POSSIBLE CORRELATION OF EXTENT OF SKIN INVOLVEMENT WITH SERUM URIC ACID LEVELS IN PATIENTS WITH PSORIASIS AT KENYATTA NATIONAL HOSPITAL - NAIROBI.

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This dissertation is my original work and has not been presented for a degree work in any other University.

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This dissertation has been submitted for examination with our approval as University supervisors.

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SUMMARY

94 patients with psoriasis were studied. It was found that psoriasis was rare before the third decade of life. Only 11.7% of patients were aged up to 20 years. No patient was found below the age of ten years. There was a higher prevalence in males (males 62.8%, females 37.2%). The tribal distribution was as expected in keeing with the geographical location of Kenyatta National Hospital. Thus the Kikuyu were the majority with 75.7 percentage. This, however, does not tell us much about the nation-wide prevalence of psoriasis amongst the various other tribes of Kenya. Just a little more than half the patients had psoriasis involving more than 10% of the skin surface, i.e. 57.4%. 29.8% had high serum uric acid. There was a strong positive correlation between serum uric acid levels and the extent of skin involvement, $r = 0.82$ and $P \leq 0.001$.

OBJECTIVES

1. To note the age, sex and tribal distribution in psoriatic patients at the Kenyatta National Hospital Nairobi.
2. To determine the extent of skin involvement in these patients.
3. To determine whether there is any correlation between the level of serum uric acid and extent of skin lesions in psoriatic patients.

INTRODUCTION

Psoriasis is a papulosquamous disease of the skin characterised by well defined pink or dull red lesions surmounted by silvery scaling. The lesions tend to become confluent and may persist indefinitely.

The cause of psoriasis is not known although there are some strongly associated factors. Psoriasis has been regarded as the expression of a disturbed environment and the resulting adaptation to this with the patient(1). There is no doubt that genetic factors may play an important role in the pathogenesis of psoriasis (2). Psoriasis has been shown to be familial (3). Mordovtsev et al have shown an incidence of 6.3% among first degree relatives, as opposed to 0.75% in the general population (3). Seventy two per cent of monozygotic twins have psoriasis as compared with only twenty two per cent in dizygotic twins (4). Brandup et al. have stated that there is a 64% incidence of psoriasis in monozygotic twins and a 14% incidence of psoriasis in dizygotic twins (5).

Hoede has shown that the frequency of the disease among siblings was 4.5% in families with non psoriatic parents and 11% when one of the parents was affected (537 cases). Based on this data he suggested that the distribution of psoriasis in these families was an autosomal dominance inheritance.

Balyavichene et al (6) examined 582 patients and revealed that in 225 cases (47%) there was a familial pattern of psoriasis.

They found that 13% of the parents, 10% of siblings and 8.9% of children of the probands were affected. Initially it was thought that the mode of inheritance of psoriasis appeared to be consistent with a simple autosomal dominant pattern with low penetrance. However, after further analysis of the above data it was found that the distribution of psoriasis among relatives of probands could not be explained on a single dominant gene suggesting that psoriasis could be multifactorial in origin.

Various immunologic, biochemical and morphologic studies have yielded some positive results to the genetic markers of psoriasis (7). It has been established, for example, that there is a strong association between the presence of HLA-B_{13} and HLA-BW_{17} and occurrence of psoriasis (3). Liden et al. have shown a greater risk of having psoriasis in people with genotype Le (a^-b^-) in Lewis system of blood groups and SS of MNSs systems of blood groups (8). Those having an A-factor of the ABO system also had a low frequency of genotypes Ss, Hp 2-1, complement 3, HLA-B_7 and HLA-B_8. Currently, it would appear that the most likely mode of inheritance in psoriasis is autosomal dominance with incomplete penetrance and variable manifestations to which postnatal and environmental factors contribute (1). Factors like sunlight, vaccinations, asthma, dysentery, endocrine diseases and streptococcal infections have been known to preceed the onset of psoriasis. Streptococcal infections may trigger psoriasis particularly in young patients in whom HLA

has its strongest influence (9). As some HLA antigens have been reported to cross-react with streptococcal antigens, it seems possible that antibodies to streptococci may cross-react with self HLA antigens and initiate the accelerated divisions of epidermal cells typical of psoriasis (10).

Emotional factors have also been known to exacerbate psoriasis in people known to have the disease. Sometimes emotional factors have been seen to change the pattern of the disease (11). The skin of an active psoriatic plaque is characterised by accelerated epidermopoesis, proliferation of capillaries of the dermal papillae, increased epidermo-dermal infolding and migration of polymorphonuclear leuco-cytosis into the epidermis. These features are associated with acanthosis, paraketatosis and the abscence of the granular layer.

The rate of cell division is greatly increased in psoriatic lesions. Both the number of cells dividing and the rate of division are increased. The mitotic index (which is the number of cell divisions per 100 cells) of the germinative cells is increased (12,13). The psoriatic plaques hence form because the rate of formation of the squamous cell is greater than the rate at which they are shed off the skin. The horny layer of the epidermis shows considerable hyperkeratosis which often alternates with parakeratosis. The air spaces intersperse between layers of poorly adherent, flattened parakeratotic cells giving the characteristic silvery appearance of the scales.

Many minor immunological modifications have been described in psoriasis including increased serum lgA levels, the presence of anti-lgA antibodies and the decrease of E rosette formation (7). The latter seem to be transitory and revert to normal after clearing of the skin lesions. More recently, however, alterations of phagocyte functions have also been described (7). Contrary to humoral and cellular immunology, enhancement of phagocyte functions seems to be independent of the condition of the skin. Thus they are believed to be primary abnormalities similar to the primary psoriatic abnormality causing the increased proliferation of the epidermal cells. The study of phagocytic function in psoriasis has thus lent support to the possibility that the psoriatic abnormality is not confined to only one type of cell, (the epidermal cell) as was previously assumed, but may be a systemic disease (7).

Psoriasis presents in various morphological types and the commonly encountered types are as follows (14):-

1. Guttate psoriasis: These are small lesions appearing generally over the body, particularly in children and young adults. The lesions are usually less than 1 cm in diameter.
2. Nummular psoriasis: This is the most common form. They vary in diameter but are usually from 1 cm and more. It is commonly seen on the limbs and trunk.
3. Rupiod psoriasis: This describes the limplet like lesions with a cone shaped hyperkeratosis seen particularly on the feet.

4. Elephantine psoriasis: This is the persistent thickly scaling plaques sometimes seen on the back, thighs and hips.

5. Flexural psoriasis: Occur in skin flexures as in the groins, axilla and submammary areas of the skin folds.

6. Exfoliative & Pustular types are quite descriptive in themselves.

The chief clinical characteristics of psoriasis are (14):

a) A silvery grey scaling papule or plaque with a sharp margin especially in chronic lesions.

b) The scaling tends to be variable and removal of the scale by gentle scratching or scotch tape stripping reveals numerous minute bleeding points - Auspitz's sign.

c) The lesion does not produce scarring and is not vesicular.

d) The psoriatic plaque may be single or numerous. There is a strong tendency to distinctive distribution of multiple lesions to the scalp, elbows, knees and lumbosacral regions.

e) Another basic characteristic which is however, not pathognomonic is the Koebner phenomenon. This is the tendency of the skin to react to physical or chemical trauma in a psoriasi-form pattern. This manifestation is seen in psoriasis developing on the scalp or any other area which had been the site of seborrhoeic eczema, on the elbows and knees at sites of repeated trauma and

also on apposing skin in obese patients as in groins or flexures.

f) Itching is not a constant feature but may be seen in lesions in which collateralor secondary dermatoses are operative as in the scalp and in the anogenital region.

g) Nail changes are common in psoriasis but are variable in severity. The most common changes are pitting of the nail plate and transverse ridging and furrows.

h) Psoriatic arthritis is said to occur in about 1% of patients with psoriasis. Other studies have given varied figures of an incidence of psoriatic arthritis of 7% (15). It is said to be common in patients with severe extensive involvement of the skin by psoriasis particularly the exfoliative type. It affects mainly the hands and feet and it is not possible to differentiate it from seronegative rheumatoid arthritis. Its typical site also is the sacro-iliac joint where its differential diagnosis includes arthritis of Reiter's Disease, ankylosing spondylitis, Crohn's Disease and ulcerative colitis.

Psoriasis was for a long time considered a rare disease in the African. This observation was due mainly to the poor medical facilities available to the African patient. With improved medical facilities now available, many African patients are now presenting themselves to hospitals and other health care centres for treatment.

Psoriasis is now a topical research subject and very few dermatologic journals exclude it from discussion. This,

however, is true in the temperate regions of the world, i.e. Europe and North America. Most of the research has been on the possible aetiological factors, pathophysiology and new methods of treatment (15).

It has been observed that patients with psoriasis (50%) have increased serum uric acid levels (15). The cause of the hyperuricaemia and its possible relation to the psoriatic process has received very little attention. In psoriasis the skin lesion is a result of hyperplasia of the epidermis in which the rate of cellular mitosis is accelerated to such an extent that maturation of the epidermal cells and the process of normal keratinization do not occur. By analogy with the hyperuricaemia seen in myeloproliferative diseases, it is possible that the hyperuricaemia of psoriasis may reflect an increased nucleic acid turnover resulting from the marked acceleration of epidermal proliferation that occurs in this disease (16).

Other important causes of hyperuricaemia include gout, hypertension, diuretics, lymphomas, multiple myeloma, congestive cardiac failure, toxaemia of pregnancy, exercise, alcohol ingestion, and shock (17). The mechanism for the latter three conditions is due to direct competition for the urate secretory mechanism by lactic acid, although an increased tubular reabsorption of uric acid may also contribute in some of these situations.

MATERIALS AND METHODS:

The study involved screening of all patients with psoriasis who attended the dermatology clinic at Kenyatta National Hospital between July, 1980 to November, 1980. Those patients who were found to have other concomitant diseases were excluded from the study. Both old and new cases of psoriasis were included in the study. Patients were treated in strict confidence and their names did not appear in the dissertation. Verbal consent was obtained and relevant vital statistics of age, sex, tribe and estimated extent of skin involvement were recorded. The estimated extent of skin involvement was obtained by the rule of nine (as used for burns patients)(18). The rule of nine divides the body into the following varied percentages with a total of 100%. The trunk is 36% of the body surface - anterior 18% and posterior 18%. The lower limbs are each 18% of the body surface. The upper limbs are each 9% of the total body surface. The head and the neck is 9% and the genitalia is 1%. Attempt was made at trying to lower the interpersonal errors by getting one of the dermatologists to make an assessment of the skin involvement and the average of the two was taken as the estimated percentage of skin involvement. A sample of 5 mls clotted venous blood was taken for uric acid analysis and personally sent ot the laboratory. A tourniquet was applied at the middle of the upper arm and the sample of blood was taken from the vein at the antecubital area.

The uric acid level was estimated by the simultaneous multiple analyser II (SMA II) (19). This is an automated adaptation of the manual uric acid procedure and uses an autoanalyser. The principle being that uric acid is oxidised to allantoin and carbon dioxide by a phosphotungstic acid reagent in alkaline solution. Phosphotungstic acid is reduced in this reaction to tungsten blue which is measured at 660 nm. Phosphotungstic acid serves both as a protein precipitating agent and as a colour reagent. A glycerine silicate reagent increases the sensitivity in this reaction and also provides alkalinity for the reduction of phosphotungstic acid to tungsten. The decrease in absorbance of light is proportional to the uric acid initially present. Sodium polyanethol-sulphonate is added to prevent turbidity (19).

RESULTS

Age Distribution:

The ages of the patients were in decades as in the graph below. There were a total of 94 patients in this study. The youngest patient was 17 years old. The oldest patient was 78 years old. The peak occurrence was in the 21-30 age group with the mean at 34.9 years. The standard deviation SD = $\pm$ 12.2.

Table 1: Age Distribution

Age groups in years	Numbers	Percentages
0 - 10	0	0.0
11 - 20	11	11.7
21-30	33	33.0
31-40	27	28.7
41-50	13	13.8
51-60	8	8.5
61-70	3	3.2
71-80	1	1.1
TOTAL	94	100

96

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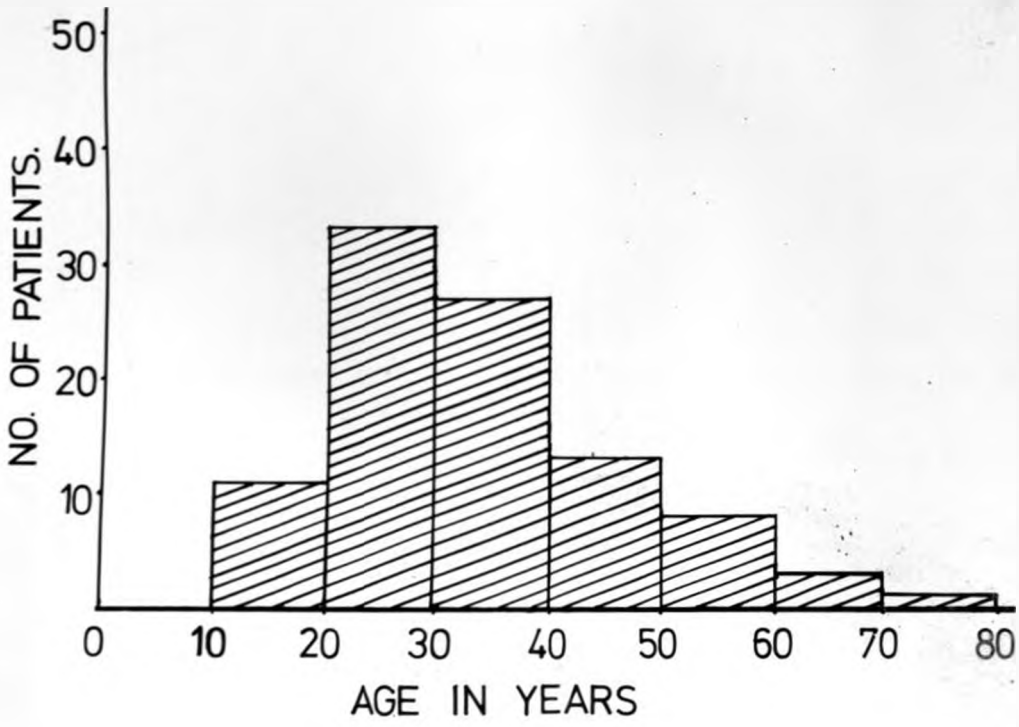
Fig. I AGE DISTRIBUTION



From the above table and figure it can be seen that there was no patient with psoriasis below 10 years of age. Also it can be seen that the peak period of psoriasis in this study was between 20-30 years which is a fairly young age. There was a higher prevalence of psoriasis in the third and fourth decade as in figure 1 above.

Table 2: Sex Distribution

Sex	Numbers	Percentages
Males	59	62.8
Females	35	37.2
Total	94	100

From the above table there were more males than females.

P = 0.04.

Table 3: Tribal Distribution

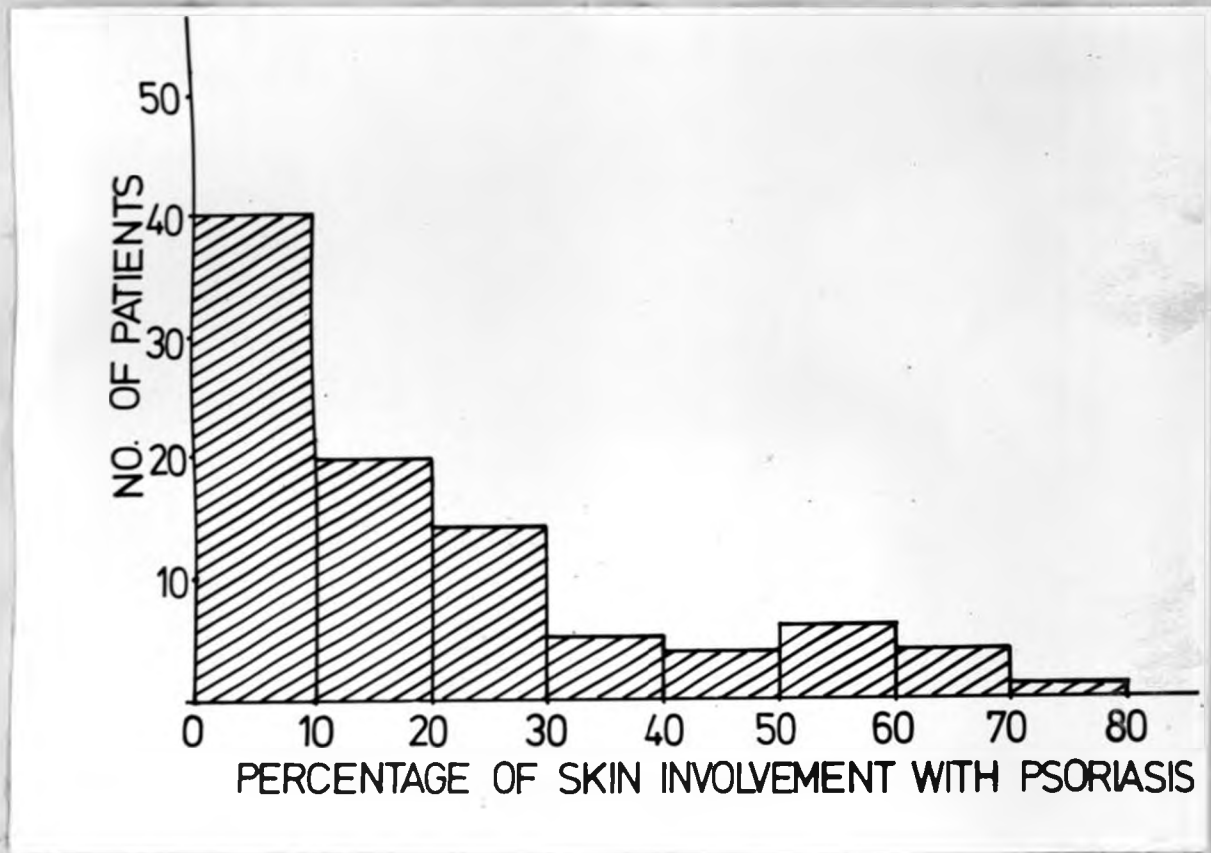
Tribe	Numbers	Percentages (%)
Kikuyu	71	75.7
Kamba	12	12.8
Luo	6	6.2
Luhya	2	2.1
Others	3	3.2
Total	94	100

The above table of tribal distribution was dominated by the Kikuyu tribe. This was as expected as Kenyatta National Hospital is situated in Nairobi which is partly in Kikuyu land.

Table 4: Skin Involvement

Skin involvement (%)	1-10	11-20	21-30	31-40	41-50	51-60	61-70	71-80	Total
Nos.	40	20	14	5	4	6	4	1	94
%	42.6	21.3	14.8	5.3	4.3	6.3	4.3	1.1	100

Figure 2: Skin Involvement

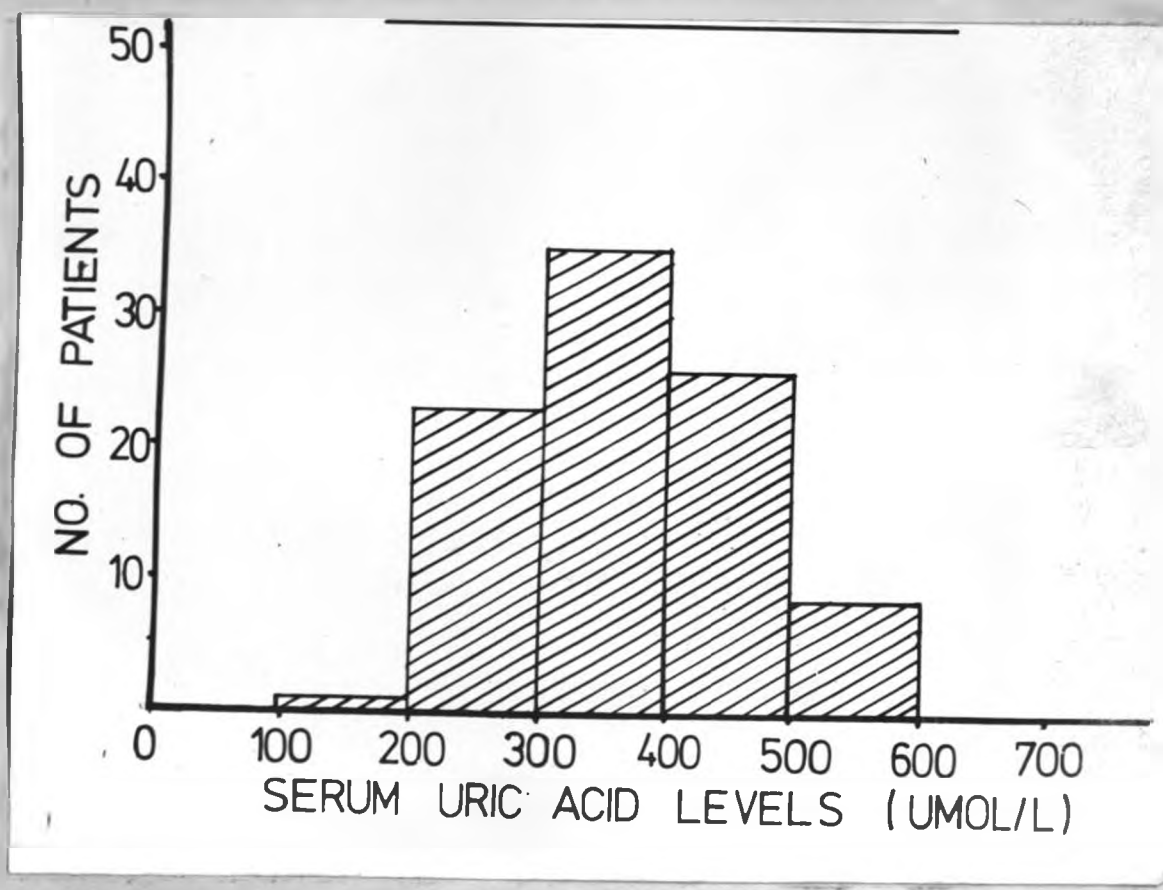


The skin involvement was recorded as shown in Table 4. The mean value of skin involvement was 21.8% with a standard deviation (SD) = $\pm$ 19.3. The large standard deviation value is due to the marked skewing of the distribution of skin involvement to the right as can be seen in Figure 2. It is also evident from this figure that many patients (42.6%) had less than 10% of skin involved with psoriasis.

Table 5: Serum uric acid levels

Uric Acid (Umol/L)	0-100	100-200	200-300	300-400	400-500	500-600	Total
Nos.	0	1	23	35	26	9	94
%	0	1.1	24.5	37.1	27.7	9.6	100

Figure 3: Serum uric acid levels



Serum Uric Acid Levels (Umol/L)

Uric acid distribution:

The standard range of normal serum uric acid values used at Kenyatta National Hospital is from 120-420 Umol/L. Any value above 420 Umol/L was considered high. In this study the serum uric acid values ranged from 190 Umol/L as the lowest upto 576 Umol/L as the highest. The mean value of uric acid was 367.4 Umol/L with a standard deviation SD = ± 92.4.

From table 5 and Figure 3 it can be seen that the peak number of patients had values between 301-400Umol/L with a normal looking distribution curve. This is quite in keeping with uric acid distribution in a normal population.

Table 6: Normal and high uric acid levels

Uric Acid Level	No.	%
Normal	66	70.2
Hyperuricaemia	28	29.8
Total	94	100

From Table 6 it is evident that majority (70.2%) of the patients had normal serum uric acid. Only 28 patients (29.8%) had high uric acid and psoriasis. Of these patients only 5 were females. Their ages were 18, 28, 35, 45 and 66 years.

Table 7: Serum uric acid and serum creatinine levels

	Normal Uric Acid		High Uric Acid	
Creatinine Level	No.	%	No.	%
Normal Creatinine	49	79.1	18	64.3
High Creatinine	17	20.9	10	35.7
Total	66	100	28	100

The upper limit of normal creatinine value used in Kenyatta National Hospital is 106 Umol/L. Any value above this was taken as high. The patients were divided into those with hyperuricaemia and those with normal uric acid values and their creatinine values analysed as shown in Table 7.

There was 35.7% with high creatinine levels among those with hyperuricaemia and 20.9% among those with normal uric acid values. The high creatinine here did not seem to be associated with any other renal disease apart from possibility of hyperuricaemia.

The coefficient of correlation for uric acid with the estimated severity of skin involvement was found to be $r = 0.82$ with $p = 0.001$. This is a significant value for the correlation between estimated extent of skin involvement and the serum uric acid levels in psoriasis patients.

DISCUSSION

Psoriasis is a chronic papulosquamous skin disease which has been known for a long time. It has the highest number of repeat visits by the patients per year of any skin disease and besides it is emotionally and physically debilitating (15).

Psoriasis is said to be rare in the African patient. It is also said that some cases on further investigations are found to have European ancestry (20). In this study all the patients were obviously people of black African origin. Therefore, the claim made earlier, that those African patients who had psoriasis were most likely of mixed (European) ancestry is untrue. This notion if maintained would put more weight on the genetic origin of psoriasis. No aetiological agent has been found in this disease so far.

It is possible that climate (sunshine) may play a big role in its aetiology as it has been observed that the disease is less common in Italy but exceedingly common in Iceland (21). Sunshine alone cannot be taken in isolation to be the aetiology as it is further observed that Europeans emigrating to the tropics to escape from their psoriasis are usually doomed to disappointment as the disease nearly always gets worse (22).

In this study there is some difference with other studies done elsewhere in the temperate regions. In one of the series where 419 consecutive patients with psoriasis were studied, the peak age of occurrence of psoriasis was between 5-9 years for girls and between 15-20 years in boys (23). In this study there was no patient seen less than 10 years in age.

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However, after taking the duration of illness into consideration, only one female patient was found to have developed the disease for the first time at the age of 6 years. The peak age of occurrence of psoriasis in this study was between 20-30 years (see Table 1 and Figure 1).

The age distribution is skewed to the right giving very low occurrence figures after the 6th decade of life (i.e. from 61-70 years age group). The age of onset of psoriasis according to Cavalieri et al, is a genetically determined trait inherited dominantly (1). But as we saw earlier on in this dissertation the disease is caused by a multiple of factors and thus its age of onset would therefore not be simply determined as postulated by Cavalieri.

From Table 2 it can be seen that psoriasis is found in a significantly higher percentage of males (62.8%) as compared to females (37.2%) ($P = 0.04$). This is in keeping with most other studies of large population groups where it was consistently found that males were frequently more affected with psoriasis than females (24). One of the studies showed that twice as many males were affected with psoriasis (25). It is known that pregnant females with psoriasis get some relief during pregnancy. Progesterone and oestradiol are significantly elevated in pregnancy and are required for its maintenance. In non pregnant females these two hormones have cyclical blood level patterns. In the males on the other hand, the main hormone is the testosterone.

It is as yet not known what actual mechanism causes the amelioration of psoriasis skin manifestations in pregnancy. However, it has been observed that steroids slow down the epidermal cell turn-over in psoriasis by a dual effect at separate stages of the cell cycle. Steroids slow down mitosis as well as decreasing the rate of DNA synthesis of cells (26). It may be that the high levels of progesterone and oestradiol in pregnancy slow down proliferation of the epidermis in psoriasis thus producing the relief of symptoms which invariably reappears some time after delivery.

The tribal distribution show as expected,that the largest number of patients (75.7%) were Kikuyus. This is due to the geographical locality of Kenyatta National Hospital, where the study was done, which is in close proximity to the heavily populated Kiambu district of Central Province. The second group was seen in Kambas with 12.8% and again this could be explained on geographical origin of Kambas, i.e., Machakos which also borders on Nairobi but is less populated than Kiambu district. Luos were 6.2%, Luhyas 2.1% and others 3.2%. In these other tribes the patients were mainly those living in Nairobi and could hardly be said to be representative of their various tribes as far as psoriasis is concerned. A rural based study of occurence of psoriasis should be done in Kenya to clearly show the tribal distribution pattern.

The estimated extent of skin involvement shows that most patients (42.6%) had less than 10% of skin involved with psoriasis.

The mean for the estimated skin involvement was 21.8% with a standard deviation SD = ± 19.3. The large standard deviation is due to the marked skewing of the distribution curve to the right as can be seen from Figure 2. It is also evident that over half of the patients (63.9%) had skin involvement of upto 20% only.

From Table 5 and Figure 3 it can be seen that the 300–400 Umol/L interval of uric acid has the largest number of patients (37.1%). The mean value of serum uric acid levels is 367.4 Umol/L with a standard deviation SD = ± 92.4. In this study the majority of the patients had normal serum uric acid levels (70.2%) with only 29.8% having raised serum uric acid levels. These results are different from some other study where it was found that 50% of the studied patients had raised serum uric acid (15). Also amongst those with hyperuricaemia there was again a striking male predominance (i.e. 82.2% males and 17.8% females). This finding is however not strange as it is known that hyperuricaemia is much less common in females - particularly before menopause. This is also exemplified in the disease gout which is quite rare in females and much more so during the child bearing ages, i.e. up to about 45 years.

Other diseases in these patients were excluded on clinical examination. All the patients were put on dithranol 0.1% to apply twice daily and 1% hydrocortisone 2% salyclic acid and 3% diodoquine to apply twice daily. Only nine patients needed additional oral antihistamines to treat pruritus.

They were given chlorpheniramine tablets 4mg twice or thrice daily depending on the severity of pruritus and occupation of the patients. One more patient (male) needed methotrexate orally for intractable psoriasis. No patient was excluded because of hypertension as none was found to have high blood pressure - diastolic greater than 95 mmHg.

Again amongst those with hyperuricaemia and those with normal uric acid levels creatinine levels were analysed as shown in Table 7. It was found that those with hyperuricaemia 35.7% of then had high creatinine levels and those with normal uric acid there were 20.9% with high creatinine levels.

Chronic hyperuricaemia is one of the aetiological problems in chronic renal failure.

There was a strong positive correlation between serum uric acid and the skin involvement ($r = 0.82$ and p 0.001). This is in slight contrast to the results found by Arthur Eisen and Seegmillar in which they found that the serum urate values correlated with the estimated percentage of skin involved with psoriasis, $r = 0.567$ (16). They also found that hyperuricaemia is frequent only in extensive psoriasis. According to Eisen and Seegmillar (1961) hyperuricaemia arises from uric acid over-production, as is indicated by excess incorporation of C^{14}- glycine 1 into urinary uric acid. The form of incorporation curve suggests an increase in nucleic acid metabolism in cutaneous lesions, where epidermal cells are rapidly renewed (27).

This study will probably suggest the possible prophylatic use of allopurinol in hyperuricaemic psoriatic patients in an attempt to lower the expected high uric acid levels in some patients to prevent the development of acute or chronic renal failure. The exact extent of skin involvement at which this prophylaxis can be used has not as yet been determined and could be taken up at another study.

Figure 4

Patient with (exfoliative) severe psoriasis.

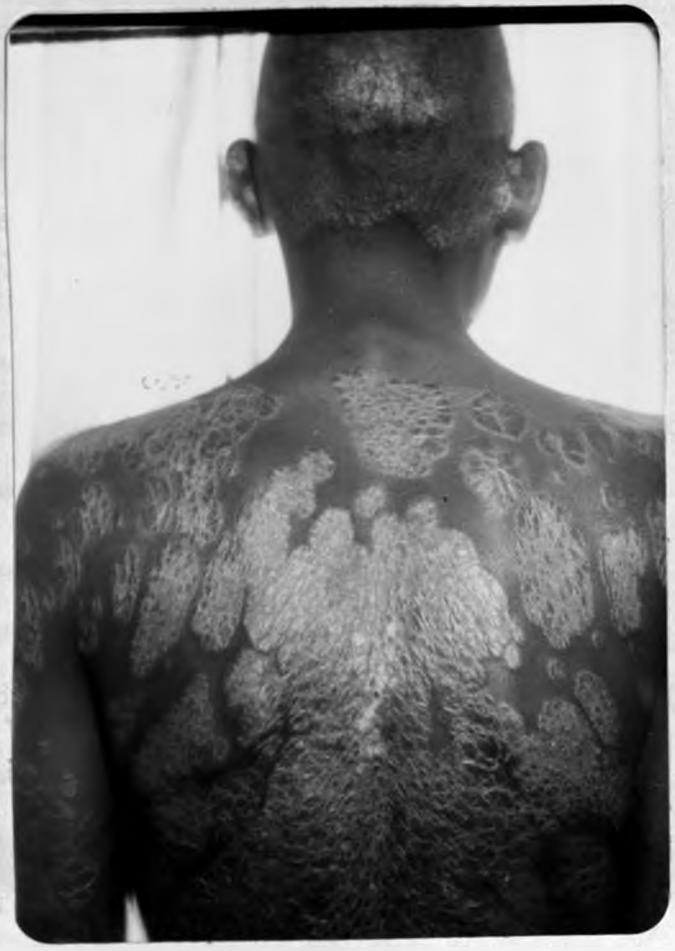

Figure 5

A close up view of the patient on Figure 4.

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