

Malakoplakia of the prostate diagnosed in patients with benign prostatic hyperplasia and elevated prostate-specific antigen

Malacoplasia da próstata diagnosticada em pacientes com hiperplasia prostática benigna e antígeno prostático específico elevado

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ABSTRACT

Malakoplakia is a rare chronic inflammatory disease which affects the genitourinary system, particularly the bladder, the kidneys and the ureters. Isolated involvement of the prostate is much rarer. Differential diagnosis of adenocarcinoma of the prostate can be extremely difficult, because of clinical and imaging similarities. Patients with frequent occurrence of benign prostatic hyperplasia and urinary tract infection need to be prepared for a diagnosis of malakoplakia.

Keywords: malakoplakia; prostatic hyperplasia; prostate-specific antigen; diagnosis, differential.

RESUMO

Malacoplasia é uma doença inflamatória crônica pouco frequente que afeta o aparelho geniturinário, particularmente a bexiga, os rins e os ureteres. O envolvimento isolado da próstata é a forma mais rara. O diagnóstico diferencial do adenocarcinoma da próstata pode ser de extrema dificuldade em virtude das semelhanças clínicas e imaginológicas. Pacientes com hiperplasia benigna de próstata e infecções do trato urinário frequentes podem alertar para o diagnóstico de malacoplasia.

Palavras-chave: malacoplasia; hiperplasia prostática; antígeno prostático específico; diagnóstico diferencial.

INTRODUCTION

The word malakoplakia comes from the Greek *malakós* (soft) and *plakós* (plaque) and represents a rare inflammatory disease that was originally described as occurring in the bladder, but which has also been detected in the genitourinary and in the gastrointestinal tract, as well as skin, lungs, bones, and mesenteric lymph nodes.

It is macroscopically characterised by the presence of soft yellow mucous plaques, with 3 to 4 cm diameter, and slightly elevated. The surrounding mucosae are normally oedematous, hyperaemic and swollen.

Microscopically, these plaques consist of spongy macrophages, of large size and intimately compressed, with occasional multinucleate giant cells intermixed with lymphocytes. The macrophages have abundant content of granular cytoplasm. The granulation is positive for Periodic Acid-Schiff and is due to the phagosomes which are brimming with membranous particles and also remnants of bacterial origin. In ad-

dition, typically there is also presence of laminated mineralised concretions known as Michaelis-Gutmann bodies, both inside the macrophages and between the cells.

Even though the exact pathogenesis is as yet unknown, it is likely to stem from an abnormal function of macrophages in response to a bacterial infection, most probably caused by *Escherichia coli*, while the Michaelis-Gutmann bodies are probably the result of deposits of phosphates of calcium and other minerals in these phagosomes, which are overloaded and could be undergoing disintegration.¹

CASE REPORT

Male patient, aged 69. He came to the Urology outpatient ward complaining of obstructive lower urinary tract symptoms (LUTS). Prostatic specific antigen (PSA) was measurement was 5.9 ng/mL, and the digital rectal exam (DRE) showed a slightly enlarged prostate. He was initially medicat-

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ed with an alpha blocker and a 5-alpha-reductase inhibitor, with satisfactory response, but then had recurring situations of bacteriuria, not showing any symptoms thereof. His medical history included chronic ischaemic cardiomyopathy, hypertension, and diabetes mellitus. He discontinued the treatment after having an acute myocardial infarction. The patient also reports that, after four years, there was worsening of prostatic complaints, while he was making irregular use of alpha blockers and 5-alpha-reductase inhibitors. His condition then progressed with acute urinary retention, which required the introduction of an indwelling vesical catheter. On the day of urinary retention, upon physical examination, he was in a fair state of health, dyspnoeic, with oedema on the lower limbs. He was submitted to medical examinations, that showed Urea at 88 mg/d, Land Creatinin at 3.2 mg/dL. Abdominal ultrasound examination showed: kidneys, nothing worthy of note; prostate weighing 98 grams. DRE showed a prostate that was double the normal size, congested, without lumps. He returned to the outpatient clinic with a PSA reading of 21.97 ng/mL. Urine Culture: *E. coli*. The patient was medicated with sulfamethoxazole 800 mg and tetramethylpyrazine 160 mg, and the catheter was maintained. A biopsy of the prostate was then performed by transrectal ultrasonography, whose results were the following: histological situation corresponding to malakoplakia, suggesting immunohistochemistry. Immunohistochemistry confirmed the diagnosis of malakoplakia. A transvesical prostatectomy was then performed, without any complications, and the anatomopathological study of the specimen obtained showed: prostate weighing 67 grams, with diagnosis of malakoplakia and myoadenomatose hyperplasia. The patient is now without a catheter, with good spurts of urine, a urea reading of 74 mg/d L and creatinin at 1.5 mg/dL, with negative uroculture. Kidney ultrasound did not shown any echographic changes, while postmictional residue is negligible.

DISCUSSION

Malakoplakia is a rare disease that can affect the prostate. Its main characteristic is being accompanied by recurring urinary tract infections that can be combined with high PSA reading^{2,3} and often leads to suspicion of carcinoma of the prostate.⁴ Recurring infections in people who have benign prostatic hyperplasia should draw attention to the diagnosis.⁵ Malakoplakia also tends to affect people with diabetes mellitus, people with liver problems (especially caused by alcohol abuse), and anyone else with a weakened immune system (such as people living with HIV and people who have undergone organ transplants) or who have lupus or ulcerative colitis.⁶

Genetic causes have been considered as a possible etiological factor.⁷

Histologically, this condition is characterised by the presence of entire coliforms in macrophages (Figures 1 and 2). The diagnosis is anatomopathological and consists in identification of pathognomic Michaelis-Gutmann bodies.^{1,8} In this regard, a biopsy is essential for a proper diagnosis, distinguishing the condition from carcinoma of the prostate.⁹

The clinical treatment for malakoplakia is based on quinolone antibiotics, or sulfa drugs, which are able to penetrate the macrophages.¹⁰⁻¹² Long-term treatment is suggested^{10,13} for complete resolution of the situation, although efficiency is hard to achieve. The addition of Vitamin C and cholinomimetic drugs seem to make the treatment more effective, through the improvement of cell activity in phagocytes.¹⁴

However, surgical treatment has been proven effective in some cases.¹²

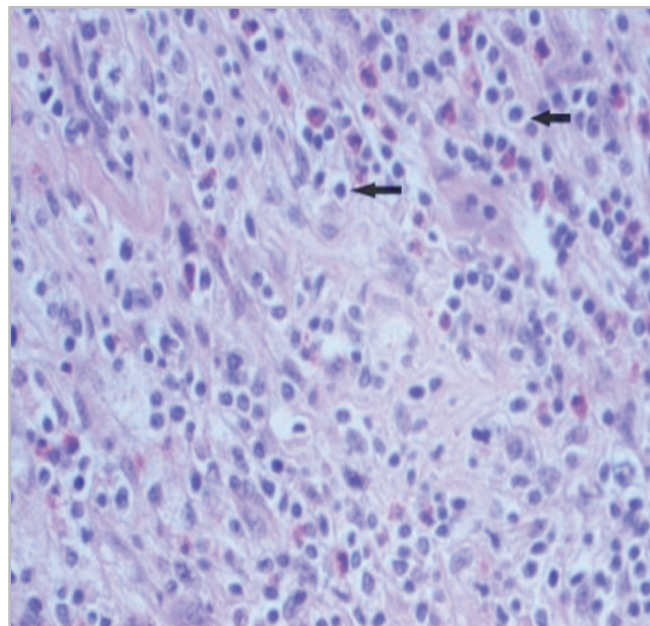


Figure 1. 40X microscopic image showing intense lymphohistiocytic infiltrate with enlarged and bulky cytoplasm containing Michaelis Gutmann bodies.

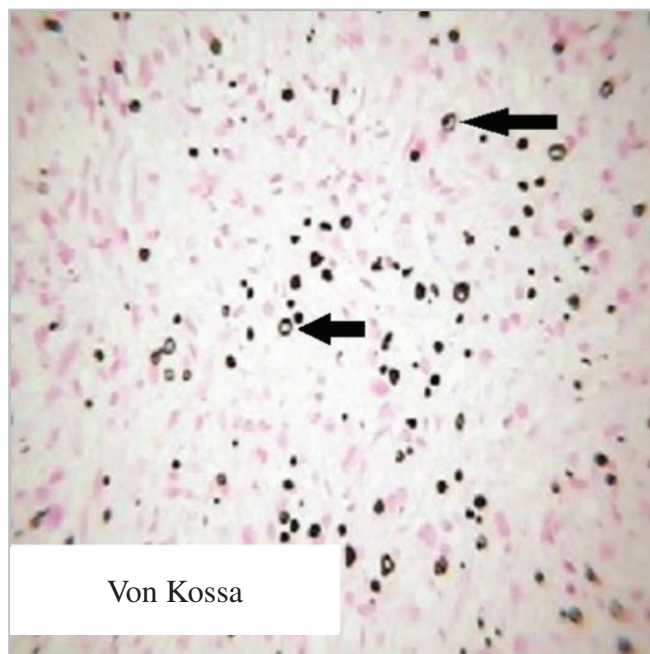


Figure 2. 400X microscopic image: one can see corpuscles positive to Von Kossa and PAS stainings.

Here, we report on a case of Malakoplakia of the prostate that was treated surgically through transvesical prostatectomy, recommended because of urinary retention after bad response to intermittent vesical catheter. The patient made a good recovery, and three months after surgery has not presented with infections, which shows the effectiveness of surgical procedures for the control of infections. The patient must now be continually monitored, considering that there is a possibility of recurrence of the disease and infections.^{15,16}

CONFLICT OF INTERESTS

The authors declare that they have no conflict of interest.

REFERENCES

1. Stanton MJ, Maxted W. Malakoplakia: a study of the literature and current concepts of pathogenesis. *J Urol.* 1981;125(2):139-46. [https://doi.org/10.1016/S0022-5347\(17\)54940-X](https://doi.org/10.1016/S0022-5347(17)54940-X)
2. Dasgupta P, Womack C, Turner AG, Blackford HN. Malakoplakia: von Hansemann's disease. *BJU Int.* 1999;84(4):464-9. <https://doi.org/10.1046/j.1464-410x.1999.00198.x>
3. Görgel SN, Balcı U, Sarı AA, Ermete M, Girgin C, Dinçel C. Malakoplakia of the prostate diagnosed by elevated PSA level and transrectal prostate biopsy. *Kaohsiung J Med Sci.* 2011;27(4):163-5. <https://doi.org/10.1016/j.kjms.2010.12.012>
4. Shimizu S, Takimoto Y, Niimura T, Kaya H, Yamamoto T, Kawazoe K, et al. A case of prostatic malacoplakia. *J Urol.* 1981;126(2):277-9. [https://doi.org/10.1016/S0022-5347\(17\)54475-4](https://doi.org/10.1016/S0022-5347(17)54475-4)
5. Sarma HN, Ramesh K, al Fituri O, Saeed SO, Majeed SA. Malakoplakia of the prostate gland report of two cases and review of the literature. *Scand J Urol Nephrol.* 1996;30(2):155-7. <https://doi.org/10.3109/00365599609180909>
6. Qualman SJ, Gupta PK, Mendelsohn G. Intracellular *Escherichia coli* in urinary malakoplakia: a reservoir of infection and its therapeutic implications. *Am J Clin Pathol.* 1984;132(1):192. [https://doi.org/10.1016/S0022-5347\(17\)49522-X](https://doi.org/10.1016/S0022-5347(17)49522-X)
7. Feldman M, Friedman LS, Brandt LJ. Sleisenger & Fordtran's gastrointestinal and liver disease. 9th ed. Philadelphia: Saunders Elsevier; 2010. p.2306-7.
8. Lewin KL, Fair WR, Steigbigel RT, Winberg CD, Droller MJ. Clinical and laboratory studies into the pathogenesis of malakoplakia. *J Clin Pathol.* 1976;29(4):354-63. <http://dx.doi.org/10.1136/jcp.29.4.354>
9. Thrasher JB, Sutherland RS, Limoge JP, Sims JE, Donatucci CF. Transrectal ultrasound and biopsy in diagnosis of malakoplakia of prostate. *Urology.* 1992;39(3):262-5. [https://doi.org/10.1016/0090-4295\(92\)90302-D](https://doi.org/10.1016/0090-4295(92)90302-D)
10. Ballesteros Sampol JJ. Malacoplaquia urogenital: presentación de 4 casos y revisión de la literatura. *Arch Esp Urol.* 2001;54(8):768-76.
11. Furth RV, van't Wout JW, Zwartendijk J, Wertheimer PA. Ciprofloxacin for treatment of malakoplakia. *Lancet.* 1992;339(8786):148-9. [https://doi.org/10.1016/0140-6736\(92\)90212-L](https://doi.org/10.1016/0140-6736(92)90212-L)
12. Dohle GR, Zwartendijk J, Van Krieken JH. Urogenital malacoplakia treated with fluoroquinolones. *J Urol.* 1993;150(5 Pt. 1):1518-20. [https://doi.org/10.1016/S0022-5347\(17\)35833-0](https://doi.org/10.1016/S0022-5347(17)35833-0)
13. Niemierko M, Kuzaka B. [Malacoplakia of the prostate]. *Przegl Lek.* 2005;62(8):825-6.
14. Abdou NI, NaPombejara C, Sagawa A, Ragland C, Stechschulte DJ, Nilsson U, et al. Malakoplakia: evidence for monocyte lysosomal abnormality correctable by cholinergic agonist in vitro and in vivo. *N Engl J Med.* 1977;297(26):1413-9. <https://doi.org/10.1056/NEJM197712292972601>
15. Velásquez JG, Vélez A, Uribe JF. Malacoplaquia en urología: reporte de una serie de casos en un hospital universitario de Medellín – Colombia. *Rev Urol Colomb.* 2006;XV(1):49-57.
16. Ferronha F, Galego P, Pardal H, Vilas Boas V, Gameiro CD, Lopes SP, et al. Malacoplaquia da próstata. *Acta Urol.* 2009;26(2):86.

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