Case 25
A 20-year-old male from Khon Kaen
Chief complaint: A 2-year history of slowly progressive brownish lesions on trunk and neck


Fig. 25.1

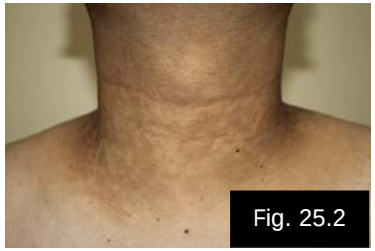
Fig. 25.2

Present illness:
The patient presented with slowly progressive asymptomatic reticulated brownish plaques on abdomen and back for 2 years. The lesions first appeared on the interscapular area then extended to abdomen, back, upper chest and progressed to neck in 1 year later. He denied previous taking of any medications or history of rapid weight gain. He also denied of previous treatment.

Past history: He had no underlying disease.

Family history: His elder brother had the same lesions on groin area.

Physical examination: Other systemic examination revealed no abnormality.

Dermatologic examination: Multiple ill-defined reticulated brownish thin plaques on back, abdomen, upper chest, and neck (Fig. 25.1-2)

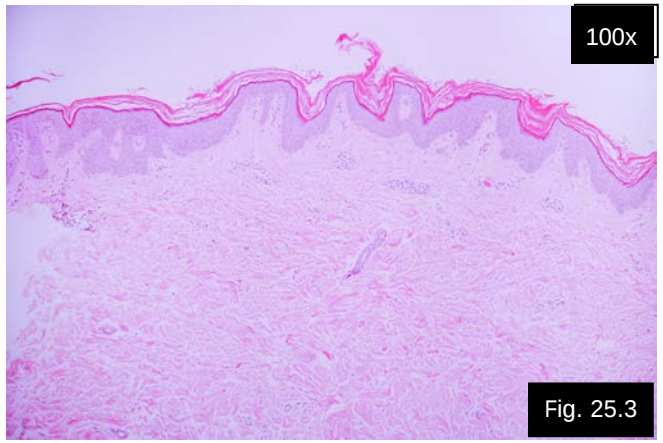

Fig. 25.3

Histopathology: (S18-3817, Abdomen)
- Papillomatosis, epidermal hyperplasia with hyperkeratosis (Fig. 25.3)

Laboratory investigations:
- KOH: Negative for yeast or hyphae

Diagnosis: Confluent and reticulated papillomatosis of Guogerot and Carteaud (CARP)

Treatment:
- Oral doxycycline 200 mg daily
- Topical 10% lactic acid cream apply once daily
- Topical 0.025% tretinoin cream apply twice daily

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Discussion:

Confluent and reticulate papillomatosis (CRP) is an uncommon condition that was described in 1927 by Gougerot and Carteaud[2] as unnamed pigmented papillomatosis. Clinically, CRP lesions initially appear as 1-2 mm erythematous papules that typically occur on the intermammary, interscapular, and epigastric regions. Subsequently, lesions can enlarge to 4-5 mm and develop a brownish hue. These papules coalesce and become confluent centrally and reticulated at the periphery. Eventually the chest, neck, shoulders, and back are involved. The cheeks, pubic area and forehead rarely are affected[1,3].

The pathophysiology of CRP is poorly understood; two prominent theories suggest an abnormal host response to Malassezia furfur or abnormal keratinization. The former theory was primarily thought that Malassezia spores were sometimes prevalent within areas of the involvement which occasionally response to antifungal therapy[4]. The majority of cases, however, demonstrate no evidence of M. furfur proliferation. Abnormal keratinization has also been postulated to be the pathogenesis of CRP due to the findings of immunohistochemical and transmission electron microscopic studies, which they show an increased expression of Ki-67, involucrin, and keratin 16[9]. Other explanations include a reaction to bacterial infection, photosensitivity, endocrinopathy, amyloidosis cutis and genetic predisposition There are also a recently reported case of CRP associated with polycystic ovarian syndrome which possibly occur more commonly in this patient population.

The differential diagnosis is made mostly with acanthosis nigricans and pityriasis versicolor[5,6,7]. Acanthosis nigricans cannot be reliably distinguished from CRP based on the presence of increased body habitus or a history of insulin resistance, as these comorbidities may coexist with CRP. Clinical clues that more consistent with CRP would be the presence of peripheral reticulation and the absence of mucosal and nail involvement. Histopathological findings show mild hyperkeratosis, papillomatosis, and focal acanthosis which indistinguishable from acanthosis nigricans. Because of their similar and overlapping clinical features, some authors consider CRP and acanthosis nigricans as one spectrum of the same entity[6].

The main treatment options for CRP are oral antibiotics[8]. Other reported treatment modalities include oral retinoids, topical salicylic acid, hydroquinone, tacrolimus[10], antifungals[11] and 5-fluorouracil.

In our patient was treated with topical 10% lactic acid, topical 0.025% tretinoin cream and oral doxycycline 200 mg daily. One month after these treatments, the lesions show partial improvement.

References

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