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Role of Finasteride in the Treatment of Recurrent Hematuria Secondary to Benign Prostatic Hyperplasia

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OBJECTIVES. We evaluated the efficacy of finasteride for the treatment of gross hematuria secondary to benign prostatic hyperplasia in a prospective fashion.
METHODS. Twelve patients with recurrent episodes of gross hematuria secondary to benign prostatic hyperplasia were treated with finasteride 5 mg/day. Before initiating treatment, we excluded other sources of hematuria using intravenous urography, cystoscopy, and urine culture.
RESULTS. Bleeding subsided within 2 weeks of treatment in all 12 patients. Minimum follow-up was 6 months. Finasteride was well tolerated by all 12 patients.
CONCLUSIONS. Finasteride appears to be effective in treating recurrent gross hematuria secondary to benign prostatic hyperplasia. This therapy should be considered an alternative to transurethral resection of the prostate or hormonal ablation in patients with recurrent hematuria and no significant obstructive uropathy or adenocarcinoma of the prostate.
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KEY WORDS: **hematuria; benign prostatic hyperplasia; finasteride**

INTRODUCTION

Finasteride is a 4-aza steroid that acts as a competitive inhibitor of the enzyme, 5α-reductase and decreases the conversion of testosterone to the more potent androgen, dihydrotestosterone [1,2]. Animal studies have demonstrated that chronic oral administration of finasteride results in a decrease in prostatic glandular and fibromuscular tissue; importantly, finasteride decreases the vascularity of the prostate [3–5].

We report a prospective, nonrandomized investigation in which the role of finasteride was determined in the treatment of patients with recurrent prostatic bleeding secondary to benign prostatic hyperplasia.

MATERIALS AND METHODS

Between July 1993 and July 1995, 12 male patients with recurrent episodes of gross hematuria secondary to benign prostatic hyperplasia were treated with finasteride 5 mg/day. Evaluation of each patient prior to treatment included a complete history and physical, urinalysis, urine bacterial culture, intravenous urogram, and cystoscopy.

Patient age range was 66–80 years. All patients had a minimum of two episodes of gross hematuria or one episode of clot retention. Three patients had four episodes of hematuria, and three patients had five episodes of hematuria. The interval between bleeding episodes ranged from 1 week to 4 months. Three patients required blood transfusion for hemoglobin <8 g/dl (normal 13.9–16.9). None of the patients had an AUA symptom score >8. Three patients had a history of prior surgery for benign prostatic hyperplasia more than 5 years ago; two patients had undergone transurethral resection of the prostate and one patient had a prior open prostatectomy. No pa-

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tient had a history, laboratory finding (PSA >4.0 ng/ml), or physical examination suspicious of carcinoma of the prostate. Excretory urography was normal and urine culture revealed no significant bacterial growth in all 12 patients. Cystoscopy revealed mild prostatic occlusive disease with engorged, friable, bleeding prostate tissue; endoscopically, the bladder and anterior urethra were normal.

RESULTS

All 12 patients had resolution of gross hematuria within 2 weeks of finasteride therapy. Minimum follow-up was 6 months. Median follow-up was 11 months, and a maximum follow-up of 24 months. No patient has experienced a recurrence of gross hematuria while taking the finasteride. One patient discontinued the medication after one year of treatment due to noncompliance. Two months after stopping the medicine, the patient had an episode of gross hematuria and clot retention. Finasteride was restarted and the patient has not experienced another recurrent episode. The medication was well tolerated by all twelve patients with no complaints of decreased libido, erectile dysfunction, or constitutional symptoms.

DISCUSSION

Evaluation of patients who present with gross hematuria includes a history and physical, urine culture, intravenous urography, and cystourethroscopy. Common causes of gross hematuria include urinary tract infection, malignancy, urinary calculus, prostatic bleeding, prior instrumentation, and coagulopathy. The diagnosis of prostatic bleeding due either to benign prostatic hyperplasia or adenocarcinoma is made when the other diagnoses have been excluded. Endoscopic evaluation of the prostate may reveal engorged blood vessels, active bleeding, friable mucosa, or a normal-appearing gland.

Treatment options for prostatic bleeding include tamponade of the prostate with a urethral catheter, endoscopic resection and fulgaration of the prostate, hormonal ablation, use of antifibrinolytic agents, or careful observation with limiting of physical activity. Marshall and Narayan [6] reported one patient with recurrent gross hematuria who was managed with finasteride. In this report, no mention was made of the medication dosage or outcome. To our knowledge, no other series in the literature reports this use of finasteride.

In our experience, we found consumption of finasteride 5 mg/day is helpful in treating patients with recurrent gross hematuria secondary to benign prostatic hyperplasia. The patients in our study were not troubled by bladder outlet obstructive symptoms and were spared endoscopic resection or other invasive approaches. To date, only one patient discontinued the medication and experienced a recurrent episode of hematuria. Based on this, our current recommendation is to continue the medication indefinitely. Perhaps, in the future, we may advise patients to stop taking the medication to evaluate whether it is necessary on a long-term basis.

The rapidity with which the finasteride helped control the hematuria experienced by our patients was striking. Animal studies have demonstrated that treatment with finasteride leads to a decrease in both the glandular and fibrovascular components of the prostate in a dose-responsive fashion. Laroque et al. [3] demonstrated decreased vascularity of the prostate stroma with replacement by loosely arranged collagen fibers, fibroblasts, and smooth muscle cells in dogs treated with increased doses of oral finasteride. Likewise, Prahalada et al. [4] demonstrated a 50% decrease in the fibrovascular content in the prostates of rats treated with finasteride. We are currently planning in vitro and in vivo studies to evaluate the mechanism of action of finasteride which accounts for the rapidity of its action in this regard.

CONCLUSIONS

Our experience is limited by the small number of patients included in the study. Many of the patients who present to our hospital with recurrent gross hematuria secondary to prostatic bleeding have concomitant obstructive uropathy or carcinoma of the prostate and are best managed with other treatment options. Therefore, no definite comparison or conclusion can be made in regard to other treatment modalities. It is clear, however, that the use of finasteride should be considered as a treatment alternative in patients with no significant obstructive uropathy or adenocarcinoma of the prostate. Further clinical investigation with finasteride is also warranted.

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