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Research Article

RELATIONSHIP OF CAROTID INTIMA MEDIA THICKNESS WITH SILENT CEREBRAL INFARCTION IN PATIENTS OF TYPE 2 DIABETES NEPHROPATHY

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ABSTRACT

Aim: To evaluate the relationship between carotid intima media thickness (CIMT) and silent cerebral infarction (SCI) and to determine whether CIMT is a predictor of SCI in patients of type 2 diabetic nephropathy.

Methods: A total of 53 patients of known type 2 diabetic mellitus with nephropathy were selected on the basis of fasting and 2-hour post-prandial blood sugar, 24 hrs urinary albumin estimation, blood urea and serum creatinine.

The selected candidates were subjected to MRI brain and carotid B mode ultrasonography to find out the presence of SCI and to find out the CIMT respectively.

Results: Study results shows 17(32.08%) cases had CIMT >0.9mm and majority had CIMT in higher range of 0.7-0.9mm in patients with type 2 diabetic nephropathy. The mean age, BMI, blood pressure (BP), macroalbuminuria, S. lipids, low GFR, duration of diabetes and CIMT were significantly higher in the subject with SCI than in those without it. Multiple logistic analysis indicated that age, BP, and CIMT were found to be significant and independent risk factors of SCI in patients of type 2 diabetic nephropathy.

Conclusion: CIMT is a surrogate and reliable predictor of higher risk of SCI among type 2 diabetic nephropathy patients.

INTRODUCTION

Silent cerebral infarctions (SCIs) are brain parenchymal lesions that posses MRI characteristics of previous infarcts but have not been associated with clinical signs or symptoms of a stroke. It is termed "silent" because it may be completely asymptomatic. It may be a precursor of symptomatic stroke and brain damage that can be associated with vascular dementia and diminished cognitive functions.

Prevalence of SCI ranges from 5.84% to 28%[4]. There is increased association between silent cerebral infarct and diabetic nephropathy. Diabetic Nephropathy (DN) is a microvascular complication in DM contributes maximally to the pool of the patients with chronic kidney disease. It is defined clinically as the presence of persistent proteinuria> 500mg in 24 hours in the setting of diabetes, but this is preceded by lower degrees of proteinuria or "microalbuminuria.". Microalbuminuria is defined as albumin excretion of 30-299 mg/24 hours. Without intervention, diabetic patients with microalbuminuria typically progress to proteinuria and overt diabetic nephropathy. This progression occurs in both type1 and type 2 diabetes. As many as 7% of patients with type 2 diabetes mellitus may already have microalbuminuria at the time they are diagnosed as diabetes**.**

The measurement of intima-media thickness of the common carotid artery (CIMT) by ultrasonography has been recognised as a powerful tool to identify subclinical atherosclerosis.

IMT is defined as distance between blood- intima interface and media-adventitia interface of carotid wall. It is measured by the B mode ultrasound as the distance between two echogenic lines corresponding the interface between lumen/intima and that of media/adventitia.

Ultrasonography guided measurement of intima-media thickness of the common carotid artery (CCA-IMT) has been recognised as a useful tool to identify subclinical

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atherosclerosis. Increased CCA-IMT is associated with patients of stroke in presence of diabetes mellitus[6-7].

Most often silent infarcts come without a symptomatic manifestation and result in a huge physical and psychosocial loss, it is always of interest to weigh the risk of silent infarcts among high risk patients.

MATERIAL AND METHODS

The study was conducted in Department of Medicine SCB Medical College, Cuttack from September 2014 to September 2015.Out of 193 cases of diabetic nephropathy finally 53 cases were taken into study after satisfying all the exclusion/inclusion criteria.

Inclusion criteria

1. Type 2 diabetes mellitus with nephropathy irrespective of duration of disease and of other risk factors (hypertension, hyperlipidaemia, obesity, etc).
2. Age between 30 and 70 years
3. Informed consent of participant.

Exclusion Criteria

Patients having clinical evidence of

1. Cerebrovascular stroke (focal neurological deficit)
2. Past history of cerebrovascular stroke, transient ischaemic attack (TIA).
3. Other neurological disorders like epilepsy, dementia, parkinsonism.etc.
4. Cardiovascular disorder like valvular heart disease, atrial fibrillation, myocardial infarction, primary myocardial disease.

A detailed history and physical examination were recorded, with emphasis on age, gender, duration of diabetes, brachial blood pressure, height, weight, BMI, waist-hip ratio, and fundoscopy. Patients were subjected to the following investigations:

1. Complete blood count
2. FBS and PPBS
3. Glycosylated HbA1c
4. Urine R/M and 24-hr urine albumin
5. ECG
6. Lipid profile
7. Blood urea and serum creatinine
8. Serum sodium and potassium
9. MRI brain (to detect SCI)
10. Ultrasonographic scanning of the carotid arteries (to determine CIMT)

Ultrasonographic scanning of the carotid arteries was performed by the ACUSON 128×P/10 machine. It was measured by a linear probe at 7.5 MHz frequency on a B-mode ultrasound and Color Doppler.

Patients were examined supine with neck extended and probe in anterolateral position. The boundaries between different layers of arterial wall are demonstrated. The boundaries appear in the longitudinal plane as two parallel echogenic lines separated by hypoechogenic central area. While this appearance is seen on both the near and far wall of the artery. The hypoechoeic arterial lumen acts as acoustic windows and allows the double echogenic lines of far wall to be seen more clearly. The first echo along the far wall is derived from the lumen/intimal interface, while the second echogenic line represents the media/adventia interface.

The point of measurement was taken 1 cm proximal to the carotid bulb at the site of maximal thickness. Four reading were taken in each carotid artery and CIMT was calculated from mean of eight measurement. Any focal thickening of 50% greater than surrounding area or intima media ≥2.0mm defined as plaque and was excluded. The ultrasound machine used had a sensitivity range of 0.1 mm i.e each division was equivalent to 0.1 mm.

Resistive Index (RI) and Pulsatility Index (PI) were measured in the common carotid artery using wall filter setting as 50Hz and Doppler frequency 7.5 MHz. The patients were divided into four equal quartiles I, II, III and IV based on increasing thickness of carotid intima media.

MRI was performed by 1.5 Tesla Machine. SCI lesions were defined as high intensity areas identified on T2-weighted image, coinciding with low intensity areas on T1-weighted image, which was ≥ 3 mm and < 20 mm in diameter. The diagnosis was made when a lesion was surrounded by a hyperintense gliotic rim on fluid attenuated inversion recovery images to exclude dilated perivascular space.

The patients were divided into two groups based on the presence of lesions of SCI, Group I (Non-infarct) and Group II (Infarct).

The statistical analysis was done using SPSS (statistical package for social sciences) version 21.0 statistical analysis software. The values were represented in number (%) and mean ± SD. The data was analysed using chi square test to study the association between various factors. Level of significance was estimated with 95% confidence intervals and p value.

Observation

The present study was conducted in the Department of Medicine in SCBMCH, Cuttack from September 2014 to September 2015. After examining 193 patients and excluding as per the criteria given finally 53 patients selected for study.

The patients who were not found having SCI on MRI were assigned Group-I and those with SCI on MRI were assigned Group-II.

Out of 53 cases 21(39.62%) were found to have SCI on MRI scan and 32(60.38%) were not having SCI.

MRI	Frequency	Percent	Cum. Percent	Exact 95% LCL
NO SCI	32	60.38%	60.38%	46.00%
SCI	21	39.62%	100.00%	26.45%
TOTAL	53	100.00%	100.00%	

The mean age of Gr-I and Gr-II were calculated separately. The mean age of Gr-I was 47.0938and that of Gr-II was 64.0952.

The table below shows 16(100%) out of 16 cases in the age range >60 were having SCI, 05(33.3%) out of 15 cases in the age range 51-60 were having SCI, 0(0%) in the age range 41-50 & 31-40 were having SCI with p value of 0.00001.14(45.16%) out of 31 male had SCI whereas 07(31.8%) out of 22 female had SCI.

AGE and SCI

AGE (in yr)	NO SCI	SCI	SCI IN MALE	SCI IN FEMALE	TOTAL
31-40	5	0	0	0	5
41-50	17	0	0	0	17
51-60	10	5	1	4	15
>60	0	16	13	3	16
TOTAL	32	21	14	7	53
Chi-square	Df	Probability			
39.0665	3	0.00001			

BMI and SCI

BMI (kg/m^2)	NO SCI	SCI	TOTAL
<25	23	3	26
25-29.9	8	14	22
>30	1	4	5
TOTAL	32	21	53
Chi-square	df	Probability	
17.2824	2	0.0002	

The above table shows 03(8.69%) out of 26 cases with BMI less than 25 had SCI, 14(63.63%) out of 22 cases with BMI 25-29.9 had SCI, 04(80.0%) out of 05 cases with BMI more than 30 had SCI with p value of 0.0002.

HTN and SCI

	SCI IN MRI		
HTN	NO SCI	SCI	Total
NO	29	13	42
Row %	69.05%	30.95%	100.00%
Col %	90.63%	61.90%	79.25%
YES	3	8	11
Row %	27.27%	72.73%	100.00%
Col %	9.38%	38.10%	20.75%
Total	32	21	53
Row %	60.38%	39.62%	100.00%
Col %	100.00%	100.00%	100.00%

	Point	95% Confidence Interval	
	Estimate	Lower	Upper
PARAMETERS: Odds-based			
Odds Ratio (cross product)	5.9487	1.3549	26.1175 (T)
Odds Ratio (MLE)	5.7309	1.3392	30.5268 (M)
		1.1437	39.0222 (F)
PARAMETERS: Risk-based			
Risk Ratio (RR)	2.5317	0.9445	6.7867 (T)
Risk Difference (RD%)	41.7749	11.9725	71.5773 (T)
STATISTICAL TESTS	Chi-square	1-tailed p	2-tailed p
Chi-square – uncorrected	6.3589		0.011679659
Chi-square - Mantel-Haenszel	6.2389		0.01249754
Chi-square - corrected (Yates)	4.7325		0.029597371
Mid-p exact		0.00868606	
Fisher exact 1-tailed		0.015306768	0.016999475

The above table shows 42 didn't had HTN and 11 had HTN, 08 (72.73%) out of 11 hypertensive had SCI, whereas 13(30.95%) out of 42 without hypertension had SCI with p value 0.008.

Total Cholesterol and SCI

TOTAL CHOLESTEROL (mg/dl)	NO SCI	SCI	TOTAL
<200	28	8	**36**
200-239	4	10	14
>240	0	3	3
TOTAL	32	21	53
Chi-square	Df	Probability	
15.0477	2	0.0005	

The above table shows 8(22.22%) out of 36 cases with total cholesterol level<200mg/dl had SCI, 10(71.4%) out of 14 cases with total cholesterol level 200-239mg/dl had SCI, 3(100%) out of 3 cases with total cholesterol level >240mg/dl had SCI with p value 0.0005.

Triglyceride LEVEL and SCI

TRIGLYCERIDE(mg/dl)	NO SCI	SCI	TOTAL
<150	20	6	26
150-260	12	14	26
>260	0	1	1
TOTAL	32	21	53
Chi-square	Df	Probability	
6.6978	2	0.0351	

The above table shows 06 (23.07%) out of 36 patients in total triglyceride in range <150 mg/dl had SCI, 14(53.84%) out of 26 patients in total triglyceride in range 150-260 mg/dl had SCI, 01(100%) out of 01 patient in total triglyceride range of >260mg/dl had SCI with p value of 0.0351.

HDLc LEVEL and SCI

HDLc(mg/dl)	NO SCI	SCI	TOTAL
<40	0	6	6
40-60	32	12	44
>60	0	3	3
TOTAL	32	21	53
Chi-square	df	Probability	
16.5195	2	0.0003	

The above table shows 12(27.27%) out of 44 cases having HDLc level of 40-60 mg/dl had SCI, 06(100%) out of 06 cases having HDLc level of <40mg/dl had SCI, 03 (100%) out of 03 cases having HDLc level of >60 mg/dl had SCI with p value of 0.0003.

LDLc and SCI

LDLc(mg/dl)	NO SCI	SCI	TOTAL
<100	30	2	32
100-129	0	10	10
130-159	2	7	9
>160	0	2	2
TOTAL	32	21	53
Chi-square	df	Probability	
38.6601	3	0.00001	

ALBUMINURIA and SCI

ALBUMINURIA(mg/24hr)	MRI		Total
	NO SCI	SCI	
<300	27	8	35
Row %	77.14%	22.86%	100.00%
Col %	84.38%	38.10%	66.04%
>300	5	13	18
Row %	27.78%	72.22%	100.00%
Col %	15.63%	61.90%	33.96%
Total	32	21	53
Row %	60.38%	39.62%	100.00%
Col %	100.00%	100.00%	100.00%
	Point Estimate	95% Confidence Interval Lower	Upper
PARAMETERS: Odds-based			
Odds Ratio (cross product)	8.775	2.3949	32.1524 (T)
Odds Ratio (MLE)	8.3433	2.3361	33.6430 (M)
		2.0507	40.1265 (F)
PARAMETERS: Risk-based			
Risk Ratio (RR)	2.7771	1.2904	5.9766 (T)
Risk Difference (RD%)	49.3651	24.4312	74.2989 (T)
STATISTICAL TESTS	Chi-square	1-tailed p	2-tailed p
Chi-square - uncorrected	12.1084		0.000501951
Chi-square - Mantel-Haenszel	11.8799		0.000567417
Chi-square - corrected (Yates)	10.1328		0.001456519
Mid-p exact		0.000386116	
Fisher exact 1-tailed		0.000703197	0.000894346

The above table shows 02 cases (6.25%) out of 32 cases having LDLc level <100mg/dl had SCI, 10 cases (100%) out of 10 cases having LDLc level 100-129 mg/dl had SCI, 07 cases (77.77%) out of 09 cases having LDLc level 130-159 mg/dl had SCI,02 cases (100%) out of 02 cases having LDLc level >160 mg/dl had SCI with p value of 0.00001.

The above table shows 08 cases (22.86%) out of 35 cases having microalbuminuria had SCI, 13 cases (77.22%) out of 18 cases having macroalbuminuria had SCI with p value of 0.000386.

GFR and SCI

GFR (ml/min)	NO SCI	SCI	TOTAL
15-29	0	9	9
30-59	12	11	23
60-90	20	1	21
>90	0	0	0
TOTAL	32	21	53
Chi-square	Df	Probability	
25.0291	2	0.00001	

The above table shows 01case (4.76%) out of 21cases having GFR > 60 ml/min had SCI, 11cases (47.82%) out of 23 cases having GFR 30 -59 ml/min had SCI, 09 cases (100%) out of 09 cases having GFR 15-29 ml/min had SCI with p value of 0.00001.

DURTION OF DM and SCI

DURATION	NO SCI	SCI	Total
<5YR	28(93.33%)	2(6.67%)	30
5-10YR	4(50%)	4(50%)	8
>10YR	0(0%)	15(100%)	15
P value	0.000		

The above table shows 02 cases (6.67%) out of 30 cases with duration of DM < 5 yr had SCI, 04 cases (50%) out of 08 cases with duration of DM 5-10yr had SCI,15 cases (100%) out of 15 cases with duration of DM >10 yr had SCI with p value of 0.000.

CIMT and SCI

CIMT(in mm)	NO SCI	SCI	TOTAL
0.6-0.69	4	0	4
0.7-0.79	15	1	16
0.8-0.9	11	5	16
>0.9	2	15	17
TOTAL	32	21	53
Chi-square	Df	Probability	
27.3357	3	0.00003	

The above table shows 0 cases (0%) out of 04 cases having CIMT range of 0.6-0.69mm had SCI, 01 case (6.25%) out of 16 cases having CIMT range of 0.7-0.79mm had SCI, 05 cases (31.25%) out of 16 cases having CIMT range of 0.8-0.9 mm had SCI, 15 cases (88.23%) out of 17 cases having CIMT of >0.9 mm had SCI with p value of 0.00003.

DURATION OF DM and CIMT

CIMT in mm	<5YR	>10YR	5-10 YR	TOTAL
<0.9	27(75%)	4(11.11%)	5(13.89%)	36(100%)
>0.9	3(17.65%)	11(64.71%)	3(17.65%)	17(100%)
TOTAL	30(56.60%)	15(28.30%)	8(15.09%)	53(100%)
Chi-square	Df	Probability		
18.5377	2	0.0001		

The above table shows 27 cases (75%) out of 36 cases having CIMT <0.9 mm were having duration of DM <5 yr, 05 cases(13.89%) out of 36 cases having CIMT <0.9 mm were having duration of DM 5-10 yr,04 cases(11.11%) out of 36 cases having CIMT <0.9 mm were having duration of DM >10 yr.

03 cases (17.6%) out of 17 cases having CIMT >0.9 mm were having duration of DM <5 yr,03 cases(17.65%) out of 17 cases having CIMT >0.9 mm were having duration of DM 5-10 yr, 11cases(64.71%) out of 17 cases having CIMT >0.9 mm were having duration of DM >10yr.The p value was 0.0001.

DISCUSSION

Out of 53 cases, SCI in MRI was found in 21(39%) cases of which 16 were of age >60 yr and 5 in the age range of 51-60 yr. Similar observation was made by D.Kumar *et al*. This indicate the need of screening of all diabetic nephropathy patients of age >50yr. The mean age of patients with SCI was 64.09. Rycharo *et al* had studied 246 patients and mean age of patient with SCI was 69±8 yr. D.Kumar *et al* found SCI in 37.5% of patients with diabetic nephropathy which is similar to our observation. But Nomura *et al* found 60.4% of DM having SCI. This may be due to more elderly patients taken in their study. In a study by Vermeer *et al* only 12% had SCI on MRI.

In our study HTN was not a common association with diabetic nephropathy. Out of 53 cases only 11 had HTN of which 8(72.73%) had SCI indicating SCI is a common finding related to HTN. Similar observation made by D.Kumar *et al*.

Nomura *et al* observed the systolic BP was higher in subject with SCI. Ricci *et al* and Vermeer *et al* also showed higher incidence of SCI with HTN. HTN in our study was more in elderly age group indicating that age and HTN increase the incidence of SCI.

Out of 53 cases 36 had normal cholesterol level (<200mg/dl), only 3 cases had cholesterol level >240mg/dl. D.kumar *et al* also had similar observation of 3.8% patients with >240mg/dl. In our study 13(24.5%) patients with SCI had borderline or severe hypercholesterolemia. Uzu *et al* found no association between cholesterol and SCI. To the best of our knowledge most of the study had not found any association between cholesterol and SCI.

Out of 53 cases only 21 (39.6%) had SCI and only 1 case had triglyceride level >260 mg/dl. Neither D.Kumar, Nomura *et al* nor Uzu *et al* found any significant association between hypertriglyceridemia and SCI.

HDLc level was normal in 44 cases (83.01%), 6 had <40 mg/dl and 3 had >60mg/dl.12 cases (57.14%) of SCI had normal HDLc level. Our study matches with that of D.Kumar *et al*., Nomura *et al* and Uzu *et al*.

In our study 32(60.3%) cases had normal LDLc level. Majority patients with SCI had normal or borderline increase in LDLc level. D.Kumar *et al* found significant difference between SCI and NON SCI group. But Nomura *et al* didn't found any such association. But it needs a larger study for further substantiation.

Majority of cases 35(66.03%) had micro albuminuria and 8 (15.09%) cases had SCI. None of the patient had urinary albuminuria in nephrotic range (>3.5gm/24 hr). There was significant difference between SCI and NON SCI group. Observation by D.Kumar *et al*, Uzu *et al* showed significant

association of macroalbuminuria with SCI.

In our study all patients had GFR <90ml/min. Majority of cases with SCI had GFR between 15-59 ml/min indicating that SCI was significantly more common in low GFR. D.Kumar *et al*, Uzu *et al* and Kobayasi *et al* also had similar observation. This may be due to low perfusion of cerebral vessels in low GFR which predisposes to SCI.

From 53 cases, 15 cases (100%) out of 15 cases with duration of DM >10 yr had SCI and 4(50%) cases out of 8 cases with duration of DM between 5-10 yr had SCI. This indicate long duration of DM directly proportional to the presence of SCI. Similar observation made by D.Kumar *et al*. Median duration for development of angiopathy in DM is around 10 yrs. In this study we have explored the relationship of target organ damage and duration of DM. The proportional risk between duration <5 yr to >5 yr is 2/19=10.52.

Recently CIMT has been emerged as predictive marker for risk of SCI. It is an easy, noninvasive and cheap method to asses atherosclerosis and its impact on cardiovascular morbidity. Since not much literature is available related to risk of SCI with DM nephropathy we explored the incidence of SCI among different range of CIMT in increasing order e.g 0.6-0.69mm,0.7-0.79mm,0.8-0.89mm, >0.9mm. It was observed that except 1 case, all with SCI had CIMT in higher range whereas patients without SCI had CIMT in lower range. Nomura *et al* and D.Kumar *et al* observed in patients with SCI that the mean CIMT was significantly higher than those without SCI. However Inoue *et al* found no significant independent risk of CIMT in prediction of SCI. Eguchi *et al* too supported these finding while Johnsen *et al* were of view that CIMT was associated with hypertensive vascular disease. Kozera *et al* indicated that presence of diabetic nephropathy but not increased CIMT can be regarded as indicator of cerebral angiopathy. The higher incidence of SCI among those with higher CIMT proves exsistence of such relationship of nephropathy with CIMT. We have not proposed any particular cut off value as it would be inappropriate to reach a conclusive cut off marker without asserting the variable profile of individual including the duration of disease, associated illness, level of complication, age, gender, lipid profile and level of glycemic control.

In our study it was observed that the patients with higher CIMT value were most with higher duration of disease. We also found a significant association between SCI and duration of DM. Thus a common association of CIMT with duration of DM could be observed. Similarly on seeking the association between infarction, urinary albumin levels and CIMT, it was observed that majority of patients with higher CIMT had SCI and/or macroalbuminuria. But all these finding needs further exploration in a large study.

CONCLUSION

SCI exist more frequently in subjects with DM than those without it and several risk factor have been identified including age, Hypertension, Diabetes Mellitus, and carotid atherosclerosis. There is steep rise of SCI in increased age. Hypertension was not a very essential criteria for presence of SCI. Hypertriglyceridemia, hypercholesterolemia is increasingly associated with incidence of SCI. Macroalbuminuria is more commonly associated with SCI than microalbuminaria. CIMT is independently associated with diabetic nephropathy and reliable predictor of higher risk of SCI in diabetic nephropathy. Therefore patients with higher CIMT should be screened for SCI which is a risk factor for stroke and dementia. More intensive preventive measure including active detection of SCI and strict treatment of multiple co risk factor especially Hypertension is needed. However a large number of patients with prolonged study is needed to throw more light on this aspect.

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