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CONTENTS

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(A Journal of continuing education in kidney diseases)

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Bangladesh Renal Journal is the official organ of the Bangladesh Renal Association. The Journal publishes two issue in a year i.e. June and December. The Journal is devoted to continuing education in kidney diseases.

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INSTRUCTION FOR AUTHORS

Renal Association Journal appears twice in a year and it publishes original articles, review articles, clinical communications, recent advances in renal diseases and letters to the editors. The editors reserve the right to select from submitted manuscripts and the right of stylistic changes or abridgements. The manuscripts may not be offered elsewhere for printing and publication; following acceptance, the publisher acquires all copywright.

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Only scientific papers written in English will be accepted. Restriction to the essential is recommended. Original papers should normally not exceed 16 type- written pages including tables, illustration and references. The arrangement of the paper should include summary, introduction materials and methods, results and discussion. Each section being clearly marked. The manuscripts must be type-written on a white paper, on one side of the sheet only and double spaced on consecutively numbered pages. Figures and illustrations, tables. captions, references, summary (15-20) lines and acknowledgement are to be submitted on separate paper. The caption should be brief and should not represent a duplication of information provided in the text.

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Grindley MF: Manual of histologic and special staining Nephrologic, Elammarion, Paris, 1965.

ABBREVIATIONS

Angstrom	A
body surface area	BSA
body weight	body wt.
centimeter	cm
celius	C
complement components	C1,C2,C3
Correlation coefficient	r
creatinine clearance	Ccr.
curie (s)	Ci
Equivalents	Eq
Fahrenheit	F

Glomerular fitration rate	GFR
gram (s)	g
Grams per cent	g/100mi
half-time	tf1/2
hour (s)	hr
inch	inch
International Unit (s)	IU
Intramuscular	im.
intraperioneal	i.p.
intravenous	i.v.
inulin clearance	Cln
Kilogram (s)	Kg
liter (s)	L
meter (s) or milli	m
microns (s) or micro	µ
milligram (s) per cent	mg/100ml
minute (s)	min
molar	M
mole (s)	mole (s)
Molecular weight	molwt
nanogram (s) (millimicrogram)	ng
nanoliter (s) (millimicroliter)	nl
normal (concentration)	N
not significant	NS
optical density	OD
osmole (s)	Osm
probability	P
second (s)	sec
standard deviation	SD
standard error	SE
standard error of the mean	SEM
ultraviolet	UV
unit (s)	U
volt	V

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Original Articles

Common Risk Factors for Diabetic Nephropathy among Patients with Diabetes Mellitus- An Experience in a Tertiary Level Hospital of Bangladesh

M Abul Kashem Khondaker[1], Manash Saha[2]

Summary:

Diabetic Nephropathy is a specific microvascular complication of Diabetes Mellitus. The level of albuminuria is highly correlated with the intraglomerular pressure which is the key determinant of Diabetic Nephropathy. This study was carried out to see the prevalence of common risk factors of Diabetic Nephropathy and to determine association between risk factors and Diabetic Nephropathy among Diabetic patients. Diabetic patients were selected from a tertiary level hospital of Bangladesh. We identified nephropathy among them by doing albumin creatinine ratio on spot urine and carried out clinical evaluation including blood pressure, body mass index and retinopathy. Finally we investigated the study population to find out metabolic risk factors like HbA1C, Triglyceride, LDL cholesterol, and 2 hr post prandial blood glucose. Assessment for proteinuria was also done in 2nd and 3rd visit during 6 months study period. Among study population 27.3% had microalbuminuria, 13.3% had macroalbuminuria and rest of them had normoalbuminuria. Hypertension, overweight& obesity, poor glycemic control, duration of diabetes, high LDL cholesterol and high blood level of triglyceride were observed to be significantly higher in nephropathy group than those in the non-nephropathy group. The result of this study suggests that among diabetic patients of a developing country, prevalence of diabetic nephropathy is high (40.6%).Several risk factors are significantly associated with albuminuria. Multidisciplinary actions should be taken to reduce the burden of the disease.

Key words: ***Albuminuria, Diabetes mellitus, Hypertension, LDL cholesterol, Intraglomerular pressure***

(Bang. Renal J. 2010; 29(2):30-36)

Introduction:

Diabetes Mellitus is one of the most alarming public heath problems of the 21st century. Bangladesh had 3.2 million people with diabetes in 2000 and by 2030 it will be 11.1 million, which will occupy the 7th position among the ten countries estimated to have the highest number of diabetic people in 2030[1,2]. About one third of those affected, will eventually have progressive deterioration of renal function[3].

About 30% of patients with type1diabetes have developed diabetic nephropathy after 20 years but the risk after this time falls to less than 1% per year, and from the outset the risk is not equal in all patients[4]. Diabetic nephropathy occurs in 20-40% of patients with diabetes and is the single leading cause of end stage renal disease.[5] It is estimated that about 20% of the type 2 diabetic patients reach End Stage Renal Disease (ESRD) during the life time.[6] Studies conducted in migrant Asian Indians in Europe have reported increased prevalence of diabetic nephropathy compared with white Caucasians.[7, 8, 9] The prevalence of diabetic kidney disease among the Asians is one of the highest in the world. Based on a survey involving 6000 people, 39% of people with diabetes were found to have microalbuminuria and another 19% had overt proteinuria[10]. The pathological changes in the kidney, clinical stages and risk factors to develop nephropathy are similar in both types of diabetes.[11,12,13] The pathogenesis of diabetic nephropathy largely depends on hyperglycemia resulting in non enzymatic glycation and the accumulation of advanced glycation end products (AGEs).

Diabetic nephropathy passes through 5 stages. 1st and 2nd stages are clinically silent. In 3rd stage, known as

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incipient nephropathy, microalbuminuria stage, blood pressure tends to rise. At stage 4, overt nephropathy, macroalbuminuria is present, blood pressure almost in variably increases and GFR progressively declines. In stage 5, renal failure is present. A variety of factors are known to play significant role in the development of diabetic nephropathy. Among them hypertension is most important one. Beside it, several studies suggested that poor glycemic control, dyslipidaemia and duration of diabetes had detrimental effect on the progression of diabetic nephropathy.

The prevalence of microalbuminuria, overt proteinuria or diabetic nephropathy and its association with risk factors have not been studied adequately in middle and lower class population. For this reason, we tried to determine prevalence of proteinuria among diabetic patients and to find out the risk factors of diabetic nephropathy such as hypertension, dyslipidaemia, obesity, duration of diabetes, anemia and poor control of diabetes among them.

Materials and methods:

This is a hospital based observational study of six months duration. Admitted diabetic patients of the department of Internal Medicine of Dhaka Medical College Hospital, Dhaka without recent history of fever, heart failure, urinary tract infection, prostatitis, pregnancy and evidence of other renal diseases were advised to attend Outpatient Medicine Department on the day of discharge for study purpose. 135 known diabetic patients were enrolled for the study during 1st phase (7 days). After taking consent, history & examination were carried out. Particulars of the patient including age, sex, residence, religion and relevant medical history including diabetes mellitus, hypertension, Diabetic nephropathy, Family history of hypertension and DM, other long-term complications of DM and drug history were compiled. Findings of Physical examination were recorded in a data sheet. Urinary albumin creatinine ratio and dipstick test for urinary protein were done from fresh morning urinary sample. Other laboratory investigations including lipid profile, HbA1C, 2 hr postprandial Blood glucose, S. Creatinine were also carried out. All patients were requested to attend the investigators on both 3rd (2nd phase) month and 6th month (3rd Phase) of the study period. During 2nd visit (2nd phase-7 days), proteinuria was assessed by doing urinary albumin creatinine ratio and dipstick test. During 3rd visit (3rd Phase-7days) physical examination was carried out. Then proteinuria was assessed and other relevant investigations like S. lipid profile, HbA1C, 2 hr postprandial blood glucose, S. Creatinine were done. 7 patients did not attend both 2nd and 3rd visit. So, total study population was 128.The investigators did not interfere on going treatment of diabetic patients given by outdoor medical officers. The patients who had albuminuria, two in three occasions were considered to have Diabetic Nephropathy. Presence of risk factors on both occasions (1st and 3rd phase) was evaluated for the association of Diabetic Nephropathy.

Operational Definitions:

Known case of diabetes was identified having prior diagnosis of diabetes by a physician or taking anti diabetic agents either oral hypoglycemic agents or insulin.

Hypertension:

Known hypertensive and individual who had systolic blood pressure > 140 mmHg and/or diastolic blood pressure > 90 mmHg were considered to have hypertension.

Known hypertensive will be defined if the patient was on antihypertensive medication at the time of evaluation.

Diabetic nephropathy: The appearance of albuminuria at least in two samples in an individual without evidence of other renal disease during study period.

Normoalbuminuria: Spot urine albumin creatinine ratio was less than 30 microgram /mg and dipstick test for protein was negative.[5]

Microalbuminuria: Spot urine albumin creatinine ratio was 30-299 microgram /mg and dipstick test for protein was negative. [5]

Macroalbuminuria: Spot urine albumin creatinine ratio was > 300 microgram /mg and dipstick test for protein was positive. [5]

Known cases of Diabetic nephropathy was based on -

1) Having prior diagnosis of Diabetic Nephropathy by a physician and/or
2) Taking treatment for Diabetic Nephropathy

Poor Glycemic control:

HbA1C >7% (Long term control-Several months), 2hour post prandial blood glucose >10 mmol/l (short term control).[5]

Dyslipidaemia:

Patient having any of underling values was considering high risk patient and dyslipidaemia. These were LDL >100gm/dl, triglyceride >150mg/dl HDL <40mg/dl.[5]

Statistical analysis:

Data were processed and analyzed with the help of computer software SPSS (Statistical Package for Social Sciences), version 11.5. The test statistics used to analyze the data were descriptive statistics, Chi-square (χ^2), ANOVA and Odds Ratio with 95% confidence interval of Odds Ratio. The data presented on categorical scale were expressed as frequency and corresponding percentages and were compared between groups using Chi-square (χ^2) Probability Test, while the data presented on continuous scale were compared among groups using ANOVA statistics. The risk factors that were found to be associated with nephropathy in univariate analysis were further subjected to multivariate binary logistic regression model to find the independent predictors of nephropathy.

Result:

About 60% of the study population had nomoalbuminuria, 27.3% had microalbuminuria and 13.3% had macroalbuminuria. Staging of chronic kidney disease (CKD) using Cockroft-Gault formula showed that 18.8% in stage – I, 14.1% in stage – II, 7.8% in stage – III, 0.8% in stage – IV disease and 58.6% of the patients were free from CKD.

A number of suspected risk factors were evaluated in this study. 41.4% of the study population was hypertensive, 36.7% had family history of diabetes, 12.5% were overweight & obese, 18.8% had family history of hypertension and 7% had family history of diabetic nephropathy. (Table-I)

Table I

Distribution of patients by presence of clinical risk factors (n = 128)

Presence of risk factors	Frequency	Percentage
Hypertension	53	41.4
Family history of DM	47	36.7
Family history of HTN	24	18.8
Overweight & obesity	16	12.5
Family history of DN	09	7.0

Demographic characteristics was compared between subjects of nephropathy and non-nephropathy groups showed that elderly subjects (> 60 years) developed nephropathy (36.5%) significantly more than the subjects with age 60 or below (18.4%) (p = 0.021). However, sex was not found to be associated with development of nephropathy (p = 0.604).

Hypertension, overweight & obesity, family history of diabetes mellitus and family history of hypertension were observed to be significantly higher in the nephropathy group than those in the non-nephropathy group($p < 0.001$, $p = 0.014$, $p = 0.027$, $p = 0.001$ respectively). Family history of diabetic neuropathy was, however, not found to be associated with nephropathy (p = 0.536). (Table-II)

Table II

Factors associated with the Diabetic Nephropathy (n=128)

Factors	Renal status		P value
	Nephropathy (n = 52)	Non-nephropathy (n = 76)	
Hypertension	36(69.2)	17(22.4)	0.001
Overweight & obesity	11(21.2)	5(6.6)	0.014
Family history of DM	27(51.9)	54(71.1)	0.027
Family history of hypertension	17(32.7)	7(9.2)	0.001
Family history of DN	4(7.7)	5(6.9)	0.536

Figures in the parentheses denote corresponding percentage.
Chi-square (χ^2) Test was employed to analyse the data.

Three lipids namely Triglyceride (TG), LDL Cholesterol and HDL Cholesterol were studied between the subjects with and with out nephropathy. Nearly 60% of the nephropathy group had TG > 150 mg/dl as opposed to 25% of the non-nephropathy group ($p < 0.001$). The presence of LDL Cholesterol more than 100 mg/dl was also observed to be significantly higher among the former group (80.8%) than that in the latter group (35.5%). The proportion of subjects with HDL Cholesterol less than 40 mg/dl was also much higher in the former group (53.8%) than that in the latter group (28.9%) (p = 0.005). (Table-III) Data showed that uncontrolled diabetes tend to be associated with the development of nephropathy significantly more than their controlled counterpart ($p < 0.001$)

Table III

Association between Dyslipidemia and Diabetic Nephropathy (n = 128)

Dyslipidemia	Renal status		P value
	Nephropathy (n = 52)	Non-nephropathy (n = 76)	
TG > 150 mg/dl	31(59.6)	19(25.0)	0.001
LDL > 100 mg/dl	42(80.8)	27(35.5)	0.001
HDL < 40 mg/dl	28(53.8)	22(28.9)	0.005

Figures in the parentheses denote corresponding percentage.
Chi-square (χ^2) Test was employed to analyse the data.

Hypertension was significantly higher in patients with microalbuminuria than that in patients without microalbuminuria and was even higher in patients with overt albuminuria ($p < 0.001$). The mean duration of diabetes was the lowest in patients with ACR < 30 (2.8 ± 0.3 years) and highest in patients with ACR e" 300 (8.3 ± 1.5 years) ($p < 0.001$). The prevalence of hypertriglyceridemia and hypercholesterolemia were also found to increase with the increase urine ACR ($p < 0.001$). (Table-IV)

Table IV

Association of risk factors with different stages of Nephropathy

RiskFactors	Urine ACR			
	< 30 (n = 76)	30 – 300 (n = 35)	> 300 (n = 17)	p value
Age > 60#	14(18.4)	16(45.7)	3(17.6)	0.007
Hypertension#	17(22.4)	23(65.7)	13(76.5)	0.001
HbA_1C χ^3 7%#	31(40.8)	22(62.9)	17(100.0)	0.001
Duration of DM¶ (yrs)	2.8 ± 0.3	7.9 ± 2.1	8.3 ± 1.5	0.001
TG > 150# mg/dl	19(25.0)	18(51.4)	13(76.5)	0.001
LDL > 100# mg/dl	27(35.5)	27(77.1)	15(88.2)	0.001
Retinopathy#	2(2.6)	9(25.7)	5(29.4)	0.001

Figures in the parentheses denote corresponding percentage.
χ^2 Test was employed to analyze the data; ¶ data were analyzed using ANOVA statistics and were presented as mean ± SEM.

Diabetic retinopathy and diabetic neuropathy was observed to be staggeringly higher in patients with nephropathy than those in patients without nephropathy (26.9% vs. 2.6%, $p < 0.001$ and 23.1% vs. 1.3%, < 0.001 respectively). IHD and CVD also demonstrated their significant presence in the former group than those in the latter group ($p = 0.015$ and $p = 0.034$ respectively). However, diabetic foot was almost identically distributed between the groups ($p = 0.537$)

The variables revealed to be significantly associated with nephropathy by univariate analyses were all entered into the Hosmer and Remeshow model directly. Of the 5 variables (Age, LDL cholesterol, hypertension, duration of Diabetes, $HbA_1C > 7\%$), LDL cholesterol, hypertension and duration of DM were found to be the independent predictors for developing nephropathy with ORs being 3.9, 3.4 and 1.3. The Odds Ratios indicate that patients with LDL Cholesterol > 100 mg/dl had 4 times higher risk of developing nephropathy than patients with LDL Cholesterol < 100 mg/dl ($p = 0.009$). Patients with hypertension were also at 3.4 times higher risk of developing nephropathy ($p = 0.014$) than those without hypertension. The likelihood of developing nephropathy in patients with longer duration of DM was 1.3 times higher than those in patients with shorter durations of DM ($p = 0.003$). (Table-V)

Table-V

Association between study subjects and disease related variables (n = 128)

Variables of interest	Univariate (p-value) analysis	Multivariate analysis Odds Ratio (95% CI of OR)	p value
Age (yrs)	0.021	2.1(0.75-6.03)	0.157
HbA1C (>7%)	0.001	2.6(0.95 – 7.1)	0.064
LDL (> 100 mg/dl)	0.001	3.9(1.4 – 11.2)	0.009
Hypertension	0.001	3.4(1.2 – 9.2)	0.014
Duration of DM (years)	0.027	1.3(1.1-1.6)	0.003

Discussion:

This was a hospital based study on 128 diabetic patients to see the risk factors of nephropathy. The prevalence of overt diabetic nephropathy was 13.3%, that of incipient nephropathy was 27.3% and 59.4% of patients were in normoalbuminuric stage. One population based study showed that prevalence of overt nephropathy was 2.2% and that of microalbuminuria was 26.9% in urban Indians[14] and 23% of Nepalese diabetic patients were having overt proteinuria[15]. A study on 6000 Asian people demonstrated that 39% of diabetic people were found to have microalbuminuria and 19% had overt proteinuria.[8] Prevalence was slightly lower in our study. A population based study in Egypt supported this prevalence. But diabetic nephropathy was higher (47%) in Pima indians and Naurians (75%) [16, 17]. The difference observed in the prevalence of nephropathy among different studies could be due to differences in study design, methodology and sample size.

There are several known risk factors of nephropathy among diabetic patients. In this study, we searched for the presence of known risk factor of nephropathy among diabetic patient and association between risk factor and diabetic nephropathy. Old age, poor glycemic control, long duration of diabetes, hypertension, and high LDL cholesterol were the risk factors for both overt and incipient nephropathy. This result is supported by other studies.

Hypertension accelerates kidney dysfunction in patients with diabetis mellitus. Normalization of blood pressure retards the progression of diabetic nephropathy. In our study, 41.4% diabetic patients had hypertension. Hypertension was significantly higher in nephropathy group (P<0.001). Among nephropathy group hypertension was higher in patients with macroalbuminuria (P<0.001). The study among urban Indian population showed that prevalence of hypertension was higher among subjects with micoralbuminuria and overt nephropathy compared with the normoalbuminuria group (P<0.001).[14] In a study of western world, it has been shown that prevalence of hypertension in older onset DM was found to be 58%[18] and in another study showed 52% hypertensive diabetic patients had proteinuria.[15]

In our study, Aged diabetic patients (>60Yrs.) was found to have more nephropathy. But in case of sex there was no significant association with nephropathy. In a study Klein et al also showed that male were more prone to develop proteinuria.[19] In Another study of 363 patients male sex and age were included among independent risk factors for diabetic nephropathy.[20]

Among other clinical risk factors, prevalence of high BMI, family history of diabetes mellitus, family history of hypertension and family history of diabetic nephropathy was 12.5%, 36.7%, 18.8%, 7% respectively in the present study. Over weight & obesity, family history of diabetes mellitus and family history of hypertension were found significantly higher among nephropathy group (P=0.014, P=0.027, P=0.001 respectively). A prospective study among 574 diabetic patients of western world found that higher BMI and male sex were significantly associated with albuminuria. [21]

Duration of diabetes mellitus plays crucial role for the development of diabetic nephropathy. Study on urban Indian population demonstrated that there was an increase in the prevalence of microabuminuria and macroalbuminuria with the increase in duration of diabetes (Duration of diabetes <1yr: 22.3%, 1-5 yrs: 25.7%, 6-10 yrs: 33.5% and > 10 yrs: 30.2%, P for trend <0.001).[16] Our study also showed similar result. The mean duration was highest in patients with macroalbuminuria (8.3±1.5 yrs). and lowest (2.8±.3) in patient with incipient nephropathy. (P<0.001)

Metabolic risk factors including poor glycemic control, triglyceride and LDL cholesterol were associated with diabetic nephropathy. Several studies in diabetic patients have revealed a beneficial effect of good glycemic control on rate of progression of microalbuminuria to overt nephropathy. These factors like Hyperglycemia, HbA1C and Dyslipidaemia have great influence on diabetic nephropathy. Study from developed country revealed that higher initial plasma value of HbA1C, LDL cholesterol, triglyceride were significantly associated with progression of diabetic nephropathy.[21]

Most studies, not all, found that poor long term glycemic control, indicated by the concentration of glycated hemoglobin, was an important predictor of the development of albuminuria both in non insulin dependent and insulin dependent diabetes.[22-25] Our study also found that about 54% of study population had poor glycemic control as indicated by HbA_1C. HbA1C was also increased (>7%) in nephropathy patients (75%) in comparison to non-nephropathy group (40%). Uncontrolled diabetes as indicated by the concentration of HbA1C and 2hr post prandial blood glucose tends to be associated with the development of nephropathy significantly more than their controlled counter part (non-nephropathy group). (P <0.001) But HbA1C was not an independent risk factor of diabetic nephropathy in our study. The prevalence of hyperglycemia in both microalbuminuria (A1C<7%: 14.5%, 7-8.9%: 22.6%, 9-10.9%: 35.1% and >10.9%: 43.4%) and overt nephropathy (A1C<7%: 0.2%, 7-8.9%: 1.1%, 9-10.9%: 3.5% and >10.9%: 5.5%) was increased with the increasing A1C levels (P for trend <0.001) among urban Indians.[14]

Dyslipidaemia may be responsible for glomerular injury and lipoproteins are also associated with microvascular changes in diabetic patient.[26] The prevalence of triglyceride more than 150 mg/dl and LDL more than 100 mg were found to increased with the increase of urinary albuminuria (P<0.001). Univariate analysis of the data from the RENAAL study confirmed that base line elevated total and LDL cholesterol and triglyceride level were associated with and increased risk of the development of ESRD in patients with type 2 diabetes mellitus.[27] Multivariate analysis of 5 risk factors (Age, LDL Cholestarol, hypertension, duration of Diabetes, HbA1C e" 7%) in our study showed that LDL cholesterol was the independent risk factor for albuminuria. In a study Ravid et al. also found similar observation that concentration of cholesterol was significantly related with the increase in urinary albumin concentration in incipient diabetic nephropathy. [21] There were 15 subjects (12%) who had renal in sufficiency. Retinopathy is one of the common complications of Diabetes Mellitus. Presence of retinopathy is a strong evidence of presence of diabetic nephropathy. In this

study, 29.4% of study population with macroalbuminuria exhibited retinopathy whereas 25.7% of the patients with microalbuminuria had retinopathy and only 2.6% of the patients with normoalbuminuria. Several studies also supported these findings. In a study on urban Indian population prevalence rate of retinopathy was 33.3%.[14] Among other complications, diabetic neuropathy, ischaemic heart disease and cerebrovascular disease were significantly higher in nephropathy group in comparison to non-nephropathy group.

To determine the predictive risk factor for diabetic nephropathy we carried out multivariate binary logistic regression analysis with Hosmer and Lemeshow Model-fit Test. The study revealed that hypertension, duration of diabetes and LDL cholesterol were the predictors for Diabetic nephropathy. Several other studies also showed similar result. Logistic regression analysis in a study by Ravid M et al high lighted the role of glucose control along with the level of total cholesterol and mean blood pressure as joint major risk factors for subsequent renal outcome on 574 diabetic patients.[21] Schmitz and co-workers showed systolic blood pressure to be an independent risk factor for the relative rate of increase of the urinary albumin concentration.[28] A higher mean arterial blood pressure was also a risk factor for development of diabetic nephropathy among the Pima Indians.[29] Mari-Anne Gall showed that male sex, presence of retinopathy, increased serum cholesterol concentration, HbA1C concentration and age were the independent risk factors for the development of nephropathy.[20]

Conclusion:

Diabetic nephropathy is a chronic illness, resulting in increased health cost and economic burden of a person as well as a nation. The result of this study suggests that diabetic nephropathy is multifactorial and prevalence rate is high. Hypertension, duration of Diabetes Mellitus and high LDL cholesterol all are independent predictors for albuminuria among diabetic patients. Poor glycemic control, family history of diabetic mellitus, family history of hypertension, high BMI and high triglyceride are also associated with diabetic nephropathy. A nation wide screening program about Diabetic Nephropathy should be started immediately to counter this disease.

Acknowledgements:

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References:

1. Wild S, Roglik G, Green A, Sicree R, King H. Global prevalence of diabetes: Estimates for the year 2000 and projections for 2030. Diabetes Care 2004; 27(5):1047-1053.
2. World health organization. The diabetes program 2004. http://www.who.int.diabetes.enl.
3. Remmuzz G, Schipath A, Rugenetiti P. Nephropathy in patients with type 2 diabetes mellitus. New Eng J Med 2002; 346: 1145-1151
4. Frier B M & Fisher B M, Diabetes Mellitus. In Davidson's Principle and Practice of Medicine. London,Churchill Livingstone. 2002; 641-682.
5. American Diabetes Association: Clinical Practice Recommendations 2007.Diabetes care 2007; 30(supp1): S19-21
6. Ayodele OE, Alebiosu CO, Salako BL: Diabetic nephropathy: a review of the natural history, burden, risk factors and treatment. J Natl Med Assoc.2004; 96:1445–1454
7. Samanta A, Burden AC, Feehally J, Walls J: Diabetic renal disease: differences between Asian and white patients. Br Med J (Clin Res Ed)1986; 293:366–367
8. Mather HM, Chaturvedi N, Fuller JH: Mortality and morbidity from diabetes in South Asians and Europeans: 11-year follow-up of the Southall Diabetes Survey, London, UK. Diabet Med.1998; 15:53–59
9. Chandie Shaw PK, Baboe F, van Es LA, van der Vijver JC, van de Ree MA, de Jonge N, Rabelink TJ: South-Asian type 2 diabetic patients have higher incidence and faster progression of renal disease compared with Dutch-European diabetic patients. Diabetes Care.2006; 29:1383–1385
10. Diabetes Voice. June 2004, 49(2) p-40, Bulletin of International Diabetes Federation.
11. Mauer SM, Chavers BM. A comparison of kidney disease in type I and type II diabetes. In: Vranic M, Hollenberg CH, Steiner G, eds. Comparison of Type I and Type II Diabetes: Similarities in Etiology, Pathogenesis, and Complications. New York, NY: Plenum Press; 1985:299-303. Advances in Experimental Medicine and Biology; Vol 186.
12. Hasslacher C, Ritz E, Wahl P, Michael C. Similar risks of nephropathy in patients of type I or type II diabetes mellitus. Nephrol Dial Transplant. 1989; 4: 859-863.
13. Pugh JA, Medina R, Ramirez M. Comparison of the course to end-stage renal disease of type I (insulin-dependent) and type II (non-insulin-dependent) diabetic nephropathy. Diabetologia. 1993; 36: 1094-1098
14. Unnikrishnan R, Rema M, Deepa M, Shantirani C S, Deepa R, Mohan V.Prevalence and risk factors of diabetic nephropathy in an Urban South Indian Population.Diabetes Care,2007:30;2019-2024
15. Aryal M and Jha B, Assessment of proteinuria as a marker of nephropathy in type 2 Diabetes Mellitus [http://www.

nmcth.edu /nmcj_articles_pdf/volume_mine/nmcj_vol8_no4/Madhukar_Aryal:pdf.(Access on 24th June, 2008)

16. Collins VR, Dowse GK, Finch CF, Zimmet PZ, Linnane AW: Prevalence and risk factors for micro- and macroalbuminuria in diabetic subjects and entire population of Nauru. Diabetes 1989; 38:1602-1610.

17. Nelson RG, Kunzelman CL, Pettitt DJ, Saad MF, Bennett PH, Knowler WC: Albuminuria in type 2 (non-insulin-dependent) diabetes mellitus and impaired glucose tolerance in Pima Indians. Diabetologia 1989; 32:870–876.

18. Klien K, Klien BE, Moss SE. De Mets Blood pressure and hypertension in Diabetes. Amer J Epidemiol 1985; 122: 75-89.

19. Klien R, Klien BC, Moss S. De Mets DL Proteinuria in diabetes. Arch Intern Med 1988, 148: 181-6

20. Gall MA, Hougaard P, Johnsen K B, Pareving H H. Risk factors for development of incipient and overt nephropathy in patients with non-insulin dependent diabetesmellitus: prospective, observational study.BMJ 1997;314(7083):783

21. Ravid M, Brosh D, Ravid D, Levy Z and Rachmani R. Main risk factors for nephropathy in type 2diabetes mellitus are plasma cholesterol levels, mean bloodpressure and hyperglycemia. Arch Intern Med. 1998;158:998-1004

22. Powrie JK, Watts GF, Ingham JN, Taub NN, Talmud PJ, Shaw KM. Role of glycaemic control in development of microalbuminuria in patients with insulin dependent diabetes. BMJ 1994; 309: 1608-12.

23. Mathisen ER, Rønn B, Storm B, Foght H, Deckert T. The natural course of microalbuminuria in insulin-dependent diabetes: a 10-year prospective study. Diabet Med 1995; 12:482-7.

24. Schmitz A, Væth M, Mogensen CE. Systolic blood pressure relates to the rate of progression of albuminuria in NIDDM. Diabetologia 1994;37:1251-8.

25. Hother-Nielsen O, Faber O, Sørensen NS, Beck-Nielsen H. Classification of newly diagnosed diabetic patients as insulin-requiring or non-insulin-requiring based on clinical and biochemical variables. Diabetes Care 1988; 11:531-7.

26. Moorhead JF, El-Nahas M, Chan MK, Varghese Z. Lipid nephrotoxicity in chronic progressive glomerular and tubulo-interstitial disease. Lancet 1982;ii: 1309-11.

27. Keane WF, Brenner BM, de Zeeuw D, Grunfeld J-P, McGill J, Mitch WE, Ribeiro AB, Shahinfar S, Simpson RL, Snapinn SM, Toto R. The risk of developing end-stage renal disease in patients with type 2 diabetes and nephropathy: the RENAAL study. Kidney Int 2003: 1499-1507.

28. Schmitz A, Væth M, Mogensen CE. Systolic blood pressure relates to the rate of progression of albuminuria in NIDDM. Diabetologia 1994; 37:1251-8.

29. Hother-Nielsen O, Faber O, Sørensen NS, Beck-Nielsen H. Classification of newly diagnosed diabetic patients as insulin-requiring or non-insulin-requiring based on clinical and biochemical variables. Diabetes Care 1988; 11:531-7.

Obstacles to Cadaver Renal Transplantation In A Tertiary Care Hospital

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Abstract:

Cadaver renal transplantation is the need of the time. The aim of the study was to find out the obstacles to cadaver renal transplantation, to evaluate the suitability of cadaver for kidney donation and to identify the social and religious barrier as well as family attitude of the brain death patients of intensive care unit of Dhaka Medical College Hospital and also to evaluate the availability of the infrastucture facilities for cadaver renal transplantation. It was a cross sectional descriptive type of observational study. It was a single centre study and available patients were taken. Total 82 patients were included in the study during 12 months period. Brain death patients admitted in the ICU of Dhaka medical college hospitals were included. These patients were thoroughly evaluated and investigated. Their family was approached for kidney donation after informed consent was obtained. 77(93.9%) families were approachable and 24.7 % (19) families had willingness to donate kidney. Social and religious barriers were also evaluated. It was noted that educated and middle class people were more easily approachable and agreed to donate kidneys. There was lack of infrastructure facilities to start cadaver renal transplantation. It can be concluded that cadaver renal transplantation will be possible in Bangladesh if we can overcome all the barriers.

Key Words: Cadaver, Renal, Transplantation, Obstacles

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Introduction:

Bangladesh is a developing country with a population of around 140 million and an annual population growth rate of 1.42%. It is one of the most populated countries of the world. Per capita income is about US $389[1]. Approximately 100-120 patients per million population (pmp) reaches ESRD every year in Bangladesh[2]. Upto 31st December 1996, 3186 patients were treated with Renal Replacement Therapy (RRT) in Bangladesh. Of them 2048 (65%) patients were treated with MHD, 800 (25%) patients were treated with IPD, 312 (9%) treated with Renal transplantation and 26 (0.8%) patients with CAPD[2]. The prevalence of RRT on a data, upto 31st December 1996, was HD- 17 pmp, PD- 7 pmp and Renal transplantation- 3 pmp[2]. In Bangladesh kidney transplantation is done only from living related donors, thus most of the ESRD patient can not avail the facility of transplantation because of scarcity of donor. Cadaver kidney transplantation will be a real hope for the ESRD patients of Bangladesh. The cadaver donor rates pmp per year are 20.7 pmp in USA, 15.9 pmp in Europe, 3-6 pmp in Poland, Turkey and Greece, 1.1 pmp in Asia, 2.6 pmp in South America and 0.05 in India[3].

In Asia, cadaveric renal transplantation comprises 10% of total kidney transplantation[4]. About 2500 to 3000 cases of renal transplantation are performed in India each year[5] only 2% of which are provided from deceased donors[6], this ratio in Korea is 5%[7].

In Bangladesh 1 year regular hemodialysis cost in private sector is about 3 lac taka[8]. So 90% of patients do not continue hemodialysis after 2-3 months of treatment as they cannot afford. After kidney transplantation, the first 3 months cost is same as hemodialysis, but after that the cost reduced to half. So kidney transplantation is the best option for RRT. Living related donor transplantation is not new in Bangladesh. To increase the donor pool use of suitable cadaver as source of kidney is the only alternative. The aim of this study was to find out the obstacles to cadaver renal transplantation, to evaluate the suitability of cadaver for kidney donation and to

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identify the social and religious barrier as well as family attitude of the brain death patients of intensive care unit of Dhaka Medical College Hospital (DMCH) and to evaluate the availability of the facilities for cadaver renal transplantation. It was a cross sectional descriptive type of observational study. It was a single centre study done in DMCH in one year.

Materials and Methods:

It was a cross sectional descriptive Type of observational study done on brain death patients admitted to Intensive Care Unit (ICU) of Dhaka Medical College Hospital over a period of November 2007 to October 2008.It was a single centre study and available patients were taken. Total 82 patients were included in the study and data were collected by structured questionnaire, clinical examination & biochemical investigations. Purposive sampling method was followed as per inclusion and exclusion criteria. The patients of both sexes between 5 and 55 years of ages, who are possible cases of brain death and whose relatives were interested to participate in the study were included in the study. The patients with Serum Creatinine > 1.4 mg/dl, having pre-existing renal diseases or other systemic diseases, HBsAg, Anti HCV & HIV positivity, chronic infective conditions, current intravenous drug abuse, potentially metastasizing malignancy, oliguric acute renal failure were excluded from the study. Measures of variables are demographic variables, occupation, level of education, socio-economic condition, serum creatinine estimation, blood urea estimation, estimation of viral markers (HBsAg, Anti HCV, Anti HIV), random blood sugar (RBS) estimation.

Operational Definition

Possible brain death patient: Critically ill patient admitted in intensive care unit (ICU) had following conditions- 1) Coma 2) On ventilation 3) Cause of coma would be known.

Brain death: A patient was declared brain death by ICU consultant, when following conditions are fulfilled. 1. Comatose patients on ventilator for more than 12 hrs. 2. Positive diagnosis of cause of coma. 3. Exclusions of following conditions- a) Primary hypothermia (<35º C) b) Drug intoxication c) Severe metabolic and endocrine disorders. 4. Clinical testing: i. Pupil dilated and fixed ii. Absence of all brainstem reflexes- a) Light reflex b) Corneal reflex c) Vestibulo-ocular reflex d) Oculo-cephalic reflex iii. There were no motor responses to adequate painful stimuli. All tests were repeated after 6-12 hours[9].

Road Traffic Accident (RTA)

Attended reported cases of RTA was defined if the patient had head injury due to fatal road traffic accident and became comatose and on ventilator admitted to Dept of Neurosurgery of DMCH and then referred to ICU for artificial ventilation.

Cerebrovascular disease (CVD)

Cerebrovascular disease or 'stroke' has been used to include episodes of focal brain dysfunction due to focal ischemia or hemorrhage as well as subarachnoid hemorrhage[10].

Primary brain tumor (PBT)

Primary intracerebral tumors are classified by their cell of origin and degree of malignancy, and vary in incidence by age and localization. Even when malignant they do not metastasise outside the nervous system[10].

Upper class- To define upper class, the group of people was included having monthly income of more than Tk. 20,000.

Middle class- To define middle class, the group of people was included having monthly income of Tk 5,000 to Tk 20,000.

Lower class- To define lower class, the group of people was included having monthly income of less than Tk 5,000

Data collection procedure: After the admission of a patient in intensive care unit (ICU), the ICU consultant declared the patient as brain death and notified the investigator. Informed consent was obtained from the close relatives of the patients and patient was thoroughly evaluated for the suitability of organ donation and medical contraindications were evaluated by history and physical examination and all the necessary investigations were done according to biochemical variables. Then the patient family was approached for kidney donation. If concerned family disagreed, underlying reason was tried to be evaluated. All the variables were noted in the data collection sheet. The infrastructure facilities to start cadaver renal transplantation were evaluated in DMCH.

Data processing plan and analysis:

Data were processed and analyzed using computer software SPSS (Statistical Package for Social Science) version 11.5. The descriptive statistics were frequency, mean and standard deviation of mean. Categorical data were expressed as percentages and evaluated using Chi-Square Test, Fisher's Exact Test and Unpaired Student's t-test.

Results:

Total 82 patients (49 were male and 33 were female) were selected from the intensive care unit (ICU) of Dhaka

Medical College Hospital (DMCH) to evaluate the suitability of their kidneys for donation as well as to assess the opinion of their close relatives about donation.

Table-I

Distribution of patients by Socio-Demographic variables (n=82)

Demographic variables	No	%
Age (Years)		
<20	11	13.4
21-30	21	25.6
31-40	24	29.3
41-50	12	14.6
>50	14	17.1
Sex		
Male	49	59.8
Female	33	40.2
Occupation		
House wife	27	35.4
Service holder	23	28
Student	13	15.9
Others	19	20.7
Education		
Primary	22	26.8
High School	24	29.3
College & University	32	39
Illiterate	4	4.9
Socioeconomic condition		
Upper class	3	3.7
Middle class	58	70.7
Lower class	21	25.6

Table-II

Distribution of patients by causes of admission (n=82)

Causes of admission	Frequency	Percentage
Road traffic accident (RTA)	52	63.4
Cerebrovascular accident (CVD)	21	25.6
Primary brain tumour (PBT)	09	11.0

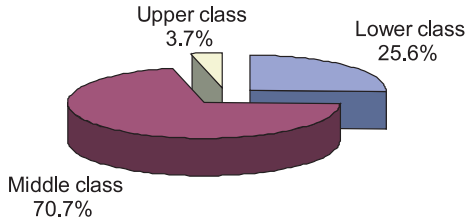

Fig.-1: *Distribution of patients by socioeconomic class (n=82)*

Table-III

Association between kidney donation and demographic features

Demographic features	Donate kidney		p value
	Agreed (n = 19)	Not agreed (n = 58)	
Age (yrs)*	37.0 ± 10.5	36.5 ± 12.7	0.873
Sex#			
Male	13(68.4)	34(58.6)	0.447
Female	6(31.6)	24(41.4)	
Residence#			
Rural	5(26.3)	16(27.6)	0.914
Urban	14(73.7)	42(72.4)	
Religion¶			
Islam	17(89.5)	56(96.6)	0.253
Hinduism	2(10.5)	2(3.4)	
Educational level¶			
Illiterate	2(10.5)	2(3.4)	0.136
Educated	17(89.5)	56(96.6)	
Occupation#			
Housewife	5(26.3)	21(36.2)	0.072
Service-holders & others	14(73.7)	37(63.8)	
Socioeconomic condition#			
Poor	3(15.8)	16((27.6)	0.306
Middle & upper class	16(84.2)	42(72.4)	
Marital status#			
Married	18(94.7)	44(75.9)	0.142
Unmarried	1(5.3)	14(24.1)	

Figures in the parentheses denote corresponding %; * Data were analysed using unpaired t-Test. # Data were analysed using χ^2 Test; ¶data were analysed using Fisher's Exact Test.

Table-IV

Logistics related to cadaver kidney transplantation present in tertiary care hospital DMCH

Logistics related to cadaver kidney transplantation	DMCH
ICU	√
Facilities for organ procurement and harvesting	×
Organ preservation facilities	×
Computerized network for organ sharing	×
Central computerized data base	×
Dedicated Transplant Team	×
Transplant Coordinator	×
Nephrology Team	√
Dedicated Team for declaration of Brain death	×
Transport facilities	×
Social service worker	√
Tissue typing Lab support	×
Donor card	×
All the logistics in the same center	×

Table-V

Prospective kidney transplant recipient information in tertiary care hospital DMCH

Recipient information	DMCH
Waiting list of the patients	
who wants to transplant kidney	×
Blood group	×
Tissue typing	×
Emergency contact no	×

Discussion:

This study was conducted to find out the obstacles to cadaver renal transplantation in a tertiary care hospital of Bangladesh, to assess the family attitude of brain death patients towards kidney donation and to evaluate the suitability of their kidneys for donation in the intensive care unit (ICU) of DMCH. At the same time, number and cause of brain death were identified.

Among them, most of the families 77 (93.9%) were approached. Out of 5 families who were not approached, 4 (80%) of the families were emotional and 1 (20%) due to miscommunication. Potential donor audit of UK including all deaths in intensive care units during 36 months period (1 April 2003 to 31 March 2006) showed that 94% of 3380 potential cadaver donor families were approached and 6% (174) were not approached [11].

Out of 77 families, who were approached to, 19(24.7%) gave consent for kidney donation. In developed countries, like Spain where in 2001 it was possible to obtain organ from 48.7% of all the brain death patients. Family acceptance for kidney donation was 84.2% among brain death patients family and 15.8% family refused to give consent for donation[12]. Among Asian countries, consent rate was 33.5% and refusal rate was 66.5% in Saudi Arabia in 2005[13]. Shroff S et al (2002) in their study showed that 19% of relatives donated the organs to their beloved ones in a major center in India. The consent rate of this study is nearer to Shroff's study of India[14].

It was found that found that Road traffic accident (RTA) and Cerebro-vascular diseases (CVD) were the two main causes of brain death. RTA were 63.4%(52), CVD 25.6%(21) and Primary brain tumor (PBT) were 11%(9) in this study. UNOS data (2006) reported that CVD (42.3%) and Head trauma (41.7%) were two main causes of brain death among Americans in 2005[15]

In this study Mean age of brain death patients was 36.1 ± 12.2 years, 29.3% of cases belong to age group 31 to 40 years. So in this study most of the donors were ideal donors (Ideal donor 18-34 years) as RTA and PBT occurred mostly in young age group. As for sex, male consisted of three-fifth of study population. UK transplant annual report 2005 showed that large portion of cadaver donor came from age group 35 to 49 years and above 50 years.[16]

Donor related obstacles in this study were illiteracy, poor socio-economic condition and lack of awareness of brain death patient's relative towards kidney donation and transplantation. The people of poor socio-economic condition were reluctant to participate in the study. As the chief earning member of most of the family is male, decision for organ donation comes from male. So in case of male brain death patient, it is difficult to get consent from other dependant members.

It was found that lack of Infra-structure facilities needed for cadaver renal transplantation was the main obstacles in our country. All the logistics for cadaver renal transplantation should be in the same center.

In this study it was found that there was lack of waiting list containing Blood group, Tissue typing and emergency contact numbers of the recipient in DMCH. There were similar obstacles as our country in other Asian countries. In contrast to developed countries, the underdeveloped nations have no organized cadaveric transplantation programme[17]. The obstacles of other countries were different from our study, as they are already running cadaveric kidney transplantation programme.

Conclusion:

From this study, it may be concluded that despite of many obstacles, around 25% of brain death patients families had willingness to donate kidneys but there was lack of infrastructure facilities to start cadaver renal transplantation in DMCH.

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References:

1. Bangladesh Bureau of Statistics (BBS), Pocket book of Bangladesh Statistics 2004. Dhaka, BBS; 2004; 38-39.
2. Rashid HU, Bangladesh renal registry report (1986-1996) Bangladesh renal J 2002; 21(1) : 25-28 .
3. Shroff S, Deceased donor program in India and the way forward. 3rd International and scientific seminar, 2007; Nov 8.
4. Cohen B, McGrath SM, De Meester J, Vanrenterghem Y, Persijn GG. Trends in organ donation. Clin Transplant, 1998; 12(6): 525-9.
5. Chugh KS & Jha V. Commerce in transplantation in third world countries. Kidney Int 1996; 49:1181-6.
6. Singh P, Bhandari M. Renal replacement therapy options from an Indian perspective: dialysis versus transplantation. Transplant Proc Sep, 2004; 36(7): 2013-4.
7. Parka JH, Parka JW, Koob YM & Kimb JH. Relay kidney transplantation in Korea-legal, ethical and medical aspects. Legal Medicine 2004; 6: 178-81
8. Rashid HU . Perspective of renal transplantation in Bangladesh. 3rd International and scientific seminar2007; Nov8..
9. Government of people's Republic of Bangladesh, 1999. Bangladesh Gadget for Organ transplantation in human body act:1999. Dhaka; Bangladesh forms and publication office.
10. Allen CMC, Lueck CJ & Dennis M. Neurological disease, In: Boon NA, Colledge NR, W alker BR & Hunter JAA editors. *Davidson's Principles and Practice of Medicine*. London: Churchill Livingstone 20th edition; An imprint of Elsevier 2006; 1200,1236.
11. Blackwell J, Hamilton C & Collett D, Potential donor audit 36- months summary report (1 April 2003 – 31 March 2006). UK transplant 2006; September (20 August 2006).
12. Organicion De Nacionale Transplante (ONT). Renal transplante 2001; Spain;ONT:2001.
13. Saudi Center for organ Transplantation (SCOT), SCOT Annual report 2005. SCOT; 2006; (20 August 2006).
14. Shroff S, Sundaram S, Abraham G, Sounddarajan, Suresh, Rao S, Thomas.P, Cadaver organ donation and transplantation in India. The Antiseptic 2002; 94:73-74.
15. UNOS, United Nations organ sharing network data annual report 2005. USA; UNOS 2006; (28 August 2006).
16. UK Transplant Registry, UK transplant activity 2005-2006; UK transplant 2006; (2004 August 2006).
17. Einollahi B, Nourbala MH, Horeh SB, Assari S, Pezeshki ML, Simforoosh N, Deceased-donor kidney transplantation in Iran: trends, barriers and opportunities. J Med Ethics 2007; 4:70-2.

Evaluation of Nutritional Status in Type 2 Diabetic Patients with Chronic Kidney Disease

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Summary:

In the present study the nutritional status of diabetic chronic kidney disease patients were assessed by anthropometric measurements and evaluation of dietary diary. The study subjects belonged to both pre-dialysis and those on dialysis. Total number included was 125 who were divided in group 1 (pre-dialysis, n=80) and group 2 (on dialysis, n=45).Both groups were matched for age, sex and BMI. Anthropometric comparisons of different skin fold thicknesses showed that dialysis patients had significantly lower MAC (mid arm circumference),TSF (triceps skin fold thickness) and SSF (subscapula skin fold thicknen) compared to the pre dialysis group. Calorie intake ideal to body weight was lower in both groups (<25 kcal/kg/d). Dietary protein intake was less than recommended 1.2g/kg/d in dialysis patients and the high biologic portion in protein (HBV) was also low in all subjects (<45%). It may be concluded that diabetic CKD patients have lower calorie intake with diet deficient in quality protein. Dialysis subjects are malnourished evidenced by reduced muscle mass and decreased subcutaneous fat. Increased macro and micronutrients intake with appropriate supplementation may help by improving their overall nutritional status.

Key words: Nutrition, DN, Diet, CKD

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Introduction:

Nutritional status is defined as the study of the condition of the body as it relates to the consumption and utilization of food. It is also the study of the relationship to determine an optimal diet. A variety of methods are used to determine nutritional status; which methods are based on series of dietary, laboratory, anthropometry and clinical measurements designed to characterize each stage in the development of a nutritional deficiency state[1].

The appropriate diet at any stage of life is one that provides sufficient energy and all the essential nutrients (carbohydrate, protein, fat, vitamins, minerals and water) in adequate amounts for health[2]. The calorie value per unit food is for 1gm of carbohydrate 4.0 kcal or 17J, 1gm of protein yields 4.0 Kcal or 17J and 1gm of fat yields 9.6 Kcal or 38. In a well balanced diet about 60-65% of the total calorie requirement may be met through intake of carbohydrates [3]. Protein requirements depend on physiological states (growth, pregnancy, lactation) of the individual. Proteins normally supply 10-15% of energy. Protein of high biological value is complete proteins and contains all essential amino acids. They may not supply all the essential amino acids or may supply them in limited amounts[4]. The protein allowance recommended is about 1gm per kilogram body weight per day. It is desirable that the energy from fat should not exceed 30% of total calorie.[5] Approximate daily energy requirement is 30-35 kcal/kg/d, 35-40 kcal/kg/d and 40-45 kcal/kg/d for sedentary, moderate and active individual repectively.[6] Vitamins are needed in small amounts but they are essential for health and well being of the body There are two kinds of vitamins: such as, fat, soluble (Vit-A, D, K), Water soluble (Vit-B complex and C). For an adult 900mg/d Vit-A, 5mg/d Vit-D,15mg/d Vit-E, 120μg/Ca+ 800mg, PO4 800 mg. Thiamin 1.3 mg/d, Riboflavin 1.6 mg/d, Niacin 2.0 mg/d, Pyridoxin1.6mg/d, 100mg/d Folic acid,1g/d Vit-B12, 40mg/d Ascorbic acid is adequate (4).For an adult 28 mg/d Iron, 15.5 mg/d Zinc, 65mg/d Chromium, 2.2 mg/d copper and 5.5 mg/d manganese is needed as trace elements[7,8]. The daily requirement for sodium is unknown but 1100-3300 mg has been suggested as a safe and adequate intake. The minimum requirement is believed to be very much less. A safe and adequate potassium intake is estimated to be 1875-5625 mg[9].

Moderate calorie restriction (250-500 calories less than average daily intake) is advised for obese individuals with Type- 2 diabetes. Protein can be 10- 20% of daily calorie intake (both animal and vegetable sources). General

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recommendation is to have 0.8-1.9g/kg/d body weight because insulin deficiency increases the rate of protein degradation. Regarding fat 10% of calories from saturated fats, <10% of calories from polyunsaturated fats, 10- 15% of calories from monounsaturated fat is advised with a limitation of dietary cholesterol to <300mg daily. Sodium chloride intake in individuals without hypertension is 6-7gm/day. Generally no need for additional vitamin and mineral supplementation for the majority of individuals with diabetes[10].

The dietary management may vary for each type of kidney disorder or renal failure and is usually individualized. The general principals of dietary management in renal disease is to achieve a balance between intake and output, alleviate symptoms, maintain adequate nutrition, retard progression of renal failure in order to postpone dialysis. [2] Protein intake is restricted to 0.8g/kg/day in patients with proteinnuia and nephropathy further restriction to 0.6/g/kg/day in advanced kidney failure. Fluid intake will be calculated from urine output.[11] Calcium and phosphorus should be supplemented to 1400-1600 and 600 -1200 mg/day. Generally 500-1000 mg/day sodium is recommended for individuals with hypertension and nephropathy.[12] Fat soluble vitamins may require supplemented due to deficiencies arising from anorexia, uremia and altered metabolism. The national Kidney Disease Outcome Quality Initiative clinical practice guidelines (KDOQI) for anemia of chronic kidney disease suggest that at least 200 mg of elemental iron for adult patient. Commercially available supplements are only in multivitamin- mineral preparations with Zinc content ranging from 15 to 25 mg per dose and if there is no response of the clinical symptoms and laboratory tests, the supplement should be discontinued [13,14].

Diabetic patients have many dietary restrictions especially those with renal failure. Poor nutritional status is a predisposing factor for increased morbidity and mortality in chronic kidney disease (CKD). This study was undertaken to evaluate the nutritional status of diabetic patients with CKD.

Methods and Material:

This was an observational study. Patients were included from both pre dialysis (group 1) and those on hemodialysis (group 2). A standard questionaire was prepared to obtain relevant information on the socio-economic status, such as age, sex, location, occupation, family size, income status etc. From the dietary history the patient's intake of calorie, protein, fat, carbohydrate, calcium, phosphorus, sodium, potassium, vitamin-A and vitamin-B was calculated by using standard food table. In this study patients were given a diet recording sheet to record food intake over a period of 3 days prior to a visit and in relevant cases cross checked by recalls. There are a lot of' vegetables, fruits, crops produced in Bangladesh. The nutritive values of different food elements have not yet been determined. For the conversion to calorie and identifying elements, different methodologies published in journals and books, both from the Subcontinent and Western countries were looked for. Dietary salt added during cooking was calculated from 20 common items cooked separately. This was averaged and added to all patients as standard daily salt consumed from cooked food.

The anthropometric measurements to assess protein energy nutritional status in study subjects will include mid arm circumferences (MAC) , biceps skin fold thickness (BSF), triceps skin fold thickness (TSF), and sub-scapular skin fold thickness (SSF), supra iliac skin fold thickness, mid-arm muscle area or circumference and body mass index (BMI). Harpenden skin calipers will be used to measure skin folds. Mean of the repeated measurements were taken. The waist and hip measurement (cm) was done using a measuring tape. Waist-hip circumferences ratio was calculated to describe the distribution of both subcutaneous and intra - abdominal adipose tissue. Waist hip ratios greater than 1.0 for man and 0.8 for woman are said to be indicative of obesity.

Results:

Total 125 diabetic CKD patients were included in the study. They were grouped into Predialysis (gr1 -80) and hemodialysis patents (gr2 - 45). Both the groups were matched for age (56±9 and 53±11years, p=NS) and BMI (25±6 and 24±5 kg/m^2, p=NS). Anthropometric comparisons of skin fold thicknesses showed group-2 had significantly lower mid arm circumference(28.2±3 and 25.8±5 cm, p <0.005), TSF (16.3±5.8 and 14±5.5mm;p <0.033) and sub scapular skin fold (25±7 and 20±9mm, p <0.006) than group-1.

Different anthropometric parameters like BSF, TSF and SSF thickness and (MAC) were measured. (Table-I)

Table- I
Anthropometrics

	Pre dialysis (CRF)	Dialysis (HD)	P value
BSF (mm)	8.6±3.4	7.6±3.3	0.128
TSF(mm)	16.3±5.8	14±5.5	0.033
SSF(mm)	25±7	20±9	0.006
MAC(cm)	28.2±3	25.8±5	0.005
Waist(cm)	88±8	84±14	0.172
Hip(cm)	95±7	89±10	0.003
W/H ratio	0.93±.05	0.95±.07	0.096

Note: BSF-Biceps Skin Fold Thickness, TSF- Triceps Skin Fold, SSF- Sub Scapular Skin Fold Thickness, MAC- Mid Arm Circumference. W/H ratio- Waist Hip ratio.

Daily calorie intake was lower in group 1 than 2 (20±5 and 24±8 kcal/kg/d, $p < 0.005$). Dietary protein intake was relatively higher in-group 2 (0.79±0.24 and 0.97±0.59 g/kg/d, $p < 0.004$) but less than recommended 1.2g/kg/d during dialysis. High biologic value (HBV) protein intake (37±14 and 45±23 %, $p < 0.038$) was also low in all subjects (<45%). (Table-II)

Table-II
Macronutrient Intakes

	Pre-dialysis (CRF)	Dialysis (HD)	P value
Calorie (cal/kg/d)	20±3	24±8	0.001
CHO (%)	63±15	55±15	0.004
PROT/kg	0.79±0.24	0.97±0.59	0.015
HBV (%)	37±14	45±23	0.038
FAT (%)	23±7	23±7	0.922

Note: CHO-Percent of Carbohydrate in diet, PROT/kg-Protein intake/kg body wt, HBV-Percent of High Biological Value protein in diet, Fat-percent of fat in diet.

When vitamin intakes was assessed in group 1 and 2, it showed that both groups had low vitamin B_1 (0.4±0.3 and 0.3±0.2 mg/d, p =NS), Vitamin-B_2 (0.9±2.1 and 1.2±0.8 mg/d, p =NS), Niacin (15±9 and 11±9mg/d, p =0.016) and folic acid (87±49and 122.55±84.59 mg/d, p =0 .012) intake than recommended in CKD. (Table-III).

Table-III
Micronutrient- Vitamins

	Pre-dialysis (CRF)	Dialysis (HD)	P value
Vitamin A (mgm/day)	1038±959	1161±1339	0.59
Thiamine(B_1) (mg/day)	0.4±0.3	0.38±0.26	0.804
Riboflavin(B_2) (mg/day)	0.99±2	1.24±0.84	0.37
Folic acid (mg/day)	87±49	122±84	0.012
Niacin (mg/day)	15±9	11±9.15	0.016

Dietary intake of sodium (1895±791 and 2364±1081 mg/d, $p < 0.014$) was high in group 2 patients while calcium (398±243 and 402±259 mg/d, p =NS), potassium (923±396 and 904±604 mg/d, p=NS) and phosphate (839±245 and 973±467 mg/d p =NS) was similar in both groups. (Table 4)

Table-IV
Micronutrients (Electrolytes):

	Pre dialysis (CRF)	Dialysis (HD)	P value
Na^+(mg/dl)	1895±791	2364±1081	0.014
K^+(mg/d)	923±396	904±604	0.86
Ca^+ (mg/d)	398±243	402±259	0.937
$PO4^-$(mg/d)	839±245	973±467	0.079

Note: Na-Sodium in diet, K- Potassium in diet, Ca- Calcium in diet, PO4- Phosphorus in diet.

Discussion:

Diet plays an important role in health and disease. Intake of different micro and macronutrients at optimum level is advised in guidelines for both healthy and chronic kidney disease patients. Evaluation of nutritional status is usually done by clinical observations and supplemented by laboratory tests. In this study the nutritional status of diabetic kidney disease patients (both pre dialysis and on dialysis) was assessed from anthropometric measurements and dietary intake (diet diary).

Body fat distribution is frequently assessed and it is a sensitive tool for evaluation of nutritional status. Comparisons of BSF, TSF and SSF showed that they were low in dialysis group than the pre dialysis patients. Similar findings have been also shown by others studies[3].

Dialysis patients are generally anorexic due to uremia related to inadequate dialysis and take less food. Also the dialysis process itself initiates catabolism which is partly

responsible for poor nutritional status leading to lower subcutaneous fat as well as decreased muscle mass [3]. In the dialysis group the mid arm circumference (MAC) is lower than pre dialysis subjects indicating ill health and wasting.

The dietary assessment from 3 day dietary diary in our study subjects showed that calorie intake in general was low in CKD patients than recommended (30-35kcal/kg/day).The protein intake in dialysis group was sub optimum (0.9g/kg/day) than the required amount (1.2g/kg/day) and their low muscle mass (MAC) was likely to be for this low protein intake [12].

The different electrolytes level assessed showed that all the study subject was taking salts much higher than the recommended amount indicating a poor compliance to salt restriction required in kidney diseases. The potassium and phosphate intake was within the range but the calcium intake was lower in both pre dialysis and dialysis group. (table4) This again may be due to dietary restrictions on vegetables and protein containing foods resulting in low protein as well as low calcium intake as meat and meat products are good source for calcium.

Micronutrients like vitamin A, B_1, B_2, niacin and folic acid intake was also assessed from dietary diary. Comparisons with patient intake to recommend allowances showed that vitamin B_1, B_2 and folic acid intake was low in study subjects. Major sources of vitamin B_1 is un milled cereals which was not being taken by most of the patients. Vitamin B_2 and folic acid are available in meat, fish, milk and some leafy vegetable. In chronic kidney disease the restriction on amount of protein and vegetables again is likely to be responsible to this less vitamin intake.

From this study it may be concluded that in diabetic chronic kidney disease patients (pre-dialysis and dialysis group) the calorie intake is generally lower than recommended. Their diet is deficient in quality protein, vitamins and calcium intake. It is also found that the dialysis subjects are malnourished as evidenced from reduced muscle mass and decreased subcutaneous fat distribution. Increased macro and micro nutrients intake with appropriate supplementation may help improving their overall nutritional status.

References:

1. Gibson R.S. Principles of nutritional assessment; determining nutritional status; chap-26, 467-478; 1990; New York: Oxford University Press.
2. Stanfield PS, Hui YH. Nutrition and Diet therapy. American Dietetic Association. Jone and Bratlell publishers, 2003; P-9-11.
3. Joshi S.A. Nutrition and Diettics; Roles of Nutrients, 2nd edition, 2002, chap-3, p-119-121; Tata McGraw-Hill Publishing Company Limited, New Delhi.
4. Gopalan C,Sastri B . V .R, Balasubamanian S.C. Nutritive value of foods, revised edition, India: National institution of nutrition. 1993; Appendix-1, p-92.
5. Antia.P.F,.Abraham P. Clinical Dietetics and Nutrition,4th edition, Oxford University Press, New Delhi 2003.
6. Robinson C H, Marilyn R L and Ann E G. Normal and therapeutic nutrition,17th edition, Macmillan publishing company, 1989, P-106.
7. Satyanarayan U. Biochemistry, Diabetes Mellitus, 2nd revised edition, Kolkata: Arunavha Sen. 2002.
8. National Research Council. Recommended dietary Allowances, 10thed, Washington, DC: National Academy Press, 1989.
9. Zeman FJ. Clinical Nutrition and Dietetics 2nd. Macmillan Publishing Company. 1991; P-26-28.
10. Dikow R, Browatzki M, Adamezak M et al. Nutritional Requirements of Diebetics with Nephropathy. American Diabetes care 2004; P-544-547.
11. Garow JS. Et,Human nutrition and Dietetics, 10th edition. Churchill livingstone. Hartcurt publisher Limited, UK. 2004.
12. Zeman F.J. Clinical Nutrition & Dietetics, 2nd edition, Macmillan Publishing Company, New York, 1983, P-315.
13. Masud T, Trace Elements and vitamins in Renal Disease, in Mitch WE, Klahrs. Hand book of Nutrition and the kidney, Lippincott Williams & Wilkins. 2004.
14. Gerald F. Combs. The vitamins, Fundamental Aspects in Nutrition & Health, Jr Academic Press, Inc, A Division of Harcourt Brace & Company, New York. 1966.
15. Ali.K.M.S. Nutritive Value of Bangladeshi foods, University of Dhaka, Institute of Nutrition and Food Science, Standard Printing & Packages. 1920.

Superior Sagittal Sinus Thrombosis and Herpes Zoster in a Patient of Steroid Dependent Nephrotic Syndrome

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Summary:

A boy of 14 years, diagnosed case of steroid dependent nephrotic syndrome, presented with severe headache and vomiting. He was normotensive and had bilateral gross papilloedema with features of cushing syndrome. His renal function was normal. MRV findings revealed superior sagittal sinus thrombosis. He was treated with standard heparin followed by warfarin. After one week he also developed herpes zoster in the back and front of the chest and intravenous acyclovir was added. Within 7 to 10 days of treatment, his condition improved and was discharged.

Key words: Thrombosis, Sagittal sinus, NS, Proteinuria.

(Bang. Renal J. 2010; 29(2): 46-48)

Introduction:

The incidence of thromboembolic complications (TEC) in nephrotic syndrome is about 1.8 – 5%, but may be an underestimation. Incidence is higher in adults and children with secondary nephrotic syndrome. The incidence is especially high in membranous nephrotic syndrome. Renal vein thrombosis, deep vein thrombosis and pulmonary embolism are the most frequently encountered TEC in children. Other venous sites of thrombosis include the superior sagittal sinus, other cerebral venous sites. Various infections can also occur, including meningitis, cellulitis, viral infections and others. Varicella is a particular concern in immunosuppressed patients and can be lethal. Prompt recognition and treatment with ACYCLOVIR (or postexposure prophylaxis with varicella-zoster immunoglobulin (VZIG) is essential.

The superior sagittal sinus is a midline venous channel, located between the inner and outer dural laminae. It receives blood from many tributaries, including the cortical, cerebral, meningeal, emissary and scalp veins. These channels can provide a collateral pathway that bypasses the thrombosis of the superior sagittal sinuses[1]. Many predisposing factors have been implicated in the development of superior sagittal sinus thrombosis. Trauma, tumours, dehydration, hypercoagulable state such as pregnancy and nephrotic syndrome are the most common causes. Nearly 20% cases of dural sinus thrombosis are idiopathic[2]. Superior sagittal sinus thrombosis is an extremely rare complication of nephrotic syndrome[3]. Non-contrast CT scan may demonstrate increased attenuation in thrombosed veins (cord sign). Contrast enhanced CT may demonstrate "empty delta sign", which occurs when the thrombus fails to enhance within the dural sinus and is outlined by enhanced collateral channels in the falx. This sign is seen in only about 25%-30% of cases, but is highly diagnostic for sagittal sinus thrombosis. False positive causes of delta sign include subdural haematoma and arachnoid granulations[4].

Case History:

Md. Rakib Hossain, 14 years old boy, developed gradual swelling of legs and face with reduction of urine volume about one year back. Prior to development of leg swelling he did not give any history of arthritis, arthralgia, skin rash and fever. He was normotensive. At that time he was thoroughly evaluated and investigated. Investigation revealed Urine R/E, Albumin +++, RBC- nil to WBC 2-4/ HPF, Granular Cast + , 24 hours total protein 5 gm, Serum urea 4.4 mmol/L, Serum albumin was 25g/l and Serum cholesterol was 320 mg/dl ,Serum creatinine 55 micro mol/ L, Serum electrolytes was within normal limit, Complete Blood picture showed Hb 13 gm/dl, ESR 55 mm in first hour, Ultra sonogram showed normal sized kidney, ANA , Anti-ds- DNA, C3, C4 was within normal limit. He was Prescribed Tab- Prednisolone 1 mg/kg body weight. Two months later proteinuria disappeared and steroid was tapered, during tapering phase, with 20 mg prednisolone, proteinuria reappeared. He was again prescribed Tab- Prednisolone 1 mg/kg body weight. And he received it for further 3 months, and steroid was gradually tapered.

On July 2008, he got himself admitted in Department of Nephrology, BSMMU, with the complaints of severe

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generalized weakness, headache, occasional vomiting. But he had no history of coma or convulsion. Physical examination revealed, his pulse was 70 beats per minute, BP 120/80 mm/Hg, severely edematous and features of cushing syndrome. Fundoscopic examination revealed bilateral gross papilloedema but his field of vision and visual acquity was normal, and no focal neurological deficit was observed. CT scan was normal, no evidence of ventriculomegaly. MRV findings were suggestive of slow flow within posterior part of superior sagital sinus with absent flow at the anterior half. Neurological consultation was done and he was diagnosed a case of superior sagittal sinus thrombosis. He was treated with standard heparin , followed by warfarin along with acetazolamide. Seven days later he developed severe Herpes Zoster in the back of the chest for that he received IV acyclovir 15 mg/kg body weight three times daily for five days followed by oral acyclovir. Within 7 to 10 days, his condition improved and he was discharged from hospital.

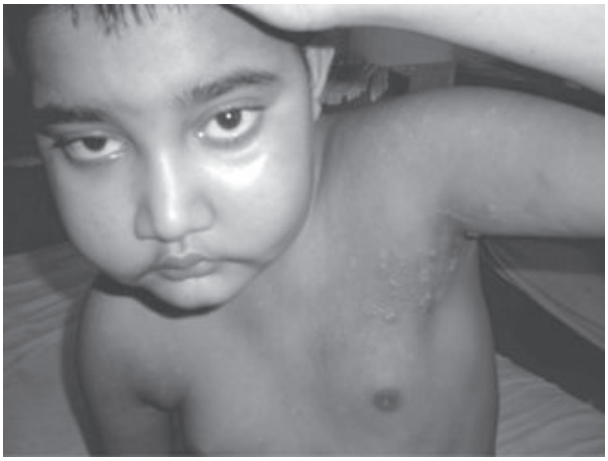

Fig.-1: *Herpes Zoster*

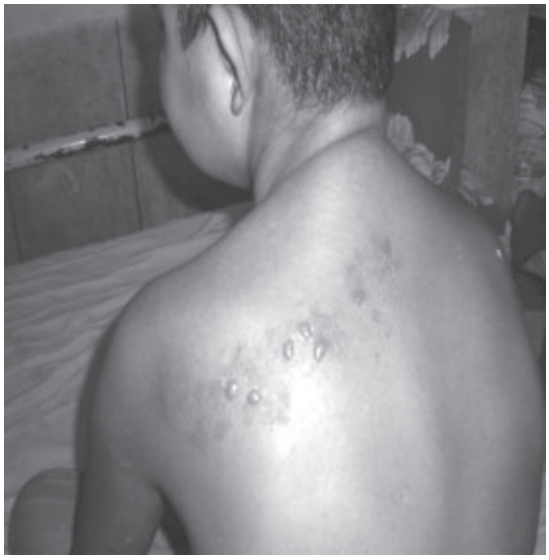

Fig.-2: *Herpes Zoster*

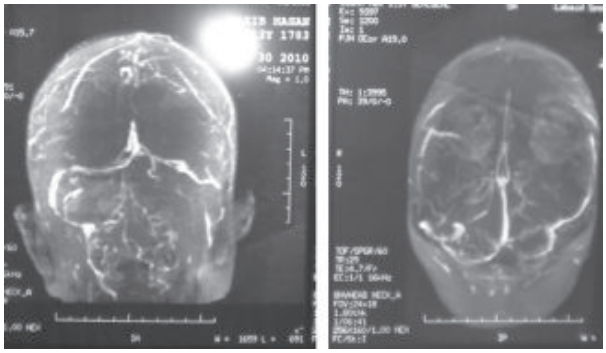

Fig.-3: *MRV (Magnetic Resonance Venography) shows, superior saggital sinus is not visible in its anterior half and narrowing of the posterior part.*

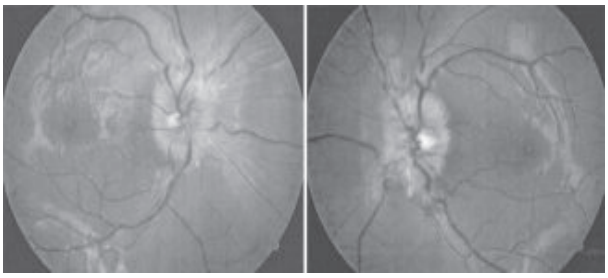

Fig.-4: *Bilateral gross papilloedema.*

Discussions:

The incidences of both venous and arterial thrombosis are much higher in patients with nephrotic syndrome compared to the general population[5]. The risk of thrombosis varies among the causes of nephrotic syndrome. The risk is highest with membranous nephropathy, followed by membrano-proliferative and minimal change disease [6]. The risk of thrombosis may also be related to severity and duration of nephrotic state and greatest within first six months[7]. Superior sagittal sinus thrombosis is rare but devastating complication of nephrotic syndrome and is associated with hypercoagulable state due to factors like elevated levels of plasma fibrinogen, and factors V, VII, VIII, X [3]. Dural sinus thrombosis manifest with diverse clinical findings. Early symptoms include headache and lethargy. As the diseases progresses, seizures, decreased mentation, and focal deficit can occur. Stroke (haemorrhagic) may develop secondary to poor venous drainage. These strokes are often bilateral and outside the normal arterial distribution reflecting the pattern of venous drainage. The patient in this case report suffered from long duration of nephrotic syndrome, he had history of persistent headache but he had no history of convulsion or focal neurological deficit.

After proper management with anti-coagulants he recovered from this complication.

Varicela Zoster infection is another devastating and potentially curable condition in immune-suppressed state. The immunocompromised patient may have a more severe illness with dissemination (35%) causing generalized skin lesions and complications like CNS, pulmonary and hepatic involvement. In this case we have found that after giving I/V ACYCLOVIR, patient improved symptomatically within few days.

References:

1. Virapongse C, Cazenave C, Quislinghling R, Sarwar M, Hunter S. The empty delta sign: frequency and significance in 76 cases of dural sinus thrombosis. *Radiology* 1987; 162: 779-785.
2. Yuh WT, Simonson TM, Wang AM, Koci TM, Tali ET, Fisher DJ, Simon JH, Jinkins JR, Tsai F. Venus sinus occlusive disease: MR findings. *AJNR Am J Neurorad* 1994; 15:309-316.
3. Ptullu MS, Deshmukh CT, Save SU, Bhoite BK, Bharucha BA. Superior sagittal sinus thrombosis : a rare complication of nephrotic syndrome. *J Postgrad Med* 1999; 45:120-122.
4. Provenzale JM, Joseph GJ, Barrborial DP. Dural sinus thrombosis : Finding on CT and MR imaging and diagnostic pitfalls. *AJR Am J Roentgenol* 1998;
5. Justich E, Fritsch G, Beitzke A, Walter GF.CT diagnosis thrombosis of dural sinus in childhood. Eur J Radiol 1984;4:294-5.
6. International classification of diseases, 9th rev., clinical modification: ICD-9-CM. 5th ed.1997 Hospital ed. Los Angels:Practice Management Information,1996.
7. Shevell MI, Silver K, O Gorman AM, Watters GV, MontesJL. Neonatal dural sinus thrombosis. Pediatr Neurol 1989;5: 161-5.

Abstract from Current Literature

Genetic polymorphisms of the renin-angiotensin-aldosterone system in Chinese patients with end-stage renal disease secondary to IgA nephropathy

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Background: Genetic variability in the renin-angiotensin-aldosterone system may modify renal responses to injury and disease progression. The angiotensin I-converting enzyme (ACE) gene insertion/deletion (I/D), the angiotensinogen (AGT) gene, M235T, the aldosterone synthase (CYP11B2) gene, C-344T, and the angiotensin II type 1 receptor (AT1R) gene, A1166C, have been shown to be associated with IgA nephropathy (IgAN) and its progression. We determined the presence of these polymorphisms in 130 Chinese patients with IgAN, including 47 patients with end-stage renal disease (ESRD) and 120 healthy Chinese subjects, to assess their impact on the susceptibility to disease and the liability of progression to ESRD.

Methods: Genotyping was performed with DNA isolated from peripheral leucocytes using polymerase chain reaction amplification of the polymorphic sequence, restriction enzyme digestion, and separation and identification of DNA fragments. Clinical data from renal biopsies were collected.

Results: ACE, AGT, CYP and AT1R genotype distributions were similar in patients with IgAN and in controls. Comparing patients with ESRD (IgAN-ESRD) and those without ESRD (IgAN-non ESRD), there was a significant increase only in the ACE DD genotype ($P < 0.05$) among the four gene polymorphisms. There was significant dominance of the male ($P < 0.05$), more marked hypertension ($P < 0.01$), proteinuria ($P < 0.01$) and increased serum creatinine during renal biopsy ($P < 0.01$) in the IgAN-ESRD group.

Conclusion: Among the ACE, AGT, AT1R and CYP gene polymorphisms, only the DD genotype may predispose the individual to increased risk of progression to ESRD in the Chinese population.

Chin Med J (Engl). 2010 Nov;123(22):3238-42.

Lupus nephropathy: clinical, immunohistochemistry and quantitative morphometric analysis studies.

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Lupus nephritis includes a wide range of parenchymal injuries and severity. Better predictors to outcome are needed for patients newly diagnosed with lupus nephritis, so that an appropriate management strategy may be selected. This study aimed to determine whether the ratio of hepatocyte growth factor (HGF) to transforming growth factor beta 1 (TGF beta1) in lupus nephritis could be a prognostic factor for response to therapy with cyclophosphamide and steroids at six months. Also, to determine whether a simple automated system for objective scoring of biopsies of lupus nephritis could be a prognostic factor for response to therapy with cyclophosphamide and steroids at 6 months. Consequently, renal biopsy findings and clinical parameters of thirty parasites-free patients with new onset lupus nephritis were recorded. Histopathologic, clinical, immune-histochemical and morphometric data at baseline served to define the predictive value for outcome after 6 months of therapy. The results showed a significant positive relationship between response to therapy and HGF IS ($P= 0.007$), HGF ES ($P= 0.026$), HGF IS/ TGFbeta1 IS ratio ($P= 0.022$) and HGF ES/ TGFbeta1 ES ratio ($P= 0.001$). A significant inverse relationship was proved between response to therapy and TGFbeta1 IS ($P= 0.025$) as well as TGFbeta1 ES ($P= 0.017$). Also, a significant inverse relationship was present between response to therapy and nuclear index, tubular index and matrix index ($P = 0.03, 0.03$ and 0.029 respectively)

J Egypt Soc Parasitol. 2010 Aug;40(2):321-35.

Systemic lupus erythematosus complicated with posterior reversible encephalopathy syndrome and intracranial vasculopathy.

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Posterior reversible encephalopathy syndrome (PRES) is a neurotoxic condition characterized by reversible vasogenic edema on neuroimaging. It is associated with various neurological manifestations, including headaches, vomiting,

seizures, visual loss, altered mental status and focal neurological deficits. PRES mainly occurs in the setting of eclampsia, hypertension, uremia, malignancy, transplantation, autoimmune diseases and/or use of immunosuppressive drugs. This syndrome has been described in patients with systemic lupus erythematosus (SLE). PRES is a potentially reversible clinical-radiological entity; however, it can be complicated with vasculopathy, infarction or hemorrhage. Vasculopathy has been demonstrated to be a common finding in patients with SLE. We report the case of a woman with lupus nephritis and PRES whose diffuse vasculopathy was present on initial neuroimaging. Subsequent brain computed tomography scan demonstrated interval development of intraparenchymal hemorrhage and subarachnoid hemorrhage. To our knowledge, this unique brain image pattern has not been reported in SLE patients

Int J Rheum Dis. 2010 Oct;13(4):e79-82. doi: 10.1111/j.1756-185X.2010.01545.x.

Clinical improvement of membranous nephropathy after a resection of rectal cancer. [Article in Japanese]

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A 71-year-old man was admitted by systemically massive edema and advanced rectal cancer. His hemoglobin or serum albumin level was 7.5 g/dL or 1.2 g/dL. Proteinuria ranged from 1.8 to 3.8 g/day. Massive effusion in chest and abdomen was obvious with low oxygenation and unstable hemodynamic state. Renal biopsy showed membranous nephropathy. Abdomino-perineal resection of the rectum was performed. Specimens showed poorly differentiated adenocarcinoma. The classification was type 1, 90 × 85 mm, pAI (seminal grand), pN3, sH0, sP0, cM0: fStage IIIb. The nephrotic syndrome was evidently improved with no urinary excretion of albumin at forty-postoperative day. The perioperative management allowed a surgical resection to be undertaken that led the clinical curability in rectal cancer as well as nephrotic syndrome.

Gan To Kagaku Ryoho. 2010 Nov;37(12):2614-6.

The changing pattern of primary glomerulonephritis in Singapore and other countries over the past 3 decades

Woo KT, Chan CM, Mooi CY, -L-Choong H, Tan HK, Foo M, Lee GS, Anantharaman V, Lim CH, Tan CC, Lee EJ, Chiang GS, Tan PH, Boon TH, Fook-Chong S, Wong KS.

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This review of 2,586 renal biopsies over the past 3 decades in Singapore documents the changing pattern of glomerulonephritis (GN) from that of a third world country to that of a developed nation. In the 1st decade, mesangial proliferative glomerulonephritis was the most common form of primary GN, just as it was in the surrounding Asian countries. In the 2nd decade, the prevalence of mesangial proliferative GN decreased with a rise in membranous, GN which is also seen in China and Thailand. In the 3rd decade, there was a dramatic increase in focal sclerosing glomerulosclerosis. This increase reflects aging and obesity in keeping with more developed countries like Australia, India, Thailand and the United States of America. IgA nephritis remains the most common GN. Apart from the geographical influence, other socioeconomic factors play a significant role in the evolution of the renal biopsy pattern. Mesangial proliferative GN remains prevalent in many Asian countries, but in Singapore the prevalence is decreasing just as it is in Japan, Korea and Malaysia. Worldwide, the prevalence of focal sclerosing glomerulosclerosis continues to increase in many countries.

Clin Nephrol. 2010 Nov;74(5):372-83.

Inhibition of capillary repair in proliferative glomerulonephritis results in persistent glomerular inflammation with glomerular sclerosis.

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The pathological process of glomerulonephritis (GN) includes glomerular capillary damage, and vascular endothelial growth factor (VEGF) has an important role in glomerular capillary repair in GN. We examined the effect of inhibition of glomerular capillary repair after capillary injury in GN. Experimental Thy-1 GN was induced in rats that were divided into two groups: rats that received anti-VEGF neutralizing antibody (50 ìg per 100 g body weight er day) and those treated with the vehicle from day 2 to day 9. We assessed the renal function and histopathology serially until week 6. Rats of the Thy-1 GN group showed diffuse glomerular mesangiolysis with ballooning destruction of the capillary network by day 3. VEGF(164) protein levels increased in the damaged glomeruli during days 5 to 10, and endothelial-cell proliferation increased with capillary repair in the vehicle-injected group. Proliferative GN resolved subsequently with decreased mesangial hypercellularity, and recovery of most of the glomeruli to the normal structure was evident by week 6. In contrast,

administration of anti-VEGF antibody significantly decreased endothelial-cell proliferation and capillary repair in glomeruli by week 2. Thereafter, glomerular mesangial-cell proliferation and activation continued with persistent infiltration of macrophages. At week 6, segmental glomerular sclerosis developed with mesangial matrix accumulation and proteinuria. Deposition of type I collagen was also noted in sclerotic lesions. We conclude that impaired capillary repair was the underlying mechanism in the prolongation of glomerular inflammation in proliferative GN and in the development of glomerular sclerosis. Capillary repair has an important role in the recovery of glomerular damage and in the resolution of proliferative GN.

Lab Invest. 2010 Oct; 90(10):1468-81. doi: 10.1038/ labinvest.2010.130. Epub 2010 Jul 19.

Profile of glomerular diseases in a public hospital of Federal District, Brazil.

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Introduction: Glomerular diseases are a frequent etiology of chronic kidney disease, especially in the developing countries.

Objective: To determine the profile of such glomerulopathies in a public hospital located in the city of Brasilia, Federal District.

Methods: 121 renal biopsies in different patients were performed by the Renal Division of Hospital Regional da Asa Norte (HRAN) between August 2005 and May 2009. Eight renal biopsies in renal-transplant patients were excluded and the medical records of 113 remaining patients were analyzed. Analyzed data: sex, age, laboratory exams, glomerular syndrome, clinical diagnosis, degree of interstitial fibrosis, immunosuppressants use, need for dialysis and clinical outcome.

Results: The age average was 34.9 ± 16.2 years-old, a predominance of male patients (51.3%). Major glomerular syndromes were: nephrotic syndrome (41.6%) and the rapidly- progressive glomerulonephritis (35.4%). Among primary glomerulopathies focal glomerulosclerosis (26.8%) followed by IgA nephropathy (25%) were predominant; and among the most prevalent secondary glomerulopathies we had lupus nephritis (50%) and diffuse exudative proliferative glomerulonephritis (34.2%).The majority of the patients used immuno-suppressants (68.1%) and almost one third of them (29.2%) needed dialysis during their hospitalization. Progressed to chronic dialysis therapy 13.3% of the patients and 10.6% died.

Conclusion: This study may contribute to better epidemiological understanding of glomerular diseases in the Federal District, guiding the adoption of public policies aiming the quick clinical treatment of such diseases

J Bras Nefrol. 2010 Jul-Sep;32(3):249-56.

Childhood idiopathic steroid resistant nephrotic syndrome in Southwestern Nigeria.

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Clinical charts of 23 Nigerian children diagnosed with idiopathic steroid resistant nephrotic syndrome (iSRNS) between January 2001 and December 2007 were retrospectively reviewed to determine their clinicopathologic characteristics and outcome. iSRNS (54.8%) was primary in 19 patients (83%) and secondary in four (17%). The mean age at diagnosis was 8.3 ± 3.5 years (2.1-13 years). Histopathology revealed membranoproliferative glomerulonephritis (MPGN) in 43.5%, focal and segmental glomerulosclerosis (FSGS) in 39.1% and mesangial proliferative glomerulonephritis in 8.7% of the patients while minimal change disease (MCD) and membranous nephropathy accounted for 4.35% each. Routine treatment protocol comprised pulse intravenous (i.v.) cylophosphamide infusion and i.v. dexamethasone lisinopril or spironolactone. Cumulative Complete Remission (CR) rate was 57.12%. The overall median time to CR from start of steroid sparing agents in 12/21 treated patients was 4.5 weeks. CR was better achieved in MPGN than FSGS (P = 0.0186). Five patients had eight relapses with the overall median relapse-free duration being four months. Cumulative renal survival at 36 months was 41.8%. The median follow-up duration was eight months. Our study revealed that there was a high prevalence of iSRNS and preponderance of non-MCD lesions, with MPGN and FSGS being the major morphologic lesions. The outcome with steroid and cyclophosphamide-based treatment for iSRNS was further enhanced with addition of either lisinopril or spironolactone

Saudi J Kidney Dis Transpl. 2010 Sep;21(5):979-90.

Beneficial effect of all-trans retinoic acid (ATRA) on glomerulosclerosis rats via the down-regulation of the expression of alpha-smooth muscle actin: a comparative study between ATRA and benazepril.

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Although ATRA is a potent renoprotective agent, relatively little is known regarding the mechanisms of its action. The present study was designed to further elucidate the mechanisms of ATRA's action to GS rats and compare that with the beneficial effect of benazepril. Male SD rats weighting 160 to 200g were used in this study. GS was induced by unilateral nephrectomy and intravenous injection of adriamycin (6mg/kg). They were divided randomly 20 ones per group into GS group, GS treated with ATRA (20mg/kg/day) group, and GS treated with benazepril (10mg/kg/day) group. The other 20 ones were taken as sham-operation group, injected normal saline into caudal vein. 12weeks later, all rats were subjected to sacrifice. As expected, the GS group exhibited significant lower serum TP and Alb, and higher BUN, Cr and proteinuria than those of the sham group. Administration of ATRA or benazepril did ameliorate these above disorders of biochemical parameters in GS rats. Extensive renal damage was observed in the GS group, such as mononuclear infiltration, mesangial proliferation, focal segment glomerular sclerosis, and tubulointerstitial fibrosis. The pathological changes in both ATRA and benazepril group were alleviated remarkably. Semiquantitative GSI was used to evaluate the degree of GS in all groups. GSI was significantly higher in the GS group than in sham group. GSI decreased from 21.9+/-6.7 in the GS group to 6.9+/-2.8 in the ATRA group and 7.0+/-2.7 in benazepril group respectively. However, no significant difference in GSI between rats treated with ATRA and rats treated with benazepril was found. RT-PCR analysis revealed the renal expression of alpha-SMA mRNA was induced substantially in GS group as compared to sham group, which could be offset completely by ATRA or benazepril administration. However, expression level of alpha-SMA mRNA in GS rats treated with ATRA was identical to that in GS rats treated with benazepril. We also examined immunohistochemical staining for renal alpha-SMA, TGF-beta1, Col IV, and FN in this model. Weak staining was observed in some glomerulus, mesangial cells, and tubular interstitium of sham rats. Staining was markedly enhanced in the majority of glomerulus, mesangial cells, and tubular interstitium of untreated GS rats. Compared with untreated GS animals, intensity and extent of staining for renal alpha-SMA, TGF-beta1, Col IV, and FN were markedly reduced in glomerulus, mesangial cells, and tubular interstitium of GS rats treated with either ATRA or benazepril. However, no significant differences existed between ATRA and benazepril with respect to the glomerular and tubulointerstitial staining scores. Interestingly, our data documented some differences of therapeutic capacities between ATRA and benazepril. In comparison with benazepril, ATRA exerted no improvement in hypoproteinemia, but more significant decrease in serum Cr level in GS rats. The reasons leading to these variations are unclear. Whatever they are, the properties of down-regulate inflammatory/proliferative programs may make ATRA an attractive potential candidate for future therapeutic use in kidney disease

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Kikuchi-Fujimoto disease: an unusual association with acute renal failure.

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Kikuchi-Fujimoto disease, also known as histiocytic necrotizing lymphadenitis of unknown etiopathogenesis, is a self-limited disease which frequently appears as feverish lymphadenomegaly, thus creating the need for differential diagnosis with lymphoma, systemic lupus erythematosus (SLE), infectious mononucleosis, cat-scratch disease, and toxoplasmosis with lymphonodal impairment. However, there are cases in which it may evolve with complications such as aseptic meningitis, cerebellar ataxia, and aseptic myocarditis. We are presenting a case of a 24-year-old man who had an initial picture of arthralgia, evening fever and adenomegaly. Kikuchi disease was diagnosed through lymph node biopsy with immunohistochemistry and evolves with severe systemic manifestations, such as pericarditis with cardiac tamponade, pneumonitis, hepatitis, and acute kidney failure - the latter has not been reported in literature yet. There was significant improvement of the clinical picture with prednisone

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Effect of the ICAM1 and VCAM1 gene polymorphisms on delayed graft function and acute kidney allograft rejection.

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Background: Immunological response following renal transplantation is a result of ischemia and reperfusion injury, which increase ICAM-1 and VCAM-1 endothelial expression. Reports suggest that there are genetic variations in ICAM-1 and VCAM-1 synthesis that can contribute to delayed graft function (DGF) and acute renal rejection after kidney transplantation. The aim of this study was to examine the effect of ICAM1 and VCAM1 gene polymorphisms on the early period after kidney transplantation.

Material/methods: The study enrolled 270 Caucasian renal transplant recipients (166 males, 104 females).

Methods: Genotyping of the rs5498 ICAM1 and the rs1041163 and rs3170794 VCAM1 gene polymorphisms was performed using real-time PCR.

Results: The distribution of genotypes and alleles of the studied polymorphisms in patients with DGF and without DGF showed no statistically significant differences. The risk of acute rejection was significantly higher in patients with the rs5498 ICAM1 GG genotype than in carriers of the AG and AA genotypes (OR 3.01; 95% CI 1.51-6.00, p=0.003).

Conclusions: These results suggest that ICAM1 and VCAM1 gene polymorphisms play a minor role in pathogenesis of DGF. The rs5498 ICAM1 gene polymorphism is associated with increased risk of acute rejection of kidney allografts

Ann Transplant. 2010 Oct-Dec;15(4):15-20.

Announcement

Apply for an ISN Educational Ambassador to Visit Your Center

ISN COMGAN and its Education Committee believe the most effective teaching takes place in face-to-face, hands on settings. ISN is now extending its traditional CME lecture program to offer renal centers in emerging countries the opportunity to invite an established expert (ISN Educational Ambassador) from outside to come to your institution and remain for a period of 1–4 weeks to provide hands on teaching and help to establish new programs that would benefit patient care. Experts may be selected directly by the center, or ISN will match the needs of the center with qualified experts who have volunteered to become educational ambassadors. Costs of travel will be covered by ISN, centers are asked to provide local accommodations.

Tired of reading journals, reviews and textbooks written by distant experts that cannot answer your questions or give advice? Invite the expert to come to you! This new program offers support for a unique opportunity to start new programs and expose your students, residents, fellows and practioners to in depth contact with an established ISN teacher whose experience and expertise matches your needs.

More information and application forms are available at http://www.nature.com /isn/society/outreach/isn_20090.html

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The ISN seeks qualified nephrologists willing to visit a renal center in a developing country for as little as 1–2 weeks to help establish or upgrade a new clinical program (e.g. peritoneal dialysis, management of AKI, pediatric nephrology, renal pathology, many others). Expertise will be matched with needs outlined in applications for assistance from developing country centers. Timing is flexible and negotiable. Both all nephrologists with a hospital or university teaching position and an existing or potential interest in international renal health care are welcomed. Travel costs are paid by ISN, and local accommodations will be provided by host centers.

You can volunteer to visit a developing renal center that needs your help in a part of the world you would not ordinarily see, experience a unique professional opportunity for service and interaction with local providers, make a contribution to improving renal care in the emerging world and promote international understanding and collaboration. If you are someone who sees yourself as a concerned citizen of the global health community, this program is a way to demonstrate that commitment by sharing your expertise where it is most needed.

If you are an emerging center that would like to benefit from the visit of an Educational Ambassador ISN is now also welcoming application requests for training! Applications must be received by May 1st.

More information and application forms are available at http://www.nature. com/isn/society/outreach/isn_20090.html

Renal Physiology for The Clinician

Fluids, electrolytes and acid-base 4-6 May 2011 Sir William Wells Atrium, Royal Free Hospital, London NW3

This course aims to integrate physiological principles with day-to-day clinical practice. It will feature formal, introductory lectures each day, and clinical case-based and interactive discussions with our faculty designed to illustrate and build upon the day's presentations. It is intended for Specialist Registrars in Nephrology, and will also be of interest to more senior General (Internal) Medicine SPRs and SPRs in Intensive Care Medicine. Consultant Nephrologists and General Physicians with an interest in fluid and electrolyte disorders are welcome to attend as a "refresher" course.

Registration Opens 1 December 2010closing Date For Registration 4 April 2011

For registration information, please contact the Course Administrator: Pamela Fong Whitehead UCL Centre for Nephrology Royal Free, Rowland Hill Street, London, NW3 2PF UK Tel: +44 (0)20 7830 2930 Fax: +44 (0)20 7317 8591 **EMAIL:** pf.whitehead@medsch.ucl.ac.uk

Course Directors: Dr Chris Laing and Professor Robert J Unwin

Dialysis 2011

A comprehensive three-day course on dialysis and the management of patients with end stage renal failure

15-17 June 2011Sir William Wells Atrium, Royal Free Hospital, London NW3

The management of end stage renal failure by dialysis forms a central component of all renal units. However, it is often the least intensively taught aspect of nephrology training. This in-depth course is a thorough introduction to dialysis, from basics to new developments. Expert faculty will provide comprehensive coverage of all aspects of dialysis and ESRF through a mixture of informal lectures and workshops. Although the course is aimed at Specialist Registrars in nephrology, it will also be of interest to other health care professionals involved in the management of patients with chronic renal failure.

Registration Opens 1 December 2010closing Date For Registration 13 May 2011

For registration information, please contact the Course Administrator: Pamela Fong Whitehead Tel: +44 (0)20 7830 2930 Fax: +44 (0)20 7317 8591 UCL Centre for Nephrology Royal Free, Rowland Hill Street, London, NW3 2PF UK **EMAIL:** pf.whitehead@medsch. ucl.ac.uk Course Directors Dr Chris Laing Dr Andrew Davenport Professor Ken Farrington Dr David Wheeler Dr Suresh Mathavakkannan