SUYASH Uro Times

A Quarterly News Letter From Suyash Nursing Home

Oct. 2015 - 9th Issue

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1st issue Oct 2013 : Lasers in urology - Dr Sharad Somani

2nd issue Jan 2014 : Managing BPH: Part 1 - Dr Sharad Somani
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Inside This Issue

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Previous Issues of Suyash Uro Times are available at www.suyashurology.com

Preface

Dear Colleagues,

Greetings.

We all know that it is very important for each one of us to remain updated in our area of specialization. However it is also true that it is very difficult to find time to abreast ourselves because of hectic schedule of work and busy schedule of our practice. Hence I feel publication of scientific news letter in urology by the name 'Suyash Uro Times' is an appreciable attempt by Dr Sharad Somani, as it is a quick and easy way to update yourself about the latest developments in urology.

The previous issues of 'Suyash Uro Times' and its subject contents, along with invitation to guest authors who are subject experts, authenticate sincerity, integrity and commitment for its academic thirst. The continuous endeavor from Dr Sharad Somani and uro times team to maintain the Gold standard of this news letter is admirable. It is said 'Well begun is half done' hopefully this beginning may prove tomorrow to be the inception of a new journal in urology from our region. Lastly, while wishing you all the best, I also convey my word of thanks to Dr Somani for inviting me to write the preface of this issue.

Yours

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From Editors Desk

Dear colleagues,

Warm greetings from team "Suyash Uro Times"

It is my immense pleasure to publish 9th issue of the newsletter.

We started this activity as a continuous medical education. I am happy to inform you that we are getting overwhelming response from the doctors all over. General practitioners, specialists & super specialists have communicated personally and appreciated the activity.

This issue highlights most common medical illnesses leading to chronic kidney disease. The articles are written by the specialists and will be definitely of benefit for the practitioners to follow the guidelines.

Please feel free to write to us on suyashnursinghome@gmail.com regarding suggestions, advice or criticism so as to make us improve on the scientific stuff.

Looking forward to communicate with you time to time through this newsletter.

Dr. Sharad Somani

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DIABETIC NEPHROPATHY

Dr. Kaustubh Sodani

Diabetic nephropathy is a clinical syndrome

Epidemiology

In the industrialized world, diabetes mellitus is the single leading cause of end-stage renal disease (ESRD). Both the incidence and prevalence of ESRD secondary to diabetes continue to rise. more than 30% of patients undergoing either dialysis therapy or renal transplantation have ESRD as a result of diabetic nephropathy, and 40% of the new (incident) cases of ESRD are attributable to diabetes.

Definition

Diabetic nephropathy is a clinical syndrome characterized by:

- Persistent albuminuria (>300 mg/d or >200 g/min) that is confirmed on at least 2 occasions 3-6 months apart
- Progressive decline in the glomerular filtration rate (GFR)
- Elevated arterial blood pressure

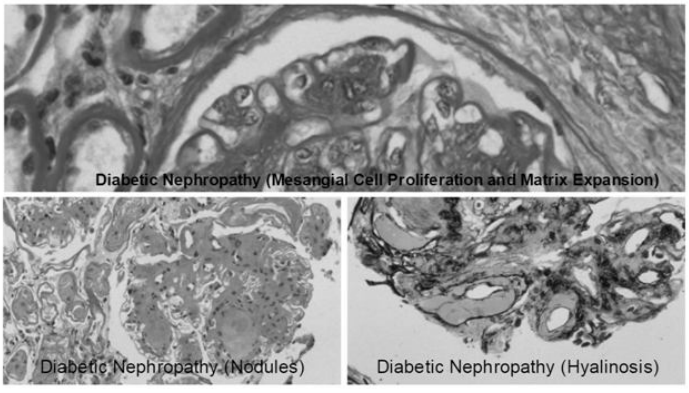
Risk factors include the following:

- Duration and degree of hyperglycemia
- Hypertension
- Dyslipidemia
- Cigarette smoking
- Certain polymorphisms affecting the renin-angiotensin-aldosterone axis
- Family history of diabetic nephropathy
- Genetic variables (decreased number of glomeruli)

Renal failure usually takes ≥ 10 yr after the onset of nephropathy to develop; however, because type 2 diabetes is often present for several years before being recognized, nephropathy often develops < 10 yr after diabetes is diagnosed.

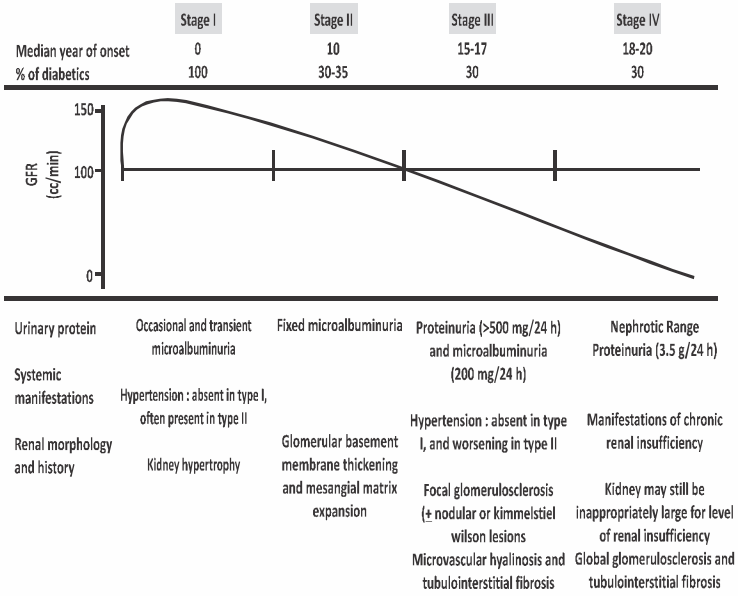
Pathophysiology

Pathogenesis begins with small vessel disease. Pathophysiology is complex, involving glycosylation of proteins, hormonally influenced cytokine release (eg, transforming growth factor-), deposition of mesangial matrix, and alteration of glomerular hemodynamics. Hyperfiltration, an early functional abnormality, is only a relative predictor for the development of renal failure.



Hyperglycemia causes glycosylation of glomerular proteins, which may be responsible for mesangial cell proliferation and matrix expansion and vascular endothelial damage. The glomerular basement membrane classically becomes thickened.

Lesions of diffuse or nodular intercapillary glomerulosclerosis are distinctive; areas of nodular glomerulosclerosis may be referred to as Kimmelstiel-Wilson lesions . There is marked hyalinosis of afferent and efferent arterioles as well as arteriosclerosis; interstitial fibrosis and tubular atrophy may be present. Only mesangial matrix expansion appears to correlate with progression to end-stage renal disease.



DN begins as glomerular hyperfiltration (increased GFR); GFR normalizes with early renal injury and mild hypertension, which worsens over time. Microalbuminuria, urinary excretion of albumin in a range of 30 to 300 mg albumin/day, then occurs. Urinary albumin in these concentrations is called microalbuminuria because detection of proteinuria by dipstick on routine urinalysis usually requires > 300 mg albumin/day. Microalbuminuria progresses to macroalbuminuria (proteinuria > 300 mg/day at a variable course, usually over years. Nephrotic syndrome (proteinuria ≥ 3 g/day) precedes end-stage renal disease, on average, by about 3 to 5 yr, but this timing is also highly variable. Other urinary tract abnormalities commonly occurring with DN that may accelerate the decline of renal function include papillary necrosis, type IV renal tubular acidosis, and UTIs. In DN, the kidneys are usually of normal size or larger.

Symptoms and Signs

DN is asymptomatic in early stages. Sustained microalbuminuria is the earliest warning sign. Hypertension and some measure of dependent edema eventually develop in most untreated patients. In later stages, patients may develop symptoms and signs of uremia (eg, nausea, vomiting, anorexia) earlier (ie, with higher GFR) than do patients without DN, possibly because the combination of end-organ damage due to diabetes (eg,

neuropathy) and renal failure worsens symptoms.

Diagnosis

- Yearly screening of all patients with diabetes with random urine albumin/creatinine ratio
- Urinalysis for signs of other renal disorders (eg, hematuria, RBC casts)

The diagnosis is suspected in patients with diabetes who have proteinuria, particularly if they have diabetic retinopathy (indicating small vessel disease) or risk factors for DN. Other renal disorders should be considered if there are any of the following:

- Heavy proteinuria with only a brief history of diabetes
- Absence of diabetic retinopathy
- Rapid onset of heavy proteinuria
- Gross hematuria
- RBC casts
- Rapid decline in GFR
- Small kidney size

Urinary protein

Patients are tested for proteinuria by routine urinalysis; if proteinuria is present, testing for microalbuminuria is unnecessary because the patient already has macroalbuminuria suggestive of diabetic renal disease. In patients without proteinuria on urinalysis, an albumin/creatinine ratio should be calculated from a mid-morning urine specimen. A ratio 0.03 mg/mg (30 mg/g) indicates microalbuminuria if it is present on at least 2 of 3 specimens within 3 to 6 months and if it cannot be explained by infection or exercise. Some experts recommend that microalbuminuria be measured from a 24-h urine collection, but this approach is less convenient, and many patients have difficulty accurately collecting a specimen. The random urine albumin/creatinine ratio overestimates 24-h collection of microalbuminuria in up to 30% of patients > 65 due to reduced creatinine production from reduced muscle mass. Inaccurate results can also occur in very muscular patients or if vigorous exercise precedes urine collection.

For most patients with diabetes who have proteinuria, the diagnosis is clinical. Renal biopsy can confirm the diagnosis but is rarely necessary.

Screening

Patients with type 1 diabetes without known renal disease should be screened for proteinuria and, if proteinuria is absent on routine urinalysis, for microalbuminuria, beginning 5 yr after diagnosis and at least annually thereafter.

Patients with type 2 diabetes should be screened at the time of diagnosis and annually thereafter.

Prognosis

Prognosis is good for patients who are meticulously treated and monitored. Such care is often difficult in practice, however, and most patients slowly lose renal function; even prehypertension (BP 120 to 139/80 to 89 mm Hg) or stage 1 hypertension (BP 140 to 159/90 to 99 mm Hg) may accelerate injury. Systemic atherosclerotic disease (stroke, MI, peripheral arterial disease) predicts an increase in mortality.

Treatment

- Maintenance of glycosylated Hb (HbA 1c) 7.0
- Aggressive BP control, beginning with angiotensin inhibition

Primary treatment is strict glucose control to maintain HbA 1c ≤ 7.0; maintenance of euglycemia reduces microalbuminuria but may not retard disease progression once DN is well established. Glucose control must also be accompanied by strict control of BP to < 130/80 mm Hg. Some experts suggest BP should be 110 to 120/65 to 80 mm Hg, particularly in patients with protein excretion of > 1 g/day; however, others claim that BP values < 120/85 mm Hg are associated with increased cardiovascular mortality and heart failure. Dyslipidemia should also be treated.

Angiotensin inhibition is first-line therapy. Thus, ACE inhibitors or angiotensin II receptor blockers are the antihypertensives of choice; they reduce BP and proteinuria and slow the progression of DN. ACE inhibitors are usually less expensive, but angiotensin II receptor blockers can be used instead if ACE inhibitors cause persistent cough. Treatment should be started when microalbuminuria is detected regardless of whether hypertension is present; some experts recommend drugs be used even before signs of renal disease appear.

Diuretics are required by most patients in addition to angiotensin inhibition to reach target BP levels. Dose should be decreased if symptoms of orthostatic hypotension develop or serum creatinine increases by more than 30%.

Nondihydropyridine Ca channel blockers (diltiazem and verapamil) are also antiproteinuric and renoprotective and can be used if proteinuria does not meaningfully decrease when target BP is reached or as alternatives for patients with hyperkalemia or other contraindications to ACE inhibitors or angiotensin II receptor blockers. In contrast, dihydropyridine Ca channel blockers (eg, nifedipine, felodipine, amlodipine) do not reduce proteinuria, although they are useful adjuncts for BP control and may be cardioprotective in combination with ACE inhibitors. ACE inhibitors and nondihydropyridine Ca channel blockers have greater antiproteinuric and renoprotective effects when used together, and their antiproteinuric effect is enhanced by Na restriction. Nondihydropyridine Ca channel blockers should be used with caution in patients taking ?-blockers because of the potential to worsen bradycardia.

Statins should be used as first-line therapy for hyperlipidemia treatment in patients with DN because they reduce cardiovascular mortality and urinary protein.

Dietary protein restriction yields mixed results. The American Diabetic Association recommends that people with diabetes and overt nephropathy be restricted to 0.8 to 1.2 g protein/kg/day. Significant protein restriction is not recommended.

Kidney transplantation with or without simultaneous or subsequent pancreas transplantation is an option for patients with end-stage renal disease. The 5-yr survival rate for patients with type 2 diabetes receiving a kidney transplant is almost 60%, compared

with 2% for dialysis-dependent patients who do not undergo transplantation. Renal allograft survival rate is > 85% at 2 yr.

Stage wise Management of Diabetic Nephropathy

Stage I	Tight glucose control; BP control—consider use of ACEI or ARB
Stage II	Tight glucose control; ACEI or ARB; BP control; Smoking cessation; Weight reduction; Exercise; Annual eye examination;
Stage III	ACEI or ARB; BP control; Restriction of dietary protein (to 0.8g/kg of ideal body weight/day); Antihyperlipidemic medications
Stage IV	Treat manifestations of nephrotic syndrome and chronic renal insufficiency; Prepare for renal replacement therapy, including prevention of abnormalities in calcium/phosphorus metabolism and prevention of anemia by early use of erythropoietin

- Diabetic nephropathy is the most common cause of end-stage renal disease. The incidence of diabetic has increased substantially over the past few years. Advanced diabetic nephropathy is also the leading cause of glomerulosclerosis and end-stage renal disease worldwide.
- Diabetic nephropathy is typically defined by macroalbuminuria or macroalbuminuria and abnormal renal function as represented by an abnormality in serum creatinine, calculated creatinine clearance, or glomerular filtration rate.
- Progressive renal disease in diabetes is a multifactorial process that begins initially via a process of glomerular hyperfiltration and increased glomerular filtration rate
- Symptoms, which may be absent until the disease is advanced, include fatigue, anorexia, and swelling of the extremities. Symptoms of retinopathy (impaired vision) and neuropathy (decreased or abnormal sensation in lower extremities) are common.
- Signs include hypertension, oedema, and findings of diabetic retinopathy and neuropathy. In clinical uraemia, nausea and vomiting, dysgeusia (altered taste), and hiccoughs supervene.
- Proteinuria is the characteristic laboratory finding. Azotaemia may develop as the disease advances.
- Microalbuminuria is now recognized as an independent cardiac risk factor, even in the absence of diabetes.
- Findings of diabetic nephropathy on kidney biopsy include mesangial expansion, glomerular basement membrane thickening, glomerulosclerosis, or a combination of these.
- Treatment includes intensive control of hyperglycaemia and hypertension with ACE inhibitors, angiotensin-receptor blockers (ARBs), or other antihypertensives. Lipid reduction, low-protein diets, and smoking cessation may be beneficial.
- Complications include hypoglycaemia due to intensive treatment of hyperglycaemia, hyperkalaemia as an adverse effect of ACE inhibitors or ARBs, volume depletion due to diuresis, and inadequate protein/caloric intake leading to malnutrition. Some patients may reach end-stage renal failure, requiring dialysis.

HYPERTENSION AND KIDNEY DISEASE

Dr Nahush Patel

Hypertension is the commonest cardiovascular disease and along with diabetes, it accounts for majority of cardiovascular deaths.

It is a major risk factor for CV mortality, CHD, CVA, CHF, and RF

The relationship between BP and risk of CVD events is continuous, consistent, and independent of other risk factors. The higher the BP, the greater the chance of heart attack, HF, stroke, and kidney diseases.

CV risk associated with HT is strongly correlated with both SBP and DBP, correlation is higher with SBP. DHT predominates before age 50, either alone or in combination with SBP elevation. The prevalence of SHT increases with age and above 50 SHT represents the most common form of HT. DBP is a more potent CV RF than SBP until the age 50, thereafter SBP is more important.

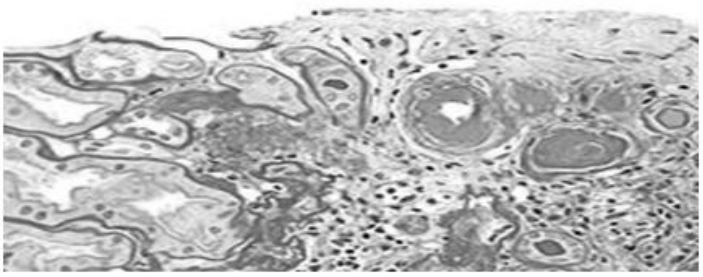
Hypertension and kidney disease are related in both ways. Long standing HT leads to hypertensive nephropathy (Hypertensive Nephrosclerosis)and intrinsic kidney diseases like glomerulonephritis, renal artery stenosis etc can cause HT.

Hypertensive Nephrosclerosis :
2 Types o I. Benign Nephrosclerosis o II. Malignant Nephrosclerosis

1. BENIGN NEPHROSCLEROSIS o Benign nephrosclerosis is the term used for the renal pathology associated with sclerosis of renal arterioles and small arteries. o The resultant effect is focal ischemia of parenchyma supplied by these vessels.The parenchymal effects include glomerulosclerosis and chronic tubulointersititial injury, producing a reduction in functional renal mass.

Pathogenesis : Two processes participate in the arterial lesions: I. Medial and intimal thickening, as a response to hemodynamic changes, aging, genetic defects, or some combination of these.

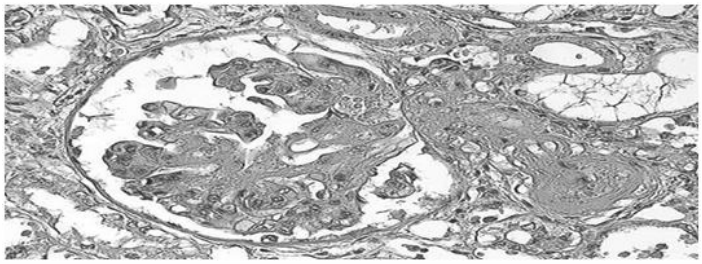
II. Hyaline deposition in arterioles, caused partly by extravasation of plasma proteins through injured endothelium and partly by increased deposition of basement membrane matrix.



MICROSCOPY: There is narrowing of the lumens of arterioles and small arteries, caused by thickening and hyalinization of the walls (hyaline arteriolosclerosis)

Corresponding to the fine surface granulations are microscopic subcapsular scars with sclerotic glomeruli and tubular dropout (Foci of tubular atrophy) with interstitial fibrosis, alternating with better preserved parenchyma

Glomerular alterations include: 1.Collapse Of The GBM, 2. Deposition Of Collagen Within The Bowman Space, 3. Periglomerular Fibrosis, and 4. Total Sclerosis Of Glomeruli



Clinical Features : It is unusual for uncomplicated benign nephrosclerosis to cause renal insufficiency or uremia. There is usually moderate reduction in renal blood flow, but the GFR is normal or only slightly reduced.

2. Malignant Nephrosclerosis : Malignant nephrosclerosis is the form of Renal Disease associated with the malignant or accelerated Phase of hypertension. Malignant hypertension may occasionally develop in previously normotensive individuals but often is superimposed on Preexisting essential benign hypertension, Secondary forms of hypertension, or an underlying chronic renal disease, particularly glomerulonephritis or reflux nephropathy associated with vesico-ureteric reflux. Malignant hypertension is relatively uncommon, occurring in 1% to 5% of all hypertensive patients.It is more often in men and in blacks.

Pathogenesis- The initial insult seems to be some form of vascular damage to the kidneys resulting in 1. increased permeability of the small vessels to fibrinogen and other plasma proteins, 2. endothelial injury, 3. focal death of cells of the vascular wall, and 4. platelet deposition. This leads to the appearance of 1. Fibrinoid necrosis of arterioles and small arteries, 2. Swelling of the vascular intima, and 3. Intravascular Thrombosis. With severe involvement of the renal afferent arterioles, the renin-angiotensin system receives a powerful stimulus; indeed, patients with malignant hypertension have markedly elevated levels of plasma renin. This sets up a self-perpetuating cycle in which angiotensin II causes intrarenal vasoconstriction, and the attendant renal ischemia perpetuates renin secretion.

Clinical Features: ● SP>200 mm Hg & DP>120 mm Hg, ● Papilledema, ● Retinal hemorrhages, ● Encephalopathy, ● Cardiovascular abnormalities, & ● Renal failure. Most often, the early symptoms are related to increased intracranial pressure and include ● headaches, ● Nausea, ● Vomiting, and ● visual impairments, particularly scotomas or spots before the eyes and episodes of loss of consciousness or even convulsions.

At the onset of rapidly mounting blood pressure, there is marked proteinuria and microscopic or sometimes macroscopic hematuria but no significant alteration in renal function. ● Soon, however, renal failure makes its appearance.

Treatment : The syndrome is a true medical emergency requiring the institution of aggressive and prompt antihypertensive therapy to prevent the development of irreversible renal lesions.

Prognosis : Before the introduction of current antihypertensive drugs, malignant hypertension was associated with a 50% mortality rate within 3 months of onset, progressing to 90%within a year. At present, however, about 75% of patients survive 5 years, and 50% survive with restoration of pre-crisis renal function.

Management of Hypertension :

JNC 8 has given guidelines for management of hypertension. There is strong evidence to support treating hypertensive persons aged 60 years or older to a BP goal of less than 150/90mmHg and hypertensive persons 30 through 59 years of age to a diastolic goal of less than 90mmHg; however, there is insufficient evidence in hypertensive persons younger than 60 years for a systolic goal, or in those younger than 30 years for a diastolic goal, so the panel recommends a BP of less than 140/90mmHg for those groups based on expert opinion. The same thresholds and goals are recommended for hypertensive adults with diabetes or non diabetic chronic kidney disease (CKD) as for the general hypertensive population younger than 60 years. There is moderate evidence to support initiating drug treatment with an angiotensin-converting enzyme inhibitor, angiotensin receptor blocker, calcium channel blocker, or thiazide-type diuretic in the nonblack hypertensive population, including those with diabetes. In the black hypertensive population, including those with diabetes, a calcium channel blocker or thiazide-type diuretic is recommended as initial therapy. ***There is moderate evidence to support initial or add-on antihypertensive therapy with an angiotensin-converting enzyme inhibitor or angiotensin receptor blocker in persons with CKD to improve kidney outcomes.***

JNC 8 Recommendations

Patient Subgroup	Target SBP (mm Hg)	Target DBP (mm Hg)
≥ 60 years	<150	< 90
< 60 years	<140	< 90
> 18 years with CKD	<140	<90
> 18 years with diabetes	<140	<90

CKD = chronic kidney disease; DBP = diastolic blood pressure; SBP = systolic blood pressure

James PA, et al. *JAMA*. 2013 Dec 18. [Epub ahead of print]

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JNC 8 Recommendations (continued)

- General nonblack population
  - Thiazides, CCB, ACEI, or ARB initially
- General black population
  - Thiazides or CCB initially
- CKD
  - Treatment should include ACEI or ARB
- Up-titrate or add therapy after 1 mo if BP goal not achieved
  - Don't use ACEI and ARB together
  - If > 3 drugs needed, refer to hypertension specialist

James PA, et al. *JAMA*. 2013 Dec 18. [Epub ahead of print]

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Presentation at West zone USICON at Goa in September 2015

Laparoscopic pyeloplasty: A nursing home experience

Dr Sharad Somani & Dr Nagesh Nagapurkar
Suyash Nursing Home
Aurangabad

Laparoscopic Boari flap repair: A nursing home experience

Dr Sharad Somani & Dr Nagesh Nagapurkar
Suyash Nursing Home
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