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MICROALBUMINURIA DURING KELOID SCARS, INCIDENTAL OR RELEVANT ASSOCIATION?

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ABSTRACT

Keloid scars are the result of an alteration in the normal healing process. Numerous studies have demonstrated the existence of cardiovascular risk factors in subjects carrying keloids.We conducted a cross-sectional study from January 2012 to December 2016 at the Brazzaville Hospital and University Center (H.U.C).Data from 34 patients with keloid scars were analyzed. The sex ratio was 1:1.8 with an average age of 37.52± 15.55 years. Pathological microalbuminuria was found in 18 diabetic and/or hypertensive patients and in 9 non-diabetic and non-hypertensive patients (p=0.6). The existence of pathological microalbuminuria in patients with keloids appears to be related to associated vascular pathologies and/or to the chronic and vascular inflammatory mechanisms involved in keloid pathogenesis.

Keywords : Micro albuminuria, Keloids, Hypertension, Diabetes

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INTRODUCTION

Keloids are benign connective tissue tumours that form as a result of a loss of substance or an inflammatory process in the skin: it is an abnormal healing process [1]. The evoked lesion mechanism is an alteration of the normal healing process with excessive production of collagen, fibronectin and chondroitin sulfate [2, 3]. Studies of keloids have led to significant advances in understanding the racial differences in the severity and morbidity of hypertension (hypertension) [4,5]. The same growth factors involved in keloid pathogenesis are involved in nephrosclerosis and atherogenesis [6]. A higher prevalence of high blood pressure and renal impairment has been associated in black subjects with cheloid disease [7, 8]. Microalbuminuria is considered a cardiovascular risk factor, an early marker of renal impairment and cardiovascular morbidity. The interest of microalbuminuria is clearly established in the monitoring of metabolic and cardiovascular diseases, but has never been evoked during keloid scarring. The authors report observations of pathological microalbuminuria found in patients with keloid scars, and discuss the incidental or relevant nature of this association.

MATERIALS AND METHOD

Patients and methods

This was a cross-sectional and analytical study carried out from January 2012 to December 2016. We followed patients with keloid scars in outpatient dermatology and nephrology consultations at the C.H.U. of Brazzaville.

The interview sought personal and family history, diabetes, hypertension.

The parameters studied were blood pressure measurement after 15 minutes of rest, blood pressure was considered pathological if greater than or equal to 140/90mm/hg on 3 occasions, fasting blood glucose, hemoglobin A1C, 24-hour microalbuminuria, 2 times at one month interval, 24-hour microalbuminuria was considered pathological if greater than 30mg/24h, uroculture, blood count, C-reactive protein.

Inclusion criteria were: patients never treated with corticosteroids, absence of infections and inflammatory syndrome. Not all patients with cheloid scars see a dermatologist. We included patients in a random and reasoned manner.

Criteria for non-inclusion, patients already treated with corticosteroids, patients with urinary tract infection, biological infectious or inflammatory syndrome, false positives: antiseptic: Chlorhexidine in the collection vessel.

The determinants studied were: Socio-demographic aspects, clinical aspects, and paraclinical aspects.

To interpret the results, we identified 3 possible age groups with statistical significance: 0 to 20 years, 21 to 40 years, 41 to 60 years. We measured microalbuminuria in all patients with keloids. Patients with keloids, who were neither diabetic nor hypertensive, and who were under 40 years of age, formed our control group.

Data analysis was performed using SPSS 23 software. The values of the quantitative parameters were expressed as means, standard deviations, ranges or numbers and percentages. The Chi-square test was used to compare categorical variables and a $P<0.05$ value was considered significant.

RESULTS AND DISCUSSION

58 patients with keloid scars were examined during the study period. 34 patients met our inclusion criteria, including 22 women and 12 men. The sex ratio 1:1.8. The mean age was 37.52± 15.55 years with extremes of 12-56 years.

The age ranges were as follows:

5 patients (14.71%) were in the 0 to 20 year age group, 13 patients (38.23%) in the 21 to 40 year age group, 16 patients (47.05%) in the 41 to 60 year age group. **Table I**

The mean age of patients with positive microalbuminuria was 35.5 years and the mean age of patients with negative microalbuminuria was 38.14 years (p=0.92).

The factors that triggered keloid were: folliculitis (n=10), spontaneous scarring (n=8), piercing (n=5) burns (n=4), surgery (n=3), prurigo (n=2), shingles (n=1) and electrocoagulation (n=1).

Cardiovascular risk factors were: hypertension (n=15), diabetes (n=6).

13 patients were neither diabetic nor hypertensive.

Positive microalbuminuria was found in 18 patients (diabetic and/or hypertensive) and 9 non-diabetic and non-hypertensive patients (p=0.6) as shown in ***Table II.a.***

Microalbuminuria was found in all patients between 0 and 40 years and in patients between 0 and 40 years (control group) without HTA and diabete (p>0,5). Table II.b.

Variations in microalbuminuria with respect to age, sex and personal history were not significant R^2 =0.288.

Discussions

Our sample size is small and is one of the limitations of the study. The small number of keloid-carrying patients studied gives the study its preliminary character. There are no studies reported in the literature evaluating microalbuminuria in patients with keloid scars.

The term keloid was used in 1806 by ALIBERT to describe a crab-shaped tumour [9]. Keloid scars are the result of an alteration in the normal healing process.

They appear in two circumstances: keloid scars after vaccine induced injuries and inflammatory skin lesions [10] or spontaneous [10], especially in the thoracic region. Family forms have also been described in an autosomal dominant inheritance pattern with incomplete penetrance and variable expression [11]. Cheloidal folliculitis of the neck, pseudo-folliculitis of the beard, especially described in the black race [12], is the most frequent trigger factor in our study. Microalbuminuria is a generalized endothelial dysfunction [13], frequently found in vascular andmetabolic diseases, and represents a major risk of renal failure and cardiovascular morbidity [14,15]. The personal and family history of hypertension and diabetes are constantly being found in microalbuminuric patients with keloids and are probably underestimated in the Congolese environment [16]. Pathological microalbuminuria is classic in the elderly and in diabetic and hypertensive subjects.

We found no statistically significant difference (p=0.92) between the mean age of patients with positive and negative microalbuminuria.

The direct relationship between keloids and microalbumunaria can be evoked because our study population is young and included a proportion of normotensive and non-diabetic patients.

The existence of microalbuminuria in these patients may be the result of vascular changes related to keloids or may be related to familial predispositions since microalbuminuria has been reported in healthy normotensive adolescents born to hypertensive parents [17]. The role of hypertension as an aggravating factor for keloids is well known [18,19]. Other genetic factors are also mentioned to explain the development of keloids [20]. The lesion mechanisms of microalbuminuria and keloid scars appear to be identical. The endothelial dysfunction mechanism implicated in microalbuminuria also appears to play a role in keloid pathogenesis [21]. The link between keloid scars and microalbuminuria may also be related to inflammation. Microalbuminuria is found in inflammatory syndromes [22]. Pro-inflammatory factors such as interleukin (IL) 1 and IL-6 are positively regulated during keloids [23] suggesting that keloids are inflammatory skin disorders [24]. Microalbuminuria can be attributed to keloid scars by chronic vascular and inflammatory phenomena.

Table 1: Characteristics of microalbuminuria by age

age / **microalbuminuria**	**0 - 20 years**	**21 - 40 yearsold**	**41 - 60 yearsold**	**P**
positivemicroalbuminuria	n=5	n=10	n=12	NS
negativemicroalbuminuria	n=1	n=3	n=3	NS

Table 2.a : Patient Characteristics by Microalbuminuria

	Diabetic and or hypertension patients	**Patients without diabetes without hypertension**	**Total patients**	**p**
patient load	21	13	34	
positive microalbuminuria	18	9	27	0,60
negativemicroalbuminuria	3	4	7	0,02

Table 2.b : Patient Characteristics by Microalbuminuria

age / **microalbuminuria**	**All Patients 0 - 40 years**	**Patients 0 - 40 years without hypertension without diabetes**	**P**
Number of patients	n=22	n=10	P>0,5
positivemicroalbuminuria	n=18	n=8	P>0,5
negativemicroalbuminuria	n=4	n=2	

CONCLUSION:

Keloid scars are a pathology of young black adults. Vascular and renal risk factors are consistently associated. The existence of microalbuminuria in patients with keloid scars appears to be

polyfactorial; secondary to associated pathologies, familial predispositions and related to the vascular and chronic inflammatory mechanisms involved in keloid pathogenesis. Larger studies are needed to significantly consider keloids as an independent vascular risk factor.

It seems logical to systematically look for microalbuminuria in patients with keloid scars even in the absence of diabetes and hypertension: to optimise the prevention of vascular risks and overall management.

Conflicts of Interest :

None

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