PSEUDOXANTHOMA ELASTICUM WITH ELASTOSIS PERFORANS SERPIGINOSA

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Summary

Three cases of pseudoxanthoma elasticum are reported. Two of them had associated elastosis perforans serpiginosa on their neck and angioid streaks in the eyes. In addition to the angioid streaks, drusen (colloid bodies) also were noted in the fundus near the optic disc in one case. The third patient had associated psoriatic lesions also.

KEY WORDS: Pseudoxanthoma elasticum, Elastosis perforans serpiginosa, perforating elastoma, Miescher's elastoma, Drusen.

Pseudoxanthoma elasticum is a recessively inherited disorder of connective tissue characterised by yellowish areas of elastotic degeneration in the skin rendering it loose, inelastic and redundant especially at flexion folds. About 24 cases have been, so far, reported from India. The disease affects multiple organs and systems of the body. The characteristic eye change is the angioid streaks which results from breaks in the elastic membrane of Bruch. The association of skin lesions of pseudoxanthoma elasticum with angioid streaks in the eyes has been called Gronblad-Strandberg syndrome. The angioid streaks develop in 50 to 75% cases of pseudoxanthoma elasticum. Yellowish white dots called drusen or colloid bodies also may rarely be seen near the optic disc[1]. Other vascular manifestations include epistaxis, hypertension, diminution or absence of peripheral arterial pulsations and bleeding from the gastro intestinal tract. Various neurological and cardiac complications also may sometimes occur.

A variety of other cutaneous diseases have been reported in association with pseudoxanthoma elasticum. These include Ehlers-Danlos syndrome, purpura, calcinosis cutis, leprosy[2] and elastosis perforans serpiginosa[3,4]. Elastosis perforans serpiginosa first described by Lutz in 1953, is a rare dermatosis. It is also called Miescher's elastoma or perforating elastoma. It is characterised by annular or serpiginous cord like lesion formed by coalescence of multiple firm papules topped by

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keratin plugs. Shedding of these plugs results in multiple tiny depressions in the lesion. The usual sites of predilection are the back and sides of the neck, chin, knees and elbows. The lesions persist for years and finally undergo spontaneous regression. The diseases reported in association with the perforating elastomas include mongolism, Ehlers-Danlos syndrome, Rothmund-Thomson syndrome, Marfan syndrome, osteogenesis imperfecta, acrogeria and pseudoxanthoma elasticum[3,4,5,6]. Disseminated lesions of elastosis perforans serpiginosa have also been reported[7,8].

The histology of pseudoxanthoma elasticum is quite typical. It is characterised by the accumulation of swollen, fragmented and irregularly clumped basophilic fibres in the middle and lower third of dermis. These fibres take up stains for elastic tissue. The basic histologic change in elastosis perforans serpiginosa is a focal increase in both the amount and the size of elastic fibres in the upper dermis and papillae. In H & E stained sections amorphous materials are seen filling the papillae along with the inflammatory cells. Verhoeff's staining for elastic tissue shows great increase in the amount and size of elastic fibres at these sites. The acanthotic epidermis shows narrow channels through which the altered elastotic material is extruded. The keratin plugged channels contain in their lower portion, thick elastic fibres. These also contain basophilic necrotic nuclei of the inflammatory cells[9]. Here, we are reporting 3 patiens with pseudoxanthoma elasticum. Two of them had associated angioid streaks in the eyes and perforating elastomas on the neck. The third patient had associated psoriasis.

Case Report

Case No. 1 – A 48 years old man was seen in the outpatient section of Medical College Hospital, Kottayam in July '82 with an asymptomatic annular skin lesion on the right side of his neck. It had started one year before as a small papule which gradually increased in size. There was no history of intermittent claudication, chest pain or malena. There was no history of consanguinity and none in the patient's family had similar skin lesions. General physical examination did not reveal any abnormality. Blood pressure and peripheral arterial pulsations were normal. Dermatological examination revealed a well circumscribed plaque measuring 3 x 2 cm on the right side of the neck formed by coalescence of multiple, firm papules capped by keratin plugs, arranged in an annular pattern with central depression containing non adherent, dry, blackish keratotic material (Fig. 1). Skin on the neck, axillae and groins were soft, yellowish, inelastic and

Fig. 1
Elastosis perforans serpiginosa. Note central depression – Case No. 1.

studded with tiny yellowish papules arranged in a linear pattern. The axillary skin was lax and redundant (Fig. 2). The mucosa was normal. Ophthalmoscopy revealed angioid streaks radiating from optic discs of both eyes. The vision was normal. Other systems were clinically normal.

Fig. 2 Elastosis perforans serpiginosa on the right of side neck. Note loose and redundant skin of axilla due to PXE. Case No. 1.

Investigations

Routine blood, urine and motion examination were normal. E.C G., X-ray of chest and limbs, serum calcium and serum cholesterol were all within normal limits Benzidine test was negative for occult blood in motion. The skin lesion on the neck was excised which on histology (H & E staining) showed amorphous materials in the papillae and upper dermis. This material stained deep with Verhoeff's stain. An inflammatory infiltrate consisting of lymphocytes, histiocytes and epithelioid cells were seen in the upper

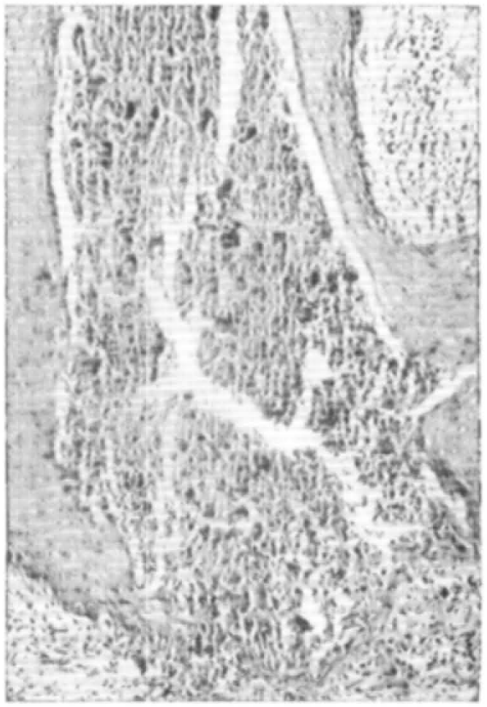

Fig. 3 Elastosis perforans serpiginosa. Histology. Note channels in acanthotic epidermis through which basophilic necrotic materials, and thick elastic fibres are extruded.

dermis around the elastotic materials. The acanthotic epidermis showed narrow channels in which basophilic necrotic materials, brightly eosinophilic thick elastic fibres and keratotic plugs were noted (Fig. 3). In the mid and deeper dermis there were many swollen, fragmented and irregularly clumped basophilic fibres which were found to be deeply stained by Verhoeff's stain.

Case No. 2 – A 21 years old male student was seen in October '82 with an asymptomatic annular lesion on the front of his neck since 9 months. There was no history of consanguinity and none in his family had similar skin lesions. General physical examination, blood pressure and peripheral arterial pulsations were within normal limits.

There was an annular lesion, 3 x 5 cm in size on the front of the neck (Fig. 4). At the margin of the lesion there were multiple, closely set, dry keratotic papules, some of them showing central depressions. The centre of the lesion was hyperpigmented, atrophic and showed multiple tiny depressions filled with keratotic material. Coarse black terminal hairs were seen in the lesion (Patient was not shaving this area for 3 months). Skin on the neck and axillae was soft, yellowish, lax and studded with numerous tiny, yellow papules arranged in a linear and reticulate pattern. Vision in both eyes was normal. Ophthalmoscopy showed angioid streaks radiating from the optic disc of right eye. In addition, there were yellowish white dots (drusen, colloid bodies) near the disc. All other systems were clinically normal. Investigations done as for case No. 1, did not reveal any abnormality. Biopsy of the skin lesion showed changes similar to that described for case No. 1.

Case No. 3 - A 35 years old housewife attended the out patient section in November '82 with generalised scaly lesions of 6 years' duration. There was no history of consanguinity and none in her family had any skin disease. Routine general physical examination, blood pressure and peripheral pulsations were normal. There were multiple, papulosquamous lesions on the trunk, scalp, neck, elbows and knees. The scales were dry, loose and micaceous. Auspitz sign was positive. The skin of the neck (Fig. 5), axillae and groins were soft, yellowish, lax and redundant. Nails showed pits on their surface. Vision was normal in both eyes. Ocular fundii were normal. Other systems were clinically normal. Investigations done as for cases 1 & 2 did not show any abnormality. Histological study of the papulosquamous lesion revealed all the features of psoriasis. Biopsy of skin from neck showed swollen, fragmented and irregularly clumped basophilic fibres in the middle and lower third of the dermis, deeply stained by Verhoeff's stain.

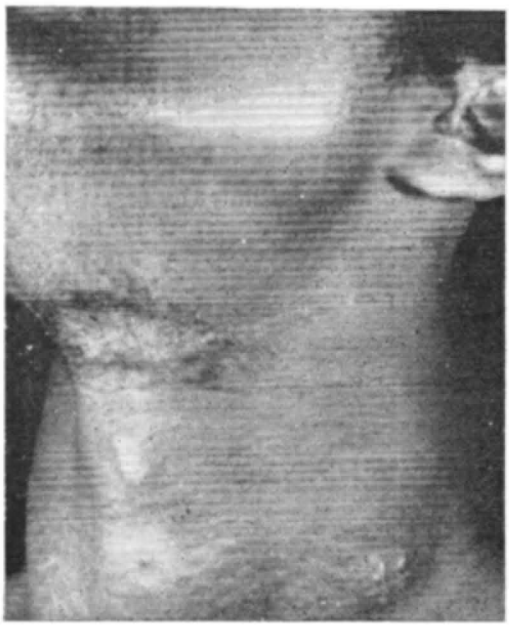

Fig. 4

Case No. 2: Note the perforating elastoma on the front of the neck. Hair growth in the lesion is unaffected.

Fig. 5
Case No. 3. Note psoriatic lesions on the neck in a patient with PXE.

Discussion

Over 328 cases of pseudoxanthoma elasticum have been reported in the world literature. Among the reported cases there have been more females than males with the sex ratio about 1 : 2[10]. In the present series there were two males and one female. In a review of 20 reported cases of pseudoxanthoma elasticum from India, Seghal et al[11] found that the males outnumbered the females. Cosmetic embarrassment due to the perforating elastomas on the neck made the first two cases attend our department. They were completely unaware of the pseudoxanthoma elasticum they had on other parts of the body. Two of our cases had associated angioid streaks in the eyes and elastosis perforans serpiginosa of the neck. One of them showed characteristic drusen near the optic disc. The coexistence of psoriasis in the third case seems to be purely coincidental.

Cases of elastosis perforans serpiginosa are only rarely reported from India[3,4]. The reported cases had associated pseudoxanthoma elasticum also. It was first described by Lutz in 1953. Many cases are associated with other congenital ectodermal or mesodermal abnormalities, thus reflecting a generalised connective tissue abnormality[12]. Cases unassociated with other connective tissue diseases have also been reported[8]. The primary event in elastosis perforans serpiginosa is the dermal elastoma which irritates and provokes a cellular response that ultimately leads to extrusion of the altered connective tissue through the epidermal channels. Mehergan suggests that a mechanism of transepidermal elimination operates here[18]. The factors determining the formation of elastoma are unknown. Although the invaginated epidermis surrounding the plugs may suggest pilosebaceous follicles and although the winding channels through which the altered elastic tissue is extruded may suggest intraepidermal sweat ducts, it seems that as a rule, the extrusion occurs without any relationship to

these structures[9]. The hairgrowth in the lesion usually remains unaffected[12]. This was quite evident in case No. 2 which showed thick coarse hairs on the lesion.

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ACTINOMADURA PELLETIERI CAUSING WHITE GRAIN MYCETOMA
- A Case Report from Madras

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Summary

Actinomadura pelletieri causing white grain mycetoma pedis in a 32 year old farmer is reported. The right foot was swollen, firm, painless, non tender and had a number of small nodules breaking down to form sinuses. The seropurulent discharge contained dirty white grains measuring 0.5 to 1 mm, composed of unfragmented filaments of bacterial width. Histologic examination of the biopsy material revealed the characteristic granule of *A. pelletieri* and culture yielded typical coral red colonies.

KEY WORDS : Actinomadura pelletieri, white grain mycetoma.

Mycetomas caused by *Actinomadura pelletieri* are known as red grain mycetomas because of the characteristic colour of the grains in the discharge. But Pardo-Castello and Trespalacios[1] had reported two cases of *A. pelletieri* mycetomas with non-red grains from Cuba. Bergeron et al[2] had even doubted the identity of those Cuban strains on account of the colour of the grains. However, Koshi et al[3] had reported two cases of *A. pelletieri* mycetomas confirmed by histopathology and culture from Vellore and in one of them, the granules were cream coloured and not red.

In this paper we present a case of mycetoma caused by *A. pelletieri* with dirty white grains instead of the usual red grains. The morphological appearance of the grains in tissue sections of the biopsy material was characteristic of *A. pelletieri* and typical coral red colonies were obtained in culture. To our knowledge, this is the second case of white grain mycetoma due to *A. pelletieri* from India.

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Case Report

A 32 year old farmer from Chengleput district attended the Surgical outpatient Department of the Government General Hospital, Madras, for a painless swelling of the right foot. The lesion had started two

years earlier as a small nodule on the dorsal aspect of the foot, that softened, burst open and discharged seropurulent material. Patient could not remember any significant injury to the affected part prior to the onset of the disease. Successively, similar nodules developed on the foot, ulcerating and discharging seropurulent material.

Clinical examination disclosed a firm, painless, non tender swelling on the dorsal aspect of the right foot with a number of small nodules breaking down to form sinuses. Inguinal lymph nodes were enlarged on both sides. X-ray showed soft tissue swelling and demineralization of all the bones.

Histopathological study

Histological sections of the biopsy material showed the presence of granules conforming to the description of *A. pelletieri*. In haematoxylin-eosin stained sections, small granules with smooth or denticulate edge, without clubs and with dense, homogenous matrix were seen in the middle of abscess cavities (Fig. 1). The constituent filaments were not seen very clearly in Gram stained sections. The granules were not acid-fast by Kinyoun's acid-fast method.

Mycological study

The seropurulent material as well as the biopsy specimen had numerous, small, soft, dirty white grains, about 0.5 to 1 mm in size. When crushed and examined in a drop of KoH, these were shown to be composed of filaments of bacterial width. The smears showed thin, delicate, long branching, Gram-positive filaments which were not acid-fast.

The granules were repeatedly washed in sterile saline and inoculated on two sets of Sabouraud's dextrose agar slants and incubated at 26°C and 37°C. Growth was very slow, appearing in 4 weeks' time. The colonies were irregularly folded and coral red in colour. Growth was better at 37°C. Microscopic examination revealed Gram positive, branched, unfragmented filaments which were not acid-fast. The organism hydrolysed casein, decomposed tyrosine crystals, and liquified gelatin, but failed to decompose xanthine or to utilize paraffin. Urease was negative. Small puff-ball like,

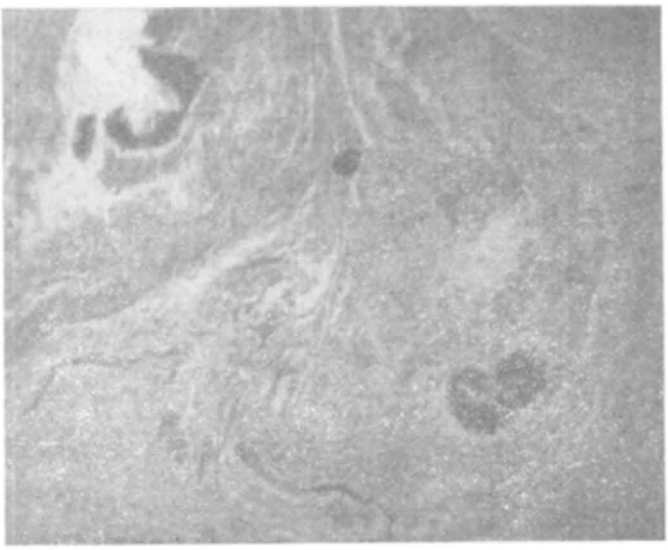

Fig. 1

Biopsy specimen of lesion showing *Actinomadura pelletieri* grains in abscess (Haematoxylin eosin × 100).

light red colonies developed in Sabouraud's dextrose broth. Acid was produced with glucose and not with lactose, arabinose, xylose, glactose and mannitol.

The isolate was identified as *A. pelletieri*.

Discussion

Mycetoma is characterized by localized swellings with multiple sinuses discharging granules or grain. Depending upon the etiologic agent, the grains are light coloured (white, yellow, cream), pink or red or dark-coloured (brown or black). The colour of the grain is useful in giving a clue to the identity of the organism; but several species may produce similar grains and the same agent may produce grains with different colours occasionally as seen in the present case and the one reported from Vellore[3].

The morphologic appearance of *A. pelletieri* grains in tissue sections is so characteristic that a specific diagnosis of *A. pelletieri* mycetomas is possible by mere histopathological examination alone[4]. In the present case, the organism is isolated in pure culture in addition. The dirty white grains, on culture yielded typical coral red, folded colonies. We had islaoted *A. pelletieri* from 2 cases of mycetoma from Madras earlier and both the patients had the characteristic red grains[5].

The exact reason for the change in the usual colour is not known. Bacterial contamination may account for a change in the colour of the grains[6], but the organism was isolated in pure culture in the present case as well as the one from Vellore[3].

The patient was given a preliminary course of tetracycline and excision of the mycetomatous tissue was carried out.

Since this is the second case of white grain mycetoma due to *A. pelletieri* from India, we are reporting the case to stress that further investigations such as histopathology and culture are always necessary for ascribing a specific cause to a case of mycetoma.

Acknowledgement

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VITILIGO IN A PIGMENTED NEVUS

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Summary

A case of vitiligo developing within a patch of pigmented nevus - an early Becker's melanosis - is being reported. The possible mechanisms of development of depigmentation are discussed.

KEY WORDS: Vitiligo, pigmentary nevus, Becker's melanosis, Self destruct theory.

Vitiligo is a common disease encountered in Dermatological practice. Though the disease has been known and studied literally from ages, its aetiology still remains obscure. Recently a number of postulations have been put forward to explain the loss of melanin from the skin, in this disease. It is interesting to note that vitiligo has occasionally been reported in association with various types of benign and malignant lesions of the melanocyte system. 'Halo nevus' is a well known entity, where leucoderma develops around a centrally placed nevus cell nevus. It has also been reported to develop around and within the skin lesions of malignant melanoma of the skin[1-2]. Kapur[3] in 1976 reported a case of vitiligo in association with congenital pigmented nevus. His patient developed vitiligo over various parts of the body including a few areas of the nevus. An almost similar case was observed by Bedi, in which depigmented macules appeared within a patch of Becker's melanosis[4]. Here, we are reporting a case in which vitiliginous macules appeared within a patch of 'pigmentary nevus' - most probably an early Becker's melanosis- on the face.

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Case Report

A sixteen years old male student attended the Dermatology section of Medical College, Kottayam for asymptomatic depigmented macules on his face which has been present for one month. There was no history of preceding trauma or inflammation at that site. He had a large hyperpigmented patch extending from the chin to the neck for two years. General physical and systemic examination did not reveal any abnormality.

Dermatological examination revealed a large hyperpigmented patch with a linear well defined upper border, extending from the middle of chin to the front and left side of neck. Close examination showed excessive growth of thin short lanugo hairs and 'goose flesh' like prominences of follicular orifices in the patch. Two small well defined, depigmented, non anaesthetic oval macules of 0.5×0.3 cm. size were seen in the hyperpigmented patch close to its upper border on the face

Fig. 1
Note two depigmented macules within a patch of pigmented nevus on the face.

(Fig. 1). There was no other skin or mucous membrane lesions.

Routine laboratory investigations were found to be normal. Histological study of the hyperpigmented patch revealed mild acanthosis and hypermelanosis of the basal layer. A few melanophages were seen in the upper dermis. The hair structures appeared normal.

Discussion

The pigmentary nevus of the face and neck in the present case is most likely to be an early Becker's melanosis because of its onset in the second decade of life, unilateral distribution and presence of mild hypertrichosis in the melanotic patch. In Becker's melanosis the lesion usually starts as a patch of pigmented skin which remains stationary for some time with subsequent appearance of hypertrichosis years after its onset[4]. Though, classically seen in the shoulder region it may rarely affect other areas[5]. The exact mechanism of development of vitiligo within these patches of pigmented nevi is not clear. One view put forward is that some form of injury to these nevi containing abundant melanin releases the antigenic material to the circulation and thereafter the body no longer considers melanin as 'self' but tries to destroy it by two basic immunologic mechanisms – humoral and cell mediated. This is autoimmunity[6]. Recently depigmentation has been noted at sites of resolution of pigmented tumours in animal models – Sinclair swine. The more recently put forward 'self destruct' hypothesis proposes that an intermediate or metabolite in melanin synthesis causes disappearance of melanocytes. Lerner[7] suggested that melanocytes have an inherent protective mechanism which leads to successful elimination of melanin precursors that are synthesised by melanocytes but toxic to them. Disruption of this labile destructive process causes vitiligo. Support for this hypothesis arises from the observation that the skin areas usually affected by vitiligo are those darkly pigmented. The development of vitiligo in Becker's melanosis – a highly pigmented area – in the present case, also favours the 'self destruct' hypothesis.

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Short Communication

Fixed drug eruption, first described by Brocq in 1894 is a type of localised cutaneous hyper sensitivity reaction following oral or parenteral administration of a drug. It is clinically characterised by recurrence of eruption at the previously affected site in each time of re-exposure to the drug, ultimately resulting in residual pigmentation. It is observed in dermatological practice that some of these cases neither give history of drug intake nor show positive results on provocation tests. Though food substances like tomatoe, egg white, etc have been mentioned as rare etiologic agents for fixed eruption, we had never come across such a case in the past. Recently a girl of 18 years came to our department with a hyperpigmented patch on the upper lip present for years with history of periodic exacerbations (characterised by itching and ring of erythema) on intake of a vegetable plant called SAUROPUS androgynus (Family Euphorbiacease) locally known as Augusti cheera; ceylon cheera - a type of spinach. The patient was asked to take a curry prepared out of this and report. Twelve hours after this patient returned to the clinic with erythema and swelling around the pigmented area on the lip. After three weeks, patient was again asked to eat boiled leaves of the same plant without adding any other ingredient to it. This time also patient reported with reaction similar to what was earlier observed.

Note the FDE of the upper lip due to the leaves of Sauropus androgynu

We favour the term 'fixed eruption' rather than 'fixed drug eruption' for these types of cutaneous hypersensitivity because in some cases a history of intake of "drug" is lacking. It is possible that some form of food substance may, at least in a minority of cases, be responsible for the eruption. A detailed dietetic history of patients is indicated in all cases of fixed eruption where there is no history of drug intake.

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Announcement...

INTERNATIONAL SOCIETY OF TROPICAL DERMATOLOGY

The V World Congress of Tropical Dermatology will be held in Mexico, October 16-20, 1984, under the direction of Dr. Ramon Ruiz Maldonado, executive president of the Congress. There will be special one day intensive courses in tropical pediatric dermatology, contact dermatitis and occupational dermatoses, and practical dermatologic cosmetology. Special lectures and symposia under the direction of a select group of experts known world-wide are being organized. There will also be clinicopathologic mini case sessions as well as poster exhibits. Abstracts for consideration for presentation should be typed and double spaced in duplicate. We would like a 300 word abstract for free communication presentations (10 minutes), and for clinicopathologic mini cases (5 minutes), a 50 word abstract. The deadline for submission of abstracts for papers and mini cases is July 31, 1984. Abstracts should be sent to the Congress Secretary, P.O. Box 101-16 Mexico 04530, D.F.

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1. Dr. Salil Kumar Panja,
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2. Dr. S. Chakraborty,
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3. Dr. B. N. Banerjee,
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4. Dr. Ranjit Kumar Panja,
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5. Dr. A. K. Chakraborty,
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6. Dr. M. A. Wali,
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