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Recurrent Milia-Like Idiopathic Calcinosis Cutis on the Upper Eyelid

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Dear Editor:

Milia-like idiopathic calcinosis cutis (MICC) is a distinctive type of idiopathic calcinosis cutis, and shows remarkable clinical and histological features. Most cases of MICC appear in children with Down syndrome, but cases of MICC unassociated with Down syndrome are occasionally reported[1]. Herein, we report a rare case of recurrent MICC after complete removal in a patient who had no evidence of Down syndrome.

A 17-year-old healthy Korean boy presented with a solitary whitish papule on the right upper eyelid for several months. Six years ago, he had complete removal of this lesion but the MICC recurred in the same area (Fig. 1). At the time of the patient's arrival at the clinic, physical examination revealed a 5 mm sized firm white papule (Fig. 1B), and it was noted to be similar to the milia that had been there before. His physical and mental development was normal, and he denied any history of previous trauma or dermatosis at the site of the lesion. Also, there were no specific findings in the past history or family history. Histologic examination of the biopsied lesion showed a condensed deposit of basophilic amorphous material within the upper dermis (Fig. 2B). Von Kossa staining showed a black colored reaction (confirmed as calcium) of the lesion, and the serial sectioning did not show the presence of an epidermal cyst. Laboratory findings, including the complete blood count, serum calcium, phosphate, and parathyroid hormone levels, were within normal limits, ruling out the diagnosis of metastatic calcinosis. With all the above findings, we diagnosed the lesion as MICC. After it was removed completely, there has been no recurrence for several months.

MICC appears as smooth, firm, whitish papules resem-

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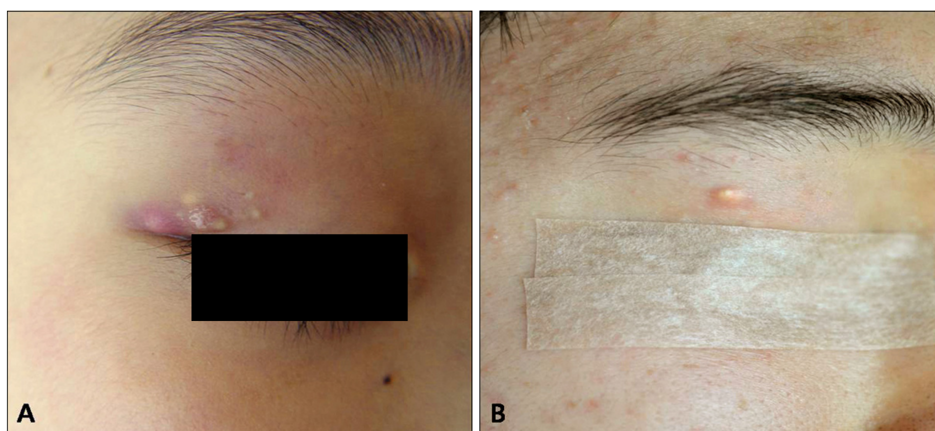

Fig. 1. (A) Several milia-like whitish papules on the right upper eyelid (6 years ago). (B) A solitary, firm, 5 mm sized whitish papule was observed in the same area (at present). The authors are indebted to the patient for his permission for publication.

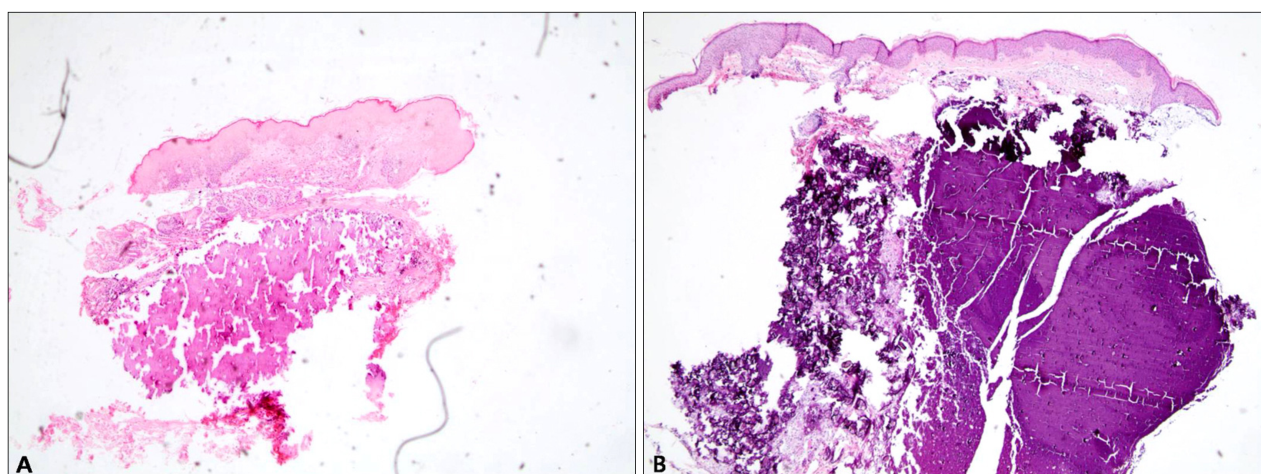

Fig. 2. (A) Amorphous basophilic material surrounded by collagen fibers and fibroblasts in the papillary dermis (H&E, ×40). (B) Amorphous homogenous and basophilic material that stained black with Von Kossa stain in the upper dermis (H&E, ×40).

bling milia, and they are occasionally surrounded by erythema and some have a central crust that indicates transepidermal elimination of calcinosis[2]. So, MICC may clinically be mistaken for warts, epidermal cysts, molluscum contagiosum, and syringomas[3]. Sites of predilection of the disease are the hands and feet, but the involvement of the face has been reported occasionally although it is rare. In addition, most cases of MICC have been reported in children with Down syndrome and these patients may have palpebral or perilesional syringomas occurring simultaneously with the MICC[2].

The pathogenesis of MICC remains unknown, but several theories have been suggested. One is the premature aging process, as in Down syndrome. Cultured fibroblasts of Down syndrome patients contain higher levels of calcium than those of controls[3]. Another theory is that eccrine sweat ducts play a role in calcium deposits, and calcified sweat ducts have been described in several patients[4]. The last theory proposes that the lesions, including the microepidermal cysts, secondarily produce a chronic inflammatory reaction and calcium deposition[2]. In our case, serum levels of calcium and phosphate were normal, and tissue or metabolic abnormalities were not observed. And the patient was a healthy boy, with was no evidence of calcified sweat ducts. So, we consider that this lesion might be calcium deposition due to a chronic inflammatory reaction of an intangible lesion.

In the case presented here, we think that this is the first description of recurrent solitary MICC of the face in a healthy person who had no evidence of Down syndrome or palpebral or perilesional syringoma. So, we announce a rare form of MICC with a review of the published literature, and alert clinical dermatologists to the existence of this rare phenomenon.

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Activation Markers CD63 and CD203c Are Upregulated in Chronic Urticaria

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Dear Editor:
Chronic urticaria (CU) is currently diagnosed clinically when patients weals and the surrounding reflex erythema persist for more than 6 weeks[1]. Autoreactivity of the CU-patients sera can be established in 30% to 50% of patients, depending on the population, through the autologous serum skin test (ASST)[2], the basophil activation test based on upregulation of CD63 (BAT)[3] or basophil histamine release assay (HR), the latter which is now available as a commercialized testing kit in the market. The ASST has only moderate specificity as a marker for functional autoantibodies against the high-affinity immunoglobulin E (IgE)-receptor FcεRIα[2]. Recent research on the field has shown significant upregulation of activation markers CD63 and CD203c on whole blood basophils from CU-patients as well as an up-regulation of expression of the high-affinity IgE-receptor FcεRIα[4]. There is a need for a robust laboratory test for the diagnosis of CU, which can persistently and objectively support the anamnestic diagnosis[1]. The purpose of this study was to confirm the measurement of *in vivo* expression of the activation markers CD63, CD203c and CD123 (interleukin-3 Rα receptor) and the high-affinity IgE-receptor FcεRIα using flow cytometry[4] as an objective complement to anamnestic diagnosis of CU. The study included 8 patients diagnosed with CU, according to the EACCI/GA(2) LEN/EDF/WAO guidelines[1], (mean age 43 years; positive HR test, n=2; negative HR test, n=3; HR test not performed, n=3) and 12 healthy control subjects with a mean age of 48 years. Within 3 hours after venipuncture, 100 μl aliquots of EDTA blood were stained at room temperature for 30 minutes with CD123 PE (BD Pharmingen 340545, 10 μl) and FceRI (Biolegend 334614, 1 μl) in tube 1, CD63 FITC (Biolegend 312004, 5 μl) and CD193 Alexa 647 (Biolegend 310710, 2.5 μl) basophils in tube 2 and CD203c (IOTest PMIM 3575, 5 μl) and CD193 Alexa 647 in tube 3. Samples were hemolyzed and washed with phosphate buffered saline. Data was acquired on a FACS Canto II (Beckton-Dickinson, San Jose, CA, USA) flow cytometer for 4 minutes at medium speed in order to obtain absolute cell numbers from the constant volume sampled[5]. Performance of the flow cytometer was moni-

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