Austin Journal of Anatomy

Open Access

Austin Publishing Group

Editorial

Chronic Kidney Disease (CKD) - Where Clinical Medicine Meets Clinical Anatomy

Muhammad S*
Co-founder and Chief in Research, The Renal Patient Support Group (RPSG), UK

***Corresponding author:** Shahid Muhammad, Co-Founder and Chief in Research, The Renal Patient Support Group (RPSG), UK

Received: June 03, 2015; **Accepted:** June 04, 2015; **Published:** June 05, 2015

Editorial

Over the last few years, collaborative efforts, enabled by a common definition of Chronic Kidney Disease (CKD) have provided a description of the epidemiology, natural history, and outcomes of this disease and have improved our understanding of its pathophysiology. There is increased recognition that CKD is encountered in multiple settings and in all age groups, and that its course and outcomes are influenced by the severity and duration of the causative event. The effect of CKD on an individual patient and the resulting societal burden that ensues from the long-term effects of the disease is attracting increasing scrutiny. There is evidence of marked variation in the management of CKD due to a lack of awareness and an absence of standards for prevention, early recognition, and intervention. These emerging data point to an urgent need for a global effort to highlight that CKD is preventable, its course is modifiable and its treatment can improve outcomes [1].

Between 2005 and 2009, the prevalence of stages (3-5) CKD increased from 0.3% to 3.9%. In 2009, 30,440 patients (1.1% unadjusted) fulfilled biochemical criteria for CKD but were not on a practice CKD register (uncoded CKD) and 60,705 patients (2.2% unadjusted) were included on a practice CKD register but did not fulfil biochemical criteria (miscoded CKD) [2]. For patients with confirmed CKD, inclusion in a practice register was associated with increasing age, male sex, diabetes, hypertension, cardiovascular disease and increasing CKD stage ($p<0.0001$) [2]. In one study, the mean age of cohort at the beginning of the study period was 64.8 years, 55% were female. In those patients with stage 3-5 CKD 83.9% were hypertensive, defined by a Pre-Pay 4 Performance Programme (P4P) Blood Pressure (BP) of >140/85 or currently taking antihypertensive medication [3]. Population BP control has improved since the introduction of P4P renal indicators, and this improvement has been sustained. This was associated with a significant increase in the use of antihypertensive medication, resulting in increased prescription cost [3].

In another study, newly diagnosed CKD patients referred to a nephrology clinic with open ended questions about their understanding of CKD causes, symptoms, and management [4]. 210 patients were surveyed. Median age was 66.5 years, 50.5% female. Prevalence of risk factors for CKD included 31% diabetic, 62% hypertension, 19% family history of CKD. CKD stage prevalence was 0 (8%), 1 (24%), 2 (11%), 3 (38.5%), 4 (18%), and 5 (0.5%). 18% were primary school educated. 82% were referred by their primary care physician and 29% had seen a nephrologist previously [4]. 16% were unsure why they had been referred. CKD causes identified by patients were unsure (40%), alcohol (29%), hypertension (16%), and diabetes (14%). Symptoms identified included asymptomatic (16%), kidney pain (17%) and other (42%).Management suggested patients were uncertain (51%), dialysis (32%) and anti-hypertensive medication (16%). 82% reported unsatisfactory education from their primary care physician. New patients referred to a renal outpatient department had poor knowledge of CKD [4]. Education of patients should begin in primary care prior to referral. Education needs to be targeted at a simplistic level and perhaps GPs should look to get more patient involvement through support groups [5].

In regard to the above, immunology, transplantation, leukocyte trafficking, chemokine receptor expression and entities of vascular inflammation have to be some of the most intriguing and crucial topics in nephrology/ cardiology generally [6-11]. Indeed the autoimmune disease Systemic Lupus Erythematosus (SLE), Immunoglobulin A Nephropathy (IgA), Nephrotic Syndrome (NS), are becoming progressively important to understand in paediatric nephrology, especially where young patients can develop vascular/ cardiovascular complications post-transplant [12]. It is also important that physicians appreciate specific laboratory techniques to retrieve data on inflammatory disease and migration patterns, for example flow cytometry and immunoassays. The decline of renal function has been associated with a major increase in serum cytokine concentration in CKD patients, [13] state:

"Creatinine clearance correlates with the circulating levels of various cytokines and their soluble receptors in patients with varying degrees of renal failure".

Clinicians should become better acquainted with diseases, which surround molecular biology [7]. Molecular biology-based subjects surrounding paediatric nephrology have long been crucial, especially with respect to podocytes and their role in renal function and protein leaking. When podocytes contract, they cause closure of filtration slits. This action decreases the Glomerular Filtration Rate (GFR) by reducing the surface area available for filtration. [14] At the University of Bristol, England UK have made grand strives with respect to developing Podocyte and Nephrin research. The investigators have been working continuously on projects to explore the function of Nephrin in order to gain insights of its expression in specific renal disease-states [14-18]. Thus molecular mechanisms of renal damage in young people with CKD and diabetic nephropathy are to be appreciated more generally. Whilst renal anatomical research is becoming more intricate, especially in light of protein leaking/ podocyte studies, longer-term follow-up of

Citation: Muhammad S. Chronic Kidney Disease (CKD) - Where Clinical Medicine Meets Clinical Anatomy. Austin J Anat. 2015;2(2): 1035.

patients with suspect/ high risk CKD will establish whether or not this translates to improved outcomes in terms of progression of overall CKD, cardiovascular disease and patient mortality [3]. This means awareness requires tighter collaborations.

References

1. Lewington AJ, Cerda J, Mehta RL. Raising awareness of acute kidney injury: a global perspective of a silent killer. Kidney Int. 2013; 84: 457-467.
2. Jain P, Calvert M, Cockwell P, McManus RJ. The need for improved identification and accurate classification of stages 3-5 chronic kidney disease in primary care: retrospective cohort study. PLoS One. 2014; 9: e100831.
3. Karunaratne K, Stevens P, Irving J, Hobbs H, Kilbride H, Kingston R, et al. The impact of pay for performance on the control of blood pressure in people with chronic kidney disease stage 3-5. Nephrol Dial Transplant. 2013; 28: 2107-2116.
4. Burke MT, Kapojos J, Sammartino C, Gray NA. Kidney disease health literacy among new patients referred to a nephrology outpatient clinic. Intern Med J. 2014; 44: 1080-1086.
5. Muhammad S, Allan M, Ali F, Bonacina M, Adams M. The renal patient support group: supporting patients with chronic kidney disease through social media. J Ren Care. 2014; 40: 216-218.
6. Fogo AB. Mechanisms of progression of chronic kidney disease. Pediatr Nephrol. 2007; 22: 2011-2022.
7. Grunfeld JP. Congenital/inherited kidney diseases: how to identify them early and how to manage them. Clin Exp Nephrol. 2005; 9: 192-194.
8. Hostetter TH, Kochis DJ, Shaffer RN, Chertow G, Harmon WE, Klotman PE, et al. World Kidney Day 2011. J Am Soc Nephrol. 2011; 22: 397-398.
9. Kogon AJ, Pierce CB, Cox C, Brady TM, Mitsnefes MM, Warady BA, et al. Nephrotic-range proteinuria is strongly associated with poor blood pressure control in pediatric chronic kidney disease. Kidney Int. 2014; 85: 938-944.
10. Shroff R, Weaver DJ, Mitsnefes MM. Cardiovascular complications in children with chronic kidney disease. Nat Rev Nephrol. 2011; 7: 642-649.
11. Ventura HO, Mehra MR. Improvement of heart failure after renal transplantation: the complex maze of cardio-renal interaction. J Am Coll Cardiol. 2005; 45: 1061-1063.
12. In the Know about Nephrotic Syndrome Seminar, Royal Manchester Childrens Hospital. 2014.
13. Pecoits-Filho R, Sylvestre LC, Stenvinkel P. Chronic kidney disease and inflammation in pediatric patients: from bench to playground. Pediatr Nephrol. 2005; 20: 714-720.
14. Coward RJ, Welsh GI, Koziell A, Hussain S, Lennon R, Ni L, et al. Nephrin is critical for the action of insulin on human glomerular podocytes. Diabetes. 2005; 56: 1127-1135.
15. Saleem MA, Ni L, Witherden I, Tryggvason K, Ruotsalainen V, Mundel P, et al. Co-localization of nephrin, podocin, and the actin cytoskeleton: evidence for a role in podocyte foot process formation. Am J Pathol. 2002; 161: 1459-1466.
16. Saleem MA, O'Hare MJ, Reiser J, Coward RJ, Inward CD, Farren T, et al. A conditionally immortalized human podocyte cell line demonstrating nephrin and podocin expression. J Am Soc Nephrol. 2002; 13: 630-638.
17. Saleem MA. Biology of the human podocyte. Nephron Exp Nephrol. 2003; 95: e87-e92.
18. Saleem MA. New developments in steroid-resistant nephrotic syndrome. Pediatr Nephrol. 2013; 28: 699-709.

Austin J Anat - Volume 2 Issue 2 - 2015
ISSN : 2381-8921 | www.austinpublishinggroup.com

Citation: Muhammad S. Chronic Kidney Disease (CKD) - Where Clinical Medicine Meets Clinical Anatomy. Austin J Anat. 2015;2(2): 1035.