Long-Term Statin Use and Risk of Breast Cancer - Letter

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Running Title: statin and gynecological malignancies: unresolved dilemma.

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Dear Editor,

we read with great interest the work of McDougall et al. recently appeared on your journal reporting that current users of statins for 10 years or longer had a 1.83-fold increased risk of invasive ductal carcinoma (IDC) and a 1.97-fold increased risk of invasive lobular carcinoma (ILC) compared with never users. Among women diagnosed with hypercholesterolemia, current users of statins for 10 years or longer had more than double the risk of both IDC and ILC compared with never users. (1)

More recently Desai et al. reported in your journal contrasting data obtained by Cox proportional hazards analyses conducted on large scale population from the WHI study (154587 post-menopausal women with 7430 breast cancer (BC) pathologically confirmed) concluding that statins were not associated with BC risk. (2)

A recent review analyzes as well the conflicting results about chemopreventive statin use in BC population concluding that even if their effects are modest, the overall good long-term tolerability and relative low-cost could make them new attractive chemopreventive agents. (3)

Considering both the increased statin use over the past few decades and the high BC incidence, morbidity and mortality, it is mandatory to well define the exact role of statins in BC risk and recurrence without underestimating the potential implications in ovarian and endometrial tissues. Because of the common hormone dependent origin and similarities in etiological factors, gene expression profiles, tumorigenic mechanisms, pathological changes and metastatic characteristics, it would be important to evaluate also the effects on ovarian and endometrial tissue. In our knowledge, only one study was properly designed to analyze “Statin use and female reproductive organ cancer risk in a large population-based setting”. Authors suggested that there is a non-
significant reduced risk of endometrial and ovarian cancers among statin users compared with nonusers. (4)

As well as was discovered Raloxifene antiproliferative effect on endometrium during the monitoring of breast antiblastic effects (making it a possible candidate to substitute tamoxifene), something similar could happen on monitoring statins effects on hormone dependent gynecological malignancies. (5)

To solve the dilemma an answer to the lingering questions regarding the association between gynecological cancer risk and statin use it will be necessary a meta-analysis combining the existing large scale studies and further perspective ones. In the same way, further perspective studies, even if conducted on BC and statins, should consider also outcomes about ovary and endometrium in order to increase the amount of data available in this field.

Conflict of interest

All Authors declare that they have not conflict of interest.
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References


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