The eGFR was calculated by CKD-EPI, MDRD4-IDMS and Cockcroft-Gault. Patients with serum creatinine below 0.4 mg/dl were excluded.

CKD-EPI was used as a reference formula to assess the concordance between the different methods of estimating, classifying patients in 3 eGFR groups according to the IV dexketoprofen SmPC: <50 mL/min/1.73 m², 50–80 mL/min/1.73 m² and >80 mL/min/1.73 m².

Results The study included 1946 patients – 54.3% men, 45.7% women – from a total population of 2052 admissions; mean age of 59.8 years (range 17–103). The mean serum creatinine concentration was 0.84 mg/dl ± 0.48 and mean eGFR, according to CKD-EPI, 83.05 ± 26.17 mL/min/1.73 m².

The following results of non-concordance were found by comparing these formulas to estimate renal function:

- CKD-EPI vs. MDRD4-IDMS: 4.5% in eGFR <50 mL/min/1.73 m² group, 23.2% in the eGFR 50–80 mL/min/1.73 m² and 15.9% in eGFR >80 mL/min/1.73 m².
- CKD-EPI vs. CG: 2.8% in eGFR <50 mL/min/1.73 m² group, 10.5% in eGFR 50–80 mL/min/1.73 m² and 7.8% in eGFR >80 mL/min/1.73 m².
- MDRD4-IDMS vs. CG: 4.5% in the group of eGFR <50 mL/min, 21.4% in group eGFR 50–80 mL/min and 17.1% in the group of eGFR >80 mL/min.

Conclusions A great difference was found in the estimates of renal function between the three methods used – CKD-EPI, MDRD4-IDMS and CG – in the three eGFR functional categories – <50, 50–80 and >80 mL/min/1.73 m² – ranging between 2.8% and 23.2%.

These results are relevant in clinical practise because the functional category determines the non-use or limited dose of dexketoprofen IV for each patient.

No conflict of interest.

PHC-026 TACROLIMUS AND IMATINIB INTERACTION. A CASE STUDY
doi:10.1136/ejhpharm-2013-000276.371

Background Tacrolimus is a drug metabolised by CYP3A4. Since imatinib increases the plasma concentrations of simvastatin, a CYP3A4 substrate, this indicates that it is an inhibitor of this enzyme and may affect other drugs.

Purpose To describe the possible interaction between imatinib and tacrolimus that result in increased blood levels of Tacrolimus.

Materials and Methods Information was collected through the SAVAC and SELENE computer systems and reviewing patient history. The variables compiled were tacrolimus blood levels, dose and dose regimen.

Results The patient had an allogeneic blood stem cells transplant from an unrelated donor, HLA and ABO compatible, presenting cutaneous sclerodermiform graft versus host disease (GVHD) on tacrolimus (2 mg/12 h) treatment and blood levels around 4 ng/ml for 12 weeks. After starting treatment with imatinib, in the following five tests tacrolimus levels ranged from 5.8 ng/ml to 8.9 ng/ml with no change in the dose of tacrolimus. After 45 days of treatment imatinib was suspended and tacrolimus levels recorded in the following test after discontinuation of imatinib fell to around 4 ng/ml.

Conclusions The increase in tacrolimus blood levels, without changing the dose, supports the possible interaction between imatinib and tacrolimus.

No conflict of interest.

PHC-027 THE PHARMACIST’S ROLE IN IMPROVING VALPROIC ACID PRESCRIPTIONS
doi:10.1136/ejhpharm-2013-000276.372
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Background Valproic acid (VPA) is 90–95% protein bound to albumin; this binding can be saturated so other parameters that can modify the free fraction of VPA should be taken into account.

Purpose To identify areas for improvement in VPA use and monitoring in a tertiary hospital where the pharmacy service does not routinely send pharmacokinetic dose adjustment recommendations.

No conflict of interest.
Materials and Methods

A retrospective study was conducted from February to April 2012. All patients treated with VPA were included and grouped depending on whether VPA was part of their home treatment or not.

Variables collected were: dose, indication, total VPA serum concentration (C), drug interactions classified as ≥C by Lexi-Comp, glomerular filtration rate (GFR), Child-Pugh score, albumin and bilirubin.

Results

80 patients were treated with VPA, 24 of whom were on VPA before admission (15 epilepsy, 9 psychiatric disorders and 1 unknown reason).

Reasons for admission were: 5 convulsions, 12 psychiatric disorders and 13 causes unrelated to VPA. At discharge 27 patients continued on VPA with a mean dose similar to the dose at admission.

C was determined in 14 patients: 5 were within the reference range (50–100 mg/L); 2 above, achieving therapeutic levels before discharge and 7 below. In these latter cases, 3 had an albumin <4.2 g/dl, but none reached C > 50 mg/L after correcting it with the J. Hermida formula which is a theoretical method for normalising C in hypoalbuminemic patients. GFR, Child-Pugh score and bilirubin were normal. Mean time between changes in dose and C determinations was 1.5 days (0–5 days).

21 drug interactions were detected in 15 patients, involving a total of 10 drugs. Only 2 interactions were reported: VPA meropenem and VPA lamotrigine.

Conclusions

Changes in free fraction of VPA, due to hypoalbuminaemia, liver or kidney disease and hyperbilirubinaemia, must be detected.

C should be measured once a steady state has been achieved (3–5 days).

Drug interactions affecting VPA should be added to the pharmacy service’s interaction notification programme.

No conflict of interest.

Table 1

<table>
<thead>
<tr>
<th>Number of patients</th>
<th>Control</th>
<th>Non-cirrhotic alcoholics</th>
<th>Cirrhosis</th>
<th>IVDA</th>
</tr>
</thead>
<tbody>
<tr>
<td>N</td>
<td>20</td>
<td>18</td>
<td>18</td>
<td>9</td>
</tr>
<tr>
<td>CL (L/h)</td>
<td>5.27 ± 1.47*</td>
<td>6.40 ± 2.16</td>
<td>4.27 ± 1.18*</td>
<td>6.53 ± 1.91</td>
</tr>
<tr>
<td>Vd(l) (L/Kg)</td>
<td>0.75 ± 0.33</td>
<td>0.64 ± 0.16</td>
<td>0.68 ± 0.10</td>
<td>0.59 ± 0.09</td>
</tr>
<tr>
<td>Initial dosage (mg/kg/day)</td>
<td>29.23 ± 5.75*</td>
<td>26.55 ± 7.35*</td>
<td>27.28 ± 9.01*</td>
<td>28.05 ± 6.12*</td>
</tr>
<tr>
<td>C50 (mg/L)</td>
<td>9.76 ± 3.49</td>
<td>7.91 ± 4.26</td>
<td>10.37 ± 4.51</td>
<td>5.30 ± 3.04</td>
</tr>
</tbody>
</table>

*p > 0.05; #p = 0.02