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Amyloidosis in the Skin

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1. Introduction

Amyloidosis is induced by deposition of amyloid proteins in various organs. Both systemic and localized type amyloidosis present with a variety of skin manifestations. Based on biochemical and immunological aspects, amyloid proteins are subdivided into several subtypes from different origins. Amyloid fibrils in primary and multiple myeloma-associated systemic amyloidosis are composed of immunoglobulin protein AL (light chain), whereas in secondary systemic amyloidosis, they are composed of a non-immunoglobulin protein (amyloid AA). In primary localized cutaneous amyloidosis, amyloid materials are derived from cytokeratin; however, in nodular primary cutaneous amyloidosis, amyloid is AL type. Dialysis-related amyloidosis is composed of β2-microglobulin.
So far, there are several reviews of skin features associated with amyloidosis [1-3]. Cutaneous amyloidosis is characterized by deposition of amyloid in the skin, which is seen in association with systemic amyloidosis and also restricted to the skin. In case of association with systemic amyloidosis, skin lesions are important as one of the extra-hematologic manifestations, because cutaneous lesions may occasionally be the initial presentation of systemic amyloidosis. Representative lesions include petechiae, purpura, ecchymoses, and eyelid translucent papulonodular lesions. By contrast, amyloidosis limited to the skin is called primary localized cutaneous amyloidosis, which is clinically classified into more common macular, papular, and the rare nodular form. Also, reports of cases showing peculiar forms of cutaneous amyloidosis are seen, depending on the different races. Additionally, amyloid deposition is secondarily seen in association with skin tumors, such as basal cell carcinoma, Bowen's disease, and other benign tumors. In this review, both primary and secondary skin lesions associated with systemic as well as cutaneous amyloidosis are discussed, making a focus on mucocutaneous manifestations.

2. Amyloid materials

Various subtypes of cutaneous amyloid are distinguished. Amyloid deposits are verified by several specific stains such as PAS, thioflavine T fluorescence, Congo red, and Dylon (Fig. 1). Light microscopy reveals amorphous materials extracellularly. Investigation by electron microscopy shows fibrillar materials (Fig. 2).

3. Systemic amyloidosis

Primary systemic amyloidosis (AL amyloidosis) is caused by plasma cell dyscrasia, and develops in 10-20% of patients with multiple myeloma. Various organs are affected such as

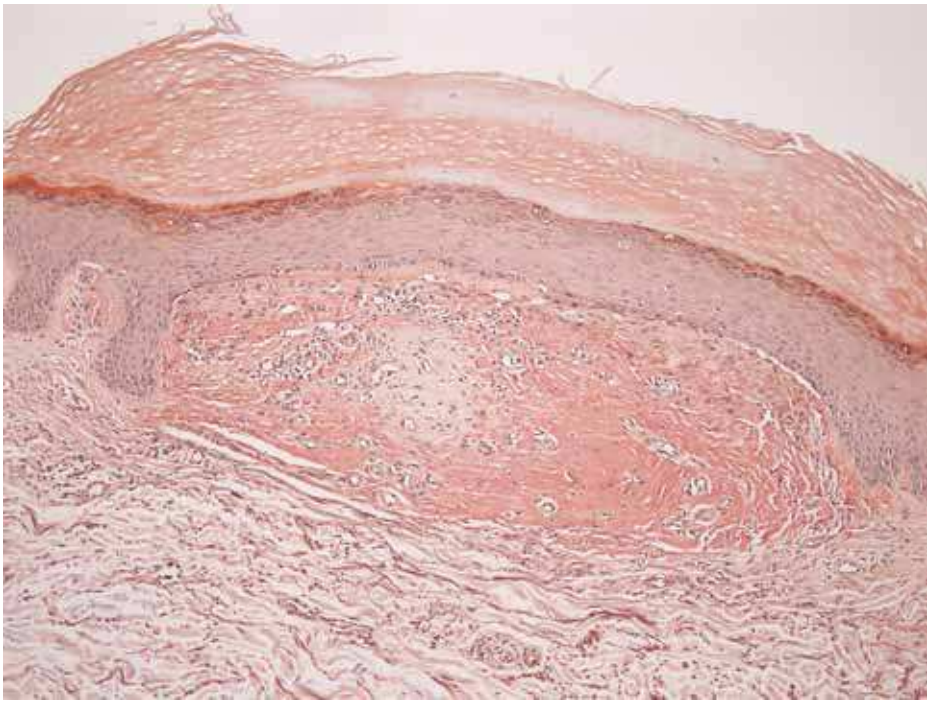


Fig. 1. Massive amyloid deposition with melanophages is seen in the papillary dermis in the lesional skin of lichen amyloidosis (Congo red staining). Hyperkeratosis of the overlying epidermis is also seen

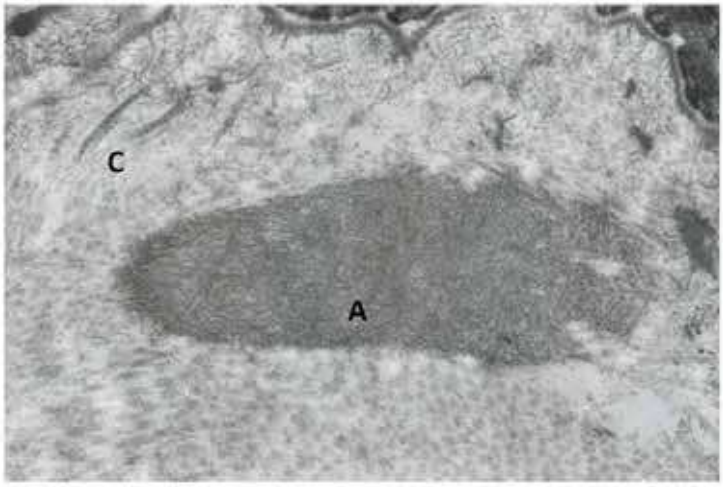


Fig. 2. Electron microscopy shows fibrillar materials consistent with amyloid (A) and normal collagen (C)

renal, cardiac, neuronal, gastrointestinal, hepatic, and splenic involvement. Skin manifestation is seen in approximately 30-40% of patients. Purpura, petechiae, and ecchymoses are induced in the skin as well as mucous membranes. Eyelid purpura is frequently seen (Fig. 3), and purpura are also seen elsewhere in the body. Purpura is caused by minor trauma, slight stimuli, or even spontaneously. Amyloid deposition is seen around the blood vessels, which causes capillary fragility. As periorbital lesions, translucent nodules, xanthomatous plaques, waxy yellowish hemorrhagic lesions are seen (Fig. 4, 5).

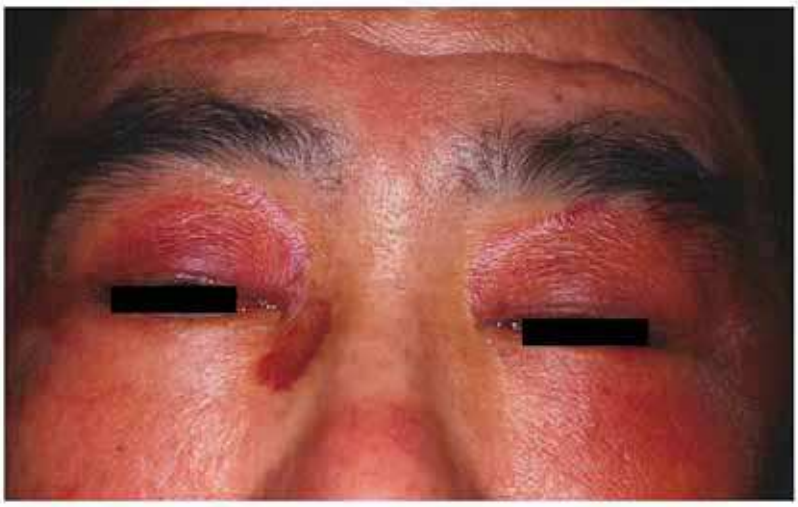


Fig. 3. Purpuric plaques around the bilateral eyelids

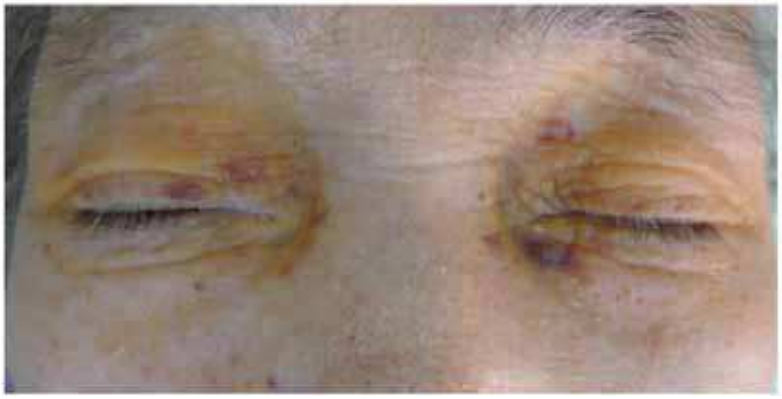


Fig. 4. Periorbital xanthomatous plaques and purpura

Other rare forms of cutaneous amyloidosis associated with systemic amyloidosis include subcutaneous nodules, whitish nodules, bullous lesions, and refractory ulcers [4, 5]. Amyloid deposition is also occasionally seen in the tongue and oral mucosa. Macroglossia is the representative sign (Fig. 6). Nail involvement is due to amyloid deposition in the nail matrix, and can be an initial manifestation of systemic amyloidosis [6]. Nail lesions present

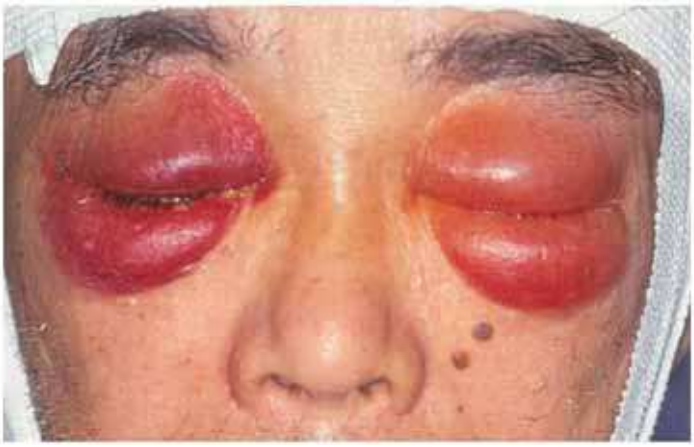


Fig. 5. Waxy, yellowish hemorrhagic lesions

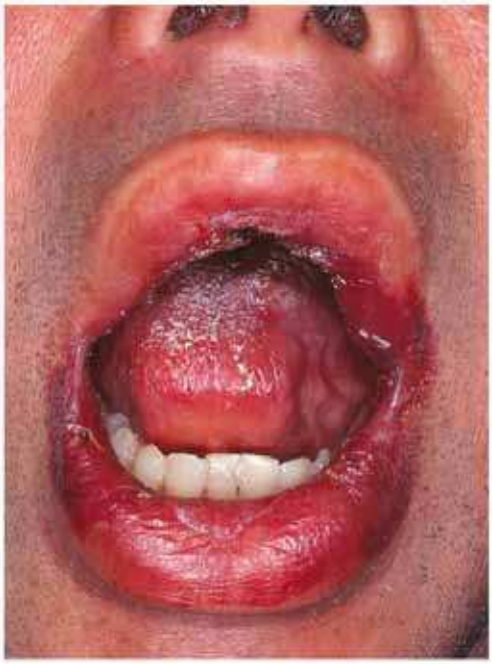


Fig. 6. Macroglossia in a patient with systemic amyloidosis

with dystrophy, thinning, whitening, banding, striations, brittleness, onycholysis, fragility, and even anonychia (Fig. 7). Alopecia may develop when amyloid deposition occurs on the hair matrix. Scleroderma-like manifestations are rarely seen, especially on the fingers, in patients with primary systemic amyloidosis [7-9]. Skin biopsy is important for the diagnosis, because amyloid deposition can be detected even in the skin of normal appearance. Blind

aspiration biopsies from the abdominal subcutaneous fat tissues or the rectal submucosa are sometimes useful for the definitive diagnosis for systemic amyloidosis.

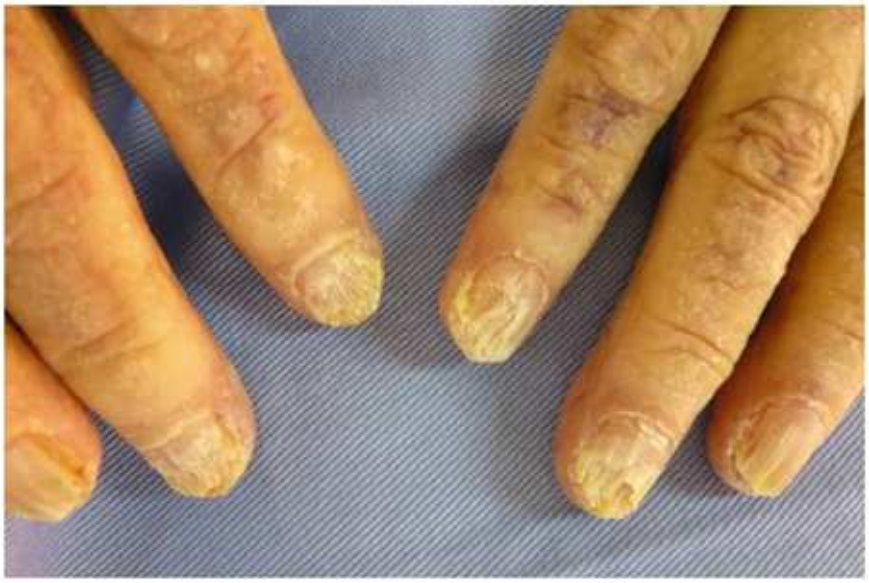


Fig. 7. Fingernails of a patient with systemic amyloidosis showing longitudinal ridging and splitting

4. Localized amyloidosis

Primary localized cutaneous amyloidosis (PLCA) is defined by deposition of amyloid in previously normal skin with no evidence of deposits in internal organs, and classified into more common macular, papular, and the rare nodular form. PLCA may be induced by chronic stimuli or minor trauma [10].

Lichen amyloidosis is frequently seen on the dorsal aspect of the lower legs and forearms, which is characterized by pruritic, firm, hyperkeratotic, reddish-brown papules or nodules (Fig. 8). Main component of amyloid in lichen amyloidosis is considered to be cytokeratin, suggesting that amyloid deposits may be derived from degenerated epithelial cells.

Macular amyloidosis is predominantly localized on the upper back, and characterized by dark pigmented macules with a rippled pattern of pigmentation (Fig. 9). In severe cases, macular amyloidosis involves all over the back (Fig. 10). Lichen amyloidosis and macular amyloidosis are occasionally seen in a single patient, and is known as biphasic forms. Unique features of macular amyloidosis are rarely seen on the upper back or exterior aspects of upper extremities. Lesions are pigmented, discrete spotty papules and not presented hyperkeratotic papules like lichen amyloidosis (Fig. 11). Those lesions are induced by prolonged scraching or rubbing with various objects such as bath sponges, brushes, towels, plant sticks and leaves, which resulted in keratinocyte degenereation. The same clinical entity includes friction amyloidosis, friction melanosis, and towel melanosis [11]. Friction amyloidosis is induced by long-term use of a nylon towel or scrub brush over

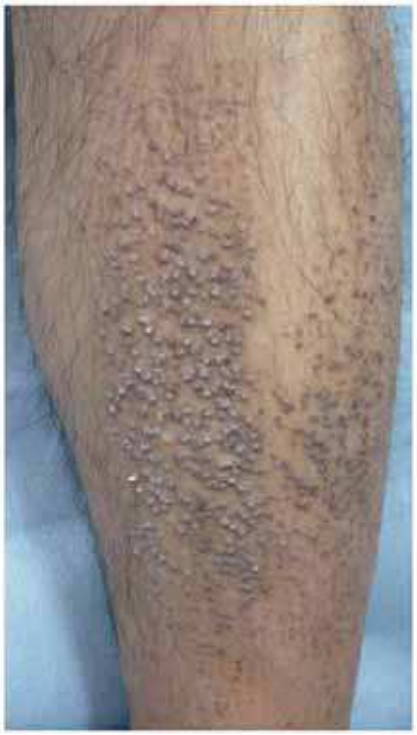


Fig. 8. Lichen amyloidosis on the lower leg

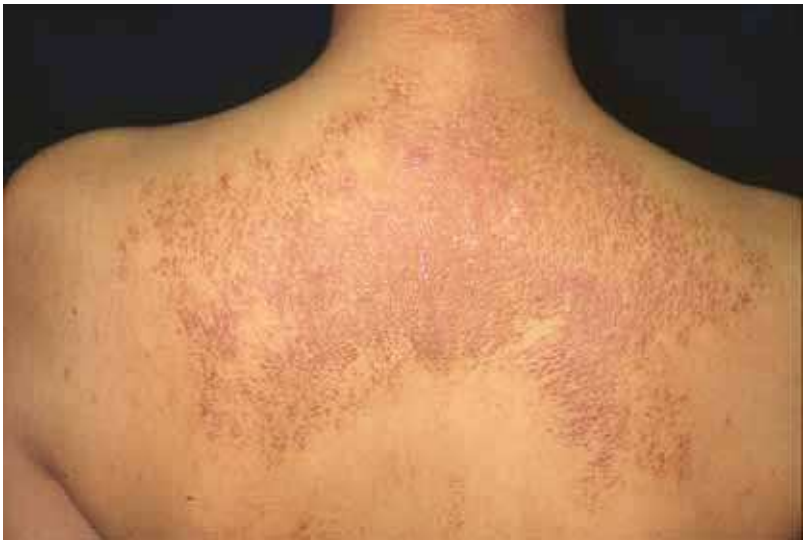


Fig. 9. Macular amyloidosis on the upper back

Fig. 10. Widespread primary cutaneous localized amyloidosis (macular form)

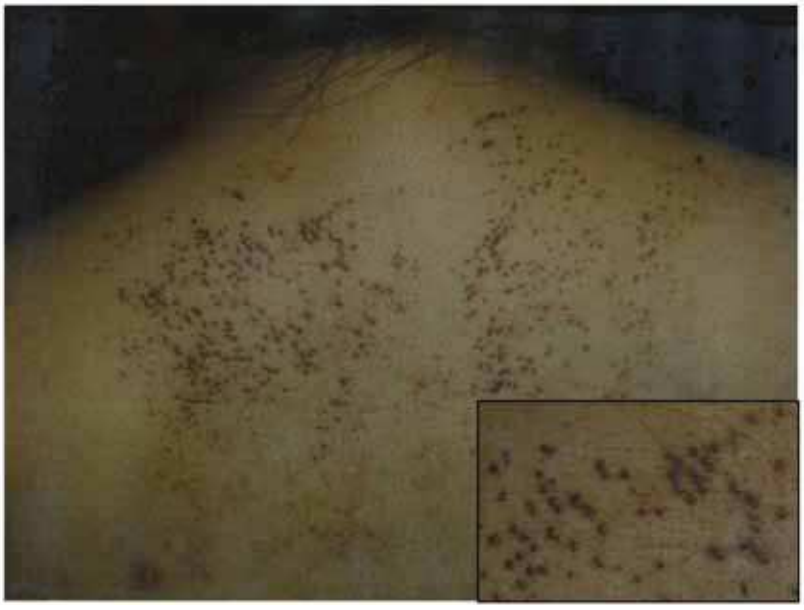


Fig. 11. Pigmented papular form of macular amyloidosis on the upper back (insert: higher magnification)

the bony regions such as the arms, forearms, clavicle, scapula, and neck [12, 13]. Amyloid deposits in the skin may be derived from degenerated epithelial keratinocytes [14], possibly through filamentous degeneration or apoptosis [15]. Histological investigation by amyloid stain show deposition of amorphous materials in the papillary dermis. Amyloid is usually detected unassociated with hair follicles, but rarely recognized around the follicles (Fig. 12).
Nodular amyloidosis presents with a single or multiple nodules on the face, trunk and extremities [16, 17], which sometimes develop following trauma (Fig. 13). Also, periorbital small, waxy nodules are multiply seen. In the nodular type, amyloid is originated from AL protein by local plasma cells, and modified β2-microglobulin is also shown to be a component of amyloid fibrils [18]. Apart from other types, amyloid materials are situated up to in the deep dermis. Plasma cell infiltration is prominent within or peripheral areas of the amyloid materials (Fig. 14). Although patients with nodular amyloidosis may develop systemic amyloidosis after long-term follow-up, recent papers indicate that the ratio is lower than previously reported [16]. In the series of 16 cases of nodular amyloidosis, 2 patients had Sjögren's syndrome, 2 had diabetes mellitus, and 3 had liver disease. In particular, association with Sjögren's syndrome is remarkable [19].
PLCA usually is unassociated with systemic disorders; however, a few cases of HCV-related amyloidosis have been reported [20, 21], one of which was biphasic PLCA [20]. Nodular lesions showing the surface atrophy are described as amyloidosis cutis nodularis atrophicans.
Additionally, other unusual variants of PLCA have been reported depending on genetic, racial, and environmental factors. Those include poikiloderma-like appearance, reticular

Fig. 12. Amyloid deposition around the hair follicle in the papillary dermis in the lesional skin of macular amyloidosis (Congo red)

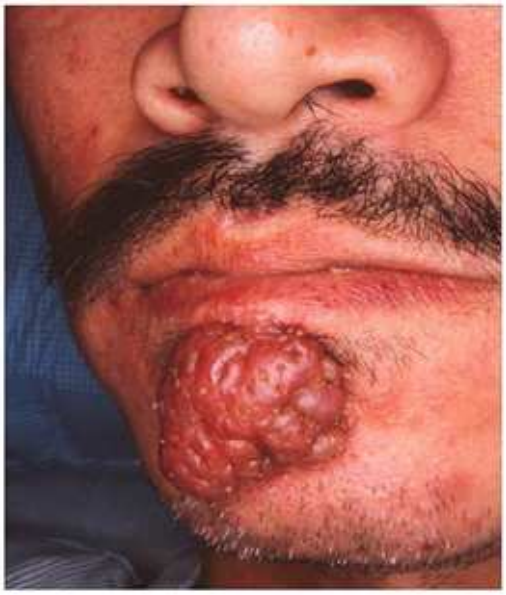


Fig. 13. Nodular amyloidosis on the chin

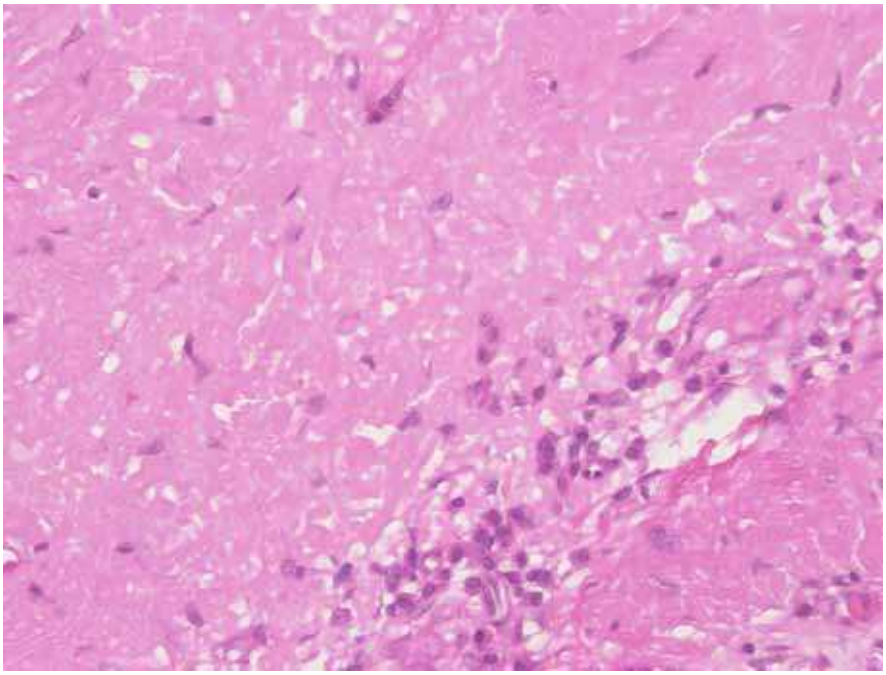


Fig. 14. Plasma cell infiltration in the lesional skin of nodular amyloidosis

form, hypopigmented, widespread diffuse pigmentation, incontinentia pigmenti-like pattern, homogenous pigmented patched, and amyloidosis cutis dyschromia [22-30]. Also, eczematous lesions [31, 32] and bullous lesions mimicking bullous pemphigoid [33] have been reported. Rare sites include ear, nose, and cheek [34-38]. Anosacral cutaneous amyloidosis is frequently seen in Asian elderly people, and hyperkeratotic, pigmented plaques are located on the bilateral outer area of the anus (Fig. 15).

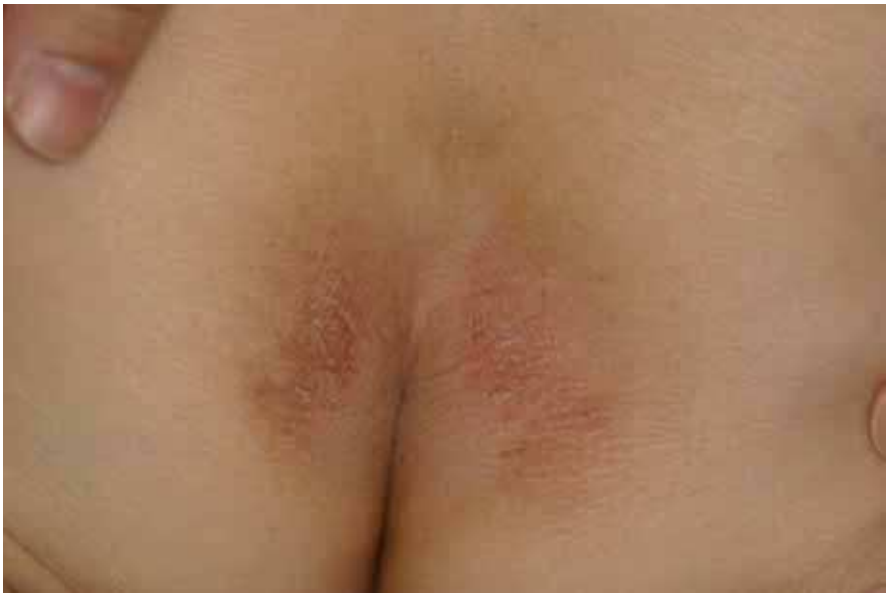


Fig. 15. Anosacral amyloidosis

Therapeutic modalities for cutaneous amyloidosis remain a challenge, although topical application of corticosteroids and dimethylsulfoxide (DMSO), phototherapy, and laser treatment are selected.

5. Secondary amyloidosis associated with various disorders

An association of cutaneous amyloidosis with various disorders (*i.e.* inflammatory disorders, autoimmune disorders, and tumors) has been reported. Amyloid deposition is occasionally seen associated with chronic inflammation, and secondary cutaneous amyloid deposition is sometimes seen in patients with atopic dermatitis [39]. Chronic stimuli by frequent scratching may play a triggering role in amyloid production from keratinocytes. Additionally, secondary amyloid deposition associated with inflammatory disorders, such as psoriasis [40] or disseminated superficial porokeratosis [41] have been reported.

Connective tissue diseases or collagen vascular diseases have been rarely seen, such as systemic sclerosis, lupus erythematosus, Sjögren's syndrome, rheumatoid arthritis, dermatomyositis, Behchet's disease, sclerodermatomyositis, generalized morphea-like scleroderma, sarcoidosis, and so on [42-47]. Association of PLCA with various autoimmune disorders suggests that underlying immune-mediated factors may be implicated [47].

Secondary amyloid deposition is occasionally associated with both benign and malignant skin tumors such as melanocytic naevi, seborrheic keratosis, calcifying epithelioma, dermatofibroma, solar keratosis, Bowen's disease, basal cell carcinoma, trichoepithelioma, and so on [48-56]. Although an epidermal origin of amyloid in secondary cutaneous amyloidosis, particularly in association with skin tumors of epithelial cell origin, is suggested in several conditions, degenerating naevus cells may contribute to the amyloid production [55]. A previous report showed amyloid deposition in Bowen's disease treated with radiotherapy [53]. It is suggested that any insult to the skin leading to degenerative cell changes could result in amyloid deposition.

6. Dialysis-related amyloidosis

In dialysis-related amyloidosis, skin manifestations often present with cutaneous or subcutaneous nodules [57, 58]. Bilateral subcutaneous masses are seen on the buttocks (Fig. 16), which are sometimes painful on sitting. Extensive deposition of β2-microglobulin amyloid is seen in the dermis to subcutis, occasionally associated with local calcification.

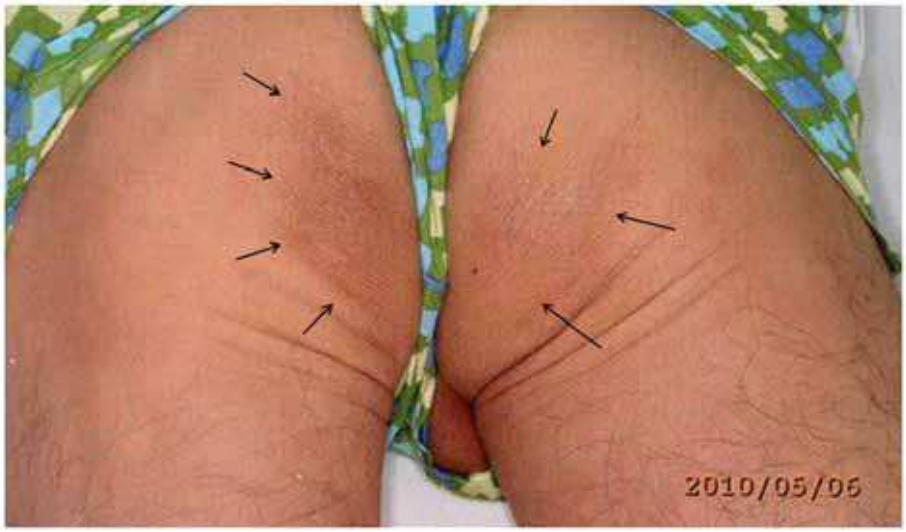


Fig. 16. Dialysis-related amyloidosis on the buttocks

7. References

[1] Steciuk, A., Dompmartin, A., Troussard, X., Verneuil, L., Macro, M., Comoz, F., & Leroy, D. (2002) Cutaneous amyloidosis and possible association with systemic amyloidosis. *International Journal of Dermatology* vol. 41, pp. 127-132.

[2] Silverstein, S.R. (2005) Primary systemic amyloidosis and the dermatologist: where classic skin lesions may provide the clue for early diagnosis. *Dermatology Online Journal* vol. 11, pp. 5.

[3] Schreml, S., Szeimies. R.-M., Vogt, T., Landthaler, M., Schroeder, J., & Babilas, P. (2010) Cutaneous amyloidoses and systemic amyloidoses with cutaneous involvement. *European Journal of Dermatology* vol. 20, pp. 152-160.

[4] Robert, C., Aractingi, S., Prost, C., Verola, O., Blanchet-Bardon, C., Blanc, F., Bagot, M., Dubertret, L., & Fermand, J.P. (1993) Bullous amyloidosis: report of 3 cases and review of the literature. *Medicine* vol. 72, pp. 38-44.

[5] Alhaddab, M., Srolovitz, H., & Rosen, N. (2006) Primary systemic amyloidosis presenting as extensive cutaneous ulceration. *Journal of Cutaneous Medical Surgery* vol. 10, pp. 253-256.

[6] Fujita, Y., Tsuji-Abe, Y., Sato-Matsumura, K.C., Akiyama, M., & Shimizu, H. (2006) Nail dystrophy and blisters as sole manifestations in myeloma-associated amyloidosis. *Journal of American Academy of Dermatology* vol. 54, pp. 712-714.

[7] Lee, D.D., Huang, C.Y., & Wong CK. (1998) Dermatopathologic findings in 20 cases of systemic amyloidosis. *American Journal of Dermatopathology* vol. 20, pp. 438-442.

[8] Cho, S.B., Park, J.S., Kim, H.O., & Chung, K.Y. (2006) Scleroderma-like manifestation in a patient with primary systemic amyloidosis: response to high-dose intravenous immunoglobulin and plasma exchange. *Yonsei Medical Journal* vol. 47, pp. 737-740.

[9] Reyes, C.M., Rudinskaya, A., Kloss, R., Girardi, M., & Lazova, R. (2008) Scleroderma-like illness as a presenting feature of multiple myeloma and amyloidosis. *Journal of Clinical Rheumatology* vol. 14, pp. 161-165.

[10] Lonsdale-Eccles, A.A., Gonda, P., Gilbertson, J.A., & Haworth, A.E. (2009) Localized cutaneous amyloid at an insulin injection site. *Clinical and Experimental Dermatology* vol. 34, pp. e1027-e1028.

[11] Siragusa, M., Ferri, R., Cavallari, V., & Schepis, C. (2001) Friction melanosis, friction amyloidosis, macular amyloidosis, towel melanosis: many names for the same clinical entity. *European Journal of Dermatology* vol. 11, pp. 545-548.

[12] Wong, C.K., & Lin, C.S. (1988) Friction amyloidosis. *International Journal of Dermatology* vol. 27, pp. 302-307.

[13] Venkataram, M.N., Bhushnurmath, S.R., Muirhead, D.E., & Al-Suwaid, A.R. (2001) Friction amyloidosis: a study of 10 cases. *Australasian Journal of Dermatology* vol. 42, pp. 176-179.

[14] Chang, Y.T., Liu, H.N., Wang, W.J., Lee, D.D., & Tsai, S.F. (2004) A study of cytokeratin profiles in localized cutaneous amyloids. *Archives of Dermatological Research* vol. 296, pp. 83-88.

[15] Maeda, H., Ohta, S., Saito, Y., Nameki, H., & Ishikawa, H. (1982) Epidermal origin of the amyloid in localized cutaneous amyloidosis. *British Journal of Dermatology* vol. 106, pp. 345-351.

[16] Moon, A.O., Calamia, K.T., & Walsh, J.S. (2003) Nodular amyloidosis: review and long-term follow-up of 16 cases. *Archives of Dermatology* vol. 139, pp. 1157-1159.

[17] Kalajian, A.H., Waldman, M., & Knable, A.L. (2007) Nodular primary localized cutaneous amyloidosis after trauma: a case report and discussion of the rate of progression to systemic amyloidosis. *Journal of American Academy of Dermatology* vol. 57, pp. S26-S29.

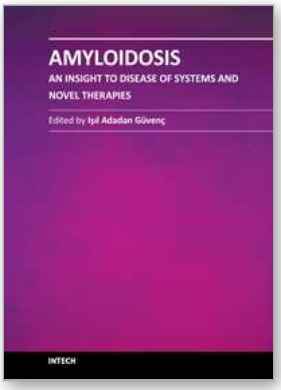
[18] Fujimoto, N., Yajima, M., Ohnishi, Y., Tajima, S., Ishibashi, A., Hata, Y., Enomoto, U., Konohana, I., Wachi, H., & Seyama, Y. (2002) Advanced glycation end product-modified beta2-microglobulin is a component of amyloid fibrils of primary localized cutaneous nodular amyloidosis. *Journal of Investigative Dermatology* vol. 118, pp. 479-484.

[19] Meijer, J.M., Schonland, S.O., Palladini, G., Merlini, G., Hegenbart, U., Ciocca, O., Perfetti, V., Leijsma, M.K., Bootsma, H., & Hazenberg, B.P. (2008) Sjögren's syndrome and localized nodular cutaneous amyloidosis: coincidence or a distinct clinical entity? *Arthritis and Rheumatism* vol. 58, pp. 1992-1999.

[20] Erbagci, Z., Erkilic, S. & Tuncel, A.A. (2005) Diffuse biphasic cutaneous amyloidosis in an HCV-seropositive patient: another extrahepatic manifestation of HCV infection? *International Journal of Clinical Practice* vol. 59, pp. 983-985.

[21] Abe, M., Kawakami, Y., Oyama, N., Nakamura-Wakatsuki, T., & Yamamoto, T. (2010) Cutaneous amyloidosis associated with HCV infection: report of 2 cases. *International Journal of Dermatology* vol. 49, pp. 960-961.
[22] Ho, M.H., & Chong, L.Y. (1998) Poikiloderma-like cutaneous amyloidosis in an ethnic Chinese girl. *Journal of Dermatology* vol. 25, pp. 730-734.
[23] Wang, C.K., & Lee, J.Y.Y. (1996) Macular amyloidosis with widespread diffuse pigmentation. *British Journal of Dermatology* vol. 135, pp. 135-138.
[24] Eng, A.M., Cogen, L., Gunner, R.M., & Blekys, I. (1976) Familial generalized dyschromic amyloidosis cutis. *Journal of Cutaneous Pathology* vol. 3, pp. 102-108.
[25] An, H.T., Han, K.H., & Cho, K.H. (2000) Macular amyloidosis with an incontinentia pigmenti-like pattern. *British Journal of Dermatology* vol. 142, pp. 371-373.
[26] Ahmed, I., Charles-Holmes, R., & Black, M.M. (2001) An unusual presentation of macular amyloidosis. *British Journal of Dermatology* vol. 145, pp. 851-852.
[27] Hung, C.C., Wang, C.M., Hong, H.S., & Kuo, T.T. (2003) Unusual skin manifestation of cutaneous amyloidosis. *Dermatology* vol. 207, pp. 65-67.
[28] Alvarez-Ruiz, S.B., Perez-Gala, S., Aragues, M., Fraga, J., & Garcia-Diez, A. (2007) Unusual clinical presentation of amyloidosis: bilateral stenosis of the external auditory canal, hoarseness and a rapid course of cutaneous lesions. *International Journal of Dermatology* vol. 46, pp. 503-504.
[29] Criado, P.R., Silva, C.S., Vasconcellos, C., Valente, N.Y., & Maito, J.B. (2005) Extensive nodular cutaneous amyloidosis: an unusual presentation. *Journal of European Academy of Dermatology and Venereology* vol. 19, pp. 481-483.
[30] Ho, M.S., Ho, J., & Tan, S.H. (2009) Hypopigmented macular amyloidosis with or without hyperpigmentation. *Clinical and Experimental Dermatology* vol. 34, pp. e547-e551.
[31] Konishi, A., Fukuoka, M., & Nishimura, Y. (2007) Primary localized cutaneous amyloidosis with unusual clinical features in a patient with Sjögren's syndrome. *Journal of Dermatology* vol. 34, pp. 394-396.
[32] Gan, E.Y., & Tey, H.L. (2010) Primary cutaneous nodular amyloidosis initially presenting with eczema. *Singapore Medical Journal* vol. 51, pp. e158-e160.
[33] Asahina, A., Hasegawa, K., Ishiyama, M., Miyagaki, T., Tada, Y., Suzuki, Y., Tanabe, T., & Saito, I. (2010) Bullous amyloidosis mimicking bullous pemphigoid: usefulness of electron microscopic examination. *Acta Dermatology and Venereology* vol. 90, pp. 427-428.
[34] Shimauchi, T., Shin, J.H., & Tokura, Y. (2006) Primary cutaneous amyloidosis of the auricular concha: case report and review of published work. *Journal of Dermatology* vol. 33, pp. 128-131.
[35] Evers, M., Baron, E., Zaim, M.T., & Han, A. (2007) Papules and plaques on the nose: nodular localized primary cutaneous amyloidosis. *Archives of Dermatology* vol. 143, pp. 535-540.
[36] Jhingan, A., Lee, J.S.S., & Kumarasinghe, S.P.W. (2007) Lichen amyloidosis in an unusual location. *Singapore Medical Journal* vol. 48, pp. e165-e167.
[37] Koh, M., Kwok, C.Y., Tan, H.W., & Mancer, J.F. (2008) A rare case of primary cutaneous nodular amyloidosis of the face. *Journal of European Academy of Dermatology and Venereology* vol. 22, pp. 1011-1012.
[38] Neff, A.G., McCuin, J.B., & Mutasim, D.F. (2010) Papular amyloidosis limited to the ears. *Journal of American Academy of Dermatology* vol. 62, pp. 1078-1079.
[39] Lee, D.D., Huang, C.K., Ko, P.C., Chang, Y.T., Sun, W.Z., & Oyang, Y.J. (2011) Association of primary cutaneous amyloidosis with atopic dermatitis: a nationwide population-based study in Taiwan. *British Journal of Dermatology* vol. 164, pp. 148-153.

[40] Wittenberg, G.P., Oursler, J.R., & Peters, M.S. (1995) Secondary amyloidosis complicating psoriasis. *Journal of American Academy of Dermatology* vol. 32, pp. 465-468.
[41] Ginarte, M., Leon, A., & Toribio, J. (2005) Disseminated superficial porokeratosis with amyloid deposits. *European Journal of Dermatology* vol. 15, pp. 298-300.
[42] Yamamoto, T., & Suzuki, Y. (2010) Primary localized cutaneous amyloidosis intermingled with papulopustular lesions in a patient with Behcet's disease. *Rheumatology International* May 15 [Epub ahead]
[43] Orihara, T., Yanase, S., & Furuya, T. (1985) A case of sclerodermatomyositis with cutaneous amyloidosis. *British Journal of Dermatology* vol. 112, pp. 213-219.
[44] Sumi, K., Yamamoto, T., Yokozeki, H., Nishioka, K. (2003) Amyloid deposition associated with generalized morphea-like scleroderma. *European Journal of Dermatology* vol. 13, pp. 509-511.
[45] Kikuchi, N., Sakai, E., Nishibu, A., Otsuka, M., & Yamamoto, T. (2010) Primary localized cutaneous amyloidosis in patients with scleroderma. *Acta Dermatology and Venereology* vol. 90, pp. 326-327.
[46] Taniguchi, Y., Horino, T., & Terada, Y. (2009) Cutaneous amyloidosis associated with amyopathic dermatomyositis. *Journal of Rheumatology* vol. 36, pp. 1088-1089.
[47] Dahdah, M.J., Kurban, M., Kibbi, A.G., & Ghosn, S. (2009) Primary localized cutaneous amyloidosis: a sign of immune dysregulation? International Journal of Dermatology vol. 48, pp. 419-421.
[48] Hashimoto, K., & King, L.E. Jr. (1973) Secondary localized cutaneous amyloidosis associated with actinic keratosis. *Journal of Investigative Dermatology* vol. 61, pp. 293-299.
[49] Peterson, W.C. Jr. (1968) Thioflavine-T reactivity in calcifying epithelioma of Malherbe. *Archives of Dermatology* vol. 97, pp. 340-341.
[50] Aso, M., Hagari, Y., Nakamura, K., Mihara, M., & Shimao, S. (1990) A case of secondary cutaneous amyloidosis: epidermal keratinocytes produce amyloid in the cytoplasm. *Journal of Cutaneous Pathology* vol. 17, pp. 176-181.
[51] Lee, Y.S., & Fong, P.H. (1990) Secondary localized amyloidosis in trichoepithelioma. A light microscopic and ultrastructural study. *American Journal of Dermatopathology* vol. 12, pp. 469-478.
[52] Weedon, D., & Shand, I. (1979) Amyloid in basal cell carcinoma. *British Journal of Dermatology* vol. 11, pp 141-146.
[53] Layton, A.M., Cunliffe, W.J., & Jones, W. (1991) Enhanced amyloid deposition in Bowen's disease treated with radiotherapy. *British Journal of Dermatology* vol. 125, pp. 606-607.
[54] MacDonald, D.M., & Black, M.M. (1980) Secondary localized cutaneous amyloidosis in melanocytic naevi. *British Journal of Dermatology* vol. 103, pp. 553-556.
[55] Hanami, Y., & Yamamoto, T. Secondary amyloid deposition in a melanocytic naevus. *International Journal of Dermatology* (in press).
[56] Quigley, B.C., Ricciuti, J., & Morgan, M.B. (2010) Amyloid light chain deposition associated with dermatofibroma: serendipity or association? *American Journal of Dermatopathology* vol. 32, pp. 298-300.
[57] Shimizu, S., Yasui, C., Yasukawa, K., Nakamura, H., Shimizu, H., & Tsuchiya, K. (2003) Subcutaneous nodules on the buttocks as a manifestation of dialysis-related amyloidosis: a clinicopathological entity? *British Journal of Dermatology* vol. 149, pp. 400-404.
[58] Takayama, K., Satoh, T., Maruyama, R., & Yokozeki, H. (2008) Dialysis-related amyloidosis on the buttocks. *Acta Dermatology and Venereology* vol. 88, pp. 72-73.



Amyloidosis - An Insight to Disease of Systems and Novel Therapies

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Amyloidosis is a benign, slowly progressive condition characterized by the presence of extracellular fibrillar proteins in various organs and tissues. It has systemic or localized forms. Both systemic and localized amyloidosis have been a point of interest for many researchers and there have been a growing number of case reports in the literature for the last decade. The aim of this book is to help the reader become familiar with the presentation, diagnosis and treatment modalities of systemic and localized amyloidosis of specific organs or systems and also cover the latest advancements in therapy.

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