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Aqueous Garlic Extract Mitigated Metabolic Syndrome in High Fat Diet-Induced Obesity: Relevance in Complementary Medicine and Obesity-Induced Nephropathy

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ABSTRACT

Background: The study investigated the effects of aqueous garlic extract (AGE) within a Wistar rat model of high-fat diet-induced metabolic syndrome and renal dysfunction. One in every four adults globally is afflicted by metabolic syndrome, with its prevalence ranging between 20% and 25% of the adult population. This condition is also well-correlated with renal dysfunction, further increasing the risk of a possible future decline in life expectancy if left unchecked.

Objective: To enhance clinical outcomes by evaluating AGE potential as an organic therapeutic intervention that could augment existing treatment or management strategies for high fat diet-induced metabolic syndrome and renal dysfunction.

Methods: Twenty Wistar rats were divided into four groups of five rats each such that AGE was administered following ad libitum access to high fat diet for a 13 weeks consecutive period. Meanwhile, the negative control group fed on normal fat diet for 13 consecutive weeks, after which the rats were euthanized in order to excise their kidneys and collect blood samples for further assessments.

Results: Ad libitum access to HFD was associated with manifestation of metabolic syndrome with the identification of four key clusters: obesity, fasting hyperglycemia, dyslipidemia and hypo-adiponectinemia. These were significantly mitigated following AGE administration ($p < 0.5$). The associated improvement of renal function was buttressed with improved renal his to-architecture (kidney reticular fibre network and general histology).

Conclusion: Aqueous garlic extract mitigated high fat diet-induced metabolic syndrome and associated obesity-induced nephropathy. This presents the extract as a potential organic therapeutic choice in complementary medicine.

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Introduction

Metabolic syndrome, characterized by the presence of at least three of five risk factors—including (i.) increased waist circumference, (ii.) diminished levels of high-density lipoprotein cholesterol, (iii.) elevated triglycerides, (iv.) hypertension, and (v.) heightened fasting blood glucose—constitutes a cluster of clinical

conditions that significantly augments the risk of developing type 2 diabetes mellitus, stroke, and cardiovascular disease [1-4]. It is noteworthy that, in experimental animal models of this condition, increased visceral adipose tissue as well as reduced adiponectin are additions to the aforementioned clusters [5-7]. An estimated one in every four adults globally is afflicted by this condition,

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with its prevalence ranging between 20% and 25% of the adult population [8,9]. While genetic predisposition, lifestyle choices, and obesity have been identified as pivotal factors influencing the prevalence of metabolic syndrome [10,11]. this study investigated an experimental model that encapsulates elements of lifestyle (high-fat diet) and obesity. The aim was to evaluate the therapeutic potential of organic complementary interventions that may impact treatment or management strategies for this condition and its associated renal dysfunction, with the objective of enhancing clinical outcomes.

Existing literature substantiates a robust correlation between metabolic syndrome and an increased predisposition to renal disease [12-15]. The pathophysiological mechanisms implicated in various studies encompass inflammation, hypertension, hyperfiltration, visceral obesity, insulin resistance and oxidative stress [12,14]. These identified pathophysiological pathways present promising targets for the development of potential treatment or management strategies that may alleviate this condition. This study undertook an experimental assessment of the extract of an organic material (garlic cloves) within a Wistar rat model of high-fat diet-induced metabolic syndrome.

Garlic cloves are significant culinary ingredients utilized as condiments in various cultural practices across parts of Asia and sub-Saharan Africa. However, due to their pronounced pungency and often irritating aroma, they are not universally embraced as a foremost choice of condiment despite their vast medicinal properties. The documented therapeutic effects of garlic cloves include, but not limited to, anti-inflammatory, antimicrobial, antibiotic and anti-oxidant activities [16-18]. Despite these remarkable pharmacological activities, there is dearth of literature on the effects of its aqueous extract within a Wistar rat model of high fat diet-induced metabolic syndrome and nephropathy. This study aimed at bridging this gap in knowledge.

Materials and Methods

Plant Materials and Diet Formulation

Fresh cloves of garlic were purchased from a commercial vendor at Lagere market of Ile-Ife, Osun state, Nigeria. They were identified by a Taxonomist at the Department of Botany, Obafemi Awolowo University, Ile-Ife, Osun State, Nigeria and a voucher specimen was archived at the herbarium.

Diet formulation was as described by Imafidon and Akomolafe [19]. While the normal fat diet (NFD) contained about 3.50% of total fat content, the high fat diet (HFD) constituted about 15.81% of total fat content. It has been established via experimental studies that normal rat feed contains about 4% of total fat content, with a total fat content of about 12% being capable to induce obesity in Wistar rats [19,20]. However, levels of up to about 15% have been previously demonstrated to be associated with nephropathy when allowed ad libitum access for a chronic period of 13 weeks [20].

Extraction Process of Aqueous Garlic Extract (AGE) And Preparation of Stock Solutions

The extraction process of aqueous garlic extract was as described in our previous study by Imafidon et al. where percentage yield was determined to be $69.47 \pm 0.51\%$ ($n=3$). The extract was stored in a desiccator until when needed [21].

According to our previous report, 200mg/kg and 400mg/kg of AGE, amongst the graded doses that were used, produced the most significant ameliorative effects in an experimental study on nephrotoxicity [21]. Building on previous experimental outcomes, this study adopted these two dose levels with the following stock solution preparations;

2g of AGE was dissolved in 20 ml of normal saline in order to prepare the stock solution for 200mg/kg of the extract. Meanwhile, 4g of AGE was dissolved in 20 ml of normal saline in order to prepare stock solution for 400mg/kg of the extract. This method of stock solution preparation was such that each 100g rat received 0.2ml of either dose level of the extract in order to prevent "fluid overload". The stock solutions were refrigerated at 5°C while fresh extracts were prepared every 48 hours, throughout the study period.

Animal Management and Experimental Protocol

The experimental protocols were in strict compliance with the guidelines for animal research, as contained in the NIH guidelines for the care and use of laboratory animals [22]. and approved by local institutional research committee.

Twenty (20) Male Wistar rats of about 8 - 10 weeks old, weighing 120 to 150g, were used for this study. The rats were divided into four (4) groups of five (5) rats each. They were housed in conventional plastic cages under natural light/dark cycle according to Table 1 and described as follows; Groups A where allowed ad libitum feeding on normal fat diet throughout the study period (13 weeks), after which they were euthanized. Group B were allowed ad libitum feeding on high fat diet for 13 weeks, after which they were also euthanized. Groups C and D where also allowed ad libitum access to HFD, as group B, while also receiving two dose-levels of Age at 200mg/kg and 400mg/kg respectively throughout the study period before they were also euthanized. Blood samples were collected under ketamine anesthesia (60 mg/kg i.m) via cardiac puncture into separate plain universal bottles in order to obtain serum samples. Thereafter, the blood samples were centrifuged at 4000 rpm for 15 mins using a cold centrifuge (Centrium Scientific, model 8881) at - 4°C. The resulting serums were decanted into separate plain bottles for biochemical assays. Thereafter, each right kidney of the rats was carefully excised and fixed in 10% formal saline solution for histological examinations while their left kidneys were transferred into separate plain bottles, maintained in a iced cooler, for the preparation of homogenates that were used to determine kidney biomarkers of pro-inflammatory cytokines and oxidative stress.

Assessment of Percentage Body Weight Change (PBWC), BMI, Fasting Blood Glucose (FBG) and Atherogenic Index

With the aid of Hanson digital weighing scale (Hanson, China), the body weight of the rats were determined while their percentage body weight change was calculated as described by the method of Ayoka et al [23]. as follows;

$$\bullet \text{ PBWC (\%)} = \frac{\text{Final body weight} - \text{Initial body weight}}{\text{Initial body weight}} \times 100\%$$

The BMI was, however, determined by the method of Novelli et al [24]. as follows;

- $$\text{BMI (g/cm}^2\text{)} = \frac{\text{Body weight (g)}}{\text{Nose-to-anus length}^2\text{ (cm}^2\text{)}}$$

- FBG was determined with the aid of a glucometer (On Call Plus II). At the end of the study period (13 weeks), before euthanization, the rats were fasted for 24 hours. Thereafter, their tail veins were gently pricked with a sterile lancet (after disinfecting the area) while drop of blood obtained was collected by capillary action into the already inserted On-call glucometer test. The values obtained were read directly in mg/dL.

- Atherogenic index was calculated by the below formula;

$$\text{Atherogenic index} = \frac{\text{Total Cholesterol} - \text{HDL}}{\text{HDL}}$$

Assessment of Serum Biomarkers of Renal Function and Adiponectin

Classical serum biomarkers of renal function including creatinine and urea were assayed using standard laboratory test kits (Randox Lab. Ltd., County Antrim, United Kingdom), according to the manufacturer’s instruction while the novel biomarkers (kidney injury molecule-1 and cystatin C) were assayed using ELISA technique with the protocol provided by Qauntikine ELISA kit (China).

Serum adiponectin concentrations were determined using Enzyme-linked Immunosorbent Assay (ELISA) technique (RIA kit, Lincon Research), according to the manufacturer’s instruction.

Assessment of Kidney Levels of Pro-Inflammatory Cytokines and Oxidative Stress Biomarkers

Standard biochemical kits for determination of pro-inflammatory cytokines in the kidneys were purchased from Abcam company using enzyme-linked immunosorbent assay (ELISA) technique and was interpreted in concentrations of pg./ml, according to the manufacturer’s protocol.

Using an electric homogenizer (S1601001), 10% homogenate in phosphate buffer (100mM) was prepared with the kidney tissues at a pH of 7.4. The homogenates were centrifuged at 3000 rpm for 20 mins and the resulting supernatants were collected for the assessment of the following biomarkers of oxidative stress and lipid peroxidation;

The kidney GSH concentration was determined according to the method of Beutler et al. [26]. as described as follows; To 1 ml of the sample, 0.5 ml of Ellman's reagent (10 mM) and 2 ml of phosphate buffer (0.2 M, pH=8.0) were added. The yellow precipitate developed was read at 412 nm against a blank containing 3.5 ml of phosphate buffer. Series of standard were treated similarly and the amount of GSH was expressed in µg/ mg protein. Whereas kidney catalase (CAT) activities were determined by the method of Sinha [27]. the SOD activity was determined by the method of McCord and Fridovich [28]. while TBARS level was by the method of Ohkawa et al [29]. as described as follows; To 0.5

ml of the sample was added 0.5 ml of phosphate buffer (0.1 M, pH 8.0) and 0.5 ml of 24% TCA. The resulting mixture was incubated at room temperature for 10 min, followed by centrifugation at 2000 rpm for 20 min. To 1 ml of the supernatant was added 0.25 ml of 0.33% TBA in 20% acetic acid and the resulting mixture was boiled at 95 °C for 1 h. The resulting pink colouring product was cooled and the absorbance was read at 532 nm.

Assessment of Serum Lipid Profile

The serum lipid profile of the rats was determined using Randox standard laboratory kit (Randox Lab. Ltd., County Antrim, United Kingdom), according to the manufacturer’s instruction and expressed in mg/dL

Histological Examinations

The 10% formo-saline fixed kidneys were subjected to histological examinations using conventional hematoxylin and eosin (H & E; for general histo-architectural appraisal) as well as Gordon and Sweet (for reticular fiber assessments) staining techniques. Tissue preparation on the slides were captured for photomicrograph assessment using a Leica DM 750 camera microscope at magnification of x 200.

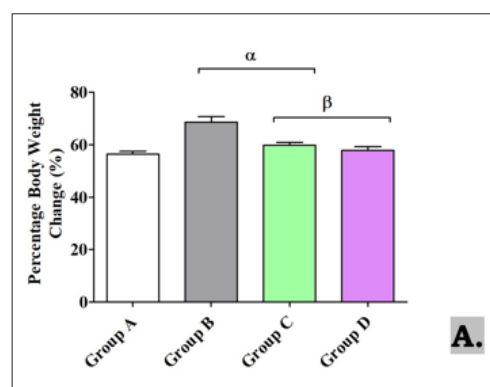
Statistical Analysis

Data were expressed as mean ± standard error of mean at p < 0.05. These were subjected to Newman Keul’s post hoc test using graph pad prism 5.03 statistical software (Graph Pad software Inc., CA, USA).

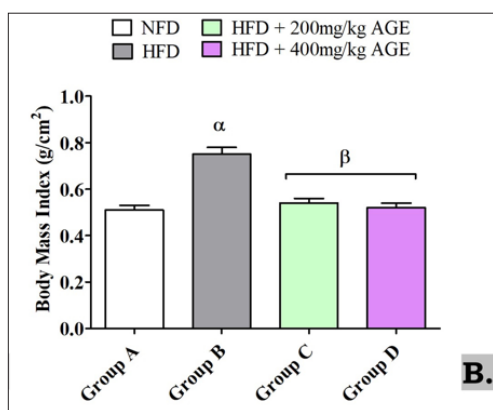
Results

Effects of High Fat Diet-Induced Obesity on PBWC, BMI, FBG and Artherogenic Index

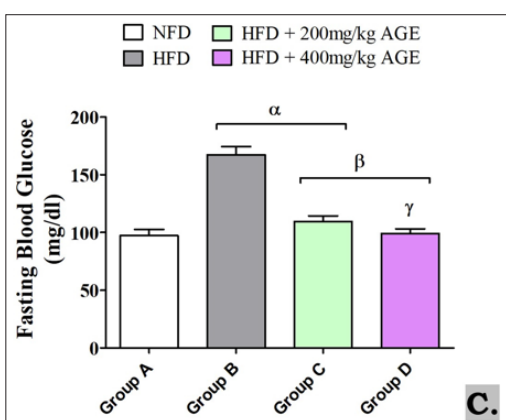
Chronic HFD consumption was associated with a significant increase in PBWC in the experimental group B when compared with the negative control group A (0.5). This was, however, significantly normalized in the AGE-treated groups (0.5) (Figure 1A).



HFD consumption induced obesity in the experimental group when compared with both the negative and treated groups (p<0.05). AGE treatment was associated with normal BMI when compared with both the negative and experimental group (p<0.05) (Figure 1B).



FBG was significantly elevated in experimental group when compared with the negative control group ($p < 0.05$). This was also significantly ameliorated following AGE treatment ($p < 0.05$) (Figure 1C).



The measured arteriogenic index (AI) was significantly elevated in group B (0.26 ± 0.02) when compared with the negative control and treated groups ($p < 0.05$). This index was significantly normalized following AGE treatment (Figure 1D).

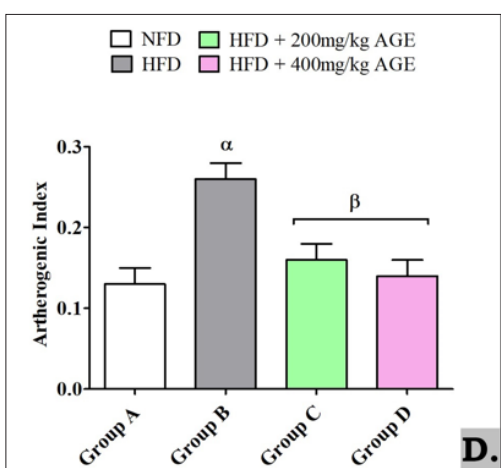


Figure 1: Effects of High Fat Diet-induced Obesity on PBWC, BMI, FBG and Arteriogenic Index.

Each bar represents mean \pm S.E.M at $p < 0.05$.

α = significant difference when compared with group A (NFD); β = significant difference when compared with group B (HFD); and γ = significant difference when compared with group C (HFD + 200mg/kg AGE).

Model of Metabolic Syndrome

The HFD formulation was adequate to exceed the minimum benchmark criteria to the clinical condition of metabolic syndrome (MetS). Ad libitum feeding on HFD for a chronic period of 13 weeks was associated with obesity, fasting hyperglycemia, dislipidemia (with elevated serum total cholesterol concentration) and significantly reduced serum adiponectin (Figure 2).

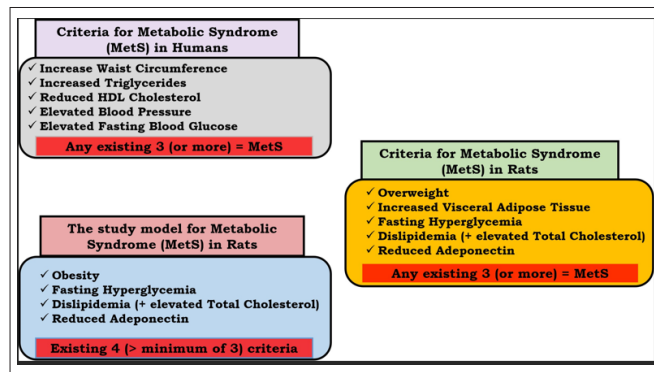


Figure 2: Risk Factors that Characterizes Metabolic Syndrome

Effects of Age on Serum Biomarkers of Renal Function and Adiponectin of Wistar Rats with High Fat Diet-Induced Metabolic Syndrome

Both the classical and novel biomarkers of renal functions were significantly elevated in the serum of the rats of the experimental group when compared with the negative control group ($p < 0.05$). These parameters were significantly mitigated following AGE administration in the treatment groups ($p < 0.05$) (Figures 3A, B, C and D).

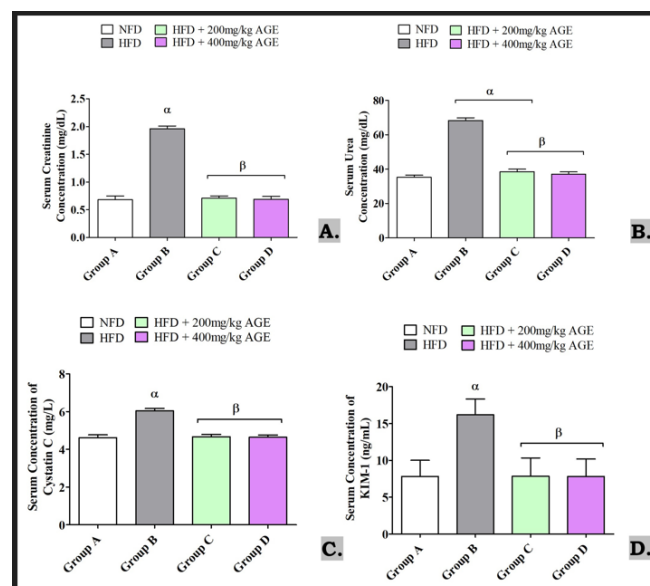


Figure 3: Effects of AGE on Serum Biomarkers of Renal Function of Wistar Rats with High Fat Diet-induced Metabolic Syndrome.

Each bar represents mean \pm S.E.M at $p < 0.05$.

α = significant difference when compared with group A (NFD); and β = significant difference when compared with group B (HFD).

Effects of AGE on Kidney Levels of Pro-Inflammatory Cytokines and Oxidative Stress Biomarkers of Wistar Rats with High Fat Diet-Induced Metabolic Syndrome

The kidney levels of pro-inflammatory cytokines were significantly elevated in the experimental group when compared with the control ($p < 0.05$). These indices were, however, mitigated by the extract in the treated groups ($p < 0.05$)

Table 1: Experimental Protocol

N = 20	Description	Duration
Group A (n =5)	Ad libitum access to NFD	13 Weeks *
Group B (n =5)	Ad libitum access to HFD	13 Weeks *
Group C (n =5)	Ad libitum HFD + 200mg/kg AGE	13 Weeks *
Group D (n =5)	Ad libitum HFD + 400mg/kg AGE	13 Weeks *

N = Total number of recruited Wistar Rats; n = Number of Rats per Group; NFD = Normal Fat Diet; HFD = High Fat Diet; AGE = Aqueous Garlic Extract; * = Point at Which Rats Were Euthanized.

The indices of oxidative stress markers as well as lipid peroxidation were deleteriously expressed in the treatment group when compared with the negative control group ($p < 0.5$). Also, following AGE administration, these levels were significantly ameliorated in the kidney homogenates of the rats ($p < 0.5$) (Table 3).

Table 2: Effects of AGE on Kidney Levels of Pro-inflammatory Cytokines of Wistar Rats with High Fat Diet-induced Metabolic Syndrome

	IL-6 (pg/ml)	CRP (pg/ml)	TNF_α (pg/ml)
Group A	72.50 ± 4.80	0.52 ± 0.02	33.21 ± 2.37
Group B	103.47 ± 6.25 ^α	0.87 ± 0.03 ^α	57.20 ± 2.49 ^α
Group C	75.05 ± 5.08 ^β	0.56 ± 0.02 ^{αβ}	33.43 ± 2.35 ^β
Group D	73.26 ± 5.32 ^β	0.53 ± 0.02 ^β	33.39 ± 2.28 ^β

Each value represents mean ± S.E.M at $p < 0.05$.

α = Significant Difference When Compared with Group A (NFD); and

β = Significant Difference When Compared with Group B (HFD).

Effects of AGE on Serum Lipid Profile of Wistar Rats with High Fat Diet-Induced Metabolic Syndrome

Table 4 depicts that the HFD-induced metabolic syndrome was associated with significant derangement in the serum lipid profile of the experimental group when compared with the negative when compared with the control ($p < 0.5$). The extract demonstrated significant normalization of serum lipid profile of the rats in the treated groups when compared with both the experimental and negative control groups ($p < 0.05$) (Table 4).

Table 3: Effects of AGE on Kidney Oxidative Stress Biomarkers and Index of Lipid Peroxidation of Wistar Rats with High Fat Diet-induced Metabolic Syndrome

	TBARS (nmol/mg protein)	CAT (μmol/min/mg protein)	SOD (mM)	GSH (μg/mg protein)
Group A	30.91 ± 2.28	2.82 ± 0.17	1.40 ± 0.10	4.51 ± 0.21
Group B	75.00 ± 2.62 ^α	1.08 ± 0.14 ^α	0.47 ± 0.13 ^α	2.32 ± 0.27 ^α
Group C	33.22 ± 3.00 ^β	2.78 ± 0.18 ^β	1.36 ± 0.12 ^β	4.47 ± 0.30 ^β
Group D	32.46 ± 2.83 ^β	2.80 ± 0.15 ^β	1.39 ± 0.12 ^β	4.50 ± 0.28 ^β

Each value represents mean ± S.E.M at $p < 0.05$.

α = Significant Difference When Compared with Group A (NFD);

β = Significant Difference When Compared with Group B (HFD); and

γ = Significant Difference When Compared with Group C (HFD + 200mg/kg AGE).

Table 4: Effects Of AGE on Serum Lipid Profile of Wistar Rats with High Fat Diet-Induced Metabolic Syndrome

	LDL (mg/dl)	VLDL (mg/dl)	TRIG (mg/dl)	HDL (mg/dl)	TC (mg/dl)
Group A	59.35 ± 4.15	28.44 ± 3.40	84.29 ± 12.53	41.08 ± 1.40	111.24 ± 13.30
Group B	151.37 ± 3.22 α	63.51 ± 3.75 α	228.67 ± 20.34 α	27.87 ± 2.06 α	280.48 ± 12.55 α
Group C	65.48 ± 3.18 $\alpha\beta$	31.73 ± 4.00 β	90.21 ± 17.25 β	38.28 ± 1.34 $\alpha\beta$	132.40 ± 11.82 $\alpha\beta$
Group D	62.09 ± 3.40 β	29.04 ± 3.55 β	86.70 ± 16.54 β	40.33 ± 1.28 β	120.07 ± /11.66 β

Each value represents mean ± S.E.M at p <0.05.

α = Significant Difference When Compared with Group A (NFD);

β = Significant Difference When Compared with Group B (HFD); and

γ = Significant Difference When Compared With Group C (HFD + 200mg/kg AGE).

Histological Effects of Age on The Kidney of Wistar Rats with High Fat Diet-Induced Metabolic Syndrome

HFD-induced metabolic syndrome was associated with marked distortion of the kidney’s reticular fibers (Figure 5A) as well as kidney interstitial vacuolation and glomerular atrophy (Figure 5B). However, AGE administration in the treatment group showed appreciable improvement of the general kidney histo-architecture of the treatment groups (Figures 5A and 5B).

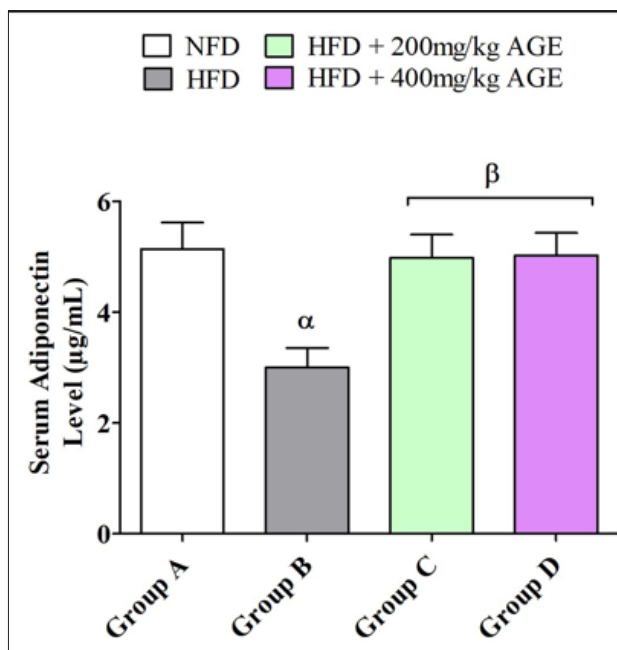


Figure 4: Effects of AGE on Serum Adiponectin of Wistar Rats with High Fat Diet-induced Metabolic Syndrome.

Each bar represents mean ± S.E.M at p <0.05.

α = significant difference when compared with group A (NFD); and

β = significant difference when compared with group B (HFD).

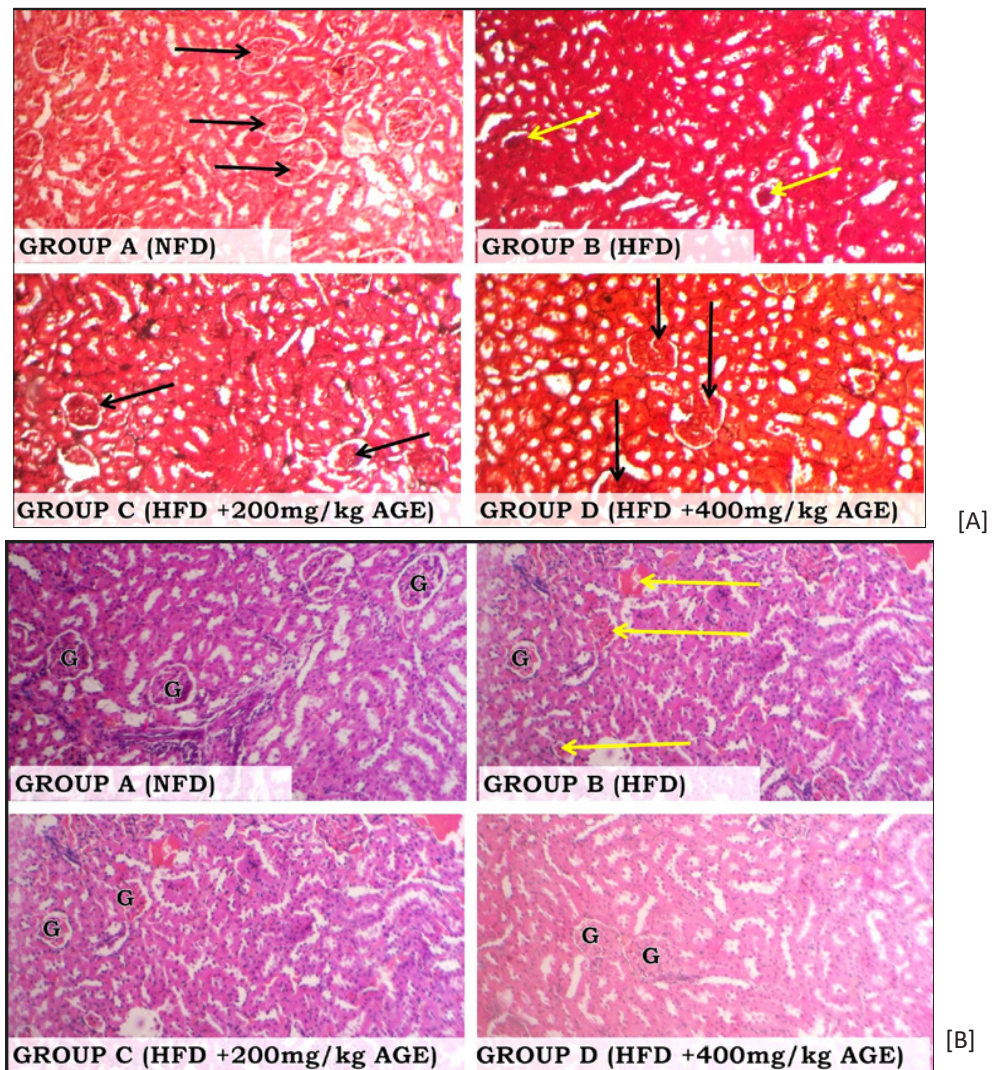


Figure 5: Histological Effects of AGE on the Kidney of Wistar Rats with High Fat Diet-induced Metabolic Syndrome.

(A.) Gordon and Sweet_Histology

(B.) H & E_Histology

Discussion

This study meticulously examined the effects of aqueous garlic extract on high-fat diet-induced metabolic syndrome and renal

dysfunction within a Wistar rat model. The objective was to enhance clinical outcomes by evaluating its potential as an organic therapeutic intervention that could augment existing treatment or management strategies for these conditions. Metabolic syndrome is defined by the presence of at least three out of five clusters of clinical manifestations (in both human and experimental animal models). This investigation identified four key clusters: obesity, fasting hyperglycemia, dyslipidemia (characterized by elevated serum total cholesterol levels), and a significant reduction in serum adiponectin.

Metabolic syndrome was correlated with notable alterations in both inflammatory and oxidative stress biomarkers assessed in this study. This underscores the involvement of both the up-regulation of pro-inflammatory pathways and oxidative stress in the pathogenesis of metabolic syndrome. However, subsequent to the administration of the extract (AGE), there was a remarkable down-regulation of pro-inflammatory cytokines (CRP, IL-6, and TNF- α) as well as a significant amelioration of oxidative stress, as indicated by normalized kidney levels of both non-enzymatic (GSH) and enzymatic (SOD and CAT) oxidative stress indices. The inference drawn from these outcomes suggests that the therapeutic effects of AGE are linked to a substantial down-regulation of pro-inflammatory pathways and the normalization of oxidative stress status.

Evident renal dysfunction was observed in association with the metabolic syndrome model. This dysfunction was characterized by significantly elevated serum levels of renal function biomarkers, encompassing both classical (creatinine and urea) and novel (Cystatin-C and Kim-1) indicators. The pronounced elevation of these biomarkers in plasma was concomitant with marked histo-architectural distortion of the kidney, as illustrated by the representative photomicrographs presented in this study. The histological anomalies included pronounced degradation of the reticular fiber network, interstitial vacuolation, and atrophy of the glomerulus. The glomerulus serves as a critical component of the glomerular filtration barrier; a physiological structure within the renal system that facilitates the elimination of metabolic wastes from circulating blood by a physiological process known as renal clearance [30]. A compromise of renal clearance can precipitate the bio-accumulation of metabolic wastes to dangerous endogenous levels, potentially leading to deleterious systemic effects if left unaddressed. Therefore, it is evident that AGE ameliorated the compromised clearance in this condition through potential repair of the reticular fiber framework and membrane stabilization; a plausible tissue-repair process culminating in an apparent enhancement of kidney histo-architecture, as depicted by the representative photomicrographs.

The deleterious effects of metabolic syndrome on oxidative stress status and inflammatory biomarkers in metabolic syndrome was well associated with hypo-adiponectinemia (decreased serum adiponectin level) as recorded in this study. Under normal condition, adiponectin is essential for the regulation of insulin sensitivity, metabolism and systemic inflammation [31,32]. The recorded elevated fasting blood glucose levels, although not within the diabetic range, suggests that a potential manifestation of type 2 diabetes is in view. This may be well-expressed if an extended chronic period of ad libitum access

to the high fat diet was allowed. Meanwhile, adiponectin regulatory function also extends to anti-atherogenic role, whereby the buildup of plaques in arteries are prevented. This study recorded a significant elevation of atherogenic index. By implication, metabolic syndrome (within the adopted animal model) is associated with an increased risk of cardiovascular disease; a health risk that was significantly mitigated by AGE administration. However, this health risk may have been exacerbated by the deleterious disturbance of the serum lipid profile that was observed.

There was a significant decrease in HDL which was associated with heightened serum levels of LDL and VLDL. These are pointers to the potential buildup of arterial plaques in this condition. The mitigating potential of the extract suggests that AGE initiates systemic mechanisms (worthy of molecular investigation) that prevents arterial plaque formation or build up, consequently reducing the risk of pre-disposition to cardiovascular disease. This was further buttressed by the significant normalization of serum triglyceride and total cholesterol concentrations.

Conclusion

Aqueous garlic extract mitigated high fat diet-induced metabolic syndrome and associated obesity-induced nephropathy via down-regulation of pro-inflammatory pathways as well as normalization of atherogenic index, adiponectin and anti-oxidant status. This presents the extract as a potential organic therapeutic choice in complementary medicine for metabolic syndrome and associated nephropathy.

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Conflict Of Interest

The authors have no conflict of interest to declare.

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