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Bicalutamide: A Novel Treatment of Seborrheic Dermatitis?

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Several treatments, both local and systemic, are available for seborrheic dermatitis (SD), but, invariably, the disease relapses once they are discontinued.

A patient of mine, an 82-year-old male, with chronic SD, was diagnosed as having a non-metastatic prostate cancer and given 150 mg daily bicalutamide. He told me that his prostate-specific antigen (PSA) was reduced from 26 to 0.31 ng/ml in 4 months. Side effects were loss of libido, impotence and a modest gynecomastia. He did not notice any improvement of his baldness, allegedly expected given the effect of bicalutamide on androgen receptors. His SD had, instead, fully cleared. At first, I attributed it to the seasonal improvement SD patients usually enjoy in summertime, but, when SD did not relapse in September and kept to be absent even in December to the following April, I had to conclude that its disappearance was due to bicalutamide.

In July, PSA started to rise again, progressively reaching 3.85 ng/ml in two months. The oncologist decided that bicalutamide was unable to stop the cancer progression and prescribed trimestral leuprorelin acetate 375 mg plus bicalutamide 75 mg/day. Testosterone blood levels that, before leuprorelin was at normal levels for a man, decreased to 0.835 ng/ml and PSA to 1.75 ng/ml in one month. However, the patient noticed a modest reappearance of SD.

Bicalutamide is a nonsteroidal competitor for androgen receptors. It is used in the non-metastatic prostate cancer either in a combined androgen blockade strategy (50 mg daily plus trimestral gonadotropin hormone-releasing hormone (GnRH) analogue) or as a daily monotherapy (150 mg/d). Bicalutamide reduces PSA levels in a dose-related way, 150-200 mg daily being the optimal dose, equivalent to castration in terms of survival in patients with non-metastatic prostate cancer [1].

Leuprorelin acetate is instead a GnRH analogue that acts as an agonist on pituitary GnRH receptors. After an initial transitory increase, testosterone blood levels drastically fall.

Because of its peripheral antiandrogen effect, bicalutamide apparently reduced in my patient the sebum production and Malassezia population. Regrettably, I did not measure the two parameters before the treatment and cannot support my hypothesis by robust laboratory data. However, the failure of SD to relapse in winter, never observed before by my patient, can be a good criterion of its efficacy. Its decreased dosage

after the introduction of leuprorelin may explain the slight relapse of SD. What is less explainable is why, though decreasing testosterone levels, leuprorelin failed to counteract SD. It is likely that androgens of adrenal origin, only partially inhibited peripherally by the lower dosage of bicalutamide, may continue to stimulate sebaceous glands.

The observation has only a theoretical importance although it may be used as an indirect measure of testosterone activity and of bicalutamide efficacy on the cancer progression. Lamentably, leaving the side effects apart that prevent it to be used in youngsters, bicalutamide is an expensive drug and I doubt that it may be even accepted among the routinary dermatological treatments for a fastidious but after all mild disease like SD.

Reference

1. Kolvenbag GJ, Nash A (1999) Bicalutamide dosages used in the treatment of prostate cancer. Prostate 9: 47-53.