CHRONIC DISCOID LUPUS ERYTHEMATOSUS WITH MULTIPLE KERATOACANTHOMA

Radha Mittal, Adarsh Chopra and S S Gill

A 62-year-old male patient had disseminated discoid lupus erythematosus and multiple nodules over the sun-exposed areas which were diagnosed as multiple keratoacanthomas. Nodules were asymptomatic and had a warty surface covered with thick scales.

Key words : DLE, Keratoacanthoma, Association.

Keratoacanthoma in a case of discoid lupus erythematosus was first reported by Farah.[1] Non-itchy, hyperkeratotic papulo-nodular lesions on the arms and hands resembling keratoacanthomata, hypertrophic lichen planus, prurigo nodularis or multiple acral fibrokeratomas may represent a distinct variant of discoid lupus erythematosus.[2]

Development of multiple keratoacanthomas in a case of discoid lupus erythematosus is very rare and our patient had disseminated discoid lupus erythematosus and multiple keratoacanthomas in addition to cytopathogenic changes typical of viral warts and this combination has probably not been reported earlier.

Case Report

A 62-year-old male was having disseminated discoid lupus erythematosus for the last $6\frac{3}{4}$ years. There was no systemic abnormality. He developed asymptomatic, scaly and warty nodules on the limbs and face one year back. One nodule left a superficial scar on accidental removal by a sharp object. Examination revealed typical lesions of discoid lupus erythematosus on the scalp, front of neck, trunk, upper and lower limbs. In addition, one nodule was present on the left malar region, four on the left forearm, one on right forearm, three on right lower leg and two on the left lower leg. Nodules were 1-4 cm in diameter, non-tender, mobile over underlying structures and covered with thick scaly warty projections. Thick scales were removed with topical retinoic acid and underlying nodules were firm, pinkish and had a warty surface with spontaneous haemorrhages at a few sites. One nodule was pedunculated. Nails of left hand showed discolouration with subungual hyperkeratosis.

Routine tests of blood, urine and stools were normal. ESR was 85 mm. VDRL test, rheumatoid factor and LE cell phenomenon were negative. Blood urea, fasting blood sugar, serum cholesterol, total serum proteins and screening chest were normal. A biopsy from upper chest was consistent with the diagnosis of discoid lupus erythematosus. Another biopsy from an early nodule on the leg showed mild hyperkeratosis, marked atrophy of epidermis and telangiectasia in the dermis with a severe lichenoid infiltrate touching the epidermis. A third biopsy from a large nodule on the forearm revealed a central keratinous crater with epithelial buttress formation, marked hyperkeratosis with horn pearls, hypergranulosis, acanthosis with pseudoepitheliomatous hyperplasia, marked papillomatosis, telangiectasia and marked cellular infiltrate in the dermis. Cytopathogenic changes in the malpighian layer, that is clumping of chromatin in the periphery of nucleus, prominent nucleoli, perinuclear halo and prominent dots in the cytoplasm and nuclei were seen. Hyalinisation with ground glass cytoplasm was seen at a few places. These changes were consistent with keratoacanthoma

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Comments

This is the first case of disseminated discoid lupus erythematosus with multiple keratoacanthomas and cytopathogenic changes characteristic of human papilloma virus. Prieto et al[3] showed influence of viral infection in the origin of keratoacanthomas. Claudy and Thivolet[4] reported that cell mediated immunity may play a major role in the defence against keratoacanthoma and its deficiency may explain the severity of the evolution of the lesions and their recurrences. Multiple keratoacanthoma and familial primary self-healing squamous epithelioma of Furguson-Smith type resemble each other closely and some dermatologists agree that these are distinct, while others think them to be variants of the same entity.[5] Multiple keratoacanthoma have been reported in association with deficient cell mediated immunity and this plays a role in the pathogenesis of viral warts and discoid lupus erythematosus. This simultaneous combination fo all the three diseases may be significant.

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MULTICENTRIC RETICULOHISTIOCYTOSIS

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This is the first reported case from India of multicentric reticulohistiocytosis, manifesting as multiple cutaneous nodules on the face, ears, elbows, wrists, knees and hands with mild pruritus. Polyarthritis involved the small and big joints. Histopathology confirmed the diagnosis. All the lesions disappeared in 1½ years time.

Key words : Reticulohistiocytosis, Multicentric.

Multicentric reticulohistiocytosis is a rare disease of skin and joints with distinctive histopathologic findings. Weber and Freudenthal in 1937[1] described systemic lesions in multicentric reticulohistiocytosis. Caro and Senear in 1952[2] reported two cases of multicentric reticulohistiocytosis and provided evidence that it was disorder of the reticuloendothelial system. Synovial membrane biopsy changes were similar to the changes seen in the skin. In 1954, Croltz and Laymon[3] used the term multicentric reticulohistiocytosis to differentiate those patients who had cutaneous nodules and definite arthritis from those with only cutaneous lesions.

Clinically, multiple, yellowish to brownish, pruritic nodules 2 to 20 mm in diameter may be present on the scalp, face, neck, ears, upper part of trunk, extensor surfaces of elbows, knees and hands. Mucosal papules are seen in about half the cases.

A symmetrical polyarthritis, insidious in onset, precedes the nodular eruption in nearly two-thirds of the cases. Women are affected more commonly. Large joints such as knees, hips and shoulders as well as smaller joints are effected. There is destruction of the interphalangeal joints which leads to accordion or concertina hands deformity. The arthritis is variable in behaviour, it persists in some cases and disappears in others without any reason. Orkin et al in 1964[4] reported that one-fifth of the patients had mutilating arthritis of the hands. Other features were weight loss, fatigue, pyrexia, lymphadenopathy, hepato-splenomegaly, tuberculosis, moderate elevation of ESR, reversal of A/G ratio, mild anaemia, hyperproteinaemia and pulmonary infiltration. Barrow in 1967[5] reported nail atrophy, longitudinal ridging, brittleness and hyperpigmentation etc. Taylor in 1977[6] treated one case of multicentric reticulohistiocytosis with systemic corticosteroids.

The striking histopathologic pattern is the presence of aggregates of histiocytic giant cells and histiocytes in the skin, synovia, mucosa and even bones. The histiocytic multinucleated giant cells can be as large 100 mm and multiple (upto 20) haphazard nuclei may be seen. Histiocytes and giant cells have finely granular pale eosinophilic cytoplasm with a ground-glass appearance. Nodular infiltrates may occupy the entire dermis and are unencapsulated. Other cell types are lymphocytes, eosinophils, plasma cells, RBCs and fibroblasts. The connective tissue stroma is pushed aside by the cellular masses and becomes vascularised. Histochemical studies indicate that the giant cells and histiocytes contain a PAS-reactive material that may be glycoprotein or mucoprotein, neutral fat, phospholipids, iron and melanin.

Case Report

A 32-year-male had rheumatoid arthritis of six months duration, first starting in the right knee and then involving the left knee, both elbows, wrists and hand joints within two

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months. At that stage, he also developed a generalised nodular eruption especially marked on the face, ears and extensor surfaces of elbows, knees and hands with mild pruritus. There was no fever or systemic illness, but he had loss of weight and fatigue. General physical and systemic examination were normal. There was swelling of the small joints of both hands, wrists, elbows and knees and painful restricted movements. Multiple, firm nodules, yellowish to brown in colour, varying in size from 5 mm to 2 cm and mobile over the underlying structures were present all over the body but were more prominent over the pinna of both ears and extensor aspect of elbows, knees and hands. There were no lesions on the mucosae. The investigations revealed, Hb 10 gm%, TLC 9700/cmm, DLC (P 69, L 26, M 3, E 2), rheumatoid factor negative twice, ESR 30 mm, serum uric acid 3 mg%, and serum cholesterol 235 mg%, X-ray both knees showed soft tissue swelling (Fig. 1). Histopathology showed large, round to oval histiocytes with abundant, eosinophilic, finely granular cytoplasm with a ground-galss appearance and with irregularly distributed multiple nuclei. There were empty spaces and fibrous tissue surrounding the giant histiocytes. Some histiocytes had single nuclei and a few inflammatory cells were also present (Fig. 2).

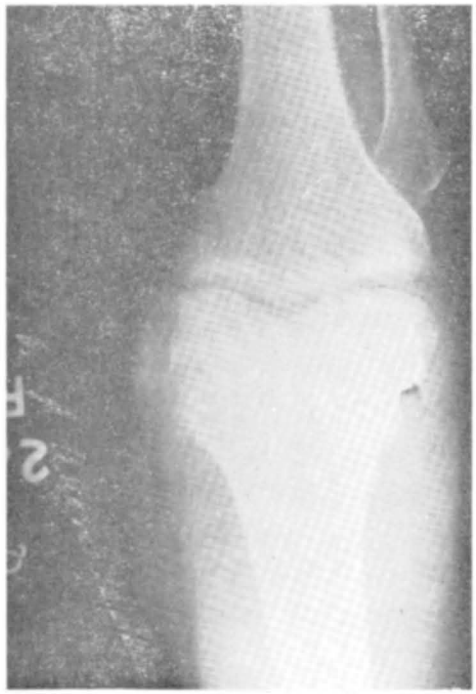

Fig. 1. Skiagram of right knee showing soft tissue swelling.

Fig. 2. Large histiocytic giant cells with peripheral empty spaces, with abundant cytoplasm and irregularly distributed multiple nuclei. Some histiocytes with single nuclei and a few inflammatory cells are also seen (H & E×400).

Comments

Exact aetiology of multicentric reticulohistiocytosis is not known. Johnson and Tilden in 1957[7] reported that though giant cells contain little or no sudanophilic lipid material, yet this condition may be related to the other histiocytic diseases such as xanthomatoses and the so-called lipoid storage diseases of the reticulo-endothelial system as many multicentric reticulo-histiocytosis

cases also have high serum cholesterol, xanthomatous lesions of eyelids and PAS positive glyco or lipoprotein and lipoid complex as in giant cells. So it was believed that mutlicentric reticulo-histiocytosis is a specific type of benign, non-neoplastic histiocytosis affecting the skin and joints as suggested by Croltz and Laymon.[3] It was thought that the histiocytic reaction may be in response to primary degeneration of the collagen.

Montgomery et al[8] however, wrote that this disorder was not an essential disorder of lipoid metabolism or rheumatic disease or neoplasia and there was no explanation why some cases improved after a few years while the disease was progressive in others. In our cases also, the skin as well as joint lesions completely disappeared after about 1½ years. Orkin et al[4] thought that histiocytosis-X shared certain facts with multicentric reticulo-histiocytosis and suggested that the suffix osis was preferable to oma since the latter signifies a neoplastic process.

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TOXIC EPIDERMAL NECROLYSIS FOLLOWING STEVENS-JOHNSON SYNDROME

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A patient who initially developed Stevens-Johnson syndrome and then toxic epidermal necrolysis is reported. The provocation with isonicotinic acid hydrazide was positive.

Key words : Stevens-Johnson syndrome, Toxic epidermal necrolysis, Isonicotinic acid hydrazide, Drug eruptions.

Stevens-Johnson syndrome (SJS) is the progression of erythema multiforme to involve several mucosal surfaces and internal organs accompanied by severe constitutional symptoms,[1] while toxic epidermal necrolysis (TEN) is characterised by wide spread erythema and detachment of epidermis resembling scalding.[2] Usually a patient suffers with either of these two during the illness but sometimes it is not so. SJS occasionally progresses to a condition indistinguishable from full-blown TEN.[1] On the other hand, TEN itself has been thought to be a maximal expression of serve erythema multiforme due to sharing of the lesions with the latter.[2] There are a few reports on the patients who presented with SJS but later developed the picture of TEN.[3,4] We herein report one more such patient who initially developed SJS and then TEN due to isonicotinic acid hydrazide (INAH).

Case Report

A 20-year-old male having scrofuloderma of two months duration in the neck was administered antitubercular therapy comprised of streptomycin, INAH and rifampicin. On the fourth day, he developed fever and generalised pruritus followed by labial, conjunctival and oral ulceration. It was associated with characteristic erythematous target lesions all over the body, palmar erythema and haemorrhagic crusting on the lips. The antitubercular treatment was withdrawn. He was diagnosed as a case of SJS and given the treatment accordingly. Two days later he developed flaccid bullous lesions all over the body. The Nikolsky's sign was positive. The skin looked scalded and at places it was coming out like a sheet leaving behind a raw bleeding area. The patient was febrile and looked toxic. This time a diagnosis of TEN was made. The patient showed uneventful recovery in two week's time with a heavy dose of systemic corticosteroids, i. v. fluids, antibiotics and supportive therapy. The antitubercular drugs were readministered one by one as described by Pasricha.[5] The provocation with INAH was found to be positive.

Comments

The SJS and TEN are usually regarded as different entities since these occur separately most of the time. The antitubercular drugs[6-8] have been reported to cause TEN. Shah et al[9] studied 17 cases of SJS. Out of these none progressed to TEN and two patients had received INAH and thiacetazone.

Due to the reporting of cases initially presenting as SJS and then as TEN,[3-4] a strong relation between the two entities is being suspected. The present case showed a similar type of transition due to INAH. Lyell[10] suggested that drug-induced TEN cases should be regarded as variants of drug-induced SJS which show this striking degree of necrolysis. Since TEN is a potentially serious skin disease with a bad prognosis, its early recognition becomes very

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vital. Assad et al[4] stated that a patient with erythema multiforme like lesions, severe involvement of conjunctivae and oral mucosa, fever and fine wrinkling of the skin over the lesions is likely to progress to TEN. We also believe that erythema multiforme, SJS and TEN constitute the spectrum of one disease in the order of severity.

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TINEA CORPORIS IN A PREMATURE INFANT

K Pavithran, A D Dharmaratnam and A Vijayalekshmy

A 16-day-old premature infant, born in the hospital developed tinea corporis due to *Trichophyton rubrum*. Her mother had tinea cruris due to the same species of fungus.

Key words : Tinea corporis, *Trichophyton rubrum*, Neonate, Dermatophytosis.

Even though tinea capitis is found to be more common in children, dermatophytic infection of the skin is only rarely seen in them. The number of cases reported in the literature of infants suffering from this infection is very small.[1-9] Here we report a case of tinea corporis due to *Trichophyton rubrum* in a 16-day-old premature infant.

Case Report

A 16-day-old infant girl was seen for a circinate patch of 7 days duration on the back. She was born prematurely by vaginal delivery in the same hospital at 32 weeks of gestation. The birth weight of the baby was only 1350 gm and she was taken to the neonatal ward for proper care. Mother noticed an erythematous circular patch on the back of her child on the ninth day of birth and the lesion gradually increased in size. (Fig. 1)

Examination revealed a well-defined, circular patch of 3 cm diameter on the back, with an active spreading border consisting of tiny papules, vesicles and crusts. Skin scrapings from the border of the lesion, examined in 10% KOH revealed the presence of numerous mycelia, some of them breaking up into arthrospores. *Trichophyton rubrum* was isolated on culture in Sabouraud's dextrose agar medium. Mother of the child had been suffering from tinea cruris since 3 months and culture of the scrapings from the lesion on her groin, also yielded growth of *Trichophyton rubrum*. There was no clinical evidence of dermatophytosis in the nursing staff or doctors of the neonatal ward. Topical application of 2% miconazole nitrate for 10 days led to complete disappearance of the skin lesions in both the mother and the infant and there was no evidence of any recurrence, when followed up for 2 months.

Comments

Jacobs et al[1] reported a case of tinea faciei due to *Microsporum canis* in a 8-day-old infant. A case of tinea corporis and tinea capitis due to *Microsporum canis* was seen by Alden and Chernita,[2] in an 18-day-old infant. *Epidermophyton floccosum* was the causative agent for

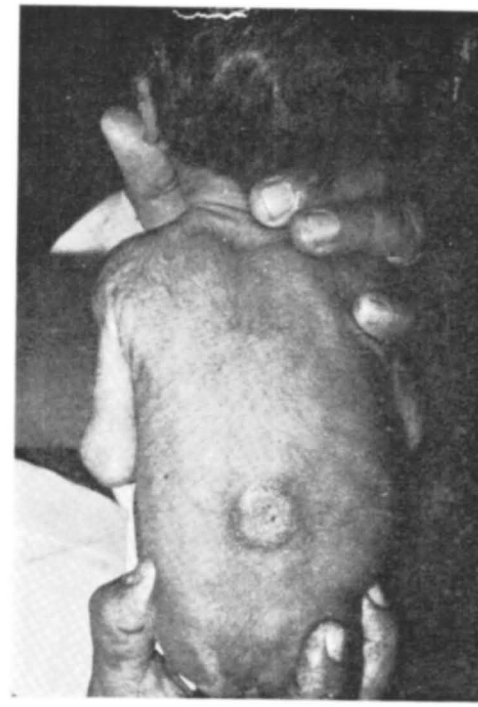

Fig. 1. The lesion on the back of the infant.

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tinea corporis in a 3-week-old infant reported by Yesudian and Kamalam.[3] Among ten cases of superficial fungal infections in infants reported by King et al,[4] two were aged only 3 weeks at the onset of the disease. Infants aged one month each were the youngest patients in the series of Mulay and Garg[5] and Sharma et al.[6] Bereston and Robinson[7] also noted this infection in a 4-week-old infant. Recently Khare et al[8] reported kerion, tinea faciei and tinea corporis due to *Trichophyton mentagrophytes* in a one and a half month old infant. The case reported by Kelly and Mackenzie[9] was a 2-months-old infant who had tinea corporis due to *Trichophyton verrucosum.*

In the present case, the source of infection was most probably the infant's mother who had tinea cruris and the fungi isolated from the skin lesions of both the infant and the mother belonged to the same species. Whether the infection occurred at the time of delivery or afterwards is not known. If the mother is presumed to be the source of infection, the maximum possible incubation period of dermatophytosis in this case is 9 days because the mother was quite sure that she first noticed the skin lesion, on the ninth day after birth. The size of the patch also suggested that this history was true.

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LETTERS TO THE EDITOR

AN UNUSUAL CLINICAL PATTERN OF CONTACT DERMATITIS DUE TO NICKEL-PLATED STRIP ON A BRIEF-CASE

Nickel is present in a variety of articles of every-day use and it is the commonest cause of contact dermatitis due to metals. Different nickel-containing articles produce different but very characteristic clinical patterns of contact dermatitis and most of us are familiar with most of these clinical patterns. There is thus generally no difficulty in recognising nickel as a cause of contact dermatitis. Sometimes however, the pattern of dermatitis can be unusual, if the mode of contact with the nickel-containing article is different. One such patient recently seen by us is described below :

A 50-year-old bank-manager had a 6-month history of itchy papular lesions involving the under-surfaces of both forearms and the medial aspects of the lower halves of the upper arms. The lesions developed first on the medial aspect of the left upper arm near the elbow, and within a month the lesions started appearing in a similar fashion on the right upper arm. Thereafter, the lesions gradually progressed to involve the medial and the under-surfaces of both forearms. Itching was moderate. He also noticed itchy lesions near the left wrist where he was wearing a watch with a metallic case and a metallic strap. Enquiries revealed that he had been carrying a brief case daily for 3 to 4 hours during his travel by bus or train, to cover a distance of 60 kilometers between his place of residence and the office. He used to keep the brief-case vertically on his thighs and rest his upper extremities on it in such a manner that the medial and the under-surfaces of his upper and forearms came in direct contact with the metal lining and the edges of the brief-case. At home and office, his upper extremities used to come in contact with the polished wooden table-tops and wooden arm-rests of chairs. He was routinely wearing half-sleeve shirts.

Patch testing revealed a 3+ reaction with nickel sulphate, while rubber, rexin of the brief-case, wooden scrapings of the chair, polish of the table-top, cobalt chloride, copper sulphate and potassium dichromate yielded negative results. He was instructed to avoid contact with all nickel-containing articles and especially the brief-case that he was carrying, to apply a topical corticosteroid cream and wear full-sleeve shirts. During follow-up, 2 weeks later, the patient was found to have marked improvement. He was wearing a full-sleeve shirt and he was still carrying the same brief-case. It seems wearing the full-sleeve shirt acted as an effective barrier between his skin and the antigenic article.

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ANGIOEDEMA DUE TO GUAVA

Urticaria and angioedema due to fruits like grape, plum, pineapple, banana, apple, strawberry and citrus fruits has been recorded,[1] but to our knowledge urticaria and angioedema due to guava has not been reported. We recently saw a 46-year-old government servant who had had 3-4 episodes of angioedema every year for the last 3 years. Each time the swelling would subside within 24 to 48 hours after taking oral antihistamines. There was no variation with the

seasons or the change of place. Exposure to heat, cold, sunlight, pressure, dust or drugs had not been recorded to precipitate the lesions. The last attack however, which occurred 3 months ago developed 3 hours after eating a guava which was bought at an orchard in Allahabad. The lesions had subsided within 24 hours after taking 25 mg pheniramine maleate orally. The patient did not eat guavas subsequently and had no lesions. In order to establish the association, we asked the patient to eat another guava. This time the patient ate a guava bought at a fruit seller's trolley. Six hours later, he developed angioedema on the scalp and the right sole which subsided within 24 hours after taking 25 mg pheniramine maleate orally. He was unable to recall if the attacks of angioedema in the past had occurred after eating guavas. We advised the patient to avoid eating guavas and he has not reported angioedema since then.

It is known that banana contains 5-hydroxytryptamine and excessive intake of bananas can precipitate/aggravate urticarial attacks. It is not known if guava also contains some vasoactive substance. Alternatively, it will be interesting to see if guava contains some histamine liberators which could non-specifically precipitate/aggravate urticarial attacks similar to those caused by aspirin. Our patient, on both the occasions, had eaten unwashed guavas making it possible that some insecticide used for spraying the fruits could also be responsible.

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CONTACT DERMATITIS TO BIDI

While reading the article "Contact Dermatitis to Bidi, 1985; 51 : 286", I found certain discrepancies.

The article concludes that the patient was sensitive to tobacco and the likely constituent of tobacco smoke which caused sensitization must be formaldehyde. My objections are : (1) The index and middle fingers of the patient were coming in contact with (a) Bidi leaf, and (b) the paper used for wrapping it, and not with the tobacco, which remains inside these two. Since the patient's fingers were not coming in contact with either tobacco, or its smoke in any way, then how could he develop allergic contact dermatitis with tobacco. Further, if tobacco smoke was sensitizer, then the mucous membrane of lips and oral cavity must also develop dermatitis. (2) If Bidi leaf and paper were sensitizers (although patch test was negative with both these) then besides fingers, lips must also have developed contact dermatitis because lips were also constantly in contact with the Bidi leaf and its paper. But this also was not the case.

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REPLY

I appreciate the critical reading by Dr. Vora of our article, 'Contact Dermatitis to Bidi'. However, I certainly disagree at the outset with the remark that we have concluded formaldehyde as the likely cause of sensitization in our case. In fact, it is a part of the discussion.

Regarding the unlikeliness of the *Bidi* smoke coming in contact with the fingers of the patients; it is a well known fact that the smokers do deve-

lop brownish yellow staining of the index and middle fingers where bidi or cigarrette is usually held.

It is also a common knowledge that contact dermatitis of the oral mucous membranes is rare except on the vermilion border of the lips. During smoking, however, this part is least likely to come in contact with the smoke. Explanations advanced for rarity of allergic contact dermatitis of mucous membrane are : (a) washing off of the antigen rapidly by the secretions, and (b) absence of well keratinised layers on the mucosae.

Routinely, we do patch testing only once, until and unless there is a strong clinical disparity. We agree that in the present situation, ideally, repeat test should have been done, because it was a new observation.

In the present case, we think that the constituents of tobacco in the form of smoke were diffusing out to produce contact dermatitis and so the tobacco gave a positive patch test.

N L Sharma