

# Prevention—

# Prevention—

CONTENTS 21.10.1 Diabetes mellitus and the kidney 4975 Rudolf Bilous 21.10.2 The kidney in systemic vasculitis 4988 David Jayne 21.10.3 The kidney in rheumatological disorders 5001 Liz Lightstone and Hannah Beckwith 21.10.4 The kidney in sarcoidosis 5012 Ingeborg Hilderson and Jan Donck 21.10.5 Renal involvement in plasma cell dyscrasias, immunoglobulin-based amyloidoses, and fibrillary glomerulopathies, lymphomas, and leukaemias 5016 Pierre Ronco, Frank Bridoux, and Arnaud Jaccard 21.10.6 Haemolytic uraemic syndrome 5027 Edwin K.S. Wong and David Kavanagh 21.10.7 Sickle cell disease and the kidney 5032 Claire C. Sharpe 21.10.8 Infection-associated nephropathies 5034 A. Neil Turner 21.10.9 Malignancy-associated renal disease 5041 A. Neil Turner 21.10.10 Atherosclerotic renovascular disease 5044 Philip A. Kalra and Diana Vassallo 21.10.1 Diabetes mellitus and the kidney Rudolf Bilous

**ESSENTIALS** Diabetic nephropathy is the commonest cause of endstage renal disease in the developed world, causing 38% of prevalent cases and 47% of incident cases requiring renal replacement therapy in the United States of America in 2016, and 18% of prevalent cases and 29% of incident cases in the United Kingdom in 2017. Most patients have type 2 diabetes, and in most countries the proportion with endstage renal disease who have type 1 diabetes is falling. Aetiology and pathology—causation is related to glycaemic control (e.g. glycation of proteins, oxidative stress, sorbitol overproduction, and alteration in growth factors), hypertension, inflammation, genetic factors, and dietary and other environmental factors. Pathological hallmarks in the glomerulus are thickening of the glomerular basement membrane and mesangial expansion, with or without nodule formation, secondary to an accumulation of extracellular matrix. Many patients have a varying severity of tubulointerstitial inflammation and fibrosis. Staging and natural history—is classically described in terms of urinary albumin excretion rate (UAER): (1) normoalbuminuria—UAER less than 20 µg/min, albumin:creatinine ratio (ACR) less than 2.5 mg/mmol (men), less than 3.5 mg/mmol (women); (2) microalbuminuria (also called incipient nephropathy, but now termed moderately increased albuminuria)—UAER 20 to 200 µg/min, ACR 2.5 to 30 mg/mmol (men), 3.5 to 30 mg/mmol (women); and (3) clinical proteinuria (sometimes called clinical nephropathy or overt nephropathy, but now termed severely increased albuminuria)—UAER greater than 200 µg/min, ACR greater than 30 mg/mmol. This staging maps better to the latest classification of chronic kidney disease based upon estimated glomerular filtration rate. Clinical features—most patients (>60%) will have a normal UAER throughout their diabetic life, but 1 to 2% of the remainder develop persistent moderately increased albuminuria each year. Once UAER exceeds 200 µg/min, there tends to be a relentless increase in proteinuria and glomerular filtration rate declines progressively at a rate that largely depends upon blood pressure control. Prevention—in both type 1 and type 2 diabetes, tight glycaemic control can prevent moderately increased albuminuria. Whether intensive blood pressure control using angiotensin-converting enzyme (ACE) inhibitors

can also prevent this remains controversial. In both type 1 and type 2 diabetes, intensive blood pressure control using ACE inhibitors or angiotensin II receptor blockers (ARBs) slows progression from moderately to severely increased albuminuria and also slows the rate of decline in glomerular filtration rate in those with severely increased albuminuria. Management—aims for (1) control of glycaemia—typical recommendations are for a glycated haemoglobin level <48 mmol/mol 21.10  
The kidney in systemic disease

---

Revision #1

Created 2026-01-22 16:42:09 UTC by Omar Ayman

Updated 2026-01-22 16:42:09 UTC by Omar Ayman