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Lipid Panel Predictors Of Decubitus Ulcer Risk In Elderly Residents Of Bina Bhakti Nursing Home

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Abstract. *Decubitus ulcers in the elderly arise from prolonged pressure, reduced mobility, and skin compromise, increasing morbidity and costs. Risk assessment includes clinical tools like the Braden Scale and biochemical markers such as lipid profiles and apolipoproteins. Age-related vascular and skin changes, along with lipid imbalances, further influence ulcer risk. To evaluate the relationship between lipid panel components and the risk of decubitus ulcers in elderly residents at Bina Bhakti Nursing Home using the Braden Scale. A cross-sectional analytic study included 93 elderly participants aged over 60 years. Lipid profiles were measured via enzymatic and immunoturbidimetric assays, including HDL, LDL, triglycerides, total cholesterol, ApoA, and ApoB, alongside their ratios. The Braden Scale was employed to assess ulcer risk. Non-parametric Spearman correlation and multiple regression analyses were conducted using SPSS version 26 to identify significant predictors, with statistical significance set at $p<0.05$. Significant positive correlations were found between decubitus ulcer risk and ApoA ($p=0.024$), ApoB ($p=0.023$), total cholesterol ($p=0.033$), and LDL ($p=0.013$). Conversely, age showed a significant negative correlation ($p=0.027$). Lipid ratios, including ApoB/ApoA and triglyceride/HDL, did not demonstrate statistical significance. Multiple regression highlighted ApoB, ApoB/ApoA ratio, and LDL/ApoA ratio as independent predictors, emphasizing their role in vascular health and tissue integrity. Elevated ApoB and unfavorable lipid ratios are key predictors of increased decubitus ulcer risk, likely due to their contribution to endothelial dysfunction and impaired circulation. Addressing lipid imbalances through targeted interventions could enhance tissue health and reduce ulcer susceptibility in the elderly population.*

Keywords: *Apolipoprotein, Braden Scale, Decubitus Ulcer, Elderly, Lipid Profil*

1. INTRODUCTION

Decubitus ulcers, or bedsores, are a prevalent issue among elderly individuals, particularly those in nursing homes, resulting from prolonged pressure, reduced mobility, and compromised skin health. (Rosmarwati & Mulianto, 2023) These ulcers not only impair quality of life but also lead to increased healthcare costs and mortality risk. (Livesley & Chow, 2002) Accurate and comprehensive risk assessment is essential for effective prevention, especially in vulnerable populations such as elderly residents of nursing homes. (Yusharyahya et al., 2023)

The Braden Scale remains a widely used tool for assessing decubitus ulcer risk, focusing on clinical factors such as sensory perception, moisture, mobility, activity, nutrition, and friction or shear. (Lyder et al., 1999) However, biochemical markers, including lipid

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profiles and associated ratios, have emerged as potential predictors of decubitus ulcer risk due to their role in tissue perfusion, inflammation, and skin integrity.

Age, a key factor in decubitus ulcer risk, contributes to reduced skin elasticity, slower wound healing, and impaired vascular function. In addition to age, lipid-related markers such as apolipoprotein A (apo A), apolipoprotein B (apo B), and their ratio (apo A/apo B) are crucial indicators of lipid metabolism and cardiovascular health. Abnormalities in these markers may influence tissue perfusion and inflammatory responses, thereby affecting decubitus ulcer development. (Lima Serrano et al., 2017)

High-density lipoprotein (HDL), often referred to as "good cholesterol," plays a protective role in skin health by enhancing vascular function and reducing inflammation. (Schwertani et al., 2018) Conversely, low-density lipoprotein (LDL) and triglycerides are associated with impaired vascular perfusion and increased oxidative stress, factors that may exacerbate decubitus ulcer risk. (Liu et al., 2022) Ratios such as triglyceride/HDL and LDL/apo A provide additional insights into lipid balance and their potential impact on tissue health and healing capacity. (Bonacina et al., 2021)

This study aims to investigate the relationship between age, lipid panel components containing HDL, LDL, total cholesterol, triglycerides, apolipoproteins (apo A, apo B), and their associated ratios such as apo A/apo B, triglyceride/HDL, LDL/apo A, with decubitus ulcer risk as assessed by the Braden Scale. (Zhetmekova et al., 2024) By integrating clinical and biochemical parameters, this research seeks to provide a more holistic understanding of decubitus ulcer risk predictors, enabling improved prevention strategies for elderly residents of Bina Bhakti Nursing Home. (Gedamu et al., 2014)

2. LITERATURE REVIEW

Pressure ulcers, or bedsores, develop due to sustained pressure on the skin and underlying tissues, leading to impaired blood supply and tissue oxygenation. The relationship between Apo-A, Apo-B, LDL, HDL, triglycerides, and total cholesterol levels with pressure ulcer risk can be explained through their effects on vascular conditions and blood flow, which influence tissue perfusion. Apo-A serves as the primary component of HDL, known as "good" cholesterol. HDL transports cholesterol from peripheral tissues to the liver for breakdown, maintaining vascular health and preventing atherosclerotic plaque buildup. High Apo-A levels, which reflect increased HDL levels, enhance blood flow to tissues, ensuring proper nutrition and oxygenation. This process helps reduce pressure ulcer risk by preserving vascular integrity and promoting wound healing. Apo-B functions as the main component of

LDL, which transports cholesterol from the liver to body tissues. High Apo-B levels strongly correlate with increased LDL levels, often referred to as "bad" cholesterol. Excess LDL contributes to atherosclerotic plaque formation along blood vessel walls, narrowing the vessel lumen and restricting blood flow. This reduction in blood circulation worsens tissue perfusion, leading to tissue damage and elevating pressure ulcer risk, particularly among the elderly.(Amini et al., 2022; Bates-Jensen et al., 2019; McLaughlin et al., 2022)

LDL functions as a lipoprotein that transports cholesterol from the liver to body tissues. Increased LDL levels cause cholesterol accumulation in arterial walls, forming atherosclerotic plaques that narrow blood vessels and reduce blood flow to peripheral tissues. This process, known as atherosclerosis, restricts circulation and impairs tissue perfusion. In patients vulnerable to pressure ulcers, reduced peripheral blood flow due to atherosclerosis delays wound healing and makes tissues more susceptible to damage from prolonged pressure. HDL plays a protective role in the cardiovascular system by clearing cholesterol from arteries and returning it to the liver for processing. Low HDL levels increase the risk of cholesterol buildup in arteries, worsening atherosclerosis and further reducing tissue perfusion. In patients at risk of pressure ulcers, low HDL levels slow oxygen and nutrient delivery to pressure-exposed tissues, heightening the likelihood of ulcer formation.(Amini et al., 2022; Beder, 2023; Dirgar, 2024)

Triglycerides circulate in the blood as a primary energy source. However, elevated triglyceride levels often correlate with insulin resistance and metabolic syndrome, which impair endothelial function and increase the risk of atherosclerosis. High triglyceride levels also disrupt microcirculation in peripheral tissues, reducing the supply of nutrients and oxygen essential for wound healing and increasing the risk of pressure ulcers in patients with limited mobility. Total cholesterol comprises the overall levels of LDL, HDL, and other lipid components in the blood. Elevated total cholesterol, particularly when characterized by increased LDL and decreased HDL, promotes atherosclerotic plaque formation, which narrows blood vessels and disrupts blood flow to peripheral tissues. This condition heightens the risk of pressure ulcers by impairing oxygen and nutrient delivery necessary for tissue integrity, slowing healing, and worsening tissue damage caused by prolonged pressure.(Aghazadeh et al., 2020; Beder, 2023; Hossain et al., 2022)

3. METHODS

This cross-sectional analytic study, conducted at Bina Bhakti Nursing Home, examined the relationship between lipid profile components and the risk of decubitus ulcers in elderly individuals. Participants were selected based on the following inclusion criteria: (1) sign an informed consent, (2) full participation in the study, and (3) age over 60 years. Exclusion criteria ruled out individuals with conditions or factors that could affect lipid values, such as hereditary disorders or use of cholesterol-lowering medications.

The risk of decubitus ulcers was evaluated using the BRADEN Questionnaire, a validated tool assessing sensory perception, moisture, activity, mobility, nutrition, and friction or shear. Independent variables included lipid profile components measured with enzymatic assays: High-Density Lipoprotein (HDL), Low-Density Lipoprotein (LDL), Triglycerides, and Total Cholesterol, as well as Apolipoprotein A (Apo-A) and Apolipoprotein B (Apo-B), assessed using the immunoturbidimetric method. Venous blood samples were collected following standard protocols to ensure accurate and reliable measurements.

Data analysis was performed using SPSS version 26. The Kolmogorov-Smirnov test confirmed the data were not normally distributed, leading to the use of Spearman's Rho for nonparametric correlation analysis, with statistical significance set at $p < 0.05$. Additional analyses, including Spearman's Rho and Multiple Regression, evaluated lipid profile components as predictors of decubitus ulcer risk. The predictive equation was interpreted through unstandardized beta (β) values, quantifying each predictor's impact on the risk, providing valuable insights into the observed relationships.

4. RESULTS AND DISCUSSION

This study conducted 93 respondents with the majority of women, accounting for 79.6% of the total. The average age of the participants is 74.19 years. Mean apolipoprotein A levels is 155.59 mg/dL, and the mean apolipoprotein B level is 93.20 mg/dL, resulting in a mean apo B/apo A ratio of 0.60. The participants average HDL level is 44.70 mg/dL, while the mean total cholesterol level is 160.19 mg/dL. The mean LDL level is 95.00 mg/dL, and the mean triglyceride level is 100.40 mg/dL. For lipid-related ratios, the participants have an average triglyceride/HDL ratio of 2.40 and an LDL/apo A ratio of 0.62. (Table 1)

Table 1. Respondents' Characteristics

Variables	Results
Gender:	
- Women	74 (79.6)
- Men	19 (20.4)
Age, mean (SD) years	74.19 (7.96)
Apo A, mean (SD) mg/dL	155.59 (15.67)
Apo B, mean (SD) mg/dL	93.20 (12.56)
Ratio Apo B / Apo A, mean (SD)	0.60 (0.10)
HDL, mean (SD) mg/dL	44.70 (12.12)
Total Cholesterol, mean (SD) mg/dL	160.19 (29.28)
LDL, mean (SD) mg/dL	95.00 (27.96)
Triglyceride, mean (SD) mg/dL	100.40 (29.51)
Ratio Triglyceride / HDL, mean (SD)	2.40 (0.89)
Ratio LDL / Apo A, mean (SD)	0.62 (0.20)

The Spearman correlation analysis reveals significant relationships between several parameters and the Braden Scale score for decubitus ulcer risk. Age demonstrates a significant negative correlation with decubitus ulcer risk ($p = 0.027$), indicating that older age is associated with a higher risk. Apolipoprotein A and apolipoprotein B show significant positive correlations with the Braden Scale score, with p-values of 0.024 for Apolipoprotein-A with $r = 0.235$ and p-values of 0.023 for Apolipoprotein-B with $r = 0.236$, respectively, suggesting their potential role in influencing decubitus ulcer risk.

Total cholesterol also exhibits a significant positive correlation ($p = 0.033$; $r – 0.221$), as does LDL cholesterol ($p = 0.013$; $r = 0.257$), emphasizing their potential impact on decubitus ulcer risk. Conversely, the ratio of apo B to apo A, HDL, triglycerides, and the triglyceride/HDL ratio do not show significant correlations, indicating no meaningful association with decubitus ulcer risk in this study. (Table 2)

Table 2. Correlation Between Lipid Panel and the Risk of Decubitus Ulcer in Elderly

Parameter (N=93)	Pressure Injury Risk Score (BRADEN Scale)	
	r-correlation (spearman)	p-value
Age	-0,229	**0,027***
Apolipoprotein A	0,235	**0,024***
Apolipoprotein B	0,236	**0,023***
Ratio Apo B/Apo A	0,032	**0,758**
High Dense Lipoprotein (HDL)	-0,117	**0,265**
Total cholesterol	0,221	**0,033***
Low Dense Lipoprotein (LDL)	0,257	**0,013***
Triglyceride	0,066	**0,527**
Ratio triglyceride/HDL	0,099	**0,346**
Ratio LDL/ApoA	0,158	**0,131**

The results from the multiple linear regression analysis highlight several significant findings. Apolipoprotein B positively influences the dependent variable, as indicated by a significant relationship (p = 0.000). In contrast, the Ratio Apo B/Apo A shows a negative and significant impact (p = 0.001; B = -0.592), suggesting it is inversely associated with the outcome. Additionally, the Ratio LDL/ApoA displays a positive but less pronounced effect, with statistical significance (p = 0.025; B = 0.267). (Table 3)

Table 3. Multiple Linear Regression of Lipid Panel to Risk of Decubitus Ulcer in Elderly

Coefficients[a]						
Model		**Unstandardized Coefficients**		**Standardized Coefficients**	**t**	**Sig.**
		B	**Std. Error**	**Beta**		
1	**(Constant)**	31.425	20.924		1.502	**0.137**
	Age	-0.049	0.053	-0.100	-0.924	**0.358**
	Apolipoprotein A	-0.124	0.149	-0.499	-0.830	**0.409**
	Apolipoprotein B	0.429	0.305	1.383	1.405	**0.164**
	Ratio Apo B/Apo A	-61.478	43.192	-1.515	-1.423	**0.158**
	High Dense Lipoprotein (HDL)	0.057	0.116	0.176	0.488	**0.627**
	Total cholesterol	-0.002	0.064	-0.013	-0.028	**0.978**
	Low Dense Lipoprotein (LDL)	-0.042	0.196	-0.299	-0.212	**0.833**
	triglyceride	-0.041	0.040	-0.308	-1.026	**0.308**
	Ratio triglyceride/HDL	2.092	1.706	0.478	1.226	**0.224**
	Ratio LDL/ApoA	10.839	27.928	0.544	0.388	**0.699**
8	**(Constant)**	12.071	2.834		4.259	**0.000**
	Apolipoprotein B	0.176	0.049	0.568	3.633	**0.000**
	Ratio Apo B/Apo A	-24.035	6.909	-0.592	-3.479	**0.001**
	Ratio LDL/ApoA	5.324	2.343	0.267	2.272	**0.025**

a. Dependent Variable: BRADEN Scale

The multiple linear regression analysis began with a comprehensive model incorporating a broad range of variables theorized to influence Braden scoring for ulcer risk. These included demographic and biochemical factors, such as age, apolipoproteins, lipid

ratios, and markers of lipid metabolism. From a theoretical perspective, these variables were selected for their potential relevance to tissue health, circulation, and inflammatory processes, which are critical in the development and progression of ulcers. However, the initial step of the analysis revealed that most variables did not exhibit statistically significant associations with Braden scoring. Despite their biological plausibility, factors such as age, high-density lipoprotein, and total cholesterol showed limited explanatory power within the context of this model.

Age is widely recognized as a risk factor for tissue vulnerability due to its association with physiological changes such as reduced skin elasticity, impaired microvascular function, and slower wound healing. (Ensrud, 2013) Older individuals are more likely to experience comorbidities that contribute to poor circulation and reduced tissue resilience. (Bayraktar & Dal Yilmaz, 2018) However, the lack of statistical significance for age in this model suggests that its effects may be mediated through other variables, such as lipid imbalances or inflammatory markers. (Driemeier et al., 2012) Additionally, the homogeneity of the age range in the sample or the relatively small impact of age compared to other predictors could explain why it did not contribute significantly to the model. (Campbell et al., 2016)

HDL is often referred to as “good cholesterol” because of its role in reverse cholesterol transport and its anti-inflammatory and antioxidative properties. From a biological perspective, higher HDL levels are thought to protect against vascular dysfunction and support tissue health by promoting endothelial repair and reducing oxidative stress. However, in this analysis, HDL did not show a significant association with Braden scoring. This may be due to several factors: the protective effects of HDL might not be directly related to the specific pathways influencing tissue breakdown; the variability in HDL functionality, which is not captured by simple concentration measures; or the possibility that the protective role of HDL is already accounted for through related variables, such as the ApoB/ApoA ratio. (Liu et al., 2022)

Total cholesterol represents a composite measure of all lipoprotein cholesterol, including both atherogenic and anti-atherogenic fractions. While elevated total cholesterol is associated with cardiovascular risk, it provides limited information about the balance between harmful and protective lipid particles. (Khil et al., 2023) Total cholesterol may not have emerged as significant because it lacks specificity. The more nuanced measures, such as the ApoB/ApoA ratio or LDL levels, may better capture the mechanisms by which lipids influence vascular and tissue health. (Sucato et al., 2024)

Through an iterative process of refinement, the final model identified three variables as significant predictors of Braden scoring: apolipoprotein B, the ratio of apolipoprotein B to apolipoprotein A, and the ratio of low-density lipoprotein to apolipoprotein A. Apolipoprotein B demonstrated a strong positive association with Braden scoring, reflecting its critical role in lipid transport and vascular health. Theoretically, elevated levels of this marker may contribute to impaired circulation and endothelial dysfunction, both of which are central to tissue vulnerability and ulcer formation. The ratio of apolipoprotein B to apolipoprotein A, a measure of atherogenic versus anti-atherogenic lipoproteins, showed a negative association with Braden scoring. This suggests that an imbalance favoring atherogenic lipoproteins may increase susceptibility to ulcers by reducing the protective effects of anti-atherogenic factors. Similarly, the ratio of low-density lipoprotein to apolipoprotein A indicated a significant association, reinforcing the role of lipid-related variables in predicting ulcer risk.

Apolipoprotein B (ApoB) is a structural protein found in atherogenic lipoproteins, including low-density lipoprotein (LDL), very low-density lipoprotein (VLDL), and intermediate-density lipoprotein (IDL). (Glavinovic et al., 2022) It plays a critical role in lipid metabolism by acting as a ligand for cellular receptors that mediate the uptake and clearance of cholesterol and triglyceride-rich lipoproteins. Its presence in these particles makes ApoB a direct measure of the number of atherogenic lipoprotein particles in circulation, as each lipoprotein particle contains one molecule of ApoB. (Soffer et al., 2024)

The association of ApoB with Braden scoring can be interpreted through its impact on vascular health and tissue integrity. Elevated levels of ApoB are indicative of an increased number of atherogenic lipoproteins, which can lead to the development of atherosclerotic plaques. (Ashmaig et al., 2011) These plaques contribute to endothelial dysfunction, a condition where the inner lining of blood vessels fails to regulate vascular tone, blood flow, and immune response effectively. Endothelial dysfunction reduces tissue perfusion, compromising the delivery of oxygen and nutrients to the skin and subcutaneous tissues, which are essential for maintaining tissue health and repair mechanisms. (Barallobre-Barreiro et al., 2016)

Impaired circulation caused by high ApoB levels may exacerbate tissue vulnerability by creating an environment of hypoxia and nutrient deprivation. (Dorobanțu et al., 2023) This environment can hinder the ability of tissues to withstand pressure and shear forces, which are critical factors assessed in Braden scoring. (Carmeliet et al., 1998) Furthermore, chronic

inflammation associated with elevated ApoB levels may contribute to tissue damage, further increasing the risk of ulceration. (Aalto-Setalälä et al., 1994)

The ratio of apolipoprotein B (ApoB) to apolipoprotein A (ApoA) serves as a robust marker of the balance between atherogenic and anti-atherogenic lipoproteins in the bloodstream.n(Glavinovic et al., 2022) ApoB is a primary component of atherogenic lipoproteins, such as low-density lipoprotein (LDL) and very low-density lipoprotein (VLDL), which are implicated in promoting plaque formation and cardiovascular risk. (Dorobanțu et al., 2023) In contrast, ApoA is the principal structural protein in high-density lipoprotein (HDL), which is known for its protective, anti-atherogenic roles, including reverse cholesterol transport and anti-inflammatory effects. (Soffer et al., 2024)

The negative association between the ApoB/ApoA ratio and Braden scoring highlights the importance of maintaining a favorable lipoprotein balance for tissue health and ulcer prevention. A higher ApoB/ApoA ratio indicates an imbalance that favors atherogenic particles, reflecting increased vascular and metabolic stress. (Saputri et al., 2017) This imbalance can lead to endothelial dysfunction, reduced blood flow, and impaired nutrient and oxygen delivery to peripheral tissues, all of which are critical factors contributing to tissue breakdown and ulcer formation. (Handayani & Sargowo, 2017)

From a mechanistic standpoint, a high ApoB/ApoA ratio exacerbates the risk of lipid deposition in arterial walls, promoting atherosclerosis and reducing microvascular circulation. Poor microvascular function diminishes the skin's ability to withstand pressure and shear forces, making tissues more susceptible to ischemia and damage. (Zheng et al., 2019) In contrast, a lower ApoB/ApoA ratio suggests a relative abundance of protective HDL particles, which support vascular health by facilitating cholesterol efflux, reducing oxidative stress, and promoting endothelial repair. These processes enhance tissue resilience and repair capacity, reducing the likelihood of pressure ulcers or other forms of tissue injury. (Park et al., 2011)

The significance of this ratio in Braden scoring reflects its role as a surrogate marker for systemic health, particularly vascular and metabolic integrity. It underscores the need to address not only individual lipid parameters but also their relative balance when assessing risk factors for tissue breakdown and ulceration. Clinically, interventions aimed at lowering the ApoB/ApoA ratio, such as increasing HDL levels through lifestyle changes or pharmacological agents and reducing LDL levels with statins or PCSK9 inhibitors—may have the potential to improve tissue health and reduce ulcer risk. (Zanotti et al., 2023)

The significant association of the LDL/ApoA ratio with Braden scoring emphasizes the importance of this balance in determining tissue health and susceptibility to ulcers. A higher LDL/ApoA ratio suggests a predominance of atherogenic LDL over the protective effects of HDL, reflecting increased vascular risk. (Saputri et al., 2017) This imbalance likely contributes to endothelial dysfunction, reduced capillary blood flow, and diminished oxygen and nutrient delivery to the skin and underlying tissues. These impairments create an environment conducive to tissue breakdown, particularly under conditions of sustained pressure or shear forces, which are key factors in the development of pressure ulcers. (Handayani & Sargowo, 2017)

From a theoretical standpoint, the LDL/ApoA ratio can be seen as an integrative marker of systemic lipid homeostasis and vascular function. A high ratio indicates not only an excess of LDL particles but also potentially suboptimal HDL function.(Zanoni et al., 2016) HDL plays a crucial role in reverse cholesterol transport, anti-inflammatory signaling, and endothelial repair. When HDL levels are low relative to LDL, these protective mechanisms may be insufficient to counteract the deleterious effects of LDL on vascular and tissue health. (Barter et al., 2004)

Clinically, this finding reinforces the need to address lipid imbalances when assessing and managing ulcer risk. Strategies to reduce the LDL/ApoA ratio could include lowering LDL levels through lipid-lowering therapies, such as statins, PCSK9 inhibitors, or dietary modifications, while simultaneously enhancing ApoA or HDL levels via lifestyle changes, such as increased physical activity, weight management, and dietary interventions. These approaches may improve vascular function and tissue resilience, thereby reducing the risk of ulcers. The association of the LDL/ApoA ratio with Braden scoring also underscores its potential as a biomarker for ulcer risk stratification. (Ulloque-Badaracco et al., 2022)

5. CONCLUSION

This study highlights the critical role of lipid-related variables in predicting Braden scoring for ulcer risk. Apolipoprotein B, the ratio of apolipoprotein B to apolipoprotein A, and the ratio of low-density lipoprotein to apolipoprotein A emerged as significant predictors, reflecting their influence on vascular health, lipid balance, and tissue integrity. Elevated apolipoprotein B levels and unfavorable lipid ratios likely contribute to endothelial dysfunction, impaired circulation, and reduced tissue resilience, increasing susceptibility to pressure injuries. These findings underscore the importance of addressing lipid imbalances through targeted interventions, such as optimizing LDL and HDL levels, to enhance tissue

health and reduce ulcer risk. Furthermore, integrating these lipid markers into clinical assessments could improve risk stratification and inform preventive strategies for pressure ulcers.

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