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Outcomes of Diabetic Kidney Disease and the Progression to Kidney Failure

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Chronic kidney disease (CKD) presents a significant global health challenge because of its prevalence and association with increased morbidity, mortality and economic burden. Chronic Kidney Disease (CKD) affects 30%–40% of diabetic patients over their lifetime.[1] The classically described natural history of diabetic kidney disease includes glomerular hyperfiltration, progressive albuminuria, declining GFR, and ultimately, end stage kidney disease (ESKD). However, this pathway more typically applies to kidney disease occurring in patients with type 1 diabetes mellitus. In type 2 diabetes mellitus (T2DM), kidney involvement often includes tubular, interstitial, and vascular damage. There is also significant variability in progression, and variable presence of albuminuria. It has also been recognised that CKD in patients with diabetes is heterogenous i.e. other processes such as hypertensive nephrosclerosis or vascular disease may also be involved. To avoid the assumption that CKD in diabetes universally results from traditional diabetic pathophysiology, the Kidney Disease: Improving Global Outcomes (KDIGO) 2022 clinical practice guideline for diabetes management in CKD adopted the term " CKD in patients with diabetes". [2] However, as long as this limitation is recognised, the term "diabetic kidney disease" is appropriate and would encompass the spectrum of patients with diabetes who exhibit albuminuria and/or reduced renal function. Overall only 10% of patients with DKD progress to kidney failure while the vast majority (90%) die from cardiovascular events or heart failure[1]. The excess CV risk is further heightened in patients with diabetes who go on to kidney replacement therapy. Strategies to minimise or prevent CV complications are therefore equally important as those targeting kidney disease progression. This talk will explore the outcomes of diabetic kidney disease and the risk factors associated with kidney disease progression. These include genetic predisposition, hyperglycaemia, hyperfiltration, hypertension, albuminuria and the increasingly recognised role of inflammation and fibrosis, altered gut microbiome, phenotypic cell transformation and programmed

cell death. References 1. Tuttle KR, Wong L, St Peter W, Roberts G, Rangaswami J, Mottl A, et al.; Diabetic Kidney Disease Collaborative Task Force: Moving from evidence to implementation of breakthrough therapies for diabetic kidney disease. Clin J Am Soc Nephrol 17: 1092–1103, 2022 10.2215/CJN.02980322 PubMed 2. Rossing P, Caramori ML, Chan JCN, Heerspink HJL, Hurst C, Khunti K, et al.: KDIGO clinical practice guideline for diabetes management in chronic kidney disease. Kidney Int 102: S1–S127, 2022 10.1016/j.kint.2022.06.008 PubMed

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