inappropriate use and healthcare costs. In addition, audit and educational feedback might strengthen the results.

REFERENCES AND/OR ACKNOWLEDGEMENTS

1. Liunbruno G, Bennardello F, Lattanzio A, Piccoli P, Rossettias G. Recommendations for the use of albumin and immunoglobulins. *Blood Transfus* 2009;**7**:216–234.

No conflict of interest.

4CPS-017 EFFICACY AND SAFETY OF TOLVAPTAN IN THE TREATMENT OF POLYCYSTIC KIDNEY DISEASE

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Background and importance Tolvaptan is the first authorised drug for the treatment of autosomal dominant polycystic kidney disease (PQRAD).

Aim and objectives To analyse the efficacy and safety of tolvaptan in the treatment of PQRAD compared with the results of the TEMPO study.

Material and methods This was a descriptive, observational and retrospective study of patients treated with tolvaptan (August 2017–April 2019). Variables studied were: age, sex, arterial hypertension, total renal volume (VRT), creatinine, serum potassium and sodium, transaminases and glomerular filtration rate (GFR). Adverse reactions were recorded. For collection of data, the electronic medical history was used. Statistical analysis was performed with the Stata14 programme.

Results We included 23 patients (8 women, 5 men), median age 46 years (31–63) years. All had VRT >1000 mL (median 1920 mL (1230–3154)). At the beginning of treatment, GFR was 49.7 mL/min/1.73 m² (25.6–102.31): 3 patients had stage 1 chronic kidney disease, 2 patients had stage 2, 10 patients stage 3A, 6 patients stage 3B and 2 patients stage 4. All patients suffered progressive deterioration of renal function during treatment: 5.25 mL/min/1.73m² (–3.61–18.29) and 8.28 mL/min/1.73 m² (–1.87–15.59) at 3 and 6 months, respectively, and 8.49 mL/min/1.73 m² (4.21–14.06) at the end of the treatment year. Tolvaptan was suspended in three patients due to impaired renal function (GFR <20 mL/min/1.73 m²); all other patients were still receiving treatment at the end of the study (five with dose reduction to 60/30 mg). All patients reported polyuria and polydipsia and no patient suffered clinically relevant alterations in serum sodium or potassium. Relative to liver function, three patients suffered specific alterations in AST, ALT and GGT above normal values (57, 76 and 63 IU/L, respectively).

Conclusion and relevance Our results, compared with the TEMPO study, showed a higher rate of renal function deterioration, measured as a decrease in GFR rate after 1 year of treatment (8.49 vs 2.7 mL/min/1.73 m²), probably in relation to the worst baseline condition of the patients included in our study. Therefore, it is essential to identify the population susceptible to receiving this drug, prioritising those patients with GFR >45 mL/min/1.73 m² and with a high risk of rapid progression.

REFERENCES AND/OR ACKNOWLEDGEMENTS

TEMPO clinical trial.

No conflict of interest.

4CPS-018 EFFICACY OF UREA IN THE TREATMENT OF HYPONATRAEMIA IN SYNDROME OF INAPPROPRIATE ANTIDIURETIC HORMONE SECRETION

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Background and importance The consequence of the syndrome of inappropriate antidiuretic hormone secretion (SIADH) is a hypotonic hyponatraemia. Urea is a well tolerated therapeutic option indicated to correct sodium levels, acting as an osmotic diuretic, eliminating a large amount of water in urine accompanied by an increase in plasma sodium concentration.

Aim and objectives To evaluate the efficacy of urea in controlling hyponatraemia due to SIADH in a third level hospital.

Material and methods This was a quasi-experimental study. Patients with hyponatraemia treated with urea in 2019 were included.

The main variable of our study was serum sodium level before treatment with urea at 24 hours, 48 hours, 14 days and 60 days. Age and sex were included as secondary variables.

There were no extreme outliers and the data were normally distributed for each measured time, as assessed by box plot and the Shapiro–Wilk test ($p > 0.05$), respectively. A one-way repeated measures ANOVA was conducted to determine whether there was a statistically significant difference in sodium concentration before and after treatment with urea. The analyses were performed using the SPSS/PC statistical programme (V.24.0 for Windows, SPSS Inc, Chicago, Illinois, USA).

Results Thirty-three patients were treated with urea for 9 months. Of these, 67% were men and mean age was 77 ± 13 years. Serum sodium levels before treatment and at 24 hours, 48 hours, 14 days and 60 days were 125 ± 4 , 127 ± 5 , 129 ± 5 , 134 ± 4 and 134 ± 4 mg/dL respectively. Time did not elicit statistically significant changes in sodium levels before and after treatment with urea ($F = 4.1$, $p = 0.074$).

Conclusion and relevance In the study, there were no significant differences in plasma sodium values before and after urea treatment, so we did not demonstrate the efficacy of urea. The main drawback in the study was the small population analysed.

REFERENCES AND/OR ACKNOWLEDGEMENTS

No conflict of interest.

4CPS-019 ALIROCUMAB AND EVOLOCUMAB: EFFECTIVENESS AFTER 3 YEARS OF FOLLOW-UP IN A REAL WORLD SETTING

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Background and importance Hypercholesterolaemia leads to a higher risk of atherosclerosis and cardiovascular events. Familial hypercholesterolaemia is more resistant to usual treatments. In 2015, the PCSK9 inhibitors (PCSK9I) alirocumab