Hepatitis B screening prior to rituximab and subsequent management to reduce the risk of reactivation

PTU-089

Hepatitis B (HBV) reactivation can occur during immune suppression in patients with serological evidence of current infection (HBsAg positive) or past exposure to HBV (HBsAg negative, anti-HBc positive). Rituximab poses a particular risk, with reported rates of reactivation >10% in those with positive HBV markers. Reactivation can result in delays to ongoing treatment and, in small numbers of cases, acute liver failure leading to transplantation or death.

Methods We performed a retrospective audit of screening for HBV markers prior to rituximab and management of patients at risk of HBV reactivation in an East London NHS Trust serving a multi-ethnic population. Our cohort was identified from a search of electronic records for a pharmacy order for rituximab placed in 201–018 across clinical specialties.

Results 461 patients were included, of whom 191 were male (41%). Ethnicity was as follows: British or Irish 169 (36.6%); Asian or Asian British 118 (25.6%); Black or Black British 44 (9.5%); Any other white background 44 (9.5%); Mixed 4 (0.9%); Chinese 1 (0.2%); Any other ethnic group 22 (4.8%); Not known 59 (12.8%). Screening was adequate in 384 patients (83.3%). 62 patients (13.4%) had undetectable HBsAg, but no record of anti-HBc. 2 patients (0.4%) were not tested for HBsAg, but were anti-HBc negative, making past/current infection unlikely. 13 patients (2.8%) had no record of either HBsAg or anti-HBc testing. 339 patients (72.7%) had undetectable HBsAg and anti-HBc. 3 patients (0.7%) tested positive for HBsAg, all of whom received appropriate antiviral prophylaxis. 42 patients (9.1%) tested positive for anti-HBc, with undetectable HBsAg. It was probable that passive transmission had occurred as a result of immunoglobulin infusions in 2 cases and was confirmed in 3 cases. Repeat anti-HBc was pending in 2 patients. 5 of these 7 patients received antiviral prophylaxis. Anti-HBc positivity was thought to be due to past HBV exposure in 35 patients (7.6%). One patient was already on Truvada for HIV infection. A further 27 patients received antiviral prophylaxis with either lamivudine, entecavir or tenofovir, although in 7 of these the prophylaxis commenced after rituximab infusions had started. No prophylaxis was given to 8 patients at risk of HBV reactivation based on serological markers (21% of at risk group). There were no episodes of reactivation during the audit period.

Conclusion In this audit of a multi-ethnic population receiving rituximab, we found that screening was adequate in only 83% of cases. Of those adequately screened, nearly 10% were at risk of hepatitis B reactivation with B-cell depleting therapies. 21% of those at risk did not receive appropriate prophylaxis. We propose cross-specialty guidelines and safety checkpoints in pharmacy and infusion units to reduce the risk of HBV reactivation in this patient group.