Author's response to reviews

Title: Unusual presentation of anetoderma: case report and brief review of the literature.

Authors:

Shahin Aghaei (shahinaghaei@yahoo.com)
Manoochehr Sodaify (sodaifym@sums.ac.ir)
Fatemeh Sari Aslani (Sariasf@sums.ac.ir)
Nazila Mazharinia (namany03@yahoo.com)

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All of the changes that have been made in the revised version of the manuscript are as following: 1-Title was corrected to: ... brief review.... 2-In abstract/case presentation "lumbar lordosis" and "dental misalignment" were deleted. 3- Replacement of the last sentence in abstract/conclusions with the second sentence in the conclusions section after the discussion was done. 4-The words haematoxylin and fibres consistently (e.g., page 4, line 4 and 4 and 5; page 6, line 10) were written in English style. 5-In Discussion the sentence "The mechanism of anetoderma... autoimmune pathogenesis" was deleted because the latter was discussed at the end of the discussion section. 6-The sentence "Although isolated and perhaps coincidental, these abnormalities could be related to the same process that produces the lesions of anetoderma..." was added to the Discussion part. 7-The Table 1 was displayed at the end of the full text. 8-About the differential diagnosis of anetoderma, several sentences were added to Discussion and according to them the References (12-16), as follows: "The differential diagnosis of anetoderma includes other focal dermal atrophies and miscellaneous diseases that must be differentiated from the skin herniation phenomenon of anetoderma [12], are shown in Table 2. Atrophoderma of Pasini and Pierini is a major source of confusion both etymologically and clinically. Patients have larger lesions with a sharp peripheral border dropping into a depression with no outpouching. On biopsy, elastin is normal, while collagen may be thickened, but this finding is difficult to quantify [12]. Perifollicular atrophoderma is most prominent on the dorsa of the hands and often is associated with multiple basal cell carcinomas and hair abnormalities in the Bazex syndrome [13]. Perifollicular atrophy also has been described in extreme forms of keratosis pilaris, in which large keratin plugs may produce a dilated patulous follicle. This condition usually found on the cheeks of young children. Both of these lesions mimic perifollicular anetoderma but lack elastin changes [12]. In focal dermal hypoplasia thinning or absence of dermis, rather than changes in elastin fibres, accounts for the proximity of the subcutis to the epidermis [12]. Cutis laxa, postinflammatory elastolysis [14], and mid-dermal elastolysis [15] share with anetoderma the property of cryptogenic loss of elastic fibres." 9-The Table 2 was created for differential diagnosis of anetoderma, and shown at the end of the manuscript. 10-The last paragraph about treatment options, the sentence "We could not find similar reports (other than anetoderma-like changes on distal extremities secondary to hamartomatous congenital melanocytic naevi) [4] of anetoderma developing on distal extremities without involvement of the upper trunk and proximal arms, in the medical literature." was transferred to first paragraph of discussion. The "nevi" was corrected to "naevi". 11-In references 20 and 21 only year, volume, and pages of the articles were written. 12- Important references such as Ghomrasseni et al. Am J Dermatopathol 2002; 24: 118-29 and Venencie et al. Br J Dermatol 1997; 137: 517-25 have been cited in the revised version as follows (from the 2nd paragraph, page 6): "Venencie et al. [38], suggested that the degradation of elastic fibres in patients with anetoderma is caused by enhanced expression of progelatinases A and B and production of the activated form of gelatinase A, and that the lack of control of these enzymes by tissue inhibitors of metalloproteinases is probably a key factor in the development and duration of anetodermic lesions. Ghomrasseni et al. [39] demonstrated that for the five samples of anetodermic skin, matrix metalloproteinase-1 (MMP-1) levels were significantly higher compared with the uninvolver cultures and the healthy samples. A significant increase of tissue inhibitors of metalloproteinase (TIMP-1) expression was also observed in the affected cultures of explants. The study demonstrated a significant increase in the production of gelatinase A (MMP-2), and no significant production of TIMP-2 in lesional skin compared with the samples from the two healthy donors." 13-Due to alteration in text, number and arrangement of references were changed. Truly yours Shahin Aghaei