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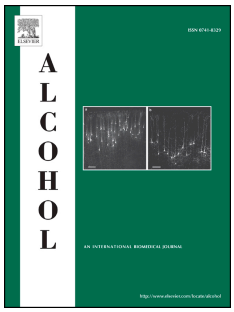

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Andrew Thompson, Paul Richardson, Munir Pirmohamed, Lynn Owens

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Alcohol related brain injury: an unrecognised problem in acute medicine

Andrew Thompson[1,2], Paul Richardson[2,3], Munir Pirmohamed[1,2], Lynn Owens[1,2,3]

[1] Wolfson Centre for Personalised Medicine, Institute of Translational Medicine, University of Liverpool, United Kingdom

[2] Liverpool Centre for Alcohol Research, University of Liverpool, United Kingdom

[3] Gastroenterology and Hepatology, Royal Liverpool University Hospital Trust, Ward 5z Link, United Kingdom

Address for correspondence:

Andrew Thompson

Wolfson Centre for Personalised Medicine

Institute of Translational Medicine

Block A Waterhouse Building

University of Liverpool

Liverpool L69 3GL

United Kingdom

Telephone: +44 151 795 5390

Email: andrew.thompson@liverpool.ac.uk

ABSTRACT

Alcohol-related brain injury (ARBI) is an unrecognised and therefore untreated consequence of alcohol use disorder. Here, we explore 12-month period prevalence of alcohol-related brain injury (ARBI) in alcohol use disorder patients. **I**npatients aged ≥18 years reviewed by the Alcohol Care Team's Specialist Nurses between 1 April 2017 and 31 March 2018 were eligible for the study (n=1276). Screening identified a high-risk subset of patients who matched at least one of the following: 1) more than three alcohol-related admissions in one year; 2) two alcohol related admissions in any given 30-day period; 3) patient or their significant other had concerns regarding cognition. The high-risk patients were assessed for evidence of ARBI using the Montreal Cognitive Assessment Tool (MoCA). The primary measure of interest was MoCA ≤23. Analysis was conducted between subgroups of the study population to identify prevalence rate ratios for matching the high-risk screening criteria, and MoCA ≤23 in high-risk patients. 205 patients were identified as high risk for ARBI. The period prevalence rates in this high-risk group for patients with a MoCA ≤23 was 36.1%. Those under the age of 35 years were significantly less likely to match the high-risk criteria. Patients staying in hostels or homeless were more likely to match the high-risk criteria and were also at increased risk of MoCA ≤23 compared with those living with family members. In summary, ARBI is common in patients with AUD attending acute hospitals. ARBI is often not diagnosed, and thus further work is required to improve screening for, and identification of, these patients to develop evidence-based clinical pathways which optimise care.

KEYWORDS: Alcohol related brain injury; Alcohol comorbidity; Alcohol liaison team

HIGHLIGHTS

- Through stratification, 205 patients were classified at increased risk of ARBI.
- We estimated the period prevalence of ARBI in this high-risk group to be 36.1%.
- Factors associated with ARBI development and progression remain knowledge gaps.

INTRODUCTION

The effects of excessive and prolonged alcohol consumption on the brain are well-recognised, but not completely understood (Abrahao, Salinas, & Lovinger, 2017). A systematic review of brain injury identified neurodegenerative changes in heavy drinkers, but also highlighted the potential for reversibility with sustained abstinence (Bühler & Mann, 2011). Alcohol Related Brain Injury (ARBI) is an umbrella term for a number of neurodegenerative complications caused by heavy drinking. Features of ARBI include executive dysfunction and behavioural changes (Saxton, Munro, Butters, Schramke, & McNeil, 2000), but there are no internationally agreed standardised criteria for determining the presence of ARBI, which is further confounded by variation in the terms used to describe the condition; these include 'alcohol-related brain damage', 'alcohol-related dementia' and 'alcoholic amnesia syndrome'. ARBI is used in this manuscript to describe cognitive impairment in the absence of other conditions known to affect cognition such as Wernicke's or hepatic encephalopathy.

ARBI typically leads to a vortex of physical and psychological harms often necessitating hospital admission, and creating distress for the patient and their families (Wilson et al., 2012). Detection of ARBI in alcohol use disorder (AUD) patients at the earliest opportunity is therefore important in facilitating more appropriate, patient-centred care. However, because ARBI is generally unrecognised and therefore untreated, it is unknown how many patients with ARBI attend alcohol treatment services. Furthermore, lack of recognition of ARBI may lead to the patient being erroneously labelled as 'non-compliant' or 'problematic' due to their diminished ability to understand, remember and therefore comply with clinical advice and treatment.

Given the gaps in knowledge and clinical resources for recognising and managing the ARBI population, we have used the Montréal Cognitive Assessment Tool (MoCA) tool to identify ARBI in AUD patients attending an acute inner city hospital over a 12-month period, with a score below cut-off considered as potential evidence of underlying ARBI (Angermann et al., 2017; Ball, Carrington, Stewart, & investigators, 2013; Carson, Leach, & Murphy, 2018; Luis, Keegan, & Mullan, 2009). MoCA is a brief cognitive screening instrument (Nasreddine et al., 2005) that has been used in different clinical settings because of its high sensitivity and specificity, and test–retest reliability of 0.92, for detecting cognitive-related defects (Copersino et al., 2009; Luis et al., 2009; Popović, Šerić, & Demarin, 2007). A considerable body of evidence suggests that the MoCA is superior to the Mini-Mental State Examination (MMSE), in detecting cognitive impairment (Hoops et al., 2009; Popović et al., 2007). Specifically, the MoCA assesses the following cognitive domains: visual-spatial abilities; executive function; language and phonemic fluency; verbal abstraction; short-term memory recall; attention; concentration; working memory; and orientation. It can be administered within 10 minutes and cut-off scores are used for detecting and categorising impairment. The specific aims of our study were to: i) explore 12-month period prevalence of ARBI in AUD patients; and ii) define if these rates are influenced by patient characteristics and/or behaviour.

MATERIAL AND METHODS

Study population and data collection

Data for this cohort study was sourced from an acute hospital in the UK, which serves a local population of approximately 465,000. The study population consisted of all inpatients aged 18 years and over who were reviewed by the Alcohol Care Team's Specialist Nurses (ASN) between 1 April 2017 and 31 March 2018. A retrospective dataset was completed by an independent administrator using patient case notes and hospital electronic information systems. Data included demographics, Alcohol Use Disorders Identification Test (AUDIT) (Babor, Higgins-Biddle, Saunders, & Monteiro,

2001), Severity of alcohol dependence questionnaire (SADQ) score (Stockwell, Hodgson, Edwards, Taylor, & Rankin, 1979), postcode data for calculating index of multiple deprivation (IMD) outcomes, and smoking. Access to the data for this project was granted by the Royal Liverpool University Hospital NHS Trust Official Trust Audit Board (ID: A03301).

MoCA screening

As part of a review by the ASN, each patient matching at least one the following was considered high risk and assessed for evidence of ARBI using the MoCA tool: 1) More than three alcohol-related admissions in one year; 2) Two alcohol related admissions in any given 30-day period; and 3) Patient or their significant other had concerns regarding cognition. Patients were not screened with MoCA if they had a breath alcohol content >0, co-morbid neurological or psychiatric diseases that might explain impaired cognition (e.g. hepatic encephalopathy, previous known stroke/TIA, dementia, Alzheimer's disease), cannabis or other illicit drug use in previous 48 hours, or prescribed any sedative in previous 48 hours. The primary measure of interest was MoCA ≤23, which triggered a referral to psychiatric liaison for further assessment. Utilisation of this cut-off was based on evidence of optimal diagnostic accuracy from previous work (Angermann et al., 2017; Ball et al., 2013; Carson et al., 2018; Luis et al., 2009).

Data Analysis

The period prevalence rate of MoCa ≤23 was estimated for the high-risk subset of patients ("high risk cohort"). Sensitivity analysis was performed to investigate period prevalence using cut-offs proposed in other condition-specific research of ≤21 (Salvadori et al., 2013) and ≤25 (Copersino et al., 2009; Nasreddine et al., 2005). The prevalence of patients in the whole cohort who met at least one of the criteria for high risk of ARBI was also estimated.

Relative rates of MoCA ≤23 in the high-risk cohort were estimated in univariable analysis stratified by gender, age bands, individual-level IMD 2015 quintiles, and smoking status. The IMD scores were skewed towards higher deprivation, and therefore the quintiles presented are based on within study bounds and are not representative of population-level cut-offs. Standardised rates were calculated for each measure using the stratum-specific proportions from the high-risk cohort. Formal analysis was undertaken using prevalence rate ratios, presented with 95% confidence intervals. A reference group was selected for each variable and null hypothesis testing conducted based on a rate ratio equal to 1. Statistical significance was set at $P<0.05$. All data analysis was conducted using R (version 3.2.5) and the R package 'fmsb'.

Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) guidelines were utilised, where applicable.

RESULTS

Cohort and period prevalence of ARBI

The entire cohort consisted of 1276 patients. Average age was 52 years (SD=14) and 885 (69.4%) were males. The mean AUDIT and SADQ scores were 20.8 (SD=8.7) and 19.3 (SD=9.9), respectively. The most common type of alcohol consumed was spirits (n=469; 36.8% of patients). Four-hundred and fifty-four were current smokers (35.6%). The 12-month period prevalence for matching at least one of the criteria for high risk of ARBI was 16.1% (n=205; 'high risk cohort'). The prevalence of MoCA ≤23 in this high group was 36.1%; which is equal to 5.8% of the entire cohort, although not all patients were assessed with MoCA meaning this figure cannot be fully validated. The estimated rate for the high-risk group was 26.3% when the MoCA cut-off was adjusted to ≤21, and 53.7% when adjusted to ≤25.

Variables associated with matching high risk criteria

Table 1 presents rates of patients matching one or more of the high-risk criteria, which resulted in MoCA assessment. The standardised rate for females and males was 15.9 and 16.2 per 100 patients, respectively, which was not significantly different ([prevalence rate ratio] 1.02; 95%CI: 0.78-1.34). Those under the age of 35 years were significantly less likely (0.51; 95%CI: 0.29-0.92) to match the 'high risk' criteria than the reference group (i.e. 35-44 years); all other age bands had similar rates. There was suggestion of a deprivation gradient, such that those from more deprived areas were at greater risk; although the only outcome that reached significance was between the least deprived and second most deprived quintiles (1.73; 95%CI: 1.15-2.59). Living in a family environment appeared to be a protective factor, with those staying in a hostel (4.68; 95%CI: 2.37-9.24) or being homeless (1.91; 95%CI: 1.02-3.59) having rates that were >1.5-fold higher than those of the reference group. None of the other investigated variables showed significant differences.

Variables associated with MoCA ≤23 in the high-risk cohort

Table 2 presents prevalence rate ratios of patients with a MoCA score ≤23 from the high-risk cohort. The standardised rate for females and males was 41.9 and 33.6 per 100 patients, respectively (0.80; 95%CI: 0.55-1.16). None of the investigated variables showed significant differences, likely due to a lack of power or screening processes resulting in a homogenous high-risk group.

Multivariate analysis was not conducted given the limited statistical rationale for such an approach based on univariate outcomes.

DISCUSSION

This study provides an estimate of ARBI in an acute care setting in the UK. We found that 16.1% of all inpatients seen by the ACT team were at increased risk of ARBI and 36.1% of this high-risk group screened positive for potential ARBI, with a MoCA score of ≤23. These findings offer insight into the size of this poorly defined clinical entity, and provide approximate baseline metrics on which to assess changes over time.

Identifying ARBI in acute care settings is important given that traditional approaches to alcohol treatment are entirely reliant on the patients' ability to retain and understand messages. Although generalist healthcare professionals are becoming more aware of the burden associated with the consequences of ARBI, there exists no standard guidance in an acute hospital setting on how to identify the presence or stratify severity of ARBI. Therefore, failure to detect the presence of ARBI results in patients being expected to comply with care pathways that they neither understand nor remember (Bates, Buckman, & Nguyen, 2013). Consequently, alternative approaches to care need to be designed to account for and manage the associated cognitive defects. These need to include: a) clear stratification of risk factors b) identification through routine screening of patients at risk, c) greater involvement of family and other carers, d) consideration of pharmacotherapy to reduce cravings, and e) close monitoring of co-morbidities. However, there needs to be further work to develop an agreed phenotypic definition of ARBI which will help better define the scale of the problem and improve clinical pathways.

The most commonly used clinical definition of ARBI is given in the Diagnostic and Statistical Manual Version IV (Frances, 1994); however this has been shown to be vague and subjective with poor utility in acute care settings (Oslin, Atkinson, Smith, & Hendrie, 1998). Oslin and colleagues attempted to

anchor the diagnosis based on duration and volume of alcohol consumption (Oslin et al., 1998), but there has been limited uptake of this definition in both clinical and research environments. Indeed, rates of ARBI in different studies have been variable (Cheng et al., 2017). For example, a 0.001% prevalence has been estimated in a general population of 300,000 based on an adapted version of the Oslin criteria (Wilson et al., 2012). By contrast, 25.6% of 39 institutionalised AUD patients had ARBI diagnosed using a battery of cognitive tests (cognitive deficit was defined as performance at least 1.5 SD below the normal control mean for MMSE, NART-R, CERAD, Trailmaking Test and/or Clock drawing) (Saxton et al., 2000). A study of homeless hostel dwellers in Glasgow reported that 21% had evidence of ARBI, defined using a cut-off of <88 on the Addenbrooke's Cognitive Examination alongside positive screens on several alcohol consumption questionnaires (Gilchrist & Morrison, 2005). There are no studies providing a directly comparable estimate of the prevalence of ARBI in an acute care setting, which highlights the dearth of research and associated knowledge gap for this condition.

The MoCA offers an alternative approach to early screening of neurodegenerative changes, and has been shown to be a viable tool in patients with AUD (Alarcon, Nalpas, Pelletier, & Perney, 2015). The MoCA has also been shown to be sensitive to changes in cognitive performance over time in patients with AUD (Pelletier, Nalpas, Alarcon, Rigole, & Perney, 2016), which adds to its clinical value. A recent study of 90 AUD patients also demonstrated the superiority of MoCA over the Brief Evaluation of Alcohol-Related Neuropsychological Impairments (i.e. BEARNI) in detecting cognitive impairment (Pelletier et al., 2018). MoCA scores of 23.6 and 21.8 were found to detect mild and moderate to severe dysfunction respectively against a standardised assessment, and the tool had a sensitivity of 0.79 and specificity of 0.65 for characterising patients. The sensitivity and specificity of MoCA for detection of ARBI still needs to be determined but is problematic given the lack of consensus on the most robust diagnostic (i.e. gold standard) criteria. When considering the

performance of MoCA across other conditions, the MMSE is often used as a comparator. The MoCA has been shown to outperform MMSE for detection of mild cognitive impairment in Parkinson's disease (Hoops et al., 2009), post-stroke vascular cognitive impairment (Dong et al., 2010), and transient ischemic attack (Pendlebury et al., 2012). Whether MoCA or MSSE is superior in the current population and context is yet to be tested.

There was minimal support for evidence of ARBI risk factors based on patient characteristics or behaviour. This is likely a reflection of the limited sample size which resulted in low power to detect differences once the groups were categorised. However, patients who were homeless or living in hostels were more likely to be screened as high risk and therefore assessed using MoCA, and there was trend towards a greater risk of ARBI in these subgroups (i.e. MoCA ≤23). This could be due to several interacting factors, including lack of support and/or poor nutrition. Nutritional deficiencies, especially low thiamine, increase the risk of ARBI susceptibility (Lovinger, 1993). For example, Lee and colleagues have shown that AUD individuals with nutritional deficiencies are at augmented risk of corpus callosum shrinkage (Lee, Jung, Na, Park, & Kim, 2005). Further investigation regarding the contribution of macro and micro nutrients to ARBI is required.

This study has highlighted that ARBI is likely to be common in acute settings. But it also highlights the need for larger studies that build on a consensus definition and moves toward randomised controlled trials that can test interventions. It is important that the link we have identified between the positive MoCA screen and ARBI risk is formally validated. The criteria against which the MoCA and other tools are assessed needs to be carefully considered given the aforementioned lack of consensus in diagnostic criteria. Our sensitivity analysis of MoCA cut-offs revealed variation in prevalence estimates, suggesting the need for further conclusive work in this area. Furthermore,

although some studies have shown that MoCA is superior than other tools, there are still gaps in the literature regarding which tool is most appropriate for this patient group.

A limitation of our study is that we were only able to calculate the period prevalence. As this pathway was newly implemented, we were unable to determine how many cases were new vs. existing, precluding the calculation of incidence. Furthermore, there might be an issue relating to inflation as frequent attenders will have been captured in this cohort and new entrants into the cohort might be rarer. Additionally, the study population in terms of IMD diversity was extremely homogenous. This precluded accurate assessment of ARBI risk by social status, but it again highlights the clustering of treatment need for AUD in less advantaged areas (Bellis et al., 2016).

In conclusion, our study which has assessed the prevalence of potential ARBI in AUD patients in an acute setting is important as it highlights a hidden problem in a patient population that is underrepresented and often misunderstood in terms of clinical care. There is a need to increase clinical awareness of ARBI alongside building skills and competence in the workforce to ensure provision of appropriate patient-centred care. We acknowledge that this is the first important step in a process and that further work is required to a) develop consensus criteria for the diagnosis of ARBI, b) fully validate the MoCA for the assessment of ARBI, c) understand its utility in this setting, and d) develop and investigate effectiveness of treatment strategies.

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Competing interests

None to Declare

Contributions

LO and PR were responsible for the wider study conception. AT and LO conceived the specific project design. All authors evaluated and approved the final analysis plan. AT conducted the data analyses and drafted the initial report. All authors contributed to interpreting the analyses and to critically revising the article, and approved the final draft. The authors would like to thank David Byrne and Kev Patterson for data management and extraction.

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Table 1: Variables predicting patients matching MoCA screening criteria

	Rate/100 patients	Risk ratio (95% CI)		P-value
Gender:				
Female	15.9	-	-	
Male	16.2	1.02	(0.78,1.34)	0.893
Age:				
<35	8.7	0.51	(0.29,0.92)	**0.020**
35-44	17.0	-	-	
45-54	18.2	1.07	(0.75,1.52)	0.711
55-64	16.7	0.98	(0.67,1.44)	0.931
≥65	15.7	0.92	(0.61,1.38)	0.694
Within study Deprivation:				
5 (least)	12.6	-	-	
4	14.6	1.15	(0.74,1.80)	0.535
3	16.2	1.97	(0.83,1.98)	0.264
2	21.9	1.73	(1.15,2.59)	**0.007**
1 (most)	15.8	1.25	(0.81,1.93)	0.320
Smoker:				
No	13.9	-	-	
Yes	16.7	1.21	(0.90,1.63)	0.216
Living environment:				
Family	13.7	-	-	
Alone	15.4	1.13	(0.79,1.59)	0.505
Homeless	23.7	1.91	(1.02,3.59)	**0.050**
Hostel	22.4	4.68	(2.37,9.24)	**<0.0005**
Supported	17.5	1.41	(0.69,2.91)	0.356

AUDIT:				
Low	14.5	-	-	
Increasing	9.2	0.63	(0.34,1.71)	0.143
High	17.9	1.23	(0.79,1.91)	0.343
Main alcoholic drink:				
Low/medium ABV beer/cider	15.6	-	-	
High ABV beer/cider	20.1	1.29	(0.87,1.90)	0.202
Spirits	14.9	0.96	(0.69,1.33)	0.796
Wine	17.3	1.11	(0.71,1.73)	0.650

Table 2: Variables associated with MoCA ≤23 in high risk cohort (n=205)

	Rate/100 patients	Risk ratio (95% CI)		P-value
Gender:				
Female	41.9	-	-	
Male	33.6	0.80	(0.55,1.16)	0.253
Age:				
<35	7.7	0.24	(0.04,1.68)	0.088
35-44	31.7	-	-	
45-54	33.8	1.07	(0.61,1.87)	0.821
55-64	41.3	1.30	(0.74,2.30)	0.357
≥65	48.6	1.53	(0.88,2.68)	0.129
Within study Deprivation:				
5 (least)	41.9	-	-	
4	38.9	0.93	(0.52,1.66)	0.801
3	30.0	0.71	(0.38,1.34)	0.300
2	35.2	0.84	(0.48,1.45)	0.539
1 (most)	35.9	0.86	(0.47,1.54)	0.609
Smoker:				
No	34.3	-	-	
Yes	35.5	1.04	(0.66,1.61)	0.876
AUDIT:				
Low	31.6	-	-	
Increasing	23.5	0.75	(0.25,2.20)	0.596
High	38.7	1.23	(0.61,2.45)	0.547

HIGHLIGHTS

- Through stratification, 205 patients were classified at increased risk of ARBI.
- We estimated the period prevalence of ARBI in this high-risk group to be 36.1%.
- Factors associated with ARBI development and progression remain knowledge gaps.

Author Statement

LO and PR were responsible for the wider study conception. AT and LO conceived the specific project design. All authors evaluated and approved the final analysis plan. AT conducted the data analyses and drafted the initial report. All authors contributed to interpreting the analyses and to critically revising the article, and approved the final draft. The authors would like to thank David Byrne and Kev Patterson for data management and extraction.