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Halting the progression of chronic kidney disease: an audit of the use of renin-angiotensin-aldosterone inhibitors and sodium-glucose cotransporter-2 inhibitors

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Abstract

Introduction

Most recently, the EMPA-KIDNEY trial demonstrated benefit in patients with chronic kidney disease with a wider range of albuminuria and glomerular filtration rate. We aim to identify the proportion of patients on renin-angiotensin aldosterone system inhibitors (RAASi) such as angiotensin converting enzyme inhibitor (ACEi), and angiotensin II receptor blocker (ARB), and sodium-glucose transporter-2 inhibitor (SGLT2i) in our Nephrology outpatients department (OPD).

Methods

A prospective audit was performed in Galway University Hospital (GUH) September to October 2023 consecutively. The audit questionnaire was completed by doctors reviewing patients in OPD. The use of ACEi/ARB/SGLT2i was recorded during the consultation.

Results

165 patients were included in the audit with a mean (±SD) age of 62.1 ± 18.6 years. The 3 most common primary renal diagnoses were diabetic kidney disease; 20/165 (12.1%), hypertensive renal disease; 18/165 (10.9%) and IgA nephropathy; 15/165 (9.1%). The mean estimated glomerular filtration rate (eGFR) was 46.8 ± 24.5 ml/min/1.73 m². Guideline directed medical therapy according to the EMPA-KIDNEY trial and UK Kidney Association Clinical Practice Guidelines would suggest_therapeutic optimisation with RAASi and SGLT2i in 65/165 (39.4%) patients.

Discussion

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Patients should be initiated on SGLT2i after being established on a RAASi early in their diagnosis to gain the maximal renoprotective effects of SGLT2i.

Introduction

Sodium-glucose cotransporters-2 inhibitors (SGLT2i) were initially developed for the treatment of type 2 diabetes. Multiple studies have demonstrated the renoprotective effect of SGLT2i, with the capacity to slow diabetic kidney disease and non-diabetic proteinuric chronic kidney disease progression. Most recently, the EMPA-KIDNEY trial demonstrated benefit in patients with chronic kidney disease with a wider range of albuminuria and glomerular filtration rate[1].

SGTL-2 cotransporters are found in the first segments of the proximal tubules. They absorb 90-97% of filtered glucose in addition to sodium at a 1:1 ratio. The remaining reabsorption of glucose is by the SGLT-1 cotransporter in the S3 segment. Chronic hyperglycaemia in diabetes results in upregulation of the SGLT-2 cotransporter and therefore increased glucose and sodium reabsorption. The reduced delivery of sodium to the distal nephron stimulates tubuloglomerular feedback, afferent arteriole dilatation, increased intraglomerular pressure, and hyperfiltration as seen in diabetic kidney disease[2, 3].

SGLT2i competitively binds to SGLT2 cotransporters with greater affinity than glucose. Inhibition of the SGLT2 cotransporters prevents glucose and sodium reabsorption. This results in glucosuria and improved glucose control. In addition, natriuresis increases sodium delivery to the distal nephron, thereby inhibiting tubuloglomerular feedback and reducing hyperfiltration[4]. Other positive effects include reduced podocyte effacement and albuminuria, improved systemic blood pressure, reduced cellular energy expenditure and oxygen consumption, and increased erythropoeitin release[5].

The renoprotective effect of SGTL2i first came to light as part of the EMPAREG trial. Though not specifically designed to look at renal outcomes, the rates of both acute renal failure and acute kidney injury were in fact lower in the empagliflozin group when compared with placebo[6]. This study was followed by the CREDENCE trial which showed a reduction in the risk of kidney failure in patients on canaglifozin with type 2 diabetes and kidney disease[7]. The DAPA-CKD trial showed the nephroprotective effect of dapagliflozin in patients with chronic kidney disease (CKD) regardless of the presence or absence of type 2 diabetes[8]. The subsequent EMPA-KIDNEY trial revealed lower progression of kidney disease in patients with proteinuric CKD on empagliflozin compared with placebo[1].

These studies have consistently shown the renoprotective effects of SGLT2i, including slowed estimated glomerular filtration rate (eGFR) decline, reduced hyperkalaemia, reduced albuminuria, and death due to kidney-related causes. Benefits are seen regardless of the individual agent, the presence or absence of type 2 diabetes, or the degree of proteinuria at initiation. Based on this growing body of evidence, SGLT2i should be started in patients with CKD with or without type 2 diabetes with an eGFR of ≥ 20 mL/min/1.73m^2 and a urinary albumin-to-creatinine ratio (uACR) of ≥ 25 mg/mmol, and those with an eGFR of 20–45 mL/min/1.73m^2 and a uACR of < 25 mg/mmol[9]. This is in addition to the already established RAAS inhibition. The KDIGO 2024 Clinical Practice Guideline for the Evaluation and Management of Chronic Kidney Disease recommends the use of RAASi in those with CKD and severely increased albuminuria without diabetes, and those with CKD and moderately increased albuminuria with diabetes. The guideline also suggests starting RAASi for those with CKD and moderately increased albuminuria without diabetes[10].

The aim of the audit is to identify the proportion of patients with chronic kidney disease with and without proteinuria attending Nephrology OPD that were prescribed renin-angiotensin-aldosterone system blocker (RAASi) and/or SGLT2i, to identify the proportion of people with an indication for use of prescribed RAASi and/or SGLT2i and to identify the proportion of people prescribed an SGLT2i not on concomitant RAASi. We would also explore reasons for non-use of SGLT2i to direct quality improvement projects in the future and to instill awareness amongst health care professionals regarding the prescribing and management of chronic kidney disease. Ultimately, we would like to implement measures to encourage prescribing of RAASi and/or SGLT2 for patients who are eligible as these are now mainstay medications used in the management of chronic kidney disease.

Methods

A prospective audit was performed in Galway University Hospital (GUH) during September 2023 and October 2023. The audit was conducted consecutively with patients attending the Nephrology OPD by doctors during the consultation. The existing use of RAASi and SGLT2i, new prescribing of RAASi and SGLT2i from the consultation and reason for non-use was recorded in the questionnaire. Inclusion criteria include all patients attending the Nephrology OPD, age 18 and over with an existing diagnosis of chronic kidney disease. We excluded patients who did not have a diagnosis of chronic kidney disease.

Results

The audit was conducted during September 2023 and October 2023. A total of 165 audit questionnaires were completed. The baseline characteristics of patients included in the audit are shown in table 1. The mean (±SD) age was 62.1 ± 18.6 years, and 99/165 (60%) were male. The mean systolic and diastolic blood pressure were 137 ± 20.7 mmHg and 78.2 ± 11.0 mmHg respectively. The mean estimated glomerular filtration rate (eGFR) was 46.8 ± 24.5 ml/min/1.73 m^2, the mean urinary protein-to-creatinine ratio (uPCR) was 113.8 ± 147.8 mg/mmol, and the mean urinary albumin-to-creatinine ratio (uACR) was 56.5 ± 97.5 mg/mmol. 40/165 (24.2%) patients had a diagnosis of diabetes mellitus. The most prevalent primary renal diagnoses were diabetic kidney disease; 20/165 (12.1%), hypertensive renal disease; 18/165 (10.9%) and IgA nephropathy; 15/165 (9.1%).

At baseline, medication use of ACEi only, ARB only, SGLT2i only and ACEi/ARB and concurrent SGLT2i at baseline were 42/165 (25.5%), 37/165 (22.4%), 5/165 (3%) and 15/165 (9.1%) respectively. None of the patients were on dual RAASi; appropriately, 15/20 (75%) patients on SGLT2i were on concurrent RAASi.

Table 1: Demographic and Clinical Characteristics of the Participants at Baseline.

	N = 165
Age – year (mean ± SD)	62.1 ± 18.6
Sex – male, n (%)	99 (60%)
Blood pressure – mmHg Systolic (mean ± SD) Diastolic (mean ± SD)	 137 ± 20.7 78.2 ± 11.0
Creatinine – µmol/L (mean ± SD)	158.7 ± 82.9
eGFR – ml/min/1.73 m^2 (mean ± SD)[a]	46.8 ± 24.5
Diabetes – yes (%) Type 2 diabetes mellitus - n (%)	40 (24.2) 33/40 (82.5)

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Type 1 diabetes mellitus - n (%)	6/40 (15)
Cystic fibrosis-related diabetes - n (%)	1/40 (2.5)

Primary renal diagnosis – n (%)	
Acute interstitial nephritis	1 (0.6)
Acute tubular interstitial nephritis	1 (0.6)
Alport's nephropathy	1 (0.6)
Transplanted – n	1
ANCA-associated vasculitis	10 (6.1)
Anti-glomerular basement membrane disease	1 (0.6)
Transplanted – n	1
Autosomal dominant polycystic kidney disease	8 (4.8)
Transplanted – n	3
Cardiorenal syndrome	2 (1.2)
Cholestrol emboli	1 (0.6)
Congenital anomalies of kidney and urinary tract	3 (1.8)
Transplanted – n	3
Diabetic kidney disease	20 (12.1)
Transplanted – n	3
Focal segmental glomerular sclerosis (FSGS)	5 (3.0)
Hypertension	18 (10.9)
Transplanted – n	4
IgA nephropathy	15 (9.1)
Transplanted – n	7
Lithium induced renal disease	3 (1.8)
Lupus nephritis	2 (1.2)
Membranoproliferative nephropathy	1 (0.6)

Transplanted – n	1
Membranous nephropathy	5 (3.0)
Microhaematuria	3 (1.8)
Minimal change disease	2 (1.2)
Monoclonal gammopathy of renal significance	1 (0.6)
Multiple myeloma	1 (0.6)
Nephrocalcinosis	1 (0.6)
Transplanted – n	1
Nephrotic syndrome	1 (0.6)
New patient - no clear diagnosis yet	2 (1.2)
Obstructive nephropathy	3 (1.8)
Post-infection glomerulonephritis	1 (0.6)
Previous acute kidney injury	5 (3.0)
Previous nephrectomy/solitary kidney	10 (6.1)
Previous preeclampsia	1 (0.6)
Recurrent urinary tract infections	2 (1.2)
Reflux nephropathy	7 (4.2)
Transplanted – n	5
Renovascular	9 (5.5)
Secondary FSGS	1 (0.6)
Tumor necrosis factor receptor-associated periodic syndrome (TRAPS)	1 (0.6)
Unclear aetiology of chronic kidney disease	14 (8.5)
Transplanted – n	2

Urolithiasis	2 (1.2)
Verocytotoxin Escherichia coli-associated Haemolytic uraemic syndrome	1 (0.6)

uPCR – mg/mmol (n=67) uPCR ≥ 35 – yes, n (%)	113.8 ± 147.8 42 (62.7%)
uACR – mg/mmol (n=42) uACR > 25 – yes, n (%)	56.5 ± 97.5 17 (40.5)
Proteinuria on urinalysis – yes, n (%)	77 (46.7)
Medications – n (%)	
ACEi only	42 (25.5)
ARB only	37 (22.4)
SGLT2i only	5 (3.0)
RAASi + SGLT2i	15 (9.1)

[a]Based on local hospital laboratory value using CKD-EPI

A total of 82/165 (49.7%) patients may have had indications for medication optimisation including dose up-titration of RAASi as tolerated, starting RAASi and/or starting SGLT2i based on level of proteinuria, most recent kidney function test, in-office blood pressure reading and presence of other indications such as Type 2 Diabetes Mellitus or heart failure. Of these, 17/82 (20.7%) had their medication optimised; 13/82 (15.9%) patients were optimised on RAASi and 4/82 (4.9%) commenced on SGLT2i (Figure 1). By using the UK Kidney Association Clinical Practice Guideline: Sodium-Glucose Co-transporter-2 (SGLT2) Inhibition in Adults with Kidney Disease 2023 Update[9], 65/82 (79.3%) did not receive optimisation of medical therapeutics with RAASi and/or SGLT2i; 26/65 (40%) neither on RAASi or SGLT2i, 33/65 (50.8%) only on RAASi but not on SGLT2i, 3/65 (4.6%) only on SGLT2i but not on RAASi, and 3/65 (4.6%) already on both RAASi and SGLT2i but dosages of RAASi not optimised as tolerated. Table 2 shows potential indication and changes to medication for medical therapeutics optimisation.

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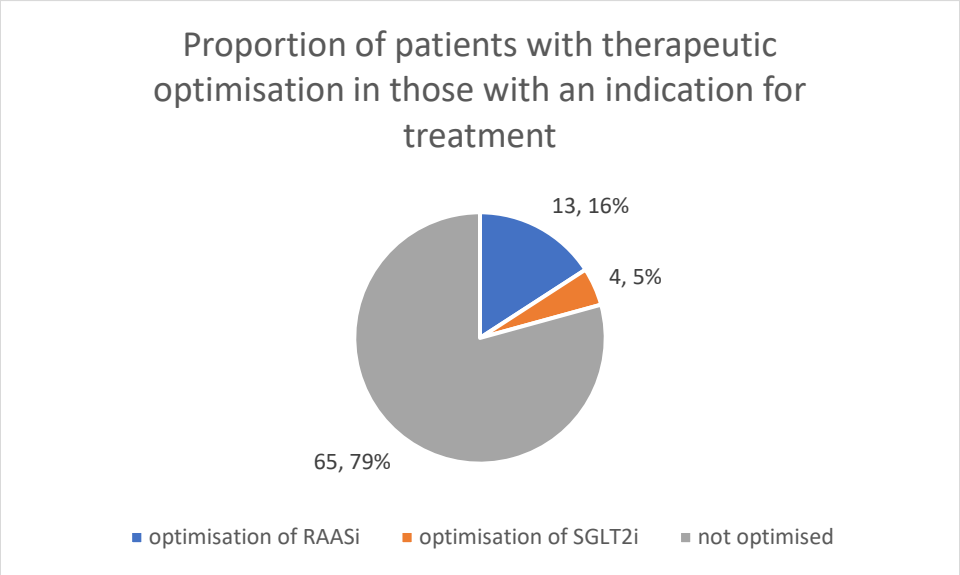

Figure 1: Proportion of patients with CKD who received therapeutic optimization with an indication for treatment.

Table 2: Potential indication and changes to medication for medical therapeutics optimisation, n = 65.*

n = 65	**Indication for RAASi**	**Indication for SGLT2i**	**Total**
eGFR criteria	0	18	18
Proteinuria criteria	3	3	6
Elevated blood pressure	29	0	29
Type 2 diabetes mellitus/heart failure	4	8	12
Total	36	29	65

*Based on level of proteinuria, most recent eGFR, in-office blood pressure reading and presence of other indication such as Type 2 Diabetes Mellitus and heart failure during the out-patient consultation

Among those who did not receive medical therapeutics optimisation with either RAASi and/or SGLT2i, 27/65 (41.5%) did not provide a reason. The majority, 38/65 (58.5%) provided a reason, including, no indication; 8/65 (12.3%), no proteinuria; 10/65 (15.4%), eGFR stable; 4/65 (6.2%), awaiting ambulatory blood pressure monitor (ABPM); 4/65 (6.2%), previous kidney injury; 1/65 (1.5%), and discussed but not yet started; 1/65 (1.5%), and patient declined use; 1/65 (1.5%). Other reasons, 9/65 (13.8%) included already on a RAASi, no medication list provided by patient, previous hyperkalaemia, awaiting uACR, has type 1 diabetes mellitus, awaiting further investigations and concern for insulinopaenia in a patient with cystic fibrosis-related diabetes mellitus. Table 3 shows a summary of reasons for non-use of RAASi and/or SGLT2i.

Table 3: Reasons for non-use of RAASi and/or SGLT2i.

	n (%)
No reason provided	27 (41.5)
No indication for use	8 (12.3)
No proteinuria	10 (15.4)
eGFR stable	4 (6.2)
Awaiting ABPM	4 (6.2)
Previous acute kidney injury with use	1 (1.5)
Use discussed but not started yet	1 (1.5)
Patient declined use	1 (1.5)
Other	9 (13.8)

Discussion

While SGLT2i were first developed for the treatment of hyperglycaemia in those with type 2 diabetes mellitus[11], subsequent trials demonstrated excellent clinical outcomes for heart failure patients across the ejection fraction spectrum[12] and in those chronic kidney disease with and without type 2 diabetes mellitus[13, 14]. Recent research has expanded their scope to include chronic kidney disease with lower eGFR and with varying degree of proteinuria[1].

In our cohort of patients, 40% had a diagnosis of diabetes but the remaining 60% did not. The majority of our cohort with diabetes mellitus had type 2 diabetes. 46.7% of patients had evidence of proteinuria on urine dipstick. Despite this, only 9.1% of patients were on both a RAASi (ACEi or ARB) and an SLGT2i, 47.9% of patients were on RAASi only and 3% of patients were on SGLT2i only. This suggests there may be a significant proportion of patients who were not receiving appropriate therapies. This is significant although the UK Kidney Association Guidelines, which were published after this audit was conducted, advocate for more cautious use of SGLT2i in those with diabetic kidney disease and type 1 diabetes. They recommend diabetes team input prior to initiation of an SGLT2i in those patients with type 1 diabetes, as well as those with type 2 diabetes at a greater risk of diabetic ketoacidosis[9].

We surveyed doctors in the OPD in an attempt to identify the reasons for the suboptimal use of SGLT2i; 41.5% provided no reason for non-use. Of those who did provide a reason, the most common was that there was no indication for use of an SGLT2i. While a proportion of patients seen in the OPD may attend without CKD, the majority would indeed have an indication as per the latest UKKA guidelines[9]. This discrepancy is likely due to lack of awareness in the trainee doctor population regarding indications and new evidence.

Under-prescribing of SGLT2i relative to other pharmacological therapies has also been observed in the cardiology setting. Studies found that Endocrinologists are more likely to prescribe these agents and this may be due to a greater familiarity, with their use being established in their field for longer[15]. This highlights a need for an active educational effort, particularly targeted at non-consultant hospital doctors who comprise the majority of the clinical staff servicing nephrology OPDs. Clinical pathways for the use of SGLT2i have been proposed as a means of enhancing translation of research to clinical practice[16]. Development and distribution of such a proforma for local use represents a low-cost intervention to address this issue and improve patient care.

When considering physician awareness and education, we must also reflect on the role of primary care clinicians. The role of primary care physicians in the care of patients with CKD is becoming increasingly important[17]. The expansion of chronic disease management in the community via an integrated model of care is key to the Irish Health Service Executive's

Slaintecare program[18]. Specialist-led educational programs directed towards community-based physicians have effectively improved prescribing rates of SGLT2i and GLP-1a among patients with diabetes, and represent a further potential strategy to expand appropriate SGLT2i prescribing in CKD[19].

It is important to recognise that if aiming to increase SGLT2i prescription rates through awareness, that physicians are also educated about their side-effect profile and contraindications to therapy[9]. Patient education represents a further challenge. For example, they must be appropriately counseled regarding sick day medication guidance so as to minimise the occurrence of adverse events[20].

Limitations of this audit include over-estimation of under-prescribing of RAASi and SGLT2i. There were 27/65 (41.5%) questionnaires which did not include the reason for non-use of RAASi or SGLT2i. There is likely a number of patients who were appropriately withheld from these therapies while awaiting further investigations including repeat blood test, blood pressure monitoring or urine test. Also, optimization of RAASi is aimed at achieving maximum tolerated dose of RAASi by the patients, therefore, the questionnaire may not have captured this accurately, hence an over-estimation of its under-prescribing. Lastly, this audit was conducted less than a year after the publication of the EMPA-KIDNEY trial[1] and soon before the 2023 Updated UK Kidney Association Clinical Practice Guidelines[9] were introduced. Therefore, change in clinical prescribing practice may not have translated to real life practice at the time of the audit. However, this audit can serve as a baseline representing the prescribing practice in our local unit and can be used as reference when this audit is repeated in the future to detect change in clinical prescribing practice.

Discussion

Chronic kidney disease is highly prevalent and this will increase in parallel with the aging population. Unfortunately, there is no cure for chronic kidney disease however efforts must be put in place to halt the progression of chronic kidney disease and development of kidney failure. There is new evidence to support the use of SGLT2i, in those with chronic kidney disease across a wide range of eGFR, and degree of proteinuria to slow its progression. SGLT2i use is underutilised in our OPD. This is attributable to lack of awareness amongst physicians regarding the indication of use of SGLT2i in chronic kidney disease, heart failure and diabetes mellitus. Ideally, these patients should be initiated on an SGLT2i after being established on a RAASi early in their diagnosis for maximal renoprotective effects of SGLT2i. Continuous efforts to raise awareness of new therapeutics available must be instigated in order to overcome prescribing

inertia of new proven therapeutics in lowering the risk of progression of chronic kidney disease. These audit findings were presented at our Departmental Journal Club to raise awareness amongst health care professionals regarding the prescribing and management of chronic kidney disease. A re-audit within the department is also planned for 2025.

Declarations of Conflicts of Interest:

J.W. Teh received speaker fees from Boehringer Ingelheim. D.W. Lappin received speaker fees from Astra Zeneca. L. Harris and N. Corcoran declare no conflict of interest.

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