



LIPID PEROXIDATION, BLOOD PRESSURE AND LIPID PROFILE STATUS OF DIABETIC ISCHAEMIC STROKE SUBJECTS

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ABSTRACT

Acute Ischaemic Stroke (AIS) is characterized by elevated level of oxidative stress indices, declined antioxidant defences and dyslipidaemia. Increased oxidative stress is thought to play a role in the development of AIS and its attendant complications. In the current study, fasting blood glucose, malondialdehyde, blood pressure, total cholesterol, triacylglycerol, very low density lipoprotein cholesterol, low density lipoprotein cholesterol, high density lipoprotein cholesterol and atherogenic index levels were estimated in 37 diabetic ischaemic stroke (DIS) subjects admitted in Neuro Medical ward of Usmanu Danfodiyo University Teaching Hospital, Sokoto, Nigeria and the

results compared with 42 non-diabetic ischaemic stroke (NDIS) subjects of comparable age and social status. The results suggested significant ($P < 0.05$) increase levels of FBS, MDA, and BP in diabetic ischaemic stroke subjects compared with non-diabetic ischaemic stroke subjects. It also revealed non-significant ($P > 0.05$) increase levels of TC, TAG, HDL-C, LDL-C, and VLDL-C and decreased AIX in DIS compared with NDIS subjects. The findings suggest that diabetic ischaemic stroke subjects had more oxidative stress and dyslipidaemia associated with acute ischaemic stroke.

KEYWORDS: Acute ischaemic stroke, lipid profile and oxidative stress.

INTRODUCTION

Diabetic ischaemic stroke is characterized by sudden loss of blood circulation to an area of the brain, resulting in a corresponding loss of neurologic function.^[1] It is also defined as a

sudden death of brain cell caused by a lack of supply in oxygen to the brain.^[2] Most strokes occur as a consequence of atherosclerosis, hypertension, or both.^[2] The brain undergoes neurodegeneration, when excess free radicals overwhelm the antioxidative defence system during senescence, head injury or neurotoxic conditions.^[3] The deleterious effects of the excessive production of free radicals or reactive oxygen species can be prevented by body's antioxidant defence mechanism which may include antioxidant enzymes such as superoxide dismutase, glutathione peroxidase and catalase and antioxidant vitamins (A, C, and E) as well as antioxidant minerals (Zn, Cu, Cr, Mn, and Se).^[4] Antioxidant defence mechanism help in neutralizing the effect of free radicals by mopping them off which further limit diabetic ischaemic stroke disease and its attending complications.^[4]

Ischaemic strokes are under chronic oxidative stress induced by excessive production of free radicals. This may overwhelm the existing antioxidant defences and deplete antioxidants including enzymes and minerals.^[5] Ischaemic stroke complications are becoming rampant and is highly life threatening most especially in elderly patients.^[6] It is expected that this study will stimulate interests, discussion and further studies on the lipid peroxidation and lipid profile vis-à-vis complications of diabetic ischaemic stroke. In this study blood pressure, serum fasting blood sugar, malondialdehyde, as well as lipid profile were determined in diabetic ischaemic strokes and the results compared with non-diabetic ischaemic strokes of comparable socio-economic status.

MATERIALS AND METHODS

Participants: The subjects employed for this study were 37 diabetic ischaemic stroke and 42 non-diabetic ischaemic stroke patients presented within 72 hours of symptom onset of both sexes who were admitted in Neuro Medical ward of the Usmanu Danfodiyo University Teaching Hospital, Sokoto, Nigeria. The consents of all the participants were sought for and obtained. Ethical committee approval was also obtained for the study.

Blood samples: Blood samples were collected by venopuncture and delivered into clean dry tubes and allowed to clot at room temperature. The samples were centrifuged at 3000 rpm for 5 minutes using bench top centrifuge and the serum separated and kept in labeled sample bottles at (-20°C) until required. Glucose was estimated immediately.

Reagents: All chemicals and reagents were of analytical grade. MDA assay kit was purchased from Enzo's Life Science United Kingdom while glucose, TC, TAG, and HDL-C assay kits were obtained from Randox Laboratory Limited, Switzerland.

Analytical Methods: Serum glucose level was determined by method of Trinder,^[7] MDA was determined by method of Chandra *et al.*^[8], BP was determined by method of Pickering *et al.*^[9], TC was determined by method of Allain *et al.*^[10], TAG was determined by method of Trinder,^[11] HDL-C was determined by method of Burstein,^[12] LDL-C was calculated using Friedewald *et al.*^[13], VLDL-C was calculated using Friedewald *et al.*^[13], and AIX was calculated using Ranjna.^[14]

Data Analysis: All results were presented as mean±SD. Levels of significance was assessed using Student t-test. Turkey-Kramer Multiple Comparison test (In stat 3 Software, San Diego, USA). Significant difference was taken at 5% ($P < 0.05$)

RESULTS

Table 1: Serum Fasting Blood Glucose, Blood Pressure and Malondialdehyde of Diabetic Ischaemic Stroke Subjects.

Parameter	Diabetic Ischaemic Stroke (n=37)	Non-diabetic Ischaemic Stroke (n=42)
FBG (mmol/L)	9.96±1.50 ^a	5.45±0.71 ^b
Systolic blood pressure (mm Hg)	160.33±13.62 ^a	157.52±22.16 ^b
Diastolic blood pressure (mm Hg)	89.65±8.74 ^a	96.57±13.17 ^b
Malondialdehyde (nmol/ml)	314.70±29.52 ^a	276.87±45.57 ^b

Values are expressed as mean±SD. Different superscripts on horizontal column indicated significant difference ($P < 0.05$), while superscripts bearing the same letters indicate no significant difference ($P > 0.05$). n= number of participants.

Table 2: Serum Lipid Profile of Diabetic Ischaemic Stroke Subjects

Parameter	Diabetic Ischaemic Stroke Subjects (n=37)	Non-diabetic Ischaemic Stroke Subjects (n=42)
Total cholesterol (mmol/L)	7.64±0.46 ^a	6.72±0.68 ^a
Triacylglycerol (mmol/L)	3.31±0.37 ^a	2.69±0.40 ^a
HDL-Cholesterol (mmol/L)	2.33±0.31 ^a	2.01±0.35 ^a
LDL-Cholesterol (mmol/L)	3.76±0.32 ^a	3.66±0.72 ^a
VLDL-Cholesterol (mmol/L)	1.48±0.18 ^a	1.21±0.18 ^a
Atherogenic index	1.96±0.30 ^a	2.14±0.47 ^a

Values are expressed as mean±SD. Superscripts bearing the same letters indicate no significant difference ($P > 0.05$). n= number of participants.

DISCUSSION

Diabetic ischaemic stroke (DIS) is characterized by elevated level of oxidative stress indices, declined antioxidant defences and dyslipidaemia.^[15] Increased oxidative stress is thought to

play a role in the development of DIS and its attending complications.^[5] Oxidative stress and thrombosis are suggested to be potential contributor to the development of acute ischaemic stroke and the associated complications.^[16] This may be connected to the fact that the antioxidant status may be inadequate in diabetic ischaemic stroke subjects. The metabolic significance of the evaluation of oxidative stress and lipid profile status in diabetic ischaemic stroke is therefore of paramount importance.

The results of the current study indicated that serum fasting blood glucose, malondialdehyde and blood pressure of the diabetic ischaemic stroke subjects were significantly ($P < 0.05$) higher than the values obtained for the non-diabetic ischaemic stroke subjects. The results further revealed no significant ($P > 0.05$) increase levels of lipid profile in diabetic ischaemic stroke subjects compared with values obtained for the non-diabetic ischaemic stroke subjects.

The increased MDA in diabetic ischaemic stroke is due to an altered intracellular ratio between free radicals and antioxidant capacity which leads to oxidative stress, which in turn stimulate cardioacceleratory center, activating the sympathetic nervous system and increasing both heart rate and stroke volume.^[17] The increased FBS is connected to hyperglycaemic related reactive oxygen species which attenuates antioxidant mechanism, creating a state of oxidative stress. The β -cells are sensitive to oxidative stress because their intracellular antioxidant defence mechanisms are weak.^[18]

The increased levels of TC, TAG, LDC-C, VLDL-C, HDL-C and AIX in DIS compared with NDIS subjects are in line previous studies of.^[19,20] The increased TC, TAG, LDL-C, VDLC-C and AIX in DIS subjects is partly explained by clustering of risk factors such as hypertension and dyslipidaemia and by direct consequences of hyperglycaemia and glycation, which favours the oxidation and modification of LDL- particles, accelerating the development of atherosclerosis.^[21] Oxidized LDL-C is thought to play a role in the development of arterial plaque.^[22]

CONCLUSION

The study indicated that, there is significant ($P < 0.05$) increase levels of FBS, BP and MDA, and also no significantly ($P > 0.05$) increased levels of lipid profile in diabetic ischaemic stroke subjects compared with non-diabetic ischaemic stroke patients. It is concluded that

diabetic ischaemic stroke subjects had higher oxidative stress and dyslipidaemia associated with acute ischaemic stroke subjects in the study area.

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