Role of 5 α -reductase inhibitors in modulation of the analgesic effects of morphine: an emerging concept in pain management

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To the Editor,

I read with great interest the recent article by Sharif et al. [1] in a recent issue of your esteemed journal. The article is highly thought provoking. Interestingly, the past few years have seen the emergence of a number of novel new agents that alter 5 α -reductase function and thereby modulate the analgesic effects of morphine.

For instance, Moradi-Azani et al. [2] have recently demonstrated that 5 α -reductase is a modulator of formalin-induced tonic pain in animal models. Central nervous system activity of 5 α -reductase is augmented by administration of morphine [3]. Not surprisingly, recent studies indicate that finasteride, as 5 alpha-reductase inhibitor, can influence and alter the perception of allodynia. Finasteride exerts its anti-nociceptive effects by augmenting the pain reducing effects of morphine and by exerting an inhibitory effect on local inflammation [4]. Finasteride also mitigates the development of opiod-induced tolerance.

Verdi and Ahmadiani [5] have recently demonstrated that naloxone-induced withdrawal symptoms can be inhibited by a single injection of finasteride. Clearly, finasteride holds significant potential in attenuating the risk of morphine dependence and decreasing the intensity of morphine withdrawal syndrome.

The above examples clearly illustrate the close relationship between finasteride, 5 α -reductase function and morphine pain modulation. Further large scale studies are needed to fully elaborate and harness the possible beneficial role of finasteride in pain modulation and morphine dependence.

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