Vaccination for hepatitis B virus in an Australian pre-biologic population with rheumatoid arthritis

Sirs,

We read with interest the review article by Meroni et al. regarding vaccination in people with rheumatoid arthritis (RA). We recently audited Hepatitis B virus (HBV) vaccination rates in patients with rheumatoid arthritis commencing biologics in all 3 public tertiary hospitals in Perth, Western Australia (1).

Patients with RA commenced on biologic therapy between January 2011 and January 2016 were identified. A chart review of all patients (electronic records, hard copy notes and clinic letters) was performed. Subjects were included in the audit if they had RA, and were commenced on a biologic between the specified dates, and screening demonstrated patients did not have prior infection, carrier status or prior vaccination.

We identified 141 patients who commenced biologic therapy between January 2011 and January 2016. 36 patients (25%) had full HBV screening (HBsAg, antiHB, antiHBC), 32 patients (23%) had no evidence of HBV screening, 73 (52%) had incomplete HBV screening (missing anti HB), thus preventing immune status from being assessed. Of the 36 patients whose immune status could be assessed, 29 patients needed vaccination.

In all 29 patients, no evidence of document ed vaccination, or physicians’ recommendation for vaccination could be found. This audit demonstrated that routine vaccination with HBV of people with RA commencing biologic therapy in WA is not occurring. Meroni’s review article recommends vaccination people with RA without immunity who are at risk, however, the 2012 ACR guidelines recommend that before starting or receiving DMARDs or biological agents, all patients should receive vaccination against HBV (1). We suspect likely barriers to vaccination include a combination of cost of vaccination to the patient (which is unfunded in Australia), questionable efficacy (as discussed in your review), and low risk of incident infection in Australia.

Perhaps more concerning, our audit identified that for the vast majority of patients with rheumatoid commencing a biologic therapy, HBV screening is incomplete, meaning that low level carriers at risk of HBV reactivation are not being identified. As Meroni points out, the use of biologics in the setting of HBV infection or low level carriage has the potential to be catastrophic. The majority (75%) of patients with RA commencing biologics in our cohort could not retrospectively be determined to have had appropriate screening to determine their risk of reactivation or exacerbation of HBV, or need for a vaccination. Whilst this figure may seem exceedingly large, it is consistent with studies in inflammatory bowel disease (2). This finding is interesting given Meroni’s assertion that suboptimal vaccination rates reported in America and Europe are likely to be a result of a lack of physician recommendations. Our audit may suggest that a lack of physician education may also play a role. Our audit is relatively small and retrospective; some documentation may be missing from the public tertiary hospital records due to screening in the private health system. It is likely that the 23% of patients with no screening is an overestimation. However, partial screening is more likely to reflect an accurate figure, as it is unlikely that partial screening was carried out in the public setting, with further screening in the private sector.

In conclusion, in Western Australia, RA patients are not being screened for or vaccinated against HBV according to international consensus guidelines, and rheumatologists require education regarding both appropriate screening and vaccination to prevent HBV infection.

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References