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Medical Uses and Adverse Effects of Paroxetine in Anxiety Disorders

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Description

Paroxetine is an effective and Selective Serotonin Reuptake Inhibitor (SSRI) with currently, it was approved for the treatment of depression, obsessive-compulsive disorder, panic disorder, and social phobia. It is also used in the treatment of generalized anxiety disorder, post-traumatic stress disorder, premenstrual disorders, and chronic headaches. Paroxetine, is derived from phenylpiperidine, which is the most potent serotonin (5hydroxytryptamine, 5HT) reuptake inhibitor than all available antidepressants, including the SSRI class. It is a very weak inhibitor of Norepinephrine (NE) approval. Paroxetine has slight affinity for the catecholaminergic, dopaminergic, or histaminergic systems, when compared with Tricyclic Antidepressants (TCAs), thus it tends reduce central and autonomic side effects. Paroxetine has specific affinity for cholinergic muscarinic receptors but more less for TCA. In addition, the adaptive changes in the somatodendritic (5HT1A) and terminal (5HT1B/1D) autoreceptors observed with paroxetine varied from those observed with TCA; it also inhibits nitric oxide synthesis.

Medical uses and adverse effects

Paroxetine acts as both a substrate and an inhibitor of the cytochrome P450 2D6 isoenzyme. Paroxetine is well absorbed orally and undergoes extensive first-pass metabolism and partially saturated. It metabolites are pharmacologically inactive in vivo. Stabilization is achieved after 4-14 days and a half-life of 21 hours is compatible with once-daily dosing. There is large inter-individual variation in the pharmacokinetics of paroxetine in adults as well as in the elderly and young, as increasing in plasma concentrations there is a slower elimination in the later stages. Elimination is also reduced in patients with severe renal and hepatic impairment. However, serious adverse effects are very rare, even in the result of an overdose. In summary, paroxetine is well accepted and effective in the treatment of depressive and anxiety disorders of all ages.

Paroxetine is almost completely absorbed after an oral dose. Its absorption is not affected by food or associated with the treatment with antacids. A first-pass effect has been described as becoming saturating and leading to higher bioavailability and non-linear pharmacokinetics after repeated dosing. With repeated use, steady-state concentrations of paroxetine are achieved within 7 to 14 days. There was no further accumulation of the drug. The distribution of paroxetine in the body is very extensive and consistent with its properties as it is lipophilic amine, only 1% of the drug remains in the systemic circulation.

The volume of distribution shows wide inter-individual variation, ranging from 3.1 to 28.0 Lkg after intravenous administration. At therapeutically relevant concentrations, paroxetine is 95% bound to plasma proteins; therefore, it should be prescribed with caution with other drugs which having high protein binding, eg warfarin. Paroxetine is extensively metabolised in rats, monkeys and humans by oxidation within the methylenedioxyphenyl ring to its major metabolites. Paroxetine is the most potent 5HT reuptake inhibitor of all available antidepressants. It is a very weak inhibitor of norepinephrine uptake, but is still more potent at this site than other SSRIs.

This paper concludes that paroxetine has shown to be effective in providing remission in all of the anxiety disorders studied. Our results suggest that the treatment with paroxetine (and possibly other SSRI antidepressants) for 2 to 12 months increases the proportion of patients in clinical remission.

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