BLOOD CORPUSCULAR PATTERN OF KELOID PATIENTS IN NORTH CENTRAL NIGERIA

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Abstract

Background: Keloid has been reported to be preponderant in Africans, non-existent before one year of age and its cause still unknown.

Aim: In search for the possible cause of this condition, differential white blood cell count in keloid patients in north centml Nigeria is studied.

Materials and Method: Eighty (80) Keloid subjects were matched with control subjects by age and sex. 5ml of venous blood was taken from each of these subjects. Using thin blood films stained with Leishman's stain, total white blood cell count and differential white cell count for each subject was done and the findings analyzed by parametric statistical methods. Student *t* test was used to determine whether there was any significant difference between leukocytes of keloid and control subjects.

Results: When the mean percentage leucocytes in Keloid and control subjects was compared, women with Keloid had significantly higher mean percentage monocytes than those in the control group ($p < 0.001$). Males with Keloid also had a higher mean percentage monocytes ($P<O.05$) than their counterparts in the control group.

Conclusion: Monocytes count was found to be significantly higher in keloid subjects compared to the control subjects. It is suggested that dietary factor is likely to be responsible for keloid formation.

Key words: Keloid, monocytes count, African diet

Introduction

Keloid is an abnormality of wound healing characterized by the deposition of an unduly large amount of collagen[1]. As of today, no specific gene or set of genes has been identified as allowing keloids to develop[2,3,4]. Although this clinical condition is found all over the world, it has been reported[5] to be preponderant in Africans and nonexistent before one year of age. In view of the fact that nutrition may play a role in several disease disorders, the purpose of this study was to find out if there is any association between keloid and white blood cells since this disease condition is nonexistent before one year of age and more common in Africans who are known to have some dietary related conditions like non-genetic neutropenia, which does not exist at birth. Finding out this will hopefully mean better preventive medicine and more effective treatment in the future.

Materials and methods

Patients with clinically diagnosed keloid attending surgical out patient clinic of the Jos University Teaching Hospital, a referral center for the states in North Central Nigeria, were studied. The ethical committee of the hospital approved the study and informed consent was obtained from all the subjects included in the study. Eighty (80) keloid subjects were matched with control subjects by age and sex. 5ml of venous blood was taken from the median cubital vein of both keloid and control subjects and placed in specimen bottles containing EDTA to prevent coagulation and then taken to the laboratory for total and differential count. Using

heparinised capillary tube and Bickerton Eaves microhaematocrit, the packed cell volume (PCV) of all the subjects were determined and recorded Using thin blood films stained with Leishman's stain, total white blood cell count and differential white cell count for each subject was done and the findings recorded. The findings were then analyzed by parametric statistical methods using excel and Number cruncher statistical system (NCSS) and the results compared were expressed as mean ± standard deviation. Student *t* test was used to determine whether there was any significant difference between leukocytes of keloid and control subjects.

Results

Table 1. Shows the age and sex distribution of the study population. The subjects comprised of 140 males and 20 females with male female ratio of 7:2. From this table, it can be seen that the females with keloid were in the younger age group. None happened to be in the age group above 26 years. Table 2. Shows the mean percentage leucocytes in keloid and control female subjects while Table 3. Shows the mean percentage leucocytes in keloid and control male subjects. In the females, those with keloid had significantly higher mean monocytes compared to their counterparts in the control group ($p < 0.001$). Amongst the males too, the mean monocyte percentage was significantly higher in those with keloid compared with the control group ($p<0.05$).

Figures 1 - 4 are histograms showing the mean percentage of leucocytes in the study population. It can be seen that women had higher lymphocytes, monocytes and eosinophils than the men. When the mean percentage leucocytes in keloid and control subjects was compared, women with keloid had significantly higher mean percentage monocytes than those in the control group ($p <0.001$). Males with keloid also bad a higher mean percentage monocytes ($P<0.05$) than their counterparts in the control group (Table 4).

Table 1. Age and sex distribution of the study population

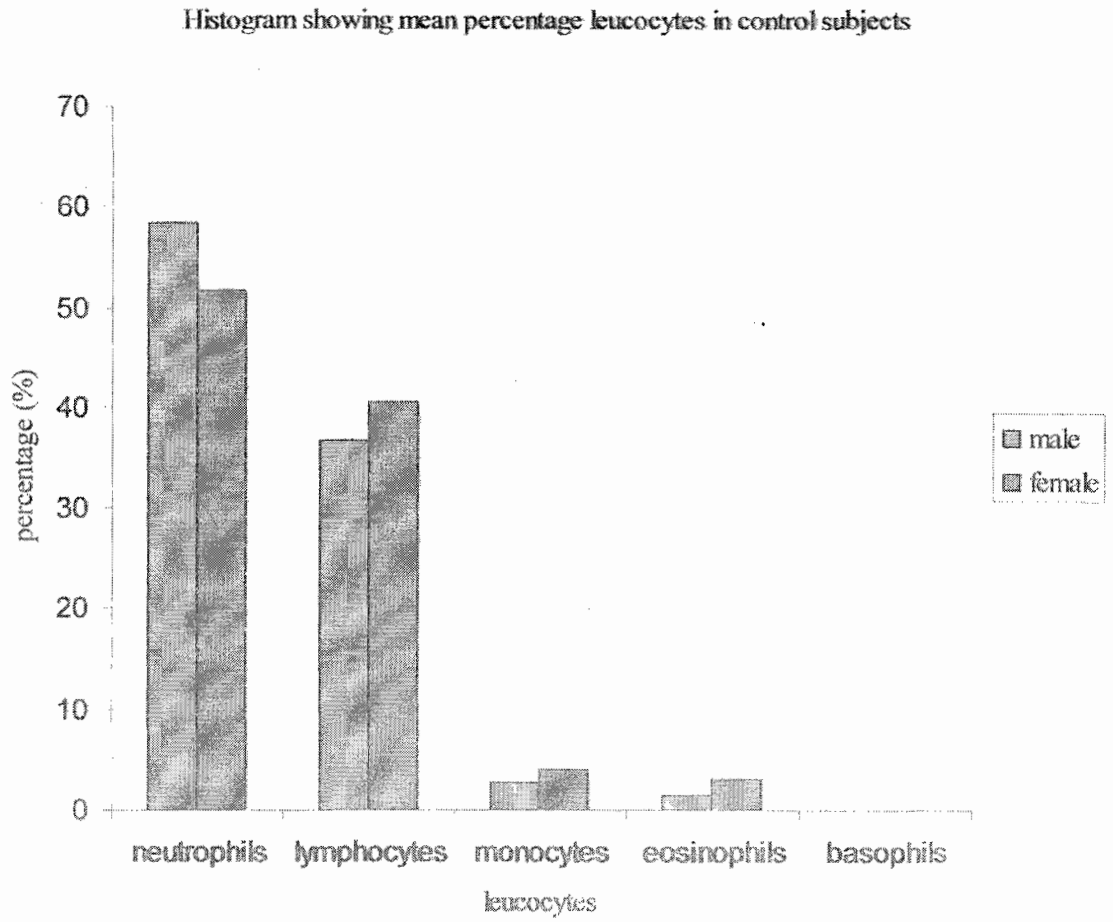
	Keloid Subject		**Control Subjects**	
Age (years)	Male	Female	Male	Female
16 - 20	-	6	-	6
21 - 25	11	4	11	4
26 - 30	24	-	24	
31 - 35	11	-	11	
36 - 40	-	-	-	-
41 - 45	8	-	8	
46 - 50	8	-	8	
51 - 55	-	-	-	-
56 - 60	-	-	-	-
61 +	8	-	8	-
Total	**70**	**10**	**70**	**10**

Table 2. Comparison of mean leukocyte count in keloid and control female subjects

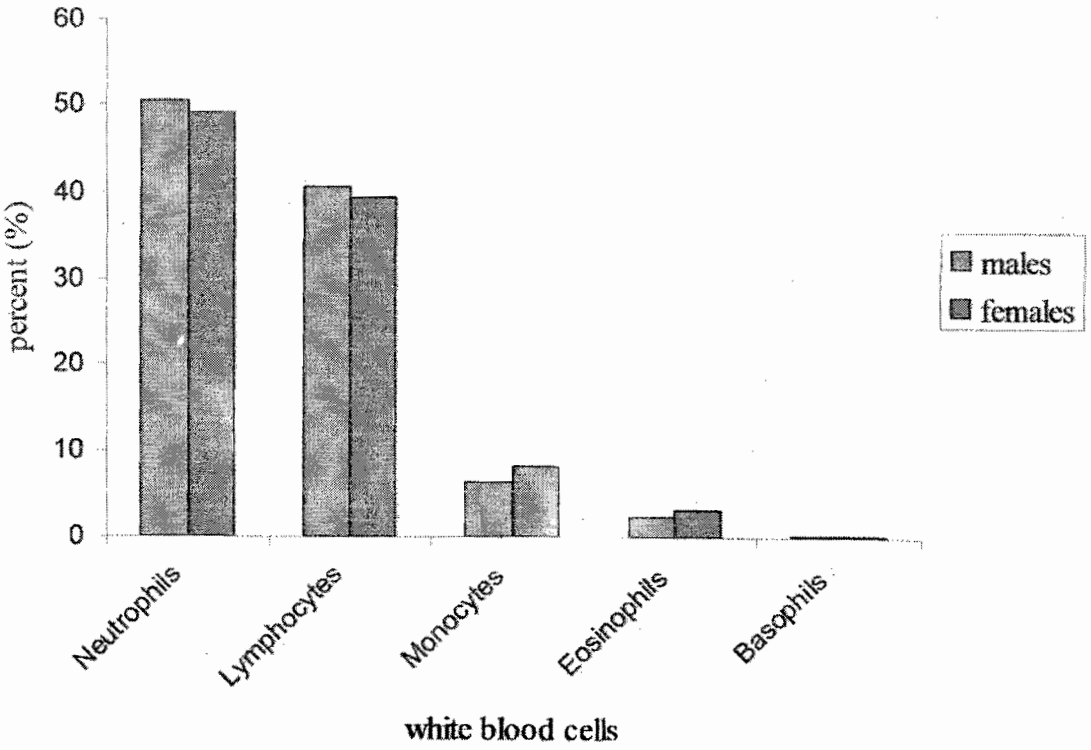
Leukocytes	Keloid	Control	*t-test*	p
Total WBC count/mm^3 (mean ± SD)	5520 ± 1808.87	5320 ± 1243.79	02037	NS
Percentage Nuetrophils (mean ± SO)	49 ± 734	58.4 ± 7.83	1.9576	NS
Percentage Lymphocytes (mean ± SD)	39.4 ± 9.4	36.8 ± 9.42	0.4363	NS
Percentage Monocytes (mean ± SD)	82 ± 1.1	2.8 ± 1.92	5.4564	0.001
Percentage Eosinophils (mean ± SD)	32 ± 4.43	1.4 ± 2.19	0.8132	NS
Percentage Basophils (mean ± SD)	02 ± 0.45	0 ± 0	0.2425	NS

Table 3. Comparison of mean leukocytes count in keloid and control male subjects

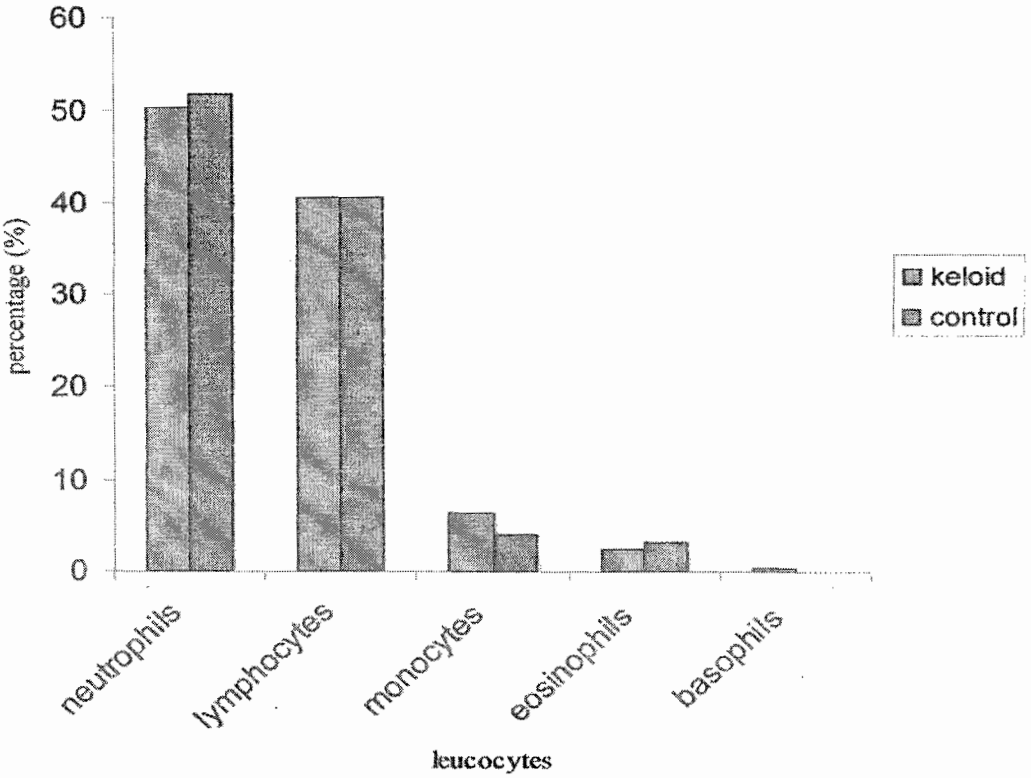
Leukocytes	Keloid	Control	*t-test*	p
Total WBC count/mm3 (mean ± SD)	6240 ± 2343.41	6140 ± 1922.50	0.0499	NS
Percentage Nuetrophils (mean ± SD)	50.5 ± 1.44	51.8 ± 1.04	0.4014	NS
Percentage Lymphocytes (mean ± SD)	40.5 ± 128	40.5 ± 1.53	0.1855	NS
Percentage Monocytes (mean ± SD)	6.4 ± 2.55	4 ± 2.83	23865	0.05
Percentage Eosinophils (mean ± SD)	23 ± 2.06	3.1 ± 238	0.1015	NS
Percentage Basophils (mean ± SD)	03 ± 0.48	0 ± 0	1.9639	NS



Histogram comparing differential count of wbc in keliod patients



Histogram showing mean percentage leucocytes in male subjects with keloid and in control group



Discussion

This study found out that patients with Keloid have high monocytes count. This finding might explain what Leibovich and Ross[6] found while studying wound repair. In their study, they reported that there was a macrophage-dependent factor that stimulates the proliferation of fibroblasts. This macrophage-dependent, fibroblast stimulating factor activity was described to be nondialyzable, heat stable (560°C for 30 minutes) and requires culture in vitro for demonstration of activity. Oladimeji et al.[7] in their elegant study on the effect of surgery on monocyte function found that peripheral monocyte counts were stable in their control group whilst in their surgical patients there were significantly higher readings at 36-48 hours suggesting that surgery has quantitative (monocyte count) effect on the mononuclear phagocytes. The presence of significantly higher monocyte count in keloid patient in the present_study supports the findings of Oladimeji et al. Since keloid does not occur before one year of age and for the fact that it is more preponderant in Africans[5] and its association with increased monocyte counts suggests that diet is more likely to be its cause than genetic factors. Why it does not occur before one year of age might because the infant at that age is still being breastfed during that period in which case receiving adequate amounts of lipids from the breast milk. The lipids present in the breast milk might have adequate cholesterol that might mobilize tissue monocytes back into the blood stream as suggested by Ogunranti[8]. But as the nutritional requirements of the baby increase with age, lipids gotten from food might not be adequate enough to keep the raised monocytes produced in response to trauma within the intravascular compartment hence they might go back to the tissue to stimulate fibroblast eventually leading to keloid formation. It is possible that as a result of low lipids in circulation in Africans due to poor dietary intake, the monocytes produced secondary to trauma escape into the tissues especially at the site of injury to stimulate fibroblast which then produces excessive collagen. Looking at lipid model as proposed by Louw[9,10,11] in the aetiology of keloid from the angle of sex, it can be seen that in the study population, males had lower monocyte count than females probably because females have higher sex steroids in circulation than males which mobilizes the monocytes from tissue into the intravascular compartment hence monocyte counts in females is higher than those in males. By this same token, females probably tend to have keloids of minimal sizes compared to males because the higher steroids in circulation protect their fibroblast partially from stimulation by the monocytes which are kept within the intravascular compartment This might also be the reason why steroids such as Triamcinolone are used in the treatment of keloid.[12,13] In conclusion, keloid patients in North Central Nigeria have significantly higher monocyte count compared to their counterparts in the control group.

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