(DDIs) but it would be interesting to know which ones are real and have clinical outcomes.

Aim and objectives The main objective of the study was to determine the prevalence of real DDIs between immunosuppressants and other drugs in transplant patients. Secondary objectives were to evaluate clinical impact, categorise the type of DDIs, identify drugs involved and propose alternative therapeutic strategies.

Material and methods A prospective, observational 1 year study (February 2018 to February 2019) was conducted at a third level hospital, including all new transplanted patients. DDIs were detected by a computer application. To determine real clinical DDIs, patient medical records were reviewed, looking for data on monitoring blood concentrations of immunosuppressive drugs and adverse drug events (ADEs) caused by DDIs. DDIs were classified in C, D or X according to the Lexi interact score (C=monitor therapy, D=consider therapy modification, X=avoid combination). The clinical importance of the real DDIs was expressed in terms of patient outcomes: percentage of patients with ADEs due to real DDIs. Data were analysed using SPSS V.17.0 (Chicago, Illinois, USA).

Results A total of 309 transplant patients were included with a mean age of 52 ± 14 years (13–79) and 69.9% were men. The prevalence of real DDIs was 21.68%. The number of real DDIs between immunosuppressants and other drugs was 71. The largest type of real DDIs was category D (52 (73.23%)).

Immunosuppressive drugs administered with antifungal azoles and tacrolimus with nifedipine had a great clinical impact due to the fluctuation in trough immunosuppressant blood concentrations (C_0) of the immunosuppressants.

The most common clinical outcomes were nephrotoxicity (12%), hyperkalaemia (10%) and hypertension (5%). Suggestions to avoid D and X for real DDIs included: immunosuppressant dose change, consider therapy modification and using paracetamol instead of non-steroidal anti-inflammatory drugs. A statistically significant linear correlation was detected between number of prescribed drugs and real and clinically important DDIs.

Conclusion and relevance There are many potential interactions described in the literature but only a small percentage have been proved to be real DDIs, based on patient outcomes, which were detected by determining the variations in C_0 of immunosuppressants and ADEs caused by DDIs. Few patients suffered ADEs due to the close pharmacokinetic monitoring of immunosuppressants. The results allow us to identify the pharmacological groups that caused real DDIs.

REFERENCES AND/OR ACKNOWLEDGEMENTS

No conflict of interest.

4CPS-119 'REAL WORLD' EXPERIENCE OF TOFACITINIB AND BARICITINIB IN THE TREATMENT OF RHEUMATOID ARTHRITIS: EFFECTIVENESS AND SAFETY EVALUATION

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Background and importance Tofacitinib and baricitinib are oral Janus kinase inhibitors (Jki) approved for the treatment of rheumatoid arthritis (RA), offering an alternative in patients who have not responded or tolerated previous treatment lines because of adverse effects, complications or other reasons. In pivotal clinical trials, patients had higher DAS28-ESR than our patients and were less pretreated with biologic disease modifying antirheumatic drugs (bDMARDs). Lower effectiveness in real world compared with clinical trials has been reported. We conducted a retrospective study to evaluate effectiveness in our patients.

Aim and objectives To assess the effectiveness and safety of Jki in patients with RA in a clinical setting.

Material and methods This observational retrospective study included patients with RA in a third level hospital, from 2016 to 2019. Clinical data were collected from the hospital medical records and 'patient and treatments registry programme' of our local government: demographic, indications, previous and current treatments, discontinuity of treatment and reasons, effectiveness and safety data.

Clinical disease impact, defined by the disease activity score, DAS28-ESR, was evaluated during patient monitoring. Mean (SD) DAS28-ESR at baseline and at follow-up after Jki treatment were analysed.

Variance analysis (ANOVA) and the χ^2 test were applied (SPSS) to evaluate treatment effect (time=0 vs follow-up data).

Results Fifty-three patients were included with a mean age of 63.9 ± 13.3 years and 46 (86.8%) were women. Previous non-bDMARDs treatments were: methotrexate (n=39, 73.6%), leflunomide (n=34, 64.2%), hydroxychloroquine (n=15, 28.3%) and sulfasalazine (n=9, 17.0%).

Disease activity was categorised as: remission (DAS28-ESR <2.6), low disease activity (2.6 < DAS28-ESR <3.2), moderate disease activity (3.2 < DAS28-ESR <5.1) and high disease activity (DAS28-ESR >5.1). At the beginning of the study, one patient had DAS28-ESR <2.6 (1.9%) and during the monitoring period, nine patients reached DAS28-ESR <2.6 (17.0%) at some point. Mean DAS28-ESR was 4.97 ± 1.32 at baseline; during follow-up, mean DAS28-ESR decreased to 0.69 ± 1.44 ($10.3 \pm 30.8\%$) ($p < 0.001$).

Patients were classified by treatment received: tofacitinib group (TofG (n=44, 83.0%)) and baricitinib group (BarG (n=9, 17.0%)). Number of bDMARDs used before Jki:

-TofG: 0 (n=7, 15.9%), 0–3 (n=22, 50.0%), >3 (n=15, 34.1%).

-BarG: 0 (n=2, 22.2%), 0–3 (n=3, 33.3%), >3 (n=4, 44.4%).

At follow-up, mean DAS28-ESR decreased: TofG, 0.64 ± 1.44 ($9.3 \pm 32.0\%$) and BarG, 0.98 ± 1.44 ($15.6 \pm 23.0\%$).

Twelve patients discontinued Jki: baricitinib (n=3, 33.3%) and tofacitinib (n=9, 20.5%). Reasons for discontinuation, baricitinib and tofacitinib, respectively, were due to:

-lack of effectiveness (n=2, 22.2%) and (n=5, 9.4%);
-lack of adherence (n=1, 11.1%) and (n=2, 3.8%);
-adverse effects (n=0, 0%) and (n=1, 1.9% oedema and dyspnoea); and

-patient's choice (n=0, 0%) and (n=1, 1.9%).

Conclusion and relevance Our study suggests that Jki could be effective in real world settings after switching from other multiple bDMARDs. The results showed a modest benefit of Jki in complicated and over treated patients with diverse backgrounds, as found in daily practice.

REFERENCES AND/OR ACKNOWLEDGEMENTS

No conflict of interest.

4CPS-120 TEN YEAR ANALYSIS OF THE USE OF INFLIXIMAB IN ULCERATIVE COLITIS

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Background and importance Analysis of data obtained in the 'real world' setting is acquiring great importance in the health-care environment. It is important to know the results obtained with different treatments outside the ideal environment offered by clinical trials.

Aim and objectives To describe and analyse the results obtained with infliximab (IFX) over a 10 year period in patients diagnosed with ulcerative colitis (UC).

Material and methods This was a retrospective descriptive study conducted in a third level hospital that included all patients diagnosed with UC who started IFX treatment between January 2005 and December 2014. Follow-up was up to 31 July 2015. The following clinical data were recorded: age, sex, time in treatment, previous biological lines, definitive or temporary suspensions of treatment and reason, dose modifications and values for C reactive protein (CRP) before starting and at the end of treatment. Dosage modifications were recorded as those that implied a treatment pattern not described in the technical data sheet. Treatment interruptions were considered to be those of a duration ≥ 6 months. Data were recorded using the OncoWin computer application and the electronic medical record stored in SAP.

Results A total of 32 patients were included in the study (59.4% men, mean age 37.7 years (12–72)). Mean follow-up time was 52.5 months (8–109); 93.8% of patients received IFX as firstline therapy. Mean baseline CRP was 19.92 mg/L (0.70–84.94), and 7.82 (0.10–30.90) at the end of treatment. A total of 71% of patients discontinued treatment definitively: 6.3% for infusional reactions, 31.2% for inefficacy, 25.0% for remission of the disease and 9.4% for other reasons. In addition, 28.7% of patients received only the three IFX induction doses, of which 78% were not able to control the disease. Only 9.4% of patients temporarily interrupted treatment, with an average interruption time of 28.7 months (7–41); 12.5% of patients required dose modifications to control the disease.

Conclusion and relevance With the present work we wanted to show the long term results of IFX in UC in clinical practice. IFX can be an effective tool to control disease symptoms in the long term. Its use, administering only three induction doses, does not seem to be useful, with about 80% ineffectiveness rate.

REFERENCES AND/OR ACKNOWLEDGEMENTS

No conflict of interest.

4CPS-121 EVALUATION OF COST AND EFFICACY OF ECULIZUMAB IN COMPLEMENT MEDIATED THROMBOTIC MICROANGIOPATHY IN THE CLINICAL SETTING

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Background and importance Complement mediated thrombotic microangiopathy (C-TMA) is caused by complement disruption that leads to haemolysis and thrombocytopenia. Eculizumab inhibits C5b-9 complex formation by binding protein C5 and has been approved for C-TMA. Nevertheless, studies on the effectiveness of eculizumab under real world conditions are scarce, even considering its high cost.

Aim and objectives To evaluate the efficacy and cost of eculizumab in clinical practice for C-TMA after 26 weeks (W) of treatment.

Material and methods Patients diagnosed with C-TMA whose treatment with eculizumab had been approved (900 mg/W for four doses and 1200 mg/W thereafter) and ongoing for >26 W were included. Clinical variables were obtained from the electronic health records. Laboratory tests were evaluated at baseline, and at 12W, 26W and 38W after initiation of treatment. C-TMA remission was defined as lactate dehydrogenase (LDH) less than the upper limit of normal (ULN), platelet count $>150 \times 10^9/L$ and $<25\%$ creatinine increase from baseline.

Results Six patients were included (1 woman) with a median age of 43 years (range 23–59); none had genetics related to complement alteration. One patient had a pulmonary transplant and one a renal transplant. Median duration of treatment was 10 (6.8–45.5) months. Two patients stopped treatment because of resolution of C-TMA. Estimated cost of eculizumab treatment for 26W was 337 300€. Median cost estimated per treatment was 160 458€ (118 055–640 870).

Haemoglobin increased from 11 (8.2–12.7) g/dL at baseline to 12 (10.2–13.20) g/dL after 26W and to 13 (9.8–13.1) g/dL at 38W. Reticulocytes decreased from 112 (90.8–190.3) to 65 (44.1–85.4) after 26W (normal values $50\text{--}100 \times 10^9/L$) ($p=0.18$).

Platelets increased from 206 (44–359) $\times 10^3$ platelets/ μL at 0W to 291 (175–305) at 38W. All patients recovered $>150 \times 10^3$ platelets/ μL within 26W.

LDH decreased in all patients: 511 (323–1787) U/L at 0W; 487 (288–1351) U/L at 12W; 491 (313–642) U/L at W26 and 430 (266–642) U/L at W38; 3 patients had LDL $>ULN$ after 38W.

Median creatinine decreased (not significant). Renal function was maintained or improved in 4/6 patients. Two patients were in dialysis and one stopped. CH50 decreased in all patients and was undetectable for most patients within 12W ($p=0.001$). There was no significant change in C3 and C4. Two patients were in remission after 26W.

Conclusion and relevance Eculizumab was effective in C-TMA based on cellular and biochemical markers (platelets, LDH, creatinine). Changes in some parameters may not have been detected because of the small sample size. Two of six patients were in remission after 26W. The estimated cost for additional C-TMA-remission was 1 011 900€.

REFERENCES AND/OR ACKNOWLEDGEMENTS

No conflict of interest.