



Correlation of Bilirubin Levels in Patients with and Without Cerebrovascular Accident

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Abstract

Stroke is a clinical syndrome of rapid onset of focal cerebral deficit, lasting more than 24 hours or leading to death, with no apparent cause other than a vascular. Bilirubin, a breakdown product of normal heme catabolism is found that in acute phase of cerebrovascular because of its antioxidant property prevents the oxidation process leading to damage of brain. In this study we correlate the total Bilirubin levels in patients with cerebrovascular accident and age and sex matched controls.

Keywords: Bilirubin , Cerebro-vascular accident, Stroke.

Introduction

Stroke is a clinical syndrome of rapid onset of focal cerebral deficit, lasting more than 24 hours or leading to death, with no apparent cause other than a vascular. Stroke is one of the leading cause of disability in developed and increasing trends in developing countries .In global perspective, stroke is the 2nd most prevalent source of death³. According to WHO estimates, 5.5 million people died of stroke in 2002, and roughly 20% of these deaths occurred in south Asia. Bilirubin, a breakdown product of normal heme catabolism is found that in acute phase of cerebrovascular accident if there is an elevated level of bilirubin is significant for prognosis of patient. Bilirubin because of its antioxidant property prevents the

oxidation process leading to damage of brain in acute phase. It is found that in acute phase of cerebrovascular accident if there is an elevated level of bilirubin is significant for prognosis of patient. Bilirubin because of its antioxidant property prevents the oxidation process leading to damage of brain in acute phase. In this study we correlate the total bilirubin levels in patients with cerebrovascular accident and age and sex matched controls.

Aims & Objectives

1. Oxidative injury is an important cause of the neurologic lesion in stroke.
2. Serum bilirubin -a natural antioxidant that may affect the prognosis of stroke.

3. To correlate the total bilirubin levels in patients with and without CVA.

Materials & Methods

Setting: Hospital based

Study design: Cross section study which was conducted in patients with acute CVA and general population admitted in south Indian rural based hospital.

Study participants: Patients with well-documented (clinical presentation and computed tomography-brain) first or recurrent acute stroke occurring within the 7 days before admission were included in the study

Study duration: 1 year

Sample size: 48 (24-cases, 24-age matched controls d = relative precision of 20%)

Inclusion criteria: Patients with well-documented (clinical presentation and computed tomography of the brain) first or recurrent acute stroke occurring within the 7 days before admission were included in the study.

Exclusion criteria: Patients who have contracted aspiration pneumonia, Transient ischemic attack, any central nervous system disease such as dementia, tumour, trauma or hydrocephalus, history of depression or other psychiatric disorders, liver disease or physically unfit for interview were excluded from the study.

After obtaining a written informed consent, a clinical examination of the CNS was performed and the case details were recorded on a special proforma. Patients will be examined for parameters RFT, LFT and CT-brain.

Data entry and analysis

The data obtained were entered in the MS excel sheet and data analysis was done using SPSS v24.0

Results

In this hospital base cross sectional study totally 48 patients were taken after ethical committee approval and participation consent from all study population. Patients with cerebrovascular

accident, brain imaging and blood samples were collected on the day of admission in the hospital. Age-related controls(OPD, In patient) were taken and their blood samples also taken for the study .The study population is sub-divided based on age grouping age of 30-39 years 5 (S-2,C-3) , 40-49 years 6 (S-3,C-3) , 50-59 years 13 (S-7,C-6) , 60-69 years 13(S-7,C-6) , 70-79 years 9 (S-4,C-5) , 80-89 years 2 (S-1,C-1). As we already know that cerebrovascular disease is age related it is seen in age groups 40-79 years, as our life expectancy is 68.35 years less number of cases are seen in 80-89.

Table 1

Age group	Group		Total
	Study	Control	
30-39	2(8.3%)	3(12.5%)	5(10.4%)
40-49	3(12.5%)	3(12.5%)	6(12.5%)
50-59	7(29.2%)	6(25.0%)	13(27.1%)
60-69	7(29.2%)	6(25.0%)	13(27.1%)
70-79	4(16.7%)	5(20.8%)	9(18.8%)
80-89	1(4.2%)	1(4.2%)	2(4.2%)
Total	24	24	48
FE test	p = 1.00(NS)		

*p<0.05 statistically significant, p>0.05 Non significant, NS

Table 2

Sex	Group		Total
	Study	Control	
Male	18(75.0%)	15(62.5%)	33(68.8%)
Female	6(25.0%)	9(37.5%)	15(31.3%)
Total	24	24	48
Chi square value(1) = 0.87, p= 0.35(NS)			

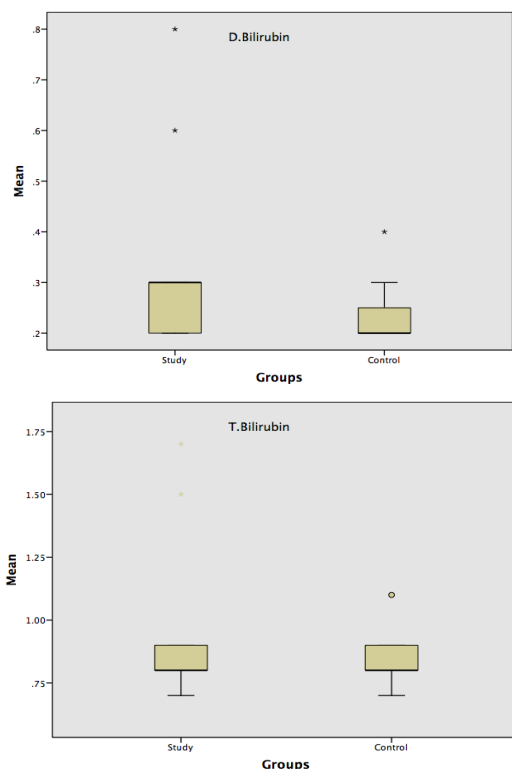
Chi square test *p<0.05 statistically significant, p>0.05 Non significant, NS

In view of sex based category it is more seen in male population in our study of 33(S-18, C-15) and female population 15 (S-6, C-9).

Table 3

	Group	N	Mean (SD)	Range	Median (Q1-Q3)	U Statistic	p-value
T Bilirubin	Study	24	0.88 (0.23)	0.70 - 1.70	0.80 (0.80 - 0.90)	283.00	0.91 (NS)
	Control	24	0.83 (0.09)	0.70 - 1.10	0.80 (0.80 - 0.90)		
D Bilirubin	Study	24	0.29 (0.14)	0.20 - 0.80	0.30 (0.20 - 0.30)	203.50	0.04*
	Control	24	0.23 (0.06)	0.20 - 0.40	0.20 (0.20 - 0.28)		

Mann Whitney U test *p<0.05 statistically significant, p>0.05 Non significant, NS



In our study the variable total bilirubin shows a mean of 0.88 with a SD of 0.23 (R=0.7 to 1.7) mean of 0.83 with a SD of 0.09 (R=0.7 to 1.1) in study and control group respectively. The variable direct bilirubin shows a mean of 0.29 with SD of 0.14 (R=0.2 to 0.8), mean of 0.23 with a SD of 0.06 (R=0.2 to 0.4) in study and control group respectively. Using Mann Whitney U test statistic is 283 & 203.5 for total and direct bilirubin with p value of 0.91 & 0.04 respectively, which shows significant statistically for direct bilirubin.

Discussion

As already known bilirubin which is the end product of heme metabolism. In an event of oxidative stress the expression of heme-oxygenase 1 will be increased. Bilirubin which has anti-oxidative stress properties was the end product of the increased heme-oxygenase expression^(4,5,6,8,11). In systemic inflammatory conditions by oxidative stress, a highly inducible protein is activated, which end products are bilirubin and carbon monoxide^(4,6,17,18).

The neuronal cells are vulnerable to oxidative stress as brain is rich in poly unsaturated fatty acids. Bilirubin thus released from the heme-

oxygenase 1 exerts cyto-protective effect^(1,2,3,5,8). Carbon monoxide another end product of heme-oxygenase 1 causes vasodilatation⁽¹⁸⁾.

Physiological levels of the bilirubin suppress the oxidation of lipids in the membranes to a greater extent than that of alpha-tocopherol. At physiological concentrations direct OH and DPPH scavenging and potent anti-oxidant activities of bilirubin are noted⁽²¹⁾. Evidence of the co-localization of HO-1 and bilirubin IX α in foam cells, suggesting a role of HO-1 induction and bilirubin in the modulation of macrophage activation in atherosclerosis^(6,7,8).

Various studies among individuals with general medical conditions including hepatic disease have suggested that Dbil levels may be of better prognostic value than Tbil levels^(15,20).

Conclusion

Bilirubin which has anti-oxidative stress properties was the end product of the increased heme-oxygenase expression.

Bilirubin thus released from the heme-oxygenase 1 exerts cyto-protective effect.

Carbonmonoxide other end product of heme-oxygenase 1 causes vasodilatation.

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