

# Diabetic Nephropathy

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## Goal

Review the pathophysiology, management and treatment of diabetic nephropathy (DN).

## Objectives

1. Identify major changes in kidney pathophysiology due to diabetes.
2. Describe the management of the diabetic patient and the prevention of DN.
3. Discuss the management and treatment of the patient with DN.

## Introduction

The worldwide prevalence of diabetes is increasing with healthcare in Australia and New Zealand being affected. Data from the AusDiab study (Chadban, Briganti, Kerr et al 2003) suggest that 7.2% of adult Australians (more than 900,000 people) have type 2 diabetes. It also showed that the prevalence of chronic kidney disease (CKD) was three times higher in those with diabetes compared with those without. Diabetic nephropathy (DN) is the leading cause of Stage 5 CKD (GFR < 15ml/min or dialysis dependent) in Australia (30%) and New Zealand (40%) (ANZDATA 2005). This is also the case for USA, Japan and most of industrialised Europe (Woredekal & Friedman 2004). Furthermore Kidney Health Australia (KHA) estimate that over 4.5 million people, in Australia alone, are at risk of developing CKD due to the presence of diabetes or hypertension. The burden of CKD relating to diabetes, particularly type 2 diabetes, is likely to increase further as both the age of the population and prevalence of type 2 diabetes are expected to rise (AIHW 2002).

## Diabetic Nephropathy

Diabetic kidney disease is a well described complication of diabetes that results from glomerular (as well as tubular and interstitial) damage secondary to continued hyperglycaemia. The key

changes in diabetic glomerulopathy is thickening of the basement membrane, mesangial cell hypertrophy and accumulation of mesangial matrix (Kimmelstiel-Wilson nodules). This leads to declining filtering efficiency in the kidney. Microalbuminuria marks the early stages of diabetic nephropathy and as diabetic nephropathy progresses, the kidneys leak larger amounts of albumin (macroalbuminuria or proteinuria). DN is characterised by persistent albuminuria, relentless decline in GFR and raised arterial blood pressure (Parving, Osterby & Ritz 2000).

DN can occur in both type 1 and type 2 diabetes, and generally takes 15 to 25 years to develop after the onset of diabetes. If not detected and well managed, it can progress rapidly and may result in end-stage kidney disease. In Australia the rapid increase in type 2 diabetes is thought to have been a major contributor to the rising incidence of treated end-stage kidney disease (ESKD) in recent years.

### Detection and Protection from Progressing DN

Several longitudinal studies have shown that microalbuminuria has an 80% positive prediction rate of development of DN in type 1 diabetics (Parving, Osterby & Ritz 2000). In addition to microalbuminuria other risk factors or markers have been suggested for the development of DN (see Table 1). The major interventions to minimise or slow down the development of DN, apart from regular repeated screening for microalbuminuria, are, glycaemic control, blood pressure control, use of angiotension converting enzyme (ACE) inhibitors and minimisation of other risk factors. Also of importance for this patient group is the assessment and management of cardiovascular disease and other diabetic complications that may arise (retinopathy, peripheral

neurovascular disease, increased risk of infection (especially urinary tract infection) and autonomic neuropathy (bladder dysfunction and postural hypotension). Ongoing nursing assessment is vital in identifying any of these early markers.

**Table 1. Risk Factors/Markers for development of DN in Type 1 & Type 2 Diabetes Mellitus Patients.**

	Type 1	Type 2
Microalbuminuria	+	+
Sex	M > F	M > F
Chronic Hyperglycaemia	+	+
Advanced Glycation End Products	+	+
Arterial Hypertension	+/-	-
Smoking	+	?
Cholesterol	+	+
Retinopathy	+	+
Familial Clustering	+	+
(ref: adapted from Parving, Osterby & Ritz 2000).		

Strict metabolic control of blood sugar levels by insulin normalises hyperfiltration and the rate of increase in albumin excretion in experimental animal studies but GFR decline, proteinuria and hypertension increase were not affected by glycaemic control in patient studies. However, the literature still suggests optimising glycaemic control (HbA1c < 7%) (Harris, Joyner, Phillips & Webster 2004).

Many studies have demonstrated the beneficial effect of ACE inhibition in

protecting against the progression of DN, both the reduction in increasing albuminuria and the decline in GFR. Use of non ACE inhibitor antihypertensives provide some protection but ACE inhibitors have been shown to be superior to  $\beta$  - blockers and calcium channel blockers whilst Angiotensin 2 receptor blockers have shown similar success (Youngman 2004).

**Management of the DN Patient in End Stage Kidney Disease**

The diabetic patient receiving renal replacement therapy (RRT) has a higher mortality risk than non-diabetics. This is primarily due to the increased risk of cardiovascular disease. Almost 40% of diabetic patients starting RRT have evidence of coronary artery disease (Parving, Osterby & Ritz 2000). Early referral and commencement of RRT has been shown to be associated with a lower morbidity, cost and mortality on haemodialysis.

Vascular access for haemodialysis is often a problem for the diabetic problem. Early referral to a vascular surgeon with appropriate assessment may result in a pre-emptive functioning fistula, which avoids the need for temporary central catheters that multiply the problem of infection in the diabetic patient. Vessels are often less suitable for fistula construction and vascular complications such as ‘steal syndrome’ need to be continually monitored. Grafts or permanent catheters may need to be employed for patients with inadequate vessels or Peritoneal Dialysis may be an option (Youngman 2004).

Fluid removal can also be problematical for these patients. This can be related to autonomic neuropathy (lack of vasoconstriction and increased cardiac output in response to decreased blood volume) and hypoalbuminaemia from gross proteinuria or poor nutrition (decreased fluid shifts between body fluid compartments because of the reduced colloid osmotic pressure, affecting vascular refilling rate). Table 2 suggests

some techniques that can be employed to ensure patient safety and comfort on haemodialysis.

**Table 2. Techniques to maximise diabetic patient safety & comfort on HD**

- Blood Volume Monitoring.
- Sodium Profiling (need to assess patient suitability carefully).
- Sequential ultrafiltration.
- Priming with colloid.
- No eating on dialysis (prevents diversion of circulating blood to gut).
- Maintaining haemoglobin (prevention of LVH).
- Omitting or decreasing antihypertensives pre dialysis.
- Exercise on dialysis to aid venous return

(ref: from Youngman 2004)

Renal transplantation still offers the best medical rehabilitation for the patient with DN. However, studies show that patient and graft survival in diabetic patients are not as good as those in non-diabetic patients (Parving, Osterby & Ritz 2000).

**Conclusion**

The combination of diabetes and renal disease presents a challenge to the renal team. Prevention of complications, especially DN, is of great importance for the diabetic patient. Early screening for microalbuminuria and aggressive preventative intervention is essential to minimise the profound effects that diabetes can have on the individual. Special care is needed once the patient has developed DN and has progressed to ESKD requiring RRT. The nephrology nurse plays an important role in reinforcement of regimes and ongoing assessment.

**References**

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**Questions**

*Mr A, a 43 year old male with a history of IDDM and hypertension is admitted to your unit. He has been admitted for investigation of his renal function.*

1. Explain to Mr A what investigations he will be undergoing.
2. What nursing assessments would you undertake to determine if he has any CKD already?

*He doesn’t have a very good understanding of the effects of diabetes on his kidneys (and the rest of his body).*

3. What information would you give him about the effects of diabetes on his kidneys as well as other complications that may arise.
4. Discuss the information you would give him that would help him manage and prevent the progression of DN.

*Mr A’s uncle died of renal failure and he is concerned about what will happen if his kidneys “stopped working”.*

5. Discuss your response to this. What information would you give him?
6. What would his options be and how can we help to keep him safe on dialysis?