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Correlation of High Sensitivity C-Reactive Protein and Carotid Intimal Medial Thickness in Patients with Ischemic Stroke

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ABSTRACT

Background: Stroke is defined as an acute neurological deficit lasting more than 24 hours and caused by cerebrovascular etiology. It is further subdivided into ischemic stroke (caused by vascular occlusion or stenosis) and hemorrhagic stroke (caused by vascular rupture, resulting in intra parenchymal and/or subarachnoid hemorrhage).

Aim: Our study was designed to study the levels of High sensitivity C reactive protein (HsCRP), which is a marker for chronic inflammation, in patients with ischemic stroke due to large vessel atherosclerosis and correlate it with carotid intimal medial thickness.

Methodology: This study was done in Department of Medicine and Department of neurology in KGMU, Lucknow, between August 2016 to July 2017, and 100 patients with ischemic stroke presenting within 72 hours of onset, who met the inclusion and exclusion criteria. HsCRP was evaluated and CIMT was subsequently measured.

Result: HsCRP is significantly increased in patients with ischemic stroke. The carotid intimal medial thickness which is a proven marker for large artery atherosclerosis is also increased in patients with ischemic stroke and it is also significantly correlated with the levels of HsCRP. The levels of HsCRP is elevated more in patients with Diabetes Mellitus and Hypertension.

Conclusion: HsCRP levels are increased in patients of ischemic stroke suggesting that it could be used as a marker for the prediction of the ischemic stroke when combined with CIMT, together these two could predict the ischemic stroke.

Key Words: High sensitivity C reactive protein, Subarachnoid hemorrhage, Carotid artery, Ischemic stroke

INTRODUCTION

Stroke is defined as an acute neurological deficit lasting more than 24 hours and caused by cerebrovascular etiology. It is further subdivided into ischemic stroke (caused by vascular occlusion or stenosis) and hemorrhagic stroke (caused by vascular rupture, resulting in intra parenchymal and/or subarachnoid hemorrhage).[1]

Progressive atheromatous plaque in the carotid artery usually at the bifurcation results in gradual narrowing of the carotid artery. This process can be symptomatic but may lead to ischemic stroke or transient ischemic attack from embolization, thrombosis, orhemodynamic compromise. To measure the extent of atherosclerotic disease we measure Carotid Intimal medial thickness, which is a proven marker for the atherosclerotic large vessel disease.

High sensitivity C reactive protein is a marker of inflammation has gained importance in the recent times as a pro-inflammatory marker. Various studies have shown that HsCRP could be used as an early marker of atherosclerosis. [2]

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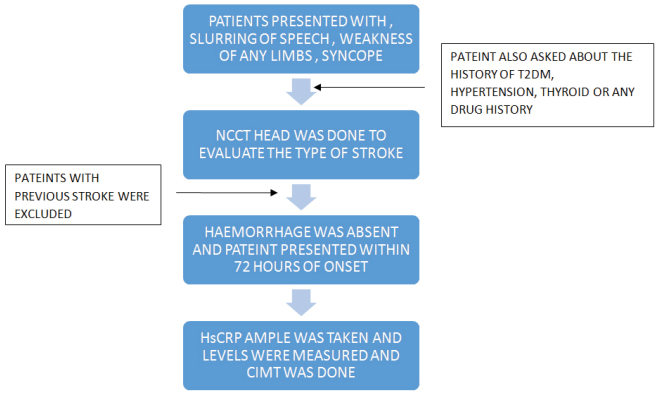
MATERIALS AND METHODS

Our study was designed to study the levels of High sensitivity C reactive protein (HsCRP), in patients with ischemic stroke due to large vessel atherosclerosis and correlate it with carotid intimal medial thickness. This study was done in Department of Medicine and Department of neurology in KGMU, Lucknow. This study was conducted in between August 2016 to July 2017, and 100 patients with ischemic stroke presenting within 72 hours of onset, who met the inclusion and exclusion criteria.

A standardized questionnaire was given that includes Name, Age, Sex or any family h/o diabetes or hypertension. Any concurrent history of hypertension, thyroid, or Diabetes mellitus was asked. All participants were asked to give informed written consent after explaining the study. The study was approved by the Ethical committee, KGMU, Lucknow. After making the diagnosis of ischemic stroke the patients were subjected to blood investigations for which approximately 5 ml venous blood sample was drawn and HsCRP levels were analyzed in those samples along with complete blood picture, S. Urea, S creatinine. Along with that if the patient wasn't in the fasting state, then sample for fasting lipid profile was also drawn after 8 hours of fasting.



Patients Particulars	
Name	
Age	
Sex	
Address	
Present History	
Family History of Such Complaints	
Drug History (Statins)	
Associated Comorbid Conditions (T2DM, Hypertension)	
Investigations Ncct Head, Fasting Lipid Profile, HSCRP, HBA1C, CIMT	

RESULTS

Statistical analysis was done by the mean, median, standard deviation and Student t-test and the significance was analyzed by the "p-value".

The mean age of the study population was 60.98±7.35. The Male-female ratio was 2.81.

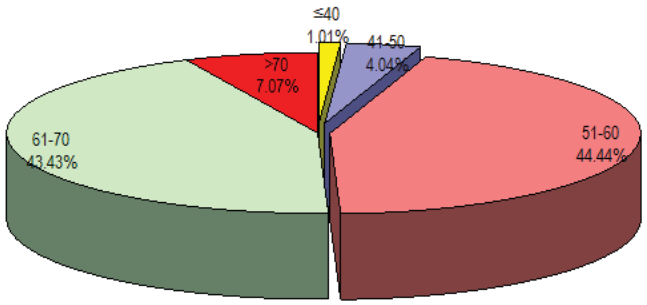


Figure 1: Showing the Age Distribution of the Pateints.

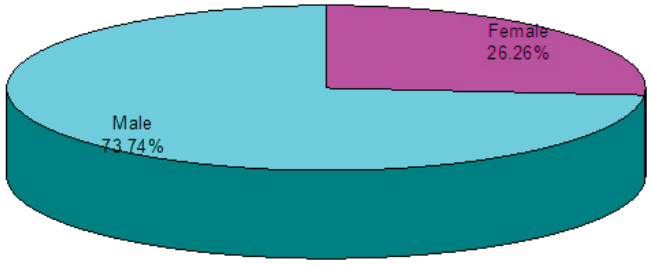


Figure 2: Sex Distribution of the Patients.

Table 1: The levels of CIMT and HsCRP in the study population

Variable	No. of patients	Min.	Max.	Median	Mean	S.D.
CIMT (mm)	99	0.4	1.2	0.70	0.708	0.128
HsCRP (mg/L)	99	1	13	3.20	4.34	2.35

CIMT of patients enrolled in the study ranged from 0.4-1.2 mm, median CIMT among our patients was 0.70 mm while mean CIMT levels were 0.708±0.128 mm.

Table 2: Association of Glycemic Control & Hypertension with CIMT and HsCRP

	No Risk (n=58)		Single Risk (n=33)		Double risk (n=8)		ANOVA	
	Mean	SD	Mean	SD	Mean	SD	F	'p'
CIMT (mm)	0.655	0.082	0.758	0.115	0.888	0.203	21.994	<0.001
HsCRP (mg/L)	2.83	0.98	5.83	1.47	9.20	1.89	127.384	<0.001

	No risk vs. Single risk			No risk vs. double risk			Single risk vs. Double risk		
	Mean	SE	p	Mean	SE	p	Mean	SE	P
CIMT	-0.102	0.023	<0.001	-0.232	0.040	<0.001	0.130	0.042	0.007
HsCRP	-3.00	0.27	<0.001	-6.37	0.47	<0.001	-3.37	0.49	<0.001

Table 3:Correlation of HsCRP and CIMT with Variables used in the study

Variables	HsCRP				CIMT			
	Correlation coeff (r)	Level of correlation	p	Sig	Correlation coeff (r)	Level of correlation	P	Sig
SBP	0.577	Moderate	<0.001	Sig	0.353	Mild	<0.001	Sig
DBP	0.335	Mild	0.001	Sig	0.359	Mild	<0.001	Sig
HbA1C	0.816	Strong	<0.001	Sig	0.569	Moderate	<0.001	Sig
Cholesterol	0.626	Moderate	<0.001	Sig	0.387	Mild	<0.001	Sig
HsCRP	–	–	–	–	0.680	Moderate	<0.001	Sig
Hb	0.103	Weak	0.311	NS	0.043	Weak	0.669	NS
HDL	-0.073	Weak	0.474	NS	-0.085	Weak	0.403	NS

The mean values of HsCRP and CIMT were increased in patients with ischemic stroke and are significantly correlated with each other. The factors which are not significant are Hb and HDL.

DISCUSSION

HsCRP which is a marker of chronic inflammation is significantly increased in patients with ischemic stroke. The carotid intimal medial thickness which is a proven marker for large artery atherosclerosis is also increased in patients with ischemic stroke and it is also significantly correlated with the levels of HsCRP. The levels of HsCRP is elevated more in patients with Diabetes Mellitus and Hypertension. As compared to the levels in patients without hypertension or diabetes mellitus, the patients having both or either one of them has increased levels. Moreover, the patients having uncontrolled Diabetes Mellitus i.e. HbA1C > 9.0 are having even more levels. The levels of HsCRP are not significantly correlated with HDL and Hb.

The results were in concordance with the following studies, **Dibyaratna Patgiri1 et al** [3] HsCRP is an acute phase reactant whose concentration rises in stroke as well as in those at risk. The rise may be identified even before the appearance of risk factors. Hence, hsCRP may be useful as a predictive and diagnostic marker in stroke. **Liu et al** [4]. We found that higher hs-CRP concentrations were associated with a higher risk of IS, particularly for non-fatal stroke, male and hypertensive subjects. In contrast, we did not observe significant associations between hs-CRP and ICH/ SAH

CONCLUSION

HsCRP levels are increased in patientswith ischemic stroke suggesting that it could be used as a marker for the prediction of the ischemic stroke when combined with CIMT, together these two could predict the ischemic stroke. As we had a small sample size, more studies need to be done to predict the role of HsCRP as a marker of ischemic stroke.

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