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ORIGINAL PAPER

Clinical purpura and elastosis and their correlation with skin tears in an aged population

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Abstract

The previous research reported the results of a prospect cohort study that used logistic regression analysis to construct a risk prediction model for skin tears in individuals aged over 65 years. The model identified three baseline individual characteristics (male gender, history of STs, and history of falls) and two baseline skin manifestations (purpura and elastosis) that predicted the risk of dorsal forearm skin tears. This paper outlines the relationships between baseline skin manifestations and the risk of skin tears. Univariable logistic regression analysis was conducted of all the baseline data collected from the same-study participants to identify variables that significantly predicted purpura and elastosis at baseline. Amongst the 173 participants, 71 (41%) developed one or more skin tears, and in these participants, 52 (73.2%) displayed purpura, 41 (57.8%) had elastosis, and 30 (42.3%) exhibited both manifestations of the dorsal forearm at baseline. Four individual characteristics (age, history of skin tears, history of falls, and antiplatelet therapy) and three skin properties (pH, subepidermal low echogenicity band of the forearms, and skin thickness) were found to predict the risk of purpura. Conversely, three individual variables (age, gender, and smoking), three clinical skin variables (uneven skin pigmentation, cutis rhomboidalis nuchae, and history of actinic keratosis) and one skin property variable (collagen type IV) predicted the risk of skin elastosis. Progressive changes to the skin's structural and mechanical properties from the underlying effects of chronological ageing, and environmental and lifestyle-related influences increased the risk of purpura and elastotic skin manifestations and concomitantly increased risk of skin tears amongst participants.

Keywords Ageing skin · Clinical manifestations · Elastosis · Purpura · Skin tears

Abbreviations

AGEs	Advanced glycation end products
AK	Actinic keratosis
CI	Confidence intervals
CML	Carboxymethyllysine
ECM	Extracellular matrix
GAGs	Glycosaminoglycans
M	Mean
MMP-2	Matrix metalloproteinase-2
OR	Odds ratio
r	Pearson's product–moment correlation coefficient
ROC	Receiver operator characteristic curve
SD	Standard deviation
SLEB	Subepidermal low echogenicity band
STs	Skin tears
TEWL	Transepidermal water loss
TNF-α	Tumour necrosis factor-alpha
UV	Ultraviolet
VE	Viscoelasticity

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Background

Identification of factors that contribute to and are proposed to predict the risk of STs in older adults has been extensively reported over the last 3 decades [43, 53, 68, 80]. Although these individual and skin-related factors include reduced dermal thickness, decreased Braden Scale score, bruising,

senile purpura, history of skin tear, ageing, and gender, they are not consistently described in the literature [44, 48, 51, 62, 80]. STs can impact on the individual in terms of morbidity, reduced quality of life, and can be an economic burden on the health care system if they develop into a chronic wound [28, 63]. The published epidemiological data indicate that Australia (41–59%) has one of the highest prevalence rates of STs among aged care residents when compared to similar North America (14–22%) and Japanese (4–14%) populations [10, 49, 80, 91]. Regardless of the geographical location of these studies, STs are reported to primarily occur on the upper extremities followed by the lower extremities [43, 49, 53, 68]. The early identification of individuals 'at-risk' of STs affords the opportunity to implement targeted and timely preventative interventions that have the potential to reduce adverse outcomes, improve the quality of life of older adults, and reduce the cost of care for health providers.

In a previous study, we reported the use of logistic regression analysis to identify variables that significantly predicted the risk of STs in a multicentre Western Australian residential care population aged over 65 years [72]. Five variables were found to significantly predict the risk of dorsal forearm STs, and included: a history of STs; a history of falls; male gender; purpura; and clinical manifestations of elastosis. The analysis yielded an area under the receiver operator characteristic (ROC) curve of 0.854 (sensitivity = 81.7%; specificity = 81.4%) which, according to Meyers et al. [57], indicated that the model showed 'very good discrimination' for correctly predicting participants with and without STs.

Despite the inclusion of clinical purpura and elastosis into the skin tear predictive model, the biological processes underlying the inclusion of these skin characteristics were not readily understood. The objective of this study was, therefore, to conduct secondary analysis to better understand the inclusion of purpura and elastotic skin manifestations into the proposed skin tear prediction model.

Materials and methods

Study design and setting

The study was a multisite 6-month prospective cohort study conducted between February 2014 and June 2015 amongst aged care residents aged over 65 years and where informed written consent was provided either by themselves or their legal guardian [72]. All residents resided in two regional and two metropolitan care facilities operated by a single not-for-profit service provider in Western Australia. The study background, design, and methodology have previously been reported with ethics approval for all the stages of this study obtained from Curtin University Research and Development Human Research Ethics Committee (RD-23-13) and the Bethanie Group Inc. Governance Committee [72].

Measurements and instruments

Two non-invasive instruments were used to assess morphological and physiological skin properties at two points in time, 6 months apart. The DermaLab Combo® (Cortex Technology, Hadsund, Denmark) assessed transepidermal water loss (TEWL), hydration, skin thickness, and elasticity. The Skin-pH-meter® (Courage + Khazaka, Cologne, Germany) evaluated skin surface pH. Collaboration with the University of Tokyo Department of Gerontological Nursing and Wound Care Management facilitated the analysis of three transepidermal secreted skin proteins [collagen IV; matrix metalloproteinase-2 (MMP-2); and tumour necrosis factor-alpha (TNF-α)]. These proteins were collected using a skin blotting technique that had been devised by the researchers at the University of Tokyo and had been implicated in skin tear studies in a Japanese population [43, 58]. These authors had previously shown that the use of these non-invasive technologies provided a safe, objective, and reliable means for quantifying the morphological and physiological ageing skin properties [73].

Participants

Two hundred residents were initially recruited to the study. The demographic and clinical profile of this population has previously been reported [71]. Twenty seven (13.5%) participants were lost to follow-up (25 participants were deceased during the 6 months and two participants declined to take part further in the study). In total, 173 residents aged between 65 and 107 years were assessed at two points in time.

Data collection

Data were collected on a broad range of individual characteristics and skin characteristics that were reported in the literature to contribute to STs and age-related skin changes [20, 74]. Non-invasive testing of skin properties was collected from the extremities (bilateral mid-dorsal forearms and upper quartile of the lateral lower legs) as they were identified as anatomical sites with the highest incidents of STs [49, 68]. Except for skin blotting, where a single measurement was collected at the initial assessment, three consecutive measurements were taken 10 mm apart at the designated test sites for each skin property (for reliability control), with the combined mean used for calculation. Skin tear occurrence data over 6 months were extracted from the service provider's integrated database.

Statistical analysis

Descriptive statistics for individual characteristics and skin characteristics were calculated as the mean (M), standard deviation (SD), and median (inter-quartile range) for continuous variables and frequency (%) for categorical variables. Chi-squared tests were conducted to evaluate the frequency data for categorical variables and the occurrence of purpura and elastosis at baseline [64]. Independent samples *t* tests were used to compare continuous data between participants with purpura and elastotic skin manifestations, and those without purpura and elastotic skin manifestations.

Univariable logistic regression was conducted to identify variables significantly associated with purpura and elastosis at baseline. Variables identified to be significant in the univariable analysis were included in the multivariable logistic models. Multivariable analysis was conducted to simultaneously test the associations between numerous variables with baseline clinical purpura or elastosis, after taking into consideration possible confounders including age, gender, history of STs, history of falls, and body mass index (BMI). The magnitude of the associations between each independent variable and the outcomes was expressed as odds ratio (OR) with associated 95% confidence intervals (CIs). The OR measured the strength of association between a variable and the baseline clinical manifestation of purpura and elastosis. The dependent variable was categorised as 0 = not having the skin characteristic and 1 = having the skin characteristic.

The correlation between skin properties was assessed using Pearson's product–moment correlation coefficient (*r*) with the alpha level set a priori at 0.05. The strength of the correlation between individual skin property values was interpreted using Hopkins [34] benchmark scale: trivial (0.00–0.01), small (0.01–0.30), moderate (0.30–0.50), large (0.50–0.70), very large (0.70–0.90), and nearly perfect (0.90–1.00).

Results

Data were collected from 173 participants at two points in time 6 months apart between February 2014 and June 2015. The population comprised of 123 (71%) females and 50 (29%) males with a combined mean age of 87.6 years (SD ± 6.7). Table 1 presents the results of the Chi-square tests that were conducted to identify significant differences between baseline individual and skin characteristics, and participants with and without purpura and skin elastosis.

Purpura was present in approximately two-thirds of all participants with 61.8% of females and 64.0% of male participants exhibiting clinical signs. Individual characteristics that were significantly associated with purpura included history of STs in the previous 12 months and history of falls in the previous 3 months. Clinical factors significantly associated with purpura included the presence of cutis rhomboidalis nuchae and a lax skin appearance.

Clinical manifestations of elastosis were identified in 30.9% of female and 66.0% of male participants. Individual characteristics found to be significantly associated with elastosis including male gender and a history of smoking. Skin characteristics that were identified to be significantly associated with the cutaneous manifestations of elastosis included uneven pigmentation, a history of actinic keratosis (AK), and cutis rhomboidalis nuchae.

The following results are reported for ease of understanding according to the specific clinical skin manifestation.

Purpura

Table 2 presents the results of the two-tailed *t* test used to evaluate the difference in baseline continuous variables between participants with clinical purpura and participants without purpura.

A significant difference was identified between participants with clinical purpura and participants without purpura for: age, number of years living in Australia, number of chronic diseases, Braden Scale score, pH extremities, skin thickness extremities, distensibility extremities, collagen IV extremities, and MMP-2 forearms.

Findings from the univariable and multivariable logistic regression for each baseline variable and the risk of baseline purpura are reported in Table 3.

Univariable analysis identified eight variables that were potential predictors of purpura. Subsequent multivariable analysis identified seven variables that were significantly associated with purpura. These variables comprised four individual variables (age; history of STs in previous 12 months; history of falls in previous 3-months; antiplatelet therapy) and three skin properties (pH upper extremities, subepidermal low echogenicity band (SLEB) upper extremities and skin thickness upper extremities) that were significantly associated with cutaneous manifestations of purpura.

Two of the clinical variables identified in the multivariable analysis were found not to be statistically associated with purpura in the univariable analysis. These clinical variables included the use of antiplatelet therapy ($p = 0.091$) and SLEB upper extremities ($p = 0.920$). Antiplatelet therapy and SLEB had been included in the multivariable analysis based on the literature and their reported biological plausibility.

The three skin properties identified in the multivariable analysis were only significant at the dorsal forearms. A Pearson's product–moment correlation coefficient was conducted to better understand the relationship between all dorsal forearm baseline skin properties in participants with purpura. The results of this analysis are presented in Table 4.

Table 1 Individual and skin characteristics that were significant in relation to clinical elastosis and purpura

Variables	Purpura			Elastosis		
	No ($n=65$)	Yes ($n=108$)	p value	No ($n=102$)	Yes ($n=71$)	p value
Individual characteristics						
Sex						
Females	47 (38.2)	76 (61.8)	0.789	85 (69.1)	38 (30.9)	< 0.001**
Males	18 (36.0)	32 (64.0)		17 (34.0)	33 (66.0)	
History of smoking						
Life-long non-smoker	34 (35.1)	63 (64.9)		67 (69.8)	29 (30.2)	0.003**
Ex-smoker	27 (40.9)	39 (59.1)	0.536	31 (46.3)	36 (53.7)	
Unknown	4 (40.0)	6 (60.0)		4 (40.0)	6 (60.0)	
History skin tears in the previous 12 months						
No	52 (61.9)	32 (38.1)	< 0.001*	50 (59.5)	34 (40.5)	0.883
Yes	13 (14.6)	76 (85.4)		52 (58.4)	37 (41.6)	
History of falls in the previous 3 months						
No	48 (48.0)	52 (52.0)	0.001**	60 (60.0)	40 (40.0)	0.745
Yes	17 (23.3)	56 (76.7)		42 (57.5)	31 (42.5)	
Skin characteristics						
Uneven pigmentation						
No	27 (43.5)	35 (56.5)	0.225	53 (85.5)	9 (14.5)	< 0.001**
Yes	38 (34.2)	73 (65.8)		49 (44.1)	62 (55.9)	
History of actinic keratosis						
No	40 (43.5)	52 (56.5)	0.087	71 (77.2)	21 (22.8)	< 0.001**
Yes	25 (30.9)	56 (69.1)		31 (38.3)	50 (61.7)	
Cutis rhomboidalis nuchae						
No	52 (42.6)	70 (57.4)	0.034*	84 (68.9)	38 (31.1)	< 0.001**
Yes	13 (25.5)	38 (74.5)		18 (35.3)	33 (64.7)	
Lax appearance						
None–mild	37 (52.9)	33 (47.1)	0.001**	37 (52.9)	33 (47.1)	0.179
Moderate–severe	28 (27.2)	75 (72.8)		65 (63.1)	38 (36.9)	

n number (%)

An asterisk (*) indicates a statistically significant difference, *$p < 0.05$; **$p < 0.01$

There was a moderate negative correlation between: skin intensity score and TEWL; skin intensity score and skin thickness; and retraction and hydration in participants with purpura. A large negative correlation was found between retraction and viscoelasticity (VE). A moderately positive correlation was identified between retraction and TEWL in participants with purpura. A large positive correlation was identified between skin thickness and the SLEB.

Elastosis

The results of the *t* test analyses to evaluate the difference in mean baseline continuous variables between participants with and without clinical elastosis are reported in Table 5.

A significant difference was identified between participants with clinical elastosis and participants without clinical elastosis for the following variables: age; number of days residing in the facility; number of years living in Australia; weight; height; Braden Scale score; TEWL of the upper and lower extremities; hydration of the upper and lower extremities; distensibility of the upper extremities; skin retraction of the upper and lower extremities; and collagen IV in the skin on the dorsal forearm. Despite the mean Braden Scale score (a tool used to evaluate the risk of developing a pressure injury) having previously been identified to be associated with STs [80], the clinical difference was not substantial between participants with and without cutaneous elastosis.

The findings from the univariable and multivariable logistic regression analyses are reported in Table 6.

Univariable analysis identified eight variables that were potential predictors of clinical elastosis. Subsequent multivariable analysis identified seven of these variables to be significantly associated with clinical elastosis. These variables consisted of three individual characteristics (age, gender, history of smoking); three skin characteristics (uneven pigmentation; history of AK; cutis rhomboidalis nuchae); and a single skin property (mean collagen IV

Table 2 Baseline variables in residents with and without clinical manifestations of purpura

Variables	No purpura ($n=65$) M (SD)	Purpura ($n=108$) M (SD)	p value
Individual characteristics			
Age	85.2 (7.7)	88.74 (5.9)	0.002**
Number of days in facility	972.4 (988.0)	920.00 (898.3)	0.721
Number of years living in Australia	72.1 (22.3)	79.46 (19.9)	0.026*
Weight in kilograms	67.6 (15.7)	66.54 (16.3)	0.686
Height in centimetres	162.6 (7.9)	161.9 (9.4)	0.608
Body mass index	25.9 (4.9)	25.3 (5.6)	0.472
ADL precise score	79.0 (17.3)	80.8 (17.3)	0.522
Number of chronic diseases	3.2 (1.5)	3.9 (1.5)	0.004**
Braden Scale score	18.9 (3.7)	17.7 (3.4)	0.042*
Total number of medications	6.8 (4.1)	7.0 (3.0)	0.738
Skin properties			
Mean melanin	30.4 (4.1)	29.6 (3.8)	0.202
Mean TEWL forearms	7.3 (2.8)	8.9 (2.9)	0.061
Mean TEWL legs	5.1 (1.5)	5.4 (1.6)	0.173
Mean hydration forearms	80.1 (32.9)	77.0 (30.9)	0.543
Mean hydration legs	87.0 (33.7)	86.2 (36.3)	0.881
Mean pH forearms	5.5 (0.5)	5.8 (0.5)	<0.001**
Mean pH legs	5.6 (0.4)	5.9 (0.5)	<0.001**
Mean SLEB forearms	290.8 (71.7)	289.5 (90.5)	0.921
Mean SLEB legs	132.7 (77.6)	152.6 (66.2)	0.076
Mean skin thickness forearms	894.4 (182.0)	783.0 (167.1)	<0.001**
Mean skin thickness legs	1149.0 (270.8)	1060.7 (242.4)	0.028*
Mean skin intensity score forearms	45.0 (12.05)	44.0 (11.0)	0.590
Mean skin intensity score legs	45.5 (15.0)	46.7 (14.5)	0.621
Mean VE forearms	4.4 (1.8)	4.1 (1.4)	0.135
Mean VE legs	2.4 (1.1)	2.5 (1.2)	0.391
Mean distensibility forearms	14.3 (2.6)	13.2 (2.6)	0.005**
Mean distensibility legs	17.1 (1.5)	16.2 (2.0)	0.002**
Mean retraction forearms	4.8 (2.9)	5.6 (3.8)	0.125
Mean retraction legs	4.0 (2.8)	3.9 (2.2)	0.832
Transepidermal skin proteins			
Mean IV collagen forearms	28.8 (19.7)	21.2 (15.8)	0.009**
Mean collagen IV legs	28.3 (18.1)	20.8 (19.5)	0.013*
Mean MMP-2 forearms	26.9 (30.7)	17.1 (23.4)	0.028*
Mean MMP-2 legs	20.3 (22.7)	16.1 (22.1)	0.240
Mean TNF-α forearms	54.6 (68.0)	72.5 (80.5)	0.118
Mean TNF-α legs	53.5 (69.4)	74.9 (82.4)	0.069

M mean, *SD* standard deviation, *TEWL* transepidermal water loss, *SLEB* subepidermal low echogenicity band, *VE* viscoelasticity

An asterisk (*) indicates a significant difference, *$p<0.05$; **$p<0.01$

dorsal forearm) that were significantly associated with the cutaneous manifestations of elastosis.

As collagen IV was only significant at the dorsal forearm, a Pearson's product–moment correlation coefficient was performed to better understand the relationship between all the upper extremity baseline skin properties and participants with clinical manifestations of elastosis. The results of this analysis are presented in Table 7.

A moderately negative correlation was identified between: hydration and TEWL; retraction and hydration, retraction and skin intensity score, collagen IV and pH, MMP-2 and pH, TNF-α and collagen IV in participatants

Table 3 Results of univariable and multivariable analyses of baseline variables associated with purpura

Variables	Purpura at baseline (univariable)		Purpura at baseline (multivariable)	
	Odds ratio (95% CI)	*p* value	Odds ratio (95% CI)	*p* value
Individual characteristics				
Age	1.08 (1.03–1.13)	< 0.001	1.08 (1.00–1.17)	0.047
History skin tears previous 12 months (yes vs. no)	9.50 (4.56–19.81)	< 0.001	14.57 (5.18–40.93)	< 0.001
Falls in previous 3 months (yes vs. no)	3.04 (1.56–5.94)	< 0.001	2.89 (1.10–7.61)	0.031
Antiplatelet therapy (yes vs. no)	1.74 (0.92–3.31)	0.091	3.40 (1.26–9.17)	0.016
Skin characteristics				
Cutis rhomboidalis nuchae (yes vs. no)	2.17 (1.05–4.48)	0.036		
Mean pH forearms	4.51 (2.18–9.33)	< 0.001	4.42 (1.60–12.19)	0.004
Mean SLEB forearms	1.00 (1.00–1.00)	0.920	1.01 (1.00–1.02)	0.014
Mean skin thickness forearms	1.00 (1.00–1.00)	< 0.001	0.99 (0.99–1.00)	< 0.001
Mean elasticity forearms	0.83 (0.73–0.95)	0.006		
Mean collagen IV forearms	0.98 (0.96–0.99)	0.008		

OR odds ratio, *CI* confidence interval, *SLEB* subepidermal low echogenicity band; vs versus

with elastotic skin manifestations. A large negative correlation was found between retraction and VE. A moderately positive association was identified between skin intensity score and hydration, VE and hydration, VE and skin intensity score, distensibility and skin thickness, collagen type IV and VE, and between MMP-2 and collagen type IV with elastosis skin changes. A large positive correlation was identified between skin thickness and SLEB.

A Venn diagram (Fig. 1) was constructed to graphically illustrate the similarities and differences in individual characteristics, skin characteristics, and skin properties that were significantly associated with the clinical manifestations of elastosis and purpura. This figure illustrates the significant differences between purpura and elastosis and helped to clarify their independent inclusion in the model predicting STs in the elderly.

Discussion

The authors had previously shown that clinical presentation of skin purpura and elastosis predicted the risk of dorsal forearm STs in an older Western Australian residential care population [72]. This study reports the findings of the secondary analysis of the baseline data that was undertaken to better understand the biological inclusion of these skin characteristics in the predictive model.

Four individual variables (ageing, history of STs in the previous 12 months, history of falls in the preceding 3 months, and taking oral antiplatelets) and three skin properties (mean pH of forearms, mean SLEB forearms, and mean skin thickness of the forearms) significantly predicted the risk of purpura over the dorsal forearm (Table 3). Three individual (ageing, gender, and smoking) variables, three clinical (uneven skin pigmentation, cutis rhomboidalis nuchae, and history of AK) variables, and one skin property (mean collagen IV in the forearm) were identified to significantly predict the clinical skin elastosis (Table 6). Ageing was the only variable that was common to both clinical manifestations.

Variables associated with dorsal forearm purpura

Ageing was associated with a 1.08 higher risk of dorsal forearm purpura in this study. The clinical presentation of purpura has been reported to increase from 2% at age 70 years to 25% for centenarians [3, 84]. Participants with a history of STs within the previous 12 months were 14 times more likely to have purpura of the forearms compared to participants without a history of STs.

Participants in this study taking antiplatelet therapy were nearly three and a half times more likely to have purpura of the dorsal forearms than participants not taking this medication. Antiplatelet therapies has been reported to interfere with the clotting process with a hypercoagulation state predisposing the older individuals to purpura and ecchymosis [85].

Participants with a higher skin surface pH of the dorsal forearms were nearly four and a half times more likely to have purpura compared to participants with a lower skin pH. Increased skin surface pH correlates with impaired skin barrier function in individuals aged over 80 years and in photoaged skin [6]. Chronic ultraviolet (UV) radiation has shown to impair the skin barrier by reducing intercellular strength, cohesion and integrity of intercellular lipids, and corneodesmosomes to decrease the skin's ability to withstand mechanical forces [6].

Table 4 Pearson's coefficient for skin properties and purpura of the dorsal forearms

Purpura	Mean TEWL	Mean hydration	Mean pH	Mean SLEB	Mean thickness	Mean skin intensity score	Mean VE	Mean distensibility	Mean retraction	Mean collagen IV	Mean MMP-2
Mean hydration	− 0.288**										
Mean pH	0.112	− 0.051									
Mean SLEB	− 0.004	0.116	0.006								
Mean thickness	0.136	− 0.150	− 0.007	0.645**							
Mean skin intensity score	− 0.358**	0.165	− 0.079	− 0.246*	− 0.305**						
Mean VE	− 0.249**	0.287**	− 0.055	0.122	0.026	0.099					
Mean distensibility	0.216*	− 0.158	0.082	0.019	0.212*	0.038	0.012				
Mean retraction	0.316**	− 0.339**	0.099	− 0.091	0.005	− 0.229*	− 0.578**	0.256**			
Mean collagen IV	− 0.226*	0.260**	− 0.247**	0.089	0.031	0.155	0.195*	− 0.020	− 0.081		
Mean MMP-2	− 0.042	− 0.127	− 0.123	− 0.005	0.067	0.061	− 0.052	0.113	0.039	0.197*	
Mean TNF-α	0.263**	− 0.009	0.126	0.046	0.067	0.121	− 0.197*	0.144	0.119	-0.298**	0.013

TEWL transepidermal water loss, *SLEB* subepidermal low echogenicity band, *VE* viscoelasticity

An asterisk (*) indicates a significant difference, *$p < 0.05$; **$p < 0.01$

Table 5 Baseline variables in residents with and without clinical manifestations of elastosis

Variables	No elastosis ($n=102$) M (SD)	Elastosis ($n=71$) M (SD)	p value
General characteristics			
Precise age	86.3 (7.4)	88.9 (5.5)	0.009**
Number of days in facility	10500.9 (1048.7)	780.0 (704.9)	0.044*
Number of years living in Australia	73.1 (20.9)	81.8 (20.4)	0.007**
Weight in kilograms	64.8 (15.5)	70.0 (16.4)	0.033*
Height in centimetres	160.2 (8.7)	165.1 (8.3)	< 0.001**
Body mass index	25.2 (5.5)	26.0 (5.1)	0.322
ADL precise score	80.6 (16.9)	79.0 (17.8)	0.654
Number of chronic diseases	3.7 (1.5)	3.5 (1.6)	0.266
Braden Scale score	17.7 (3.9)	18.9 (2.8)	0.021*
Total number of medications	6.9 (3.2)	6.9 (3.7)	0.971
Skin properties			
Mean melanin	29.6 (3.6)	30.2 (4.3)	0.352
Mean TEWL forearms	7.2 (2.6)	8.9 (2.9)	< 0.001**
Mean TEWL legs	5.0 (1.4)	5.6 (1.7)	0.010**
Mean hydration forearms	84.4 (31.2)	69.3 (30.2)	0.002**
Mean hydration legs	92.2 (34.9)	78.4 (34.4)	0.011*
Mean pH forearms	5.7 (0.5)	5.8 (0.5)	0.444
Mean pH legs	5.8 (0.5)	5.8 (0.5)	0.738
Mean SLEB forearms	285.2 (79.7)	296.9 (89.3)	0.370
Mean SLEB legs	137.3 (73.8)	156.3 (66.0)	0.084
Mean skin thickness forearms	810.3 (265.1)	845.8 (179.0)	0.205
Mean skin thickness legs	1087.8 (265.1)	1102.6 (244.5)	0.709
Mean skin intensity score forearms	45.7 (11.7)	42.5 (10.6)	0.067
Mean skin intensity score legs	47.2 (15.4)	44.9 (13.5)	0.323
Mean VE forearms	4.3 (1.7)	4.1 (1.3)	0.286
Mean VE legs	2.6 (1.2)	2.3 (1.2)	0.178
Mean distensibility forearms	13.2 (2.7)	14.2 (2.3)	0.015*
Mean distensibility legs	16.6 (2.0)	16.5 (1.7)	0.752
Mean retraction forearms	4.6 (2.5)	6.4 (4.5)	0.002**
Mean retraction legs	3.6 (2.1)	4.4 (2.9)	0.047*
Transepidermal skin proteins			
Mean collagen IV forearms	21.7 (15.6)	27.4 (20.1)	0.048*
Mean collagen IV legs	21.5 (14.9)	26.6 (24.0)	0.088
Mean MMP-2 forearms	21.3 (30.6)	20.1 (20.2)	0.781
Mean MMP-2 legs	18.1 (24.0)	17.0 (20.0)	0.752
Mean TNF-α forearms	70.1 (78.8)	59.6 (72.7)	0.377
Mean TNF-α legs	72.4 (81.7)	58.8 (73.0)	0.261

M mean, *SD* standard deviation, *TEWL* transepidermal water loss, *SLEB* subepidermal low echogenicity band, *VE* viscoelasticity

An asterisk (*) indicates a significant difference, *$p<0.05$; **$p<0.01$

A thicker SLEB of the dorsal forearm was associated with a significantly higher risk of purpura compared to participants with a thinner SLEB. The SLEB is considered a biological marker for collagen degeneration of the papillary dermis and a reliable indicator of both ageing and elastotic skin changes [25]. Chronic UV radiation, however, is reported to have a stronger influence on dermal echogenicity and the formation of the SLEB across exposed skin than chronological ageing [81].

This study found that, as skin thickness declined, there was a significant increased odd (1.01) for purpura of the dorsal forearms, which is consistent with the previous research [16, 25]. Skin thickness directly relates to the proportion of dermal collagen, elastin, and glycosaminoglycans (GAGs)

Table 6 Results of univariable and multivariable analyses of baseline variables associated with elastosis at baseline

Variables	Elastosis at baseline (univariable)		Elastosis at baseline (multivariable)	
	Odds ratio (95% CI)	*p* value	Odds ratio (95% CI)	*p* value
Individual characteristics				
Age	1.06 (1.01–1.11)	0.016	1.12 (1.03–1.22)	0.006
Sex (males vs. females)	4.34 (2.16–8.74)	< 0.0005	3.65 (1.27–10.52)	0.016
History of smoking (ex-smoker vs. life-long non-smoker)	2.68 (1.40–5.13)	0.003	2.61 (1.06–6.44)	0.037
Skin characteristics				
Uneven pigmentation (yes vs. no)	7.45 (3.35–16.58)	< 0.001	8.09 (2.71–24.16)	< 0.001
History of actinic keratosis (yes vs. no)	5.45 (2.81–10.57)	< 0.001	2.95 (1.24–7.01)	0.014
Cutis rhomboidalis nuchae (yes vs. no)	4.05 (2.03–8.08)	< 0.000	3.56 (1.32–9.64)	0.012
Skin properties				
Mean elasticity forearms	1.17 (1.03–1.32)	0.016		
Mean collagen IV forearms	1.02 (1.00–1.04)	0.042	1.05 (1.01–1.08)	0.004

CI confidence interval

with chronological and photoageing processes contributing to decreased procollagen I and III syntheses [78]. The loss of structural collagen reduces the skin's mechanical supporting properties and leaves it vulnerable to trauma [60]. Decreased dermal collagen is associated with the reduced scaffold support for small blood vessels, which predisposes them to rupturing, even in the event of minor trauma [12]. While the nutritional status of study participants was not extensively examined, deficiencies in macro- and micro-nutrients and, in particular, Vitamin C, may have contributed to the decline in dermal collagen and the occurrence of purpura. Vitamin C is an important cofactor for enzymes involved in the synthesis of collagen with deficiencies leading to the fragility of blood vessels and the manifestation of purpuric and ecchymotic skin lesions [1, 18, 36, 37]. Researchers have shown that exposure to UV radiation severely depletes the cutaneous levels of vitamin C [14, 50].

Skin properties associated with dorsal forearm purpura

Five skin properties including skin surface pH, skin thickness, distensibility, collagen IV, and MMP-2 were found to significantly differ between participants with and without purpura of the dorsal forearm (Table 2).

pH

As previously noted, participants with a higher skin surface pH were significantly more likely to display purpura than participants with a lower skin pH. A Pearson's correlation identified a small negative correlation between increased skin surface pH and decreased collagen IV [$r\ (106) = -0.247$, $p < 0.01$] in participants with clinical purpura of the dorsal forearms. Skin surface pH is reported to increase with both ageing and photoageing, which suggests a degree of collagenase degradation of the extracellular matrix (ECM) [75, 86]. The degradation of the ECM and, presumably, the basement membrane may increase the risk of purpura from loss of structural proteins.

Distensibility

The mean skin distensibility differed significantly between participants with and without purpura of the dorsal forearm. A small positive correlation [$r\ (106) = 0.212$, $p < 0.05$] was found between distensibility and skin thickness. Participants with purpura of the forearm had significantly decreased skin thickness ($p < 0.01$) and significantly more rapid distension compared to participants without purpura. Increased distensibility suggests a loss of structural collagen support and a decline in the skin's mechanical properties that increase the risk of purpura and STs across the forearm surfaces. Age-related and photoage-related skin changes are reported to decrease the mechanical stability properties of skin by reducing skin elasticity [19, 77].

A small positive correlation [$r\ (106) = 0.216$, $p < 0.05$] was found between distensibility and TEWL. Increased TEWL is associated with altered skin barrier function [38], while increased distensibility and loss of elasticity suggests a decline in connective tissue elastin [87]. The increased distensibility and marked tissue displacement, which characterises ageing skin, may adversely decrease the skin's ability to resist friction and shearing forces [17, 22]. These physiological changes have the potential to lead to purpura and predisposition susceptible individuals to STs.

Table 7 Pearson's coefficient for clinical elastosis and skin properties of the dorsal forearms

Elastosis	Mean TEWL	Mean hydration	Mean pH	Mean SLEB	Mean thickness	Mean skin intensity score	Mean VE	Mean distensibility	Mean retraction	Mean collagen IV	Mean MMP-2
Mean hydration	− 0.332**										
Mean pH	0.190	− 0.252*									
Mean SLEB	− 0.109	0.164	− 0.077								
Mean thickness	0.071	− 0.157	0.029	0.529**							
Mean skin intensity score	− 0.281*	0.340**	− 0.095	− 0.166	− 0.229						
Mean VE	− 0.122	0.409**	− 0.160	0.034	− 0.105	0.342**					
Mean distensibility	0.250*	− 0.270*	0.044	− 0.063	0.311**	− 0.067	− 0.074				
Mean retraction	0.118	− 0.324**	0.230	− 0.114	− 0.043	− 0.316**	− 0.592**	0.230			
Mean collagen IV	− 0.223	0.291*	− 0.337**	0.100	0.006	0.100	0.348**	− 0.009	− 0.227		
Mean MMP-2	− 0.074	− 0.045	− 0.403**	0.033	− 0.091	− 0.047	0.040	− 0.123	− 0.177	0.450**	
Mean TNF-α	0.212	− 0.102	0.164	0.124	0.045	− 0.060	− 0.227	0.151	0.169	− 0.326**	− 0.217

TEWL transepidermal water loss, *SLEB* subepidermal low echogenicity band, *VE* viscoelasticity

An asterisk (*) indicates a significant difference, *$p < 0.05$; **$p < 0.01$

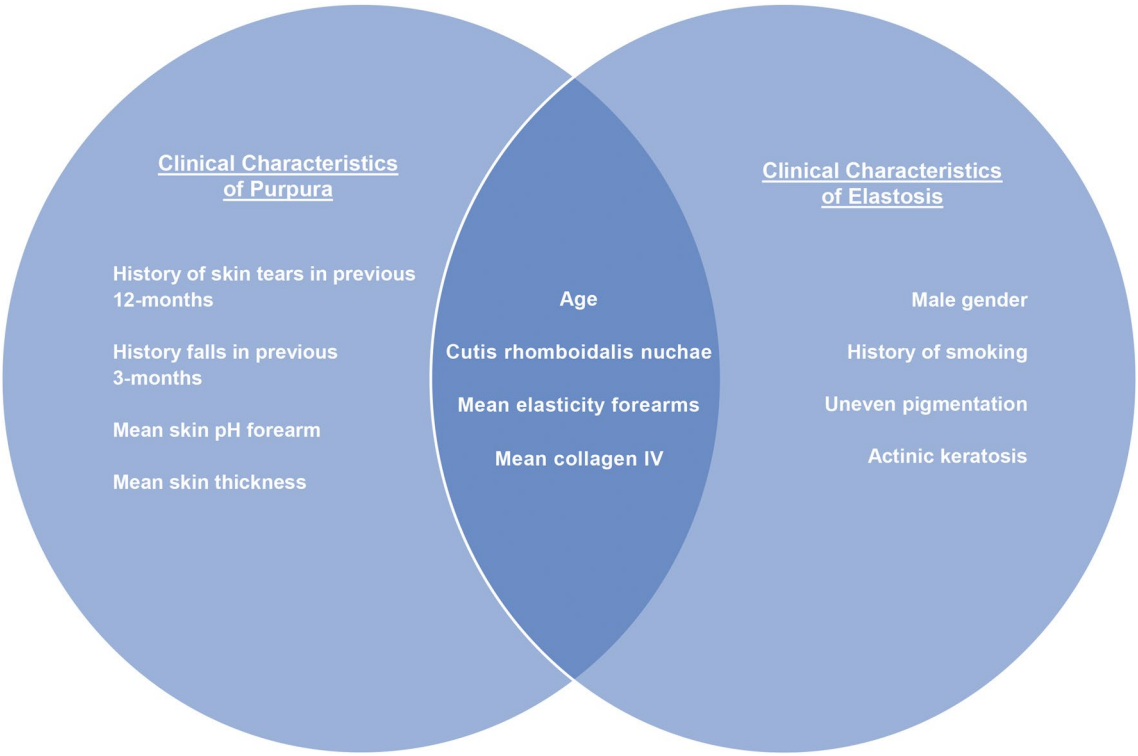

Fig. 1 Venn diagram illustrating characteristics associated with clinical purpura and elastosis

Collagen IV, MMP-2, and TNF-α

The mean collagen IV and MMP-2 levels were significantly lower in participants with purpuric lesions compared with participants without purpura. Collagen IV is a ubiquitous, network forming extracellular basement membrane protein found in a range of tissue types including skin and blood vessels [8]. The decline in collagen IV suggests the loss of stability and functional integrity of the vascular basement membrane with concomitant extravasation of blood into the dermal space. Research indicates that MMP-2 can catalyse collagen IV and degrade elastin across exposed skin surfaces [92]. In addition, MMP-2 also inhibits dermal collagen synthesis that results in a decline in skin elasticity and flexibility [69].

A negative correlation was found between collagen IV and TEWL and with pH. A positive correlation was noted between collagen IV and hydration and with VE. These findings show the overall interaction between the various skin properties and the decline in the skin barrier function and hydration status of ageing skin. In spite of a Pearson's correlation showing a correlation between TNF-α, TEWL, VE, and collagen IV, the independent samples *t* test did not identify any significant difference between participants with and without purpura for these variables.

Variables associated with elastosis of the dorsal forearms

The clinical spectrum of cutaneous manifestations of elastosis is reported to differ between skin types and the extent to which skin is subjected to UV radiation [93]. Variables identified in this study to significantly predict clinical elastosis indicate extrinsic age-related skin changes. Six of these variables were broadly categorised as either genetic (ageing, male gender), environmental (uneven skin pigmentation, cutis rhomboidalis nuchae, presence of AK), and lifestyle (history of smoking)-related factors. The seventh variable, collagen IV, was the only skin property identified to be directly associated with clinical elastosis.

In this study, age as a factor was associated with a 1.12 increased risk of participants sustaining skin elastosis and is consistent with the other studies [40, 82]. The clinical presentation of elastosis arises from degenerative changes within the ECM of the epidermis and dermis [78]. Three main classes of biomolecules form the ECM, and include the structural proteins (collagen and elastic fibres), specialised proteins (fibrillin, fibronectin and laminin), and proteoglycans, which are composed of GAGs covalently attached to core proteins [45]. Individually, these biomolecules provide cellular adhesion (fibrillin, fibronectin, and laminin), resist

tensile forces (fibrillar collagens), compressive forces (proteoglycans), and confer elastic (elastic fibres) behaviours to the skin through their mechanical properties [56, 61]. The mechanical properties of skin are dependent on its structural properties, which determine its tensile strength, integrity, and the degree to which it can expand without tearing [52, 67].

Aged-related and photoaged-related skin changes cause architectural and structural remodelling, which alter the mechanical properties of the biomolecules and the gross appearance of skin [56]. In the initial stages, photo-damaged skin is reported to thicken, while, in the later stages, the skin has a fragile appearance from atrophic changes associated with a decline in the cellular structures [76].

The ECM proteins of skin have a long lifespan and low turnover that predisposes them to modification from the cumulative and deleterious effects of time and UV radiation [19]. The half-life of skin collagen is estimated to be about 15 years, whereas elastin is about 70 years [83, 89]. The longevity of these ECM proteins makes them susceptible to the cumulative effects of ageing and UV radiation, which progressively degrade the dermal architecture of the skin to cause loss of functional structural skin proteins, altered biomechanical behaviour, and gross clinical manifestations [89].

Male participants in this study were more than three and a half times more likely to display the clinical manifestations of elastosis than female participants and are consistent with the previous findings [9, 35]. The inclusion of males in the statistical model suggests that male participants most likely had a higher level of UV radiation exposure than female participants.

Participants with a history of smoking were over two and a half times more likely to display the clinical signs of elastosis than participants who did not smoke. Smoking is an independent risk factor for elastotic skin changes [41]. In this study, 66.7% of males who had previously smoked displayed the clinical manifestations of elastosis compared to 43.2% of females. The previous research showed that smoking was strongly associated with the male gender and independent of UV exposure [15]. Exposure to UV radiation and smoking have equally been shown to accelerate elastotic skin changes through thickening and destruction of elastic fibres by up-regulating MMPs involved in the synthesis of collagen and elastic fibres, and the degradation of proteoglycans [39, 41].

Participants with cutis rhomboidalis nuchae were more than three and half times more likely to display the signs of clinical elastosis of the forearms compared to participants without this manifestation. Cutis rhomboidalis nuchae, a clinical variant of elastosis [32], was evident on the posterolateral aspect of the neck of 77.8% of males and 52.2% of females with elastotic skin changes of the dorsal forearm. Cutis rhomboidalis nuchae is specific to the nape of the neck and is a clinical sign of generalised prolonged exposure to UV radiation and is predominantly seen in mature aged males as a result of their characteristically shorter hair styles [5]. A pathological facet of cutis rhomboidalis nuchae is the deposition of anti-carboxymethyllysine antibody-positive substances in the dermis from elastotic skin changes [94]. Carboxymethyllysine (CML) is a major advanced glycation end products (AGEs), which accumulates within sun-exposed skin and contributes to the glycoxidation modification of longevity ECM proteins [26].

Advanced glycated end products arise from the non-enzymatic reaction between free amino groups in proteins and a reducing sugar, which induces a complex series of reorganisation and dehydration, and the formation of an irreversible cross-linked product [65]. Modification of longevity skin proteins from photoageing and the deleterious formation of cross-links can inhibit normal tissue function by causing the fibres to pathologically stiffen [2]. These molecular changes conceivably have the potential to cause tearing and disruption to the integrity of skin tissue.

Participants with uneven skin pigmentation were eight times more likely to show the clinical manifestations of elastosis than participants without pigmented skin changes. Uneven skin pigmentation is reported to be a clinical marker of photo-damaged skin that increases with age [78].

A documented history of actinic keratosis (AK) was associated with nearly three times increased risk of clinical elastosis compared to participants without a documented history and is consistent with the previous studies [13, 88]. Regardless of gender, participants exhibiting the clinical elastosis of the dorsal forearm were significantly ($p < 0.001$) more likely to have a history of AK than participants without a history of AK.

Participants with higher collagen IV levels had significant higher odds (1.05) of having clinical elastosis compared to participants with a lower collagen IV level. As previously noted, collagen IV is a ubiquitous basement membrane protein and is the principle component of the dermal–epidermal junction, and provides mechanical stability and structural support to the skin [8].

Higher levels of skin collagen IV are associated with increased thickening of the basement membrane [66]. Excess collagen IV production and the concomitant thickening of the basement membrane are influenced by both ageing and chronic UV exposure, through glycation of the longevity dermal ECM proteins (collagen and elastic fibres) and the accumulation of cytotoxic AGEs [24, 66]. The accumulation of cytotoxic AGEs alters the skin biomechanical properties through cross-linking of adjacent collagen fibres, increased tissue stiffness, reduced skin elasticity, loss of skin plasticity, reduced susceptibility of MMPs to regulate protein renewal, and impaired lateral

extensibility that decrease the resistance of collagen to mechanical forces [30, 66]. The findings from this study suggest an indirect association between increased collagen IV and STs. This result differs to the initial work by Koyano et al. [43] who identified that decreased collagen IV was directly associated with STs in a small sample of ageing Japanese. A subsequent prospective study by Koyano et al. [44] however, failed to identify any association between collagen IV and STs.

The difference in collagen IV levels and the risk of STs between the two studies suggests that the samples differed in terms of the influence associated with the Fitzpatrick skin type, chronological ageing, skin photoageing, environmental exposure, and lifestyle behaviours had on the structural integrity and mechanical properties of Asian and Caucasian skin.

It is, therefore, more likely that the risk of STs and acquiring clinical purpura and/or elastosis relates to the skin type and degree and extent to which skin is subjected to the above influences. The influence of these factors may help to explain the different skin tear incident rates reported between Australian (40.6–59.4%) and Japanese (3.8–14.1%) aged populations [10, 43, 44, 71, 80]. Research by Lagarrigue et al. [46] found the quantity of dermal papillae to be significantly lower with ageing and in participants with Fitzpatrick skin types I–II compared to darker pigmented skin. The decline in dermal papillae in lighter skin types may reduce the interdigitation of the dermis into the epidermis to leave the layers of skin at greater risk of separating or tearing.

Skin properties associated with elastosis of the dorsal forearms

Five skin properties including collagen IV, TEWL, hydration, distensibility, and retraction significantly differed between participants with and without clinical elastosis (Table 5). Results of the Pearson's correlation showing the relationship between all skin properties in participants with the clinical manifestations of elastosis of the dorsal forearms (Table 7) are discussed in greater detail in the following section.

Collagen IV

Participants with cutaneous manifestations of elastosis had significantly higher levels of collagen IV. Higher levels of collagen IV negatively correlated with a lower skin pH. While the precise reason for this association is unknown, the decline in skin surface pH may reflect changes to the structural components of the skin in participants with elastotic skin manifestations.

TEWL

The mean TEWL across all extremities was significantly higher in participants with clinical manifestations of elastosis compared with participants without elastosis. The TEWL is the insensible water loss from the skin surface, and is considered a surrogate means for evaluating the effectiveness of the skin barrier and the stratum corneum integrity [79]. Conversely, the measurement of hydration assesses the structural and behavioural integrity of skin as water is bound to the proteoglycans, which influence the skin's VE properties [70].

The higher TEWL and lower hydration measurements obtained across the dorsal forearm of participants with clinical skin elastosis suggest a degree of disruption to the skin barrier. This result is similar to findings of the previous studies which show photoageing alters the permeability function of the skin barrier across exposed skin surfaces [31, 33]. A small but positive correlation [$r\ (171) = 0.209$, $p = 0.006$] was found between age and TEWL of the dorsal forearms. As age only accounted for 4.4% of the variation in the TEWL result, it is conceivable that the higher TEWL level may relate to changes to the skin barrier from photoageing.

A Pearson's correlation also recorded a moderate inverse relationship between TEWL and hydration [$r\ (69) = -0.332$, $p = 0.05$] in participants with elastotic skin manifestation of the dorsal forearms (Table 7). Recent research entailing repeated exposure of human skin samples to UV radiation reported similar findings with TEWL increasing as the skin moisture content declined [86]. These physiological properties suggest that the integrity of the skin barrier was diminished in participants with clinical elastosis compared to participants without elastosis.

Hydration

The mean hydration of the dorsal forearm was significantly lower ($p = 0.002$) in participants with clinical elastosis (M 69.23, SD 30.18) compared to participants without elastosis (M 84.35, SD 31.23) (Table 5). Corneocyte hydration contributes to an effective skin barrier function by conferring suppleness, elasticity, flexibility, plasticity, and softness that bestows skin with the tensile strength to withstand deformation and tearing under mechanical stress [21].

The lower hydration level findings for the dorsal forearm in participants with clinical manifestations of elastosis are consistent with the previous studies that examined photoaged skin surfaces [7, 55]. A small negative significant correlation was found in the present study between hydration and skin surface pH [$r\ (71) = -0.252$, $p = 0.05$] across the dorsal forearm. Increased skin surface pH is associated with reduced hydration as lipid processing is controlled by the acidity level of the ECM [54]. Lipid hydrolases diminishes

as skin surface pH increases to compromise the homeostatic function of the epidermal barrier [54]. Increased skin surface pH activates proteases that degrade corneodesmosomes to further disrupt the integrity of the stratum corneum [29].

The dermal contentment of GAGs diminishes with age to alter both hydration and the mechanical properties of skin [47]. While the level of GAGs is reported to increase in photoaged skin compared to chronologically aged skin they are disproportionally amassed on the elastotic material rather than generally distributed [4]. The accumulated GAGs bind weakly with water molecules and contributes to the increased amounts of unbound water in the dermis and the typically dry and furrowed appearance that is characteristic of photoaged skin [90].

Distensibility and retraction

The DermaLab Combo® elastic suction probe measured three variables: distensibility, retraction and VE. The mean skin distensibility and retraction time of the dorsal forearms significantly differed between participants with cutaneous manifestations of elastosis compared with participants without elastosis. In participants with clinical elastotic skin, the mean distensibility values were increased, indicating that skin was stiffer compared to participants without elastotic skin manifestations. Likewise, the mean retraction was delayed (as indicated by increased values) in participants with clinical signs of elastosis compared to participants without cutaneous manifestations of elastosis. The reduced distensibility and delayed retraction (recoil) time for participants with cutaneous manifestations of elastosis compared with participants without elastosis suggests some loss of functionality. This result is similar to the findings of the previous studies that showed delayed skin distensibility and retraction is related to photoaged-related skin changes [27, 77]. Research findings indicate that AGEs contribute to the gradual decline in skin elasticity and resultant loss of mechanical properties in individuals as they aged [11].

The measurement of VE in the present study was not directly associated with clinical elastosis of the dorsal forearms. A moderate positive correlation [$r\ (71) = 0.348$, $p = 0.01$] was, however, identified between mean VE and collagen type IV in participants with clinical elastosis of the dorsal forearms (Table 7). This finding suggested that, in participants with clinical elastosis, increased collagen IV inhibited the VE behaviour of skin. Photoageing and associated elastotic skin changes have histologically been likened to fibrotic and more rigid tissue [59].

SLEB, skin thickness, and skin intensity score

The SLEB is considered an objective biological marker for quantifying skin photoageing as its thickness and density increase proportionally with age and exposure to UV radiation, in particularly across the dorsal forearms [16, 25]. The SLEB is reported to correspond to the formation of non-functional elastotic material and arises when elastin and GAGs accumulate in the papillary dermis [25, 42].

While descriptive analysis showed the mean SLEB (M 296.9, SD 89.3) was higher in participants with the clinical signs of elastosis compared to participants without visible manifestations (M 285.2, SD 79.7) of elastosis, the independent t test did not identify any significant difference ($p = 0.370$) (Table 5). Moreover, there was no significant correlation between age and the SLEB [$r\ (69) = 0.153$, $p = -0.171$] in participants with elastosis of the forearm. No significant difference was found between skin thickness (M 845.8, SD 179.0) of the dorsal forearms in participants with the clinical signs of elastosis (M 810.3, SD 181.1) compared to participants without elastosis (Table 5). A Pearson's correlation [$r\ (69) = 0.529$, $p \leq 0.01$] showed a moderate positive correlation between the mean skin thickness of the dorsal forearm and the mean SLEB in participants with elastotic skin manifestation (Table 7). The positive correlation between skin thickness and the SLEB across the dorsal forearm is consistent with the previous findings showing the width of the SLEB reflected the magnitude and severity of skin photoageing [23, 25]. This result suggests that photo-related skin changes may have had a greater influence on the formation of the SLEB and concomitant increase in skin thickness in this study population than the other age-related skin changes.

Conclusion

Changes to the structural and mechanical properties of skin from chronological ageing, photoageing, variation in Fitzpatrick skin types, environmental exposure, and lifestyle-related behaviours are the most likely explanation for clinical purpura and elastosis of the dorsal forearm and connotation increased risk of STs in older adults. Variations in the clinical manifestations reported in the literature and the ability to predict the risk of STs may relate to different ethnicity and geographical locations and the degree and extent to which the study samples were influenced by these factors. The findings from this study suggest that both these clinical skin manifestations increase the risk of STs. External validation of these clinical skin manifestations will confirm their role in providing a simple but promising means for health care providers to predict the risk of STs in older individuals.

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Compliance with ethical standards

Conflict of interest R. Rayner was a recipient of a 2013 Australian Postgraduate Award, Curtin University Postgraduate Scholarship, and a Wound Management Cooperative Research Centre (CRC) PhD stipend. The School of Nursing, Midwifery and Paramedicine, Curtin University, and the Silver Chain Group, Western Australia are participants in the Wound Management Innovation CRC. No conflict of interest exists among the authors.

Ethical approval All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards.

Informed consent Informed consent was obtained from all individual participants included in the study.

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