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Screening and Early Intervention on Mood and Neurocognitive Impairments for Stroke Patients undergoing In-patient Rehabilitation: Initial Results of the Integrated Mood and Neuropsychological (iMAN) Scheme

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Abstract

Mood problems and cognitive impairment are common sequelae of stroke and require clinical attention. This study investigated the effectiveness of psychometric screening service in stroke rehabilitation between April 2014 and March 2016. A total of 457 non-aphasic stroke in-patients were screened and clinical psychology service was provided for indicated patients. Results showed that patients had marked improvement in mood and cognitive functioning when discharged. The clinical psychology service was found to contribute to such improvement by providing intensive mood treatment and neurocognitive rehabilitation respectively. Long-term follow-up on the mood and cognitive status of discharged patients and alternative psychological treatment modality were recommended.

Keywords: Stroke, depression, screening, psychotherapy, neurocognitive intervention

Stroke often happens suddenly, leaving huge and multiple impacts on patients' daily life, such as abilities to move, communicate, self-care, work, and maintain financial independence and family roles. Research suggested that up to 55% of acute stroke patients suffered from post-stroke depression and the percentage was similar even months after stroke (Moncayo, Bogousslavsky & Bogousslavsky, 2008). However, these mood issues may go undetected if stroke is only formulated as a medical condition. Many stroke patients revealed that their emotional issues received little to no attention during hospitalization (Hackett, Yapa, Parag & Anderson, 2005). It is possible that stroke patients may not be able to express their emotional needs due to lethargy or difficulties

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in verbal communication. Psychomotor retardation and shortened attention span could be misinterpreted as part of stroke symptoms rather than signs of mood disturbance. Mood screening could thus be a systematic and effective way to screen for needed patients (NICE, 2013).

Studies also revealed that stroke patients may develop cognitive deficits in areas including attention, language, psychomotor speed, visuospatial ability, memory, reasoning, and executive functions (Lo Coco, Lopez & Corrao, 2016; Middleton, Lam, Fahmi, Black, Mcllroy, Stuss, Danells, Ween, & Turner, 2014; Tatemichi, Desmond, Stern, Paik, Sano, & Bagiella, 1994). Jokinen and colleagues (2015) found that most stroke patients showed deficits in at least one of these domains, and 50% of the patients were affected in more than three domains. These impairments were often not revealed in brain scans but more detectable by a comprehensive neurocognitive assessment.

There is growing evidence supporting that early attention to stroke patients' mood and cognitive functioning is essential, and timely access to psychological and neurocognitive interventions are of great benefit to patients. The NICE Guideline (2013) explicitly stated that stroke patients should be screened for cognitive deficits and emotional difficulties as part of the rehabilitation routine before planning for treatment. Studies also supported this recommendation (Burton & Tyson, 2015; Jokinen et al, 2015). Although holistic care and multidisciplinary services were advocated in recent public health care development, no such practice has been reported or documented in Hong Kong public hospitals. To enhance the stroke care pathway of Haven of Hope Hospital, the Department of Clinical Psychology has strengthened its role in the multi-disciplinary team from April 2014, incorporating a hospital screening scheme, namely the Integrated Mood and Neuropsychological (iMAN) Screening Scheme for Stroke Patients. Stroke patients referred to iMAN would be screened with valid and reliable mood and cognitive measures usually within three days upon admission. Clinical psychology service would be recommended for patients based on the screening results. It was hoped that the scheme could supplement ward observations of potential mood and neurocognitive problems. It was hypothesized that (1) there would be a change in mood and neurocognitive functioning for stroke patients from admission to discharge; (2) mood or neurocognitive functioning would be improved for the indicated patients who were referred for psychological intervention or individualized neuro-rehabilitation training; (3) treatment intensity, i.e. dosage effect, would bring about the change in mood and neurocognitive functioning.

Method

Procedure

Stroke patients were recruited via the iMAN referral protocol (Figure 1). They were then screened by a trained patient care assistant (PCA) for mood and cognitive functioning within three days upon admission (T1). If indicative (i.e. they scored beyond cut-offs of either mood or cognitive measures, or significant issues were noted during screening), case doctor would be recommended to refer such patients to clinical psychology service, including assessment and intervention for mood problems and neurocognitive training. Prior to discharge, the PCA would assess the patients again for progress evaluation (T2).

Due to the early discharge of some patients, and the shortage of manpower for screening during peak seasons, not all patients would be assessed for T2 measures. With the best effort, only 17.1% of patients had completed both T1 and T2.

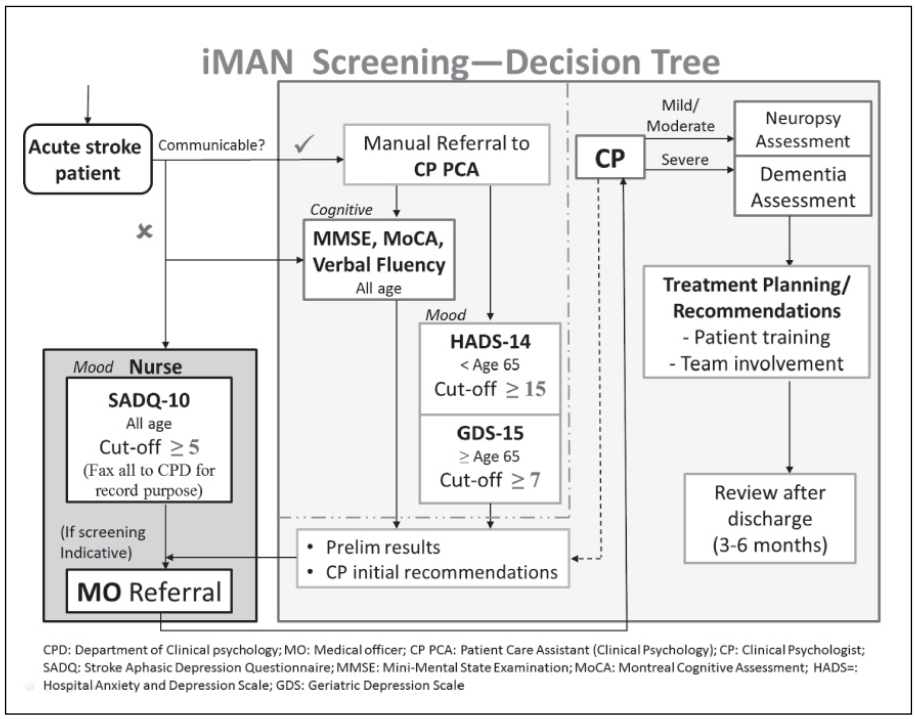

Figure 1: Integrated Mood and Neuropsychological (iMAN) Scheme's clinical decision-making tree

Instruments

Mood screening. Two screening tools were adopted according to age of patients. Either the Chinese versions of Hospital Anxiety and Depression Scale (HADS) or Geriatric Depression Scale 15-item Short Form (GDS-15) was used for mood screening, with the former to provide cut-off score for patients aged below 65 while the latter for age 65 or higher.

HADS. It was developed by Zigmond & Snaith (1983) to screen for depression and anxiety. It was translated into Chinese by Leung, Ho, Kan, Hung & Chen (1993). HADS consisted of seven items of anxiety subscale and seven items of depression subscale. The anxiety and depression subscales showed internal consistency with Cronbach's alpha 0.81 and 0.74 respectively. The cut-off used for each subscale was four out of 21 (Tang, Ungvari, Chiu, & Sze, 2004; Sagen, Vik, Moum, Mørland, Finset, & Dammen, 2009). Higher scores indicated higher level of anxiety or depression.

GDS. It was designed by Yesavage and colleagues (1982) for elderly to rate for depression symptoms. A shorter version (GDS-15) which consisted of 15 items was later developed by Yesavage & Sheikh (1986) and translated into Chinese by Chiu et al. (1994). The scale showed good reliability with a Cronbach's alpha of 0.84. The cut-off used was seven out of 15 (Chiu et al., 2004). Higher scores suggested higher level of depression.

Neuropsychological screening. The Cantonese versions of the Mini-Mental State Examination (MMSE) and the Montreal Cognitive Assessment (MoCA) were used to screen for cognitive impairment.

MMSE. It was developed by Folstein, Folstein and McHugh (1975) and translated into Cantonese (Chiu, Lee, Chung & Kwong, 1994). It measured orientation to time and place, registration, recall, attention, language, ability to follow commands and visual construction. Cronbach's alpha was 0.78. The cut-off used was 24 out of 30 (Wong et al., 2009). Lower scores indicated potential cognitive impairment.

MoCA. It was developed by Nasreddine (2000) and translated into Cantonese by Wong and colleagues (2009). It was initially designed to screen for mild cognitive impairment (MCI) and early stage of dementia. It measured visuospatial and executive functions, naming, memory, attention, language, abstraction, delayed recall and orientation. One extra mark was given to patients with less than six years of education. The cut-off was 22 out of 30. Lower scores indicated potential cognitive impairment.

Participants

Out of 841 stroke patients admitted to the hospital from April 2014 to March 2016, a total of 457 non-aphasic patients (about 54.3%) were screened during the period and included in the present study. Two hundred and twenty-seven screened patients (about 49.7%) exceeded one or more cut-offs and were then referred to clinical psychologist for further management. Table 1 showed their demographic information.

Table 1
Demographics of the Stroke Patients

	N	%
Gender		
Male	252	55.1
Female	205	44.9
Age Group		
30-39	2	0.4
40-48	18	3.9
50-59	44	9.6
60-69	94	20.6
70-79	107	23.4
80-89	162	35.4
90-99	29	6.3
100-109	1	0.2
Education		
None to Primary	265	58.0
Secondary	107	23.4
Tertiary	23	5.0
Not documented	62	13.6
Handedness		
Right-handed	408	89.3
Left-handed	7	1.5
Not documented	42	9.2

Of note an extra sample of 27 patients treated before the start of the iMAN scheme was also included to allow broader sample size for comparisons in the treatment outcome analyses. The demographics, namely age, gender, education, handedness, of these patients were not significantly different from the 457 patients. Hence, a total of 484 patients were used for analysis, in which 180 of them (about 37.2%) received low intensity (less than

3 sessions) treatment (usually composed of intake assessment and pre-discharge review), 55 of them (about 11.4%) received intensive mood treatment (mean number of sessions was 5.1; ranging from 3 to 15) and 19 of them (about 3.9%) received intensive neurocognitive intervention (mean number of sessions was 8.8, ranging from 3 to 33).

Plan of Data Analysis

Paired-sample t-tests were conducted to compare the measures upon admission and discharge. Among those patients who received intervention by clinical psychologist with both pre- and post-treatment scores available, within-subject ANOVAs (pre-post vs treatment-active-control) were computed and interaction effects were looked into for specific treatment effect. The low intensity treatment group (i.e., < 3 sessions) could not serve as a valid control group as their baseline scores on mood (e.g. means of HADS/GDS were 18.28 and 8.85 for the low intensity group versus 20.69 and 9.31 for the mood treatment group) and cognitive measures (e.g., means of MMSE/MoCA were 18.43 and 16.71 for the low intensity group vs 19.16 and 15.73 for the neurocognitive treatment group) were different from the baselines of intensitive mood treatment group and intensitive neurocognitive intervention group respectively. In addition, waitlist control was not feasible in a hospital setting. Thus a proximate was to use the neurocognitive treatment group (with comparable exposure to clinical psychology service) to serve as the active control group for mood treatment group and vice versa.

The net effect sizes were first computed by subtracting the scores in T2 by T1, and the resulting means of the change in scores of both treatment groups and control groups were used to calculate the Cohen's ds.

Dosage effect was calculated by computing step-wise hierarchical regression for the variance explained for the predicted treatment outcomes by treatment intensity controlling the baseline score.

Results

Mood and cognitive functioning scores upon admission (T1) and prior discharge (T2) were compared in Table 2 below.

Table 2
Comparisons of Mood and Neurocognitive Measures Upon Admission (T1) and Before Discharged (T2)

Measures	T1 (Full sample)			Matched-sample					
					T1		T2		
	N_{full}	M	SD	$N_{matched}$	M	SD	M	SD	*p*
HADS	80	17.23	8.07	31	19.58	7.68	16.13	6.12	.009**
Anxiety	80	8.46	4.79	30	10.80	4.54	8.27	3.27	.004**
Depression	80	8.76	4.26	30	8.83	4.08	8.10	3.86	*ns*
GDS	336	7.62	3.86	50	8.82	3.32	8.00	4.36	*ns*
MMSE	405	18.44	6.42	77	20.18	5.09	22.91	5.30	.000***
MoCA	89	16.96	7.11	37	16.30	6.27	19.19	6.22	.000***

* p <.05, ** p <.01, *** p<.001

Mood & cognitive functioning upon admission and prior discharge

Upon the samples with both T1 and T2 measures available, results showed that there was a significant difference between the HADS total scores of T1 (M_{T1}=19.58, SD_{T1}=7.68) and T2 (M_{T2}=16.13, SD_{T2}=6.12), t(30), p < .01. The effect size (d) was .50 (large). Significant difference was also found for the anxiety subscale (M_{T1}=10.80, SD_{T1}=4.54; M_{T2}=8.27, SD_{T2}=3.27), t(29), p < .01, d=.64 (large), but not for the HADS depression subscale (M_{T1}=8.83, SD_{T1}=4.09; M_{T2}=8.10, SD_{T2}=3.86), t(29), *ns*, d=.18 (small). No significant difference was found for GDS between T1 (M_{T1}=8.82, SD_{T1}=3.32) and T2 (M_{T2}=8.00, SD_{T2}=4.36), t(49), *ns*, d=.21 (small).

Results suggested that there was an improvement on cognitive functioning when patients were discharged from the hospital as compared with their admission status. Significant differences between T1 (M_{T1}=20.18, SD_{T1}=5.09) and T2 (M_{T2}=22.91, SD_{T2}=5.30) were found for MMSE, t(76), p < .001, d= -.53 (large); and MoCA (M_{T1}=16.30, SD_{T1}=6.27; M_{T2}=19.19, SD_{T2}=6.22), t(36), p < .001, d= -.46 (moderate).

Outcomes of treatment groups

Among those patients who completed mood intervention, only 21 (38% of 55 treated patients) of them have completed both pre- and post-treatment measures (n=5 for HADS, and n=16 for GDS) of both mood and cognitive scales. For those completed neurocognitive intervention, 24 entries were available (n=14 for MMSE, n=10 for MoCA; some patients might contribute to both scores; 74% of 19 treated patients). Table 3 showed the outcomes of patients who received intensive mood treatment and neurocognitive treatment.

Mood. For HADS total scores, there was a significant main effect of time, F(1,11)= 6.75, p <.05, but the time x treatment type interaction effect was not significant, F(1,11)=.36, *ns*. The effect size (d), as calculated by comparing the change in score (T2-T1) between the treatment group and control group (neurocognitive treatment group as active control), was .31 (moderate) (same applied to subsequent effect size reporting; see Table 4). There was a significant main effect of time, F(1,11)= 6.89, p <.05, but no interaction effect was found for the anxiety subscale, F(1,11)= .37, *ns, d*= .32 (moderate). No significant main effect of time (F(1,11)= 2.01, *ns*) and interaction effect (F(1,11)= .11, *ns*) was found for the depression subscale. The effect size (d) was .17 (small). The main (F(1,19)= 2.91, *ns*) and interaction effect (F(1,19)= .56, *ns*) for GDS was not significant, with effect size (d) of .42 (moderate). No significant differences of T2-T1 were revealed by t-test for all mood measures.

Cognitive functioning. There was a significant main effect of time (F(1,33)= 28.49, p <.01) but not time x treatment interaction effect (F(1,33)= .72, *ns*) for MMSE. The effect size (d) was .31 (moderate). Similarly, the main effect of time for MoCA was significant, F(1,18)= 31.05, p <.01, while the interaction effect was marginally significant, F(1,18)= 3.63, p= .073. The effect size (d) was .85 (large). No significant differences of T2-T1 were revealed by t-test for all neuropsychological measures.

Outcomes and treatment intensity

It was found that while HADS depression score at baseline (T1) predicted 27.3% of the treatment outcome (T2 score), F (1, 25)= 9.33, p <.01, treatment intensity predicted an additional 13.7% of variance, F (2,24)= 8.29, p <.01. The β for T1 HADS depression score was .47, p <.01; the β for treatment intensity was -.37, p <.05. The sample was too small for any meaningful analysis for GDS.

Dosage effect was not found for neurocognitive intervention.

Table 3
Results of Within-subject ANOVAs (pre-post vs treatment-active-control) for Mood and Neurocognitive Treatments

	Treatment				Control				Time			Time x Treatment		
	T1		T2		T1		T2		*df*	*F*	*p*	*df*	*F*	*p*
	N	M *(SD)*	*N*	M *(SD)*	*N*	M *(SD)*	*N*	M *(SD)*						
Mood Treatment														
HADS	5	22.20 (7.82)	5	17.00 (3.54)	8	19.38 (7.67)	8	16.13 (5.64)	1,11	6.75	.025*	1,11	.36	*ns*
Anxiety	5	11.60 (4.78)	5	8.40 (3.05)	8	10.88 (3.91)	8	8.88 (2.17)	1,11	6.89	.020*	1,11	.37	*ns*
Depression	5	10.60 (3.44)	5	8.60 (3.58)	8	8.50 (4.72)	8	7.25 (3.81)	1,11	2.01	*ns*	1,11	.11	*ns*
GDS	16	9.94 (2.77)	16	9.31 (3.14)	5	7.80 (3.96)	5	6.20 (5.59)	1,19	2.91	*ns*	1,19	.56	*ns*
Neurocogitive Treatment														
MMSE	14	20.50 (3.67)	14	24.50 (3.08)	21	19.67 (4.16)	21	22.57 (5.55)	1,33	28.49	.00***	1,33	.72	*ns*
MoCA	10	16.20 (4.13)	10	21.30 (3.23)	10	13.90 (5.92)	10	16.40 (6.67)	1,18	31.05	.00***	1,18	3.63	.073 (*ns*)

* p <.05, ** p <.01, *** p<.001

Table 4
Comparison of Score Differences (T2-T1) in Mood and Neurocognitive Measures with Effect Sizes

	Treatment		Control					
	N	M *(SD)*	*N*	M *(SD)*	*df*	*t*	*p*	Cohen's *d*
HADS	5	-5.20 (8.04)	8	-3.25 (3.77)	11	.60	*ns*	.31
Anxiety	5	-3.20 (4.60)	8	-2.00 (2.62)	11	.61	*ns*	.32
Depression	5	-2.00 (5.29)	8	-1.25 (3.06)	11	.33	*ns*	.17
GDS	16	-0.63 (2.70)	5	-1.60 (1.82)	19	-.75	*ns*	.42
MMSE	14	4.00 (2.51)	21	2.90 (4.37)	33	.85	*ns*	.31
MoCA	10	5.10 (3.18)	10	2.50 (2.92)	18	1.91	.073 (*ns*)	.85

* p <.05, ** p <.01, *** p<.001

Table 5
Hierarchiacl Regression of T2 HADS Depression score on Baseline (T1) score and Treatment Intensity

	B	*SE(B)*	β	$\triangle R^2$
Step 1				.272**
T1 HADS depression score	.48	.16	.52**	
Step 2				.137**
T1 HADS depression score	.44	.15	.47**	
Treatment intensity	-.31	.13	-.37*	

* p <.05, ** p <.01, *** p<.001

Discussion

Change in mood and cognitive functioning upon discharge

Subacute rehabilitation is a vital stage for recovery among stroke patients. It is encouraging to see that from the selected sub-samples of an in-patient cohort over two years, promising improvement in mood and neurocognitive functioning from the time of admission to discharge was evident among HADS, MMSE and MoCA results (but not in GDS). These positive changes reflected clinical value for screening for early intervention in both mood and neurocognitive problems. It would be ideal to study the relationship between the improvement in mood or cognitive functioning, and other outcomes such as motor functioning, adaptive daily living (ADLs), known protective factors such as family support, medical conditions. To enable such comparison and analysis, a protocol of standard outcome matrix would be needed in the future.

Limitations

Input from clinical psychology service suggested small effect size for mood treatment and medium to large effect size for neurocognitive intervention, although only some of the comparisons reached statistical significance given the small sample sizes. The lack of randomized control also limited the interpretation of the findings, as the present active control group could only balance the attention effect (both receiving clinical psychology service) but their presenting issues were very different. For ethical reason, it is not feasible to have a waitlist control for identified cases as the length of stay of each patient could not be extended for the sake of research design in public hospitals. A possibility is to compare the outcomes in another hospital of a similar size where it does not provide screening and early intervention programme. The limitation of resources including low psychologist-to-patient ratio may affect the effectiveness for the treatment as evidenced by the treatment dosage effects in mood treatment. The low PCA-to-patients-ratio has limited the completion rate of T1 and T2 measures.

Future directions

Treatments usually took place when the patients were hospitalized, and most often only short-term treatment with limited sessions could be provided. The impairments, however, lasted longer than patients' stay in hospital. For example, Jokinen and colleagues (2015) reported that even for stroke patients with satisfactory physical recovery at discharge, cognitive impairment was still detected in 71% of these patients in subsequent 3-month follow-up. Besides cognitive recovery, mood disturbance may still persist as patients will be facing adjustment issues post-stroke (Moncayo et al., 2008). At present, only a small percentage of patients could receive post-discharge care such as day-hospital rehabilitation or specialized out-patient clinic service. There is no existing research data on the long term functioning of those discharged patients in Hong Kong. The essence of screening and early intervention, for example, the iMAN scheme, ensures indicated patients would not be missed out for support and training during their stay in hospital.

Aphasic patients did not benefit much from self-reported screening as they have lost their abilities to communicate verbally and often associated with difficulties in reading and writing. The 10-item version of the Stroke Aphasic Depression Questionnaire (SADQ-10) was used in iMAN scheme and their ward observed behaviours were rated by duty nurse. Using the cut-off of five as suggested by Bennett, Thomas, Austen, Morris & Lincoln (2006) , less than 20% of patients were indicated. Such percentage was much lower than that of non-aphasic stroke patients suggested possibility of under-reporting of depression in patients with aphasia. Research in local validation of mood screening for aphasic stroke patients is highly needed.

A rather weak effect size for talk therapy, namely supportive counseling and cognitive behavioral therapy, to stroke patients with mood problems was revealed. Apart from the limitations discussed above, it is also possible that conventional psychotherapy may not be as effective as expected for treating stroke patients with depression (e.g. Lincoln & Flannaghan, 2003; also see NICE, 2013). Alternatively, other mood treatment modality may be considered. For instance, transcranial direct current stimulation (tDCS) was reported to be effective in reducing depressive symptoms in a large randomized-controlled trial (Brunoni et al., 2013), and was recently recommended by NICE (2015) as a safe and effective means of treatment for depression. The technique is yet to apply to both aphasic and non-aphasic stroke patients with mood symptoms, and warrants further research.

The current study has shown mood and neurocognitive improvements among stroke patients from admission to discharge, and clinical psychology service is found to have contributive effect. Long-term follow-up would be desirable to document the rehabilitation trajectory and identify late emergence of crises. The tDSC may be considered to increase the effectiveness of mood treatment adjunct to conventional psychopharmacological and psychological interventions.

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摘要

中風病人在復康醫院的綜合情緒認知甄別評估與及早治療初步結果

中風後患者往往會出現需受關注的情緒及認知障礙。本研究旨在探討心理甄別評估在中風復康住院的成效，在二零一四年四月至二零一六年三月間共招募了457個中風住院病人，並在評估後為其中有需要的病人提供臨床心理服務。結果指出在出院時病人的情緒及認知能力整體有臨床上的顯著進步。通過提供心理治療或腦神經復康訓練，臨床心理服務為病人的復康帶來正面的影響。文末亦探討了在病人出院後作長期跟進的臨床價值，以及談話治療以外的其他心理治療模式。

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