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Short communication

Psychiatric comorbidities associated with a positive screening using the Montreal Cognitive Assessment (MoCA) test in subjects with severe alcohol use disorder

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ABSTRACT

Background: Patients with severe alcohol use disorder (SAUD) frequently show cognitive deficits that can be efficiently identified using screening tools such as the Montreal Cognitive Assessment (MoCA) test. These cognitive deficits, which reduce the efficacy of therapeutic interventions and may contribute to dropout and relapse, could be, however, partly due to a treatable other cause. Accordingly, this exploratory study examined whether psychiatric comorbid disorders can induce a positive MoCA screening among recently detoxified SAUD subjects.
Methods: One hundred recently detoxified patients with SAUD were divided into two groups according to whether they presented cognitive deficits using the MoCA. Groups were compared for demographic data, SAUD severity, impulsivity, and psychiatric comorbidities. The significant parameters were then introduced in a logistic regression model to establish their relative contributions in a positive MoCA status in SAUD subjects.
Results: Among the significant parameters revealed by the bivariable analyses, agoraphobia and current depressive episode were found to be significant predictors of the MoCA status in the multivariable comparisons.
Conclusions: A positive MoCA screening for cognitive impairments among post-detoxification SAUD patients could also be related to comorbid agoraphobia and depressive episode rather than to SAUD itself. A comprehensive psychiatric assessment must be performed in SAUD patients so that other potential causes of cognitive deficits, in particular with regard to mood and anxiety disorders, can be identified and treated.

1. Introduction

Severe alcohol use disorder (SAUD) has been associated with widespread cognitive deficits (Stavro et al., 2013), which may affect at least half of SAUD patients (Alarcon et al., 2015; Fein et al., 1990; Martin et al., 1986), impair the efficacy of therapeutic interventions (Bernardin et al., 2014; Copersino et al., 2012) and contribute to dropout and relapse (Czapla et al., 2016; Durazzo et al., 2008; Noël et al., 2002; Parsons, 1994; Rupp et al., 2016). Several screening tools have been proposed to identify these alterations in SAUD populations, particularly the Montreal Cognitive Assessment (MoCA) test (Nasreddine et al., 2005), a practical and useful tool for post-detoxification screening (Alarcon et al., 2015; Ewert et al., 2018) and for assessing the longitudinal recovery process in subjects with SAUD (Pelletier et al., 2016). After a positive MoCA screening in SAUD patients, it is usual to confirm the suspected deficits using a wider and more complex battery of cognitive tests. Then, symptom-focused treatments such as cognitive training programs can be proposed for patients with alcohol-related cognitive deficits (Bates et al., 2013).

However, the MoCA test also constitutes an efficient screening tool to detect cognitive deficits in other neuropsychiatric disorders, e.g., depression (Moirand et al., 2018) or schizophrenia (Musso et al., 2014;

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Wu et al., 2014). Thus, even though neuropsychological tests confirm the cognitive impairments screened with the MoCA test among patients with SAUD, they cannot ascertain their SAUD-related origin. In other words, the cognitive deficits identified in SAUD patients could be partly due to psychiatric comorbidities, which are indeed highly prevalent in these populations (Fein, 2015). If psychiatric comorbidities are not assessed and, therefore, not treated, this could limit or even prevent the improvement of cognitive abilities and, in the end, constitute a lost chance for the concerned patients. Consequently, the present study aimed to explore, in a sample of recently detoxified patients with SAUD, which psychiatric comorbid disorders can be significantly associated with a positive MoCA screening.

2. Materials and methods

2.1. Participants

A total of 100 patients (32 females) with SAUD were included in this study. Participants were diagnosed with severe AUD according to DSM-5 criteria and recruited in two detoxification centers. They had all been abstinent for 10–15 days to avoid any influence of acute alcohol withdrawal. Benzodiazepine dosing had to be 20 mg per day of diazepam (or equivalent) or less (https://www.benzo.org.uk). Participants had to provide informed consent and to be aged more than 18 years.

2.2. Qualitative and quantitative variables

Face-to-face interviews were conducted by trained psychiatrists. Age and sex were collected and, SAUD severity was measured by considering the mean alcohol consumption in alcohol units (=10 g of pure ethanol) per week one month before detoxification and the number of previous alcohol detoxification treatments. Then, detection of cognitive impairments was performed using the MoCA (Nasreddine et al., 2005). To assess impulsivity, participants completed the Barratt Impulsiveness Scale (BIS-11, Patton et al., 1995; French version, Baylé et al., 2000). The Personality Diagnostic Questionnaire-4+ (PDQ-4+, Hyler, 1994; French version, Bouvard, 2002) was used to screen for antisocial and borderline personality disorder, and the Adult ADHD Self-Report Scale (ASRS, Kessler et al., 2005) was used for attention deficit hyperactivity disorder (ADHD). The Mini International Neuropsychiatric Interview (MINI, French version 5.0.0) assessed the following psychiatric comorbidities: cannabis dependence or abuse, cocaine dependence or abuse, heroin dependence or abuse, current or past depressive episode, current or past isolated psychotic episode, current or past recurrent psychotic episodes, current or past mania, agoraphobia, dysthymia, social phobia, panic disorder, post-traumatic stress disorder, and suicide attempt history.

2.3. Groups creation and statistical analyses

Two groups were constituted: "MoCA+" (n = 51), including patients with scores below the usual cutoff level, i.e., 26 (Ewert et al., 2018; Nasreddine et al., 2005), indicating cognitive deficits, and "MoCA–" (n = 49), including patients with scores above 25, indicating an absence of cognitive deficits.

The statistical analyses were performed using R studio version 1.1.419 (RStudio, Inc., Boston, MA.). For all analyses, the thresholdof significance was set at $p < 0.05$. The normality of the distribution of quantitative parameters was assessed with Shapiro–Wilk tests. Groups were compared for demographic data, SAUD severity, impulsivity, and psychiatric comorbidities using: (i) Wilcoxon rank-sum tests or independent *t*-tests (or Welch's *t*-test when the assumption of homogeneity of variance was not met) for quantitative variables when the distributions of these variables were significantly different from normal or not, respectively; and (ii) Pearson chi-square tests for qualitative variables. In this case, the p-value of the Fisher's exact test is reported when one of the expected frequencies was below 5%.

Parameters significantly associated with MoCA status in the bivariable analyses were then introduced in a logistic regression model with MoCA status as a dependent variable. A sensitivity analysis was also performed using all the variables with a p-value of $p < 0.1$ in the bivariable comparisons. Case-wise diagnostics were performed by examining basic residuals statistics: leverage, studentized residuals, and DFBeta values. Testing for multicollinearity was carried out by computing VIF and tolerance values. Testing for linearity of the logit was also conducted for scores at the BIS-11 by rerunning the logistic regression analysis with all variables and the interaction term of the BIS-11 and its logit.

2.4. Ethics procedures

The protocol of the study was approved by a French official ethics committee (Comité de Protection des Personnes: avis 2014 A00132 45).

3. Results

Except for the mean alcohol consumption in alcohol units per week one month before detoxification and the number of detoxification treatments, distribution normality was confirmed for all the quantitative variables in both groups. Group comparisons (Table 1) revealed that patients from the MoCA+ group, who represented fifty-one percent of the whole sample, reported more impulsivity than patients from the MoCA− group, $t(95.16) = -2.93$, $p = 0.004$, $r = 0.29$. Moreover, significant associations were found between the MoCA status and ADHD, $\chi^2(1) = 7.443$, $p = 0.006$, current depressive episode, $\chi^2(1) = 12.460$, $p < 0.001$, and agoraphobia, $\chi^2(1) = 11.565$, $p < 0.001$.

Subsequently, a logistic regression analysis was conducted to establish the relative contributions of these four parameters in a MoCA+ status among patients with SAUD. The results are presented in Table 2. Introducing the predictors detailed above produced a significant improvement in the fit of the model, $\chi^2(1) = 25.26$, $p < 0.001$. Unlike ADHD, $b = 0.60$, $z = 1.09$, $p = 0.277$, and the score at the BIS-11, $b = 0.01$, $z = 0.61$, $p = 0.540$, both agoraphobia, $b = 1.47$, $z = 2.04$, $p = 0.042$, and current depressive episode, $b = 1.53$, $z = 2.63$, $p = 0.009$, were significant predictors of the MoCA status. Agoraphobia multiplies the odds of MoCA deficit by 4.36; a current depressive episode multiplies the odds by 4.60.

A sensitivity analysis of this logistic regression modeling included the same variables as above plus PTSD and the mean pre-detox alcohol consumption. In this second model also, only agoraphobia (OR = 4.72; 95% CI [1.22–24.27]) and current depressive episode (OR = 4.01; 95% CI [1.27–14.45]) were significantly associated with a positive MoCA test.

All cases had DFBetas less than 1, and leverage statistics were close to the expected average value of 0.05, suggesting that no influential cases were having an effect on the model. Less than 5% of studentized residuals lay outside ± 1.96 since only one case had a studentized residual with a value lower than −1.96 (−2.27). There was no collinearity within the data as suggested by VIF values (ADHD = 1.357, agoraphobia = 1.105, BIS = 1.436, current depressive episode = 1.020) and tolerance values (ADHD = 0.737, agoraphobia = 0.905, BIS = 0.697, current depressive episode = 0.980). Finally, the assumption of linearity of the logit was met for BIS-11 scores.

4. Discussion

We assessed whether comorbid psychiatric conditions are associated with a positive screening for cognitive impairments using the MoCA test in a post-detox population of SAUD subjects. The results revealed that comorbid agoraphobia and depressive episode increased the probability for a positive MoCA screening. This is consistent with the literature that

Table 1
Comparisons between groups showing cognitive deficits (MoCA+) or not (MoCA–) for demographic data, AUD severity, impulsivity, and comorbidities.

Variables	MoCA–	MoCA+	Group comparison
Demographic variables			
Sex ratio (M/F)	(33/16)	(35/16)	$\chi^2(1) = 0.019$, $p = 0.891$
Age in years [M (SE)]	40.59 (1.18)	44.55 (1.10)	$t(97.16) = -1.64$, $p = 0.104$, $r = 0.16$
AUD severity			
Mean alcohol consumption in IAU per week before detoxification [Mdn]	84	140	$W = 985.5$, $p = 0.068$, $r = -0.18$
Number of detoxification treatments [Mdn]	1	1	$W = 1084$, $p = 0.605$, $r = -0.05$
Impulsivity			
BIS-11 [M (SE)]	60.37 (1.78)	67.25 (1.53)	$t(95.16) = -2.93$, $p = 0.004$, $r = 0.29$
Comorbidities			
Agoraphobia	6%	33%	$\chi^2(1) = 11.565$, $p < 0.001$
Antisocial personality disorder	2%[2]	4%	$\chi^2(1) = 0.265$, $p = 1$[a]
Attention deficit hyperactivity disorder	24%	51%	$\chi^2(1) = 7.443$, $p = 0.006$
Borderline personality disorder	31%	45%[2]	$\chi^2(1) = 2.128$, $p = 0.145$
Cannabis abuse	20%	18%	$\chi^2(1) = 0.124$, $p = 0.725$
Cannabis dependence	10%	12%	$\chi^2(1) = 0.062$, $p = 0.803$
Cocaine abuse	2%	12%	$\chi^2(1) = 3.630$, $p = 0.112$[a]
Cocaine dependence	6%	4%	$\chi^2(1) = 0.255$, $p = 0.675$[a]
Current depressive episode	10%	41%	$\chi^2(1) = 12.460$, $p < 0.001$
Current isolated psychotic episode	0%	6%	$\chi^2(1) = 2.971$, $p = 0.243$[a]
Current mania	0%	2%	$\chi^2(1) = 0.970$, $p = 1$[a]
Current recurrent psychotic episodes	0%	6%[1]	$\chi^2(1) = 3.032$, $p = 0.242$[a]
Current tobacco consumption	88%[1]	92%	$\chi^2(1) = 0.591$, $p = 0.517$[a]
Dysthymia	12%	14%	$\chi^2(1) = 0.048$, $p = 0.826$
Heroin abuse	2%	4%	$\chi^2(1) = 0.304$, $p = 1$[a]
Heroin dependence	2%	4%	$\chi^2(1) = 0.304$, $p = 1$[a]
Panic disorder	14%	20%	$\chi^2(1) = 0.502$, $p = 0.479$
Past depressive episode	15%	12%	$\chi^2(1) = 0.172$, $p = 0.678$
Past isolated psychotic episode	0%	6%	$\chi^2(1) = 2.971$, $p = 0.243$[a]
Past mania	8%	6%	$\chi^2(1) = 0.200$, $p = 0.712$[a]
Post-traumatic stress disorder	4%	16%	$\chi^2(1) = 3.740$, $p = 0.092$[a]
Past recurrent psychotic episodes	0%	6%	$\chi^2(1) = 2.971$, $p = 0.243$[a]
Social phobia	10%	12%	$\chi^2(1) = 0.062$, $p = 0.803$
Suicide attempt history	38%[1]	39%	$\chi^2(1) = 0.031$, $p = 0.861$

Note. [a]The p-value of the Fisher's exact test is reported when one of the expected frequencies was below 5%; [1]1 missing value; [2]2 missing values; IAU = International Alcohol Unit; M = mean; SE = standard error of the mean; Mdn = median.

Table 2
Results of the logistic regression modeling based on the parameters associated with a positive MoCA test in the bivariable comparisons.

	B (SE)	95% CI for odds ratio		
		Lower	Odds ratio	Upper
Included				
Constant	−1.72 (1.41)			
ADHD	0.60 (0.56)	0.61	1.83	5.51
Agoraphobia	1.47 (0.72)*	1.16	4.36	21.56
BIS-11	0.01 (0.02)	0.97	1.01	1.06
Current depressive episode	1.53 (0.58)**	1.55	4.60	15.75

Note. $R^2 = 0.18$ (Hosmer–Lemeshow), 0.22 (Cox–Snell), 0.30 (Nagelkerke). Model $\chi^2(1) = 25.26$, $p < 0.001$. ** $p < 0.01$; * $p < 0.05$.

links both depression and anxiety disorders with cognitive deficits (Castaneda et al., 2008; Ferreri et al., 2011; McDermott and Ebmeier, 2009; Rock et al., 2014), even though direct evidence is lacking regarding agoraphobia. Thus, cognitive deficits in SAUD populations could be linked to alcohol or SAUD and comorbid agoraphobia and depressive episode, combined effects of SAUD and these comorbidities, or even to a latent factor common to all the disorders. SAUD and psychiatric comorbidities could also have causal relationships: for instance, anxiety or mood disorders can be side effects of alcohol withdrawal so that cognitive deficits would result from an indirect action of SAUD. Because of the intertwined nature of psychiatric conditions and SAUD, the temporality of disorders is, therefore, an important factor to be considered. In particular, the results of the present study may not reflect a stable picture of the patient. To further specify the potential causes of cognitive impairments in those populations, future studies should determine whether the psychiatric disorder is constituted before the SAUD and persists four weeks after abstinence (Substance Abuse and Mental Health Services Administration, 2015).

Moreover, while bivariable analyses showed significant links between the MoCA status and self-reported impulsivity as well as ADHD, multivariable analyses did not confirm these results, as both BIS-11 scores and ADHD were found not to be significant predictors of a positive MoCA screening. Regarding impulsivity, this is in line with the results of a recent study (Czapla et al., 2016) in which SAUD patients reported more impulsivity than controls, while BIS scores were not significantly correlated with cognitive functioning. This is more surprising regarding ADHD, which is frequently associated with AUD (Faraone et al., 2007; Huntley and Young, 2014), as the MoCA explores dimensions altered in ADHD, such as attention and concentration (Kaplan and Stevens, 2002; Knouse et al., 2008) or executive functions (Boonstra et al., 2005). Although we cannot rule out a lack of statistical power, this result questions the specific impact of ADHD on the MoCA score in SAUD patients, which should be investigated more closely in future studies.

In conclusion, this study is the first to highlight that a positive screening of cognitive deficits using the MoCA test in SAUD populations is probably not related solely to alcohol or SAUD but also to psychiatric comorbidities and that further investigations are warranted to disentangle the different contributing factors better. In clinical practice, a comprehensive psychiatric assessment, in particular regarding mood and anxiety disorders, is necessary before cognitive screening in SAUD. Indeed, our results stress the need to assess and, if possible, to treat psychiatric comorbidities, as this could benefit the rehabilitation of the cognitive abilities of concerned patients and, in this way, might

optimize their chance to achieve and maintain abstinence.

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Author contributors

FD, RJ, and BR drafted the study design. FD, CL, OM, OC, and BG recruited the participants. FD, PM, and BR conducted the statistical analyses. All authors substantially contributed to writing the first draft of the manuscript, and the final version was also approved by all the authors.

Conflict of interest

No conflict declared.

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