Box 21.10.1.2 Clinical features suggestive of nond

Box 21.10.1.2 Clinical features suggestive of nondiabeticrenal disease

21.10.1 Diabetes mellitus and the kidney 4983 estimate, although the UKPDS suggests that rates are similar to type 1 (Table 21.10.1.2). Up to 7% of newly diagnosed type 2 patients in the United Kingdom will have a urinary albumin concentration above 50 mg/litre, and 1% will be above 300 mg/litre. Some studies have reported a reduction in UAER with initial glycaemic correc- tion, but many patients have a sustained increase suggesting estab- lished nephropathology at diagnosis. GFR As previously mentioned, GFR at diagnosis of type 1 and type 2 diabetes is increased in 40 to 45% of patients. It returns to normal in most following glycaemic correction, although a significant minority maintain persistently high values (hyperfiltration). In nondiabetic humans, the GFR declines by 1 ml/min per year after the age of 40, and it does so also in normotensive diabetic patients who have normal UAER. As the UAER approaches and exceeds the severely elevated albuminuria threshold, there tends to be a steady decline. This is particularly so in hypertensive patients, in whom the rate of loss of GFR varies considerably. In those with poorly con- trolled hypertension, the average decline was 10 ml/min per year in historical series, leading to endstage renal disease in 7 to 10 years. More recently, the rate of decline is 2–4 ml/min per year in patients with well-controlled systemic blood pressure, effectively delaying endstage renal disease by 15–20 years. Patients with type 2 diabetes and a normal UAER tend to have a much slower rate of loss of GFR. It is now recommended that all people with diabetes have an eGFR performed annually using the Modification of Diet in Renal Disease (MDRD) or Chronic Kidney Disease Epidemiology Collaboration (CKD-EPI) equations. Blood pressure In patients with type 1 diabetes, blood pressure is virtually always normal at diagnosis. This is not the case in type 2 diabetes, where over one-third will have blood pressure higher than 160/95 mmHg and many more are hypertensive by recent criteria. Type 1 patients who go on to develop moderately elevated albuminuria have signifi- cantly higher blood pressures than those who remain with a normal UAER, although the averages remain below 140/90 mm Hg in both groups. Patients with newly developed moderately elevated albu- minuria show a steady increase in blood pressure such that over 45% exceed 140/90 mmHg within

4 years. Most type 1 and type 2 patients with severely elevated albuminuria are hypertensive and on therapy. Clinical concomitants of nephropathy Many patients with diabetic nephropathy will also have retinopathy and neuropathy, which will tend to progress. Both of these com- plications can be reversed or at least ameliorated by improved gly- caemic control. There is an increased incidence of cardiovascular, cerebrovascular, and peripheral vascular disease; intensive manage- ment of modifiable cardiovascular risk factors is essential (see later). Amputation rates in patients with diabetic nephropathy are high; careful foot surveillance and preventative podiatry are essential. Differential diagnosis It is important to remember that not all renal or urinary tract dis- ease in diabetic patients is due to diabetic nephropathy. Urinary tract infection is more common in diabetic women compared to age-matched nondiabetic controls. Infection is often asymptom- atic and culture should always be performed in any patient with an isolated positive urinalysis for protein, blood, leucocytes, or nitrite. A positive result is much more likely if two or more of these tests are positive. Papillary necrosis has been described in women with long- standing type 1 diabetes and is a recognized complication of hyperosmolar coma in patients with both types of diabetes. Atheromatous renovascular disease is also common in diabetes, but the precise prevalence of functionally significant renal artery sten- osis is uncertain. Whereas the vast majority of type 1 patients with moderately in- creased albuminuria who undergo renal biopsy are found to have histologically proven diabetic glomerulopathy, the situation is less certain in type 2 diabetes. Up to 10% of such patients have evi- dence of nondiabetic pathologies, many have nonspecific ischaemic changes, and only a few have classic diabetic lesions. The presence of diabetic retinopathy is helpful as those with it are almost certain to have diabetic glomerulopathy and those without it much less so. Even so, there are few cases of specifically treatable glomerular dis- ease in those with nondiabetic lesions, hence management is un- likely to be significantly different; although those with nonclassic lesions tend to have slower rates of decline of GFR and may be at lower risk of endstage renal disease. Clinical investigation Type 2 diabetes is becoming more common and as a result the chance of concomitant nondiabetic renal or urological disease is increased. The need to exclude urinary tract infection has already been mentioned. Current United Kingdom guidelines suggest investigation and possible referral of all diabetic pa- tients with persistent nonvisible or visible haematuria. An atyp- ical presentation of proteinuria, or an unusual clinical course such as rapidly deteriorating GFR, or the presence of features of other systemic diseases should prompt referral and inves- tigation (Box 21.10.1.2). Current United Kingdom guidelines suggest expert review of all with an eGFR of less than 30 ml/ min per 1.73 m2. Box 21.10.1.2 Clinical features suggestive of nondiabetic renal disease • Increased UAER/clinical proteinuria/nephrotic syndrome in absence of retinopathy • Low GFR with normal UAER • Rapidly declining GFR (>15 ml/min per year or >25% in 1 year) • Rapidly increasing proteinuria • Refractory hypertension (use of four or more agents)—consider renal artery stenosis • Presence of active urinary sediment (red cells, cellular casts) • Signs or symptoms of other systemic disease • A greater than 30% reduction in GFR within 2–3 months of initiation of renin–angiotensin system blocking agents—consider renal artery stenosis

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Revision #1
Created 2026-01-22 16:41:47 UTC by Omar Ayman
Updated 2026-01-22 16:41:47 UTC by Omar Ayman