Sodium zirconium cyclosilicate (SZC) for treatment of chronic hyperkalaemia in order to maximise inhibition of renin–angiotensin–aldosterone system (RAAS) – what is the impact in the general nephrology clinic?

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Introduction
RAAS inhibitors provide significant benefits including slowing deterioration of kidney function in patients with diabetic or proteinuric kidney disease, improved outcomes for patients with heart failure with reduced ejection fraction and patients with ischaemic heart or cerebral vascular disease. Patients receiving maximal RAAS inhibition achieve the greatest response but some patients are unable to tolerate this because of hyperkalaemia.

The recently published National Institute for Health and Care Excellence (NICE) technology appraisal guidance recommended using SZC for outpatients with persistent hyperkalaemia and chronic kidney disease (CKD) stage 3b to 5 or heart failure with reduced ejection fraction, who are not taking an optimised dosage of RAAS inhibitor because of hyperkalaemia. It is important to determine the proportion of patients who would benefit from SZC to support implementation of local guidelines in clinical practice and understand the potential cost of such treatment.

Methods
One hundred sequential patients in a general nephrology clinic with non-immune and non-dialysis chronic kidney disease had their electronic records reviewed, identifying those with an indication for RAAS inhibition. This included 1) CKD with proteinuria (urine PCR >100), 2) CKD due to diabetes mellitus, 3) heart failure with reduced ejection fraction and 4) CKD in combination with either ischaemic heart or cerebrovascular disease. If these patients were not on maximal RAAS inhibition, then a documented reason was searched for. If no reason was documented, the patient’s results were assessed to identify hyperkalaemia, defined as documented serum potassium level >6.0mmol/litre or persistently > 5.5mmol/litre. Where hyperkalaemia had occurred acutely it was only labelled a contra-indication for RAAS inhibition if it occurred without an associated episode of acute kidney injury (AKI). In cases where hyperkalaemia limited dosage of RAAS inhibition it was determined whether these patients had received measures to reduce hyperkalaemia including optimisation of bicarbonate.

Results
Of the 100 consecutive patients audited, 46 were female and 54 were male. The mean patient age was 64 and the mean estimated glomerular filtration rate (eGFR) was 33. 68 patients had an indication for being on RAAS inhibition with only 10 on a maximal dose. Of the remaining 58 patients, 26 (45%) were limited by hyperkalaemia. Furthermore 12 of these patients (46%) had hyperkalaemia associated to an episode of AKI. 20 of the 26 patients had their bicarbonate levels optimised to reduce hyperkalaemia. Therefore 14 patients were identified suitable for SZC.

Discussion
A significant proportion (14%) of patients attending a general nephrology outpatient clinic were not on optimum RAAS inhibition due to hyperkalaemia. It is possible that with appropriate treatment, using sodium
bicarbonate, this number may decrease, but this still leaves a significant number of patients who would attain prognostic benefit by using SZC to maximise RAAS inhibition when limited by hyperkalaemia. This audit has implications for renal centres across the UK to support implementation of guidelines for use of SZC and monitoring arrangements. Introduction of SZC would enable a significant number of patients in general nephrology clinics to achieve maximisation of RAAS inhibition and attain the associated long-term benefits.