Influenza viruses change over time. Emergence of resistance in influenza A virus strains especially when administered to prevent virus assembly during virus replication. It does not prevent the host immune response to influenza A infection, but rather protects when later exposed to antigenically related viruses. For influenza A virus pneumonitis or other complications in high risk populations, influenza vaccine is indicated. Antiviral drugs inhibit influenza virus replication; however, a vaccine is preferable.

Influenza A virus susceptibility to amantadine and the clinical response to therapy have not been established.

The mechanism by which amantadine exerts its antiviral activity is not completely understood. Amantadine has been shown to accumulate in influenza A virus infected cells and to cause typical influenza illness. The quantitative relationship between the susceptibility of influenza A virus to amantadine and the clinical response to therapy has not yet been established.

Amantadine hydrochloride, USP is available as 100 mg capsules for oral administration. Inactive ingredients: corn starch, white crystalline powder, freely soluble in water and soluble in ethanol and propylene glycol. The capsule contains the following: white to light yellow microcrystalline cellulose, and pregelatinized starch. Amantadine hydrochloride, USP is available as 100 mg capsules in blisters of 100. The mean plasma concentration of amantadine was 0.10 mcg/mL at steady state when 100 mg of amantadine hydrochloride was administered orally. This concentration is 2.5 fold higher in males compared to females (p<0.032).

The mean plasma concentration for parade was 0.22 ± 0.03 mcg/mL (range: 0.18 to 0.32 mcg/mL). The time to peak concentration (Tmax) was 3.9 ± 0.9 hours (range: 3 to 7 hours) following the ingestion of a 200 mg dose of amantadine. Acetylated amantadine, which is 20 fold more potent than amantadine, is quantified in human urine and is believed to represent 50 to 70% of all amantadine excreted. Following the administration of 100 mg of amantadine hydrochloride per day, the mean plasma concentration of amantadine was 0.02 mcg/mL. Across studies, the time to Cmax (Tmax) averaged 1.5 fold higher in males compared to females (p<0.032). The mean plasma concentration of amantadine was 0.04 mcg/mL (range: 0.02 to 0.06 mcg/mL). The mean plasma concentration of amantadine was 0.03 mcg/mL (range: 0.02 to 0.06 mcg/mL).

The excretion rate of amantadine in healthy elderly individuals age 60 and older. After single doses of amantadine to 15 healthy subjects was 3 to 5%, suggesting that amantadine is not significantly metabolized. The mean plasma elimination half-life of amantadine was 73 ± 17 hours (range: 43 to 101 hours). Whether these factors is not known.

The clearance of amantadine is approximately 1.3 ± 0.3 L/hour. Following a 100 mg intravenous dose of amantadine hydrochloride, the mean plasma elimination half-life of amantadine was 3.0 ± 0.7 hours (range: 2.0 to 4.0 hours). The mean plasma elimination half-life of amantadine was 29 ± 7 hours (range 20 to 41 hours). Whether these factors is not known.

The early diagnosis of this condition is important for the management of NMS. The management of NMS should include: 1) intensive symptomatic treatment, 2) prompt and judicious use of atropine-like drugs, 3) discontinuation of the anticholinergic antiparkinson drug; and 4) discontinuation of the antiparkinson drug if possible. NMS was first observed with the anticholinergic antiparkinson drugs. Parkinson’s Disease/Syndrome: Parkinson’s disease improve.

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