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Short Communication

Basics of the Management of Chronic Kidney Disease for the General Physician

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Abstract

Adaptive hyperfiltration is the main mechanism leading to progression of chronic kidney disease (CKD) after an insult/injury, even if this injury is cured. The kidney responds to an injury by increasing the Glomerular Filtration Rate (GFR) to the normal nephrons, this effect can keep the creatinine (Cr) at a normal level initially, but with time, the kidney function deteriorates due to the chronic high pressure in the nephrons induced by raised GFR, this deterioration is monitored by worsening proteinuria and decline in Cr level.

The rationale of using medications like SGLT2/ACE inhibitors or ARB in CKD is based on this adaptive hyperfiltration mechanism, these medications help in reducing GFR and hence reducing proteinuria and intrarenal pressure and preserving the kidney function or at least minimizing the rate of decline in kidney function.

Keywords: Acute Kidney Injury; Hyperkalemia; Renal disease; Glomerular filtration rate.

Abbreviations: CKD: Chronic Kidney Disease; GFR: Glomerular Filtration Rate; Cr: Creatinine; CVD: Cardiovascular Disease; BP: Blood Pressure; ESRF: End Stage Renal Failure; HPT: Hyperparathyroidism.

Introduction

Adaptive hyperfiltration is the main mechanism leading to progression of chronic kidney disease (CKD) after an insult/injury, even if this injury is cured. The kidney responds to an injury by increasing the Glomerular Filtration Rate (GFR) to the normal nephrons, this effect can keep the creatinine (Cr) at a normal level initially, but with time, the kidney function deteriorates due to the chronic high pressure in the nephrons induced by raised GFR, this deterioration is monitored by worsening proteinuria and decline in Cr level [1].

The rationale of using medications like SGLT2/ACE inhibitors or ARB in CKD is

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based on this adaptive hyperfiltration mechanism, these medications help in reducing GFR and hence reducing proteinuria and intrarenal pressure and preserving the kidney function or at least minimizing the rate of decline in kidney function [2].

Progressive decline in renal function is asymptomatic initially and usually discovered on routine blood test, however, when patient reach stage 4/5, this is when symptoms occur, for example, fluid overload due to oliguria, hyperkalaemia, metabolic acidosis, anaemia, hypertension, and bone disorders.

End stage renal failure usually manifests with uraemia, signs of uraemia include nausea and vomiting, encephalopathy, pericardial effusion/pericarditis, and peripheral neuropathy. The rate of progression is variable among patients and depends on many factors [3]. The stage of CKD has an impact on the rate of progression, stage 3 progress faster to ESRF, compared to the rate of progression of stage 1 to stage 2, it appears that once the GFR is below 60 the rate of progression of CKD is faster with an expected decline in GFR by 1.5% annually [4]. Another factor which affects the rate of decline in GFR is the presence or absence of proteinuria, a low GFR below 60 with proteinuria have a rapid course of progression of CKD compared to either alone [5]. Just to recap, the definition of CKD is a decline in renal function or albuminuria of 30 mg/day or more, for three months or more. Classification of CKD is important as it helps with management planning and risk stratification. Classification is based on the cause of kidney injury, GFR, and presence or absence of proteinuria [6]. GFR in mL/min/1.73 m2 over 90 in presence of kidney injury is G1, below 90 to 60 is G2, below 60 to 45 is G3a, below 45 to 30 is G3b, below 30 to 15 is G4 and below 15 is G5 or end stage renal failure [6]. Degree of albuminuria if present, A1 is absence of albuminuria (below 30 mg/day), A2 is 30 to 300 mg/ day, used to be called microalbuminuria, and A3 is albuminuria more than 300 mg/day [6].

Discussion

CKD is an independent risk factor for cardiovascular disease (CVD) and patients with CKD do commonly have other risk factors for CVD like diabetes, hypertension, and metabolic syndrome. Aggressive risk factors management is strongly indicated to reduce the risk of CVD and related events [7]. The management of CKD is focused on reducing risk factors to prevent CVD and progression of CKD, and treatment of CKD complications as will be mentioned below.

Intensive blood pressure (BP) control below 130/80 can reduce the rate of progression of CKD with proteinuria, and no effect in the progression of the rate of CKD in nonproteinuric patients, while the intense BP control reduce the risk of CVD in both groups [8]. Treating proteinuria is another essential step in reducing the rate of CKD progression to end stage renal failure (ESRF), ACE inhibitors and ARB are first line treatment for patients with CKD and proteinuria, their effect on patients with CKD without proteinuria is like other classes of antihypertensives. SGLT2 inhibitors have been shown to be beneficial for patients with CKD and proteinuria in presence or absence of diabetes [9].

Other factors which help in declining the rate of progression of CKD are bicarbonate

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therapy in patients with CKD and metabolic acidosis, smoking cessation, dyslipidaemia, and strict glycaemic control if safe to do so, glycaemic control can reduce the rate of decline in GFR and prevent or reduce the rate of proteinuria, GLP-1 RA as well as SGLT2 inhibitor can reduce the CKD progression rate [2]. It is explained so far, the classification of CKD, reason for CKD progression, and the important risk factors to treat to prevent the progression of CKD, below it will discuss the management of complications which occur with patients with CKD. Fluid overload is very common in patients with ESRF, however, patients with GFR below 60 are prone to fluid overload as their kidneys are less capable of dealing with the rapid intake of sodium and water, recommendations are sodium restriction below 2 g/day and loop diuretics for fluid overload [6]. Hyperkalaemia is common in advance renal disease, the rationale is due to three factors, firstly, potassium excretion is reduced with oliguria, which is common in advance CKD, secondly, the degree of hypoaldosteronism which can occur in patients who are on ACEi/ARB, and finally, the degree of metabolic acidosis. Management of hyperkalaemia is potassium restriction in diet, reverse acidosis, and avoid medications which can increase potassium like, ACEi/ARB/NSAIDs, or Trimethoprim [10]. Metabolic acidosis is common in advanced CKD, some evidence that bicarbonate levels can slow the progression of CKD, aim to keep bicarb level between 20-22 mEq/L and caution with fluid status as bicarbonate supplements contain sodium [11].

Hyperphosphatemia is common in advanced CKD due to phosphate retention and leads with hypocalcaemia from low Vitamin D to secondary hyperparathyroidism (HPT) [12]. HPT leads to the development of renal osteodystrophy including osteitis fibrosa. Management aims at reducing HPT by oral phosphate binders with dietary restriction of phosphate. Calcitriol is an active metabolite of vitamin D and levels are reduced in poor renal function, usually GFR below 40 and secondary to phosphate retention, this leads to hypocalcaemia.

Alfacalcidol, calcium supplements and cinacalcet (Calcimimetics) can all help to reduce HPT and mineral bone disease in CKD [13]. Normocytic anaemia (normal MCV, reduced Hb below 12 in men and 11 in women) is common in progressive CKD stage 4/5 due to reduced erythropoietin levels [14]. Iron deficient anaemia (IDA) should be ruled out first before initiating erythropoietin treatment for anaemia of CKD, serum ferritin below 100 with transferrin saturation below 20% are indications for IDA [15]. Statin therapy is strongly indicated in patients with CKD, statins are safe and efficient in reducing LDL in general population and in patients with CKD who carry a high risk of CVD, and as mentioned before CKD is independent risk factor for CVD [16].

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