Alcohol-associated liver disease and the COVID-19 pandemic in New Zealand—a single centre retrospective analysis

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Alcohol-associated liver disease (ALD) is a public health problem that has been exacerbated by the COVID-19 pandemic.[1,2] Several studies have reported an increase in alcohol misuse during the COVID-19 pandemic due to lockdown restrictions, and this has resulted in a corresponding rise of alcohol-related harms, which include ALD.[3,4] Some of these may stem from reduced access to abstinence services, increased anxiety, social isolation and easier access to alcohol through e-commerce.[5,6] This trend is more pronounced in younger age groups with pre-existing alcohol use disorder and may have important public health consequences for years to come.[1,3,6]

In comparison, we demonstrated that New Zealand's public health response to COVID-19, perhaps somewhat surprisingly, did not appear to increase alcohol-related harms, as we showed alcohol-related acute hospital presentations were unchanged pre-pandemic compared to matched periods during the pandemic lockdown.[7] Given the discordance with international literature, it was suggested that admissions specifically for ALD during these periods should be examined in more detail, particularly clinical severity and adverse outcomes, to determine the influence of COVID-19 lockdown restrictions on this important subgroup.

We therefore conducted a single centre retrospective case-controlled analysis in the Waitematā district, which has a catchment of approximately 650,000 people. We reviewed all ALD cases separated into alcoholic hepatitis (AH), defined as patients admitted with hepatitis related to alcohol without previously established cirrhosis, and alcoholic cirrhosis (AC), which we defined as patients presenting with alcohol-related hepatitis with a previously established diagnosis of alcohol-related cirrhosis. Patients' presentations were initially extracted from clinical coding using ICD-10-AM, Eleventh Edition, between 1 January 2019 to 2 December 2021, with each case presentation individually reviewed by three doctors. Cases were only included if acute alcohol intake was deemed to be the primary cause of admission, based on history and laboratory values. This time period includes the year immediately preceding the commencement of the COVID-19 lockdown to serve as the control group for comparison. Data on demographic, clinical and admission variables were collected. The pre-COVID group (before 25 March 2020) was compared to the COVID-19 group, and the same comparisons were also performed for two separate sub-cohorts of AH and AC. Inter-group comparisons of normally distributed continuous variables were conducted using the independent samples *t*-test, following confirmation with Shapiro–Wilk normality testing ($p>0.05$). Non-normally distributed continuous data were compared using the Mann–Whitney U test, and categorical data analysed using the $\chi 2$ and Fisher's exact tests. Local ethics approval was granted (ID: RM15128).

A total of 68 and 92 ALD presentations pre and during COVID-19 lockdown, respectively, were analysed. There were no significant differences in demographic variables between the two groups aside from lower Māori and higher Asian patient presentations during COVID-19 lockdown within the AC sub-cohort (Table 1). Clinical severity as determined by MELD, MELD-Na or Glasgow Alcoholic Hepatitis scores were not significantly different between the two groups. Child–Pugh scores were not significantly different pre and during COVID-19 lockdown for the AC sub-cohort. Despite this, there was a significant trend of less steroid treatment during COVID-19 lockdown overall ($p=0.04$). Adverse outcomes (acute kidney injury, hepatic encephalopathy, ascites, variceal bleed, ICU admission and intubation), mortality and re-hospitalisation rates were not significantly

different between the two groups overall or in the sub-cohorts.

ALD presentations in Waitematā, New Zealand were overall not significantly influenced by the COVID-19 pandemic lockdown restrictions. The trend of less steroid treatment for ALD during COVID-19 lockdown may represent concerns around additional immunosuppression during this time. The clinical severity and adverse outcomes of ALD presentations, however, were similar pre and during COVID-19 lockdown. This stands in contrast to the rise in severe ALD cases, even requiring liver transplantation, seen in other developed countries during COVID-19 lockdowns.[8–10] An Australian study has found ALD rates significantly increased since COVID-19 lockdown, but not the clinical severity.[11] Our findings may reflect survey data on alcohol consumption, which demonstrated varying consumption levels, with 47% of respondents unchanged and 34% actually decreasing alcohol consumption during the first lockdown.[12] We hypothesise that potentially social support structures implemented by the government helped to mitigate potential harms. Longer follow-up to assess alcohol-related harms is required to assess for any longer-term impact, in particular as inflationary economic pressures of these policies are translated to higher cost of living for our population.

The strength of this study is its focussed aim and high level of detail on each clinical presentation, with individual case review. The main limitation is its retrospective design and single centre nature, and lack of quantification of excess alcohol consumed. The AH cohort of patients is heterogenous and includes a range of patients without cirrhosis, from those without liver disease to those with possible advanced fibrosis. Our results are generalisable to other large New Zealand cities but not to other countries, given New Zealand's unique characteristics, including its relative geographic remoteness and diverse ethnic composition.

In conclusion, this study is consistent with our prior results and supports that New Zealand is comparatively unaffected by the negative impact of COVID-19 lockdown on alcohol-related harms seen in other countries.

Table 1: Alcohol-associated liver disease presentation characteristics. Data are presented as mean ± SD, median (IQR) or number of patients (% of patients).

Characteristic	All			Alcoholic hepatitis			Alcoholic cirrhosis		
	Pre-COVID (68)	COVID-19 (n=92)	p	Pre-COVID (n=28)	COVID-19 (n=45)	p	Pre-COVID (n=40)	COVID-19 (n=47)	p
Age (years)	46.3±13.2	50.2±11.7	0.33	39.5±12.3	48.0±11.3	0.07	52.1±12.4	48.2±7.3	0.77
Male sex	44 (64.7%)	67 (72.8%)	0.30	19 (67.9%)	32 (71.1%)	0.80	25 (62.5%)	35 (74.5%)	0.25
Ethnicity									
European	51 (75.0%)	76 (82.6%)	0.13	16 (57.1%)	36 (80.0%)	0.25	35 (87.5%)	40 (85.1%)	0.02
Māori	6 (8.8%)	1 (1.1%)		2 (7.1%)	1 (2.2%)		4 (10.0%)	0 (0.0%)	
Pacific peoples	1 (1.5%)	1 (1.1%)		1 (3.6%)	1 (2.2%)		0 (0.0%)	0 (0.0%)	
Asian	9 (13.2%)	14 (15.2%)		8 (28.6%)	7 (15.6%)		1 (2.5%)	7 (14.9%)	
Other	1 (1.5%)	0 (0.0%)		1 (3.6%)	0 (0.0%)				
Length of admission (days)	6 (2–10)	6 (2–10)	0.93	10 (7–16)	12 (7–15)	0.82	5 (1–9)	6 (3–14)	0.88
Cost of admission (NZ$)	8,230 (3,300–13,400)	8,190 (3,400–16,820)	0.48	8,130 (3,010–22,920)	9,100 (6,800–17,110)	0.87	7,300 (2,600–12,880)	8,940 (4,630–25,990)	0.49
MELD score	16 (13–21)	17 (12–21)	0.76	15 (8–21)	17 (12–20)	0.29	17 (13–21)	18 (14–24)	0.75
MELD-Na score	18 (14–26)	20 (14–24)	0.98	18 (8–26)	20 (15–23)	0.99	18 (14–25)	21 (13–25)	0.83
GAHS score	7 (6–8)	7 (6–8)	0.97	6 (5–8)	7 (6–8)	0.51	7 (6–8)	6 (6–7)	0.56
Child–Pugh score	-	-	-	-	-	-	7 (6–8)	6 (6–9)	0.66

Table 1 (continued): Alcohol-associated liver disease presentation characteristics. Data are presented as mean ± SD, median (IQR) or number of patients (% of patients).

Charlson Comorbidity Index	1 (0–2)	1 (0–2)	0.15	1 (0–1)	1 (0–2)	0.80	1 (0–2)	1 (0–1)	0.07
Previous abstinence	37 (55.2%)	54 (58.7%)	0.75	13 (46.4%)	26 (57.8%)	0.47	19 (40.4%)	28 (59.6%)	>0.99
Hazardous drinking	59 (86.8%)	81 (88.0%)	0.81	26 (92.9%)	40 (88.9%)	0.70	33 (82.5%)	41 (87.2%)	0.56
COVID-19 infection	0 (0%)	13 (14.1%)	<0.01	0 (0%)	4 (8.9%)	0.27	0 (0%)	9 (19.1%)	0.01
Adverse outcomes									
Acute kidney injury	11 (16.4%)	26 (28.3%)	0.09	5 (17.9%)	11 (24.4%)	0.57	6 (15.0%)	15 (31.9%)	0.08
Hepatic encephalopathy	14 (20.6%)	22 (23.9%)	0.70	3 (10.7%)	6 (13.3%)	>0.99	11 (27.5%)	16 (34.0%)	0.64
Ascites	40 (58.8%)	49 (53.3%)	0.52	8 (28.6%)	16 (35.6%)	0.61	32 (80.0%)	33 (70.2%)	0.33
Variceal bleed	4 (5.9%)	7 (7.6%)	0.76	2 (7.1%)	2 (4.4%)	0.64	2 (5.0%)	5 (10.6%)	0.45
Endoscopy	19 (27.9%)	29 (31.5%)	0.73	5 (17.9%)	13 (28.9%)	0.40	14 (35.0%)	16 (34.0%)	0.93
Steroid treatment	7 (10.3%)	2 (2.2%)	0.04	7 (25.0%)	0 (0.0%)	<0.001*	0 (0.0%)	2 (4.3%)	0.50
Intensive care unit admission	1 (1.5%)	4 (4.3%)	0.40	1 (3.6%)	2 (4.4%)	>0.99	0 (0.0%)	2 (4.3%)	0.50
Intubation	1 (1.5%)	2 (2.2%)	>0.99	1 (3.6%)	1 (2.2%)	>0.99	0 (0.0%)	1 (2.1%)	>0.99
Continuous veno-venous haemodialysis	0 (0.0%)	0 (0.0%)	>0.99	0 (0.0%)	0 (0.0%)	>0.99	0 (0.0%)	0 (0.0%)	>0.99
Mortality									
Inpatient mortality	5 (7.4%)	7 (7.6%)	>0.99	0 (0.0%)	3 (6.7%)	0.28	4 (10.0%)	3 (6.4%)	0.70

Table 1 (continued): Alcohol-associated liver disease presentation characteristics. Data are presented as mean ± SD, median (IQR) or number of patients (% of patients).

30-day mortality	5 (7.4%)	7 (7.6%)	>0.99	0 (0.0%)	3 (6.7%)	0.28	5 (12.5%)	4 (8.5%)	0.73
1-year mortality	14 (20.5%)	19 (20.7%)	>0.99	4 (14.3%)	4 (8.9%)	0.47	10 (25.0%)	15 (31.9%)	0.64
Re-hospitalisation									
30-day re-hospitalisation	24 (37.5%)	32 (37.2%)	>0.99	8 (28.6%)	11 (24.4%)	0.79	16 (40.0%)	21 (44.7%)	0.67
90-day re-hospitalisation	33 (51.6%)	46 (53.5%)	0.87	11 (39.3%)	17 (37.8%)	>0.99	22 (55.0%)	29 (61.7%)	0.66

COMPETING INTERESTS
None.

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