Giant prostatic hyperplasia. A case report and literature review

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ABSTRACT
Giant prostatic hyperplasia (GPH) is an unusual pathology that should be suspected during the study of solid pelvic cavity tumors in men. There are fewer than 30 reports in the literature of cases in which the entity weighs more than 500 g. The rare GPH case described here presented in a 73-year-old man with few urinary symptoms, an episode of hematuria and no significant prostate specific antigen (PSA) elevation. He was treated with enucleation of the adenoma by vesicocapsular approach. The surgical specimen weighed 600 g. Patient postoperative progression was adequate.

Key words: giant prostatic hyperplasia, vesicocapsular prostatectomy, Mexico.

RESUMEN
La Hiperplasia Prostática Benigna Gigante, (HPB-G) es una patología inusual, que deberá ser sospechada durante el estudio de los tumores sólidos del hueco pélvico en varones. Se informa sobre el caso de una patología poco común, HPB-G, de la cual existen menos de 30 casos informados en la literatura; con peso mayor de 500 g. Presentamos el caso de un paciente de 73 años de edad, quien cursaba con pocos síntomas urinarios, con un episodio de hematuria y sin elevación importante del APE. Fue tratado con enucleación del adenoma por abordaje vésico-capsular, y con un peso de 600 g de la pieza. Presentó una evolución posoperatoria adecuada.

Palabras clave: Hiperplasia prostática benigna gigante, prostatectomía vésico-capsular, México.
INTRODUCTION

Prostatic hyperplasia is without a doubt the most frequent tumor in men after 50 years of age. From a histological perspective, it is characterized by glandular, muscle or stromal tissue proliferation according to the activation of the distinct cell lines in its structure in such a way that the intensity with which that cellular development is generated results in the degree of prostate growth.

Giant benign prostatic hyperplasia (G-BPH) is characterized by a growth exceeding 500 g, even though different authors consider a size of 200 g to constitute G-BPH. There are fewer than 30 cases exceeding 500 g reported in the international literature.

CASE PRESENTATION

Patient is a 70-year-old man who sought medical attention at the urology service presenting with irritative and lower obstructive urinary syndrome of 1-year progression that was characterized by a reduction of strength and quality of urine stream, straining, intermittence, terminal dribbling, suprapubic pain and 5 x 5 micturitions in a 24-hour period. One month prior to hospital admittance patient presented with an episode of macroscopic hematuria that stopped in two days with no specific treatment. During medical history interview patient referred to chronic constipation with pain upon defecation but no weight loss. During physical examination full bladder was not palpated, genitals were adequate for age and sex, digital rectal examination (DRE) revealed external hemorrhoids and normotonic sphincter. Prostate was very enlarged. Neither its base nor its lateral edges could be defined and surface was nodular and adenomatous. Temperature was normal and there was slight pain.

Laboratory work-up: Urinalysis: cloudy urine, density 1.020, leukocyturia, erythrocyturia, bacteria +++. Prostate specific antigen (PSA): 13 ng. Prostate size could not be adequately measured with transrectal ultrasound (US) (13 cm x 10 cm); there was loss of the usual echotexture that enables peripheral, central and transitional zone definition. Sextant biopsy provided histopathological report of prostatic hyperplasia.

Vesicocapsular prostatectomy with prior Santorini plexus ligature and later enucleation of the large 10 cm x 10 cm x 8 cm adenoma was carried out. Sutures (0 chromic catgut) were placed at the five and seven o’clock positions on the bladder neck and it was trigonized with 2-0 chromic catgut for later surgical suturing of the prostate capsule and bladder on two planes with the same type of 0 suture. Transurethral 22 F Couvelaire catheter was placed after 1 chromic catgut sutures were placed on bladder neck at the 12 o’clock position to reduce its diameter. Postoperative progression was satisfactory and management was conventional with continuous irrigation with physiological solution during the first 12 postoperative hours. Catheter and stitches were removed after 10 days. Pathology report confirmed the presence of prostate tissue with fibrous myoglandular components with important stromal component and no evidence of malignancy in relation to prostatic hyperplasia and weighing 600 g (Images 1 and 2).

DISCUSSION

BPH prevalence is directly proportional to increase in age. At 80 years of age the possibility of presenting with it is 95%. These prostatic changes begin at 40 years of age and prostate volume increases around 0.6 mL per year and is associated with a reduction of the mean urinary flow at the rate of 0.2 mL per second. This is not necessarily a rule, since prostate growth and the severity of its symptoms are erratic in each case. Known as prostatism these clinical symptoms are characterized by the presence of irritative and obstructive lower urinary tract symptoms (LUTS) which are evaluated in a general manner through the international prostate symptom score (IPSS). In this regard, it is fundamental to remember that prostate size is not directly proportional to degree of symptom severity.

Thanks to prostate studies carried out over the last fifty years, the general population is now more informed about this organ and attaches importance to prostate study, especially in regard to the natural progression of
diseases affecting it after the age of 40, such as prostate cancer, BPH and prostatitis.

The prostate maintains a supposed balance between the influence of hormonal stimuli controlled by the hypothalamic-pituitary-adrenal axis through luteinizing hormone-releasing hormone (LH-RH) secretion by the hypothalamus.

This stimulates the adenohypophysis for luteinizing hormone (LH) production, acting on the testes that produce 95% of testosterone and on the adrenal glands that produce 5%. LH acts at the prostate level by breaking down into dihydrotestosterone (DHT), under the effect of 5-alpha reductase (5-AR) enzyme, with which prostate cell DNA is stimulated and messenger RNA (mRNA) is activated, and fires both stimulation and inhibition growth factors that are in perfect homeostasis. In this way, cytokines, epidermal growth factor and platelet-derived growth factor induce prostate growth by producing amplification of the intracellular signal that stimulates mitosis. Transforming growth factor beta (TGF-β) inhibits cell division in addition to inducing cell death programmed through apoptosis that is induced by different genes, including B-cell lymphoma 2 (Bcl-2) and Bcl-2-associated X protein (BAX).

G-BPH genesis is not known. However, an exaggerated overexpression of growth factors in addition to the absence or reduction of inhibitory factors, breaking that balance, can be inferred. The mutation of certain proto-oncogenes such as Ras and c-erbB-2 may also be involved, developing a continuous cellular proliferation signal or the loss of influence of the p-53 suppressor gene through its mutation or deletion, which would allow for abnormal cell proliferation.

G-BPH is an extremely rare entity that was first reported in 1908 by Freyer. He extirpated an adenoma weighing more than 500 g. Up to 2004 there have been 16 reported cases, the largest of which weighed 2400 g.

Patients can clinically present with obstructive urinary symptoms or with extrarinary symptoms characterized by compression of neighboring structures due to exaggerated prostate growth. G-BPH, ectopic prostate tissue and sarcoma should always be considered in the differential diagnosis of pelvic cavity solid masses. Likewise, the possibility of tumors of neighboring structures to the prostate should be considered, regardless of patient age and the many or few symptoms presenting since there have been reports of this pathology in patients under 40 years of age. Severe prostatic hyperplasia is confirmed by imaging studies such as US, tomography and magnetic resonance, though they can be imprecise in defining tumor origin.

G-BPH treatment is open surgery employing either transcapsular retropubic approach or transvesical suprapubic approach. Vesicocapsular approach with penile dorsal complex ligature was used in the present case, allowing for adequate control during surgery. A case of G-BPH with a tumor larger than 200 g was reported on in which the tumor was resected endoscopically by means of electrovaporization and carried out in several surgical interventions. The present authors consider open surgery to be the best option.
option with respect to best resolution percentage and fewer complication possibilities when dealing with large prostates.

One of the disadvantages of open surgery is greater volume of intraoperative bleeding in comparison with endoscopic procedures. Reduction of this bleeding by means of hypogastric clamping techniques (vascular control) prior to adenoma enucleation has been described. The present authors have never had to carry out such vascular control in open surgery nor was it necessary in the case presented here. Simple ligature of the penile dorsal complex before the procedure and the placement of sutures at the 5 and 7 o’clock positions on the bladder neck once the adenoma was enucleated were sufficient. Intraoperative bleeding was 700 mL and did not merit blood transfusion, the same as in other G-BPH reports.

**CONCLUSIONS**

G-BPH is an extremely rare entity and the mechanisms of its genesis are not known. It should be considered in the differential diagnosis of pelvic cavity tumors in men regardless of their age. The fact that there is a greater stromal component in these large prostates explains the lack of PSA elevation over 25 ng, a condition that could perhaps be expected due to the known volume/PSA relation in the benign pathology of the prostate.

**BIBLIOGRAPHY**