Multiple Flesh-colored Papules on the Face of a 53-year-old Woman
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CASE REPORT
A 53-year-old woman visited our clinic due to many flesh-colored lesions on her cheeks for 3 years. The lesions were not itchy or painful but caused some social embarrassment. Except having moderate facial acne in early adulthood, her medical history was unremarkable. No family member had similar skin problem. On examination, she had multiple 1-3 mm, firm, flesh-colored papules on her cheeks. (Fig. 1) Histological examination of skin specimen from her left cheek revealed bones with lamellar features in the dermis. (Fig. 2) The bone formations encircle spaces containing adipocytes without marrow cells. There was no obvious inflammation in the dermis and the epidermis was relatively normal. Laboratory investigations, including complete blood cell count, serum calcium, phosphorus, alkaline phosphatase and parathyroid hormone were within normal range.

Fig. 1
Numerous small, firm, flesh-colored papules about 1-3 mm in diameter on cheeks.

Fig. 2
Histologically, sheets of compact osseous tissues in the reticular dermis. Bony formation encircles adipose tissue forming a marrow-like cavity. (H&E, 40X)
DIAGNOSIS: Multiple Miliary Osteoma of the Face (MMOF)

DISCUSSION

Multiple miliary osteoma of the face (MMOF) is a form of osteoma cutis presenting as multiple asymptomatic flesh-colored or bluish papulonodules on the face. It has a propensity to occur in women for unclear reasons and no direct evidence shows that hormones play a role in the development of these lesions. It is debating in whether it is a primary form or a sequela of acne vulgaris scars. Acne is now considered by most as a precipitating rather than an etiologic factor, and MMOF is grouped as primary osteoma cutis.1-3

The histologic presentation of MMOF is similar to other osteomas, which have true bones with lamellae and osteocytes. The bones were formed through calcium phosphate mineralization of protein network synthesized by mesenchymal cells. It stains reddish with hematoxylin-eosin, which is different form the deep blue color of cutaneous calcinosis. The bones may encircle adipose tissue, producing a central marrow-like cavity. Osteoblasts, seen along the peripheral margin of the bone, become osteocytes after being included into the bone tissue they produced. Osteoclasts are often absent, and sometimes Haversian canals may be seen.4

The pathogenesis of MMOF is not well known. Either a hamartomatous growth of primitive mesenchymal cells or the metaplasia of matured mesenchymal cells into osteoblasts induced by factors such as osteonectin have been proposed. Dynamic bone studies by Glodminz et al. indicate a high rate of bone remodeling in MMOF, particularly within the internal surface than in the periphery. However, diphosphonate therapy has failed to show any improvement.

Treatment of MMOF is difficult, and multiple modalities have been tried. Topical agents such as tretinoin or adapalene may help with transepidermal elimination of the tiny bone fragments. More invasive procedures include direct punch or scalpel excision with/without pretreatment with dermabrasion for larger lesions.6 Newer modalities utilizing carbon dioxide or erbium: YAG laser may provide more cosmetically acceptable results.7,8 Erbium: YAG laser may have better result because of limited non-additive thermal damage with less pigmentary change or scarring. Superficial soft tissue X-ray examination or ultrasonography can demonstrate the extent and the depth of lesions, which may be helpful for choosing appropriate therapeutic modalities.

REFERENCES