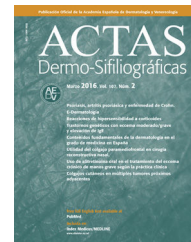




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RESIDENT'S FORUM

FR- Risk of Nonmelanoma Skin Cancer in Patients on Hemodialysis[☆]



FR- Riesgo de cáncer cutáneo no melanoma en pacientes hemodializados

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Wang et al.¹ recently published an interesting article in the *British Journal of Dermatology* on the risk of skin cancer in patients on chronic hemodialysis. That study found an increased risk of skin cancer in patients undergoing chronic hemodialysis of 1.58 compared to that of the general population. The authors mainly attribute this increased risk to the increase in inflammatory biomarkers caused by the uremic

pruritis suffered by these patients. This situation is analogous to the association between inflammatory bowel disease and colorectal cancer. It was also found that uremia can lead to the impairment of DNA repair,² a finding that had been published 4 years earlier on an initial review of their database. In their database, this risk is higher in patients aged between 20 and 39 years and falls gradually with age.

We would like to report the experience of our research group, which, in 1995, published a multivariate analysis of aging markers in patients on hemodialysis.³ In this study, we were able to show that hemodialysis is a technique that accelerated skin aging in our patients with chronic kidney disease. In our patients, we found reduced hydration of the stratum corneum, increased Favré-Racouchot

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senile elastosis with cysts and comedones, and increased actinic keratosis independent of age. The sample size did not allow us to establish a statistically significant link with basal cell carcinoma. Nevertheless, all signs of photoaging found in our patients presented a link with the duration of hemodialysis. Renal failure thus produces accelerated pathologic aging. The prognosis for patients with chronic kidney disease continues to be extremely poor and remains so despite improvements in treatment options. We therefore hypothesize that immunosuppression is age-related and therefore affected by hemodialysis.⁴

White et al.⁵ recently corroborated the similarity between the physiological aging process and hemodialysis in patients with chronic kidney disease. The principal molecular mechanisms affected in both processes and which lead us to believe that there is a crossover between them include impaired proteostasis, mitochondrial dysfunction, post-translational protein modification, and senescence and telomere shortening. We agree that uremia is the key process in the pathophysiology of this accelerated aging process and induces increased apoptosis, necroptosis, and autophagy, and that dialysis may even accelerate the processes of apoptosis per se.

In light of these arguments, we believe that skin cancer in patients on hemodialysis must also be considered as an aging

marker and the increased incidence due to the inflammatory cascade caused by the uremia should not be considered as the sole causal factor. It is part of a multifactor process that is more complex and similar to the physiological aging process, where oxidative stress is one more factor to take into account.

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