ABSTRACTS FROM CURRENT LITERATURE

Metastatic squamous cell carcinoma in linear porokeratosis of Mibelli, Lozinski AZ, Fisher BK, Walter JB et al: J Amer Acad Dermatol, 1987; 16: 448-451.

Linear porokeratosis usually appears in childhood and presents as a linear verrucousnevus like lesion, usually on the extremities. Here the authors report a 45-year-old woman with linear porokeratosis of Mibelli who developed squamous cell carcinoma in lesional sites. She had skin lesion on the back of calf since 9 years. In 1970 a skin biopsy from the lesion showed features of squamous cell carcinoma and had been treated with local radiation. In 1980 she was seen for evaluation. Pathological study of the enlarged inguinal lymph node of the right groin showed features of welldifferentiated squamous cell carcinoma. glands were excised and she was given radiotherapy. It is believed that porokeratosis evolves from a clone of abnormal epidermal cells at the base of coronoid lamella. These cells are more sensitive to ultraviolet light and often show degenerative and dysplastic changes. nancy is thought to develop from these areas of epidermal dysplasia.

K Pavithran

Coexistant leprosy and lupus erythematosus, Posner DI and Guill III MA: Cutis, 1987; 39: 136-138.

A 42-year-old woman developed erythematous anaesthetic plaques on the knee and elbow, while on systemic corticosteroid therapy since 8 years for associated systemic lupus erythematosus. The cutaneous lesions of SLE showed regression by corticosteroid therapy, but the lesions on the elbow and knee were recalcitrant. Histopatho-

logical study of the plaque revealed lympho-histiocytic granulomas in the dermis with perineural and perivascular involvement. Special staining showed acid-fast bacilli. Since, she was found to have glucose-6-phosphate dehydrogenase deficiency, she was put on clofazimine 100 mg thrice weekly. She is tolerating clofazimine without any difficulty, though there is development of mild hyperpigmentation of the plaques. Occurrence of leprosy and SLE in the same patient is probably purely coincidental. Authors believe that his patient acquired earlier, but did not become clinically apparent until she received long-term corticosteroid therapy which suppressed her cell-mediated immunity and allowed leprosy to manifest.

K Pavithran

Pityriasis rosea—An immunological study, Hafez KA and Deyab Z: Internat J Dermatol, 1987: 26: 231-233.

Immunologic studies were carried out in 31 patients with pityriasis rosea. Sera and heparinised peripheral blood were obtained from patients and controls and submitted to following Blood count by standard methods, tests. determination of serum immunoglobulins G, A and M and serum complement factors C3 and C₄ measured by single radial immuno-diffusion. T-lymphocytes with surface receptors for sheep erythrocytes, detected with a rosette technique and B-lymphocytes by presence of fluorescence on their surface, when treated with anti-wholeimmunoglobulins conjugated with fluorescein. The response of peripheral blood mononuclear cells to phytohaemagglutinin was measured in the migration inhibition test. The migration index was calculated using the standard formula.

Epithelioid and polygonal cells in histoid

leprosy, Porichha D and Bhatia VN: Ind J

Tissue biopsy from skin lesions of 3 patients were taken and frozen sections submitted to direct immuno fluorescence for identification of 1gG, 1gA, 1gM, C₃ and fibrinogen. Normal levels of serum IgG and IgA but higher levels of 1gM were obtained. Serum C₃ values decreased while C₄ was normal. B lymphocytes showed no difference from controls but T cell counts were lower. Migration inhibition indices Direct immunowere higher than controls. fluorescence tests were negative in the three cases studied. Possible implication of a viral infection in the reported changes is discussed.

N Sasi

Topical clindamycin phosphate compared with oral tetracycline in the treatment of acne vulgaris, Katsmbas A, Towarky AA and Stratigos J: Brit J Dermatol, 1987; 116: 387-391.

Sixty patients, aged between 12-30 years, suffering from moderate acne vulgaris were enrolled in a 12-week double-blind randomized study to compare the efficacy of clindamycin phosphate 1% solution topically, with oral tetracycline 500 mg twice daily. Forty four patients, 22 in each group were available for evaluation at the end of the study. All patients experienced significant reduction in the number of pustules, papules and inflamed nodules and there were significant differences between the two groups. Both treatment regimens were well tolerated and no systemic side effects were reported. Clindamycin treated group gave a significantly higher improvement rating than the tetracycline group. This study demonstrates that clindamycin phosphate 1% topical solution is a safe and effective alternative to oral tetracycline for the treatment of moderate acne vulgaris.

leprosy, histopathologically characterized by presence of large number of spindle shaped

Leprosy, 1987; 59: 191-193.

Histoid leprosy is a subtype of lepromatous histiocytes. Salient histopathological features of 36 cases of histoid leprosy are described. The granuloma in all the cases were circumscribed and hypercellular and consisted of mostly spindle shaped cells disposed in compact whorls and interlacing bundles. Well-formed pseudocapsule could be seen in only 6 cases. Epidermis showed pressure atrophy due to the underlying granuloma. Subepidermal clear zone could be seen in all cases. Small but prominent circumscribed islets of epithelioid cells were amidst the spindle cell mass in 5 cases. Such a feature has been described by Wade also who called this as tuberculoid contamination. Here the authors found such cells in the deeper aspect of dermal granuloma whereas Wade found them mostly in the subcutaneous nodule. Epithelioid cells with absence of bacilli suggest some amount of cell mediated immunity in these foci or a possible development of histoid features in a pre-existing borderline leprosy. The actual nature of epithelioid cells in these lesions needs to be explored. Presence of polygonal cells which indicate early histoid lesion, were observed in 12 cases. Such an admixture of spindle cells and polygonal cells has previously been designated as mixed lepromatous leprosy.

K Pavithran

More about clofazimine—3 years experience and review of literature, Kumar B, Kaur S, Kaur I et al: Ind J Leprosy, 1987; 59:63-74.

Clofazimine is one of the drugs used in the treatment of leprosy. Side effects of this drug were observed in 514 patients with leprosy and 26 patients with vitiligo who had been getting treatment with clofazimine for periods ranging

N Sasi

from 3 months to 2 years. Dose of clofazimine varied from 100 mg alternate days to 300 mg daily.

Commonest side effect noted was skin pigmentation of reddish brown and black hue (77.8%). The pigmentation was noticed 3-4 weeks after starting therapy and it became marked after 6 weeks. The pigmentation was lighter in flexures but prominent over exposed areas. In an equal number of patients ichthyotic changes were noted on the peripheral parts of the symptoms occurred body. Gastro-intestinal in only 20 (0.04%) patients. Of 21 patients in whom absorption parameters and jejunal biopsies were studied, Schilling's and d-xylose tests remained normal. Jejunal mucosa was normal in all biopsies, clofazimine crystals were seen in lamina propria of one patient. No correlation was found between the abnormality in mucosal pattern, crystal deposition, absorption parameters or symptomatology and the dose of clofazimine. Other minor side effects noted were reddish colouration of sweat, urine and tears. No abnormality in the EKG or serum or biochemistry occurred even after prolonged therapy. This study shows that clofazimine is reasonably free from serious side effects in the usual dosage and even in high doses for shorter periods.

K Pavithran

Congenital cytomegalovirus infection, Best JM: Brit Med J, 1987; 3:469-470.

Congenital cytomegalovirus infection and resultant congenital anomalies in the new borns remains a global problem. Though congenital rubella has been partly controlled with rubella vaccine, there is no effective vaccine to prevent cytomegalovirus infection. Recent advances in radio-immuno-assay technique has made us to detect the infection in new born infants. It is not clear whether severe handicaps are more

likely to result from maternal infection in early or late pregnancy. A recent study showed that although congenital infection may occur throughout pregnancy, permanent damage is more likely to result if the mother is infected in the first half of pregnancy. Other studies have shown that serious defects may result from infection in both early and late pregnancy. Congenital infection may result from either primary or recurrent infection in the mother. Serious handicaps are more likely after primary rather than recurrent infection. But neurological damage and bilateral hearing loss may be seen in children born to mothers with recurrent CMV infection.

Perhaps women who transmit the virus to the foetus have defective immunological responses and are unable to limit replication of CMV. Author concludes by stating that we must wait to see whether genetically engineered vaccines will be able to stimulate the necessary immune responses in such women.

K Pavithran

Generalised vitiligo following Sezary syndrome, Alcalay J, David M, Shohat B et al: Brit J Dermatol, 1987; 116:851-856.

Even though vitiligo has clinical association with auto-immune diseases, it is rarely seen in association with internal malignancies. Here a case of generalised vitiligo following Sezary syndrome is reported. Initially, the patient was admitted with erythroderma and they couldn't find any cause for the same. Patient was followed up by them but no abnormalities were detected. Two and a half years later depigmentation developed all over the body. Then the clinical, histological and laboratory findings were compatible with Sezary syndrome. This patient had persistent bacteraemia also without any infective foci. Patient responded dramati-

cally with chemotherapy. Authors suggest an immune mechanism as the cause of vitiligo.

K Sobhanakumari

House dust mite allergy and atopic eczema, a case report, Barnetson St C, Maefaslane HAF and Bentar EC: Brit J Dermatol, 1987; 116: 857-860.

Authors reported a female patient with history of atopic eczema since infancy. She had asthma and angio-oedema following ingestion of certain foods. Initially when she was seen, the eczema was active and concentrations of total serum IgE and antibody to house dust mite were very high. Subsequently during examination the antibody to house dust mite decreased during subsidence of disease and increased during worsening of disease. serum IgE concentration didn't show much Those results suggest that large variation. amounts of house dust mite antigen may be scratched into the skin during clinical exacerbation of eczema, this in turn aggravate the disease because of hypersensitivity to mite.

K Sobhanakumari

Dysplastic nevi and malignant melanoma, Goldsmith MF: JAMA, 1987; 257: 894-895.

There is a definite relationship between dysplastic nevi and malignant melanoma. These atypical pigmented moles are found to be the precursors of malignant melanoma. Rigel and his colleagues have developed a method of ranking the risk of developing melanoma for persons who have many dysplastic nevi. They studied 452 persons who came with unusual pigmented lesions. Those who were found to have malignant melanoma were not included. Patients were grouped into 5 groups. Type A had the lowest risk of developing malignancy and Type D₂ had the highest. Type A has no

personal or family history of melanoma and moles do not run in family (sporadic dysplastic nevus syndrome). Type B has no personal or family history of melanoma and moles do run in family. Type C has personal or family history of melanoma and moles do not run in family. Type D has personal or family history of melanoma and moles run in family. In type D₂ 2 or more family members have had melanoma and moles run in family. A patient who already has a melanoma runs a 4% chance of developing it again. Early detection of malignant melanoma is important. By light microscopy it is sometimes difficult to differentiate between dysplastic nevi and malignant melanoma. So a human derived monoclonal antibody directed against a cytoplasmic antigen has been tried. Even some rapidly growing benign nevi could pick up the antibody. But the two types showed some difference in staining. Persons with moles of size more than 6 mm are at a higher risk. So the conclusion is that careful follow up of patients with dysplastic nevi is important to reduce the risk of advanced melanoma.

K Anitha

Nasal carriage of staphylococci and streptococci, Barth JH: Internat J, Dermatol, 1987; 26: 24-26.

A thorough knowledge of the pattern of nasal carriage of Staphylococcus aureus and Streptococcus haemolyticus is important in dealing with recalcitrant skin sepsis. Nasal carriage is acquired faster when topical antibiotics are used. It is increased in the presence of specific staphylococcal nasal disease. Skin infections associated with nasal carriage of the same phage type are predominantly on the head and neck. When the axilla and legs are affected there is no nasal carriage or a different phage type. S. aureus can always be isolated from the nose in systemic infections. Persistent carriers are

colonized by an individual phage type whereas intermittent carriers have different phage types on each successive examination. The degree of binding of S. aureus to nasal mucosa is increased in subjects with atopic eczema. The most important mechanism of staphylococcal transfer is by manual dissemination, and not by breathing, talking, coughing or Topical neomycin reduces nasal carriage. Systemic antibiotic therapy does not eradicate it. The main value of nasal therapy is to control an epidemic in an institution. Treatment of intermittent carrier states is useful for a patient with recurrent staphylococcal boils or atopic eczema who is infected by himself or a close contact. The use of nasal antibiotics is associated with the carriage of resistant strains.

Haemolytic streptococci are uncommon in the nose. Nasal carriage is increased after tonsillectomy. Nasal carriers are important in spreading streptococcal infection. They are eradicated by systemic antibiotics. Treatment is necessary for limiting the spread of infection to the patient's contacts.

Joyce Mathew

Pelvic inflammatory diseases—Its pathogenesis, diagnosis and treatment, King AL: Postgraduate Medicine, 1987; 81: 105-114.

The major pathogens responsible for PID are Neisseria gonorrhoeae, Chlamydia trachomatis, acrobic bacteria like Gardnerella vaginalis, Gram negative Enterobacteriaceae and Group B streptococci, anaerobic bacteria like peptococcus peptostreptococcus, bacteroides and mycoplasma. There are both major and minor diagnostic criteria. The major criteria include lower abdominal pain and tenderness, tenderness on cervical motion, adnexial tenderness and

rebound tenderness of lower abdomen. In acute PID the clinical picture is attributed to the ascending spread of microorganisms from the lower to the upper genital tract. The various regimes recommended by Centers for Disease Control for treatment include drugs that are effective against all these causative agents. The treatment of unruptured tubo-ovarian mass is either prompt surgery or conservative line of treatment. In all cases the preservation of the reproductive capacity should be the main aim of the treatment.

Molykutty Francis

Necrotising dermatitis in patients receiving cancer chemotherapy, Driezen S, McCredie KB, Bodey GP et al: Postgraduate Medicine, 1987; 81: 263-271.

The necrotising dermatitis can be microbial, occlusive, toxic, idiopathic or neoplastic in The microbial group include Pseudoorigin. monas aeruginosa, Staphylococcus aureus, Clostridium perfringes, Phycomycetes, Aspergillus etc. The occlusive causes are due to underlying diabetes mellitus, non-bacterial thrombotic endocarditis or thromboangitis obliterans. The toxic agents include chemotherapeutic drugs, anticoagulant drugs and spider venom. congenital or acquired deficiency of antithrombotic protein C resulting in excess fibrin production and subsequent thrombo-embolism is the proposed mechanism for anti-coagulant induced dermal gangrene. This has a predilection for areas of abundant subcutaneous fat. Pyoderma gangrenosum is one of the idiopathic causes. The failure to maintain adequate blood supply in all parts of the tumour can also lead to necrotising dermatitis.

Molykutty Francis

Porokeratosis: Immunosuppression and exposure to sunlight, Bencini PL, Crosti C and Sala F: Brit J Dermatol, 1987; 116: 113-116.

Porokeratosis is characterised by one or many atrophic, keratotic patches surrounded by a distinct, raised ridge showing the coronoid lamella as a characteristic histopathologic finding. Four cases of histopathologically proved disseminated superficial actinic porokeratosis are reported. All the four cases were on immunosuppressive drugs for renal transplant in 3 cases and one for chronic active hepatitis. There appears to be a clear relationship between the immunosuppressive treatment and the development of epithelial pathology. Immunosuppression either favours or triggers porokeratosis. The fact that all the four patients had actinic type of porokeratosis with associated actinic keratosis and papilloma virus infection (verruca and condyloma) suggests a possible relation between exposure to sunlight and viral infection. The synergistic effect of exposure to sunlight and immunosuppressive therapy might influence the development of a clone of mutant cells or might favour or cause viral activation. Further study of the relationship of porokeratosis to immunosuppression should lead to better understanding of its pathogenesis.

N Surendran Pillai

Surgical treatment of speckled skin caused by dyschromatosis symmetrica hereditaria, Taki T, Kozuka S, Izawa Y et al : J Dermatol, 1986; 13: 471-473.

Dyschromatosis symmetrica hereditaria is characterized by mottled areas of depigmentation. There is no effective treatment for this condition. Here the authors took split skin autografts from the abdomen to replace speckled skin of the dorsum of the hands, caused by dyschromatosis symmetrica hereditaria in a 20-year-old man. Speckled skin was removed with a hand dermatome, mostly to a thickness

of 0.1 to 0.2 mm, in order to get a uniform bleeding surface. The raw surfaces were covered with skin grafts taken from the lower abdomen. Normal skin colour appeared later in the recipient area and there was no recurrence of depigmentation in the treated area. The authors suggest that although the cause of depigmentation in dyschromatosis symmetrica hereditaria is unknown, the recovery of normal colour in the areas of skin grafts suggests that either the cause of depigmentation was removed and/or the factors which repair the depigmentation were introduced by the skin grafting.

K Pavithran

Porokeratotic eccrine ostial and dermal duct nevus, Driban NE and Cavicchia JC: J Cutan Pathol, 1987; 14: 118-121.

Nevus comedonicus has been reported in palm or sole. But their occurrence in these sites where normally there are no pilosebaceous structures is quite interesting. Hence, the authors studied the sections taken from a clinically diagnosed nevus comedonicus lesion of the palm. But light microscopy and electron microscopy showed that the structures in this nevus were related to eccrine sweat gland ducts. The ducts were made up of 2 and 3 rows of epithelial cells with tonofilaments and numerous desmosomes, with characteristic microvilli in the lumen covered with an amorphous substance. In connection with these structures, the lesion contained typical granules of the eccrine glands. Light microscopy showed a large expansion of a probable ductus sudori-The authors believe that from the ferous. clinical and microscopic characteristics, this case should be included under the designation porokeratotic eccrine ostial and dermal duct nevus.

Generalised pustular psoriasis (Von Zumbusch) responding to cyclosporin A, Meinardi MMHM, Westerhof W and Bos JD: Brit J Dermatol, 1987; 116: 269-270.

Generalised pustular psoriasis is a serious form of psoriasis which may occasionally become lethal. Etretinate is effective in this condition but this cannot be used in patients with liver damage. A case of generalised pustular psoriasis following withdrawal of methotrexate he had been receiving for severe psoriasis and arthralgia, is reported. Methotrexate was withdrawn because of liver damage. Cyclosporin A was used in this patient because hepatotoxic effects of cyclosporin A have not been reported. In this patient cyclosporin A produced a comparable dramatic improvement in about the same time as that reported with etretinate. There were no side effects except for a slight rise in plasma creatinine level, which was controlled by lowering the dosage of cyclosporin A. Cyclosporin A thus represents a valuable contribution to the treatment of severe and recalcitrant forms of psoriasis.

N Surendran Pillai

Transmission of leprosy (editorial), Job CK: Ind J Leprosy, 1987; 59: 1-8.

Recent advances in the study of the source and transmission of leprosy and their implications are briefly discussed. Naturally acquired disease caused by *M. leprae* exists in wild armadillos, a mangabey monkey and a chimpanzee. According to a recent survey, 2% of the armadillos in Louisiana have lepromatous leprosy. A sick armadillo can harbour 10¹² *M. leprae* in its body. They shed bacilli from ulcers in their skin, nose and mouth just like humans. These armadillos are often killed by automobiles on the road and their carcasses are eaten by other wild animals and birds. Thus, several trillions of bacilli are disseminated into the soil and the environment. *M. leprae* can survive in the

environment for upto 9 days and in the moist soil even upto 46 days. This introduces a very important factor in the transmission of the disease.

Experimental studies show that the two common routes of entry of *M. leprae* are the skin and the nasal mucosa and that the organisms multiply at the site of entry and produce a local lesion before the disease is disseminated. It has been postulated that in humans when *M. leprae* enter through the nasal mucosa lepromatous disease results and that skin entry of *M. leprae* causes tuberculoid disease. But in experimental leprosy both skin and nasal entry of *M. leprae* have resulted in lepromatous or borderline disease. Tuberculoid leprosy has so far not been produced in animal models.

Placental transmission of leprosy is a possibility in human beings. *M. leprae* have been found to infiltrate and multiply in uterine muscle cells, endometrial tissue and placental villi of pregnant armadillos.

K Pavithran

Nerve involvement in Hansen's disease, early diagnosis and treatment, Pfaltzgraff RE: The Star, 1987; 46: 1-3.

The most important aspect of the management of leprosy is to control nerve involvement, as almost all of the deforming and disabling aspects of leprosy are directly due to what happens to nerves. If nerve involvement is controlled, while continuing specific therapy, leprosy becomes an insignificant infection. Here, the author gives an outline of the clinical aspects of neuritis and its management. Most leprosy workers think of neuritis only when there is unquestioned pain, tenderness or loss of function. But there are also patients with mild or so called silent neuritis that is painless. Early nerve involvement should be started at the earliest. It is

always best to give the patient the benefit of the doubt. Progress of motor involvement can be followed by voluntary muscle testing (VMT).

One of the steroid regimens recommended is prednisolone 25 mg daily in a single dose, reduced by 5 mg per day on a monthly basis. Another alternative is clofazimine therapy. Clofazimine, in addition to its antimycobacterial action has profound anti-inflammatory and immunosuppressive effects.

The corticosteroid gives the most rapid response and begins to give relief within 6 hours. For thalidomide it takes 24 to 48 hours and with the clofazimine it is only evident after 6 to 8 days. So when neuritis is severe corticosteroid is the drug of choice. Anti-leprosy drugs should be continued. Clofazimine and thalidomide are used to wean corticosteroid.

K Pavithran