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Supporting Information

Supplementary methods and results

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Appendix to: O'Beirne J, Skoien R, Leggett BA, et al. Diabetes mellitus and the progression of non-alcoholic fatty liver disease to decompensated cirrhosis: a retrospective cohort study. *Med J Aust* 2023; doi: 10.5694/mja2.52104.

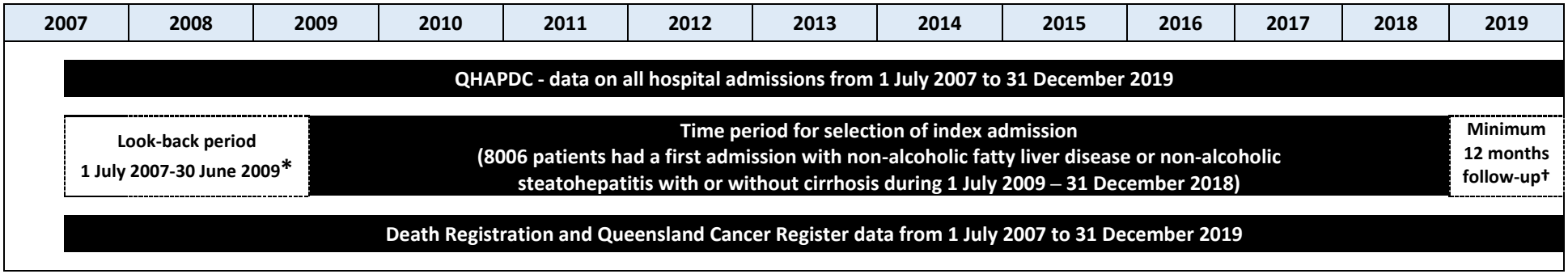
Supplementary methods

1. Selection of cases and index admission

A case of non-alcoholic fatty liver disease (NAFLD) or non-alcoholic steatohepatitis (NASH) with or without cirrhosis was defined by at least one hospitalisation with any of the following International Classification of Diseases 10th revision – Australian modification (ICD-10-AM) codes as 'principal diagnosis' or 'other diagnosis': NAFLD (K76.0), NASH (K75.8) or other and unspecified cirrhosis of liver (K74.6). As Hagstrom et al[1] and Petta et al,[2] we excluded patients who ever had other liver diseases recorded in a hospitalisation during 1 July 2007 to 31 December 2019, namely alcoholic liver disease, viral hepatitis, autoimmune liver disease, hemochromatosis, Wilson's disease, alpha-1-antitrypsin deficiency, Budd-Chiari syndrome, chronic hepatitis, unspecified, secondary/unspecified biliary cirrhosis.

We identified patients with a first hospital admission with NAFLD/NASH during 1 July 2009 to 31 December 2018 (index admission), discharged alive and survived for at least 30 days. We excluded patients who had a code for liver decompensation (ascites, jaundice, hepatic encephalopathy, or oesophageal variceal bleeding) prior to 1 July 2009. Patients with a history of liver transplant or hepatocellular carcinoma prior to the index admission were also excluded. All patients had a look-back period of minimum two years (1 July 2007 to 30 June 2009) and a minimum follow-up period of one year (1 January 2019 to 31 December 2019; **Figure 1**). As about 50% of patients with decompensated cirrhosis die within two years, the look-back period of two years is likely to identify the first admission for decompensation in most cases. Patients with an episode of decompensation (hospital admission due to cirrhosis) prior to the two-year look-back period are likely to have reverted to a compensated stage. The minimum follow up period of one year is adequate for capturing relevant outcome events (death, hospital admissions).[3]

Figure 1. Timeline for case ascertainment and data availability



QHAPDC = Queensland Hospital Admitted Patient Data Collection.

*Minimum look-back period for index admissions in 2009 was 2 years; † Minimum follow-up period for index admissions in 2018 was 12 months

Data sources

Socio-demographic data obtained from the Queensland Hospital Admitted Patient Data Collection (QHAPDC) included: age group at admission, gender, marital status, country of birth, place of residence, interpreter required, and Indigenous status (patients were coded as Indigenous if identified in at least one of their records within the study period). Place of residence was categorized according to the level of remoteness of residence (Accessibility and Remoteness Index of Australia, ARIA+)[4] and socioeconomic advantage and disadvantage status (Index of Relative Advantage and Disadvantage).[5]

The data includes all admitted patient separations from public hospitals and private hospitals in the state of Queensland. A separation can be a formal separation (e.g. discharge, transfer) or a statistical separation when there is a change in episode type (e.g. when patients are transferred from the emergency department to a ward). The latter definition was used. When patients were transferred within the same hospital (e.g. from the emergency department to award) or to another hospital, we considered these episodes of care as one hospital stay. Hospital sector for each hospital stay was categorized as 'public hospital only' or 'private only or mix'.

Comorbidity at index admission was measured using the Charlson Comorbidity Index[6] using validated coding algorithms.[7] Major cardiovascular events included the four items from the Charlson Comorbidity Index, namely: (item 1) myocardial infarct, (2) congestive heart failure, (3) peripheral vascular disease, and (4) cerebrovascular disease. Diabetes mellitus included two items, namely: (10) diabetes mellitus, and (13) diabetes mellitus with end organ damage. Obesity and hypertension, not included as items in the Charlson Comorbidity Index, were identified using the Elixhauser co-morbidities grouping using validated coding algorithms.[7] Disorders of lipoprotein metabolism was identified by the presence of IDC-10-AM code for disorders of lipoprotein metabolism and other lipidaemias (E78). Features of the metabolic syndrome included in the analysis comprised obesity, disorders of lipoprotein metabolism and other lipidaemias, diabetes mellitus, and hypertension.

The primary outcome was progression to decompensated cirrhosis identified by the first decompensation event identified by having hospital admission with an ICD-10-AM code for ascites, hepatic encephalopathy, or oesophageal variceal bleeding (as described in **Table 1**) as 'principal diagnosis' or 'other diagnosis'.

2. Accuracy of ICD-10-AM codes for identification of patients and study measurements

The accuracy of identification of patients with NAFLD/NASH (grouped ICD-10-AM codes) in the QHAPDC is reported to have a 91.2% positive predictive value (PPV), 84.4% negative predictive value (NPV) (**Table 1**).[8] While codes for NAFLD/NASH had 97.7% specificity, they underestimated the prevalence of NAFLD/NASH by 42.9% (sensitivity 57.1%). The accuracy of identification of patients with other and unspecified cirrhosis of liver has been reported to have a 96% PPV and 33% NPV.[9] Regarding the accuracy of risk factors examined here, Hayward et al reported that while the specificity of ICD-10-AM codes for obesity was high (95.9%), codes underestimated the prevalence of by 45.3%.[8]

Table 1. ICD-10-AM codes used for identification of patients with non-alcoholic fatty liver disease or non-alcoholic steatohepatitis with or without cirrhosis and of decompensated cirrhosis

ICD-10-AM code		PPV* (95% CI)	NPV* (95% CI)
NAFLD/NASH			
K75.8	Other specified inflammatory diseases of the liver (non-alcoholic steatohepatitis)	0.82 (0.79-0.85)	0.92 (0.80-0.97)
K76.0	Fatty (change of) liver, not elsewhere classified	0.72 (0.71-0.74)	0.90 (0.53-0.99)
	Grouped K75.8, K76.0	0.84 (0.81-0.89)	0.91 (0.81-0.96)
NAFLD/NASH-related cirrhosis			
K74.6	Other and unspecified cirrhosis of liver	0.96 (0.93-0.99)	0.33 (0.28-0.38)
Cirrhosis decompensation			
I85.0	Oesophageal varices with bleeding	1	0.58 (0.54-0.62)
I98.3	Oesophageal varices with bleeding in diseases classified elsewhere	1	0.60 (0.55-0.64)
R18	Ascites	0.97 (0.94-0.99)	0.76 (0.71-0.80)
G31.2	Degeneration of nervous system due to alcohol	0.37 (0.21-0.55)	0.74 (0.70-0.78)
G93.4	Encephalopathy, unspecified	0.71 (0.54-0.84)	0.77 (0.73-0.80)
	Grouped hepatic encephalopathy (G31.2, G93.4)	0.55 (0.43-0.67)	0.78 (0.74-0.81)

ICD-10-AM = International Classification of Diseases 10th edition – Australian Modification; NAFLD = Non-alcoholic fatty liver disease; NASH = Non-alcoholic steatohepatitis.
* Positive predicted values (PPV) and negative predicted values (NPV) were obtained from a study reported by Hayward et al.[8]

3. Supplementary data analysis

Multivariable Cox regression analysis reported in terms of hazard ratios (HRs) with associated 95% confidence intervals (CIs), was used to assess the differences in cumulative incidence of decompensated cirrhosis according to selected sociodemographic and clinical characteristics. We built two models, namely: (**Model 1**) including patients without cirrhosis at index admission, and (**Model 2**) patients with cirrhosis. The variables describing the patients' sociodemographic and clinical characteristics at index admission included in the main article **Box 1** were taken into account for these models except 'Charlson comorbidity groups' and 'Metabolic syndrome (features)'. These variables were not considered for inclusion in the models because they contained information about individual item variables that were considered for inclusion (eg. diabetes mellitus is an item in the Charlson comorbidity index and the Elixauser comorbidities).[7] Diabetes mellitus was included as an indicator of comorbidity risk, as it is common in this patient population and is associated with higher risk of morbidity.[10] The individual variables obesity, disorders of lipoprotein metabolism and other lipidaemias, diabetes mellitus, and hypertension were considered for inclusion as representing the measure for the metabolic syndrome.

First, unadjusted HRs from Cox regression are presented. Secondly, a Least Absolute Shrinkage and Selection Operators (LASSO) penalised regression cox proportional hazards model was used to identify a parsimonious model including a set of variables that had the strongest association with the outcome (decompensated cirrhosis).[11] The LASSO procedure was used due to the high number of predictors and potentially complex patterns of collinearity among predictor variables. Variables included in the model were checked to ensure that they adhered to the assumption of proportional hazards over time (Shoenfeld residuals). The vce(robust) option was used to obtain robust standard errors for the parameter estimates to control for mild violation of underlying assumptions.

In **Model 1** including patients without cirrhosis at index admission, portal hypertension was not considered for inclusion in the model because the numbers were too small (33 patients (7 progressed to decompensated cirrhosis) had a code for portal hypertension). The following variables were included in the final model: age group, diabetes mellitus, major cardiovascular events, cancer (excluding hepatocellular carcinoma), hypertension, hospital sector, remoteness of residence, interpreter required, and Indigenous status. We have explored whether the inclusion of the interaction between diabetes mellitus and obesity affected the risk of decompensation. Including the interaction term diabetes mellitus and obesity in the final model did not alter the adjusted hazard ratios (aHR), the interaction term was not

statistically significant (p=0.50) and did not add any extra information to the model (Akaike information criterion (AIC)= 2412 in the model with the interaction term and AIC=2411 in the model without). Therefore, the interaction term of diabetes mellitus and obesity was not included in the model.

In **Model 2** including patients with cirrhosis at index admission, portal hypertension was considered for inclusion in the model. The following variables were included in the final model: age group, diabetes mellitus, cancer (excluding hepatocellular carcinoma), portal hypertension, hypertension, obesity, rurality of residence, socioeconomic status, and country of birth. Similar to Model 1, the interaction term was not statistically significant (p=0.22), did not add any extra information to the model (AIC=2583 in the model with the interaction term and AIC=2582 in the model without), and therefore was not included in the model.

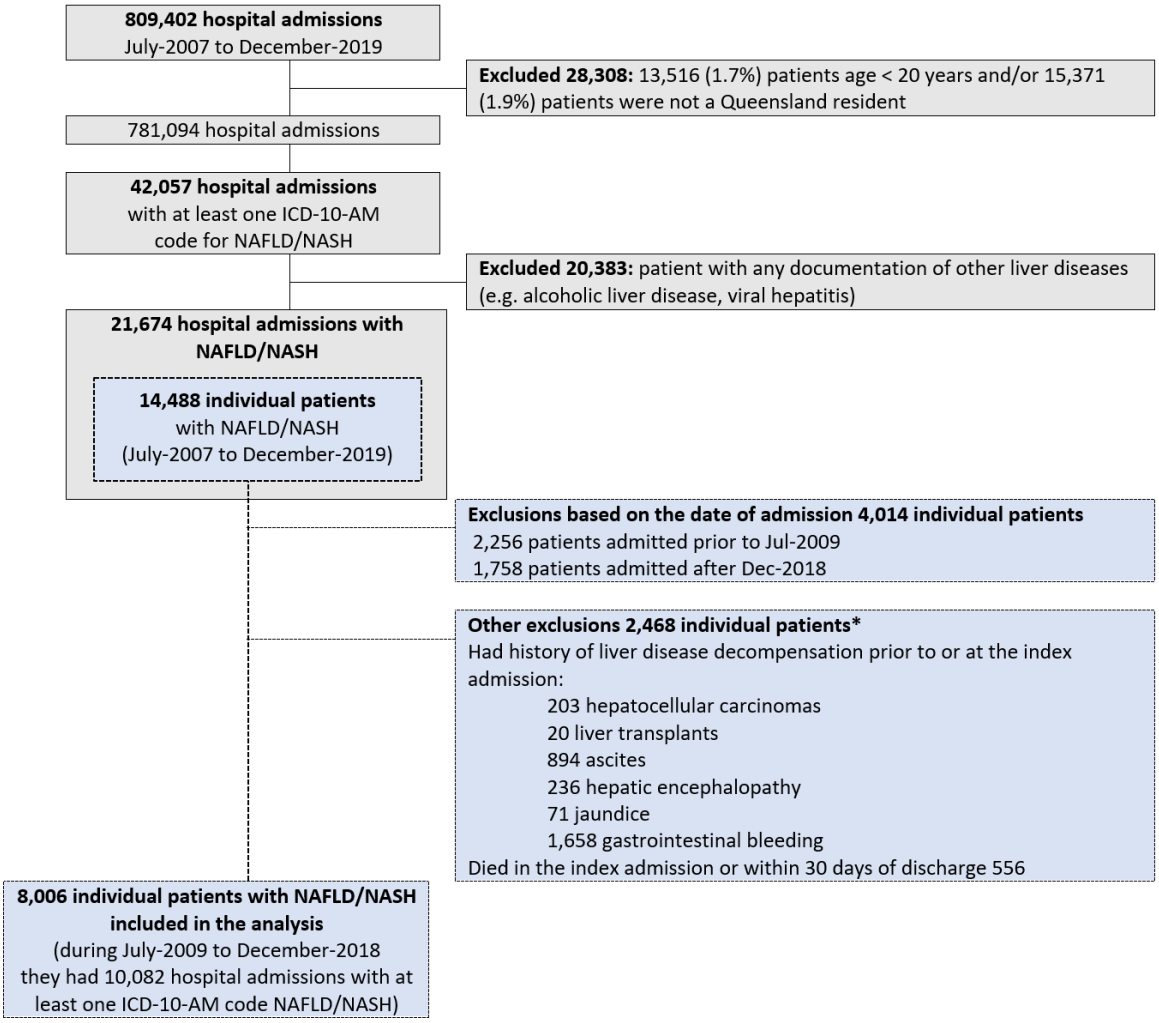
Sensitivity analyses was conducted to assess whether hospital admissions prior to the index admission was associated with progression to decompensated cirrhosis. Restricting the analyses to patients who had at least one prior admission, the analyses were repeated including the variable 'prior admission with a recorded diagnosis related to severity of liver disease' (namely, major cardiovascular events, diabetes mellitus, portal hypertension, and varices).

Supplementary results

4. Cohort ascertainment

The study cohort was identified via a comprehensive list of ICD-10-AM diagnosis and procedure codes provided to the Statistical Analysis Linkage Unit of the Queensland Department of Health. The dataset obtained for this study included 809,402 hospital admissions from 1 July 2007 to 31 December 2019. A total of 28,308 admissions were excluded (13,516 (1.7%) patients age < 20 years; 15 371 (1.9%) patients were not Queensland residents). There were 42,057 hospital admissions in which patients were >20 years, resided in Queensland, and had at least one ICD-10-AM code for NAFLD/NASH. After excluding 20,383 admissions of patients with any documentation of other liver diseases (e.g. alcoholic liver disease, viral hepatitis),[3] there were 21,674 hospital admissions with NAFLD/NASH from 14,488 unique individuals (**Figure 2**). We excluded 2256 patients admitted prior to 1 July 2009 and 1758 patients admitted after 31 December 2018, patients with liver transplants, hepatocellular carcinoma, or liver decompensation prior to the index admission, and 2468 patients who died in hospital or within 30 days of discharge. A total of 8006 individual patients were included in the analysis.

Figure 2. Flowchart for case ascertainment



Non-alcoholic fatty liver disease (NAFLD) or non-alcoholic steatohepatitis (NASH) with or without cirrhosis; International Classification of Diseases 10th revision – Australian modification (ICD-10-AM).
* Patients may been excluded for more than one reason.

Table 2. Cox regression analysis of factors associated with progression to decompensated cirrhosis among patients with non-alcoholic fatty liver disease or non-alcoholic steatohepatitis with cirrhosis at index admission (1106 patients)

	No progression	Progression	Hazard ratio (95%CI)	Adjusted hazard ratio (95%CI)*
Number of people	906	200		
Gender				
Male	461 (50.9%)	105 (52.5%)	Reference	N/A
Female	445 (49.1%)	95 (47.5%)	0.9 (0.7-1.2)	
Age group				
20-39 years	38 (4.2%)	8 (4.0%)	Reference	Reference
40-49 years	66 (7.3%)	8 (4.0%)	0.6 (0.2-1.5)	0.6 (0.2-1.6)
50-59 years	165 (18.2%)	39 (19.5%)	1.1 (0.5-2.2)	1.0 (0.5-2.1)
60-69 years	255 (28.1%)	69 (34.5%)	1.3 (0.6-2.6)	1.2 (0.6-2.5)
70 years and over	382 (42.2%)	76 (38.0%)	1.3 (0.6-2.5)	1.2 (0.6-2.5)
Marital status				
Married/De Facto	387 (52.2%)	111 (61.7%)	Reference	N/A
No partner	355 (47.8%)	69 (38.3%)	0.9 (0.6-1.2)	
Missing data	164	20		
Country of birth				
Australia	695 (76.9%)	155 (77.5%)	Reference	Reference
New Zealand, Oceania/Antarctica	39 (4.3%)	6 (3.0%)	0.7 (0.3-1.6)	0.8 (0.4-1.7)
Europe and Americas	133 (14.7%)	29 (14.5%)	1.0 (0.7-1.5)	1.0 (0.7-1.5)
Asia, Africa, Middle East	37 (4.1%)	10 (5.0%)	1.1 (0.6-2.1)	1.2 (0.6-2.4)
Missing data	<5	0		
Indigenous status				
Non-Indigenous	857 (94.7%)	190 (95.0%)	Reference	N/A
Indigenous	48 (5.3%)	10 (5.0%)	0.9 (0.5-1.7)	
Missing data	<5	0		
Remoteness of residence (ARIA +)				
Major city	553 (61.0%)	119 (59.5%)	Reference	Reference
Inner regional	190 (21.0%)	53 (26.5%)	1.3 (0.9-1.8)	1.4 (1.0-1.2)
Outer regional/remote/very remote	163 (18.0%)	28 (14.0%)	0.9 (0.6-1.3)	1.0 (0.6-1.5)
Socioeconomic status (IRSAD)				
Q1 most affluent	142 (15.7%)	28 (14.0%)	Reference	Reference
Q2	146 (16.1%)	44 (22.0%)	1.6 (1.0-2.5)	1.5 (0.9-2.5)
Q3	165 (18.2%)	31 (15.5%)	1.1 (0.7-1.8)	1.1 (0.6-1.8)
Q4	216 (23.8%)	42 (21.0%)	1.1 (0.7-1.8)	1.1 (0.6-1.8)
Q5 most disadvantaged	237 (26.2%)	55 (27.5%)	1.2 (0.8-1.9)	1.2 (0.7-1.8)
Hospital sector				
Public	551 (60.8%)	112 (56.0%)	Reference	N/A
Private or mix	355 (39.2%)	88 (44.0%)	1.0 (0.8-1.3)	
Interpreter required †	20 (2.3%)	7 (3.6%)	0.6 (0.3-1.3)	N/S
Portal hypertension	93 (10.3%)	33 (16.5%)	1.9 (1.3-2.8)	1.8 (1.3-2.7)
Charlson comorbidity groups				
CCI=0	361 (39.8%)	80 (40.0%)	Reference	N/A
CCI=1	157 (17.3%)	29 (14.5%)	1.1 (0.7-1.7)	
CCI=2	205 (22.6%)	49 (24.5%)	1.3 (0.9-1.9)	
CCI=3	183 (20.2%)	42 (21.0%)	1.6 (1.1-2.4)	

Metabolic syndrome (features)				
Nil	436 (48.1%)	91 (45.5%)	Reference	N/A
1	310 (34.2%)	66 (33.0%)	1.2 (0.9-1.7)	
2	133 (14.7%)	39 (19.5%)	1.7 (1.2-2.5)	
3-4	27 (3.0%)	< 1.0% [g]	0.8 (0.3-2.1)	
Diabetes mellitus	387 (42.7%)	99 (49.5%)	1.5 (1.1-2.0)	1.5 (1.1-2.0)
Obesity	138 (15.2%)	37 (18.5%)	1.4 (1.0-1.9)	1.3 (0.9-1.9)
Hypertension	124 (13.7%)	20 (10.0%)	0.8 (0.5-1.3)	0.6 (0.4-1.0)
Major adverse cardiovascular events ‡	136 (15.0%)	14 (7.0%)	0.7 (0.4-1.1)	N/A
Renal disease	85 (9.4%)	10 (5.0%)	0.8 (0.4-1.4)	N/A
Chronic pulmonary disease	53 (5.8%)	8 (4.0%)	0.7 (0.4-1.5)	N/A
Extrahepatic cancers	76 (8.4%)	21 (10.5%)	1.9 (1.2-3.1)	1.8 (1.1-2.9)
Disorders of lipoprotein metabolism	11 (1.3%)	< 1.0% [g]	0.4 (0.1-2.9)	N/S

CI = confidence interval; ARIA + = Accessibility and Remoteness Index of Australia;[6] IRSAD = Index of Relative Socioeconomic Advantage and Disadvantage;[7] Quintile (Q); Charlson Comorbidity index (CCI); N/A = not applicable (variable not included in the final model).

* Adjusted for age group, country of birth, rurality of residence, socioeconomic status, portal hypertension, diabetes mellitus, obesity, hypertension, and extrahepatic cancers (include any cancer except hepatocellular carcinoma).

† Missing data for 36 patients (no progression: 29; progression 7).

‡ Four items from the Charlson comorbidity index: myocardial infarction, congestive heart failure, peripheral vascular disease, cerebrovascular disease.

§ Any cancer apart from hepatocellular carcinoma.

5. Sensitivity analyses

A total of 6755 patients (86%) had at least one hospital admission prior to the index admission (5798 patients without cirrhosis and 977 patients with cirrhosis). Among 5798 patients without cirrhosis, when the variable 'prior admission with a recorded diagnosis related to severity of liver disease' was included in the model, it was significantly associated with the outcome (aHR, 2.0; 95%CI 1.2-3.3), the variable diabetes mellitus at index admission had a lower hazard ratio when compared to the main analysis (changing from aHR, 2.8; 95%CI 2.0-3.9 in the main analysis to 1.8; 95%CI 1.1-3.0), and the other hazard ratios were similar to the main analysis (data not shown). Moreover, this variable 'prior admission with a recorded diagnosis related to severity of liver disease' did not add any extra information to the model (AIC=2059 with and AIC=2063 without this variable in the model). Among 977 patients with cirrhosis at index admission, when the variable 'prior admission with a recorded diagnosis related to severity of liver disease' was included in the model, it was significantly associated with the outcome (aHR, 1.7; 95%CI 1.2-2.5), the variable diabetes mellitus at index admission was no longer associated with the outcome (aHR, 1.2; 95%CI 0.8-1.6), and the other hazard ratios were similar to the main analysis (data not shown). This variable did not add any extra information to the model (AIC=2224 with and AIC=2229 without this variable in the model). Moreover, there was a significant correlation between the variables 'diabetes mellitus at index admission' and 'prior admission with a recorded diagnosis related to severity of liver disease' ($r=0.64$, $p<0.001$). Therefore, the variable 'prior admission with a recorded diagnosis related to severity of liver disease' was not included in the model.

Table 3. Distribution of cancer type according to progression to decompensated cirrhosis among patients with a recorded diagnosis of cancer (excluding hepatocellular carcinoma; 339 patients)

	No progression	Progression	Total
Number of people	**298**	**41**	**339**
Cancer type among all patients with cancer at the index admission*			
C00-C14 Lip, oral cavity and pharynx	<1.0% †	<1.0% †	<1.0% †
C15-C26 Digestive organs	117 (39.3%)	28 (68%)	145 (42.8%)
C30-C39 Respiratory system	16 (5.4%)	0	16 (4.7%)
C40-C41 Bone and articular cartilage	<1.0% †	0	<1.0% †
C43-C44 Melanoma and other skin	7 (2.3%)	0	7 (2.1%)
C45-C49 Mesothelial and soft tissue	<1.0% †	0	<1.0% †
C50-C50 Breast	24 (8.1%)	<5% †	26 (7.7%)
C51-C58 Female genital organs	12 (4.0%)	<5% †	14 (4.1%)
C60-C63 Male genital organs	17 (5.7%)	<10% †	21 (6.2%)
C64-C68 Urinary tract	17 (5.7%)	<5% †	18 (5.3%)
C69-C72 Eye, brain and other central nervous system	9 (3.2%)	0	9 (2.7%)
C73-C75 Thyroid and other endocrine glands	<1.0% †	0	<1.0% †
C76-C80 Ill-defined, secondary and unspecified sites	9 (3.0%)	0	9 (2.7%)
C81-C96 Lymphoid and haematopoietic	64 (21.5%)	<10% †	68 (20.1%)
Cancer type among patients with cancer of the digestive organs*	**117**	**28**	**145**
C22.1 Cholangiocarcinoma (intra hepatic bile duct)	<5.0% †	<5% †	7 (4.8%)
C23 Gallbladder	<5.0% †	0	<5.0% †
C24 Other parts of biliary tract	<5.0% †	<5% †	<5.0% †
C25 Pancreas	20 (17.1%)	5 (18%)	25 (17.2%)
C15 Oesophagus	<5.0% †	0	<5.0% †
C16 Stomach	7 (6.0%)	<10% †	9 (6.2%)
C17 Small intestine	5 (4.3%)	0	5 (3.4%)
C18-C21 Colorectal	70 (59.8%)	18 (64%)	88 (60.7%)
C26 Ill-defined digestive organs	<1.0% †	0	<1.0% †

* International Classification of Diseases 10th edition – Australian Modification (ICD-10-AM) codes as 'primary diagnosis' or 'other diagnosis'; † Numbers suppressed to preserve anonymity.

Table 4. Cox regression analysis examining the association between colorectal cancer and progression to decompensated cirrhosis among patients with non-alcoholic fatty liver disease or non-alcoholic steatohepatitis stratified according to cirrhosis status at index admission

	No cirrhosis	Cirrhosis
Number of people	**6900**	**1106**
	Hazard ratio (95%CI)	**Hazard ratio (95%CI)**
	9.9 (5.1-19.2)	1.6 (0.5-3.2)
	Adjusted hazard ratio (95%CI) [b]	**Adjusted hazard ratio (95%CI)** [c]
	10.5 (5.2- 21.2)	1.4 (0.7-2.8)

CI = confidence interval

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