

COLLOID BODIES IN DERMATOSES OTHER THAN LICHEN PLANUS

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Abstract. In a previous study on lichen planus the morphology and significance of the colloid bodies were discussed (13). In the present study a description is given of 30 dermatoses with colloid bodies, observed among unselected patients. The colloid bodies develop as a result of damage to the epithelium caused by circulatory disorders with subsequent hypo- or anoxia. Lichenoid cell infiltration into the connective tissue—nearly always present—aggravates the process. Dyskeratosis, pyknosis and fibrinoid necrosis of the damaged cells occur and the nuclei disintegrate. The resultant colloid bodies may coalesce or scatter or be expelled from the epithelium according to the principles of apoptosis (6, 7). Besides circulatory disorders, purely local damage and pathological processes involving the tissues should be regarded as important factors. Although the colloid bodies are characteristic for various skin diseases, they do not have an absolutely clear diagnostic significance. In doubtful cases their presence may nevertheless be considered a valuable contribution and supplement to the diagnosis.

Key words: Colloid bodies; Fibrinoid necrosis; Lichenoid dermatoses; Mesenchymal colloid bodies

In the course of studies on paraproteins we found that these are often not uniform compounds, but various mixed proteins. They were found in the vicinity of mammary tumours or basalomas in the form of amyloid-like substances bound to the elastica. In the cells such a substance appears after certain kinds of damage, bound to fibrinoid. In the skin, no amyloid could be demonstrated in the epithelial cells, but fibrinoid mixed with horny substances (keratofibrinoid) was present (12). This material was also demonstrated in the colloid or hyaline bodies (CB) of lichen planus. At first these colloid bodies were regarded as characteristic of this disease, but subsequently they were observed in certain other skin diseases too. They were mentioned in a previous publication and will be discussed in greater detail here (see 13).

MATERIALS AND METHODS

All samples sent for histological examination were subjected to routine staining. Suitable cases were examined

with trichrome stainings (Ladewig, Goldner), fibrin stainings (Weigert, Kockel, Heidenhain), alcian blue, Congo red and PAS reaction. For fluorescence microscopy, acridine orange, thioflavin S, thioflavin T and eosin were used as fluorochromes, (for references to the methods, see 12). The pathological processes were divided into groups and the diseases were studied separately.

RESULTS

*Group A. Dermatoses Where CB or Similar Elements Have Previously Been Reported**Lupus erythematosus*

In this disease, CBs are 'extremely rare', according to Pinkus in his 'Guide' (11). Subsequently, Lever (8) and other investigated this phenomenon and—as in my own findings—observed these elements more frequently. It became evident that nearly all cases with CB belonged to the group called *lichen-like LE by Montgomery* (9).

In haematoxylin-eosin preparations, these cases show marked liquefaction degeneration, the subepidermal cell reaction is characterized by band-shaped confluence, though not as marked as in lichen planus. Formation of the CB starts as usual approximately halfway through the Malpighian layer in damaged epithelial cells. Signs of dyskeratosis are seen. The involved nuclei become pyknotic, the protoplasm stains intensive red. The nucleus is usually expelled, a clear empty space being seen in the cell. Subsequently this space fills with eosinophilic material and the cell shrinks further to become a CB. On the one hand, the CB may wander upwards and disappear into the horny layer. At the same time an apoptotic process starts (7); many CBs wander downwards and are expelled downwards. Some disintegrate, while others coalesce. They may accumulate in small fissures or vesicles. They undergo complete necrosis, take on a reddish-grey to grey colour and disappear in the exudate.

Trichrome staining: with Ivemark's modification of Ladewig's method (12) the dyskeratotic cells and CBs stain brilliant red, as does the basement membrane. The horny layer stains an intense orange. Once the further regressive changes have occurred, small bluish-red inclusions are often seen in the dyskeratotic cells. In the CBs the colour becomes reddish-blue to bluish-grey, until it disappears among the closely packed round cells.

After staining with Goldner's method the same changes are seen, with colours ranging from bright red to greenish blue. With van Gieson's method the CBs are seen as bright yellow elements that do not change colour. *After trichrome staining the sections do not show fluorescence.*

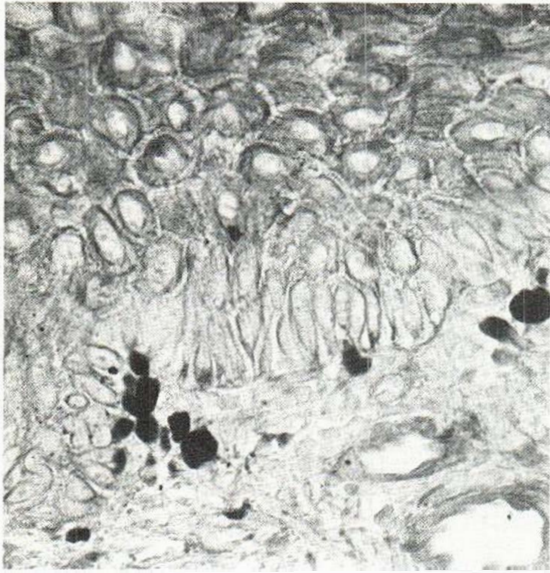


Fig. 1. Expelled colloid bodies in the dermal-epidermal junction, stained black after Kockel's method. Lichenoid erythematodes. $\times 400$.

The PAS-reaction was always positive for the CBs, although often weakly so. Congo red always yielded negative results and so did polarization, thionine, toluidine blue and alcian blue.

Fibrin staining: with Weigert's method, keratin and the dyskeratotic cells stain intensely. However, below the granular layer the colour disappears immediately. With the haematoxylin methods, fibrin and fibrinoid, erythrocytes and the striated muscles stain normally. The damaged cells and the CBs stain black, deep grey, or light grey. The intensity of staining is probably related to the keratin content. Parallel with increasing fibrinoid necrosis and degradation of the horny components the colour faded rapidly. Staining after Kockel remains persistently positive (fig. 1), while Heidenhain's method yields barely weakly positive results (0 to +).

Fluorochromes: the best results are obtained with acridine orange. In Wood's light the horny layer normally fluoresces with a beautiful golden-yellow colour, sometimes with a reddish tinge. Normal epithelium does not show fluorescence. The dyskeratotic cells and the CB display bright fluorescence similar to that of the horny layer or often even more intense. This decreases deeper down and shows a greenish tinge. In the CBs in small vesicles or fissures the tints described are often seen in the closely packed CBs, building co-called *mosaic structures* or *piebald structures* (12). These indicate phasic changes. The fluorescence persists for a long time. Rhodamine B stains all horny matter a silvery red; intracellular degradation products are hardly coloured or not at all. At most a pale green fluorescence is seen. The same applies to Taft's method. Thioflavins are unreliable.

Poikiloderma atrophicans

In the first so-called inflammatory stage a subepithelial band-like infiltrate with isolated mature CBs is seen. These bodies, first described by A. Civatte (3), are sparsely distributed in the thin epithelium and lie at the dermo-epidermal junction. They are even seen in chronic cases without exudate. Stains and fluorescence microscopy yield the same results as in lichen planus.

Pityriasis varioliformis lichenoides acuta (Mucha-Haberman)

The disease develops relatively rapidly. Again, regressive changes in the cells as described above are observed and CBs develop if there is abundant exudate. The complete picture is somewhat more varied, the PAS reaction is ++, fibrin staining yields rather more strongly positive results. Piebald structures are rarely seen.

Group B. Cases of Superficial Inflammatory Processes

In such cases lichenoid tissue reaction can often occur. They range from erythema multiforme and toxic erythemas from simple drug reactions, to serious cases of Lyell's disease.

Erythema multiforme

This can be caused by a variety of external and internal noxious factors and is often characterized in the acute stage by epithelial damage (epidermal type), formation of subepidermal vesicles and a monocytic—often lichenoid— infiltrate in the dermis. Eosinophilic epithelial necrobiosis dominates and there is also necrosis of the cells, with shrinkage. Small fragments, partly expelled and morphologically corresponding to CBs, are also seen. Piebald structures are rare. Fibrin staining remains positive for longer periods than in the above-mentioned cases, except for Heidenhain's method.

Lichenoid toxic reactions

In these cases the changes are variable and mostly closely related to drug-induced eczema. CBs are mostly found in the oedematous basal cells or just below these.

Drugs may cause marked lichenoid infiltrates, often after treatment with hypotensive agents (hydantoin-hydralazine preparations). Formation of CBs has been reported by A. Civatte (3). In our cases penicillin was the drug most often responsible. In all cases with lichenoid tissue reaction, dyskeratosis and CBs were observed. In the horny layer and in the CBs the stains demonstrated few differences and alcian blue showed blue tinges, indicating the presence of acid polysaccharides (rapid course of the process).

Toxic necrolytic dermatosis (Lyell)

This group is not uniform and cases range from imitation of erythema multiforme or drug-induced eczema, to toxic processes that are serious from the outset. In our observation of fatal cases, mercury preparations, quinidine and gold as an antirheumatic preparation were involved. At

the margin of the process, small groups with "water-clear" finely vacuolized cells are sometimes seen in the vicinity (12, 13). These indicate a disturbance of the water balance as a consequence of hypoxia. Central coagulation necrosis takes place. Necrosis of single cells and formation of CBs are seen. Apoptosis takes place only when there is immediate contact between the necrotic process and the connective tissue and only in the presence of a lichenoid inflammatory process.

Group C. Bullous Dermatoses

Some skin diseases in this group have already been discussed in the studies on apoptosis and fibrinoid necrosis of the skin (13). They will therefore, be described only briefly.

In cases of *herpes zoster*, *herpes simplex* and *varicellae*, intra-epithelial virus vesicles are found and in their immediate vicinity the water-clear cells described above are observed. Despite the morphologically fairly severe lesion, these never show fluorescence and they are therefore fully viable. In the neighbourhood of the vesicle, necrosis of individual cells and CBs are seen in the otherwise normal epithelium. The damaged cells and the CBs may be expelled into the vesicle. There, piebald structures are often seen and the stages between keratinization, fibrinoid necrosis and complete cellular death can readily be studied. The staining reaction and fluorescence are as described above.

In *pemphigus vulgaris*, variable dyskeratosis is seen around the vesicle, in the epithelial cells and sometimes also in the vesicular contents. Some CBs are occasionally also found in the lumen. The other variants of pemphigus only show uncertain results as regards CBs. In the process as a whole, fibrinoid necrosis appears to dominate.

In a case of *stomatitis epidemica* we found CBs in the buccal mucosa around the vesicle.

In *dermatitis herpetiformis* CBs are sometimes found in the same distribution pattern as for the other vesicular dermatoses.

Porphyria cutanea tarda

The skin shows hyperpigmentation, the vesicles develop mostly in those part of the skin exposed to sunlight. Alcoholism with or without cirrhosis and syphilis were very common, according to personal observations (14). In a recent case we observed many dyskeratoses with subsequent pyknosis and expulsion of the nuclei in the covering epithelium of the vesicle and its surroundings. Typical CBs staining was red with Ladewig's method (Fig. 2) and intense fluorescence was also seen with fluorochromes.

Group D. Cases of Dermatoses with Special or Rarely Occurring Lichenoid Infiltrate

One such disease is *hyperkeratosis lenticularis perstans*, first described by Flegel. Of our 2 cases, one has been described in detail elsewhere (10). Both the patients showed typical histological features: thin epithelial lining, hyperkeratosis and a narrow lichenoid infiltration consisting of lymphoid cells. Several such cases were described

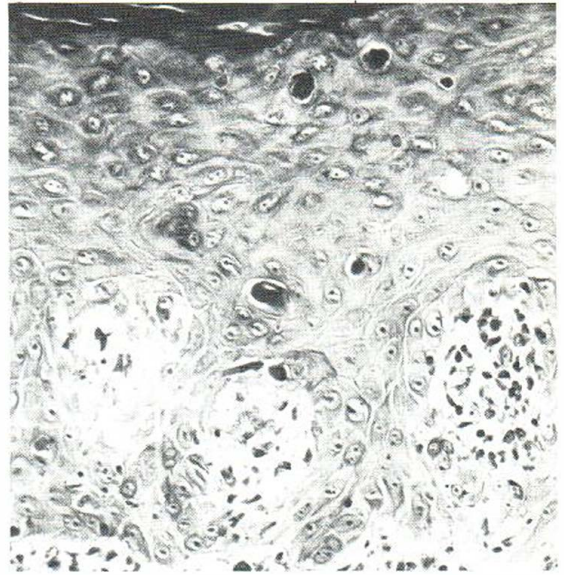


Fig. 2. Porphyria cutanea tarda. Intra-epithelial colloid bodies in the neighbourhood of one vesicle. Stained bright red according to Ladewig's method. $\times 320$.

by Nasemann. They are regarded as lichen ruber variants, called *lichen ruber lenticularis*. Very few CBs were found in the epithelium or expelled downwards. These showed the changes already described.

In the literature (Montgomery) (9) a description of such infiltrates is found in which the infiltrate does not consist of inflammatory cells. Such phenomena are sometimes seen in *sarcoidosis of the skin*. The miliary 'tubercles' form a superficial chain round cells. The CBs are found at the usual sites, although some are exceptionally found in the tubercles.

In *subcorneal pustular dermatosis* (Sneddon-Wilkinson's disease) and in *psoriasis pustulosa* there are similar findings. In the vicinity of the pustule epithelial damage is found and in the pustule itself necrobiotic epithelial cells, horny fragments and some homogeneous CBs are seen. The fluorescence is the most marked uppermost in the pustule, fading further downward. The appearance of CBs and signs of apoptosis may be related to the increased number of mitoses in psoriasis.

Group E. Diseases with Suprabasal Clefts *Keratosis follicularis Darier*

In typical cases acantholysis, suprabasal clefts, and damage to the keratinization process with consecutive formation of round bodies and grains are seen. The epithelium shows initial signs of dyskeratosis in the form of very small groups of slightly enlarged cells staining intense red with eosin and having pyknotic nuclei. They stain red with Ladewig's method and show intense fluorescence after fluorochromation. Such changes are also seen in the clefts and often lateral to the lesion, either with or even without nuclei. The round bodies may also become dyskeratotic.

Finally, these cells undergo fragmentation. These bodies which are therefore formed from the round bodies or also in the direct line from the dyskeratotic cells correspond to the grains, i.e. the final stage of dyskeratosis. The grains all show the staining reactions of the CBs, although often they still have shrunken remnants of nuclei. Around the periphery of the lesions CBs were still found—evidence of the severe epithelial damage.

Familial benign chronic pemphigus (morbus Hailey-Hailey)

The predominant histological feature is acantholysis, with suprabasal clefts and small vesicles. According to the literature, signs of a partial premature keratosis are 'sometimes' or 'rarely' seen. They are mentioned by J. Civatte (3) and were present in all of our cases.

Usually the nuclei stain weakly, the cell membrane fluoresces and some CBs form, especially close to the vesicle. The connective tissue seen to contain some lichenoid cell infiltration. The dyskeratosis probably does not belong to the basal disease; its presence can be explained by hypoxaemia (12).

Transient acantholytic dermatosis Grover

This peculiar disease was first described in 1958 (4). Usually there are papulo-vesicles and the course of the disease is relatively brief (a few weeks to a month or more). The number of variants of this disease is particularly remarkable. In addition to cases with a chronic course, four types have been described (Grover) (2): spongiotic-acantholytic, Darier-like, Hailey-Hailey-like and pemphigus-like. One case has been published where the oral cavity was involved (18).

The main symptoms are acantholysis, spongiosis and dyskeratosis. In cases of the spongiform type a very extensive spongiosis is seen through the entire depth of the epithelium. Locally, small acantholytic vesicles develop.

Dyskeratotic cells are also seen and scattered CBs are very easily visualized by fluorescence microscopy. In the dermis a very slight inflammatory reaction is observed. The impression is gained that dyskeratosis and apoptosis are caused not only by hypoxaemia of the vicinity, but also by extensive epithelial damage (severe spongiosis). This is the case even for the fibrinoid necrosis observed. In cases of the pemphigoid type, small suprabasal vesicles were seen, with expelled acantholytic and dyskeratotic cells in the lumen and in the wall of the vesicle. Such cells and some CBs were also found in the vicinity of the lesion, where they were particularly clearly visible by fluorescence microscopy.

Darier-like variant: the phenomena seen are roughly the same as those called focal acantholytic dyskeratosis by Ackerman (1), i.e. also round bodies and grains with staining reactions corresponding to CBs. The differential diagnosis from true Darier's disease may be difficult and only the clinical course can confirm it.

In a Hailey-Hailey-like variant we found only acantholysis, little spongiosis at the margin of the lesion and some CBs in the vicinity. There was no dyskeratosis.

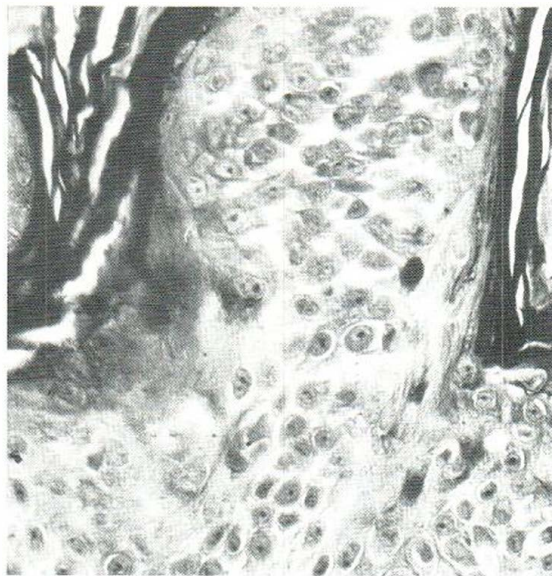


Fig. 3. Some colloid bodies in a case of epidermolytic hyperkeratosis Ladewig's stain. $\times 320$.

Group F. Dermatoses with Predominantly Physical Causes

Epidermolytic hyperkeratosis

A complex of symptoms whose nature has not yet been determined with certainty, (Ackerman (1), Pinkus (11)). There is a certain relationship to ichthyosiform erythrodermia. It may be found in cases of epidermal or ichthyosiform naevi, or on a small scale over dermatofibroma and in sweat gland units. It may also occur over larger areas.

There is swelling in small groups of cells of the granular layer, with rupture and formation of small vesicles. Ladewig's staining is positive (Fig. 3). With fluorescence microscopy, weak fluorescence of the affected cell membranes is seen as a sign of dyskeratotic cellular damage. The scattered 'eosinophilic bodies' (Ackerman (1)) also show fluorescence and can be grouped among the CBs. Further studies are necessary.

Similar changes can be caused by *large subepithelial cysts* close to the covering epithelium. The epithelium in the wall of the cyst remains normal; subsequently round-cell infiltration takes place and in the covering epithelium dyskeratotic cells and some CBs are seen. They are more numerous in the vicinity.

Polymorphic actinic eruption

The patients had usually sun-bathed for several days. In the upper epithelial layer, eosinophilic cell necroses, oedema and dyskeratosis are seen, while around the lesion itself some strongly fluorescing CBs are often found. M. Thyresson & Wennersten (16) examined the skin of such patients who for diagnostic reasons were tested with ul-

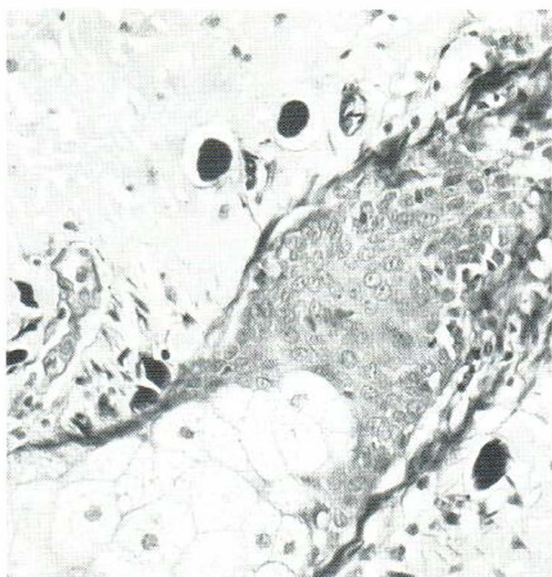


Fig. 4. Mesenchymal colloid bodies in lichenoid toxicoderma. Dark blue staining by Ladewig's method. $\times 250$.

traviolet light in order to determine their actinic sensitivity. They kindly supplied the histological preparations. In the upper epithelial layer we found eosinophilic necrobiosis, dyskeratosis, signs of fibrinoid necrosis and numerous intensely fluorescing CBs, even in small vesicles.

Group G. Colloid Bodies of Connective Tissue Origin: ('mesenchymal CBs')

In the studies described, peculiar CB-like formations were sometimes observed in the dermis. These are very similar to the epithelial CBs but do not display the usual staining reactions. They occur in small groups and the epithelium covering them is undamaged. They would appear to originate from the connective tissue. They are not always as sharply rounded as the CBs mentioned previously and often appear in connection with damaged basement membranes, or collagen bundles. The description of such bodies was established long ago. Their filamentous structure can be seen under the light microscope, whereas for the other CBs the electron microscope is necessary.

Eosin staining yields the same results for both forms, as does fluorescence. Ladewig's and Goldner's techniques with their red hues are negative here, i.e. the CBs stain a vivid blue or sometimes only lilac resp. greenish (Fig. 4). With van Gieson's method they stain red—as does normal connective tissue—or sometimes only orange. The colours seen by fluorescence microscopy shift towards the yellow. It can therefore be concluded that under certain circumstances still not clear, not only the epithelial cells but also the connective tissue is damaged by regressive processes to such an extent that the end products are rather similar, although fundamental differences are

found. Epithelial and mesenchymal CBs can appear simultaneously in the same histological preparation. With routine staining they are usually difficult to differentiate.

COMMENTS AND DISCUSSION

The dermatoses described here were divided into groups and encompass 30 different pathological processes.

The first three groups contain superficial inflammatory processes having a chronic or more acute course. Formation of CBs starts with dyskeratotic changes of the epithelial cells. Eosinophilic necrosis of the cells is seen, with pyknotic nuclei. Marked fluorescence of these cells is evident and CBs developing from them. The CBs are mostly transported downwards (apoptosis 6, 7) and expelled by the epithelium: they disintegrate and disappear in the neighbouring exudate. Another regressive process is also seen. The red hue seen on staining after Ladewig shifts towards blue-lilac, and the fluorescence colours are tinged with green. This process corresponds to a fibrinoid necrosis. From the hues varying from red-lilac-grey or golden yellow-greenish yellow-grey in Wood's light, one may infer a phasic course of the disease. Expect for substances closely related to fibrinoid, the damaged cells and CBs also contain degraded and premature horny material, as was confirmed by Thyresson & Moberger (15) in experimental studies. The mixed protein described was called keratofibrinoid and corresponds to the material in the CBs (Sümeği) (12).

The amyloid-like mixed proteins we found in liver cells affected by fibrinoid necrosis or in cancers of the breast and basalomas could not be demonstrated in the lesions described, with the techniques used. Gueft (5) and others demonstrated the close relationship between keratin, fibrinoid and amyloid by ultrastructural and X-ray diffraction studies. The phasic course of the pathological process and the piebald structures are more often found in the chronic cases of group A than in the more acute cases of group B, which indicates the importance of the time factor.

It is very striking that in these three groups the formation of CBs is most marked in cases where a lichenoid reactive infiltrate develops. In the close vicinity of the pathological process—most clearly so in group C—signs of circulatory disturbance are seen: stasis, dilatation of the small vessels, oedema,

small groups of finely vacuolized cells in the epithelium; in short: signs of hypoxia/anoxia. In the literature the appearance of the finely vacuolized cells has been called cellular hydropsy or urticaria of the cells and is attributed to an expansion of the endoplasmic reticulum. The injured cells apparently lose their ability to eliminate water and the hypoxia may contribute to the development of dyskeratosis. Therefore the lichenoid infiltrate should be considered to play an important role in the development of hypoxia and circulatory disorders in various diseases. However, specific properties (porphyria, zoster, etc.) should also be considered. On purely morphological grounds it is difficult to determine what are the respective degrees of importance of circulatory diseases and specific properties of the manifold diseases in the formation of CBs.

Certainly, in all cases the factors mentioned are present in varying proportions. The different morphology of the lichenoid infiltrate and causes in the organism that can affect the characteristic formation there still await an explanation (11). The morphology and the staining of the CBs are yet relatively constant.

The cases with suprabasal clefts show a much milder inflammatory reaction and are morphologically somewhat different keratinization anomalies. In Darier's disease the uncomplicated cases show hardly any lichenoid changes. The marked dyskeratosis, round-bodies, grains and transition to CBs are nearly always seen, however. Pinkus described the grains as "cornified bodies . . . and there is no obvious reason for not assuming that corps ronds and grains are progressing through a granular stage to a shrunken non-nucleated keratinized body". These bodies show all the reactions of the CBs. It should be stressed again that fluorescence is related to the viability of the cell, the corps ronds do not fluoresce in Wood's light, as long as they contain nuclei, but only after incipient necrosis. The grains and the CBs also show a more intense fluorescence after expulsion of their nuclei.

In Hailey-Hailey's disease inflammation in the connective tissue is mostly absent. The occurrence of CBs must be explained by the nutritional disorders of the epithelium, caused by the extensive acantholysis, formation of clefts and vesicles and spongiosis.

Grover's disease is an interesting entity, occurring in four variants, as far as is known. The reactive inflammation is very variable and here again

the presence of CBs must often be explained predominantly by local damage. Nevertheless, their morphology is fairly uniform. It might be assumed that the various manifestations of the disease can be affected by the immunobiological state, the internal secretion, and previous diseases of the damaged organism. The common element, however, is the injury of the epithelium and the possible appearance of CBs. External damage to the epithelium as a result of sunlight and ultraviolet light can possibly play a similar role. The consequence is poor nutrition of the epithelium and the protein and lipid reserves of the keratinocytes become depleted. The circulation is also damaged.

Not only can the epithelium be markedly damaged, but also the connective tissue. Under circumstances that have not yet been studied in any great detail, peculiar CBs may appear. Purely morphologically, these CBs are similar as end products of different origins, but significant differences are also found. To determine these, routine methods are not sufficient—the differences on staining described should be carefully considered. CBs of differing origins may occur in the same preparation. The studies show that both indicate damage to the epithelial cells or connective tissue.

CONCLUSION

In the present study a description is given of 30 diverse dermatoses with colloid bodies (CBs). These colloid bodies develop as a result of damage to the epithelium, e.g. circulatory disorders with subsequent hypo/anoxia. Lichenoid cell infiltration, if present, aggravates the process. Besides circulatory disorders, purely local damage of the tissue or general pathological changes of the organism should be regarded as important etiological factors.

The onset of the process, with dyskeratosis, the appearance of fibrinoid necroses and the degeneration of the CBs discussed. CBs may originate even from the connective tissue. The CBs characteristic of various skin diseases, yet they do not have any clear diagnostic significance. In doubtful cases their presence must be considered as a valuable detail to establish and support the most precise diagnosis.

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REFERENCES

1. Ackerman, A. B.: Diagnostic Inflammatory Skin Diseases.: Lea & Febiger, Philadelphia, 1978.
2. Chalet, R., Grover, R. W. & Ackerman, A. B.: Transient acantholytic dermatosis. *Arch Dermatol* 113: 431-435, 1977.
3. Civatte, J.: Histopathologie cutanée. Flammarion, Paris 1967.
4. Grover, R. W.: Transient acantholytic dermatosis. *Arch Dermatol* 101: 426-434, 1970.
5. Gueft, B.: The analogy of amyloid and keratin as suggested by X-ray, aminoacid and ultrastructural analysis. *Mount Sinai J Med* 39: 91-102, 1972.
6. Hashimoto, K.: Apoptosis in lichen planus and several other dermatoses. *Acta Dermatovener (Stockholm)* 56: 187-210, 1976.
7. Kerr, J. F., Wyllie, A. H. & Currie, A. R.: Apoptosis: A basic phenomenon with wide ranging implications in tissue kinetics. *Br J Cancer* 26: 239-257, 1972.
8. Lever, W. F. & Lever-Schaumburg, G.: Histopathology of the Skin (ed. V. Lippincott). Philadelphia, 1975.
9. Montgomery, H.: Dermatopathology. Harpér & Row, New York, 1967.
10. Neumann, E., Nilsson, R. & Paul, U.: Hyperkeratosis lenticularis perstans Flegel. *Acta Dermatovener (Stockholm)* 54: 475-478, 1974.
11. Pinkus, E. & Mehregan, A. H.: A Guide to Dermatohistopathology, 2nd edn., Appleton, New York, 1976.
12. Sümegi, I.: Fibrinoid necrosis of the epithelial cells of the skin. *Acta Pathol Microbiol Scand [A]* 84: 109-119, 1975.
13. — Fibrinoid necrosis and downward motion of colloid bodies in lichen planus (Apoptosis). *Acta Dermatovener (Stockholm)* 59: 27-31, 1979.
14. Szodoray, L. & Sümegi, I.: Über die Nosologie der chron. kutan. Porphyrie. *Dermatologica (Basel)* 90: 224-230, 1944.
15. Thyresson, N. & Moberger, G.: Cytologic studies in lichen ruber planus. *Acta Dermatovener (Stockholm)* 37: 191-204, 1957.
16. Thyresson, M. & Wennersten, G.: Unpublished investigations.

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