Letter to the Editor

Comment on “Accentuation of Tumor Growth Secondary to Morphine Administration: The Proneoplastic Role of Morphine besides Its Role in Pain Management”

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I read with great interest the recent article by Luk et al. in a recent issue of your esteemed journal [1]. The article is highly thought provoking. Interestingly, the past few years have clearly revealed the connection between morphine administration and tumor development and progression. For instance, morphine acts on the μ-opioid receptors and increases intracellular cyclin D1 and activates G protein receptors resulting in accentuation of the mitogen-activated protein kinase (MAPK) pathway and thereby enhances angiogenesis in breast tumor cells [2]. In fact Farooqui et al. have recently demonstrated that morphine administration in breast tumor animal models activates prostaglandin E2 thereby enhancing tumor metastasis also. Similar effects are noted in prostate cancer tissue [3]. For instance, patients who undergo radical prostatectomy for prostate carcinoma and who receive opioid treatment in conjunct with general anesthesia have a 57% higher risk of tumor recurrence in comparison to patients who receive general anesthesia in conjunction with epidural anesthesia [4]. Similarly, Mathew et al. have shown that morphine administration enhances tumor growth in lung cancer models [5]. Interestingly, methylnaltrexone inhibits activation of RhoA by inhibiting transactivation of VEGF receptors [6]. As a result it attenuates morphine-induced angiogenesis and thus inhibits tumor growth. PD98059 is another new agent that inhibits the MAPK pathway and thereby decreases opioid-induced tumor cell proliferation [7]. Similarly, celecoxib attenuates opioid-induced stimulation of cyclooxygenase-2 receptors and thereby exerts antineoplastic effects [3].

These examples clearly illustrate the role of morphine in enhancing tumor angiogenesis and growth. Clearly, there is a further need to identify further such opioid receptor antagonists besides methylnaltrexone that can inhibit morphine-mediated carcinogenesis.

References

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