63 - Treatment of post stroke depression

Treatment of post-stroke depression

Depression and anxiety disorders CHAPTER 3 Post-stroke depression Depression is a well--established risk factor for stroke.1,2 In addition, depression is seen in at least 30–40% of survivors of stroke3,4 and post-stroke depression is known to slow functional rehabilitation.5 Antidepressants may reduce depressive symptom severity6 and thereby facilitate faster rehabilitation.7 They may also improve global cognitive functioning,8,9 enhance motor recovery10 and even reduce mortality.11 Despite these benefits, post-stroke depression often goes untreated.12 Prophylaxis of post-stroke depression The high incidence of depression after stroke makes prophylaxis worthy of consideration. Pooled data suggest a prophylactic effect for antidepressants.13,14 Nortriptyline, fluoxetine, escitalopram, duloxetine, mirtazapine and sertraline appear to prevent post- stroke depression.13,14 A large cohort study suggested that mirtazapine and venlafaxine may be associated with an increased risk of a new stroke compared with SSRIs or TCAs.15 Mianserin seems ineffective in the treatment of post-stroke depression.16 Amitriptyline17 and duloxetine are effective in treating central post-stroke pain. Routine use of antidepressants for the prevention of post-stroke depression is, however, not recommended.18 A Cochrane review19 suggests that there may be a benefit but regards the supporting evidence as poor. RCTs published since the Cochrane review suggest that the risks of prescribing fluoxetine (bone fractures, falls, seizures) may outweigh uncertain reduction in incident depression.20–22 Prophylactic use may be justified in individual cases. Treatment of post-stroke depression Treatment is complicated by medical comorbidity and by the potential for interaction with other co-prescribed drugs. Antidepressants that enhance serotonin release can be expected to reduce platelet adhesion and increase the risk of bleeding, especially when co-prescribed with aspirin or anticoagulants (see section on SSRIs and bleeding in this chapter). There is also potential for pharmacokinetic interaction between antidepressants and anticoagulants. Overall, contraindication to antidepressant treatment is more likely with tricyclics than with SSRIs.23 Fluoxetine,24,25 citalopram/escitalopram8,26–28 and nortriptyline29,30 are probably the most studied31 and seem to be effective and safe in respect to pharmacokinetic interaction. RCTs also support the use of other antidepressants, including sertraline,27 mirtazapine32 and agomelatine.33 Reboxetine (which, like nortriptyline, does not affect platelet activity) may also be effective and well tolerated,34 although its effects overall are doubtful.35 Vortioxetine may be of particular benefit because of its additional benefits for

cognition, independent of any effects on depressive symptoms. It also does not appear to adversely affect cardiovascular parameters or interact with warfarin or aspirin, but data to support its use specifically in post-stroke depression are limited to a single pilot study.36 Despite their anti-platelet effects, SSRIs seem not to increase risk of stroke37,38 (at least post-stroke), although some doubt remains.39,40 Depression itself increases the risk of stroke, so initiation of an antidepressant post-stroke may appear to be associated

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