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Opinion Article

Anti-Tubercular Drug-Induced Multi-Organ Toxicities

Shui Kurokiwa*

Department of Cardiovascular Science and Medicine, Chiba University of Medicine, Chiba, Japan

DESCRIPTION

An ailment called tuberculosis kills millions of people worldwide and affects about one-third of the world's population. The highest mortality and morbidity rates worldwide are also attributed to it. According to statistics from 2019, this illness claimed the lives of 1.4 million individuals overall (WHO). However, the incidence of TB is declining globally at a rate of 2% per year as a result of effective management and treatment using the DOTS (Directly Observed Treatment, Short Course) strategy. According to the World Health Organization (WHO), TB patients should receive medication treatment for six months. A two-month "intense" therapy phase should come after which there should be a four-month "continuation" phase. Isoniazid (INH), Rifampin (RIF), Ethambutol (EMB), and Pyrazinamide are the first-line Anti-Tubercular Medicines (ATDs) utilised in the DOTS strategy for humans (PZA). Past research has shown that ATD are cytotoxic in nature and can interfere with normal tissue function. Hepatotoxicity is one such frequent side effect of the treatment for ATDs.

Numerous studies have been published in the literature that point to the possibility that ATDs could harm the liver and result in hepatitis. ATDs also cause nephrotoxicity, neurotoxicity, phototoxicity, ocular toxicity, and reproductive toxicity in addition to hepatotoxicity. DOTS is a World Health Organization-recommended interventional technique that combines various drug combinations to treat tuberculosis. The mortality rate has dropped since it is the most effective and economical way. The majority of the classifications for the various drug types utilised in the DOTS method are based on how well they work against Mycobacterium tuberculosis. Previous research has demonstrated that the first line ATDs' potential for increased cytotoxicity when used in combination.

Anti-tubercular medication therapy generates oxidative stress, which releases free radicals, primarily Reactive Oxygen Species (ROS), which damage mitochondria and activate the apoptotic pathway by releasing cytochrome C from mitochondria. The main reasons for the histological changes brought on by anti-tubercular medications in hepatocytes include oxidative stress, mitochondrial malfunction, and activation of the apoptotic pathway by ATDs. Anti-tubercular medicines' reactive metabolites produce free radicals, which then promote lipid peroxidation, which destroys membrane lipids, the endoplasmic reticulum, and other structures that are rich in Polyunsaturated Fatty Acids (PUFA).

Malonaldehyde (MDA), which can damage hepatocytes and induce a loss of cell membrane integrity, will be produced as a result. Due to enhanced lipid peroxidation in the anti-tubercular drug-treated group of rats, the toxicant group develops an anti-oxidant defence mechanism to eliminate all free radicals brought on by the drug. The liver has a remarkable capacity for toxin elimination, however on the flip side; same toxin removal also harms the liver's cells. Hepatocyte cell membrane oxidative damage will result from the high susceptibility of anti-tubercular medicines to hepatocyte cell membrane. Along with INH and RIF, the PZA is linked to a rise in the frequency of toxicity. In all animals given anti-tubercular medication, liver damage and patchy necrosis result. In groups treated with drugs, the current investigation also found liver damage with patchy necrosis. In DS groups, the combined use of first-line anti-tubercular medications was more responsible for the deterioration. Drug-sensitive populations produce more micro nucleated and apoptotic cells in the bone marrow.

Correspondence to: Shui Kurokiwa, Department of Cardiovascular Science and Medicine, Chiba University of Medicine, Chiba, Japan, E-mail: Kurokiwa@gmail.com

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