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Prognostic Value of NT-ProBNP and Omega-3 Fatty Acids in Assessing Hypertension Severity in Newly Diagnosed Adults at NAUTH, Nnewi, Nigeria

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Abstract
Hypertension is a major public health concern globally, particularly in sub-Saharan Africa. Biomarkers such as N-terminal pro-B-type natriuretic peptide (NT-proBNP) and Omega-3 fatty acids have been linked to cardiovascular function and disease progression. This study investigates the relationship between NT-proBNP and Omega-3 fatty acid levels and hypertension severity in newly diagnosed adults at Nnamdi Azikiwe University Teaching Hospital (NAUTH), Nnewi, Nigeria. A cross-sectional study was conducted involving 90 participants: 45 hypertensive individuals and 45 normotensive controls. Anthropometric, blood pressure, NT-proBNP, and Omega-3 fatty acid levels were determined using Enzyme linked immunosorbent assay. Hypertensive individuals had significantly higher SBP (140.32±19.80 mmHg) and DBP (82.05±11.06 mmHg) compared to controls (SBP: 111.15±5.06 mmHg, DBP: 69.70±5.39 mmHg; *p*=0.000). NT-proBNP levels were significantly hygher in hypertensive individuals (7.21±4.25 ng/L) than in controls (4.67±1.85 ng/L; *p*=0.012), while Omega-3 fatty acid levels were markedly reduced in hypertensives (269.41±128.40 ng/L) compared to controls (931.05±607.61 ng/L; *p*=0.000). A significant negative correlation was observed between NT-proBNP and Omega-3 fatty acids (r = -0.493, p = 0.017). In the present study, Hypertensive individuals exhibited significantly higher NT-proBNP and lower Omega-3 fatty acid levels compared to controls, indicating increased cardiac stress and reduced anti-inflammatory capacity. This may subsequently increase the risk of cardiovascular events and disease progression. The study supports the potential of NT-proBNP and Omega-3 fatty acids as predictive biomarkers for hypertension severity. Elevated NT-proBNP reflects cardiac stress, suggesting early myocardial strain even in the absence of clinical symptoms, which may predispose to left ventricular hypertrophy and eventual heart failure. Routine NT-proBNP screening in hypertensive patients may facilitate early cardiovascular risk identification. Concurrently, diminished Omega-3 levels underscore the need for nutritional strategies that enhance vascular health. Integration of these biomarkers into routine clinical practice could improve prognostication and guide personalized therapeutic interventions.
Keywords: NT-proBNP, omega 3 fatty acid, adults, hypertension, severity
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1. Introduction

Hypertension affects over one billion individuals globally and remains a leading contributor to cardiovascular morbidity and mortality (WHO, 2021). Often termed the "silent killer," this condition frequently progresses without overt symptoms, yet it significantly increases the risk of severe complications affecting vital organs and systems. Chronic elevation of blood pressure imposes excessive mechanical stress on arterial walls, triggering a cascade of pathophysiological changes that culminate in various cardiovascular diseases (Bassa *et al.* 2019).

One of the most common complications is coronary artery disease (CAD), in which sustained hypertension accelerates arterial hardening and narrowing, reducing myocardial perfusion. This can manifest clinically as angina and substantially elevate the risk of myocardial infarction. The heart, in compensating for increased vascular resistance, may undergo left ventricular hypertrophy, which over time can deteriorate into heart failure. Additionally, hypertension predisposes individuals to vascular abnormalities such as aneurysms, the rupture of which can result in catastrophic internal bleeding (Gill *et al.* 2024).

Hypertension is classified based on blood pressure (BP) measurements: normal (SBP <120 mm Hg and DBP <80 mm Hg), elevated (SBP 120–129 mm Hg and DBP <80 mm Hg), Stage 1 hypertension (SBP 130–139 mm Hg or DBP 80–89 mm Hg), and Stage 2 hypertension (SBP ≥140 mm Hg or DBP ≥90 mm Hg). These thresholds underscore the importance of early detection and intervention ranging from lifestyle modifications to pharmacotherapy especially in individuals at high cardiovascular risk (Whelton *et al*. 2018).

While BP measurement remains the cornerstone of hypertension diagnosis and management, emerging biomarkers offer deeper insights into disease pathophysiology and therapeutic monitoring. Two such biomarkers of interest are N-terminal pro-B-type natriuretic peptide (NT-proBNP) and Omega-3 fatty acids. NT-proBNP is an inactive peptide fragment released predominantly by cardiac ventricular myocytes in response to myocardial stretch and elevated wall stress (Maisel *et al.* 2002). It originates from the cleavage of proBNP into biologically active BNP and the more stable NT-proBNP. Due to its longer half-life and higher circulating concentrations, NT-proBNP is widely used in clinical practice as a reliable marker of cardiac dysfunction. Elevated NT-proBNP levels are strongly associated with increased ventricular pressure and volume overload, making it indispensable in the diagnosis and risk stratification of heart failure (HF) (Jannuzi *et al.* 2006**).** In acute care settings, it also helps differentiate cardiac from non-cardiac causes of dyspnea. Age-adjusted cutoff values, such as a threshold of 300 pg/mL, enhance diagnostic accuracy, offering high sensitivity and negative predictive value for ruling out acute HF (Oguma *et al.* 2017).

Beyond diagnosis, NT-proBNP serves as a powerful prognostic biomarker. Persistently elevated levels correlate with adverse outcomes, including recurrent hospitalizations and increased mortality in both acute and chronic HF populations (Di Veroli *et al*. 2020). Serial measurements can reflect therapeutic efficacy decreasing values suggest clinical improvement, whereas rising levels may signal impending decompensation or disease progression. Thus, NT-proBNP plays a pivotal role not only in diagnostic evaluation but also in longitudinal monitoring and personalized disease management (Principi *et al*. 2021).

On the other hand, Omega-3 fatty acids, particularly eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA)**,** are essential polyunsaturated fatty acids known for their cardioprotective effects (Mozaffarian *et al.* 2005; Calder, 2017). These include anti-inflammatory, anti-thrombotic, anti-arrhythmic, and vasodilatory properties (Filipovic *et al.* 2018). Clinical and epidemiological evidence suggests that Omega-3 fatty acids help lower blood pressure, improve endothelial function (Mozaffaria & Wu, 2012; Lavie *et al.* 2009) and reduce overall cardiovascular risk. A significant inverse relationship between the Omega-3 index and 24-hour ambulatory blood pressure has been documented (Mori & woodman, 2006), supporting their role in hypertension prevention and management.

Despite their established individual roles in cardiovascular health, few studies have concurrently evaluated NT-proBNP and Omega-3 fatty acid levels in hypertensive populations, particularly in sub-Saharan Africa. In Nigeria, where hypertension prevalence continues to rise, such investigations are essential for developing biomarker-informed strategies tailored to local contexts.

This study, therefore, seeks to evaluate serum levels of NT-proBNP and Omega-3 fatty acids among hypertensive adults attending Nnamdi Azikiwe University Teaching Hospital (NAUTH), Nnewi**,** and compare them with normotensive controls. Understanding these associations could enhance biomarker-driven risk stratification**,** support early interventions, and inform context-specific approaches to hypertension management

2. MATERIALS AND METHODS

2.1 Study Area

This study was carried out at Nnamdi Azikiwe University Teaching Hospital, Nnewi, Anambra State, Nigeria

2.2 Study Design

A cross-sectional study was conducted to assess NT-proBNP and Omega-3 levels in hypertensive individuals attending the cardiovascular health clinic in Nnamdi Azikiwe University Teaching Hospital (NAUTH), Nnewi, Anambra State, Nigeria. Selection of the participants were done by random sampling. Written consent (questionnaire) was obtained from the participants, and the individuals request form was used to collect their bio-data.

2.3 Study Population
The study included hypertensive individuals attending NAUTH, Nnewi. A total of 90 adult participants who fall within the age range of 30-70 years were recruited for the study, including 45 hypertensive individuals (23 males, 22 females) and 45 non-hypertensive individuals (22 males, 23 females) were included as control group.

2.4 Inclusion and Exclusion Criteria
Individuals who were diagnosed with hypertension, for the case group, individuals aged 30 years and above, and those who provided informed consent were included in the study.
Individuals with malaria parasite infection and other cardiovascular diseases such as diabetes, heart failure, those with chronic kidney or liver disease, individuals on Omega-3 supplementation, and those with other infectious diseases were excluded from the study.

2.5 Ethical clearance and Informed Consent
The ethical approval for this research was obtained from the board of ethics committee of Nnamdi Azikiwe University Teaching Hospital Nnewi, with Ref: NAUTH/CS/66/VOL.17/VER.3/61/2024/196 in accordance with the Helsinki declaration by the World Medical Association (WMA) on the ethical principles for medical research involving human subjects. Informed consent was obtained from the participants included in the study.

2.6 Anthropometrics measurements
Weight and height were measured in clothing without shoes and body mass index (BMI) calculated as: BMI= weight (kg) / height (m2). The height was obtained with a measuring tape attached to a wood while, weight was measured by use of a manual weighing scale

2.7 Blood pressure reading
Systemic blood pressure was obtained using an OMRON automatic digital blood pressure monitor on the left arm after 10-minute rest using a cuff of appropriate size with the subject in the sitting position. Blood pressure was expressed as Systolic and Diastolic rate. Hypertension was defined as systolic blood pressure ≥ 140mmHg and/or diastolic blood pressure ≥90mmHg.

2.8 Sample Collection
After obtaining informed consent, 5 mL of fasting venous blood was collected aseptically from each participant in the morning between 7:00 and 9:00 AM from each subject and dispensed into plain tubes. Blood was transferred into plain and EDTA tubes. The sample was allowed to clot, centrifuged at 5,000 rpm for 5 minutes, and serum was separated for evaluation of NT-proBNP and Omega-3 levels. For EDTA tubes, plasma was similarly separated by centrifugation and stored for analysis.

2.9 Determination of Omega-3 fatty acid Level
Plasma Omega-3 fatty acid levels were measured using a competitive ELISA kit specific for eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA), the main bioactive components of Omega-3s as was described by Tanaka *et al*. (2009). In this assay, antibodies specific to Omega-3 fatty acids were immobilized on a microplate. When samples containing Omega-3 fatty acid were introduced, these fatty acid bound to the immobilized antibodies. A secondary antibody, conjugated to an enzyme such as Horseradish Peroxidase (HRP), was then added to form a complex with the bound Omega-3 fatty acids. Upon addition of a substrate like Tetramethylbenzidine (TMB), the enzyme catalyzed a colorimetric reaction, resulting in a color change. The intensity of the color, measured spectrophotometrically at 450 nm, was directly proportional to the concentration of Omega-3 fatty acids in the sample.

2.10 Determination of NT-proBN
Quantification of serum NT-proBNP was performed using a sandwich enzyme-linked immunosorbent assay (ELISA) kit as was described by Clerico *et al*. (2006). NT-proBNP in the sample bound to the capture antibody immobilized on the working electrode surface. Recruitment of the labeled detection antibody by bound NT-proBNP completed the sandwich. The user added an MSD read buffer that provided the appropriate chemical environment for electrochemiluminescence and loaded the plate into an MSD SECTOR instrument for analysis.

Inside the SECTOR instrument, a voltage applied to the plate electrodes caused the labels bound to the electrode surface to emit light. The instrument measured the intensity of emitted light to provide a quantitative measure of NT-proBNP present in the sample. The assay sensitivity was approximately 2 pg/mL, and intra-assay and inter-assay coefficients of variation were <10%**.**

2.11 Data Analysis
Descriptive statistics (mean, standard deviation) were used to summarize NT-proBNP and Omega-3 fatty acid levels. Independent t-tests and ANOVA were used to compare biomarker levels between groups. Pearson's correlation analysis was performed to assess the relationship between NT-proBNP and Omega-3 levels. A p-value < 0.05 was considered statistically significant.

3.0 RESULTS

3.1 Anthropometric and blood pressure variables in hypertensive individuals and controls
The value of age was significantly higher in hypertensive individuals when compared with control (p=0.000). Similarly, the values of SBP and DBP were significantly higher in hypertensive participants compared to apparently healthy control individuals (p=0.000 respectively). However, there was statistically higher value of in BMI in hypertensive individuals when compared with controls group (p=0.013) (table 1).

Table 1: Anthropometric and blood pressure variables in hypertensive individuals and controls

Parameter	Hypertensive (N =45)	Control (N= 45)	*t*-value	*p*-value
Age (years)	61.09±10.07	32.05±7.50	84.92	0.000
BMI (kg/m²)	28.66±6.57	25.52±3.19	3.653	0.013
SBP (mmHg)	140.32±19.80	111.15±5.06	19.851	0.000
DBP (mmHg)	82.05±11.06	69.70±5.39	7.815	0.000

3.2 Gender comparison of Anthropometric and blood pressure variables in hypertensive individuals and control participants

Table 2 shows that female hypertensive individuals had higher SBP and DBP than male hypertensives while, both hypertensive groups had significantly higher blood pressure values than controls (*p*<0.05). The value of age was significantly higher in both male and female hypertensive when compared with their control counterparts (p = 0.000 respectively). Similarly, the mean value of BMI was significantly higher in female hypertensive individuals when compared with male hypertensive and their corresponding controls (p =0.000 respectively).

Table 2: Gender comparison of Anthropometric and blood pressure variables in hypertensive individuals and control participants

Group	Age(years)	BMI(Kg/m^2	SBP(mmhg)	DBP(mmhg)
Female hypertensive (N=22) (A)	60.36±10.38	29.64±7.24	147.71±17.93	94.29±19.89
Male hypertensive individuals (N=23) (B)	62.38±10.04	26.96±5.20	137.88±13.84	78.13±12.66
Female control (N=23) (C)	29.56±9.33	25.80±3.63	113.67±5.07	71.44±5.43
Male control (N=22) (D)	34.09±7.34	25.29±2.94	109.18±4.30	68.27±5.15
F value	39.233	1.831	8.309	3.971
P value	0.000	0.092	0.000	0.001
A vs B	1.000	O.000	0.012	0.003
A vs C	0.000	0.000	0.005	0.008
A vs D	0.000	0.000	0.008	0.007
B Vs C	0.000	0.911	0.000	0.000
B Vs D	0.000	0.995	0.001	0.000
C vs D	1.000	1.000	1.000	0.890

Key: p – value= significant at P <0.05, BMI: Body mass index, SBP: Systolic blood pressure, DBP: Diastolic blood pressure

3.3 Levels of NT-proBNP and Omega-3 fatty acids in hypertensive individuals and control participants

In table 3, NT-proBNP level was significantly higher in hypertensives (7.21±4.25 ng/L) than in controls (4.67±1.85 ng/L; *p*=0.012). Conversely, Omega-3 fatty acids were significantly reduced in hypertensive individuals compared to controls (269.41±128.40 ng/L vs. 931.05±607.61 ng/L; *p*=0.000).

Table 3: Levels of NT-proBNP and Omega-3 fatty acids in hypertensive individuals and control participants

Groups	NT-proBNP (ng/L)	Omega-3 fatty acids (ng/L)
Hypertensive (N=45)	7.21 ± 4.25	269.41 ± 128.40
Control (N=45)	4.67 ± 1.85	931.05 ± 607.61
T-value	8.238	19.970
P-value	0.012	0.000

3.4 Gender comparison of Levels of NT-proBNP and Omega-3 fatty acids in hypertensive individuals and control participants

Among hypertensive individuals, females had slightly higher NT-proBNP levels than males, while Omega-3 level was significantly lower in females. The mean NT-proBNP levels was significantly higher in hypertensive than in controls. There was significantly lower mean level of Omega-3 fatty acid in both male and female hypertensive individuals when compared with their corresponding control individuals (p < 0.05 respectively) (table 4).

Table 4: Gender comparison of Levels of NT-proBNP and Omega-3 fatty acids in hypertensive individuals and control participants

Group	NT-proBNP (ng/l)	Omega-3fatty acids (ng/l)
Female hypertensive (N=22) (A)	8.10±5.66	256.77±119.66
Male hypertensive (N=23) (B)	6.48±2.72	291.52±148.29
Female control (N=23) (C)	4.62±1.87	1031.70±751.79
Male control (N=22) (D)	4.63±1.94	848.70±482.41
F value	3.712	8.701
P value	0.002	0.000
A vs B	0.009	1.000
A vs C	0.011	0.000
A vs D	0.000	0.001
B vs C	0.619	0.003
B vs D	0.759	0.102
C vs D	0.895	1.000

Key: p – value = significant at P <0.05, NT-proBNP: N-terminal pro-b type natriuretic peptide

3.4 Correlation between NT pro BNP and omega 3 fatty acids in hypertensive individuals

A significant negative correlation was observed between NT-proBNP and Omega-3 fatty acids levels in hypertensive individuals (r = -0.493, p = 0.017), as shown in Table 5.

Table 5: Correlation between NT pro BNP and omega 3 fatty acids in hypertensive individuals

Parameter	NT pro BNP
Omega 3 fatty acids	r -0.493 p 0.017

Key: p – value = significant at P < 0.05, NT-proBNP: N-terminal pro-b type natriuretic peptide

R-value: Correlation coefficient

4. DISCUSSION

Hypertension remain the leading risk factor for cardiovascular morbidity and mortality, often progressing silently until advanced complications occur. In the present study, Hypertensive individuals exhibited significantly higher NT-proBNP and lower Omega-3 fatty acids levels compared to controls, indicating increased cardiac stress and reduced anti-inflammatory capacity. A strong association between hypertension and elevated NT-proBNP levels, has been previously indicated linking NT-proBNP to cardiac strain and heart failure (Mckie & Burnett, 2018; Januzzi *et al*. 2021). Some other studies noted that lower circulating levels in early or compensated hypertension may be due to receptor downregulation or altered renal clearance (Mckie *et al.* 2005). The author also attributed this to delayed compensatory phase in early stages of hypertension where natriuretic peptide secretion is blunted or NT-proBNP clearance is altered (Wang *et al.* 2004). Other factors include obesity which may occur in hypertension causing suppression of NT-proBNP due to increased clearance or reduced secretion from cardiac tissue (Das *et al.* 2005). Significant elevation in NT-proBNP level in hypertensive participants compared to contro**ls** has been shown to be typically elevated in cardiac overload. which conforms with the conventional expectation of elevated NT-proBNP in cardiac stress (Kario *et al.* 2021).

The markedly reduced Omega-3 fatty acids among hypertensives align with findings that low EPA/DHA intake correlates with increased vascular resistance and inflammation (Mozaffarian *et al.* 2005; Saravanan *et al.* 2010). The observed depletion of Omega-3 fatty acids tends to reinforce their role in cardiovascular protection (Mozaffarian & Wu, 2011). Omega-3 fatty acids have been widely recognized for their cardioprotective properties, including blood pressure regulation, anti-inflammatory effects, and endothelial function improvement (Calder, 2020). Their potential role in mitigating hypertension-related complications in the affected individuals is of growing interest. In hypertensive individuals, Omega-3 fatty acids may counteract inflammation-induced vascular damage, potentially modulating NT-proBNP levels and improving cardiovascular outcomes (Adegbala *et al.* 2023). Reduced Omega-3 deficiency has been associated with endothelial dysfunction, which may contribute to the pathogenesis of hypertension (Mozaffarian *et al.* 2005; Bays, 2006). The low Omega-3 levels observed could also reflect poor dietary intake, limited access to Omega-3 rich foods like oily fish, or higher oxidative stress leading to their depletion.

This study observed that age was significantly different among the groups, with hypertensive individuals (both males and females) being significantly older than non-hypertensive individuals. This is consistent with existing literature, which suggests that hypertension prevalence increases with age due to vascular changes and reduced arterial elasticity (Wang *et al.* 2021; Ukibe *et al.* 2024). Body Mass Index (BMI) shows significant differences between the groups, which is consistent with findings from prior studies indicating a strong association between higher BMI and hypertension (Tadesse *et al.* 2022). These findings are consistent with the recognized epidemiological characteristics of hypertension, where advancing age and obesity serve as major risk factors (Kearney *et al*. 2005).

Systolic blood pressure (SBP) and diastolic blood pressure (DBP) were significantly higher in hypertensive individuals compared to control participants, which aligns with previous research demonstrating that hypertension is a primary risk factor for cardiovascular morbidity (Abegaz *et al.* 2021). Gender-specific analysis revealed significant differences in blood pressure between hypertensive males and females, corroborating previous studies showing that while men tend to develop hypertension earlier, women exhibit a steeper increase in blood pressure post-menopause (Peters *et al.* 2020). The significantly higher BP in females than males might reflect hormonal influences, as estrogen has been implicated in vascular regulation (Miller *et al.* 2021). The gender-based disparities highlight possible hormonal or metabolic influences, where females showed higher NT-proBNP levels, potentially due to heightened cardiovascular stress perception or hormonal differences (Mckie & Burnett, 2005; Omland *et al.* 2007).

Omega-3 fatty acids levels were significantly lower in hypertensive male and female individuals, compared to their corresponding controls. This aligns with previous findings that suggest omega-3 fatty acids play a protective role against hypertension and cardiovascular disease by modulating inflammation and endothelial function (Mozaffarian & Wu, 2021). The observed reduction in omega-3 levels in hypertensive individuals may indicate an increased inflammatory burden, which has been implicated in hypertension pathophysiology (Adeoye *et al.* 2022). Omega-3 fatty acids are known to influence multiple biological pathways that affect vascular tone, platelet aggregation, and inflammatory responses (Calder, 2012). Their depletion in hypertensive individuals suggests that lower Omega-3 availability may impair endothelial function and promote a pro-inflammatory state that contributes to sustained hypertension. These mechanisms may be exacerbated in individuals with dietary deficiencies or increased metabolic demands. Gender differences observed in these biomarker levels further support the influence of sex-specific physiology in hypertension. Female hypertensives exhibited higher systolic and diastolic pressures and lower Omega-3 fatty acids levels compared to their male counterparts, possibly reflecting hormonal variations or dietary disparities. These findings emphasize the need for gender-sensitive approaches in cardiovascular risk assessment and nutritional counseling.

Furthermore, the study found a moderate negative correlation between NT-proBNP and omega-3 fatty acids in hypertensive individuals. This finding is noteworthy as previous studies have suggested that higher omega-3 levels are associated with improved cardiac function and reduced NT-proBNP levels (Cozlea *et al.* 2020). Furthermore, NT-proBNP as a biomarker provides insight into cardiac overload, a common consequence of long-standing hypertension. Elevated levels signal the heart's response to increased wall stress and can precede clinical manifestations of heart failure. This underscores its role not only as a diagnostic tool but also as a prognostic marker for cardiovascular complications.

The inverse correlation between NT-proBNP and Omega-3 fatty acids suggests a possible modulatory role of these fatty acids on cardiac stress and endothelial function, supporting the cardioprotective effects of Omega-3 supplementation (Bays, 2006). This suggests that as Omega-3 fatty acids levels decrease**,** there is a tendency for NT-proBNP levels to increase**,** and vice versa. The inverse relationship observed may reflect the protective cardiovascular effects of Omega-3 fatty acids**.** These actions collectively help to reduce cardiac wall stress and may influence the release or suppression of natriuretic peptides like NT-proBNP. In hypertensive individuals, lower Omega-3 levels may impair these protective mechanisms, potentially leading to increased blood pressure, worsening left ventricular strain, and subclinical myocardial dysfunction. Such conditions are usually associated with elevated NT-proBNP due to increased myocardial wall tension (Mckie *et al.*2016) The significant negative correlation, still implies that Omega-3 fatty acids may be indirectly linked to cardiac neurohormonal balance and could modulate natriuretic peptide expression in a compensatory manner

Future studies with larger sample sizes, longitudinal follow-up, and interventional trials assessing Omega-3 supplementation effects on NT-proBNP levels and hypertension outcomes will be essential to validate these findings.

5. Conclusion

Elevated NT-proBNP reflects cardiac stress, while diminished Omega-3 indicates increased cardiovascular risk. The inverse correlation between these biomarkers suggests a potential interrelationship that could influence cardiovascular risk and disease progression. Low Omega-3 levels could serve as an early risk indicator for cardiac stress in hypertensive patients. Omega-3 supplementation might be beneficial in modulating NT-proBNP expression and improving cardiovascular outcomes. Combined monitoring of NT-proBNP and Omega-3 levels could enhance the predictive accuracy of subclinical cardiac dysfunction in hypertensive populations. Given the modifiable nature of Omega-3 fatty acids levels through diet and supplementation, public health strategies that promote the consumption of Omega-3 rich foods may contribute to improved blood pressure control and cardiac function. Additionally, NT-proBNP could serve as a supplementary biomarker for stratifying hypertensive patients, although further validation is necessary. Future research should include larger, multicentric studies with longitudinal follow-up to explore causality and evaluate the therapeutic potential of Omega-3 supplementation in modifying NT-proBNP levels and cardiovascular outcomes in hypertensive populations.

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References

1. World Health Organization. Hypertension. 2021 (accessed 23 May 2025). Available from: https://www.who.int/news-room/fact-sheets/detail/hypertension
2. Bassa, F.K., Essé, C., Koné, S., Acka, F., Laubhouet-Koffi, V., Kouassi, D. *et al.*, 2019. Epidemiological links between malaria parasitaemia and hypertension: findings from a population-based survey in rural Côte d'Ivoire. *Journal of Hypertension*, 37(7), pp.1356–1364.

3. Gill, D., Seidler, T., Troughton, R.W., Yandle, T.G., Frampton, C.M., Richards, M. *et al.*, 2024. Vigorous response in plasma N-terminal pro-brain natriuretic peptide (NT-ProBNP) to acute myocardial infarction. *Clinical Science*, 106(2), pp.135–139.
4. Whelton, P.K., Carey, R.M., Aronow, W.S., Casey, D.E. Jr, Collins, K.J., Himmelfarb, C.D. *et al.*, 2018. A report of the American College of Cardiology/American Heart Association Task Force on clinical practice guidelines. *Hypertension*, 71(6), pp.13–115.
5. Maisel, A.S., Krishnaswamy, P., Nowak, R.M. *et al.*, 2002. Rapid measurement of B-type natriuretic peptide in the emergency diagnosis of heart failure. *New England Journal of Medicine*, 347(3), pp.161–167.
6. Januzzi, J.L. Jr, van Kimmenade, R., Lainchbury, J. *et al.*, 2006. NT-proBNP testing for diagnosis and short-term prognosis in acute destabilized heart failure. *European Heart Journal*, 27(3), pp.330–337.
7. Oguma, J., Ozawa, S., Kazuno, A., Nitta, M., Ninomiya, Y. and Yatabe, K., 2017. Clinical significance of the depth of invasion in superficial Barrett's esophageal adenocarcinoma. *Journal of Rare Diseases Research & Treatment*, 2(6).
8. Di Veroli, A., De Bellis, E., Rossi, V., Biagi, A., Rapisarda, V., Maurillo, L. *et al.*, 2020. Clinical use of ESAs in low-risk myelodysplastic syndromes. *Journal of Rare Diseases Research & Treatment*, 2(6).
9. Principe, M., Borgoni, S. and Novelli, F., 2021. Commentary: "Alpha-enolase (ENO1) controls alpha v/beta 3 integrin expression and regulates pancreatic cancer adhesion, invasion, and metastasis". *Journal of Rare Diseases Research & Treatment*, 2(5).
10. Mozaffarian, D., Geelen, A., Brouwer, I.A. *et al.*, 2005. Effect of fish oil on blood pressure: a meta-analysis of randomized controlled trials. *Hypertension*, 45(4), pp.432–439.
11. Calder, P.C., 2017. Omega-3 fatty acids and cardiovascular disease: evidence explained and mechanisms explored. *Clinical Science*, 131(1), pp.1–11.
12. Filipovic, M.G., Aeschbacher, S., Reiner, M.F. *et al.*, 2018. Whole blood omega-3 fatty acid concentrations are inversely associated with blood pressure in young, healthy adults. *Journal of Hypertension*, 36, p.1548.
13. Mozaffarian, D. and Wu, J.H.Y., 2012. (n-3) Fatty acids and cardiovascular health: are effects of EPA and DHA shared or complementary? *Journal of Nutrition*, 142(3), pp.614S–625S.
14. Lavie, C.J., Milani, R.V., Mehra, M.R. and Ventura, H.O., 2009. Omega-3 polyunsaturated fatty acids and cardiovascular diseases. *Journal of the American College of Cardiology*, 54(7), pp.585–594.
15. Mori, T.A. and Woodman, R.J., 2006. The independent effects of eicosapentaenoic acid and docosahexaenoic acid on cardiovascular risk factors in humans. *Current Opinion in Clinical Nutrition and Metabolic Care*, 9(2), pp.95–104.
16. Tanaka, N., Ishizaki, T. and Yoshida, H., 2009. Development of an ELISA system for quantitative determination of omega-3 fatty acid levels in human serum. *Journal of Nutritional Science and Vitaminology*, 55(4), pp.345–350.
17. Clerico, A., Emdin, M. and Giannoni, A., 2006. Analytical validation of immunoassay methods for NT-proBNP: impact on clinical practice. *Clinical Chemistry and Laboratory Medicine*, 44(3), pp.274–283.
18. McKie, P.M. and Burnett, J.C., 2016. NT-proBNP: the gold standard biomarker in heart failure. *Journal of the American College of Cardiology*, 68(22), pp.2437–2439.
19. Januzzi, J.L., Butler, J., Fonarow, G.C., Rame, J.E. and Zile, M.R., 2021. NT-proBNP as a biomarker of cardiovascular risk. *European Heart Journal*, 42(15), pp.1465–1473.
20. McKie, P.M. and Burnett, J.C., 2005. NT-proBNP: the gold standard biomarker for cardiovascular disease. *Heart Failure Clinics*, 1(3), pp.321–332.
21. Wang, T.J., Larson, M.G., Levy, D. *et al.*, 2004. Impact of obesity on plasma natriuretic peptide levels. *Circulation*, 109(5), pp.594–600.
22. Das, S.R., Drazner, M.H., Dries, D.L. *et al.*, 2005. Impact of body mass and body composition on circulating levels of natriuretic peptides: results from the Dallas Heart Study. *Circulation*, 112(14), pp.2163–2168.
23. Kario, K., Hoshide, S. and Kanegae, H., 2021. NT-proBNP as a biomarker for cardiovascular risk in hypertension. *Hypertension Research*, 44(2), pp.97–109.
24. Saravanan, P., Davidson, N.C., Schmidt, E.B. and Calder, P.C., 2010. Cardiovascular effects of marine omega-3 fatty acids. *The Lancet*, 376(9740), pp.540–550.
25. Mozaffarian, D. and Wu, J.H.Y., 2011. Omega-3 fatty acids and cardiovascular disease: effects on risk factors, molecular pathways, and clinical events. *Journal of the American College of Cardiology*, 58(20), pp.2047–2067.
26. Calder, P.C., 2020. Omega-3 fatty acids and cardiovascular disease: current evidence and future perspectives. *Nutrition, Metabolism and Cardiovascular Diseases*, 30(3), pp.284–293.

27. Adegbala, O.M., Zhang, Z. and Yano, Y., 2023. Prevalence of elevated NT-proBNP and its prognostic value by blood pressure treatment and control—National Health and Nutrition Examination Survey, 1999–2004. *American Journal of Hypertension*, 36(5), pp.396–404.
28. Bays, H.E., 2006. Safety considerations with omega-3 fatty acid therapy. *American Journal of Cardiology*, 98(4), pp.35i–43i.
29. Wang, J., Li, Y., Franklin, S.S., Safar, M.E. and Ji, Y., 2021. Aging and hypertension: mechanisms and interventions. *Journal of Clinical Hypertension*, 23(8), pp.1457–1472.
30. Ukibe, N.R., Alex, J.C., Osakue, N.O., Ukibe, E.G., Ukibe, B.C., Ukibe, V.E. *et al.*, 2024. Linking malaria and hypertension: unveiling the interconnected pathophysiological nexus. *Journal of Scientific Research*, 9(1), pp.12–19.
31. Tadesse, M., Alemu, T. and Dessie, Y., 2022. Obesity, hypertension, and cardiovascular risk: a community-based study. *BMC Public Health*, 22(1), p.1124.
32. Kearney, P.M., Whelton, M., Reynolds, K., Muntner, P., Whelton, P.K. and He, J., 2005. Global burden of hypertension: analysis of worldwide data. *The Lancet*, 365(9455), pp.217–223.
33. Abegaz, T.M., Tefera, Y.G. and Abebe, T.B., 2021. Hypertension and cardiovascular risk: a systematic review. *Journal of Hypertension Research*, 38(2), pp.112–126.
34. Peters, S.A.E., Huxley, R.R. and Woodward, M., 2020. Gender differences in hypertension and cardiovascular outcomes. *Hypertension*, 75(2), pp.432–441.
35. Miller, V.M., Reckelhoff, J.F., Romero, D.G. and Sandberg, K., 2021. Sex differences in hypertension and cardiovascular disease. *Hypertension Research*, 44(4), pp.556–568.
36. Omland, T., de Lemos, J.A., Sabatine, M.S. *et al.*, 2007. A sensitive cardiac troponin T assay in stable coronary artery disease. *New England Journal of Medicine*, 361(26), pp.2538–2547.
37. Mozaffarian, D. and Wu, J.H.Y., 2021. Omega-3 fatty acids and cardiovascular disease: effects and mechanisms. *Journal of the American Heart Association*, 10(12).
38. Adeoye, A.O., Onanuga, O.A. and Akindele, A.A., 2022. Inflammatory markers and cardiovascular risk in malaria patients. *Journal of Infectious Diseases*, 225(3), pp.425–439.
39. Calder, P.C., 2012. Mechanisms of action of (n-3) fatty acids. *Journal of Nutrition*, 142(3), pp.592S–599S.
40. Cozlea, D.L., Farcas, D.M., Nagy, A., Keresztesi, A.A., Tifrea, R., Cozlea, L. and Carașca, E., 2020. The impact of omega-3 fatty acids on cardiovascular health: a meta-analysis. *Clinical Nutrition*, 39(5), pp.1330–1340.
41. Bays, H.E., 2007. Safety considerations with omega-3 fatty acid therapy. *American Journal of Cardiology*, 99(6A), pp.35C–43C. doi:10.1016/j.amjcard.2006.11.008.
42. McKie, P.M. and Burnett, J.C., 2016. NT-proBNP: the gold standard biomarker in heart failure. *Journal of the American College of Cardiology*, 68(22), pp.2437–2439. doi:10.1016/j.jacc.2016.10.002.