needed to increase early recognition of alcohol-related liver disease and assessment for cirrhosis to improve clinical outcomes through early treatment.

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3. Alcohol-associated hepatocellular carcinoma

3.1 Background

Primary liver cancer, in particular hepatocellular carcinoma (HCC), represents a major global health challenge, ranking as the sixth most common cancer worldwide and the third leading cause of cancer-related mortality(19). The global burden of HCC varies significantly by geographical region, reflecting differences in the prevalence of underlying liver disease.

While hepatitis B virus (HBV) remains the predominant cause in Asia and sub-Saharan Africa and hepatitis C virus (HCV) has historically been the leading cause in many Western countries, alcohol-related liver disease (ALD) has emerged as a major cause of HCC-related mortality in Europe and globally(20-22).

Australian data on alcohol-associated HCC (AL-HCC) remain limited. A large multi-jurisdictional study reported that ALD accounted for 40.5% of cases between 2000 and 2017, with a disproportionate burden among First Nations peoples(23). In addition, two contemporary Australian population studies found prevalence to be 33-39%.(24, 25) However, alcohol exposure is frequently under-recognised as a key driver of chronic liver disease, in part because alcohol intake is commonly under-reported and poorly documented(26).

International studies have demonstrated that AL-HCC patients typically present with more multi-focal disease, worse performance status, poorer liver function, younger age at diagnosis and are more likely to be male(20) Importantly, people with AL-HCC are also more likely to be diagnosed at a later stage, often outside of formal surveillance programs, which significantly impacts their treatment options and prognosis(27). This pattern of late presentation has been attributed to multiple factors, including suboptimal engagement with healthcare services, lower rates of surveillance uptake, and challenges in maintaining abstinence from alcohol(21). The cascade of care for HCC encompasses several critical steps: recognition of at-risk patients through timely diagnosis of cirrhosis, timely referral to specialist services, enrolment in surveillance programs, and access to appropriate treatment modalities. For patients with ALD, there is growing evidence that breakdowns occur at multiple points in this cascade, leading to suboptimal outcomes(28-30).

The primary objective of this study was to provide a comprehensive analysis of AL-HCC epidemiology in a large, multi-centre Australian cohort. Specifically, we aimed to: 1) describe the prevalence of ALD among people with incident HCC diagnosed in Greater Melbourne; 2) describe the socio-demographic and clinical characteristics of people with AL-HCC compared to those with HCC due to other causes; 3) describe the cascade of care among people with AL-HCC compared to those with HCC due to other causes, including rates of cirrhosis diagnosis, surveillance enrolment, and specialist care linkage prior to HCC diagnosis; and 4) describe mortality among people with AL-HCC compared to non-AL HCC.

3.2 Methods

Study Design and Setting

This was a multi-centre observational cohort study conducted across the six major tertiary health networks which collectively provide specialist hepatology and oncology services to the Greater Melbourne population (approximately 5 million people in Victoria, Australia). The study comprised retrospective case ascertainment from 1 January 2018 to 31 December 2019 and prospective case ascertainment from 1 January 2020 to November 2022; outcomes were followed through to 31 December 2023.

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Study Population and Case Identification

All consecutive adult patients (≥ 18 years) with a new diagnosis of HCC managed at the participating health networks during the study period were included. Incident HCC cases were systematically identified through weekly multidisciplinary team meetings, which represent the standard of care for cancer diagnosis and management in the Australian healthcare system(31). All cases required a confirmed HCC diagnosis, established by histology or by characteristic imaging features, including LI-RADS criteria (Liver Imaging Reporting and Data System).

Data Sources and Collection

The HOMER2 database collates information from multiple sources, including HCC MDT notes and databases, electronic medical records, pathology and radiology reports, and dedicated HCC clinical databases maintained by individual health networks.

Key Definitions

The primary exposure variable was *AL-HCC*, defined as a clinician diagnosis of ALD as a primary or significant contributing factor to the underlying chronic liver disease, as documented in MDT notes and electronic medical records. This definition was based on clinical assessment by specialist hepatologists and was not mutually exclusive, recognising that patients may have multiple contributing aetiologies for their liver disease. The comparator group consisted of patients with non-alcohol-related HCC.

Liver disease severity was evaluated using the Child-Turcotte-Pugh (CTP) score and class (calculated based on serum albumin, bilirubin, INR, and the presence of ascites and encephalopathy). Model for End-Stage Liver Disease (MELD) score was calculated using the standard formula incorporating serum creatinine, bilirubin, and INR. Albumin-Bilirubin (ALBI) grade was calculated and categorised into grades 1, 2, and 3 based on established cut-off values.

The presence of *liver cirrhosis* was determined based on histological examination, disease-specific liver stiffness measurement using vibration controlled transient elastography, characteristic imaging features, or clinical evidence of portal hypertension. Clinically significant portal hypertension was defined by a liver stiffness of >25 kPa on transient elastography, presence of oesophageal or gastric varices, splenomegaly with thrombocytopenia, or other clinical signs of portal hypertension(32).

Tumour staging was comprehensively documented according to the Barcelona Clinic Liver Cancer (BCLC) staging system. Early-stage HCC was defined as BCLC stage 0 or A, while advanced-stage disease encompassed BCLC stages B, C, and D. Additional tumour characteristics included the number and size of lesions, presence of portal vein thrombosis, and evidence of extrahepatic metastases.

Cascade of Care Assessment

Engagement in the HCC cascade of care was systematically assessed through multiple indicators. Enrolment in HCC surveillance was defined as participation in a formal surveillance program with regular abdominal ultrasound examinations, with or without alpha-fetoprotein measurement, according to national guidelines(31). Surveillance adequacy was categorised as adequate (ultrasound surveillance within last 6 months), partial (ultrasound surveillance within last 12 months), or none.

The mode of HCC diagnosis was categorised as surveillance-detected (HCC initially identified on routine surveillance imaging) or incidental/symptomatic (HCC diagnosed following investigation of symptoms or incidentally on imaging performed for other indications). The source of referral to the MDT was also documented.

Treatment Classification

Curative treatments included surgical resection, liver transplantation, and ablative therapies (radiofrequency ablation, microwave ablation, percutaneous ethanol injection or stereotactic ablative

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body radiotherapy [SABR]). Palliative treatments included transarterial chemoembolisation (TACE), SABR, external beam radiotherapy, selective internal radiation therapy (SIRT), and systemic therapies (tyrosine kinase inhibitors, immunotherapy, or combination regimens). Best supportive care was defined as palliative symptom management without tumour-directed therapy.

Details of statistical analysis are provided in **Appendix 3.**

Ethics Approval

The study protocol was approved by the Human Research Ethics Committee of St. Vincent's Hospital Melbourne (the lead site) and received governance approval from all participating health networks (Project ID 82225, LRR 322/21

3.3 Results

Prevalence and incidence of AL-HCC

Between January 2018 and November 2022, 1194 incident HCC cases were recorded across the six participating health networks, of which 453 (37.9%) had AL-HCC and 741 (63.1%) attributed to other aetiologies (**Table 3.1**). Among patients with AL-HCC, alcohol was the sole recorded liver disease aetiology in 199 patients (43.9%), while 254 patients (56.1%) had alcohol recorded in combination with at least one other concurrent chronic liver disease aetiology. Poisson regression of annual case counts showed no evidence of a temporal trend during the study period (p=0.56, **Figure 3.1**). The proportion of AL-HCC among all incident HCC cases varied minimally year-to-year (36.8–40.4%; mean 37.9%).

AL-HCC Demographic and Social Characteristics

Compared with non-AL-HCC, patients with AL-HCC were younger at diagnosis (median 64 years [IQR 59.0 – 71] compared with 68 [60.0 – 76.0] years; p<0.001). The highest proportion of people with AL-HCC were in age categories 46–55 (14.8% vs 8.9%; p=0.001) and 56–65 (40.4% vs 27.7%; p=0.001). Men predominated among people with ALD-HCC (89.0% vs 75.0%; OR 2.7, 95% CI 1.9–3.8; p<0.001) and a higher proportion of people with ALD-HCC were Caucasian (89.7% vs 71.2%; OR 10.0; p=0.03) or First Nations Australian (2.0% vs 0.7%; OR 23.4; p=0.01). A higher proportion of people with AL-HCC lived in smaller towns in Greater Melbourne (UCL 2 32% AL-HCC vs 21% non-AL-HCC, p<0.001). Interestingly, socioeconomic disadvantage measured by IRSD decile did not differ by ALD status(19). A non-English-speaking background was less common among AL-HCC (7.4% vs 22.5%; p<0.001).

Harmful alcohol consumption was near-universal in AL-HCC (60.1% current; 39.9% previous) but uncommon in non-AL HCC (7.3% current; 10.6% previous; 82.1% none). Current smoking was more frequent in AL-HCC than in non-alcohol cases (29.8% vs 15.4%; p<0.001), and 48.1% people with AL-HCC were former smokers compared with 68.0% of the non-alcohol group. The prevalence of type 2 diabetes was more common among people with AL-HCC (14.8% vs 13.4%; p=0.001), whereas obesity prevalence was similar (13.0% vs 12.2%; p=0.66). A history of intravenous drug use trended higher among alcohol-associated cases (14.3% vs 9.9%; p=0.06)

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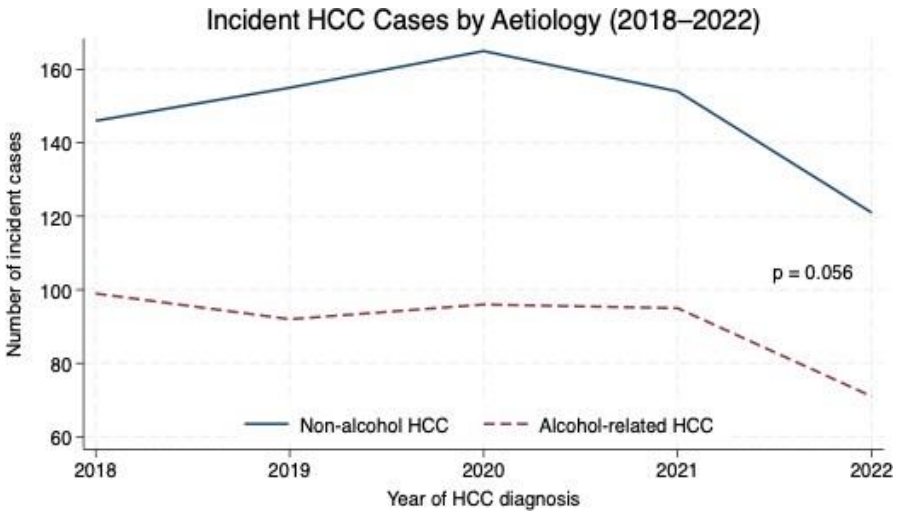


Figure 3.1. Annual number of incident hepatocellular carcinoma cases 2018–2022, stratified by alcohol and non-alcohol aetiology.

Table 3.1. Demographic and clinical characteristics at hepatocellular carcinoma diagnosis, stratified by alcohol and non-alcohol associated aetiology

Variable	AL-HCC (n=453)	Non-alcohol HCC (n=741)	Crude OR (95% CI)	p-value
Age (years, median, IQR)	64.0 (59.0-71.0)	68.0 (60.0-76.0)	1.0 (0.99 – 1)	0.001
Age Category				**0.001**
≤ 45	4 (0.5%)	34 (7.5%)	Reference	0.001
46 – 55	67 (14.8%)	66 (8.9%)	8.6 (2.9 – 25.7)	0.001
56 – 65	183 (40.4%)	205 (27.7%)	7.6 (2.6 – 21.8)	0.001
66 – 75	139 (30.7%)	250 (33.8%)	4.7 (1.6 – 13.6)	0.004
>75	60 (13.2%)	186 (25.1%)	2.7 (0.9 – 8.0)	0.066
Sex				
Male	403 (89.0%)	556 (75.0%)	2.7 (1.9 – 3.8)	0.001
Ethnicity				**0.001**
Caucasian	402 (88.7%)	525 (70.9%)	Reference	
African	1 (0.2%)	13 (1.8%)	0.1 (0.01 – 0.8)	0.027
Asian	36 (7.9%)	194 (26.3%)	0.2 (0.2 – 0.4)	0.001
First Nations	9 (2.0%)	5 (0.7%)	2.4 (0.8 – 7.1)	0.13
Non-English Speaking				
No	411 (90.7%)	557 (75.2%)	Reference	

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Variable	AL-HCC (n=453)	Non-alcohol HCC (n=741)	Crude OR (95% CI)	p-value
Yes	33 (7.3%)	167 (22.5%)	0.3 (0.2 – 0.4)	0.001
IRSD – disadvantage deciles				0.5
1-2 (Most disadvantaged)	86 (19.0%)	158 (21.3%)	Reference	
3-4	92 (20.3%)	142 (19.2%)	1.2 (0.7 – 1.4)	0.4
5-6	93 (20.5%)	175 (23.6%)	1.0 (0.7 – 1.4)	0.9
7-8	97 (21.4%)	136 (18.4%)	1.3 (0.9 – 1.9)	0.2
9-10 (Least disadvantaged)	82 (18.1%)	130 (17.5%)	1.2 (0.8 – 1.7)	0.4
BMI Category				0.4
Urban-locality data (ABS)				<0.001
Category 1 (>10,000 people) **Category 2 (>900 < 10,000)**	260 (68%) 123 (32%)	493 (79%) 131 (21%)	Reference 1.8 (1.3-2.4)	
<18.5	2 (6.1%)	1 (1.7%)	4.2 (0.3 – 54.1)	0.3
18.5 – 24.9	8 (24.2%)	17 (28.3%)	Reference	
25 – 29.9	12 (36.4%)	28 (46.7%)	0.9 (0.3 – 2.7)	0.9
>30	11 (33.3%)	14 (23.3%)	1.7 (0.5 – 5.3)	0.4
ECOG				0.3
0	258 (58.1%)	437 (60.6%)	Reference	
1	121 (27.3%)	165 (22.9%)	1.2 (0.9 – 1.7)	0.1
2	44 (9.9%)	72 (10.0%)	0.9 (0.6 – 1.5)	0.9
3	19 (4.3%)	39 (5.4%)	0.9 (0.5 – 1.5)	0.6
4	2 (0.5%)	8 (1.1%)	0.4 (0.1 – 2)	0.3
Harmful alcohol use				0.001
Previous	180 (39.7%)	77 (10.4%)	Reference	
Current	271 (60.1%)	53 (7.2%)	2.2 (1.5 – 3.3)	0.001
Smoking				0.001
Current	135 (29.8%)	114 (15.4%)	2.7 (2.0 – 3.6)	0.001
Previous	218 (48.1%)	491 (66.3%)	1.6 (1.2 – 2.2)	0.005

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Variable	AL-HCC (n=453)	Non-alcohol HCC (n=741)	Crude OR (95% CI)	p-value
No	218 (50.0%)	491 (69.0%)	Reference	
T2DM	140 (31.1%)	298 (40.6%)	0.7 (0.5 – 0.8)	0.001
Mental Health diagnosis	65 (14.8%)	97 (13.4%)	1.1 (0.8 – 1.6)	0.5
Intravenous drug use				0.06
Current	13 (3.0%)	16 (2.2%)	1.4 (0.7 – 3.0)	0.3
Previous	51(11.3%)	57 (7.7%)	1.6 (1.1 – 2.3)	0.03
Never used	367(81.0%)	645 (87.0%)	Reference	

**CI – Confidence interval; IQR – interquartile range; BMI - Body mass index; ECOG – Eastern Cooperative Oncology Group; T2DM – type 2 diabetes mellitus; IRSED – Index of Relative Socio-economic Disadvantage; SEIFA – socio-economic indexes for areas.*

Liver Disease Severity and Portal Hypertension

Compared with non-AL-HCC, patients with AL-HCC were more likely to have cirrhosis (96.5% compared with 73.7%, p<0.001). Patients with AL-HCC presented with more advanced chronic liver disease *(*Table 3.2*)*. Clinically significant portal hypertension was more frequent (63.1% vs 37.7%; p<0.001), with a higher median liver stiffness (median 29.7 kPa vs 19.3 kPa; p<0.001) and consequent complications were also more common, including ascites (27.0% vs 12.3%; p<0.001) and hepatic encephalopathy (8.2% vs 4.4%; p=0.01).

Consistent with this, Child–Pugh scores were higher in AL-HCC than non-AL-HCC (median 6.0 vs 5.0; p<0.001) and decompensated Child–Pugh B class (32.0% vs 20.2%; OR 2.1; p<0.001) and Child-Pugh C class (12.8% vs 5.7%; OR 3.0; p<0.001) liver disease occurred more frequently in people with AL-HCC. Similarly, liver function measured by median ALBI score was also poorer in AL-HCC (−1.67 vs −2.00; p<0.001).

Tumour Characteristics and Staging

Tumour characteristics in AL-HCC showed distinct patterns (Table 2). People with AL-HCC were more likely to present with advanced BCLC stage (59.4% vs 48.2%; p<0.001; table 1). Portal vein thrombosis was also more frequent (24.2% vs 16.9%; p=0.001), however median size and number of nodules did not differ to those with non-AL-HCC.

Table 3.2. Hepatic function and portal hypertension features by aetiology (alcohol-associated hepatocellular carcinoma compared with non-alcohol associated hepatocellular carcinoma)

Variable	AL-HCC (n=453)	Non-alcohol HCC (n=741)	Crude OR (95% CI)	p-value
Fibroscan LSM (kPa)	29.7 ± 19.8	19.3 ± 13.2	1.04 (1.01 – 1.06)	<0.001
Portal hypertension	284 (63.1%)	276 (37.7%)	2.8 (2.2 – 3.6)	<0.001
Ascites	120 (27.0%)	89 (12.3%)	2.6 (1.9 – 3.6)	<0.001
Encephalopathy	36 (8.2%)	32 (4.4%)	1.9 (1.2 – 3.1)	0.010

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Variable	AL-HCC (n=453)	Non-alcohol HCC (n=741)	Crude OR (95% CI)	p-value
Child-Pugh Score (median, IQR)	6.0 (5.0-8.0)	5.0 (5.0-7.0)	1.0 (1.01– 1.01)	<0.001
Child-Pugh Class				<0.001
A	250 (55.2%)	549 (74.1%)	Reference	
B	145 (32.0%)	150 (20.2%)	2.1 (1.6-2.8)	<0.001
C	58 (12.8%)	42 (5.7%)	3.0 (2.0 – 4.6)	<0.001
MELD (median, IQR)	9 (7-12)	8 (6-11)	1.04 (1.00 – 1.09)	0.01
ALBI (median, IQR)	-1.67 (-2.2 - - 0.69)	-2.00 (-2.51 –- 0.99)	1.1 (1.00 – 1.14)	<0.001
ALBI (grade)				<0.001
Grade 1 (≤ −2.60)	43 (9.3%)	140 (18.8%)	Reference	
Grade 2 (> −2.60 to ≤ −1.39)	226 (49.1%)	377 (50.5%)	2.0 (1.3 – 2.9)	<0.001
Grade 3 (> - 1.39)	94 (20.4%)	68 (9.1%)	4.5 (2.8 – 7.1)	<0.001
BCLC Stage				
Early (0-A)	183 (40.6%)	383 (51.8%)	0.63 (0.5 – 0.80)	<0.001
Advanced (B-D)	268 (59.4%)	356 (48.2%)		

**HCC – hepatocellular carcinoma; ALBI – Albumin-Bilirubin score; BCLC – Barcelona Clinic Liver Cancer Staging; LSM – Liver stiffness measurement; MELD – Model of End-Stage Liver Disease*

Cascade of Care Uptake

Engagement with the HCC cascade of care differed significantly between AL-HCC and non-AL-HCC (Table 3.3). AL-HCC cases were more commonly referred from inpatient hospital teams (25.0% vs 18.6%; p=0.006) or peripheral hospitals (11.3% vs 7.6%; p=0.008), rather than via established liver specialist clinics, and were consequently less likely to be linked into specialist liver services at the time of HCC diagnosis (Table 3.3).

People with AL-HCC were more likely tobe diagnosed with cirrhosis for the first time at the time of HCC presentation (36.2% vs 26.0%; p<0.001). Patients with AL-HCC were also less often enrolled in HCC surveillance than those with non-AL-HCC (36.2% vs 43.0%; p=0.027); when enrolled in surveillance, AL-HCC were less likely to be detected by scheduled surveillance imaging (32.9% vs 42.2%; p<0.001).

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Table 3.3. Uptake of the hepatocellular carcinoma cascade of care among people with alcohol-associated hepatocellular carcinoma and non-alcohol associated hepatocellular carcinoma

Variable	AL-HCC (n=453)	Non-alcohol HCC (n=741)	Crude OR (95% CI)	p-value
Cirrhosis Status				<0.001
No	16 (3.5%)	195 (26.3%)	Reference	
Yes – prior diagnosis	273 (60.3%)	353 (47.6%)	9.4 (5.5 – 16.1)	<0.001
Yes – new diagnosis	164 (36.2%)	193 (26.0%)	10.4 (6.0 – 18.0)	<0.001
HCC screening program				
Enrolled HCC surveillance	164 (36.2%)	319 (43.0%)	0.8 (0.6 – 1.0)	0.027
Within 6 months prior	121 (26.7%)	225 (30.4%)	13.6 (9.3 – 19.9)	<0.001
Within 12 months prior	109 (24.1%)	240 (32.4%)	22.6 (13.9 – 34.4)	0.029
Mode of diagnosis				
Ultrasound surveillance	149 (32.9%)	313 (42.2%)	0.7 (0.5 – 0.9)	<0.001
Symptomatic / other indication	304 (67.1%)	428 (57.8%)		
Referral				
General practitioner	41(8.9%)	92 (12.3%)	Reference	
Liver clinic	85 (18.5%)	180 (24.1%)	1.1 (0.7- 1.7)	0.8
Surgical clinic	16 (3.5%)	43 (5.8%)	0.8 (0.4 – 1.7)	0.6
Private specialist	98 (21.3%)	170 (22.8%)	1.3 (0.8 – 2.0)	0.3
Inpatient team	115 (25.0%)	139 (18.6%)	1.9 (1.2 – 2.9)	0.006
Peripheral hospital	52 (11.3%)	57 (7.6%)	2.0 (1.2 – 3.5)	0.008

**HCC – hepatocellular carcinoma; AL-HCC – alcohol associated HCC.*

Treatment Allocation and Access to Curative Therapies

Patients with AL-HCC were less likely to receive any curative therapy (30.0% vs 38.3%; p=0.03), including surgical resection (7.2% vs 20.0%; p<0.001; **Table 3.4**). However, when adjusted for BCLC stage, AL-HCC was not independently associated with receipt of curative therapy, suggesting that any difference in AL-HCC receiving curative therapy was due to advanced BCLC stage at diagnosis rather than alcohol status per se. The BCLC-adjusted predicted probability of receiving curative therapy was 36.1% for non–alcohol-related HCC versus 34.1% (95% CI −6.5 to 2.6; p=0.40). Consistent with more advanced liver disease, BCLC stage and worse performance status at presentation, people with ALD-HCC were more often managed with best supportive care (27.2% vs 19.4%; p<0.001). However, referral for liver transplantation did not differ by AL-HCC status (3.9% vs 2.8%; p=0.50).

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Table 3.4. Hepatocellular carcinoma treatment modalities, stratified by alcohol-associated compared with non-alcohol associated aetiology

Variable	AL-HCC (n=453)	Non-alcohol HCC (n=741)	Crude OR (95% CI)	p-value
First treatment type				
Liver transplant referral	18 (3.9%)	21(2.8%)	1.3 (0.6 – 2.4)	0.500
Surgical resection	33 (7.2%)	149 (20.0%)	0.3 (0.2 – 0.5)	0.001
Laparoscopic / open MWA	16 (3.5%)	18 (2.4%)	1.5 (0.7 – 2.9)	0.270
Percutaneous MWA	73 (15.9%)	103 (13.8%)	1.2 (0.9 – 1.6)	0.300
TACE	120 (26.1%)	187 (25.1%)	1.1 (0.8 – 1.4)	0.630
SIRT	11 (2.4%)	25 (3.4%)	0.7 (0.3 – 1.5)	0.350
SABR/EBRT	3 (0.7%)	19 (2.6%)	0.3 (0.1 – 1.2)	0.082
Systemic TKI	32 (7.0%)	56 (7.5%)	0.9 (0.6 – 1.5)	0.750
Immunotherapy	22 (4.8%)	28 (3.8%)	1.2 (0.7 – 2.3)	0.370
Clinical trial	12 (2.6%)	14 (1.4%)	1.4 (0.6 – 3.1)	0.383
Best supportive care	125 (27.6%)	144 (19.4%)	1.6 (1.2 – 2.1)	0.001
Curative treatment	136 (30.0%)	286 (38.6%)	0.7 (0.5 – 0.9)	0.030

*****MWA – Microwave ablation; TACE – transarterial chemoembolization; SIRT – Selective internal radiation therapy; SABR – Stereotactic ablative body radiotherapy; EBRT – External beam radiotherapy; TKI – Tyrosine kinase inhibitors.*

Survival Outcomes and Prognostic Factors

Median overall survival after HCC diagnosis was 4.2 years (95% CI 3.6–5.6; Figure 3.2A);

4.9 years in people with non–AL-HCC (95% CI 4.0–not reached) versus 3.3 years in alcohol-related HCC (95% CI 2.5–4.7; p=0.009; Figure 3.2B). However, on multivariable Cox regression adjusting for age, BCLC stage, Child–Pugh class, ECOG performance status, and smoking status, AL-HCC was not independently associated with overall mortality (HR 1.1, 95% CI 0.9–1.3; p=0.27) (Table 3.5).

Sex-specific differences in ALD-HCC risks, clinical presentation and outcomes

We compared differences in AL-HCC presentation and outcomes between men and women. In sex-stratified analyses restricted to patients with AL-HCC (n=453), women comprised 11.0% of cases and men 89.0%. Age at diagnosis was similar by sex (median 63 years [IQR 57–69] in women vs 64 [59–72] in men; p=0.26), with no significant differences in age-category distribution or the prevalence of concurrent liver disease aetiologies recorded alongside alcohol (HBV, HCV, and MASLD; all p>0.05) (Table 3.6).

Behavioural and psychosocial comorbidity profiles differed by sex. Women were more likely to be current smokers than men (46.0% vs 29.0%; overall p=0.031) and had a higher prevalence of a documented mental health diagnosis (26.0% vs 13.3%; OR 2.28, 95% CI 1.14–4.58; p=0.017) (Table 3.6). History of

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intravenous drug use also differed, with women more frequently reporting current use (8.3% vs 2.4%, p=0.034). In contrast, diabetes and obesity did not significantly differ by sex.

Liver disease severity and tumour stage at diagnosis were similar by sex, including median Child–Pugh score (6 [IQR 5–9] in women vs 6 [5–8] in men; p=0.52), MELD score (10 [6–12] vs 9 [7–12]; p=0.81), Child–Pugh class distribution (overall p=0.065), and BCLC stage (early [0–A] 49.0% in women vs 39.6% in men; p=0.21) (**Table 3.6**). Sex-stratified survival outcomes for AL-HCC are summarised in Kaplan–Meier curves (**Figure 3.3**). Median overall survival was 5.1 years (95% CI [4.6 – 5.5]) in women and 4.6 years (95% CI [4.2 – 4.8]) in men, with survival distributions not differing on log-rank testing (p=0.26).

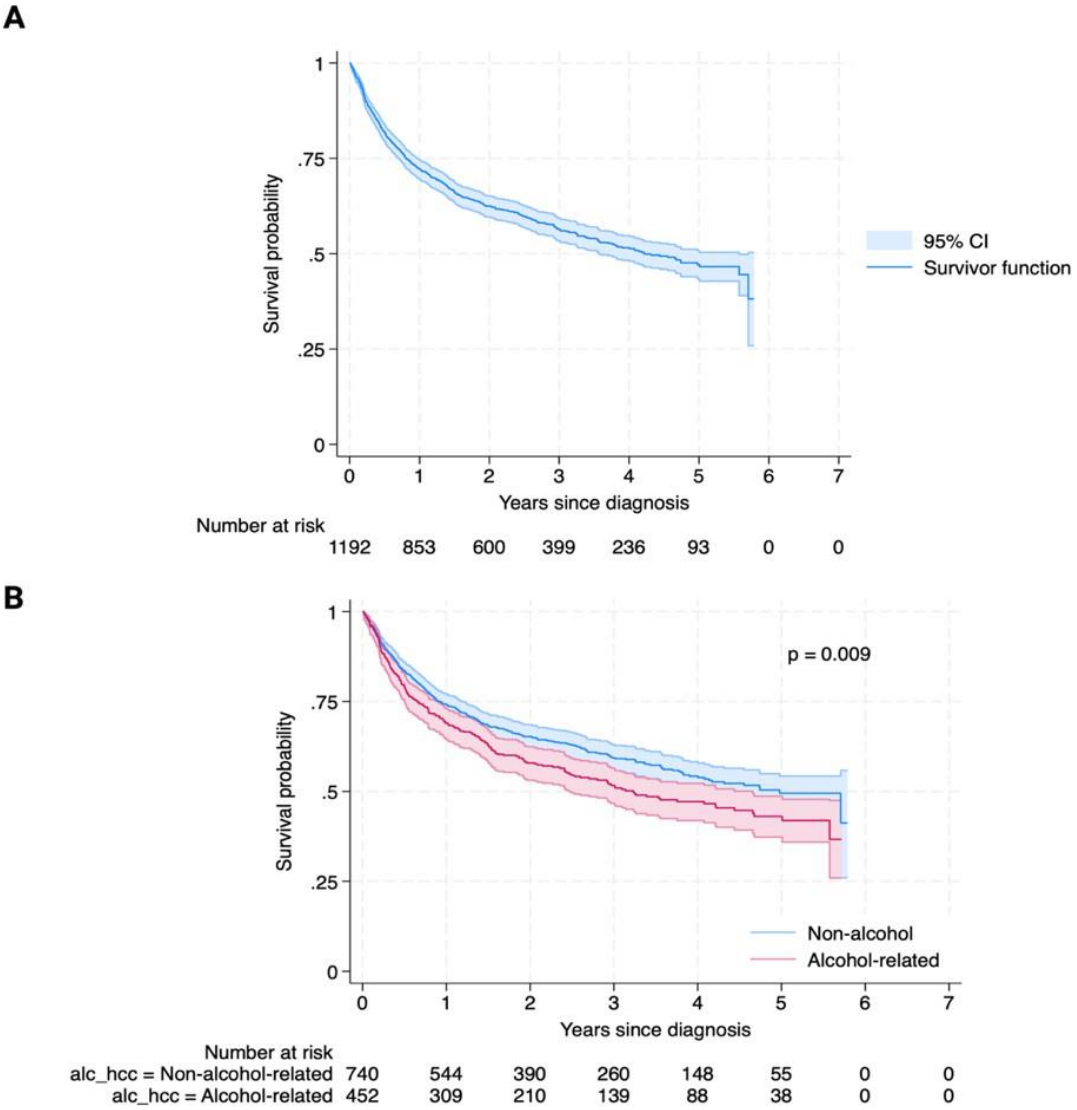


Figure 3.2. Overall survival after hepatocellular carcinoma diagnosis in years: (A) entire cohort; (B) by aetiology (alcohol-related vs non-alcohol related hepatocellular carcinoma)

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Table 3.5: Multivariable Cox-proportional hazards model for independent predictors of overall survival in people with alcohol associated and non-alcohol associated hepatocellular carcinoma

Characteristics	Univariate		Multivariate	
	HR (95% CI)	p-value	HR (95% CI)	p-value
Alcohol liver disease	1.3 (1.1 – 1.5)	0.010	1.1 (0.9 – 1.3)	0.270
Age	1.0 (1.0 – 1.0)	0.020	1.0 (0.99 – 1.0)	0.130
BCLC Stage				
Early (0-A)	0.3 (0.2 – 0.4)	0.001	0.4 (0.3 – 0.5)	0.001
Late (B-D)	Reference			
Child-Pugh Class				
A	Reference	Reference	Reference	
B	2.6 (2.2 – 3.1)	0.001	1.8 (1.4 – 2.2)	0.001
C	2.7 (2.1 – 3.6)	0.001	1.6 (1.2 – 2.2)	0.003
ECOG Performance Status				
0	Reference	Reference	Reference	
1	2.4 (2.0 – 2.9)	0.001	1.7 (1.4 – 2.1)	<0.001
2	2.9 (2.2 – 3.8)	0.001	1.6 (1.2 – 2.2)	0.001
3	4.0 (2.8 – 5.5)	0.001	1.6 (1.1 – 2.5)	0.008
4	5.9 (2.9 – 12.0)	0.084	2.5 (1.2 – 5.5)	0.020
Smoking Status				
No	Reference		Reference	
Yes	0.9 (0.7 – 1.1)	0.810	0.9 (0.7 – 1.1)	0.200

**BCLC – Barcelona clinic liver cancer staging; ECOG – Eastern Cooperative Oncology Group*

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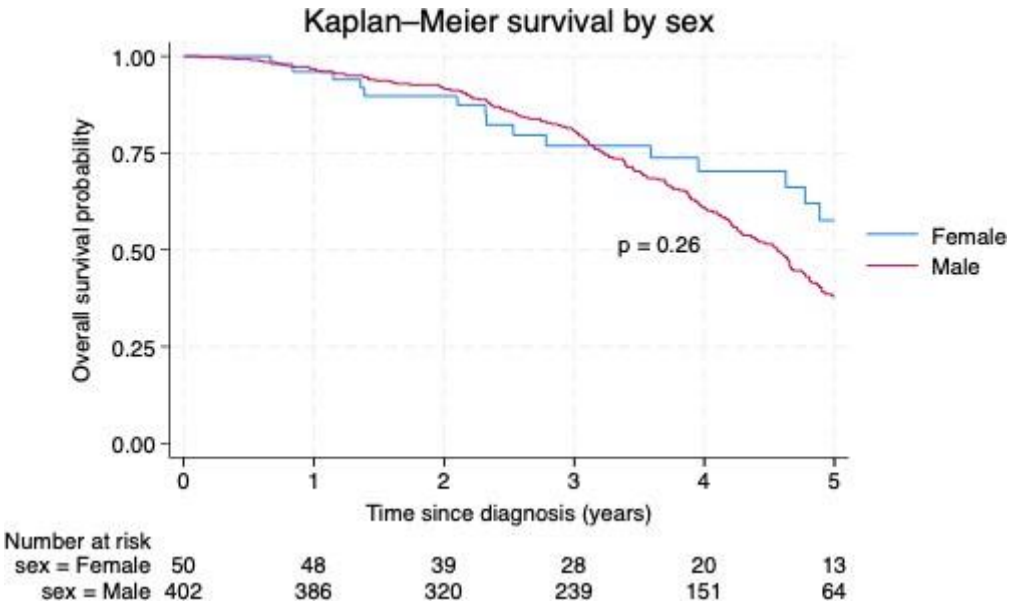


Figure 3.3. Overall survival since diagnosis in people with alcohol-associated hepatocellular carcinoma (years), stratified by sex.

Table 3.6: Sex-stratified distribution of sociodemographic and clinical variables in patients with AL-HCC

Variable	Male (n=403)	Female (n=50)	Crude OR (95% CI)	p-value
Age years (median, IQR)	64 (59-72)	63 (57-69)	0.98 (0.95 – 1.01)	0.26
Age Category				
36 – 45	3 (0.7%)	1 (2.0%)	2.32 (0.23 – 23.24)	0.48
46 – 55	59 (14.6%)	8 (16.0%)	0.94 (0.40 – 2.22)	0.89
56 – 65	160 (39.7%)	23 (46.0%)	Reference	Ref
66 – 75	127 (31.5%)	12 (24.0%)	0.66 (0.31 – 1.37)	0.26
>75	54 (13.4%)	6 (12.0%)	0.77 (0.30 – 2.0)	0.60
Place of residence				
UCL 1 > 10,000 people	232 (69%)	28 (62%)	Reference	
UCL 2 900-10,000 people	106 (31%)	17 (38%)	0.75 (0.39-1.43)	0.39
Harmful alcohol use				
Previous	155 (38.7%)	25 (50.0%)	1.59 (0.88 – 2.87)	0.13
Current	246 (61.3%)	25 (50.0%)	Reference	
Smoking				0.031
Current	112 (29.0%)	23 (46.0%)	Reference	
Previous	78 (20.2%)	5 (10.0%)	0.31 (0.11 – 0.86)	0.024

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Variable	Male (n=403)	Female (n=50)	Crude OR (95% CI)	p-value
No	196 (50.8%)	22 (44.0%)	0.55 (0.29 – 1.0)	0.06
T2DM	128 (32.0%)	12 (24.0%)	0.67 (0.34 – 1.33)	0.25
Obesity	55 (14.2%)	4 (8.2%)	0.54 (0.19 – 1.55)	0.25
Mental Health diagnosis	52 (13.3%)	13 (26.0%)	2.28 (1.14 – 4.58)	0.017
Intravenous drug use				0.034
Current	9 (2.4%)	4 (8.3%)	Reference	
Previous	43 (11.2%)	8 (16.7%)	0.42 (0.10 – 1.70)	0.22
Never used	331 (86.4%)	36 (75.0%)	0.24 (0.7 – 0.83)	0.025
Child-Pugh Score (median, IQR)	6 (5 – 8)	6 (5 – 9)	1.0 (0.99 – 1.01)	0.52
Child-Pugh Class				0.065
A	222 (55.1%)	28 (56.0%)	Reference	Ref
B	134 (33.2%)	11 (22.0%)	0.65 (0.31 – 1.35)	0.25
C	47 (11.7%)	11 (22.0%)	1.8 (0.86 – 3.99)	0.11
MELD (median, IQR)	9 (7 – 12)	10 (6 – 12)	1.02 (0.95 – 1.09)	0.81
BCLC Stage				
Early (0-A)	159 (39.6%)	24 (49.0%)	1.47 (0.81 – 2.66)	0.21
Advanced (B-D)	243 (60.4%)	25 (51.0%)	Reference	Ref

3.4 Conclusion

AL-HCC represents a major health challenge in Australia. Alcohol-related liver disease accounts for 38% of all HCC cases in Greater Melbourne, with stable incidence over the past 5 years. In contrast to HCC due to other causes, people with AL-HCC are younger, with more severe liver disease and more often present with advanced, non-curative HCC. This results in people with AL-HCC experiencing an almost two-year lower median survival compared with people with non-AL-HCC.

People with AL-HCC are a vulnerable population who present with advanced disease and face challenges to engagement with the healthcare system. The finding that survival outcomes are primarily determined by disease stage at diagnosis rather than alcohol aetiology itself provides hope that targeted interventions to improve the cascade of care could significantly improve outcomes for people with ALD. Addressing these challenges will require coordinated efforts across multiple levels of the healthcare system, from individual patient care to broader public health policy interventions.

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4. Conclusion

4.1 Summary of data and implications

Alcohol-related liver disease is one of the most rapidly progressive liver diseases and a major cause of morbidity and mortality in Australia. Alcohol-associated hepatitis, cirrhosis and HCC represent the severe end outcomes of progressive alcohol-related liver disease and affect people with alcohol use disorder across the age spectrum (summarised in **Table 4.1**). While cirrhosis and HCC are later complications that arise after years of alcohol-related liver disease, alcohol-associated hepatitis can occur at any age and stage of alcohol use. Alcohol-related liver disease may also coexist with other chronic liver diseases, acting synergistically to worsen outcomes.

A significant proportion of people with complications of alcohol-related liver disease are under 40 years of age, with the majority experiencing life-threatening complications in middle age, resulting in major repercussions for work and family life. Alcohol-related liver disease complications also affect people across socio-economic strata, with highest morbidity seen in both low and high socio-economic areas.

Alcohol-related liver disease is most common among people born in Australia, however culturally safe and acceptable approaches to support people born overseas are increasingly needed. Similarly, the increasing burden of alcohol-related liver disease among women requires urgent research and advocacy to ensure alcohol support services meet the needs of women with alcohol-related liver disease.

Table 4.1. Comparison of key features of alcohol-associated hepatitis, alcohol related cirrhosis and alcohol associated hepatocellular carcinoma (HCC) in Victoria

	Alcohol-related Hepatitis	Alcohol-related cirrhosis	Alcohol-related HCC
Cohort size and sample	4111, Victorian hospital admissions	1420, Victorians FibroscanΨ	453, HCC cases Greater Melbourne
Prevalence	-	19%	38%
Median Age (IQR)	48 (39, 57)	-	64 (59, 71)
Proportion < 40 yrs	14%	-	8%
Males Females	63% 37%	21% 13%	89% 11%
Ethnic Group: Caucasian Asian African	 77% 10% 7%	-	 89% 8% <5%
Aboriginal/ Torres Strait Islander	<3%		<5%
Median IRSD deciles: 1-2 (most disadvantaged) 3-4	 8% 27%	-	 19% 20%

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	Alcohol-related Hepatitis	Alcohol-related cirrhosis	Alcohol-related HCC
5-6	14%		21%
7-8	18%		21%
9-10 (least disadvantaged)	33%		18%
Alcohol Use Disorder:		-	
Current:	100%		60%
Past:			40%
Mental health condition	68%	-	15%
Cirrhosis (Y/N)	53%	100%	97%
Cirrhosis Stage*		-	
A	13%		55%
B	32%		32%
C	55%		13%
Mortality (%)	4%	-	59%

ΨFibroscan, transient elastography

**In people with cirrhosis, Child Pugh stage A (compensated liver function) to C (severe decompensation)*

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Appendix 1

Statistical Analysis AAH

We described the distribution of socio-demographic and clinical variables for the VAED and SVHM cohorts, overall and stratified by gender. Non-parametric continuous variables were reported as medians with IQRs and parametric continuous variables as means +/- sd; categorical variables as counts and percentages. Differences were assessed using Wilcoxon rank-sum test for non-parametric continuous variables and chi-square or Fisher's exact test for categorical variables.

To evaluate trends in monthly AAH admission rate pre and post COVID-19 in Victoria, we used interrupted time series (ITS) analysis with Poisson regression for SVHM data and generalized linear models (Gaussian family, identity link) with Newey-West standard errors for VAED data to compare the mean monthly rate of AAH admissions pre, during and post COVID-19 pandemic restrictions in Victoria. Models assessed changes in level and mean rate of AAH admissions per month between March 2020 and November 2021, overall and stratified by male and female gender. In the SVHM cohort, Poisson regression was also used to model the annual count of unique individuals and the number of admissions per person over time. Survival analyses were performed using Kaplan-Meier method and log-rank test.

Cox proportional hazards models were built to identify variables associated with mortality. All analyses were performed using Stata/BE 18.0 (StataCorp LLC, College Station, TX, USA). A two-sided P-value <0.05 was considered statistically significant.

Supplementary Table S1.1 Multivariate Cox model for 90-day all-cause mortality

Characteristics		Univariate		Multivariate	
	N	HR (95% CI)	p-value	HR (95% CI)	p-value
COVID Strata					
Overall	340	0.6 (0.3 – 1.1)	0.1	0.6 (0.3 – 1.2)	0.2
Age					
<40	77	Ref	Ref	Ref	Ref
40-59	199	3.7 (1.1 – 12.2)	0.03	3.1 (0.9 – 10.3)	0.07
≥60	64	2.5 (0.6 – 10.1)	0.2	2.7 (9.7 0- 10.9)	0.2
Male	237	1.1 (0.6 – 2.4)	0.7	1.4 (0.7 – 3.0)	0.4
Risk scores (median,IQR)					
Maddrey DF	24 (9 – 46)	1.0 (1.0 – 1.0)	<0.001	1.0 (1.0 – 1.0)	0.001

**HR, Hazard Ratio; CI, confidence interval*

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Appendix 2

Statistical analysis- alcohol-related cirrhosis

Count and categorical variables were described as numbers (proportion) and continuous variables described using mean and standard deviation (normally distributed data) or median and interquartile range (IQR, non-parametric data).

The number of Fibroscans performed during the study period was reported, overall and stratified by year and liver disease indication.

Trends over time described as the proportion of cirrhosis range Fibroscan cases per year overall and stratified by liver disease indication were compared using Chi square test. The number of new cirrhosis cases diagnosed by Fibroscan per month was calculated to provide a monthly cirrhosis incidence rate and linear regression modelling used to compare the difference in the slope of cirrhosis incidence per month between different liver disease aetiologies.

The number and proportion of individuals who have repeat Fibroscans over time (without cirrhosis) described overall, then stratified by liver disease indication and compared by Chi square test. The median time interval in years between repeat Fibroscans will be calculated overall and stratified by liver disease indication, with time to cirrhosis estimated and compared between aetiologies using Cox proportional hazards modelling.

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Appendix 3

Statistical Analysis- AL-HCC

Descriptive statistics were used to summarise the characteristics of the study cohort, with results presented overall and stratified by alcohol-related versus non-alcohol-related HCC. Comparisons between groups were performed using Student's t-test for normally distributed continuous variables and the Wilcoxon rank-sum test for non-normally distributed variables and Chi square test for proportions.

Temporal trends in HCC incidence were assessed using Poisson regression models, with the number of incident cases as the outcome variable and year as the primary predictor. Separate models were fitted for alcohol-related and non-alcohol-related HCC, and interaction terms were used to test for differences in temporal trends between the two groups.

Overall survival was defined as the time from HCC diagnosis (date of first MDT discussion) to death from any cause or last follow-up for those alive at last contact. Patients alive at last contact were right-censored Survival data were obtained from hospital records, state death registries, and clinical follow-up. Survival curves were generated using the Kaplan-Meier method and compared using the log-rank test. The proportional hazards assumption was assessed visually using Nelson-Aalen plots and formally tested using Schoenfeld residuals.

A multivariable Cox proportional hazards regression model was constructed to assess the independent association between AL-HCC and overall survival. Variables were selected for inclusion based on clinical relevance and statistical significance in univariable analyses. A backward, likelihood-ratio step-down procedure was employed to identify the most parsimonious model, with variables retained if their removal resulted in a significant deterioration in model fit ($p < 0.05$).

A multivariable logistic regression model was also developed to identify independent predictors of having alcohol-related HCC. The model building process followed similar principles, with variables selected based on clinical relevance and statistical significance. Model performance was assessed using the area under the receiver operating characteristic curve (AUC) and the Hosmer-Lemeshow goodness-of-fit test.

All statistical analyses were performed using Stata, version 17 (StataCorp, USA). A two-sided p-value of < 0.05 was considered statistically significant for all analyses.

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