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QUESTION

Question: Can escitalopram or problem solving therapy prevent post-stroke depression?

Patients: 176 non-depressed participants within 3 months of haemorrhagic or ischaemic stroke. Exclusions were DSM-IV based major or minor depression (research criteria); >11 on the Hamilton-17 Depression Rating Scale; severe neuropsychological deficits; other life threatening medical conditions; neurodegenerative disorders; and DSM-IV alcohol or substance abuse or dependence within the past 12 months.


Intervention: One of three 2 month regimens: escitalopram (10 mg/day if <65 years old, 5 mg/day if ≥65 years old; taken in the morning), placebo pill or problem solving therapy (PST) (six sessions over the first 12 weeks and six reinforcement sessions up to 12 months). PST was delivered by therapists who had completed training in the manual based intervention and all sessions were videotaped and assessed for adherence.

Outcomes: Major or minor depression (defined as meeting DSM-IV criteria for either of these diagnoses, as assessed with the Structured Clinical Interview for DSM-IV, and having a score >11 on the Hamilton-17 Depression Rating Scale).

Patient follow-up: 149 of the 176 randomised participants (85%) began treatment; 15/149 (10%) dropped out; 100% were analysed. There were no differences between those that completed the study and those that dropped out in terms of characteristics at baseline.

METHODS

Design: Randomised controlled trial.

Allocation: Concealed.

Blinding: Drug treatment was double blind; PST was single blind (assessors blinded).

Follow-up period: 12 months (treatment period only).

MAIN RESULTS

Escitalopram and PST reduced the risk of depression compared with placebo, following adjustment for past history of depression (AR for developing depression: 8.5% with escitalopram vs 11.9% with PST vs 22.4% with placebo; adjusted HR for escitalopram vs placebo: 4.5, 95% CI 2.4 to 8.2; p < 0.001; adjusted HR for PST vs placebo: 2.2, 95% CI 1.4 to 3.5; p < 0.001; proportional hazards Cox regression model). These differences remained after further adjustment for potential confounders (eg, age, sex and severity of impairment). A conservative intention to treat analysis that considered all participants randomised but not entered into treatment to have developed depression also found that escitalopram was associated with lower risk of depression but that PST was not different from placebo.

CONCLUSIONS

Escitalopram and PST are effective in preventing the development of depression following stroke.

NOTES

There were no differences between those that completed the study and those that dropped out. The single blind nature of the PST was compromised by all participants telling raters of their group allocation.

ABSTRACTED FROM


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Post-stroke depression (PSD) is an important and common consequence of stroke and may affect functional outcome. Today, interventions to prevent the development of PSD are still inconclusive. Recently, Robinson and colleagues reported a favourable effect of escitalopram and partially of problem solving therapy in preventing PSD. The study is very intriguing. However, some issues may limit generalisation of the results. In particular, there are some discrepancies with several features from other studies with case series of stroke survivors. The first discrepancy is related to the mean age of the sample, which seems particularly low (especially for a group treated with escitalopram, 61.3 years). It is not only lower than that found in other similar studies (as also reported by the authors) but also compared with mean age of stroke onset in several stroke registries. 2 3

The second point is related to the percentage of patients with bilateral white matter lesions (WML) (nearly 38% of the sample), which seems unusually high. Even if WMLs, essentially associated with small vessel disease, are frequent in stroke patients, it should be taken into account that they could be asymptomatic or a predictor of risk for subsequent stroke. The third point is related to the degree of disability, which is very mild in this series, as revealed by the baseline FIM score (nearly 115 out of maximum score of 126). Because disability is recognised as a relevant factor for developing PSD, this case series arguably does not represent the optimal target for studying this problem.

In conclusion, Robinson’s data suggest that, in selected cases, it is possible to prevent the development of PSD. Further studies, analysing a more appropriate case series, are necessary to generalise these results.

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