key points

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Proteinuria originates from the kidney and occurs as a result of injury to either the glomerulus or the renal tubule or both. It is relatively common in the general population with reported point prevalence of up to 8% but the prevalence falls to around 2% on repeated testing.

Chronic glomerular injury resulting in proteinuria may be secondary to prolonged duration of diabetes or hypertension. A tubular origin of proteinuria may be associated with inflammation of renal tubules triggered by prescribed drugs or ingested toxins. In the absence of obvious clues to the cause of persistent proteinuria on history or clinical examination it is worthwhile reviewing the patient's prescribed drugs to identify any potentially nephrotoxic agents e.g. NSAIDs.

NICE guidelines recommend screening for proteinuria in individuals at higher risk for chronic kidney disease (CKD). These include patients with diabetes, hypertension, cardiovascular disease, connective tissue disorders, a family history of renal disease and those prescribed potentially nephrotoxic drugs. Microalbuminuria, the earliest clinically detectable sign of diabetic nephropathy, represents low level albuminuria and is not detected by dipstick urinalysis but requires measurement by urinary albumin:creatinine ratio (ACR).

In patients with CKD the amount of proteinuria tends to be directly correlated with a faster rate of decline of kidney function as well as an associated substantially increased cardiovascular disease risk. Blood pressure measurement and kidney function estimation are both important because of the association of proteinuria with hypertension and CKD. Patients with sudden onset of lower limb oedema and associated proteinuria should have a serum albumin level measured to exclude the nephrotic syndrome. Renal tract ultrasound will measure kidney size, and detect scarring associated with chronic pyelonephritis or prior renal stone disease which can cause proteinuria.

For patients with persistent proteinuric renal disease the main therapeutic option is treatment with an ACE inhibitor or angiotensin receptor blocker (ARB). Addressing components of overall cardiovascular risk is vital in the management of all proteinuric renal conditions. In the management of CKD, the aim is to up-titrate ACE inhibitor or ARB therapy to the maximum tolerated dose in order to reduce proteinuria to a urinary ACR <70. Patients should be warned to stop these drugs temporarily in the presence of intercurrent illnesses leading to volume depletion. If an ACE inhibitor or ARB is being prescribed care should be taken with other medications such as NSAIDs, potassium-sparing diuretics and trimethoprim which can contribute to serious hyperkalaemia.