Cost Savings and Luteinizing Hormone–Releasing Hormone Agonist

By Steven Tucker, MD

In the March 2006 issue of the *Journal of Oncology Practice*, Wagmiller et al¹ describe significant potential cost savings for use of an individualized luteinizing hormone-releasing hormone agonist (LH-RHa) strategy based on serum testosterone triggers rather than strict manufacturer guidelines. It is clear and well documented that gonadal suppression persists for months after LH-RHa therapy

is held, and even minimal androgen recovery may be delayed by more than 12 months after longer than 2 years of continuous therapy. Given the substantial economic impact and preliminary suggestion of a noninferior outcome, efforts to define the real world savings should be evaluated prospectively.

It should be noted that reimbursement changes brought about by the Medicare Modernization Act (reimbursement of LH-RHa therapy at average sales price [ASP] instead of average wholesale price [AWP]) have already brought relative change to this field. For example, Wagmiller et al cite an

AWP of \$1796.51 for a 3-month leuprolide injection. However, the current ASP is 60% lower, at \$708.39.² Re-evaluation using current ASP figures would likely re-demonstrate substantial, albeit relatively lower, savings. This model remains important as future work should assess the increasing use of androgen-deprivation therapy (ADT) in localized disease, the adjuvant setting, and in biochemical-only relapse. Currently, the overwhelming majority of men receiving ADT today do not have androgen independent prostate cancer and do not have osseous metastases.³

However, advocates of this model should be cautioned regarding the serious difficulties in the accurate measurement of serum androgens, as well as medical definitions of “castrate.” Available androgen immunoassays become unreliable at low levels.⁴ Although Wagmiller et al identified existing controversies in the serum values to define castration, it has become clear that mechanistically, most patients retain active androgen receptor signaling even at testosterone levels less than 20 ng/mL.⁵ In the future, additional savings may be realized with alternative LH-RHa depot preparations, use of novel anti-androgens, risk-adapted strategies for clinically favorable patients, and identification of molecular triggers for adjusting therapy versus serum testosterone alone.

Steven Tucker, MD, is the director of the Prostate & GU Oncology Program at The Angeles Clinic & Research Institute in Los Angeles, California. Contact him at stucker@theangelesclinic.org.

References

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