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Letters to the Editor

Without endothelial proliferation UNT has been reported to be associated with hyperestrogenemic states such as puberty, pregnancy and alcoholism.[2-5] This is explained by the hypothesis that a humoral agent, probably estrogen, stimulates the end target organs that are distributed congenitally in a dermatomal pattern in order to produce telangiectasia.[2]

There are a few cases of UNT with serologic evidence of hepatitis C supporting the hormonal theory, which is based on hyperestrogenemic state, but this theory does not apply in all UNT cases.[6,7] Cases of UNT occurring in the presence of normal serum estrogens and liver function are documented in literature.[8-10] Tok et al. reported a case of UNT related to pregnancy with no estrogen and progesterone receptors, and suggested that the receptor assays were not sensitive enough to detect estrogen and progesterone levels in the skin when compared with the breast tissue.[11]

It is known that normal skin contains low levels of estrogen receptors and it is difficult to explain the lack of estrogen receptors in case of increased circulating estrogen.[12] Although we were not able to report the serum estrogen and progesterone levels of our patient, the lack of estrogen and progesterone receptors supports the theory that physiologic hormonal or hyperestrogenemic states do not always apply for the etiopathogenesis of the UNT. It was suggested that UNT might result from a somatic mutation in the presence of normal hormonal status,[2] but further data need to be supplied for its pathogenesis.

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Eruptive lichen planus in a child with celiac disease

Sir,
A 6-year-old boy had diarrhea and abdominal distension since the age of 1 year. He was investigated and was found to have celiac disease. His complete blood count, liver and renal function tests and fasting blood sugar were within normal limits. His IgG anti-gliadin antibody and IgA anti-transglutaminase antibodies were negative, and duodenal biopsy showed evidence of celiac disease (Marsh grade 2). He was started on gluten-free diet and his celiac disease improved. Two months before seeking dermatology consultation, he started developing showers of itchy violaceous papules all over his body. He was not on any active medication for celiac disease except the gluten-free diet. On examination, he had multiple violaceous monomorphic papules all over with evidence of koebnerization [Figure 1]. A punch biopsy from one of the papules showed classical changes of lichen planus (LP). He was given 20 mg prednisolone per day orally with which the skin lesions healed within 3 weeks.

Celiac disease is a chronic T-cell-mediated enteropathy against ingested gluten in genetically predisposed
Unlike previously thought, recent epidemiological studies have shown celiac disease to be a common disorder affecting 1 in 250 persons in the US and UK populations. It has been associated with HLA-DQ2, expressed in more than 80% of patients or HLA-DQ8.

Many extra-intestinal associations have been observed in celiac disease patients, including dermatological manifestations. Most of the dermatological manifestations are secondary to nutritional deficiency. A few autoimmune dermatological manifestations have also been described, most important among which is dermatitis herpetiformis. Others include linear IgA bullous disease, vitiligo, alopecia areata, dermatomyositis, etc. However, there is only one report of a single case of erosive oral LP with celiac disease.

It is surprising that association of cutaneous LP and celiac disease has never been reported previously, although both are not very uncommon and are T-cell-mediated autoimmune disorders. Perhaps, the different genetic linkages of LP (HLA-DQ1) and celiac disease (HLA-DQ2 and HLA-DQ8) may explain this. This report will increase awareness of this association amongst dermatologists and pediatricians so that more such instances may be identified in the future.

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Figure 1: Violaceous papules with koebnerization over dorsum of hand