Kyrle Disease
Kyrle's disease is a rare disorder, described by Kyrle in 1916. There is controversy as to whether it represents a...
This eruption presents with a large number of papules, some coalescing into plaques, numbering in the hundreds and often are found in the vicinity of these lesions. Linear lesions related to possible koebnerization have been described.
Histopathology

The essential histopathologic findings include (a) a follicular or extrafollicular cornified plug, (b) keratinocytes displaying marked acanthosis, dyskeratosis, and/or vacuolization extending to the basal cell zone, (c) absence of demonstrable collagen and elastin, (d) irregular epithelial nuclei, (e) perforating elastic fibers stains to exclude perforating elastic fibers, as in elastosis perforans serpiginosa, or collagen fibers, as in RPC.
**Histogenesis**

The primary event is claimed to be a disturbance of epidermal keratinization characterized by the formation of dyskeratotic foci and acceleration of the process of keratinization. This leads to the
formation of keratotic plugs with areas of parakeratosis. Because the rapid rate of
differentiation and keratinization exceeds the rate of cell proliferation, the parakeratotic column
gradually extends deeper into the abnormal epidermis, leading in most cases to perforation of
the parakeratotic column into the dermis. Perforation is not the cause of Kyrle's disease, as
originally thought, but rather represents the consequence or final event of the abnormally
sped-up keratinization. This rapid production of abnormal keratin forms a plug that acts as a
foreign body, penetrating the epidermis and inciting a granulomatous inflammatory reaction. A
certain similarity exists between the parakeratotic column in Kyrle's disease and that observed
in porokeratosis of Mibelli. In both conditions, a parakeratotic column forms as the result of
rapid and faulty keratinization of dyskeratotic cells, but, whereas in Kyrle's disease the
dyskeratotic cells are often used up so that disruption of the epithelium occurs, the clone of
dyskeratotic cells can maintain itself in porokeratosis Mibelli by extending peripherally