Original Article

Journal of Addictions Nursing • Volume 30 • Number 3, 159–168 • Copyright © 2019 International Nurses Society on Addictions

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Risk Factors for Severe Alcohol Withdrawal Syndrome in an Acute Hospital Population

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Abstract

Background: The literature lacks consensus to the factors that increase the risk of a patient developing severe alcohol withdrawal syndrome (SAWS).

Aim: The study set out to identify the variables that increase the risk of SAWS in patients who have alcohol dependence syndrome.

Methods: A case–control study was designed to investigate the variables associated with SAWS in an acute hospital setting. Three hundred eighty-two case and 382 control patients were randomly selected retrospectively from referrals to the acute addiction liaison nursing service during a 12-month period (January 1, 2015, to December 31, 2015). Statistical significance ($p < .05$) and association with SAWS were calculated using chi-square, Cramer's V test, odds ratio, and Levene's test.

Results: Twenty-four variables have been identified as associated with SAWS development. Five of the 24 variables had a moderate-to-strong association with SAWS risk: Fast Alcohol Screening Test, Glasgow Modified Alcohol Withdrawal Scale score, AWS admission, hours since the last drink, and systolic blood pressure. The study also identified that comorbidity was associated with not developing SAWS.

Conclusion/Recommendations: These findings confirm that noninvasive variables collected in the emergency department are useful in identifying a person's risk of developing SAWS. The results of this study are a useful starting point in the exploration of SAWS and the development of a tool for use in the emergency department that can stratify risk into high and low and is the next stage of this program of work.

Keywords: Alcohol, Alcohol-Related Seizures, Alcohol Withdrawal Syndrome, Delirium Tremens, General Hospital

INTRODUCTION

Excessive alcohol use is a global phenomenon that contributes to approximately 6% of worldwide deaths and 5% of health conditions (World Health Organisation, 2014). The annual costs associated with alcohol-related ill health are £270 billion for the European Union, $185 billion for the United States, £25 billion for the United Kingdom, and £3.5 billion for Scotland (Carlson et al., 2012; Rehm et al., 2009; The Department of Health, 2008; The Scottish Government, 2009). Most of the expenditure connected to alcohol-related ill health is spent on hospital treatment, most of which is attributable to inpatient costs (Forsythe & Lee, 2012; Manasco, Chang, Larriviere, Hamm, & Glass, 2012; Stehman & Mycyk, 2013; The Department of Health, 2008; Waye, Wong, & Lee, 2015). Although patients presenting to accident and emergency departments and intoxicated are recognized, the primary alcohol-related reason for hospital admission is alcohol withdrawal syndrome (AWS; Maldonado et al., 2015; Pecoraro et al., 2013; Vardy et al., 2016).

AWS is a consequence of alcohol cessation in people who drink in a dependent way and where the severity of withdrawal is congruent with the person's level of alcohol dependence syndrome (ADS; James, Hussain, Moonie, Richardson, & Waring, 2012; Morgan & Ritson, 1998; National Institute for Health and Care Excellence, 2011; Riddle, Bush, Tille, & Dilkhush, 2010). When a person who has ADS stops drinking, it triggers pathophysiological changes within the brain, which present themselves as physiological and psychological symptoms that, in the most extreme circumstances, can result in death (Heymann, Nachtigall, Goldmann, & Spies, 2010; Munchie, Yasinian, & Oge, 2013). The symptoms associated with AWS are evident as early as 6 hours post last drink and peak within the first 48 hours (Heymann et al., 2010; Munchie et al., 2013; Victor & Adams, 1953). However, severe alcohol withdrawal syndrome (SAWS) that includes alcohol-related

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The authors report no conflicts of interest. The authors alone are responsible for the content and writing of the article.

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DOI: 10.1097/JAN.0000000000000287

seizures (ARSs) and delirium tremens (DTs) is experienced by as few as 10% of people who have ADS on cessation of drinking (Feeney et al., 2015; Morgan & Ritson, 1998; Perry, 2014; Riddle et al., 2010; Sutton & Jutel, 2016).

In a hospital setting, SAWS is associated with poor alcohol assessment, identification, and treatment, a practice that is improved with the employment of specialist alcohol nurses (Awissi, Lebrun, Coursin, Riker, & Skrobik, 2013; Stephens et al., 2014; Sutton & Jutel, 2016; Swift, Peers, Jones, & Bronson, 2010; The Royal College of Physicians, 2010; Williams & Mitchell, 2013). However, short-stay hospital admissions are on the increase, with patients who have ADS admitted in case they develop SAWS (Dolman & Hawkes, 2005; Information Services Division Scotland, 2017; Maldonado et al., 2015; National Health Service [NHS] England, 2016; Pecoraro et al., 2013). Although there are a plethora of tools available for screening the level of a person's alcohol use and severity of his or her symptoms, there are a paucity of tools for identifying the patients at a low risk of SAWS (Dolman & Hawkes, 2005; Maldonado et al., 2015; Pecoraro et al., 2013). The development of a tool to stratify SAWS risk is not supported by a lack of agreement in the literature to the factors that increase this risk (Booth & Blow, 1993; Dolman & Hawkes, 2005; Eyer et al., 2011; Ferguson et al., 1996; Lee et al., 2005; Lukan, Reed, Looney, Spain, & Blondell, 2002; Maldonado et al., 2015; Mennecier et al., 2008; Monte et al., 2010; Pecoraro et al., 2013; Wright, Myrick, Henderson, Peters, & Malcolm, 2006).

The systematic literature review carried out by the authors and published previously identified a number of flaws in the SAWS literature (Benson, Roberts, McCallum, & McPherson, 2019). The flaws included inconsistent data coding and databases that were not recognized or validated, which made it difficult to extrapolate to a wider population. In addition, comorbidities and cotherapies were not documented or included in the analysis. The literature presented findings based on evidence from studies that failed to include a validated screening questionnaire, treatment tool, and data generation from staff competent in alcohol assessment (Booth & Blow, 1993; Eyer et al., 2011; Ferguson et al., 1996; Lee et al., 2005; Lukan et al., 2002; Mennecier et al., 2008; Monte et al., 2010; Wright et al., 2006). Although poor alcohol assessment is a frequent criticism of general hospital clinicians, the competence levels of the staff who generated the primary alcohol data within the literature were rarely explored (Benson et al., 2019; Griffiths, Stone, Tran, Fernandez, & Ford, 2007; Schwarz & Oyserman, 2001; Stehman & Mycyk, 2013; The Royal College of Physicians, 2010; Williams & Mitchell, 2013).

The aim of this study, described in this article, is to identify the risk factors that were statistically significant and associated with SAWS development in an acute general hospital population in Glasgow. The case–control study was the second stage of a three-stage program of work to develop a risk stratification tool. The risk stratification tool would support the identification of patients at a low risk of SAWS and who could be discharged from the emergency department. The first stage of the program of work was a systematic literature review; and the third, a cohort study.

PATIENTS AND METHODS

The population studied in the study resided in Greater Glasgow and was admitted to one of the five Glasgow hospitals within NHS Greater Glasgow and Clyde (NHSGGC): Southern General, Victoria Infirmary, Gartnavel General, Western Infirmary, and Glasgow Royal Infirmary. Glasgow is the largest city in Scotland and accommodates approximately 600,000 of the five million population. The Scottish Government article "Changing Scotland's Relationship with Alcohol" estimates that 10% (n = 60,000) of the Scottish population has ADS (The Scottish Government, 2009). Therefore, the sample size determination for this study was calculated using an electronic sample calculator and based on 60,000 people (Creative Research Systems, n.d.). The Glasgow population representative of those with ADS who achieve a 95% confidence level and 5% confidence interval was calculated at 382 in the case and control groups.

The retrospective design was unanimous within the literature and supported the investigation of SAWS. SAWS is the consequence of years of drinking in patients who have ADS and where the development of SAWS is rare and evident in as few as 10% of patients (National Institute for Health and Care Excellence, 2016). In addition, the retrospective design allowed the matching of patients who developed SAWS and those who did not, which would not have been possible prospectively (Booth & Blow, 1993; Eyer et al., 2011; Ferguson et al., 1996; Lee et al., 2005; Lukan et al., 2002; Mennecier et al., 2008; Monte et al., 2010; Wright et al., 2006).

The study included patients older than 16 years admitted to the NHSGGC acute adult hospitals and who had ADS. In NHSGGC, patients who have an alcohol problem are referred to the acute addiction liaison nursing service (AALNS). The AALNS is a team of specialist alcohol nurses who have undertaken a formalized alcohol care and treatment competence program (McPherson & Benson, 2011). During a 12-month period (January 1, 2015, to December 31, 2015), all patients referred to the AALNS (n = 4,852) were assessed for inclusion in the study against the criteria of ADS. In NHSGGC, patients who have a Fast Alcohol Screening Test (FAST) score of ≥9 are deemed to have probable ADS and commenced on the Glasgow Assessment and Management of Alcohol (GAMA) guideline (McPherson, Benson, & Forrest, 2012). The GAMA is a four-page guideline that includes screening for alcohol use and dependency using the FAST (Hodgson, Alwyn, John, Thom, & Smith, 2002) and guidance for vitamin prophylaxis and treatment of Wernicke's encephalopathy. In addition, risk stratification is incorporated for staff, with guidance provided on the use of fixed-dose or symptom-triggered treatments in the management of AWS. The tool also includes a simple numeric score, the Glasgow Modified Alcohol Withdrawal Scale (GMAWS), to assess the symptoms of AWS and provide treatment guidance (McPherson et al., 2012). However, in addition to the FAST and to increase the sensitivity of case and control identification, the International Classification of Diseases (ICD), 10th Revision discharge diagnosis was also used.

The case group was randomly selected from the patients referred to the AALNS and who had an ICD-10 discharge

diagnosis of ADS (F10.2), withdrawal from alcohol (F10.23), and alcohol delirium (F10.231; $n = 1{,}114$), whereas the control group was randomly selected from patients who had an ICD-10 discharge diagnosis of ADS (F10.2; $n = 1{,}879$) only. Although patients who have ADS will have frequent hospital admissions, only the first admission for each patient was used. The selection, randomization, and data checking processes are shown in Table 1.

Randomization for the study was performed using the electronic program Research Randomiser (Urbaniak & Plous, 2015). The case and control groups were matched on gender and based on the percentages referred to the AALNS during the study period (80% male, 20% female). The study sample consisted of 308 male patients and 74 female patients within the case and control groups. Ethical approval for the case–control study was granted by the NHS North East Research Ethics Committee (16/NE/0243).

The 55 risk factors of interest implicated in the development of SAWS and investigated in the study were identified through exploration of the SAWS literature (Booth & Blow, 1993; Eyer et al., 2011; Ferguson et al., 1996; Lee et al., 2005; Lukan et al., 2002; Mennecier et al., 2008; Monte et al., 2010; Wright et al., 2006). In NHSGGC, these risk factors were located on two systems: paper assessments and electronic patient records stored on Clinical Portal.

Extraction of the data was manually performed by G. B. using a data extraction tool. The data extraction tool was reflective of the variables of interest and its ease of use piloted by a single rater (G. B.) on a small sample of patient records. The paper assessment completed by the AALNS included alcohol history variables: time since the last drink, weekly alcohol consumption, previous ARS, SAWS, DTs, detoxification, and number of detoxifications. The electronic patient record included demographics, FAST, GMAWS, benzodiazepines required, comorbidities, blood results, seizure during admission, and length of hospital admission.

During the study period (January 1, 2015, to December 31, 2015), all patients with suspected ADS and perceived to be at risk of AWS were managed using the GAMA guideline. Benzodiazepines were used for symptoms of AWS and directed by the GMAWS (McPherson et al., 2012). Thiamine was administered to all patients and guided by the GAMA. The GAMA reflected SAWS with a GMAWS ≥ 4 and no SAWS with a GMAWS < 4 (McPherson et al., 2012).

ANALYSIS

Statistical analysis for this study was performed using the Statistical Package for Social Sciences program (Version 22). Complete data were collected for all variables except gamma-glutamyl transferase. The biological blood results were dichotomized as high and low in accordance with the parameters employed by NHSGGC and the United Kingdom Accreditation Service (Clinical Biochemistry Services in NHSGGC, 2016). In addition, pulse was dichotomized as high and low in accordance with physiological text books (Waugh & Grant, 2014). The chi-square test was employed to identify statistical significance for the categorical variables and SAWS, whereas the odds ratio measured whether SAWS as an outcome was expected or not. The Cramer's V test supported the chi-square and calculated the strength of statistical significance for the categorical variables. Moreover, as the continuous variables did not follow a normal distribution, the Levene's test was used with statistical significance set at $p = .05$ or 5%. The analysis and crude odds ratio supported the removal of variables that showed no statistical significance in the development of SAWS in the NHSGGC population. Only the statistically significant variables would be studied further in Stage 3 of this program of work and a cohort study.

RESULTS

The demographic descriptive and statistical analysis results are shown in Table 2. Ninety percent of participants were White Scottish, 24% were married, and 8% were in employment, and whereas ethnicity and marital status did not increase a patient's risk of SAWS, employment did ($p < .036$). The case group was younger (47 vs. 50 years old) than the control group that did not develop SAWS, although age was not statistically significant in the development of SAWS ($p = .188$). Deprivation where 1 = *the most deprived* and 5 = *the least deprived* was associated with SAWS development ($p < .004$). In addition, although more than half of all participants lived in the most deprived postcode areas, people residing in the second least deprived area were four times as likely to develop SAWS than not. Moreover, admission with an alcohol-related reason, namely, ARS, AWS, and DTs, was statistically significant in the development of SAWS (all ps $< .01$).

Comorbidities were prevalent in the control group that did not develop SAWS (8 vs. 4) and included alcohol liver disease, cardiac disease, cerebral vascular disease, cancer, diabetes, and pancreatitis (all ps $< .05$). In addition, four biological blood measures were statistically significant in the development of SAWS, namely, alanine aminotransferase ($p < .001$), aspartate aminotransferase ($p < .001$), platelets ($p < .001$), and potassium ($p < .022$), whereas albumin decreased the risk ($p < .001$).

Table 3 shows that patients who developed SAWS presented with higher FAST and GMAWS scores at the emergency department, scored higher on the GMAWS throughout their admission, and required greater levels of pharmacological treatment. The case group consumed more alcohol (238 vs. 216 units) and had their last drink almost twice as long ago (35 vs. 19 hours) than the control group. Although the level of a person's alcohol consumption was not statistically significant ($p = .159$) in the development of SAWS, the time since the last drink was ($p < .001$). The alcohol history of previous SAWS ($p < .001$), ARS ($p < .001$), and DTs ($p < .003$) was statistically significant and associated with SAWS, whereas previous detoxification and number of detoxifications were not. The physical observations of systolic blood pressure (SBP) and pulse rate were statistically significant and associated with developing SAWS ($p < .001$), whereas that of diastolic blood pressure (BP) was not ($p < .704$). The patients who did not develop SAWS had a hospital admission that was 1 day less than the case group (5 vs. 6 days, respectively).

The analysis shown in Table 3 identified 29 of 55 variables as statistically significant in the development of SAWS. However,

TABLE 1 Identification and Randomization Steps for the Case–Control Sample	
Step	**Process**
Step 1	PIMS electronic report exported that identified referrals to the AALNS for the period from January 1, 2015, to December 31, 2015 (n = 4,852)
Step 2	PIMS report identified that 4,852 referrals were generated by 4,100 patients. Only the first admission for each patient was included (males= 3,239, female = 861)
Step 3	Electronic spreadsheet for the 4,100 potential participants developed to include chi-number, name, gender, and admission date
Step 4	ICD-10 discharge codes located in the 4,100 patients' general practitioner (GP) letters accessed through Clinical Portal
Step 5	ICD-10 discharge code from GP letter recorded beside the corresponding patient information on the electronic spreadsheet
Step 6	Removal of records from the electronic spreadsheet that had no GP letter (n = 615) or ICD-10 diagnosis (n = 492). Total removed: N = 1,107
Step 7	Remaining 2,993 potential participants identified and stratified by gender
Step 8	Potential case participants: ADS (F10.2) and AWS/delirium (F10.23/F10.231; n = 1,114; male = 869, female = 245 Potential control participants: ADS (F10.2; n = 1,879; male = 1,466. female = 413)
Step 9	Random sampling: potential population numbered on the electronic spreadsheet. Male controls (1–1,466), male cases (1,467–2,335), female controls (2,336–2,748), and female cases (2,749–2,993)
Step 10	Electronic sampling (Research Randomiser) performed through Microsoft Excel and using the allocated patient number (1–1,466/1,467–2,335/236–2,748/2,749–2,993)
Step 11	Random sample case population identified (n = 382; male = 308, female = 74) Random sample control population (n = 382; male = 308, female = 74)
Step 12	Removal of records from the electronic spreadsheet that were not randomly sampled for the study
Step 13	Sample number allocated on entry to the study. Additional column created with the new study number. Case participants were numbered 1–382; and control participants, 383–764. The original numbers were removed from the spreadsheet.
Step 14	Paper data abstraction tool generated for each participant and included study number, chi-number, name, gender, and date of admission from the electronic spreadsheet.
Step 15	Alcohol history gathered from the AALNS paper assessment that corresponded with the date of admission and inputted into the paper data abstraction tool
Step 16	Data from AALNS assessment recorded on the paper data abstraction tool as it appeared in the notes; previous SAWS (yes/no), time since the last alcoholic drink (22 hours), etc.
Step 17	Other history obtained during the patient's hospital admission generated from the electronic patient record and accessed through Clinical Portal
Step 18	Data from the patient's hospital admission recorded on the paper data abstraction tool as it appeared in the electronic record: SBP = 142 mmHg, FAST = 14, etc.
Step 19	Electronic spreadsheet expanded to include data abstraction tool variables
Step 20	Electronic spreadsheet anonymized by removing identifiers such as the chi-number, with identification made through the allocated study number
Step 21	Data dichotomized where appropriate during transfer from hard copy data abstraction tool to electronic spreadsheet. For example, biological blood results were inputted as high or low based on NHSGGC biochemistry and hematology guidance.
Step 22	Data inputted into the electronic spreadsheet and checked against the hard copy data abstraction tool after every 20 records to identify and correct coding errors
Step 23	Data abstraction tool stored in locked cupboard away from other study materials

AALNS = acute addiction liaison nursing service; ADS = alcohol dependence syndrome; FAST = Fast Alcohol Screening Test; ICD-10 = International Classification of Diseases, 10th Revision; NHSGGC = National Health Service Greater Glasgow and Clyde; SAWS = severe alcohol withdrawal syndrome; SBP = systolic blood pressure.

TABLE 2 Demographic Descriptive and Inferential Characteristics and Results for the Case and Control Groups

Variable	Case (n = 382) Descriptive	Control (n = 382) Descriptive	Odds Ratio (*OR*)	p	Cramer's Statistic
Mean (*SD*) age at admission (years)	46.7 (11.54)	49.8 (12.32)	N/A	.188[a]	N/A
Male/female	308/74	308/74	N/A	N/A	N/A
SIMD groups, *n* (%)				.004[b]	0.141
First quintile (most deprived)	233 (61)	263 (69)	0.88		
Second quintile	65 (17)	67 (17)	1.00		
Third quintile	45 (12)	35 (9)	1.31		
Fourth quintile	23 (6)	6 (2)	4.00		
Fifth quintile (least deprived)	14 (4)	11 (3)	1.10		
Ethnicity, *n* (%)				.135[b]	0.103
White Scottish	331 (87)	350 (92)	0.95		
White: other British	34 (9)	21 (5)	1.60		
White Irish	2 (0.5)	0 (0)	2.00		
White Polish	12 (3)	6 (1.5)	2.00		
Asian	3 (0.5)	5 (1.5)	0.62		
Marital status, *n* (%)				.121[b]	0.087
Married	91 (24)	89 (23)	1.04		
Single	237 (62)	222 (58)	1.07		
Divorced	51 (13)	60 (16)	0.81		
Widowed	3 (1)	11 (3)	0.27		
Employed, *n* (%)	34 (9)	23 (6)	1.75	.036[b]	0.076
Comorbidity, *n* (%) history					
Alcohol liver disease	46 (12)	68 (18)	0.68	.025[b]	0.081
Liver disease	15 (4)	11 (3)	1.36	.425[b]	0.029
Respiratory	56 (15)	63 (16)	0.89	.485[b]	0.025
Cardiac	33 (9)	53 (14)	0.62	.022[b]	0.083
Mental health	160 (42)	143 (37)	1.12	.209[b]	0.045
Cerebral vascular disease	6 (1.5)	16 (4)	0.38	.031[b]	0.078
Sepsis	17 (4)	10 (3)	1.70	.170[b]	0.050
Cancer	6 (1.5)	15 (4)	0.40	.046[b]	0.072
Head injury	22 (6)	28 (7)	0.79	.380[b]	0.032
Diabetes	14 (3.5)	34 (9)	0.41	.003[b]	0.108
Pancreatitis	24 (6)	44 (11.5)	0.55	.011[b]	0.092
Illicit drug use	59 (15)	56 (15)	1.05	.761	0.011
Reason for admission, *n* (%)					
Alcohol-related seizure	133 (35)	0	N/A	.001[b]	0.204
Alcohol withdrawal syndrome	241 (63)	121 (32)	1.99	.001[b]	0.315
Delirium tremens	8 (2)	4 (1)	2.00	.003[b]	0.042

Note. Data are expressed as mean (*SD*) for continuous variables and as frequency/*n* (%) for categorical variables.
N/A = not applicable.
[a]Levene's test.
[b]Chi-square.

TABLE 3 Statistically Significant Variables

Variable	Case (*n* = 382) Descriptive	Control (*n* = 382) Descriptive	Odds Ratio (*OR*)	*p*	Cramer's Statistic
SIMD groups	N/A	N/A	N/A	.004[a]	0.141
Employed, *n* (%)	34 (9)	23 (6)	1.75	.036[a]	0.076
Alcohol liver disease, *n* (%)	46 (12)	68 (18)	0.68	.025[a]	0.081
Cardiac, *n* (%)	33 (9)	53 (14)	0.62	.022[a]	0.025
Cerebral vascular disease, *n* (%)	6 (1.5)	16 (4)	0.38	.031[a]	0.045
Cancer, *n* (%)	6 (1.5)	15 (4)	0.40	.046[a]	0.050
Diabetes, *n* (%)	14 (3.5)	34 (9)	0.41	.003[a]	0.032
Pancreatitis, *n* (%)	24 (6)	44 (11)	0.55	.011[a]	0.108
Alcohol-related seizure (ARS), *n* (%)	133 (35)	0.0	N/A	.001[a]	0.204
Alcohol withdrawal syndrome, *n* (%)	241 (63)	121 (32)	1.99	.001[a]	0.315
Delirium tremens (DTs), *n* (%)	8 (2)	4 (1)	2.00	.003[a]	0.042
High alanine aminotransferase	158 (41)	106 (28)	1.49	.000[a]	0.143
High aspartate aminotransferase	273 (71)	218 (57)	1.25	.000[a]	0.150
Low platelets	139 (36)	89 (23)	1.56	.000[a]	0.190
Low potassium	75 (20)	62 (16)	1.21	.022[a]	0.100
Low albumin	83 (22)	149 (39)	0.56	.000[a]	0.190
Fast Alcohol Screening Test, mean (*SD*)	14.8 (1.49)	13.9 (2)	N/A	.000[a]	0.305
Glasgow Modified Alcohol Withdrawal Scale (GMAWS), mean (*SD*)	3.4 (1.63)	0.0	N/A	.000[a]	0.949
Highest GMAWS, mean (*SD*)	4.6 (1.01)	0.0	N/A	.000[a]	1.000
Hours since the last drink, mean (*SD*)	35.4 (6.6)	18.8 (14)	N/A	.000[b]	N/A
Previous SAWS, *n* (%)	57 (15)	28 (7)	2.04	.001[a]	0.121
Previous ARS, *n* (%)	198 (52)	121 (32)	1.64	.000[a]	0.204
Previous DTs, *n* (%)	60 (16)	37 (10)	1.62	.012[a]	0.090
Systolic blood pressure	138.2 (22)	127.0 (23)	N/A	.000[b]	N/A
Pulse > 100 beats per minute (%)	200 (52)	136 (36)	1.47	.000[a]	0.177
Benzodiazepine in the first 24 hours, mean (*SD*)	62.6 (41)	0.0	N/A	.000[b]	N/A
Total benzodiazepines, mean (*SD*)	115.3 (66)	0.0	N/A	.000[b]	N/A
Length of hospital admission, mean (*SD*)	6.2 (8)	5.1 (6)	N/A	.039[b]	N/A
Seizure during admission, *n* (%)	7 (2)	0.0	N/A	.008[a]	0.096

Note. Data are expressed as mean (*SD*) for continuous variables and as frequency/*n* (%) for categorical variables.
N/A = not applicable; SAWS = severe alcohol withdrawal syndrome.
[a]Chi-square.
[b]Levene's test.

the variables benzodiazepines in the first 24 hours, total benzodiazepines, seizure during admission, highest GMAWS score during admission, and length of hospital admission were not variables that would stratify a person's risk of developing SAWS, leaving 24 variables requiring further study.

DISCUSSION

AWS is a potential consequence for people who have ADS on cessation of their drinking. AWS spans a spectrum that ranges from mild and, in the most extreme circumstances, severe that includes DTs and ARS. However, the SAWS literature presents findings that are conflicting and inconclusive, prompting this study (Benson et al., 2019). Accounting for the limitations of the SAWS literature, in this analysis, we compared a group of patients who had ADS and developed SAWS or not in a randomly selected acute hospital population.

This study identified 24 variables that were implicated in SAWS risk, although the strength of association for 19 of the

categorical variables was poor with a Cramer's V test < 0.3 shown in Table 3 (Gravetter & Wallnau, 2012). The categorical variables with a moderate-to-strong association were GMAWS, $\chi^2(9, N = 764) = 687.98$, $p < .0001$; FAST, $\chi^2(6, N = 764) = 71.92$, $p < .0001$; and AWS admission, $\chi^2(1, N = 764) = 75.60$, $p < .0001$, as well as the noncategorical variables, namely, hours since the last drink, $F(1, N = 762) = 75.58$, $p < .0001$, and SBP, $F(1, N = 762) = 4.27$, $p < .0001$.

This study corroborates the importance of an accurate and complete alcohol history, although we propose that, in the development of SAWS, the association with alcohol history may be more related to the quality of assessment than the strength of the variables. Not surprising, the reason for admission being AWS, ARS, and DTs was associated with SAWS development in the randomly selected population. However, of note was that almost one third of the group in our study that did not develop SAWS or score on the GMAWS had AWS documented in the emergency department as their reason for admission. There are three possible reasons for this finding. First, in NHSGGC, the emergency department is frequently failing to achieve the emergency department waiting times target, where 95% of people will be admitted or discharged within 4 hours of their presentation, putting pressure on clinicians to make a decision quickly (Information Services Division Scotland, 2017). Second, the symptoms of AWS are, for the most part, self-reported and, in a pressurized emergency department environment, not explored. Third, the comprehensive assessment carried out by the AALNS resulted in the reason for admission at the emergency department differing from the reason for admission documented at discharge, a practice previously reported (Ambrosini & Bowman, 2009; Désy, Howard, Perhats, & Li, 2010; McPherson et al., 2012; National Institute for Health and Care Excellence, 2011; The Royal College of Physicians, 2010; Ward, Murch, Agarwal, & Bell, 2009).

The realignment of reason for admission was supported in our study by the employment of alcohol screening and treatment tools (Awissi et al., 2013; Stephens et al., 2014; Sutton & Jutel, 2016; The Royal College of Physicians, 2010; Williams & Mitchell, 2013). Unlike the SAWS literature, our study utilized an alcohol protocol—the GAMA guideline (Booth & Blow, 1993; Dolman & Hawkes, 2005; Eyer et al., 2011; Ferguson et al., 1996; Lee et al., 2005; Lukan et al., 2002; Maldonado et al., 2015; Mennecier et al., 2008; Monte et al., 2010; Pecoraro et al., 2013; Wright et al., 2006). The GAMA included the FAST for identifying ADS and the GMAWS for recognizing escalating AWS (McPherson et al., 2012). The usefulness of tools for identifying AWS symptoms has been recognized in other studies (Dolman & Hawkes, 2005; Eyer et al., 2011; Maldonado et al., 2015; Mennecier et al., 2008; Pecoraro et al., 2013). The advantage of using the GMAWS and FAST is that they are quick to use and noninvasive. In addition, both the GMAWS and FAST have been validated in the NHSGGC population for the identification and treatment of SAWS (McPherson et al., 2012).

Although our study, like others, identified age as statistically significant in the development of SAWS, unlike these studies, the development of SAWS in our study was associated at a younger age (47 years) than those not developing SAWS (50 years; Booth & Blow, 1993; Eyer et al., 2011; Ferguson et al., 1996; Lee et al., 2005; Lukan et al., 2002; Mennecier et al., 2008; Monte et al., 2010; Vardy et al., 2016). However, the association with age in our study may be because in part of greater levels of comorbidity, lower alcohol consumption, and lesser participants who had experienced previous SAWS, ARS, and DTs in the group that did not develop SAWS. Although comorbidity does not necessitate having no SAWS, in the older population, preexisting health issues may reduce the levels of alcohol consumed and risk of SAWS on cessation of drinking.

Similar to other published literature, more alcohol was consumed by the group that developed SAWS (Booth & Blow, 1993; Eyer et al., 2011; Ferguson et al., 1996; Maldonado et al., 2015), although in our study, the difference ($n = 20$ units) was not statistically significant. We also showed that, unlike the literature (Eyer et al., 2011; Monte et al., 2010), having a comorbidity was a statistically significant ($p < .05$) factor associated with not developing SAWS. In addition, whereas alcohol liver disease was associated with not developing SAWS, the liver function test bloods, namely, alanine aminotransferase and aspartate aminotransferase, were statistically significant (all ps < .0001) in its development. The prominence of abnormal liver function test bloods in the development of SAWS supports a greater capacity to damage healthy liver cells than abnormal liver function test bloods being associated with SAWS.

The time since the last alcoholic drink is an indicator that divides the literature (Eyer et al., 2011; Ferguson et al., 1996; Lee et al., 2005; Lukan et al., 2002; Maldonado et al., 2015). Although most studies used the blood and breath alcohol concentration to measure the time since the last alcoholic drink, in our study, this variable was self-reported. Of note is that blood and breath alcohol concentration is most useful if the time since the last drink is less than 24 hours, whereas SAWS is most prevalent beyond 24 hours (Munchie et al., 2013). Moreover, as discussed previously, AWS spans a spectrum, where as few as 10% of people will develop SAWS on cessation of alcohol use (Feeney et al., 2015; Morgan & Ritson, 1998; Perry, 2014; Riddle et al., 2010; Sutton & Jutel, 2016). Therefore, if time since the last drink is applied as a single indicator, patients are potentially admitted to a hospital just in case they develop SAWS. Subsequently, in our study, SAWS was statistically significant ($p < .0001$) and associated with the last drink consumed beyond 36 hours and fits with the SAWS time frames (Munchie et al., 2013). Although it could be argued that failure to develop SAWS in our study was because of the time since the last alcoholic drink being within those same time frames, the retrospective design and collection of ICD-10 discharge classification strengthens the results of this study.

Our study identified that people who have ADS, live in the three least deprived areas of Glasgow, and were admitted to a hospital are at a greater risk of developing SAWS than not developing SAWS. In addition, we identified that being employed was statistically significant ($p < .05$) in the development of SAWS. Although the alcohol literature (Eyer et al., 2011; Ferguson et al., 1996; Lee et al., 2005; Lukan et al., 2002; Maldonado et al., 2015) proposes that people living in the most

deprived areas are eight times more likely to be admitted to a hospital for an alcohol reason, our study found that the development of SAWS was greater among those living in the least deprived areas. Nonetheless, although this appears to be findings not presented by the literature, the numbers living in the three least deprived areas and in employment were small compared with those living in the most deprived areas and unemployed.

Patients who developed SAWS like the studies by Monte et al. (2010), Lee et al. (2005), and Lukan et al. (2002) had elevated BP and pulse. However, of note is that elevated physiological markers are synonymous with overactivity of the autonomic nervous system and a natural response to the trauma of withdrawal (Becker, 2008; Carlson et al., 2012; McKeon, Frye, & Delanty, 2008). Added to this, in our study, the control group was older and aligned with more comorbidity than the case group, and therefore, any association between physiological markers and SAWS should be taken with caution. Caution is advised because a consequence of this comorbidity may have been the use of medication with a sedater effect or the use of beta blockers, both of which may have had a counter effect on the overactivity of the autonomic nervous system, maintaining normal SBP, diastolic BP, and pulse (Chen, Chaugai, Zhao, & Wang, 2015; Ferguson et al., 1996).

Although our study reemphasized the complicated relationship between risk factors and SAWS development, it provided a number of important findings. First, the use of an alcohol screening tool (FAST) and alcohol withdrawal symptom tool (GMAWS) in the emergency department can support the identification of not only a high risk of SAWS but also a low risk. Second, in addition to FAST and GMAWS, time since the last alcoholic drink is a useful indicator of SAWS risk. Third, whereas comorbidity is associated with not developing SAWS, the association between liver function tests and SAWS appears more related to a healthier liver. However, the association between deprivation, employment, and physiological markers is more precarious and should be accepted with caution.

Despite the valuable implications of this study, we have to account for some limitations. This study focused on a single healthcare system in Glasgow where the quantities of alcohol consumption and disproportionate health problems experienced by the population may reduce the generalizability of the study findings to other populations. Second, although the use of ADS only in the control group supported identification of the variables synonymous with SAWS, it precluded the patients who developed mild–moderate AWS (GMAWS < 4). Third, the study was limited by the use of a single researcher (G. B.) who was responsible for data collection, coding, and analysis that made it difficult to discount selection bias.

Notwithstanding these limitations, our study provides an important starting point for further investigation into the risk factors synonymous with SAWS. This case–control study is the second stage of a program of work to develop a tool that can help clinicians to stratify risk and support the discharge of patients identified as having a low risk of SAWS from the emergency department.

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