

Chemotherapy and Immune Suppression

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In areas where hepatitis B virus (HBV) is endemic, the increased use of cytotoxic or immunosuppressive therapy and bone marrow transplantation (BMT) has resulted in an increased incidence of liver-related morbidity and mortality due to HBV reactivation in patients infected with the virus. As the hepatitis is preceded by HBV virological reactivation, administration of effective anti-viral therapy to HBV (anti-HBV) such as lamivudine pre-emptively before or at the time of conditioning regimen for BMT and to cover the entire period of immunosuppression, has greatly reduced the risk of liver-related morbidity and mortality due to HBV reactivation. However, such an early, "pre-emptive" approach runs the risk of over-treating patients who might not be suffering from HBV reactivation with nucleoside analogue. In addition, the duration of therapy with nucleoside analogue, such as lamivudine, would be longer with this approach. Taken together, such an indiscriminant pre-emptive approach could result in an increased risk of developing HBV viral resistance. Indeed, severe liver damage due to the development of mutations in the polymerase gene related to lamivudine, namely at M204V and at L180M, has been reported in hepatitis B surface antigen (HBsAg)-positive recipients of allogeneic bone marrow transplantation who were treated with pre-emptive lamivudine. In order to further optimize the management of post-chemotherapy HBV reactivation, more studies aimed at identifying the risk factors for HBV reactivation after chemotherapy should be undertaken.