or atazanavir/ritonavir [ATZ/r] as the second option for IND/r-intolerant patients). Analysis was by intention-to-treat (ITT) and per protocol (OT), allowing changes of NNRTI or boosted PI within the same class of antiretrovirals.
**Results:** Sixty-six patients were randomised: 34 received an NNRTI-based regimen and 32 received a boosted PI-based regimen. 50% had had C events. Median (range) CD4+ and PVL at baseline were 40 (1–99) cells/mm3 and 5.5 (4.0–>6) log10/mL, respectively. All patients completed 3 years of follow-up. Five patients died (NNRTI, 4; PI, 1) and 12 developed a C event (NNRTI, 6; boosted PI, 6), most of them within the first six months. Seventeen patients changed EFV (NVP, 3; LPV/r 1) or IDV/r (LPV/r 9, ATZ/r 3, EFV 3, NVP 1) because of adverse events and eight patients stopped HAART or were lost for follow-up (NNRTI, 1; PI, 7). There were 12 virological failures (NNRTI, 5; PI, 7). PVL < 200 copies/mL for NNRTI/PI arms at 1, 2 and 3 years was 71%/65%, 68%/49% and 65%/29% ($p = 0.05$ at the last time point) by ITT (M=F) and 73%/77%, 73%/69% and 73%/49% (p=NS at all time points) by OT analysis, respectively. Median CD4+ cell increase after 1, 2 and 3 years by OT analysis was +186/+136, +226/+162 and +303/+220 cells/mm3 (p=NS for all time points) for NNRTI/PI groups, respectively. At 1 and 2 years, immune activation (CD8+CD38+) was significantly lower in the NNRTI group ($p = 0.004$). There were no differences in T-cell subsets, proliferative responses to mitogens and recall antigens between both study arms.
**Conclusions:** At 3 years, the virological response and the magnitude of the immune reconstitution induced by an NNRTI-based regimen was at least as potent as that induced by a boosted PI-based regimen.

### P1916 The evolution of the avidity of HIV-1-specific antibodies is prevented by early treatment started during primary HIV-1 infection

*M. Selleri, M. Zaniratti, N. Orchi, A. Corpolongo, P. Zaccaro, G. Ippolito, M. Capobianchi, E. Girardi (Rome, IT)*

**Objectives:** Avidity of anti-HIV antibodies progressively increases after primary HIV-1 infection (PHI). It has been reported that an Avidity Index (AI) <0.80 identifies recent (<6 months) infections, whereas anti-HIV antibodies with an AI $\geqslant$ 0.90 indicates long-standing infections. We evaluated if the administration of highly active antiretroviral therapy (HAART) during PHI may affect AI evolution.
**Methods:** The AI and Western blot (WB) evolution patterns were retrospectively analysed on serial serum/plasma samples from 13 individuals with symptomatic PHI, defined as detectable viral load and negative or indeterminate HIV-1 antibody tests. Five patients have never been treated, eight individuals had initiated HAART at the time of diagnosis (range 0–46 days after presentation), of whom, 4 discontinued HAART after a variable time lapse from PHI ($\geqslant$ 1 year).
**Results:** At diagnosis, the range of HIV viraemia was $0.003–38 \times 10^6$ copies/mL. In untreated patients viraemia reached the set-point in 4–6 months, while in all patients receiving HAART complete suppression of viraemia (<50 RNA copies/mL) was achieved early, lasting for the entire observation period (12 months). At presentation, the median AI was 0.42 (range 0.33–0.43) in untreated patients, and 0.44 (range 0.40–0.72) in subsequently treated patients. At 3, 6 and 12 months the median AI was 0.75, 0.89, 0.97 in untreated patients, and 0.42, 0.49, 0.54 in treated patients. In the 4 patients who stopped HAART, AI increased after interruption, reaching the value of 0.80 in 6 months. WB pattern transiently/partially reversed during HAART in 2 patients.
**Conclusions:** During PHI, early HAART, accompanied by rapid virologic suppression, prevents the gradual increase of AI, while resumption of viral replication is paralleled by a prompt increase of AI. This altered pattern of maturation of the antibody response in patients achieving rapid control of viral replication may suggest a complex mechanism of immune response to HIV not yet fully investigated.

### P1917 Antiretrovirals adverse reactions from a prospective HIV/AIDS cohort study in Bogotá, Colombia

*J.F. Vesga, C. Alvarez, J.R. Tamara, J.A. Cortes, P. Guerrero, F. Gil (Bogota, CO)*

**Background:** Adverse Drug Reactions (ADR), lead not just to a high percentage of therapy abandonment, but also to a poor adherence to treatment instructions resulting in failure of therapy. The purpose of this study was to show the relation between the use of drugs and ADRs, their impact on treatment and the severity of reaction as defined by the DAIDS scale.
**Methods:** Making use of a database designed in Access (ver 11.5 2003, Microsoft, USA), information from ADR to Antiretrovirals from 384 HIV positive patients between June 2005 and June 2006 was analysed. Patients were seen routinely every 4 weeks, and data was collected from such consults. Patient was asked about the presence of ADRs, lot and drug manufacturer, the active commencement of the antiretroviral, the effect of the reaction on the overall treatment scheme, and laboratories related to the negative reaction. For abnormal laboratory results a DAIDS grade was established. Data was then analysed using programme Stata (Ver 9.0).
**Results:** In the consequent year (4608 month/patient), 672 ADRs were reported to have occurred with the following frequency: metabolic reactions 28.1%, haematologic 28%, gastrointestinal 26.5%, neurologic and psychological 13.5%, dermatological 3.28%, hepatic 2.39%, renal and urological 0.9%.
Regarding intensity of ADRs, 85.2% were mild, 14.4% moderate and 1.7% severe. The most frequent ADRs were macrocytosis, nausea, hypertrygliceridaemia, combined dyslipidaemia and diarrhoea. Among all ADRs 89.5% were classified as DAIDS grade I, 8.8% grade II, 1% grade III and 0.4% grade IV. The 89.9% of ADRs mean no change in the scheme of treatment, 6.3% required a change of the drug, 1.3% quit the treatment, and 2.4% of treatments were transitorily suspended.
**Conclusions:** ADRs were frequent in our study, however the most of it were reported as mild reactions and didnt necessitate change or suspension of the treatment. As expected, metabolic reactions like dyslipidaemia where most frequently found among protease inhibitors and NNRTs than in NRTs.

### P1918 Pharmacokinetic parameters of 400/100 mg indinavir/ritonavir in HIV-infected Thai patients

*S. Jaruratanasirikul (Songkhla, TH)*

**Introduction:** The dosage recommendations of 800/100 mg indinavir/ritonavir in the Thai population may lead to more severe side effects. This may be due to higher drug levels.
**Objective:** The aim of this study was to assess the steady state pharmacokinetics of reduced doses of indinavir boosted with ritonavir in HIV infected Thai patients.
**Methods:** Our study was conducted in ten HIV infected patients. All patients received 400/100 mg indinavir/ritonavir combined with lamivudine and stavudine or zidovudine every 12 h. After 4 weeks of starting antiretroviral therapy, indinavir pharmacokinetics studies were carried out and HIV-1 RNA viral load was determined at 24 weeks.
**Results:** The pharmacokinetic parameters are shown as below and all patients had a HIV-1 viral load less than 400 copies/mL after 24 weeks follow up.

| Parameter | Mean (SD) | Units |
|---|---|---|
| $C_{max}$ | 7.51 (3.57) | mg/L |
| $C_{min}$ | 0.86 (0.59) | mg/L |
| $AUC_{0-12}$ | 38.14 (15.17) | mg·h/L |
| $T_{1/2}$ | 2.94 (0.90) | h |
| $T_{max}$ | 2.10 (1.31) | h |