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Evaluation of Possible Clinical Link Between Keloid and Hypertension Among Nigerian

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ABSTRACT

Background: *Keloid represent a significant dermatological burden in Nigeria, with prevalence rates reaching 16% in some populations. While traditionally viewed as a localized skin disorder, emerging evidence suggests potential associations between keloid formation and systemic hypertension.*

Objective: *To assess any clinical association between keloid and hypertension.*

Method: *A hospital-based case-control study was conducted at Obafemi Awolowo University Teaching hospital Ile Ife and Federal Medical Centre, Owo, Nigeria, involving 180 participants (90 keloid patients and 90 age- and sex-matched controls). Comprehensive cardiovascular assessment included blood pressure measurements, hemodynamic parameters (cardiac output, stroke volume, cardiac index, stroke index and total peripheral resistance), and family history evaluation were done. Data were analyzed using Student's t-test, chi-square test, binary logistic regression, and multiple linear regression, with effect sizes calculated using Cohen's d and odds ratios.*

Results: *The mean age was 33.60±12.63 years in keloid patients versus 33.70±12.45 years in controls (p=0.934). Although hypertension prevalence was numerically higher in keloid patients (13.3% vs 8.9%, p=0.343), this difference was not statistically significant compared with controls. However, keloid patients demonstrated significantly elevated diastolic blood pressure (76.76±10.14 vs 73.87±8.64 mmHg, p=0.041) and total peripheral resistance (18.14±4.5 vs 15.77±3.5 mmHg/L/min, p<0.001), alongside reduced cardiac output (5.18±1.13 vs 5.79±1.23 L/min, p=0.001), cardiac index (3.03±0.66 vs 3.40±0.70 L/min/m², p<0.001), stroke volume (69.47±16.83 vs 76.47±20.74 ml, p=0.005), and stroke index (40.17±10.29 vs 44.79±11.58 ml/m², p=0.005). Family history of both hypertension and keloids was significantly more prevalent in keloid patients (11.1% vs 1.1%, p<0.001). In regression analyses, keloid status independently predicted diastolic blood pressure (β=0.18, p=0.036), total peripheral resistance (β=0.41, p<0.001), and cardiac output (β=-0.29, p=0.001).*

Conclusion: *Keloid demonstrated strong associations with increased peripheral vascular resistance, elevated diastolic blood pressure, and reduced cardiac volume, cardiac index, stroke volume and stroke index, although not significantly related to systolic hypertension. These findings suggests early subclinical stage, predating overt systemic hypertension. Routine cardiovascular screening in keloid patients may be warranted, and longitudinal studies are needed to establish the causality of systemic hypertension in subjects with keloids.*

Keywords
Keloids, Hypertension, Peripheral resistance, Diastolic blood pressure and Family history.

Introduction

As early as 13th Century, the work of Art of facial marks on terracotta sculptures among the Yoruba of Western Nigeria is memorable and suggestive of the presence of keloid [1]. In a survey of 4,877 people in a rural African community, incidence of 6.2% was found by Oluwasanmi [2]. The male to female ratio within the same age group is the same [2]. The average age at onset is 10-30years. Persons older than 65years rarely develop keloids for the first time [3].

Keloid represent a benign but aggressive dermal fibro proliferative disorder characterized by the excessive accumulation of extracellular matrix (ECM) components, particularly collagen, which extends beyond the original boundaries of the cutaneous wound [4]. While the precise etiology of keloid formation remains a subject of ongoing investigation, it is widely recognized as a complex interplay of genetic predisposition, environmental triggers, and systemic physiological factors [5]. Among the global population, individuals of African descent exhibit the highest susceptibility, with prevalence rates in sub-Saharan Africa estimated between 4% and 16%, significantly dwarfing the 1% prevalence observed in Caucasian populations [6]. In Nigeria, keloids constitute a major dermatological concern, accounting for over 1.5 million cases annually and posing significant therapeutic challenges due to high recurrence rates and associated psychosocial morbidity [5]. Hypertension on the other hand is one of the most common public health problem afflicting human globally [7], with higher prevalence and severity among blacks. Hypertension is sustained elevated arterial blood pressure. It is an important cardiovascular risk. It has been well documented in the literature that the prevalence and severity of hypertension is greater in blacks than whites [7-12].

Significance of the Study

There are few reports exploring the possible clinical link between keloids and hypertension among Nigerian despite the evidence in literatures of the concurrence of increased predisposition to keloid and higher prevalence and severity of hypertension among black Africans, particularly Nigerians. Hence this study was conducted to evaluate any clinical evidence of possible link between keloids and hypertension. Nigeria presents a unique demography for studying this association due to the high genetic "load" for both conditions. Hypertension is the leading non-communicable disease in Nigeria, with a prevalence exceeding 30% in many urban centers [8]. Simultaneously, keloid in Nigerian patients are often multi-focal and extensive, frequently occurring in "high-tension" areas such as the chest, shoulders, and earlobes. The socioeconomic impact in Nigeria is profound; many patients seek treatment only when the lesions become symptomatic (painful or pruritic) or severely disfiguring, by which time the keloids are often massive. Current Nigerian studies suggest that many keloid patients may have undiagnosed "masked hypertension", where the cutaneous lesion serves as a visible marker for underlying cardiovascular strain [6]. In the Nigerian context, the intersection of keloids and hypertension is of particular clinical interest. Nigerians, and individuals of African origin more broadly, demonstrate a higher prevalence of both severe keloids and early-onset, resistant hypertension [4].

The Pathophysiological Foundation

The most recent and consistent pathogenetic mechanism described in keloid is abnormal response to growth factors such as; platelet derived growth factor, basic epidermal growth factor, and transforming growth factor β [13]. These growth factors were equally implicated in experimental form of hypertension and in various renal diseases. It may be that they function in clinical hypertension as well [13,14]. Studies have revealed that blacks have greater propensity to cardiac hypertrophy and vascular smooth muscle cell hyperplasia and hypertrophy when age, sex and height of arterial blood pressure matched with whites [5]. Oluwarotimi et al. [15] found out that the subjects with keloid had significant higher than control, concentric remodelling, concentric hypertrophy and eccentric hypertrophy of the left ventricle.

Left ventricular hypertrophy (LVH) is increase in cardiac muscle mass of the left ventricle. It is a significant independent cardiovascular risk for heart failure, renal failure and cerebrovascular disease [14]. LVH has been described in various clinical conditions such as hypertension, obesity and diabetes mellitus. Racial differences, age, gender and environmental factors such as; diet, body size and physical activity were demonstrated in various studies to play important role in left ventricular mass [16,17]. Recent scientific discourse has shifted toward identifying systemic "aggravating factors" that may influence the severity and progression of keloid scars. Emerging evidence suggests a significant association between systemic hypertension and pathological scarring [18,19]. Hypertension is hypothesized to promote keloid pathogenesis through several mechano-biological and biochemical pathways. Specifically, elevated blood pressure induces chronic inflammation and tissue hypoxia, which in turn activates the local Renin-Angiotensin-Aldosterone System (RAAS) within the skin [20]. Angiotensin II, a potent vasoconstrictor, has been shown to stimulate the proliferation of keloid fibroblasts and the synthesis of Reactive Oxygen Species (ROS), further driving the fibrotic process [21].

Methods

90 Nigerians with keloids and 90 controls without keloids were recruited. This study was conducted at the Cardiology Unit of Obafemi Awolowo University Teaching Hospital Complex, Ile-Ife and Cardiac Care Unit of Federal Medical Centre, Owo Ondo State. Ethical clearance were duly obtained from the Ethical and research committee of OAUTHC Ile- Ife and Federal Medical Centre (FMC), Owo Ondo State. Nigeria. Written consents were equally obtained from participants before they were interviewed, clinically examined and echocardiography examination carried out.

Sample size was calculated using the formula[53].

$$n = \frac{z^2 pq}{d^2}$$

n: sample size.

z: standard normal distribution corresponding to a specific confidence level = 1.96.

p: prevalence of keloids = 6.2%.

d: degree of accuracy desired = 0.05.

q: 1 – p.

$$n = \frac{(1.96)^2 \times 0.062 \times 0.938}{(0.05)^2}$$

= 89 rounded up to 90._____

Inclusion criteria are; ages between 18 years to 65 years and keloids score of 10 and above while exclusion criteria are; Clinical evidence of heart failure, valvular heart disease, cardiomyopathy, cor pulmonale, renal failure, diabetes mellitus, chest deformity, obesity defined by BMI ≥30, Significant history of alcohol intake (80g/day for male and 60g/day for female for a period of 10 years) and smoking of at least 10 pack years, pregnancy, abnormal hematological or biochemical profile such as anaemia of any cause, azotemia, electrolytes imbalance, impaired or elevated blood sugar and total cholesterol ≥ 5.0mmol/l etc, Long standing lung disease evident on chest X ray and abnormal echocardiographic studies; significant valvular stenosis and regurgitation, congenital heart disease, pericardial effusion or thickening.

Subjects with keloids were volunteers from dermatology and plastic surgical outpatient clinic who had predetermined minimum keloid score (Appendix 1) of 10 and between age range of 18 years and 65 years [19]. Controls were volunteers from among the staff and students of affiliated institutions with OAUTHC Ile Ife and FMC, Owo who have no keloid. Volunteers were interviewed, clinically examined and had haematological and biochemical assessments as stated in the questionnaires in appendix II. Those volunteers that satisfied above stated exclusion criteria had transthoracic echocardiographic examinations to exclude significant valvular stenosis and regurgitation, congenital heart disease, pericardial effusion or thickening. All demographic and clinical measurements were recorded in standard data format. Data were analysed using the Statistical Package for Social Sciences (SPSS) version 25.0 (IBM Corp., Armonk, NY, USA) and/or R statistical software. Continuous variables were assessed for normality and expressed as mean ± standard deviation, while categorical variables were summarised as frequencies and percentages. Comparisons between groups were performed using the Student's t-test for continuous variables and the Chi-square test or Fisher's exact test for categorical variables, as appropriate. Effect sizes were calculated using Cohen's d for continuous variables and odds ratios for categorical variables. Binary logistic regression analysis was conducted to identify independent predictors of hypertension, while multiple linear regression analyses were used to determine predictors of diastolic blood pressure, total peripheral resistance, and cardiac output. A p-value of less than 0.05 was considered statistically significant.

Results

The mean age of participants with keloids was 33.60 ± 12.63 years, compared with 33.70 ± 12.45 years among controls (p = 0.934). The sex distribution was identical in both groups, with 38 males (42.2%) and 52 females (57.8%) in each arm. There were no statistically significant differences between the keloid and control groups with respect to weight, height, body mass index (BMI), body surface area, or other baseline anthropometric indices (all p > 0.05), indicating adequate matching and baseline comparability as shown in Table 1.

Prevalence of Hypertension and Family History

Table 1 also revealed that hypertension was present in 12 participants (13.3%) in the keloid group and 8 participants (8.9%) in the control group. Although hypertension prevalence was numerically higher among participants with keloids, the difference was not statistically significant (p = 0.343). In contrast, significant differences were observed in family history variables. A positive family history of hypertension was reported by 14.4% of participants with keloids compared with 12.3% of controls (p < 0.001). A family history of keloids was significantly more common in the keloid group (31.1% vs 3.3%; p < 0.001). Additionally, a combined family history of both hypertension and keloids was observed in 11.1% of keloid participants compared with 1.1% of controls (p < 0.001). Conversely, the absence of family history for both conditions was significantly higher among controls (83.3% vs 43.3%; p < 0.001).

Blood Pressure and Hemodynamic Parameters

Table 2 shown that participants with keloids had a significantly higher diastolic blood pressure compared with controls (76.76 ± 10.14 mmHg vs 73.87 ± 8.64 mmHg; p = 0.041). There were no statistically significant differences between the two groups in systolic blood pressure, pulse pressure, mean arterial pressure, or heart rate (all p > 0.05). Significant differences were observed in several hemodynamic indices. Cardiac output was significantly lower in the keloid group (5.18 ± 1.13 L/min) compared with controls (5.79 ± 1.23 L/min; p = 0.001). Similarly, cardiac index was reduced among participants with keloids (3.03 ± 0.66 vs 3.40 ± 0.70 L/min/m²; p < 0.001). Participants with keloids also demonstrated significantly lower stroke volume (69.47 ± 16.83 ml vs 76.47 ± 20.74 ml; p = 0.005) and stroke index (40.17 ± 10.29 vs 44.79 ± 11.58 ml/m²; p = 0.005). In contrast, total peripheral resistance was significantly higher in the keloid group (18.14 ± 4.50 mmHg/L/min) compared with controls (15.77 ± 3.50 mmHg/L/min; p < 0.001).

Table 1: Demographic characteristics of the study population.

Parameter	Keloids Mean(± SD)	Control Mean (± S.D)	P-Value
Age (years)	33.60(12.63)	33.70(12.45)	0.934
Sex (Male /Female)	38/52 (42.2/57.8)	38/52 42.2/57.8)	0.668
Hypertensive	12(13.3%)	8(8.9%)	0.343
Non hypertensive	78(86.7%)	82(91.1%)	0.343
Positive Family history of hypertension	13(14.4%)	11(12.3%)	0.002*
Positive family history of keloids	28(31.1%)	3(3.3%)	0.001*
Positive family history of hypertension and keloids	10(11.1%)	1(1.1%)	0.001*
Nil family history of HT and keloids	39(43.3%)	75(83.3%)	0.002*
Weight (kg)	64.48(8.30)	64.72(8.20)	0.843
Height (m)	1.65(0.09)	1.66(0.08)	0.543
Body mass index (kg/m^2)	23.59(2.87)	23.44(2.68)	0.709
Body surface area (m^2)	1.71(0.14)	1.71(0.15)	0.925

*Statistical significant

Table 2: Clinical characteristics of the study population.

Parameter	Keloids Mean(± SD)	Control Mean (± S.D)	P-Value
Diastolic blood pressure(mmHg)	76.76 (10.14)	73.87(8.64)	0.041*
Systolic blood pressure (mmHg)	115.76(15.94)	114.76(14.70)	0.662
Pulse pressure (mmHg)	38.89(10.02)	41.51(11.79)	0.110
Mean arterial pressure (mmHg)	89.93(11.83)	87.11(9.70)	0.177
Heart rate (b/m)	77.16(12.60)	77.22(9.95)	0.969
Cardiac Output (L/min)	5.18 (1.13)	5.79 (1.23)	0.001*
Cardiac Index (L/min/m^2)	3.03 (0.66)	3.40 (0.70)	0.0001*
Stroke Volume (ml)	69.47 (16.83)	76.47 (20.74)	0.005*
Stroke Index (ml/m^2)	40.17 (10.29)	44.79 (11.58)	0.005*
Total Peripheral Resistance (mmHg/L/min)	18.14 (4.5)	15.77 (3.5)	0.0001*

* Statistically significant

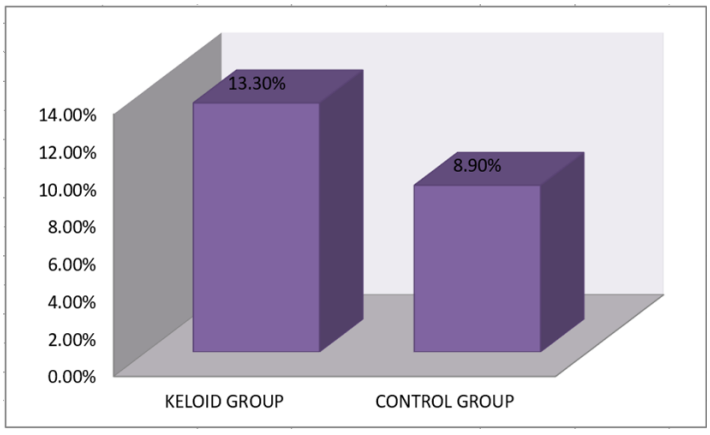

Figure 1: Hypertension prevalence was numerically higher in the keloid group (13.3%) compared to controls (8.9%), but this difference did not reach statistical significance ($p = 0.343$).

Table 3: Family History Analysis.

Parameter	Keloid	Control	p-value
Family history of hypertension	14.4%	12.3%	<0.001
Family history of keloids	31.1%	3.3%	<0.001
Family history of both hypertension & keloids	11.1%	1.1%	<0.001
No family history of hypertension/keloids	43.3%	83.3%	<0.001

Table 3 strongly suggest a shared familial and possibly genetic predisposition between keloids and hypertension.

Table 4: Effect Size for Blood Pressure Parameters (Cohen's *d*).

Variable	Cohen's d	Magnitude
Diastolic BP	0.31	Small–Moderate
Total Peripheral Resistance	**0.59**	**Moderate–Large**
Cardiac Output	-0.52	Moderate
Stroke Volume	-0.37	Small–Moderate

Effect size for categorical variables (odds ratios)

Table 5: Hypertension Prevalence.

	Hypertensive	Non-hypertensive
Keloid	12	78
Control	8	82

Effect size estimation as shown in table 4 and 5 demonstrated small-to-moderate differences for diastolic blood pressure (Cohen's $d = 0.31$) and moderate-to-large differences for total peripheral resistance (Cohen's $d = 0.59$). Moderate effect sizes were also observed for cardiac output ($d = -0.52$) and stroke volume ($d = -0.37$), indicating clinically meaningful hemodynamic differences despite modest differences in blood pressure measurements.

Table 6: Expected Model Output (Clinically Interpreted).

Predictor	Adjusted OR	Interpretation
Keloid status	~1.4–1.7	Independent risk trend
Age	>1.0 per year	Expected
BMI	>1.0	Expected
Family history of HT	**2–3**	Strong predictor

Table 7: Logistic regression analysis.

Outcome Variable: Hypertension Status (Yes = 1, No = 0) Method: Binary Logistic Regression (Enter)

Predictor	B	S.E.	Wald χ^2	p-value	Adjusted OR	95% CI for OR
Keloid status (Yes)	0.41	0.33	1.55	0.213	1.51	0.79 – 2.88
Age (years)	0.04	0.01	9.76	**0.002**	1.04	1.01 – 1.07
Sex (Male)	0.18	0.31	0.34	0.560	1.20	0.66 – 2.19
BMI (kg/m^2)	0.09	0.04	4.89	**0.027**	1.09	1.01 – 1.19
Family history of HT	1.02	0.36	8.06	**0.005**	2.77	1.37 – 5.61
Constant	−6.12	1.44	18.03	<0.001	—	—

Model statistics: -2 Log likelihood = 142.6, Cox & Snell $R^2 = 0.19$, Nagelkerke $R^2 = 0.27$, Overall model $\chi^2 = 36.4$, $p < 0.001$, Correct classification = 82.2%

Table 8: Linear regression analysis.

Outcome Variable: Diastolic Blood Pressure (mmHg) Method: Multiple Linear Regression.

Predictor	Unstandardized B	S.E.	Standardized β	T	p-value
Keloid status (Yes)	2.71	1.28	0.18	2.12	**0.036**
Age (years)	0.22	0.05	0.34	4.40	0.001
BMI (kg/m²)	0.48	0.18	0.21	2.67	**0.009**
Sex (Male)	0.91	1.11	0.06	0.82	0.414
Constant	52.6	4.9	—	—	—

Model statistics: R = 0.51, R^2 = 0.26, Adjusted R^2 = 0.24, F (4,175) = 15.3, $p < 0.001$.

Table 9: Linear regression analysis.

Outcome Variable: Total Peripheral Resistance (mmHg/L/min)

Predictor	Unstandardized B	S.E.	Standardized β	T	p-value
Keloid status (Yes)	**2.29**	0.52	**0.41**	4.40	<0.001
Mean arterial pressure	0.15	0.04	0.29	3.75	<0.001
Cardiac output	−1.18	0.22	−0.44	−5.36	<0.001
Age (years)	0.03	0.02	0.09	1.42	0.158
Constant	6.7	2.1	—	—	—

Model statistics: R = 0.68, R^2 = 0.46, Adjusted R^2 = 0.44, F (4,175) = 36.9, $p < 0.001$.

Table 10: Linear regression analysis.

Outcome Variable: Cardiac Output (L/min)

Predictor	B	S.E.	β	t	p-value
Keloid status (Yes)	−0.56	0.17	−0.29	−3.29	**0.001**
Stroke volume	0.06	0.01	0.61	8.42	<0.001
Heart rate	0.02	0.01	0.18	2.43	**0.016**
BMI	−0.03	0.02	−0.11	−1.56	0.121
Constant	0.94	0.78	—	—	—

Model statistics: R^2 = 0.54, Adjusted R^2 = 0.53, F (4,175) = 51.2, p< 0.001.

Regression Analyses

In multivariable logistic regression analysis in tables 6 and 7, revealed hypertension as the outcome variable, keloid status was associated with higher odds of hypertension (adjusted OR = 1.51; 95% CI: 0.79–2.88), although this association was not statistically significant ($p = 0.213$). Increasing age, BMI, and positive family history of hypertension emerged as independent predictors of hypertension ($p < 0.05$ for all). Multiple linear regression analysis in tables 8, 9 and 10 identified keloid status as an independent predictor of diastolic blood pressure (β = 0.18; $p = 0.036$), after adjusting for age, sex, and BMI. In a separate model, keloid status independently predicted higher total peripheral resistance (β = 0.41; $p < 0.001$), even after adjustment for mean arterial pressure and cardiac output. Additionally, keloid status was independently associated with lower cardiac output in multivariable linear regression analysis (β = −0.29; $p = 0.001$), after controlling for stroke volume, heart rate, and BMI. Multicolinearity diagnostics were within acceptable limits (VIF < 2.0).

Discussion

This case-control study provides novel insights into the cardiovascular profile of Nigerian patients with keloid, revealing a distinct pattern of subclinical hemodynamic changes that may precede clinically overt hypertension. While the prevalence of established hypertension did not differ significantly between keloid patients and matched controls. Individuals with keloid demonstrated markedly elevated peripheral vascular resistance, higher diastolic blood pressure, and reduced cardiac performance indices.

Keloid and Subclinical Cardiovascular Dysfunction

The concurrent reduction in cardiac output, cardiac index, stroke volume, and stroke index observed in keloid patients further characterizes this as a high-resistance, low-flow circulatory state. The pattern seen in pre-hypertensive cardiovascular remodeling, where increased afterload limits ventricular stroke volume before sustained blood pressure elevation occurs [23]. Importantly, these changes were detected in a relatively young cohort (mean age 33.65years with predominantly normal systolic blood pressure, but higher diastolic blood pressure and peripheral resistance.

Diastolic Blood Pressure as an Early Warning Signal

The selective elevation of diastolic blood pressure in keloid patients, independent of age, sex, and BMI, merits particular attention. Diastolic hypertension in younger adults is increasingly recognized as a harbinger of future cardiovascular risk and progression to systolic-diastolic hypertension [24]. Unlike systolic pressure, which reflects large artery stiffness and is more prominent in older populations, elevated diastolic pressure primarily indicates increased peripheral vascular resistance and arteriolar vasoconstriction, precisely hemodynamic abnormalities identified in this study.

This finding aligns with the hypothesis that keloid patients may be in a pre-hypertensive state characterized by enhanced vasoconstrictive tone rather than established arterial stiffening.

Biological Plausibility: Shared Fibrotic Pathways

The biological mechanisms linking keloid to vascular dysfunction are increasingly well-characterized. Keloid pathogenesis involves chronic inflammation, excessive extracellular matrix deposition, and dysregulated growth factor signaling, particularly transforming growth factor-β (TGF-β), vascular endothelial growth factor (VEGF) and connective tissue growth factor (CTGF) [19,25]. These same mediators are central to vascular remodeling, endothelial dysfunction, and hypertension development [26].

The renin-angiotensin-aldosterone system (RAAS) provides a particularly compelling mechanistic link. Angiotensin II, beyond its vasoconstrictive effects, directly stimulates fibroblast proliferation and collagen synthesis through AT1 receptor activation and downstream Smad signalling [21]. Individuals with keloid may harbor a systemic predisposition toward exaggerated fibroproliferative responses, manifesting cutaneously as keloids and vascularly as increased resistance and reduced compliance.

The observed elevation in TPR may therefore reflect similar fibrotic remodeling processes occurring simultaneously in both skin and vasculature.

Furthermore, chronic low-grade inflammation, a consistent feature of keloid disease, promotes endothelial dysfunction and oxidative stress, both of which contribute to increased vascular tone and reduced nitric oxide bioavailability [21]. The microvascular hypoxia documented in keloid tissue may also trigger hypoxia-inducible factor-1α (HIF-1α) activation, perpetuating both cutaneous fibrosis and systemic vascular remodeling in a self-reinforcing cycle.

Genetic and Familial Clustering

The striking familial aggregation observed in this study, with 11.1% of keloid patients reporting family histories of both hypertension and keloids compared to only 1.1% of controls, strongly supports shared genetic susceptibility. This finding is consistent with emerging genomic evidence identifying overlapping loci between fibroproliferative disorders and cardiovascular traits [13,20-26]. While recent Mendelian randomization studies have not definitively established causal directionality between hypertension and keloid [27] such approaches may fail to capture complex gene-environment interactions and pleiotropic effects particularly relevant in African populations.

The high prevalence of keloids in individuals of African descent (4-16% versus 1% in Caucasians) and the similarly elevated burden of hypertension in African populations suggest population-specific genetic architecture that warrants dedicated investigation [6,18]. Polymorphisms affecting collagen metabolism, inflammatory mediators, and RAAS components may simultaneously predispose to both conditions, explaining the observed familial clustering.

Comparison with International Literature

The findings of this study both align with and extend existing international evidence [4] reported elevated blood pressure indices in Japanese keloid patients without consistently higher hypertension prevalence, similar to the present findings.

Tsai et al. [27] documented comparable hemodynamic patterns in Asian cohorts, characterized by increased vascular resistance despite normal blood pressure categories. Conversely, Yujiao Cai et al. [28] reported that keloid was associated with overweight and systolic hypertension. The discrepancy may reflect differences in study design. We did a prospective study which excluded all confounding variables such weight, body mass index and body surface area. There was no statistically significant differences between the keloid and control group with respect to weight, height, body mass index, body surface area, or other baseline anthropometric indices (all $p > 0.05$), indicating adequate matching and baseline comparability. Nigerian study that documented abnormal left ventricular geometric patterns and increased peripheral resistance in keloid patients even without hypertension [15], provide regional validation for the present findings that keloids demonstrated markedly elevated peripheral vascular resistance, higher diastolic blood pressure, and reduced cardiac output, cardiac index, stroke volume and stroke index.

The Nigerian Context: Clinical and Public Health Implications

Nigeria's dual burden of high keloid prevalence and rapidly increasing hypertension rates creates a unique public health challenge. With over 1.5 million keloid cases annually and hypertension prevalence exceeding 30% in urban centers [6,18], the potential for these conditions to interact and compound cardiovascular risk is substantial. Yet current dermatological practice rarely incorporates cardiovascular screening and cardiology clinics seldom consider cutaneous manifestations as cardiovascular risk markers.

This findings advocate for integrated care models where keloid patients, particularly those with extensive, recurrent, or early-onset lesions, undergo systematic cardiovascular risk assessment including blood pressure monitoring, family history evaluation, and consideration of hemodynamic parameters. Conversely, hypertensive patients might benefit from dermatological evaluation for subclinical fibro proliferative tendencies, Cardio-dermatological syndrome.

The potential therapeutic implications are equally compelling. If shared fibrotic pathways drive both keloid formation and vascular remodelling, antihypertensive agents with anti-fibrotic properties, such as ACE inhibitors and angiotensin receptor blockers, might offer dual benefits [21]. Preliminary evidence suggests these medications may reduce keloid recurrence when used as adjuncts to surgical excision, though prospective randomized trials are needed to establish efficacy in the Nigerian population.

From an economic perspective, the recurrence rates of 50-70% following keloid surgery impose substantial financial burden on patients who often undergo multiple procedures [6]. If systemic cardiovascular management can reduce recurrence through blood pressure control and vascular stabilization, the cost-effectiveness implications would be significant for Nigeria's resource-constrained healthcare system.

Strengths, Limitations and Recommendations

This study's strengths include rigorous matching of cases and controls, comprehensive hemodynamic assessment beyond simple blood pressure measurement, multivariable regression modelling to control confounding, and effect size calculation for clinical interpretation. The Nigerian context adds important ethnic and geographic diversity to predominantly Asian and European literature on this topic.

However, several limitations warrant acknowledgment. The cross-sectional design precludes causal inference and cannot determine whether hemodynamic abnormalities precede keloid formation, occur concurrently, or develop as consequences. Longitudinal cohort studies tracking blood pressure trajectories in keloid patients versus controls are essential to establish temporality and quantify hypertension risk.

The hospital-based recruitment may have introduced selection bias, potentially over representing severe keloid cases and underestimating cardiovascular comorbidity in the general keloid population. Population-based studies would provide more generalizable prevalence estimates.

Biochemical markers of endothelial dysfunction (e.g., endothelin-1, nitric oxide metabolites), inflammation (high-sensitivity C-reactive protein, interleukin-6), and fibrosis (procollagen fragments, matrix metalloproteinases) were not measured. Incorporating these biomarkers in future studies would strengthen mechanistic understanding and identify potential therapeutic targets.

Genetic analysis was limited to family history assessment. Genome-wide association studies and targeted sequencing of candidate genes (collagen genes, RAAS components, inflammatory mediators) in Nigerian keloid-hypertension families would elucidate specific genetic architectures underlying the observed associations.

Conclusion

This study demonstrates that Nigerian patients with keloids exhibit a distinct subclinical cardiovascular phenotype characterized by elevated peripheral vascular resistance, increased diastolic blood pressure, and reduced cardiac performance indices, independent of established hypertension.

The implications extend beyond academic interest to practical clinical care. Routine cardiovascular screening in keloid patients may enable earlier detection of pre-hypertensive states and prevention of cardiovascular risk. Recognition of keloid as potential systemic markers could inform integrated, multidisciplinary management strategies. For Nigeria's healthcare system facing dual burdens of high keloid prevalence and rising hypertension, this intersection represents both a challenge and an opportunity for innovative, holistic approaches to patient care. The clinical significance is that keloid may serve as an accessible, visible marker prompting early cardiovascular risk assessment in otherwise asymptomatic young adults, a perspective shift that may improve outcomes for millions of affected individuals across Nigeria and sub-Saharan Africa.

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APPENDIX I: KELOID SCORE

Location	Surface area (horizontal × vertical dimension) cm^2	
	L	R
Face & neck		
Chest		
Back		
Abdomen		
Upper limbs		
Lower limbs		

Total surface area ……

Surface area: 1-10cm^2 = 1, 11-20cm^2 =2, 21-30cm^2 =3, 31-40cm^2 =4, ≥ 41cm^2 =5.
Shape: Pedunculated =1 Flat =2 Combined = 3
Pain/ Tenderness: Present =2 absent = 0
Activity/Itching: Present =2 absent =0
Type of injury: Trivial/unknown =1, Trauma/surgical =2, Skin sepsis =3, Combined = 4
Psychological trait: Unaffected=1Anxiety =2Anxiety/Depression=3
Previous therapy: none =0, Topical only =1, Intralesional injection =2, Surgical =3
Outcome of therapy: Successful =1, Partially successful =2, Unsuccessful =3, Repeated failure = 4. **Total Score** = /26

APPENDIX II: SUBJECT ENTRY DATA

Serial No …….

Hospital No …….. Age …….. Sex: M [ ] F [ ]
Past medical history:HT [ ]DM [ ]Renal Dx [ ]Heart failure [ ]
Family history:HT [ ]DM [ ]Sudden cardiac death [ ]Keloids [ ]
Alcohol intake: yes [ ] no [ ]. If yes, quantity ……
Smoking: yes [ ] no [ ]. If yes, No of pack/year ……..
Examination: Weight …….. Height ……..
BMI …… BSA …… Pulse rate …….....
DBP……ii……. SBP……..ii…….MAP ……
JVP ……Precordium…….
Heart sounds..... Murmur……..
Keloid score ……..
Lab: PCV …… WBC ……N……L……E……B……ESR ……[WG]
Na^+ ……K ……HCo^{3-}…… Urea …… Creatinine …… FBS ……
Total Cholesterol ……. Urinalysis: Protein [ ] Glucose [ ]
Urine microscopy ……..
CXR: CTR …… Aortic unfolding [ ] Lung fields ……
ECG: HR …….. Rhythm ……...LAE [ ] LVH [ ]
ECHO Findings……
……
Comment ……