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# Should Oral Sodium Bicarbonate Be Used to Prevent Chronic Kidney Disease Progression?

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Dear Editor,

In the October edition of Irish Medical Journal (IMJ), our group identified the high burden of metabolic acidosis of chronic kidney disease (MA-CKD) in nephrology outpatient setting at a tertiary centre. After adjusting for potential contraindication, only 19% of the eligible patients were on oral sodium bicarbonate (OSB)[1]. We are for the first time reporting the preliminary results from our ongoing study aiming to identify the practice of prescribing OSB in the management of MA-CKD in patients with serum bicarbonate levels below 22mmol/L from the majority of the nephrology centres across the Republic of Ireland using the national renal database (eMED). Initial results indicate 267 patients had serum bicarbonate level below 22mmol/L of which 57 (22.1%) were on OSB, during the study period between 2018-2019. This multicentre cohorts' initial results correlate with our previously published work suggesting limited usage of OSB in practice.

The question is why is it important to actively manage MA-CKD? The principles of reducing chronic kidney disease (CKD) progression are based around managing hypertension, proteinuria and glycaemic control. However, in the last decade correcting MA-CKD and maintaining serum bicarbonate levels above 22mmol/L has shown in randomised control trials (RCT) to reduce the progression of CKD[2]. A low protein diet with high fruits and vegetables is recommended, but in CKD patient the propensity to develop severe hyperkalaemia limits fruits and vegetables. Therefore, oral alkali agent, commonly OSB is suggested to be used once overt MA-CKD has developed [2]. The use of OSB increases overall salt load which can potentially worsen volume status and blood pressure thus many nephrologists remain reluctant to prescribe OSB.

The recently published, The Use of Bicarbonate in Chronic Renal Insufficiency (UBI) study, the largest multicentre RCT assessing if the use OSB to treat MA-CKD preserved renal function with secondary safety outcomes[3]. The study involved 740 participants with CKD stage 3-5 and serum bicarbonate (HCO3) between 18-24mmol/L over mean follow up of 30 months. Patients with congestive heart failure (NYHA III or IV) and blood pressure > 150/90 were excluded. OSB was given to the intervention group (IG) with the aim to maintain HCO3 between 24-28mmol/L compared to standard therapy (ST) who received only dietary input of low protein diet, low phosphorus diet and controlled sodium diet.

The primary endpoint, doubling of serum creatinine occurred in 87 participants of which 25(6.6%) were in the IG compared to 62 (17%) in ST. A 64% lower hazard rate of doubling serum creatinine when OSB was used (IG). Secondary endpoints all favoured IG compared to ST, including a slower decline in creatinine clearance at each CKD stage, patients progressing to renal replacement therapy, all-cause mortality and hospitalisations. It is also important to note no significant change in blood pressure or body weight was seen in IG.

The study, however, was underpowered to identify mortality differences. It was unblinded and risk of selection bias exists regardless of centralisation of randomisation.

Combined with previous RCTs, the UBI study adds mounting evidence for nephrologists to actively manage MA-CKD and maintain HCO3 above 22mmol/L to prevent progression of CKD[4].

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