Factitial dermatitis, also known as dermatitis artefacta, consists of self-inflicted cutaneous lesions. It was first described in 1951 by Asher. Factitial dermatitis may be associated with borderline personality disorder, post-traumatic stress disorder, and eating disorders. It is more common in women. The reported clinical morphology is diverse and includes erosions, blisters, ulcers, erythema, and ecchymoses. Lesions are usually multiple, are located in sites accessible to the patient, and may demonstrate bizarre or geometric configurations. Methods of injury include excoriation, thermal burn, application of topical caustics, blunt trauma, and foreign body injection. When confronted, patients usually deny any role in inducing the skin lesions. Bullous factitial dermatitis has been described secondary to aerosol spray, garlic burn, and curling iron injury.

Herein, we report a case of bullous factitial dermatitis with potentially misleading histopathology. A 35-year-old female presented with a one-day history of sharply demarcated, rectangular erythematous plaques on her flanks, lower back, right arm, chest and thighs (Figs. 1, 3 & 4). One plaque included a large, tense blister containing serous fluid. A biopsy demonstrated vacuolar interface dermatitis with copious eosinophils and superficial epidermal necrosis accentuated at follicular ostia (Fig. 5). There were numerous multinucleated giant cells in the epidermis with up to 16 nuclei/cell (Figs. 2 & 6). The nuclei possessed prominent nucleoli and nuclear membranes with heterochromatic nucleoplasm. Viral inclusions and chromatin margination were absent. Immunohistochemistry for herpes simplex virus was negative (Fig. 7). There were no cutaneous immunoreactants identified by direct immunofluorescence. The patient was admitted to the hospital for observation and there was no progression of the lesions overnight, and the patient requested discharge the following morning. She reported complete resolution at 7 days with use of clobetasol ointment twice daily. Further clinical investigation revealed a history of anxiety and depression. A diagnosis of factitial dermatitis was made based on the geometric configuration of her lesions, her psychiatric history, the presence of superficial epidermal necrosis, the observed spontaneous improvement, and lack of laboratory evidence of infection or autoimmune reaction. At two months, the patient had no new or...
Fig. 2. A biopsy revealed numerous multinucleated keratinocytes within the epidermis.

recurrent lesions and continued to deny any role or knowledge in the induction of the lesions.

Epidermal multinucleated giant cells may be misconstrued as specific evidence of a viral exanthem. The pathologist who initially reviewed our patient’s biopsy suspected herpetic dermatitis. Instead, correlation with the clinical context led to the diagnosis of bullous factitial dermatitis. We posit that epidermal multinucleated giant cells may represent a consequence of a variety of mechanical, physiologic, and infectious insults and can be a false clue to viral infection. Bi- and trinucleate keratinocytes have been described in a broad variety of non-neoplastic dermatoses, but epidermal multinucleated giant cells with more than eight nuclei are uncommon, in our experience. Epidermal multinucleated giant cells can be seen with increased frequency in pruritic cutaneous diseases. They are also described in active esophagitis, particularly secondary to gastroesophageal reflux disease. These findings suggest to us that external mechanical or caustic stressors can induce keratinocyte multinucleation. In our patient’s case, the superficial epidermal necrosis, eosinophilia, and accentuation of necrosis around follicular openings, where an externally applied agent might be more concentrated, suggested that an external allergic or irritant caustic had been applied to the skin.

Multinucleated giant cells can be formed via cell-cell fusion or by failure of cytoplasmic splitting during the last phase of mitosis. Multinucleation in benign and reactive conditions is usually a result of cell-cell fusion, whereas multinucleation in malignancies often reflects imperfect cell division, or, more colorfully, mitotic catastrophe. Herpesviruses trigger production of viral glycoproteins in infected cells that promote cell-cell fusion. Labeled cell cultures of giant cell tumor of tendon sheath have shown an admixture of nuclei within giant cells, suggesting the giant cells are formed via fusion of pre-existing mononuclear cells. Additionally, cultures of human blood monocytes form giant cells in response to gamma interferon. These monocyte polykaryons are physiologically less active than their mononuclear compatriots.

The mechanism of formation of epidermal multinucleated giant cells in non-viral skin diseases remains unknown. Kimura and Hatano have suggested that dyskeratotic tonofilaments become entangled with the spindles of the mitotic apparatus, preventing proper cell division. We believe the multinucleated giant cells in our case are more likely due to cell-cell fusion than mitotic failure. A lack of MIB-1 (Ki-67) reactivity in our case provides indirect evidence in favor of cell-cell fusion rather than mitotic error (Fig. 8). Histopathologists should be aware of potential viral mimicry in cases of factitial dermatitis with prominent multinucleated giant cells to ensure diagnostic accuracy and to avoid unnecessary medical treatment.

Figs. 3 and 4. There are rectangular erythematous plaques across the back and left posterior shoulder.

Fig. 5. Necrosis of outer ostial follicular epithelium was noted, as was an eosinophilic infiltrate.

Fig. 6. In another area, the biopsy demonstrated an interface dermatitis with copious eosinophils and epidermal multinucleated giant cells.

Fig. 7. Immunohistochemistry for herpes simplex virus yielded a negative result.

Fig. 8. The multinucleated giant cells were MIB-1 nonreactive.

References
6. Friedman T, Shalom A, Westreich M. Self-inflicted garlic burns: our experience and
Cover Quizlet


