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Perspective

Association of Pediatric Acute Lymphoblastic Leukemia and Hyperbilirubinemia in Chemotherapy

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DESCRIPTION

By using current treatment protocols, the majority of children with Acute Lymphoblastic Leukemia (ALL) can be cured. Acute and long-term negative effects, however, which may complicate therapy delivery or have an impact on health and quality of life both during and after treatment, outweigh the benefits of survival. Hepatotoxicity is a well-known consequence that usually occurs in the initial stages of ALL treatment and is primarily assessed in clinical trials by evaluation of hyperbilirubinemia and transaminasemia. Surprisingly, there aren't many thorough papers documenting the prevalence and effects of hepatotoxicity on the general course of pediatric ALL. During the course of treatment, 527 people (28%) out of 1872 pediatric ALL patients treated with the ALL IC-BFM 2002 regimen experienced 934 grade 3 or higher hepatotoxicity episodes, according to the National Cancer Institute's (NCI) Common Toxicity Criteria. According to a prior single institution study, hyperbilirubinemia rarely affected Event-Free Survival (EFS), but treatment changes, such as delays, were frequently made. When exposed to certain anti leukemic medications, such as L-asparaginase or antimetabolites, high total serum bilirubin levels - with or without transaminasemia - may indicate liver dysfunction. By identifying risk-associated genetic variations, Genome-Wide Association Studies (GWAS) have made it possible to more precisely quantify the risk of various diseases, including pediatric ALL. In the current study, the clinical effects of hyperbilirubinemia in a sizable cohort of pediatric ALL patients and used a GWAS approach to find genetic variations impacting chemotherapy-related hyperbilirubinemia were assessed.

In clinical trials for pediatric ALL, routine hepatotoxicity monitoring is carried out by classifying increased bilirubin and liver transaminase levels as being absent, mild, moderate, severe, or life-threatening/fatal in accordance with the NCI CTC criteria. Although it is usual practice to evaluate laboratory values for hepatotoxicity, it is debatable how well such measurements represent liver disease. Alternative classifications incorporate more clinical data to improve the phenotypic characterization in relation to aberrant laboratory readings. Similar to this, genetic biomarkers have the potential to support techniques for a better assessment of hepatotoxicity. Only a small number, nevertheless, are now utilized in clinical practice to direct genotype-adapted dose of particular chemotherapeutic drugs and hence minimize side effects. Regular hepatotoxicity monitoring in pediatric ALL clinical trials involves categorizing elevated bilirubin and liver transaminase levels as being absent, mild, moderate, severe, or life-threatening/fatal using the NCI CTC criteria. Although assessing test results for hepatotoxicity is common practice, it is questionable how accurately such assessments reflect liver illness.

More clinical information is incorporated into alternative classifications to enhance phenotypic characterization in connection to abnormal laboratory results. Similar to this, genetic biomarkers may support methods for a more accurate evaluation of hepatotoxicity. Though few, they are being used in clinical settings to target genotype-adapted doses of specific chemotherapy medicines and hence reduce side effects. It is widely known that genetic variation in UGT1A influences enzymatic glucuronidation activity and modulates the metabolism of numerous xenobiotics as well as endogenous metabolites. Only UGT1A1 out of nine functional isoforms is important for bilirubin glucuronidation. The phenotype of heritable illnesses of bilirubin metabolism is determined by various functional UGT1A1 mutations that cause a partial or total reduction in enzyme activity. The (TA) 7 variant allele UGT1A1*28, an insertion polymorphism in the TATA box of UGT1A1, is the most frequent genetic cause of reduced bilirubin conjugation. Homozygosity for this allele confers a reduced transcriptional activity of 18% to 33%, which corresponds to the residual glucuronidation activity of 30%found in patients with Gilbert's syndrome (GS).

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