Microalbuminuria[1,2]

Diabetic kidney disease or nephropathy is the most common cause of end stage renal disease (ESRD) or kidney failure in the Western World. One of the earliest markers of diabetic nephropathy is the presence of small amount of the protein albumin in the urine. This is called MAU or microalbuminuria (urinary albumin excretion of 30-300 mg/24 hours). Microalbuminuria may progress over a span of a number of years to overt nephropathy characterized by the presence of larger amounts of the protein albumin leaking through the kidneys' filter mechanism into the urine. This is called macroalbuminuria (urinary albumin > 300 mg/24 hours). The presence of macroalbuminuria indicates more serious kidney disease. Progression to ESRD or kidney failure may then occur within several years. Once overt kidney failure has developed 2 year survival is approximately 50%.

Studies have shown that presence of microalbuminuria is reversible with interventions to tightly control blood sugar and blood pressure. Microalbuminuria may sometimes resolve on its own. Specific medications including angiotensin converting enzyme inhibitors (ACE inhibitors) and angiotensin receptor blockers (ARBs) have been shown to halt and reverse the presence of MAU and delay the progression to ESRD and the need for dialysis. In some cases a combination of both agents is indicated to protect the kidneys. This may be indicated when the creatinine clearance (a measure of kidney function) is $\leq$ 60 mL/min. Screening for MAU is recommended in all post-pubertal type 1 with diabetes $\geq$ 5 years duration and all type 2 diabetics on an annual basis.

MAU is measured on spot early morning urine collections, timed urine collections or as a ratio of albumin to creatinine the urine (ACR). The ACR is the preferred method as it does not require early morning or timed collections, it correlates with the 24-hour urine values over a large range of proteinuria, it is cheap to perform, and repeat values can be easily obtained to be certain ascertain that microalbuminuria, if present, is persistent. A patient is considered to have diabetic nephropathy if 2 of 3 measurements of ACR are elevated above 2.0 mg/mmol in men or 2.8 mg/mmol in women. A false reading for ACR may occur after vigorous exercise, in the presence of fever, urinary infection, congestive heart failure, acute severe elevations of blood pressure or blood sugar or menstruation.

Stages of kidney involvement according to the urinary albumin level			
Stage of nephropathy	Urine dipstick for protein	Urine ACR (mg/mmol)	24 hour urine collection for albumin
Normal	Negative	< 2.0 (men) < 2.8 (women)	< 30 mg/day
Microalbuminuria	Negative	2.0-20.0 (men) 2.8-28.0 (women)	30-300 mg/day
Overt nephropathy (Macroalbuminuria)	Positive	> 20.0 (men) > 28.0 (women)	> 300 mg/day

MAU is associated not only with increase risk of kidney disease but of cardiovascular disease in patients with diabetes and hypertension. Type 2 MAU diabetics with MAU have a 50% change of having a coronary event with in 7 years. Microalbuminuria reflects vascular damage and appears to be a marker of early arterial disease and endothelial dysfunction. In one recent study the presence of microalbuminuria indicated a risk of developing coronary heart disease (CHD) of 1.36 in patients without known CHD and a risk of all cause mortality of 1.61 in patients with known CHD. Screening for microalbuminuria is not yet routinely recommended in non-diabetic patients with or without hypertension.

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1 Canadian Diabetes Association Clinical Practice Guidelines Expert Committee. Canadian Diabetes Association 2003 Guidelines for the Prevention and Management of Diabetes in Canada. *Can J Diabetes* 2003; 27(suppl 2):S66-71.

2 Rose BD, Bakris GL. Microalbuminuria and Cardiovascular disease In: *UpToDate*, Rose, BD (Ed), *UpToDate*, Wellesley, MA, 2004.

3 Yuyan MF, Khaw KT, Welch A, Bingham S, Day NE, Wareham NJ. A Prospective Study of Microalbuminuria and Incident CHD and its Prognostic significance in a British Population: The EPIC-Norfolk Study. Am J Epidemiol. 2004 Feb 1; 159(3):284-93.