## CASE 20

A 65-year- old civil servant form Chonburi

**Chief complaint:**

Edematous, brownish peau d' orange plaque overlying both anterolateral tibias for 6 months.

**Present illness:**

The patient presented with localized brownish and thickened infiltrative plaques accompanied with edema on both lower legs for 6 month duration. The lesion initially involved both pretibial areas, but then gradually progressed to the lateral aspect of both legs. They were asymptomatic, however developed secondary impetiginization on one occassion. This resolved after treatment with Augmentin 625 mg three times per day for 2 weeks.

**Past history:**

She has several underlying medical conditions consisting of long standing diabetes mellitus type 2 and hypertension, which lead to chronic renal insufficiency, proven by ultrasound of KUB system. She also has old lacunar infarction, again possibly a macrovascular complication of her diabetes and hypertension.

**Physical examimation :**

| | |
|---|---|
| Vital signs: | Pulse 60/m regular rhythm, RR 20/min,BP 150/90 mmHg |
| GA : | An elderly Thai woman, afebrile, alert |
| HEENT: | mildly pale, no jaundice, no exophalmos, no opthalmoplegia, thyroid gland not enlarged |
| LN: | not palpable anywhere |
| Heart & Lungs : | normal |
| Abdomen: | slightly distended , liver is just palpable, spleen is not palpable, shifting dullness is negative |
| Extremities: | no varicose veins, pitting edema 2+, no tremor |
| DTR: | 2+ all with normal relaxation |

Skin: well-demarcated , thick,infiltrative, brownish verrucous plaques with peau d' orange surface, overlying both anterolateral tibias

Nails: no acropachy

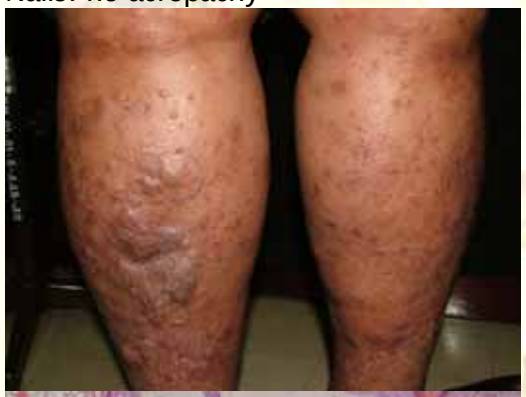

Fig. 20.1

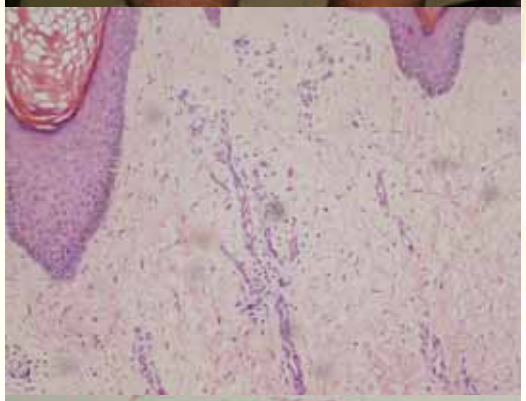

Fig. 20.2

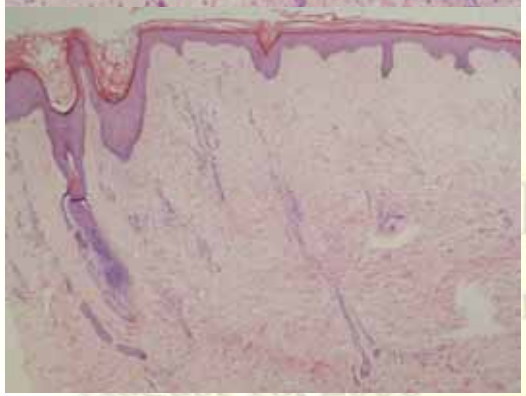

Fig. 20.3

**Histopathology:** S 06-09075

Mild hyperkeratosis and epidermal hyperplasia.Diffuse edema of the entire dermis. Sparse lymphocytic infiltrate and scattered fibroblast in the dermis.

The section was further sent for special staining alcian blue pH 2.5 and toluidine blue. The results are pending.

**Laboratory findings:**

- CBC: Hb 11.1 g/dl, Hct 33.8 vol%, normochromic normocytic, WBC 4,360 / $mm^3$, Plt 167,000/$mm^3$
- Bl. Chem: BUN 46 mg/dl, Cr 2.9 mg/dl, Serum albumin 33.9 g/dl
- TFT: FT3 1.09 pg/ml ( 2.57-4.43), FT4 0.81 ng/dl (0.93-1.17), TSH 2.19 uIU/ml (0.27-4.20)
- Microsomal Ab (Anti-TPO): negative
- Antithyroglobulin: 18.25 IU/ml (0-115.0IU/ml) (Compatable central hypothyroidism)

**Diagnosis:** Pretibial myxedema, Central Hypothyroidism
**Presenter:** Kumutnart Prabudhanitisarn
**Consultant:** Penpun Wattanakrai

**Discussion**

Pretibial myxedema refers to localized thickening of the pretibal area due to accumulation of acid mucopolysacharides, mainly hyaluronic acid[1]. The same process can be demonstated in the dorsum of hallux, lower abdomen, arms, shoulders, neck and pinna, hence, unusual location can occur[2]. It is an infrequent manifestation of autoimmune thyroiditis, particularly Graves' disease, occuring 1-10% of these patients. Pretibial myxedema is considered one of the triad of Graves'disease, consisting of exophthalmos, acropachy and pretibial myxedema[3]. However, there are several reports of euthyroid pretibial myxedema and pretibial myxedema associated with other conditions such as chronic stasis dermatitis and chronic obesity[4]. A recent demographic report of 178 patients with pretibial myxedema revealed that 91% were classified as hyperthyroid, 3.9% hypothyroid, 2.8% euthyroid and 2.2% were initially hypothyroid and subsequently turned to hyperthyroid during follow up[5].

There are many clinical variants of pretibial myxedema. In a large series, a review of 150 cases, revealed that 58% had non-pitting edema, 21% had plaques, 20% had nodules, 7% had polypoid or elephantatic type lesion. Lesions varied in color from flesh to purple[6].

The definite etiology of pretibial myxedema is unclear. It was demonstrated that pretibial myxedema patients have increase hyaluronic acid production by cultured fibroblast from pretibial area. Thyroid stimulating hormone (TSH) receptors have been indentified in fibroblasts from pretibial and orbital regions of patients with Graves' disease. However, there were no obvious correlation between clinical manifestation and the presence of TSH receptor antibody[7]. Moreover, this certainly does not explain the presence of myxedema in euthyroid and hypothyroid patients. Another study demonstrated that pretibial myxedema patients have circulating immunoglobulin $A_2$($IgA_2$) fibroblast antibody capable of binding to dermal fibroblast cell line[8].

Histologic finding in pretibial myxedema shows thickened dermis especially in mid and deep dermis with extensive deposit of acid mucopolysaccharide.

Treatment includes observation in mild form[5]. Topical glucocorticoids, with or without occlusive dressing, or intralesional glucocorticoid injection may be useful. Intravenous immunoglobulin, plasmapheresis and surgical therapy have all been tried. New therapy with octreotide, insulin analog (that supresses TSH receptor like growth factor activity) and gradient pneumatic compression have been used with variable results.[1,9]

## References


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