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Commentary

Prospects for Drug-Induced Liver Injury

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DESCRIPTION

Drug Induced Liver Injury (DILI) is caused by drugs (prescription or over-the-counter), herbs and dietary supplements, or other heterologous organisms that cause liver test abnormalities or liver dysfunction. It cannot be explained by any other cause. There are two types of drug induced liver injury they are endogenous and specific. Endogenous drug induced liver injury refers to predictable and dose-dependent hepatotoxicity induced by drugs such as acetaminophen. The infrequent idiosyncratic DILI is associated with inconsistent dose toxicity relationships and a wider variety of symptoms.

The true prevalence of DILI is difficult to determine because premarket clinical trials are too weak to demonstrate the presence of idiosyncratic DILI. However, the annual incidence of DILI is estimated to be 10 to 15 per 10,000 to 100,000 people taking prescription drugs. As a result, about 44,000 people in the United States experience DILI annually, which is not only for human exposure but also for medical costs. This prevalence is expected to increase with the spread of HDS.

It occurs as an unpredictable event. Drugs can be harmful to the liver of sensitive people due to genetic and environmental risk factors. These risk factors alter hepatic metabolism and excretion of DIL pathogens, leading to inability to adapt to cell stress, cell death, activation of adaptive immune responses and progression to overt liver damage. Idiosyncratic DILI is a relatively rare liver disease, but it is severe, sometimes fatal and can exhibit a variety of phenotypes that mimic other liver diseases. The diagnosis of DILI is based on the exclusion of other causes of liver disease, as certain biomarkers are still deficient. Clinical scales such as CIOMS/RUCAM can support the diagnostic process but need improvement. Several clinical variables validated in future cohorts can be used to predict more serious DILI outcomes. Although there is no well-tested pharmacological treatment in randomized clinical trials, corticosteroids may be useful in new forms of DILI, especially related to immune checkpoint inhibitors in cancer patients.

Although DILI is rare, a predisposition has been identified that may increase a patient's risk of developing DILI. Patient risk factors that may contribute to DILI include genetics, age (aged and younger), gender (female), race, pregnancy, malnutrition, intestinal micro flora, hormonal status, obesity, and diabetes mellitus. It includes pre-existing liver disease or comorbidities including HIV and indications for treatment (eg, hepatitis C). Environmental factors that may increase the risk of 2, 3, 2225 DILI include smoking, alcohol consumption, infections or inflammatory episodes.

DILI includes the whole spectrum from asymptomatic and transient elevation in liver tests to ALF. DILI due to paracetamol overdose and idiosyncratic drug reactions remain the most common causes of ALF in the western world. DILI is a diagnosis of exclusion and there is an urgent need to develop diagnostic biomarkers. Antimicrobials remain the most common cause of idiosyncratic DILI although recent studies show a significant increase in DILI due to herbal and dietary supplements. Hepatocellular DILI is more likely to progress to ALF compared with cholestatic or mixed DILI. DILI management is symptomatic with rapid discontinuation of the causative agent and early referral of liver transplantation after the onset of ALF, especially in patients with specific DILI.

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