CASE REPORT

Presentation of a functional pituitary adenoma as a significant decrease in prostate-specific antigen level in a patient followed for prostate cancer

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The stimulatory role of testosterone in the production and release of prostate-specific antigen (PSA) has been well characterized. Testosterone production by the testes is dependent on a functional hypothalamic-pituitary-gonadal axis. High prolactin levels have been shown to disrupt this axis, resulting in decreases in gonadotropins and testosterone levels. We report a patient with prostate cancer and elevated PSA levels followed with "watchful waiting" for several years who experienced a precipitous decrease in PSA level over a 3 month period. The patient was found to have an asymptomatic prolactin-secreting pituitary macroadenoma.

Key Words: prostate specific antigen, prolactinoma, prostate cancer

Case report

An 87-year old white male being followed with moderate prostate obstructing symptoms presents with a rising PSA level (9.0 ng/mL; 0-4.0ng/mL). PSA levels over the previous 4 years were stable (5.0 ng/mL-7.0 ng/mL). Transrectal ultrasound-guided prostate needle biopsy revealed a Gleason 3+3 prostatic adenocarcinoma in the right apex involving 5% of one core. A skeletal survey was negative for metastatic disease. The patient had no significant medical and was taking tamsulosin 0.4 mg. He had no significant findings on physical examination.

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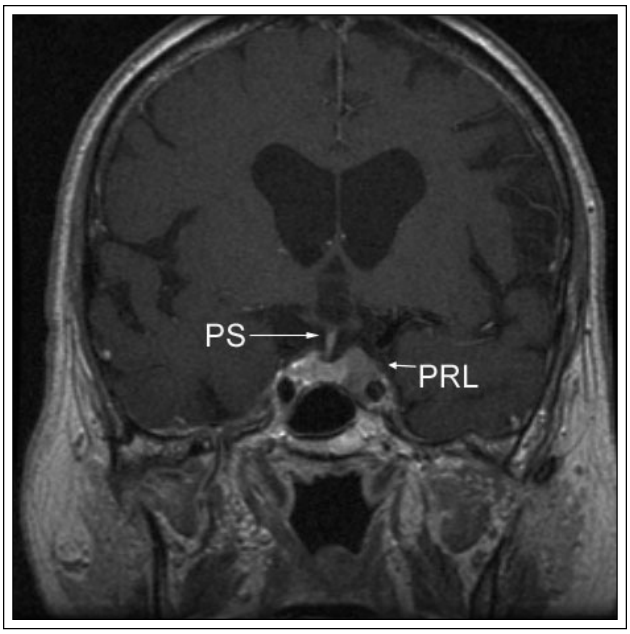

Figure 1. T1 weighted MRI coronal view of the brain: Arrow demonstrates pituitary adenoma 0.6 cm x 1 cm x 1.2 cm in size (Arrow labeled PRL). There is invasion of this mass into left cavernous sinus. The pituitary stalk is minimally deviated to the right side. (Arrow labeled PS)

After diagnosis his PSA level continued to increase from 7.2 ng/mL to 12.6 ng/mL. He opted not to receive hormonal therapy. After 18 months of steadily rising PSA levels, his PSA dropped to 3.7 ng/mL; verified 2 weeks later at 3.4. His review of systems was unremarkable; he had no changes in his urologic symptoms and changes in medications, diet, or general health. Endocrinologic evaluation demonstrated a testosterone of 262 ng/dL (350 ng/dL-1030 ng/dL), a luteinizing hormone 3.1 MIU/ml (1.3 MIU/ml -12.0 MIU/ml), and a prolactin 366 ng/mL (2.1 ng/mL -17.7 ng/mL).

A magnetic resonance imaging of the brain demonstrated a pituitary macroadenoma measuring 0.6 cm x 1.2 cm with left cavernous sinus invasion. See Figure 1. No visual field defects or neurologic symptoms were observed. The patient was treated medically with cabergoline, a prolactin-inhibiting agent.

Three months after initiating therapy his prolactin was 1.7 and his PSA level rose to 6.3 ng/mL. The testosterone levels remained low (244 ng/dL.).

Discussion

Secretion of prolactin by pituitary adenomas has been shown to disrupt the pulsatile release of gonadotropins, resulting in decreased levels of LH and testosterone. PSA expression has been shown to be dependent on testosterone production. In our patient with prostate cancer with an elevated PSA (max = 12.6 ng/mL), a prolactin secreting adenoma caused a 70.6% decrease in PSA value over 12 weeks. Abnormal serum hormone findings confirmed dysfunction of the hypothalamic-pituitary-gonadal axis.

Medical therapy was indicated with this significantly elevated prolactin level. Surgical intervention is an option reserved only for macroadenomas with suprasellar extension and those patients who fail dopamine agonist therapy.[1] Medical suppression of the prolactinoma was successful but PSA and serum testosterone levels remained low. While serum prolactin levels normalize with medical therapy in 85% of cases, in only 50%-65% of patients' serum testosterone return to normal due to global pituitary dysfunction.[2] As in our patient, testosterone and PSA may not rebound after adequate medical therapy for prolactinoma.

Yoon et al recently reported on a patient diagnosed with a non-functional pituitary adenoma by observing a serial decrease in PSA value over a 2 year period.[3] Our case demonstrates that a precipitous drop in serum PSA should trigger screening for hypogonadism and prolactin secreting adenoma. Even after successful treatment men with prolactinomas may challenge the urologist's ability to monitor prostatic adenocarcinoma by PSA. □

References

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