Graziottin A.
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Abstracts of the 13th World Congress on the Menopause, Roma, Italy, June 8-11, 2011, Climacteric, vol. 14, suppl. 1, June 2011, p. 215

Menopause, inflammatory markers and pain: what is new

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Background

Is menopause (also) an inflammatory condition? And what is the impact of fluctuations and loss of estrogens on local and systemic inflammatory/degenerative processes? Mast-cells are the director of the inflammatory and pain orchestra: what is the link then between sexual hormones and mast cells activity? Abundant evidence clearly demonstrates that women are at substantially greater risk for many clinical pain conditions, and there is some suggestion that postoperative and procedural pain may be more severe among women than men. Current human findings regarding sex differences in experimental pain indicate greater pain sensitivity among females compared with males for most pain modalities, including more recently implemented clinically relevant pain models such as temporal summation of pain.

Aim of the presentation

To analyze current evidence on the association between menopause, increase of inflammatory molecules and pain.

Method

Review of the literature and clinical experience.

Results

Estrogens fluctuations are credited to trigger mast cells degranulation, increasing the release of inflammatory molecules. This contributes to local and systemic vulnerability to pain, at least in the subset of women who report perimenstrual flares of pain, and worsening of pain syndromes during the climacteric. During and after the menopause, a definite increase in inflammatory markers has been documented in osteoarthritis (that triples after the menopause), in bone resorption leading to osteopenia /osteoporosis, in cardiovascular diseases and aging, in obesity, in dyspareunia, and also in depression, until recently considered a non inflammatory condition. New evidence indicates that approximately one-half of the women with depression reported pain of mild intensity. Pain intensity was significantly correlated with the severity of depression ($r^2 = 0.076$; $P = 0.04$) and tended to be correlated with the severity of anxiety, ($r^2 = 0.065$; $P = 0.07$), and the number of depressive episodes ($r^2 = 0.072$; $P = 0.09$). Other researches re-read depression as a systemic disease and a systemic inflammatory conditions, where flooding of the brain with inflammatory molecules may contribute to the complex depressive symptomatology.

Conclusions

Increase of inflammatory molecules and pain is documented in different conditions that worsens across and after the menopause. The presentation will focus on the new evidence of menopause as an inflammatory disease and on the relationship between sexual hormones changes, mast cell activity, inflammation and pain.