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Osteoporosis in Pemphigus: Steroids Are Not the Only Story

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To the Editor,

I read with interest the recent study by Kaya et al. (Dermatol Pract Concept. 2025;15(2):5050), on bone mineral density (BMD) in pemphigus vulgaris (PV) patients receiving glucocorticoids. While the topic is clinically relevant, several methodological concerns warrant caution in interpretation.

Most notably, the absence of a control precludes causal attribution of observed BMD reductions to glucocorticoids, especially since PV itself is an inflammatory state linked to cytokine-driven bone loss independent of steroid exposure, likely via chronic inflammation and elevated cytokine levels such as IL-6 and TNF-α that stimulate osteoclastogenesis and impair bone formation [1,2]. Inflammatory mediators like RANKL, IL-17, and TNF-α have been implicated in both the pathophysiology of PV and the development of osteoporosis, emphasizing the need to account for disease-driven bone loss when evaluating treatment-associated effects [3,4].

The study also omits disease severity scores (e.g., PDAI) and autoantibody levels (anti-Dsg1/3), both of which can correlate with inflammatory burden and may influence BMD. Emerging data suggest that incorporating these parameters enhances the predictive value of fracture risk assessment tools and allows earlier identification of patients who may benefit from anti-resorptive therapy. Categorizing steroid exposure by daily dose alone, rather than cumulative or prednisone-equivalent dose, likely oversimplifies treatment impact [5].

The study's bone health assessments rely only on lumbar and femoral T-scores, neglecting important adjuncts such as trabecular bone score (TBS), vertebral fracture assessment (VFA), or bone turnover markers. These tools offer deeper insight into bone microarchitecture and remodeling status, particularly useful in PV patients, who may have discordantly normal BMD but impaired structural integrity. I also note the absence of fracture data and lack of application of validated prediction tools like FRAX®, despite their utility in determining real-world fracture risk and guiding therapy. Given that fracture, not merely low BMD, is the clinically meaningful endpoint, this is a missed opportunity to link structural bone changes with functional consequences.

Furthermore, while the authors note a significant increase in vitamin D levels post-treatment, the absence of baseline deficiency prevalence, seasonal variation data, or compliance monitoring renders this finding difficult to interpret. Other

key confounders, such as smoking, dietary calcium intake, sun exposure, physical activity, and concurrent medications, were also not accounted for.

While this work highlights a key clinical concern, future studies would benefit from incorporating disease activity, broader skeletal assessments, and a matched comparator group to better define osteoporosis risk in PV.

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