Genital lichen sclerosus (lichen sclerosus et atrophicus) in childhood and adolescence

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Introduction

Lichen sclerosus (lichen sclerosus et atrophicus: LS) was first described by Hallopeau[1] in 1887 and its typical histology defined by Darier[2] in 1892. The lesions are ivory-white papules and plaques, often showing central delling, atrophy, telangiectatic speckling and purpura (Figure 1). The histology is characterized by a thin flat epidermis, a sub-epidermal zone of oedematous and hyalinized collagen and a deeper band of lymphocytes (Figure 2). The elastic tissue in later stages tends to disappear. Where the skin has become thickened in response to rubbing the epidermis is hyperkeratotic and acanthotic and the rete ridges strikingly pointed and irregular.

Both clinically and histologically there are marked resemblances and associations between LS, lichen planus and morphoea (localized scleroderma); it is not established whether one should regard the three as part of a spectrum or as three distinct but significantly associated conditions.

LS is a dermatosis which may affect all areas of the body, at all ages and in both sexes. Its most common manifestation is in the genital and anogenital area in women; hence much confusion arising from separate consideration in gynaecological literature. Its aetiology is unknown although various factors are thought to be involved[3]. It is established that there is a significant association with autoimmune disease[4-6]. HLA findings have been inconclusive; the latest report[7] found a significant association with HLA A29 and B44 separately and, even more strongly, together; discrepancies in reports might perhaps be ascribed to differences in techniques, populations, and sex and clinical patterns in the patients. Familial cases are well recognized.

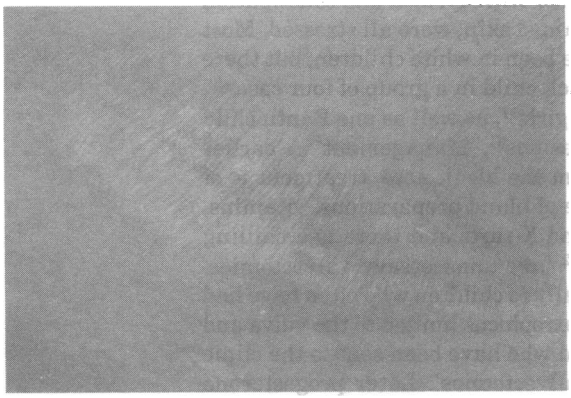

Figure 1. Lichen sclerosus on trunk showing typical plaques

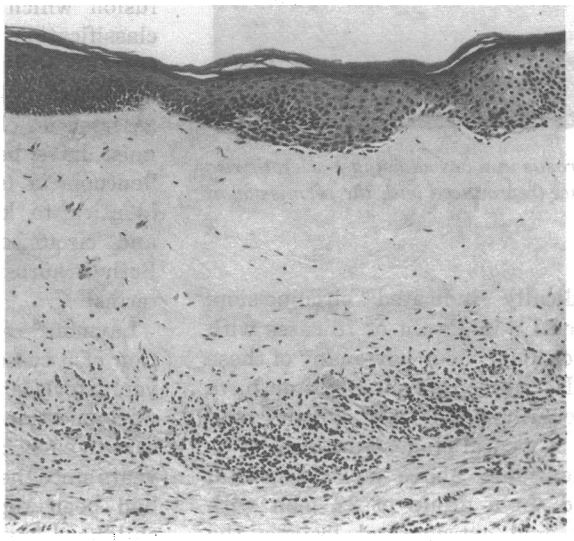

Figure 2. Histology: atrophic epidermis, hyalinized dermis, band of inflammatory cells (H&E×60) (reproduced with the permission of C H Buckley)

The recognition of its occurrence in the genital area and assessment of its natural history in childhood and adolescence have slowly evolved. These aspects of the condition will now be considered with the aim of clarifying, if not of fully answering, some of the questions which persist. A review of the earlier literature will be followed by presentation of a personal series of cases in girls and then by a survey of the current position.

Genital lichen sclerosus in boys

On the subject of genital LS in boys there is relatively little to say. Early reports stress its rarity, and those discussing LS in girls constantly emphasize how much rarer the condition appears to be in boys. Yet Rickwood *et al.*[8] showed that, whereas the prepuce in male children circumcised only for religious reasons appeared to be normal, there was good evidence of LS in the prepuce of 20 out of 21 boys aged 4-11 years with phimosis, all of whom showed scarring at the tip of the prepuce; one child in this group had possibly, in addition, meatal involvement. Furthermore, Chalmers *et al.*[9] in 100 prepubertal boys referred for medically indicated circumcision demonstrated LS in 14, all but one between the ages of 5 and 11 years; the patients with LS usually showed a sclerotic rim at the tip of the prepuce and some had meatal stenosis and lesions of the glans (Figure 3). Later reports have given figures of LS in 6 out of 140[10] and in 9 out of 59[11] boys

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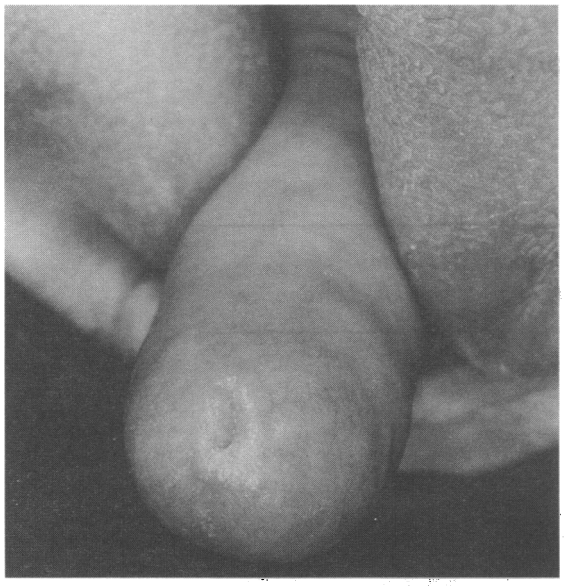

Figure 3. Lichen sclerosus in a boy showing a sclerotic rim at the tip of the prepuce (reproduced with the permission of Dr R Chalmers)

referred for medically indicated circumcision; another[12] mentioned LS in 15 out of 78 cases with phimosis but did not make clear how many of these were in children. Ten other cases in children have been described, five with meatal involvement[13], and a further example of meatal stenosis in a child[14].

In the series mentioned above no extragenital lesions were found. In a group of 22 men with genital LS[15] none had extragenital lesions; the author however has seen one adult patient with genital and extragenital lesions, and six such have been noted previously[16]. Of interest is the apparent absence of perianal lesions in boys (and adult males) contrasting with their frequent presence in females of all ages. The only possible exception is a somewhat unusual case reported by Loening-Baucke[17], a boy previously circumcised (reason not disclosed) with 'hypopigmented and sclerotic' (not biopsied) lesions perianally with, later, erosions on the penis and also showing what was thought to be LS on the fingers. Reported cases appear to have been in white children but one review mentions the diagnosis in a 9-year-old black boy[18].

The mode of presentation of LS in boys makes it likely that treatment will be by circumcision but it is probable that, as in adult males, treatment with the super-potent topical corticosteroid clobetasol propionate would be effective. There is no information on long-term follow-up in these reports, understandably so when the vast majority of the children appear to be free of trouble following circumcision. In a series of 22 adult men[15] no patients with lesions of the glans gave a past history of circumcision; another report[19] found that in 19 cases of LS in men not one had been circumcised in infancy; so it is conceivable that early circumcision would be a protection against the condition. The position appears unlike that in women where many young patients give a history of vulval problems as children.

Although it is likely that LS in male children, as in male adults, is indeed much rarer than in the female, many cases are clearly missed since those seeing the children are urologists or paediatricians rather than dermatologists, and histological examination of removed foreskin is not routinely carried out.

Anogenital lichen sclerosus in girls

For the female, there is a much larger body of literature. Early case reports[20-23] are of small numbers and stress the apparent rarity of the condition. For example a 1940 report[24] described 45 examples of LS, eight men and 38 women, but of these only one was a child, a girl with vulval lesions appearing at 10 years of age. Terminology is another factor to be considered. Although in 1922 Ormsby & Mitchell[25] had clearly recognized vulval 'kraurosis' as indistinguishable from LS, yet later authors still discuss 'kraurosis' as something quite different, a confusion which long dogged the steps of rational classification. A similar confusion in terminology surrounded the term 'leukoplakia', and two of the early cases of LS in children, as it can be confidently asserted on clinical and histological grounds they must have been reported under the headings of 'leucoplacia' (sic)[20] (considered by the authors to be identical to 'kraurosis' and similar to scleroderma) and 'circumscribed scleroderma or leukoplakia'[21]. Both 'kraurosis' and 'leukoplakia' are now obsolete terms[26,27].

Laymon[28] reporting three cases, says 'the observation of 3 cases of lichen sclerosus et atrophicus in young girls is noteworthy' and 'many dermatologists were questioned but none could remember seeing a case in early childhood'. Kindler[29] found that in the years since the 1940 account 54 cases of LS in women had been reported, including only four with a childhood onset of genital LS. She described eight cases of her own, six with anogenital lesions, and made the shrewd comment 'The observation within a comparatively short period of eight cases of lichen sclerosus in female children . . . suggests that the condition is not quite so rare at this age as is generally assumed. Possibly it is frequently overlooked owing to lack of symptoms, reluctance to examine these parts and inadequate knowledge of the disorder'.

Gradually LS in girls was divested of the shadows surrounding it and the clinical features became clear. Thus the occasional presence of extragenital lesions, the Koebner phenomenon in such lesions, the frequent involvement of the perianal area, the absence of symptoms in some children with anogenital lesions, and the frequent presentation of these patients with secondary infection or with dysuria and constipation because of the inflamed skin, were all stressed. Most cases reported have been in white children; but there are reports of a black child in a group of four cases[30], five cases in black girls[17], as well as one Bantu child with anogenital lesions[31]. Management in earlier reports ranged from the bleak note 'treatment is of no avail' to the use of bland preparations, vitamins, thyroid extracts and X-rays; and there is a chilling comment in 1962[32] '. . . unnecessary vulvectomies. This applies especially to children who often have had lichen sclerosus et atrophicus limited to the vulva and perianal region and who have been sent to the clinic for unnecessary vulvectomies'. Later progesterone cream, which was considered as a safer hormone preparation for children than the testosterone often advocated in adult women, was used. Gradually topical corticosteroids came to take a dominant role.

Of later case reports it is worth singling out those which make a particular point. One has suggested a

significant association with other abnormalities[33], the author finding 11 cases of gonadal dysgenesis in 32 girls with LS; this is so striking as to raise the possibility of some selection bias; an earlier report[34] found one child with nephrosis, two with congenital defects of the genitourinary tract and three with recurrent urinary tract infection in its 24 patients, but association with such abnormalities has not been further reported. It has been shown[35] how LS can be mistaken for child sexual abuse, a possibility also commented on much earlier[16]. Clearly child sexual abuse and lichen sclerosus may coexist; it is conceivable that sexual abuse could itself trigger off LS as a Koebner effect although this is generally held to be unlikely[36,37]. Others have added important information to the known familial tendency of LS[16,32], by reporting its onset in a close time relationship in both identical[38] and non-identical twins[39]. Autoimmune disease is reported in some cases[40] as one would expect.

Squamous cell carcinoma of the vulva is a well recognized risk of LS in adults, although one which is difficult to quantify. Authenticated examples of carcinoma in young subjects with LS are few, but likely to be of significance, and in carcinomas reported in other young patients the presence of LS may have gone unrecorded. Wallace[16] described carcinoma in two young adults, one with known and one with suspected LS since childhood. Pelisse[41] noted two cases, in girls of 17 and 18 years, on previously unrecognized LS. Cario[42] described an example in a (black) patient aged 18 years with long-standing lesions of LS, and Roman *et al.*[43] in a 22-year-old patient who was found to have LS. Although a malignant melanoma has been reported in a 14-year-old girl[44] with LS, a presumably coincidental occurrence, it is recognized that junctional and compound naevi may be easily mistaken for melanoma when found in association with genital LS in childhood and adolescence[45].

Natural history of lichen sclerosus

There are a few reports where follow-up of fairly large numbers was attempted. Of Kindler's six cases[29] none remitted under observation, although only one was followed over puberty. Ditkowsky *et al.*[46] described eight LS patients with histological confirmation, all carefully followed up. Two were said to show some signs of LS at the age of 12 years whereas all the others showed no signs of disease on examination at ages ranging from 6 to 14 years. Huffman[47] said that in two girls 'the vulvar lesions disappeared spontaneously during the early post-menarchal years'. Clark and Muller[34] described 24 cases, with a follow-up, in part by questionnaire, on 22. Of their anogenital cases, eight were reported as still affected and eight showed 'involution' though two noted 'easy irritability of the anogenital skin'. However, it is uncertain how many were actually examined. Clearance appeared to be without any definite relation to the menarche. Wallace[16] discussed the outcome of LS in 34 girls with anogenital lesions and noticed that symptoms improved with the menarche. He described the LS as persisting in 15 but in the others, in some of whom symptoms abated, it is not clear whether or not signs also improved. Török *et al.*[48] reported on 33 girls and again symptoms improved with time; in about 25% they noted resolution but these children also showed relapses with haemorrhagic blisters and they were not followed up over the menarche.

Dewhurst[49] noted 'no sign of diseased skin' at the age of 14 years in one out of six cases. Burova *et al.*[50] reported 12 girls with anogenital LS between the ages of 14 and 16 years; lesions had been present for many years but around puberty 'the scleroatrophic lesions disappeared completely' in eight of them. Berth-Jones *et al.*[51] noted that the lesions in one of their cases 'completely resolved'. Helm *et al.*[40] reviewed cases retrospectively. With an age at first attendance ranging from 3 to 30 years, and age at onset before 18 years in 33 patients, this series included 46 women in whom vulval lesions were present in 32 and perianal lesions in 10. The follow-up was retrospective and by telephone or questionnaire. The total of 52 patients included male adults and some with no anogenital lesions; the figures quoted of 'resolution' in 44% overall are therefore in the present context insufficiently precise to be of assistance. Another recent report[17], again relying at least in part on

Table 1. Details of 43 girls with lichen sclerosus

Age at onset (mainly of symptoms; occasionally of change in appearance)	2-13 years
Race	4 Asian, 1 Afro-Carribean/white, remainder Caucasoid
Biopsy confirmed	25 (biopsy not diagnostic in one clinically typical case)
Screening for autoimmune disease (blood count, liver function test, random glucose, thyroid function, organ specific auto-antibodies)	Performed 37 Positive results: 1 smooth muscle antibody 3 thyroid antibodies 1 smooth muscle & thyroid antibody
Autoimmune disease	3 (alopecia areata)
Family history of LS	2: 1 mother with LS 1 father with LS (and 1 patient had a brother circumcised at the age of 5)
Family history of autoimmune disease in first degree relatives	5: parents - 1 vitiligo, 2 thyroid, 1 alopecia areata sibling - 1 vitiligo
In grandparents, aunts or uncles	13
Extragenital lesions	2: 1 extensive and morphoea-like 1 only at the age of 20

telephone follow-up, noted that the condition in two pre-menarchal girls 'cleared completely'.

While all these reports present much clinical material of interest, reliable information on prognosis is limited, with few apparently definite findings. Clearly with a condition which is so often symptomless an objective assessment is all important. A point confirming that resolution is at all events not invariable is the history given by those presenting with LS as young adults; in one series[16] 19 out of 32 patients gave a history of vulval itching in childhood, and in another[6] there were 31 patients with onset in childhood, of whom 11 were assessed as adults and gave this history.

A personal series of girls with LS

These points will now be amplified with reference to a personal series of 43 girls with genital or anogenital lesions (Table 1). Of these 17 were included in an earlier study[6] relating to autoimmune disease.

Clinical features (Figures 4-6)

Management has been, in general, with emollients and mild topical corticosteroids. One girl had such marked loss of tissue on presentation that she had an examination under anaesthesia to exclude internal congenital abnormality; later she developed a peri-clitoral accumulation of secretion under the fused tissue. Such changes perhaps could now be minimized by the use of the super-potent topical corticosteroid clobetasone propionate, 0.05%, which has been used recently in some patients with great benefit.

Child sexual abuse was suspected in three patients and may well have occurred in one of these although proof was not achieved. There was a low incidence of autoimmune disease and of positive organ-specific antibodies, accounted for possibly by their youth. The same applied to first degree relatives, compared with the figures in a series largely consisting of adults; there was however a quite large incidence of auto-immune disease in grandparents; as the parents of these children are themselves fairly young, it may be that they will develop evidence of autoimmune disease in the course of time.

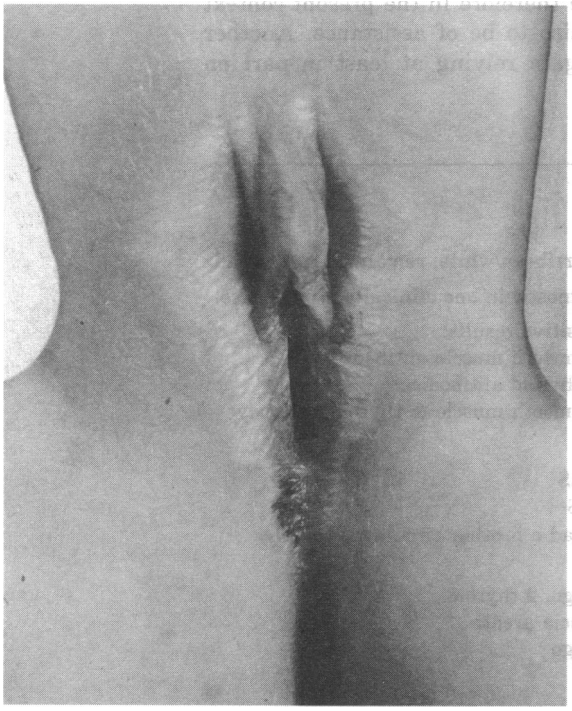

Figure 4. Typical lichen sclerosus in a girl showing pallor and atrophy

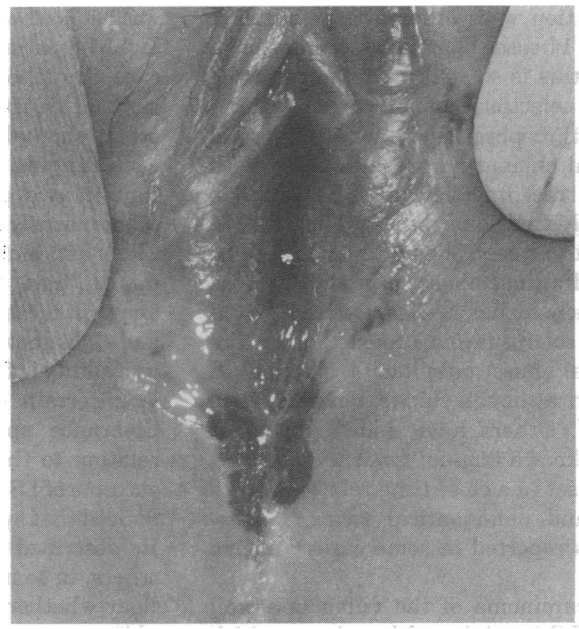

Figure 5. Lichen sclerosus showing pallor, atrophy and haemorrhagic areas

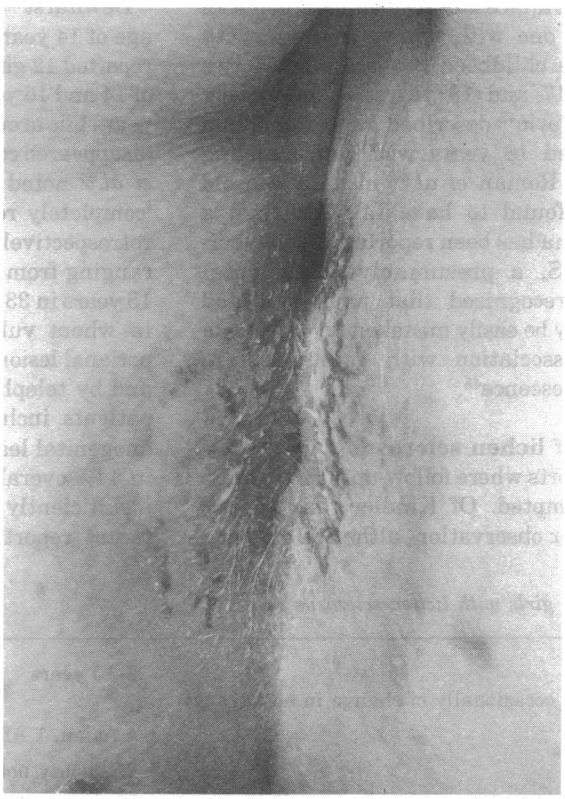

Figure 6. Lichen sclerosus showing perianal inflammation and infection

Several children had dysuria or constipation. In two the reason for the constipation was long unrecognized and inappropriate forms of treatment were employed including, in one, referral to a psychiatrist and development of a tendency to senna addiction and in the other admission to hospital and administration of enemas. In the latter child perianal lesions continued to be a problem after diagnosis, complicated

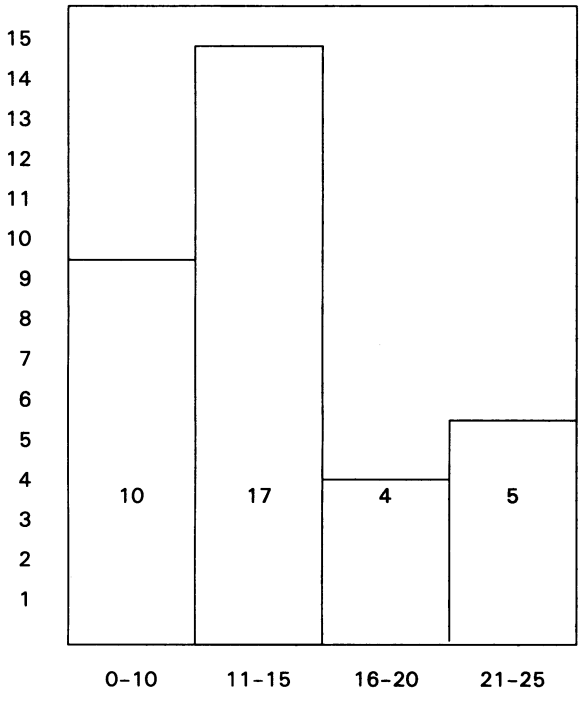

Figure 7. Age distribution (at time when last seen) of girls with persisting lichen sclerosus (36 patients)

by threadworm infestations and attacks of streptococcal dermatitis.

Two patients, although with biopsy confirmed LS, had lesions which from time to time appeared as festooned papules looking very much like lichen planus.

Follow-up data

Patients were followed up for periods ranging from a few months to 14 years. Six cases were lost to follow-up; they were last seen in childhood and all then had obvious LS; they will now be aged 7, 10, 11, 11, 13 and 15 years.

In the remaining 37 cases, signs of the condition have persisted in 36; their age distribution is shown in Figure 7. All these girls have unequivocal vulval changes, that is pallor and/or atrophy and/or loss of tissue; some have perineal and perianal signs. Many, however, are symptom free.

One girl, now aged 16 years, has, however, only minimal pallor of doubtful significance.

Discussion

The clinical picture of genital or anogenital LS in childhood and adolescence is now well delineated. Paediatricians and gynaecologists as well as dermatologists should be aware of it to prevent inappropriate management of presenting symptoms and confusion with child sexual abuse. It has been shown[51] that the diagnosis is made more expeditiously if a patient is referred to a dermatologist rather than to a paediatrician or a gynaecologist. The diagnosis can usually be made clinically. In view of its chronicity and uncertain long-term outlook it would seem wise to have a confirmatory biopsy to refer back to. This should not be pressed if it would cause distress; however it can often be achieved satisfactorily with a topical anaesthetic cream containing a eutectic mixture of prilocaine and lignocaine (EMLA) followed by painless injection of lignocaine and a 3 mm or 4 mm punch biopsy, even in quite young children. It is unfortunately the case that occasionally biopsy, even of a carefully selected site in a typical case, fails to give definite confirmation. If biopsy is not feasible a photograph is recommended. As regards current views on management, discussion with parents (perhaps with the help of an explanatory leaflet) is essential. It is important to enquire about a family history of autoimmune disease and advisable to carry out screening tests for autoimmune disease. Where there has been anxiety about child sexual abuse this should be allayed.

Emollients and mild topical corticosteroids with or without topical or systemic antibiotics are effective and non-controversial therapies. The use of the super-potent corticosteroid, clobetasone propionate 0.05% is justifiable for severe symptoms not related simply to secondary infection, and for those girls in whom the condition is severe and associated with a tendency to gross destruction of the vulval architecture. In the hands of experienced dermatologists, its use in milder, or even symptomless, cases is proving encouraging. When used close supervision, in monitoring a reducing regimen of application and the total quantity used, is mandatory to prevent, for example, hazardous absorption or striae and atrophy of non-affected areas; as well as, conceivably, a 'steroid vulva' analogous to 'steroid facies', where steroid-induced erythema leads to increased use and perpetuation of the problem; in practice this would seem to be rare. Although there are anecdotal reports of worsening of LS in pregnancy it is not adversely affected in that state in general, and no problem has been encountered in labour even in patients with a severely affected vulva. It is important that the obstetrician should be told of the background.

As regards the course of LS in childhood, it is important to stress that since there is no good evidence to suggest that LS is directly related to hormonal factors (the only formal study[52], measuring serum hormone levels in adults, being somewhat inconclusive) what one is really discussing when speaking of improvement in LS is the effect of time rather than that of any hormonal change.

The Vulval Invasion & Premalignancy Project instigated by the British Gynaecological Cancer Society aims at a prospective survey amongst dermatologists and gynaecologists of LS (and of vulval intraepithelial neoplasia in addition) and will yield large numbers for assessment. However, there are intrinsic difficulties in resolving the problem. Deficiencies in knowledge may arise from reliance on the patients' impressions rather than on careful examination, since signs may be florid but symptomless; a further impediment has been the disinclination of pubescent and adolescent girls to come to be examined. Even when the patient is so examined, signs may be minimal and the area 'almost normal'. Biopsy might help in these cases but would be unethical. Furthermore, should one assume that if the area appears 'quiet', ie pale and atrophic, and not obviously affected with papules, haemorrhage and so forth, that this counts as resolution? Who is able to assess activity of the disease process? Equally, should one accept the condition as 'resolved' even if gross destruction of tissue has been left in its wake? Moreover, the use of topical clobetasol propionate 0.05%, with its dramatic effects on the tissue both clinically and histologically[53], may actively modify the natural history.

Perhaps in trying to achieve some definite answer one is pursuing an ignis fatuus. However, on the basis of the previous reports and of the personal series presented, one may conclude that while in a very few patients the disease perhaps remits completely (possibly a recur later) certainly in most it persists into adult life.

Conclusion

The true incidence of LS in boys will only be established if lesions are recognized before operation or if tissue removed at the time of medically indicated circumcision is submitted to histological examination. Recognition at the earlier stage would permit a trial of medical treatment. Follow-up studies would be of considerable interest.

In girls, genital and anogenital LS is far from rare. Diagnosis and management are of considerable clinical importance. There is a very strong case for prolonged observation through adolescence and into adult life, from the point of view both of treating any symptoms that may appear and of seeing at an early stage any malignant change.

LS is a good example of those dermatological conditions which may have a multidisciplinary presentation. Genital lesions in young patients may present to physicians in genitourinary medicine, paediatricians, general surgeons, urologists and gynaecologists. There is a need for the paediatrician to be versed in some dermatological matters and here the growth of paediatric dermatology as a discipline will be beneficial. Clinics where dermatologists work alongside urologists and physicians in genitourinary medicine would be useful for male patients. Vulval clinics with a dermatologist and a gynaecologist and often a physician in genitourinary medicine are already well established. The incipient plans for linked training in dermatology and venereology will facilitate such interchanges.

Such cooperation is an argument not for less specialization within any one discipline but for more, so that we can join together in a position of strength to achieve the most insight into a given condition and hence its best management for the patient.

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Forthcoming events

FAIR Seminar: Influence and Stress Related Issues
22 March 1993, Royal Society of Medicine, London
Family Action Information Rescue (FAIR) is a voluntary organization founded in 1976. It offers support and information to individuals affected by cult organizations.
Further information from: BCM Box 3535, PO Box 12, London WC1N 3XX (Tel: 0689 853128; Fax: 0689 862531)

Aesthetic Surgery of the Face
25-27 March 1993, San Francisco, USA
Further details from: (see entry for 11-13 February 1993)

Advanced Cardiac Pathology
29 March 1993, London
Further details from: Postgraduate Education Centre, National Heart & Lung Institute, Dovehouse Street, London SW3 6LY (Tel: 071 351 8172; Fax: 071 376 3442)

Lung Tumours
30-31 March 1993, London
Further details from: (see entry for 29 March 1992)

British Association of Oral and Maxillofacial Surgeons: Spring Meeting
31 March to 3 April 1993, Cardiff
Further details from: The Honorary Secretary, British Association of Oral and Maxillofacial Surgeons, Royal College of England, 35/43 Lincoln's Inn Fields, London WC2A 3PN (Tel: 071 405 8074; Fax: 071 430 9997)

Mediastinal Tumours
1-2 April 1993, London
Further details from: (see entry for 29 March 1993)

Tinnitus and its Management
4-8 April 1993, Nottingham University
Further details from: Mrs Jean Willoughby, Course Administrator, c/o Institute of Hearing Research, University Park, Nottingham NG7 2RD

Trace Elements and Free Radicals in Oxidative Diseases
5-9 April 1993, Chamonix, France
Further details from: Professor A Favier/Mme A Alcaraz, Laboratoire de Biochimie C, Hopital A Michallon, BP 217X, 38043 Grenoble Cedex 09, France (Tel: 76 76 54 07; Fax: 76 42 66 44)

Postgraduate Course in General Surgery
15-17 April 1993, San Francisco, USA
Further details from: Extended Programs in Medical Education, University of California, Room LS-105, San Francisco, CA 94143-0742, USA (Tel: 415 476 4251)

7th British Knee Instability Course
20-23 April, Oswestry
Further details from: Erica Wilkinson, Symposium Secretary, Institute of Orthopaedics, Robert Jones & Agnes Hunt Orthopaedic & District Hospital, Oswestry SY10 7AG (Tel: 0691 655311)

26th Annual Advances and Controversies in Clinical Pediatrics
13-15 May 1993, San Francisco, USA
Further details from: (see entry for 15-17 April 1993)

9th Annual Current Issues in Anatomic Pathology
26-28 May 1993, San Francisco, USA
Further details from: (see entry for 15-17 April 1993)

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