P1249
Poster Session V
Immunology, vaccination and host defences

IGG2 ANTIBODY RESPONSE TO A PRIME-BOOST VACCINE STRATEGY COMBINING PCV13 FOLLOWED BY PPV23 VERSUS PPV23 ALONE IN HIV-INFECTED ADULTS

C. Sadlier[1], N. Conlon[2], C. Rock[1], A. Brown[1], S. O'Dea[1], J. Dunne[2], C. Bergin[1]

[1]St James's Hospital, GUIDE, Dublin, Ireland ; [2]St James's Hospital, Department of Immunology, Dublin, Ireland

Background:

Protection against *streptococcuspneumoniae* infection is based on humoral immune function and more specifically IgG2 subclassantibodies. Impaired production of IgG2antibodies in HIV-infected patients is thought to add to risk of pneumococcal infection which continues to cause significant morbidity and mortality in this patient group in the era of HAART.

In this single centre randomised controlled trial, we compared IgG2 subclass response to a prime boostvaccine strategy combining the 13-valent conjugate pneumococcal vaccine (PCV13) followed by the 23-valent polysaccharide pneumococcal vaccine (PPV23) versus PPV 23 alone in HIV-infected adults.

Methodology:

Pneumococcal vaccine naïve HIV-infected adults $\geq$18 yearswith CD4 count > 200 cells/mm^3 were randomised to receive PCV13at week 0 followed by PPV23 at week 4 (prime boost group, n=27) or PPV 23 alone atweek 4 (un-primed group, n=33).

IgG2 response to the 23 vaccine serotypes contained inPPV23 was measured at week 0, 8 and 28 using ELISA.

Fold increase in IgG2 geometric mean titre (GMT) and proportion of'responders' (patients with $\geq$2-fold increase in IgG2) postvaccination were measured and compared between vaccine groups. Wilkoxon and x^2tests were used to compare IgG2 levels and categorical variables as appropriate.**Results:**

60 patients (mean age [SD] 37 [9] years, 92% male, meanCD4 Tcell count 503 [209] cells/mm^3, 47% on HAART, mean HIV RNA4.5log$_{10}$ copies/ml) were recruited.

Overall IgG2 GMT [CI] increased significantly post-vaccination;

IgG 2 GMT week 0 = 12.08[9.82-14.86]ug/ml,

IgG 2 GMT week 8 = 63.60[51.84-77.52]ug/ml, (p<0.001)

IgG2 GMT week 28 = 33.08 [26.22-41.73]ug/ml, (p<0.001)

At week 8, 93% of the un-primed and92% of the prime-boost group had a $\geq$2 fold increase in IgG2.

Fold increase inIgG2 GMT in the un-primed versus prime-boost group was (mean [SD]; 6.35 [3.88] versus 7.04[6.77], p=0.64).

At week 28, 76% of the un-primed group and 68% of the prime-boost group had $\geq$2 foldincrease in IgG2 (p=0.56).

Fold increase in IgG2 GMT in the un-primed versus the prime-boostgroup was (mean [SD]; 3.55 [2.87] versus 3.56 [2.84], p=0.99).**Conclusions:**

93% ofstudy participants had a significant IgG2 response at week 8 however by week 28 the proportion of IgG2 responders (72%) along with IgG2 GMT(33.08ug/ml) decrease significantly (p=0.01 and <0.001 respectively). This indicatesa poor durability of IgG2 antibody protection in this patient cohort.

There was no detectable difference in magnitude or durability of IgG2 responsein the prime-boost versus the un-primed vaccine groups.

Clinical end-point trials are needed to clarify the optimal pneumococcalvaccine strategy and schedule in HIV-infected adults.