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Brief report

Cognitive and anxiety symptoms in screening for clinical depression in diabetes
A systematic examination of diagnostic performances of the HADS and BDI-SF

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ABSTRACT

Background: Little systematic research into the diagnostic performance of instruments used to screen for clinical depression is available for people with diabetes. The objective of this study was to compare performances of the HADS and BDI-SF and their components in association with a standard diagnostic interview.
Methods: In a sample of 298 French outpatients from a diabetes clinic (165 men, aged 59.4 ± 10.7 years), we assessed diagnoses of clinical depression (CD, $n = 42$) and major depression (MD, $n = 30$) using the MINI and administered the HADS and BDI-SF.
Results: Cognitive symptoms from the BDI-SF (BDIcog) were more closely associated with MD than CD. BDIcog and HADS total scores performed best overall in identifying clinical depression (AUCs under ROC curve 85%). For identification of CD, the sensitivity/specificity of BDI cognitive symptoms was 88/71% (cutoff 3+) and for the HADS 83/65% (cutoff 13+). For identification of MD, BDIcog scored 83/80% (cutoff 4+) and HAD-A 80/76% (cutoff 9+). Logistic regression analyses further suggested that BDIcog and HAD-A discriminated between depressed and non-depressed patients better than the somatic and anhedonia items present in the same scales. The depression subscale of the HADS performed poorly.
Limitations: The consecutive nature of the sample may limit the generalizability of our findings.
Conclusion: Results suggest that, in addition to depressed mood, both negative thoughts and anxiety are core elements for the correct identification of clinical depression in chronic illnesses such as diabetes. It may be more appropriate to use the total score when applying the HADS and distinguish non-somatic symptoms within the BDI.

1. Introduction

Although studies have consistently emphasized the impact of depression in people with diabetes, little systematic research has as yet focused on the assessment procedures used to identify depression in this population (McHale et al., 2008). Depression is frequent in people with diabetes, with prevalence ranging from 8% to 20% (Anderson et al., 2001; Chou and Chi, 2005). Compared with patients with diabetes alone, patients with diabetes and depressive symptoms exhibit higher risks of morbidity and mortality (Katon et al., 2005). It is recommended to screen systematically for depression in patients with this condition (ADA, 2009).

Research into the taxonicity of depression suggests that symptoms making this diagnosis are not equally important when it comes to diagnostic performance (Beach & Amir, 2003). The task of identifying depression reliably might be made easier by focusing on specific symptoms such as depressed

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mood, anhedonia or suicidality, which seem to represent the core disturbances observed in clinical depression.

But assessing depression in the medically ill is a challenge because any increase in prevalence may reflect either a genuine depression or physical symptoms associated with the medical illness. One method used to correct this type of confound has been to exclude features common to ill patients in the assessment of depression: weight loss, sleep disturbances, fatigue and so on. This strategy was adopted in the development of the Hospital Anxiety and Depression Scale (HADS, Zigmond & Snaith, 1983). Another method is to give symptoms that seem to characterize depression better (e.g. cognitive symptoms or anxiety) a higher weighting than less discriminating symptoms such as distress. This was the approach adopted when developing the Beck Depression Inventory-Short Form (BDI-SF) with the aim of providing clinicians with an instrument capable of detecting depression in medically ill patients in primary care (Beck and Beck, 1972; Beck et al., 1997). A focus on cognitive symptoms and anxiety is justified in diabetes since the illness is known to encourage a negative self-image and worry about the future (Rubin and Peyrot, 2001).

The objective of this research was to compare the diagnostic performances of the BDI-SF and HADS in the identification of clinical depression, with a particular focus on anxiety and the cognitive symptoms of depression.

2. Method

2.1. Participants

The sample comprised a group of 302 consecutive type 2 diabetes outpatients visiting the Diabetes Department at Pitié-Salpêtrière Hospital, Paris, France between September 2006 and November 2007. Analyses were based on 298 patients, since questionnaires were missing for 4. The study was proposed to 370 patients but 68 refused (18.4%) because of lack of time (Table 1). The patients who refused did not differ from the final sample on age or gender. Inclusion criteria corresponded to a longitudinal follow-up approach currently employed in the department: Type 2 diabetes identified at least one year prior to inclusion (ADA, 2006), age between 20 and 75 years and no major comorbidity

Table 1
Sample description according to clinical diagnosis status.

	MINI standard diagnostic				
	Non-depressed	Major depression (MD)	Dysthymia (DYS)	Depressed (MD+DYS)	Total
	$N=256$	$N=30$ (10.1%)	$N=12$ (4%)	$N=42$ (14.1%)	$N=298$
Personal					
Gender: Men (%)	145 (57%)	15 (50%)	5 (42%)	20 (48%)	165 (55%)
Age (M ± SD)	59.8 ± 10.8	55.7 ± 9.8	61.6 ± 8.4	57.4 ± 9.7	59.4 ± 10.7
Education					
Primary	46 (18%)	9 (30%)	4 (33%)	13 (31%)	59 (20%)
High school	138 (54%)	15 (50%)	3 (25%)	20 (48%)	158 (53%)
College/university	72 (28%)	6 (20%)	5 (42%)	9 (21%)	81 (27%)
BMI	28.9 ± 7.0	31.3 ± 5.9	30.0 ± 4.1	30.9 ± 5.4	29.2 ± 6.8
Medical					
HbA_{1C}	8.3 ± 1.7	9.1 ± 2.5	9.2 ± 1.7	9.1 ± 2.3 [a]	8.4 ± 1.8
Diabetes duration	11.7 ± 9.2	11.6 ± 7.9	13.3 ± 8.5	12.1 ± 8.0	11.8 ± 9.0
Treatment					
Oral	118 (46%)	13 (43%)	4 (33%)	17 (40%)	135 (45%)
Insulin	138 (54%)	17 (57%)	8 (67%)	25 (60%)	163 (55%)
Diabetes complications					
Yes	132 (52%)	15 (50%)	6 (50%)	21 (50%)	153 (51%)
No	124 (48%)	15 (50%)	6 (50%)	21 (50%)	145 (49%)
Retinopathy	67 (26%)	8 (27%)	2 (17%)	10 (24%)	77 (26%)
Nephropathy	41 (16%)	4 (13%)	3 (25%)	7 (17%)	48 (16%)
Neuropathy	59 (23%)	8 (27%)	3 (25%)	11 (26%)	70 (23%)
Macrovascular	65 (25%)	4 (13%)	3 (25%)	7 (17%)	72 (24%)
Depression scales					
BDI-SF (α = .80)	4.6 ± 3.7	12.5 ± 5.7 [c]	8.5 ± 5.0 [b]	11.5 ± 5.8 [c]	5.6 ± 4.7
BDIcog (α = .78)	1.8 ± 2.4	7.7 ± 4.4 [c]	3.7 ± 3.0 [a]	6.6 ± 4.4 [c]	2.5 ± 3.2
BDIsom (α = .70)	2.8 ± 2.1	4.8 ± 2.5 [c]	4.4 ± 2.6 [b]	4.7 ± 2.5 [c]	3.1 ± 2.3
HADS (α = .78)	10.9 ± 5.0	18.8 ± 5.6 [c]	18.2 ± 5.4 [c]	18.6 ± 5.5 [c]	12.0 ± 5.7
HAD-D (α = .71)	4.3 ± 2.8	8.0 ± 3.3 [c]	8.2 ± 3.2 [c]	8.0 ± 3.2 [c]	4.8 ± 3.2
HAD-A (α = .69)	6.6 ± 3.1	10.8 ± 3.4 [c]	10.1 ± 3.0 [c]	10.6 ± 3.3 [c]	7.2 ± 3.4

Note. Correlation between BDIcog and BDIsom was .41. BDIcog and BDIsom correlated .89 and .78 respectively with BDI-SF. Correlation between HAD-D and HAD-A was .54. HAD-D and HAD-A correlated .87 and .89 with HADS. In the full sample, the BDI-SF identified 'moderate to severe depression' (8+) and 'severe depression' (16+) in 77 (25.8%) and 16 (5%) cases respectively. On the HAD-D, 'probable' (8+) and 'definite depression' (11+) were identified in 58 (19.5%) and 18 (6.4%) cases respectively.

[a] $p<.05$.
[b] $p<.01$.
[c] $p<.001$.

apart from diabetes-related complications. The study protocol received full Institutional Review Board approval.

2.2. Materials and procedure

2.2.1. Demographic and clinical variables

Physicians from the department invited eligible patients to participate. Participants were then directed to a psychologist intern who obtained their informed consent and administered an initial structured clinical interview. The examiner was unaware of the somatic or psychological status of the patient. Self-reports were then completed by the patients and given back the same day. The demographic items were assessed by means of specially designed questions. Clinical variables were obtained from the patients' medical records after other data had been collected.

2.2.2. MINI

All patients were administered the Mini-International Neuropsychiatric Interview version 5.0.0 for the DSM-IV (MINI, Modules A and B) as a measure of clinical depression (Sheehan et al., 1998). Interns had been trained in the administration of the MINI prior to data collection. In the present study, we used the diagnosis of Clinical Depression (CD) to refer to either Major Depression (MD) or Dysthymia (DYS). CD or MD, diagnosed with the MINI, was considered to be the standards against which other self-report instruments were evaluated.

2.2.3. BDI-SF and HADS

Participants completed the 13-item BDI-SF. Factor analyses (O'Connor, 2000; Waller, 2009) of BDI-SF items suggested a two-factor solution with the first factor (BDIcog) consisting of eight cognitive and affective symptom items: Self-hate, Sense of failure, Guilt feeling, Body image, Pessimism, Suicidal ideas, Sadness, Lack of satisfaction, and the second factor (BDIsom) consisting of five somatic and social symptom items: Fatigue, Difficulty working, Appetite change, Indecisiveness, Social withdrawal. Patients completed the 14-item HADS. The depression subscale focuses on the measurement of anhedonia, which the authors considered to be a central feature of those depressive disorders that are associated with antidepressant drug response. Factor analyses of HADS items confirmed the bidimensional structure of the instrument. These factor structures justify the comparison of the six scales and subscales in further analyses (Mykletun et al., 2001; Shafer, 2006).

2.3. Statistical analyses

Prior to the analyses, missing data were imputed by means of two-way imputations (Van Ginkel and Van der Ark, 2005). We performed logistic regressions to examine the ability of the scales to predict the odds of clinical depression. We compared the classification performance of the scales and used a univariate *z*-test of the difference between the areas under the two ROC curves (Metz et al., 1998; Metz, 2006). Statistical analyses were performed using SPSS for Windows 14.0 (SPSS Inc. Chicago, Illinois, USA). An α value of .05 was used to indicate statistical significance.

3. Results

We examined the ability to predict clinical diagnoses of each screen in turn after adjusting for controls: Gender, Age, Insulin treatment, Complications, Diabetes duration and HbA_{1C}. Among these, only metabolic control (HbA_{1C}) was associated with CD (OR = 1.29, 95%CI 1.07–1.55, $p < .01$). All six depression scales significantly predicted CD to a greater extent than the personal and clinical data (Mean OR = 1.43, 95%CI 1.26–1.62, $ps < .001$). The highest level of discrimination was achieved by the HADS (Wald = 40.28; $\chi^2 = 62.93$) and BDIcog (Wald = 36.19; $\chi^2 = 49.89$) followed by BDI-SF (Wald = 37.45; $\chi^2 = 49.55$), HAD-D (Wald = 35.85; $\chi^2 = 48.49$), HAD-A (Wald = 35.82; $\chi^2 = 46.26$) and BDIsom (Wald = 17.30; $\chi^2 = 19.48$). We performed the same analyses to predict the probability of MD and found that the highest level of discrimination were obtained by BDI-SF (Wald = 39.45; $\chi^2 = 61.17$) and BDIcog (Wald = 38.33; $\chi^2 = 61.56$). These scales discriminated MD even better than they did CD. The values for the other scales were lower.

When comparing ROC curves in association with the diagnosis of CD, BDI-SF and BDIcog (AUCs = .85 95%CI .78–.91) outperformed BDIsom (AUC = .72 95%CI .64–.80) ($z > 2.95$, $p < .01$), whereas the performance of BDI-SF was similar to that of BDIcog. HADS (AUC = .85 95%CI .79–.92) outperformed HAD-A (AUC = .81 95%CI .73–.87) ($z = 2.10$, $p < .05$). We also found a tendency for HADS to outperform HAD-D ($p = .08$). This suggests that the HADS tended to be more accurate in identifying clinical depression when anxiety was considered in addition to depression items. We also found that the performances of BDI-SF and HADS were similar, as were those of BDI-SF and HAD-D, on the one hand, and BDIcog and HAD-D on the other.

A comparison of AUCs for the diagnosis of MD showed that BDI-SF (AUC = .90, 95%CI .81–.94) and BDIcog (AUC = .90, 95%CI .82–.94) outperformed HAD-D (AUC = .80 95%CI .71–.88) and HAD-A (AUC = .81 95%CI .71–.89) with *z*-values over 2.13 ($p < .05$), but did not perform better than HADS (AUC = .85 95%CI .77–.91, $z = 1.31$, $p = .19$). Other comparisons produced results that were comparable to those observed when predicting CD.

Table 2 reports comparisons of scales considered at cut-points. Among measures with high sensitivity, BDIcog 3+ had the highest screening ability. Between 78% and 98% of depressed patients had a score of 3+ at a confidence level of 95%. With regard to the published cutoffs, BDI-SF 4+ exhibited the greatest sensitivity. However, the use of this measure would lead to a high rate of false positives (specificity 47%). The next highest sensitivity rates were found for HADS 13+ and HAD-A 8+. The usual cutoff of 8+ on the depression subscale HAD-D yielded a low sensitivity of 50%. These results suggest that cognitive symptoms may be used to screen for depression and that anxiety should be included in the HADS in order to optimize the rate of depressed patients detected. When considering MD alone, we found three measures with higher levels of sensitivity: BDI-SF 4+, BDIcog 4+ and HADS 13+. BDIcog 4+ outperformed the other two measures.

To summarize, the best trade-offs between sensitivity and specificity while maximizing sensitivity for the purposes of screening for CD and MD were provided by BDIcog 3+ (CD) and

Table 2
Diagnostic performance of self-report scales for detection of depression at optimal and published cut-points.

	Cut-point	Sensitivity (%)	Specificity (%)	PPV (%)	NPV (%)	Agreement (%)	κ
Detection of clinical depression: 256 Non-depressed vs 42 with clinical depression							
BDI-SF	4+	95 (89–100)	47 (41–53)	22 (16–29)	98 (96–100)	54	.18
	8+ (optimal)	71 (57–85)	81 (76–86)	38 (27–48)	95 (92–98)	80	.38
	16+	29 (15–43)	98 (97–100)	75 (54–96)	90 (86–93)	74	.37
BDIcog	3+ (optimal)	88 (78–98)	71 (66–77)	33 (24–42)	97 (95–100)	74	.35
	4+	73 (60–87)	80 (75–85)	37 (27–48)	95 (92–98)	79	.38
BDIsom	4+ (optimal)	69 (55–83)	68 (62–74)	26 (18–34)	93 (89–97)	68	.22
HADS	13+	83 (72–95)	65 (59–71)	28 (20–36)	96 (93–99)	67	.26
	15+ (optimal)	79 (66–91)	79 (73–84)	38 (27–48)	96 (93–98)	79	.39
	19+	52 (37–67)	93 (90–96)	55 (40–70)	92 (89–96)	76	.36
HAD-D	7+ (optimal)	71 (58–85)	79 (74–84)	36 (25–46)	94 (91–97)	78	.36
	8+	50 (35–65)	86 (81–90)	36 (24–49)	91 (88–95)	81	.31
	11+	26 (13–39)	97 (95–99)	61 (39–84)	89 (85–93)	87	.31
HAD-A	8+	81 (69–93)	64 (58–70)	27 (19–35)	95 (92–98)	66	.25
	10+ (optimal)	71 (58–85)	84 (79–88)	42 (30–53)	95 (92–98)	82	.42
	11+	57 (42–72)	88 (84–92)	44 (31–58)	93 (89–96)	84	.41
Detection of major depression: 256 Non-depressed vs 30 with major depression							
BDI-SF	4+	100 (100–100)	47 (41–53)	18 (12–24)	100 (100–100)	53	.16
	8+ (optimal)	77 (62–92)	81 (76–86)	32 (22–43)	97 (94–99)	81	.36
	16+	37 (19–54)	98 (97–100)	73 (51–96)	93 (90–96)	92	.45
BDIcog	4+ (optimal)	83 (70–97)	80 (75–85)	33 (22–43)	98 (96–100)	80	.38
BDIsom	4+ (optimal)	73 (58–89)	68 (62–74)	21 (13–29)	96 (93–99)	69	.20
HADS	13+	83 (70–97)	65 (59–71)	22 (14–29)	97 (95–100)	67	.21
	17+ (optimal)	77 (62–92)	84 (80–89)	37 (25–48)	97 (95–99)	84	.41
	19+	57 (39–74)	93 (90–96)	49 (32–65)	95 (92–98)	89	.46
HAD-D	7+ (optimal)	73 (58–89)	79 (74–84)	29 (19–39)	96 (94–99)	78	.31
	8+	53 (35–71)	86 (81–90)	30 (18–43)	94 (91–97)	82	.29
	11+	27 (11–42)	97 (95–99)	53 (28–79)	92 (89–95)	90	.31
HAD-A	8+	80 (66–94)	64 (58–70)	21 (13–28)	96 (94–99)	66	.20
	9+ (optimal)	80 (66–94)	76 (66–94)	28 (18–37)	97 (95–99)	76	.31
	11+	67 (50–84)	88 (84–92)	40 (26–54)	96 (93–98)	86	.43

Note. Numbers in parentheses are the 95% confidence interval; PPV = positive predictive value; NPV = negative predictive value. Optimal cut-points determined by ROC curve analyses minimizing the sum of squared complement of sensitivity and specificity to the unity. Sensitivity: rate of test positives among depressed patients; Specificity: rate of test negatives among non-depressed; Positive Predictive Value: rate of depressed among test positives; Negative Predictive Value: rate of non-depressed among test negatives.

4+ (MD). The next best trade-offs were provided by HADS 13+ (CD and MD).

4. Discussion

Two sets of results emerge from these analyses. First, by examining the ability of scales to predict the probability of depression and systematically comparing ROC curves, we were able to confirm that cognitive symptoms are central in the BDI-SF and that anxiety symptoms are necessary in the HADS if this scale is to accurately identify depression in people with diabetes, particularly in the case of MD. Our results are consistent with studies which have suggested the relative superiority of various versions of the BDI over the depression scale of the HADS in mixed somatic samples (Golden et al., 2007). The results are also at odds with the idea that all instruments should perform equally well in the medically ill (Williams et al., 2002).

Second, when examining indices of CD or MD identification performance at cutoff values, we found that BDIcog and HADS or even HAD-A performed better than BDIsom or HAD-D. This underlines the central role of cognitive symptoms and depressed mood in any attempt to screen accurately for depression in somatic patients (Parker et al., 2002; Clark et al., 1998). These results provide additional confirmation of the value of using anxiety symptoms when measuring depression and are consistent with studies in the medically ill suggesting that HAD-A performs well in screening for depression (Strik et al., 2001; Katz et al., 2004). In diabetes, given the threatening progression of the illness, it is likely that the experience of depression will be marked by anxiety. In contrast, relying primarily on anhedonia to identify clinical depression, as we did when using the HAD-D alone, yields poor results.

Overall, our results suggest that depression in diabetes should be approached in terms of three core aspects: depressed mood and anhedonia (e.g. sadness, lack of satisfaction), cognitive symptoms (e.g. sense of failure, self-hate) and anxiety (e.g. worrying, feelings of panic) (see Joiner et al., 2005). They suggest that we should use not the HAD-D but the HADS which includes both depression and anxiety (Razavi et al., 1990) and that the use of the BDI-SF subscales is advisable in the medically ill since its components have different levels of validity when external diagnostic criteria are used.

However, the cutoffs that were examined here involved low PPVs with the result that a small percentage of the people screened as positives will actually be depressed. This raises the question of whether this percentage is higher than the depression base rate in people with diabetes. We computed a chance-corrected PPV (Kraemer, 1992)[1] and found that the increases in

[1] The equation for computing chance-corrected PPV is: QPPV = (PPV − base rate) / (1 − base rate).

diagnostic value for BDIcog 3+ were: 27% and 16% (8% and 20% prevalence). This means that it is more effective to use this screen than simply to rely on chance.

Some limitations must be acknowledged. First, we did not use a randomly selected sample, a fact which might limit the generalizability of our findings. Second, the instruments used were not developed to comply with the DSM-IV diagnostic criteria. Consequently, discrepancies with the structured interview might be due to differences in the time history of reported symptoms.

To conclude, we found that cognitive symptoms, e.g. a sense of failure or an experience of self-hate, appear to be an important aspect of depression which is not confounded with medical condition, and confirmed the important idea that symptoms of anxiety may constitute a central aspect of clinical depression in serious chronic illness. In clinical practice with somatic patients, it may be more appropriate to use the total score when applying the HADS and distinguish non-somatic symptoms within the BDI.

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Conflict of interest

No conflict declared.

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