Lichen Sclerosus in a Breast Cancer Survivor

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We thank Dr. Vieira-Baptista for his letter, which underscores one key point made in our clinical vignette: more research about the causes and management of lichen sclerosus is necessary.

Lichen sclerosus can be insidious or aggressive;[1] therefore, we disagree with the assertion that our patient's disease must have been present prior to the initiation of the aromatase inhibitor.

We do not believe that Dr. Vieira-Baptista's arguments contradict our suggestion that women may be more susceptible to developing lichen sclerosus in exaggerated hypoestrogenic states, as seen with aromatase inhibitors. The bimodal distribution of the disease (in prepubescent and post-menopausal women) actually corroborates the possibility of association, as estrogen levels will be low in both of these groups.

We agree that lichen sclerosus, once established, does not respond to administration of exogenous estrogen (hormone replacement therapy) in postmenopausal women, as we reported in our article. It is therefore not surprising that lichen sclerosus symptoms are also not ameliorated by endogenous estrogen increases seen during pregnancy, menstrual cycle fluctuations, and puberty. However, these observations do not refute the idea that hypoestrogenemia is associated with initial development of the disease; in fact, epidemiological research recently published by Drexel University continues to support this association.[2]

That said, it was not our intention to assert that hypoestrogenemia is the only risk factor for the development of lichen sclerosus. We maintain that the causes of lichen sclerosus are not well understood, and several risk factors have been associated with it, including autoimmunity. Our clinical experience and anecdotal observations of peers suggest an increased incidence in women taking aromatase inhibitors, and we chose our case to present this hypothesis. We hope that more research will be conducted to determine the validity of this theory. It is possible that aromatase inhibitors contribute to the development of lichen sclerosus via an unknown mechanism unrelated to hypoestrogenemia. It is also possible that hypoestrogenemia is a confounding risk factor contributing to some of the other causes mentioned both in our article and in Dr. Vieira-Baptista's letter.

Our intention in the publication of this article was to generate discussion about lichen sclerosus and to encourage additional research on its causes and treatments. We maintain, until further research is published, that patients prescribed aromatase inhibitors should undergo annual vulvo-vaginal examinations to rule out lichen sclerosus and other hypoestrogen-related problems.

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REFERENCES

1. **Pugliese J, Morey A, Peterson A.** Lichen Sclerosus: Review of the Literature and Current Recommendations for Management. J Urol. 2007;178:2268–2276.
2. **Nyirjesy P, Leigh RD, Mathew L, Lev-Sagie A, Culhane JF.** Chronic vulvovaginitis in women older than 50 years: analysis of a prospective database. J Low Genit Tract Dis. 2012;16(1):24–9.

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