to treat SIADH. There is a lack of studies about the prevalence of SIADH as an Adverse Drug Reaction (ADR).

**Purpose** To classify by the Naranjo Algorithm (NA) and to determine the prevalence of SIADH in hospitalised patients caused by ADR and treated with tolvaptan.

**Materials and Methods** Two-year descriptive, retrospective, longitudinal, historical cohort study of 33 patients (15 men, 18 women). We sought patients and their clinical characteristics (age, sex, pre-treatment in the week prior to tolvaptan with drugs that could cause SIADH as ADR) using pharmacotherapy management software SINFHOS, Silicon, IANUS and BOUT Plus. To determine the probability of ADR, we used the NA. Probability levels based on total score are: definite (>9), probable (5–8), possible (1–4), doubtful (0).

**Results** 12 of the 33 (6 men, 6 women) patients were treated in the week prior to tolvaptan with drugs that could cause SIADH as ADR. 16 treatments with 10 drugs that could cause SIADH as ADR (1.3 drugs per patient) were found in the week prior to tolvaptan. The 16 treatments detected were classified as possible (12 times), probable (once) and doubtful (three times); average score was 2.6. SIADH could have been caused by a drug in 10 patients and was classified as possible (4 men, 5 women, 7 older than 65, 2 younger), and probable (1 man over 65). Prevalence was 50%, 40% in men and 54% in women, 28% in people older than 65 and 40% in younger people.

**Conclusions** Our results suggest that some drugs may contribute to the development of SIADH, and there seems to be a greater risk in male patients under 65. Further research is required to evaluate the prevalence and the relative risk of suffering from SIADH as ADR.

No conflict of interest.

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**Abstract CPC-133 Table 1**

<table>
<thead>
<tr>
<th>Anaemia</th>
<th>69%</th>
<th>Grade 2 (8.0 – &lt;10.0 g/dL)</th>
<th>54%</th>
<th>Grade 3/4 (&lt;8.0 g/dL)</th>
<th>8%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Thrombocytopenia</td>
<td>89%</td>
<td>Neutropenia</td>
<td>77%</td>
<td>Hyperbilirubinaemia</td>
<td>46%</td>
</tr>
<tr>
<td>Increased triglycerides</td>
<td>46%</td>
<td>Increased ferritin</td>
<td>54%</td>
<td>Increased GGT</td>
<td>23%</td>
</tr>
<tr>
<td>Fatigue</td>
<td>100%</td>
<td>Depression</td>
<td>69%</td>
<td>Reduction appetite</td>
<td>77%</td>
</tr>
<tr>
<td>Nausea</td>
<td>30%</td>
<td>Diarrhoea</td>
<td>46%</td>
<td>Vomiting</td>
<td>38%</td>
</tr>
<tr>
<td>Haemorrhoids</td>
<td>77%</td>
<td>Rash and pruritus</td>
<td>69%</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

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**CPC-134 THE EFFECT OF ABACAVIR ON CARDIOVASCULAR RISK OF A SPANISH HIV-INFECTED COHORT**

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**Background** There is evidence that antiretroviral therapy (ART) increases cardiovascular risk (CVR). There is controversy over the effect of abacavir (ABC) on CVR. The use of abacavir has been associated with a higher incidence of myocardial infarction in several cohort studies, but data from clinical trials are not conclusive.

**Purpose** To determine the effect of exposure to ABC and exposure time (ET) to ABC in CVR of a HIV-infected cohort on ART from the northwest of Spain.

**Materials and Methods** Cross-sectional study including HIV patients on ART who were treated at our hospital between March-May 2012. We recorded demographics, ART history and CVR risk factors. CVR was estimated using the Framingham function calibrated for Spanish population (REGICOR). CVR categories were: low (<5%); intermediate (5–9%); high (10–14%); very high (>15%). Three ABC exposure groups were defined: a) no abacavir exposure (No ABC); b) exposure to abacavir but not to indinavir (ABC); c) exposure to abacavir and indinavir (ABC + Ind).

**Results** 89 HIV patients were included in the study (83.1% males, mean age 47.4 ± 7.8 years). Smoking prevalence was 51.7%, hypertension 39.3%, dyslipidaemia 24.7%, low HDL cholesterol 67.4%, diabetes 4.5%. Mean global CVR was 4.01%±2.50. Proportion of patients with low CVR was 70.8%; intermediate 25.8%; high 10%–14%; very high (>15%). Three ABC exposure groups were defined: a) no abacavir exposure (No ABC); b) exposure to abacavir but not to indinavir (ABC); c) exposure to abacavir and indinavir (ABC + Ind).

**Conclusions** Apparently, ABC exposure does not increase CVR in our HIV-infected population. More prospective controlled studies are needed to evaluate any association between ABC and increased CVR.

No conflict of interest.