



ISONIAZID IS ONE OF THE FIRST-LINE DRUGS USED IN TUBERCULOSIS

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Annotation

Tuberculosis (TB) is a chronic granulomatous disease caused by *Mycobacterium tuberculosis*. *Mycobacterium avium* complex (MAC) infections are not only more common but also rapidly progress in these patients. First-line drugs are superior in efficacy to second-line drugs. Most patients can be treated successfully with these drugs.

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Isoniazid (INH). It is the most effective and cheapest primary antitubercular drug. It is effective both in acidic and alkaline medium. INH is tuberculocidal for rapidly multiplying bacilli but static for resting bacilli. INH destroys: Intracellular bacilli as it freely penetrates into the cells, i.e. tubercle bacilli in macrophages, and bacilli multiplying in the walls of the cavities. Thus it is effective against both intra- and extracellular organisms. If used alone, mycobacteria develop resistance to it. Hence, it should be used in combination with other drugs.

Mechanism of Action. INH inhibits the synthesis of mycolic acids which are important components of the mycobacterial cell wall. The cell wall of mycobacteria differs from other bacteria in having large amounts of mycolic acids which form essential components of mycobacterial cell wall. INH, a prodrug, freely enters the mycobacteria and is converted to an active form by an enzyme catalase-peroxidase (Kat G) present in the mycobacteria. This active form covalently binds certain enzymes and thereby inhibits mycolic acid synthesis. Resistance to INH is seen



when there is over production of the enzymes that are inhibited by INH. Mutations of inhA and Kat G enzymes also result in resistance.

Pharmacokinetics. INH is completely absorbed orally, penetrates all tissues, tubercular cavities, ascitic fluid, necrotic tissues, caseous material and CSF. It is metabolised by acetylation and this is genetically determined. Patients can be fast or slow acetylators depending on the genetic inheritance—slow acetylators responding better. The $t_{1/2}$ in slow acetylators is 3–5 hours while in fast acetylators it is 1 hour. Peripheral neuropathy is more common in slow acetylators while hepatotoxicity is more likely in fast acetylators. If INH is given once weekly in fast acetylators, adequate therapeutic concentrations may not be attained. Metabolites of INH are excreted in the urine.

Adverse Effects. Peripheral neuritis due to interference with utilization and increased excretion of pyridoxine can be avoided by giving prophylactic pyridoxine (10–50 mg) with INH. However, it is uncommon with therapeutic doses and is seen in higher doses with an incidence of 10– 20% and in patients with comorbid conditions like AIDS, diabetes and malnutrition. Hepatitis is another major adverse effect, more common in alcoholics and in the elderly. If hepatitis is mild, INH may be continued, but in a small percentage of patients. INH can cause hepatic necrosis with anorexia, nausea, vomiting and jaundice—can sometimes be fatal. In such patients with the first signs of hepatic necrosis, INH should be withdrawn. INH can cause CNS toxicity including psychosis and seizures but is less common— epileptics are more prone to this effect and pyridoxine helps these patients. Other minor effects like anorexia, gastrointestinal discomfort, fever and allergic reactions can occur. Haemolysis can occur in patients with G6PD deficiency.

In Conclusion,isoniazid is a bactericidal agent active against organisms of the genus Mycobacterium, specifically M. tuberculosis, M. bovis and M. kansasii. It is a highly specific agent, ineffective against other microorganisms. Isoniazid is bactericidal when mycobacteria grow rapidly and bacteriostatic when they grow slowly.



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