



Clinical case

Unusual metastasis of prostate cancer: a rare case report

Y.Dkhissi, A. Mai, M.Haloua, B. Alami, Y. Alaoui Lamrani, M. Maaroufi, M. Boubbou

Abstract

Prostate cancer is the second death causing men cancer after lung cancer [1]. It is the most common men cancer over the age of 50. Metastasis for this type of cancer are mainly lymph node and bone. Visceral and cerebral metastasis are unusual, rare and terminal, reported in less than 4% of cases and often post mortem [2]. We report the case of a patient suffering from prostate adenocarcinoma with a high-risk according to the AMICO classification, who has been operated (open prostatectomy without preservation of neurovascular bundle). The patient presented two years after radical treatment a biological recurrence and started complaining headache and visual problems that led to the discovery of dural metastasis.

Keywords: cancer, prostate, dural metastasis.

Résumé

Le cancer de prostate représente la 2^{ème} cause de décès chez l'homme après le cancer des poumons [1]. C'est le cancer le plus fréquent chez les hommes de plus de 50 ans. Les métastases pour ce type de cancer sont principalement ganglionnaires et osseuses. Les métastases viscérales et cérébrales sont inhabituelles, rares et terminales, retrouvées

dans moins de 4% des cas et souvent en post mortem [2]. Nous rapportons un cas d'un patient suivi pour un adénocarcinome de prostate à haut risque selon la classification AMICO, qui a été opéré (prostatectomie sans préservation des bandes neurovasculaires). Le patient a présenté deux ans après le traitement radical une récurrence biologique et a accusé des maux de tête et des troubles visuels qui ont conduit à la découverte de métastases dures.

Mots clés : cancer, prostate, métastase durale.

Introduction

Prostate cancer is the most common men cancer over 50 years. It represents the first urological cancer and the second after lung cancer [3-4]. It is a global disease whose incidence increases with age and varies by races and regions [3-5]. The diagnosis is based on digital rectal exam and an abnormal increase of prostate specific antigen (PSA), which may envisage and predict tumor extension (PSA>100ng/ml) [5,6]. The anatomopathological study confirms the diagnosis and specifies the type and grade of histology. Prostatic cancers are dominated in 95% by adenocarcinomas [7]. Less than 2% of patients

develop brain metastasis. Autopsy series have certified the rarity of brain secondary locations [8]. The most common metastasis are lymph node and bone (reaching 40% of patients at the time of diagnosis and 86% at the terminal stage). Visceral and brain metastasis are rare and terminal [2].

Clinical case

A 54-year-old patient with history of operated shoulder fracture 9 years ago, presented low urinary tract symptoms (poor stream, sensation of incomplete bladder emptying) associated to approximately eight kilograms of weight loss. The initial clinical examination objectified an enlarged asymmetrical prostate (60g) with areas of firmness and induration. The PSA level was 18 ng/ml.

A trans rectal ultrasound was performed and showed an increased prostate volume with diffuse

hypo echogenic areas. The kidneys were normal, without dilatation. The prostatic biopsy revealed a moderately differentiated prostatic adenocarcinoma with a Gleason score of 7 (4+3). This ranks the patient as a high-risk patient according to the AMICO classification. The initial extension CT didn't objectify any remote secondary locations. The patient was operated (open prostatectomy without preservation of neurovascular bundle). The patient presented a good clinical, biological and radiological response. Two years later, the patient had a biological recurrence with a high PSA level superior to 0.2 µg/L, he was programmed for radiotherapy sessions. Meanwhile, the patient experienced headaches with a decrease in visual acuity. A cerebral CT scan was performed with evidence of dural and skull metastasis (Figure 1 and 2).

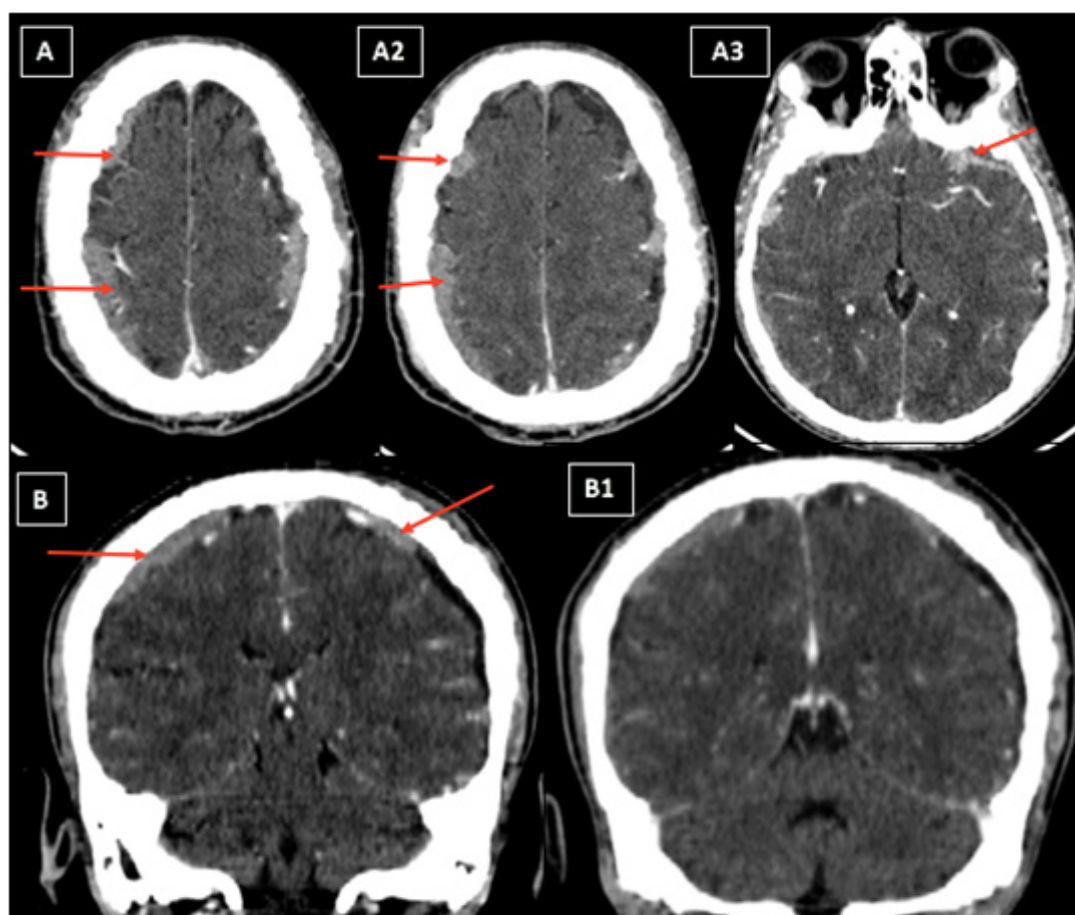


Figure 1 : Axial contrast enhanced cerebral CT (A,A1,A2) with sagittal reconstructions (B,B1) : Thickness and enhancement of the dura of the convexity and the base respecting the dura of the falx cerebri and tentorium cerebelli (Red Arrows).

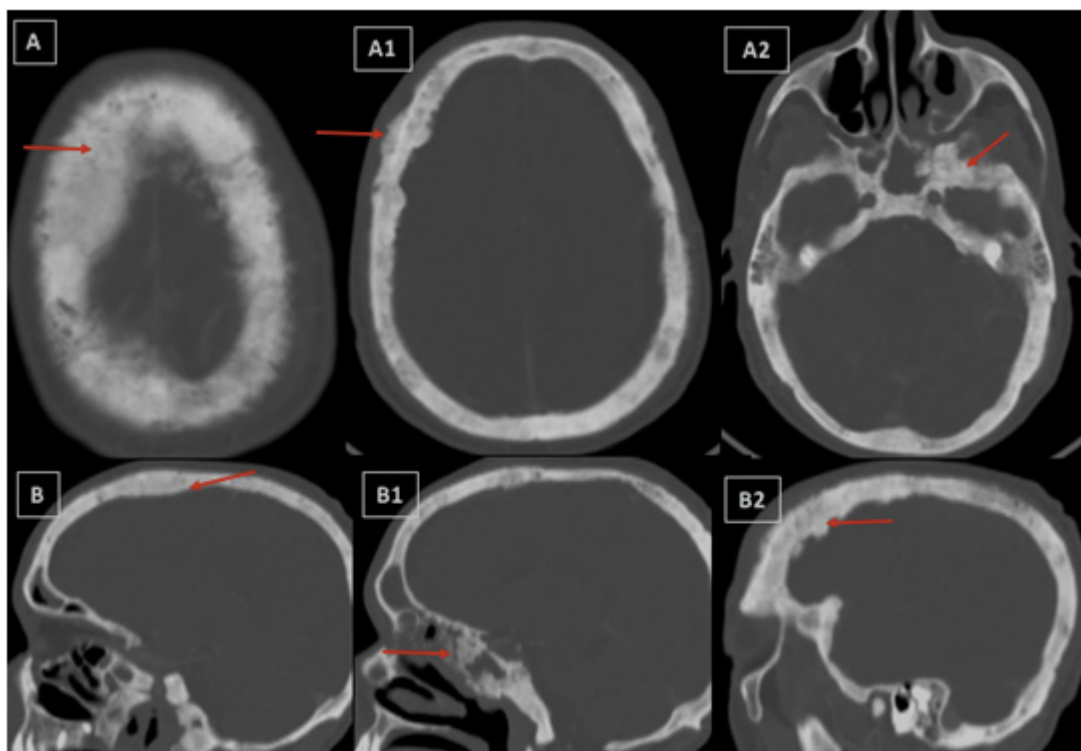


Figure 2 : Axial Cerebral bone window CT (A,A1,A2) with sagittal reconstructions (B,B1,B2): Diffuse osteoblastic lesions of the cranial vault and the skull base, irregular bone thickening and loss of differentiation between the inner, outer tables and the spongy bone of the diploë. (Red Arrows).

Discussion

The pathogenesis of dural metastasis is not yet elucidated [8]. There are two main routes of dissemination: Hematogenous diffusion from a cerebral localization or from the cervical lymph nodes through the cranial foramina [10,11]. Theoretically, the cancer cell detaches itself from the primary tumor and crosses several barriers to reach the target organ which is the dura [12].

Prostate cancer presents a great tropism for dura [13]. It is responsible of 9% of dural metastasis and comes behind breast cancers 32% and melanomas 11% [14].

Diffuse dural metastatic localizations are exceptional and associated with a diffuse metastatic infiltration of the cranial vault and skull base; these localizations are observed in prostate cancer; bone metastatic involvement is represented by diffuse osteoblastic lesions of the skull base, bone thickening and loss of differentiation between the inner, outer tables and the spongy bone of the diploë. The dura being in

contact with the infiltrated cranial vault and skull base appears thickened and enhanced after injection of the contrast agent, focal thickening is possible and can simulate meningioma; subcutaneous tissues can also be infiltrated; the falx cerebri and tentorium cerebelli are preserved [15].

A diffuse thickening of the dura of the convexity and possibly of the base respecting the dura of the falx cerebri and tentorium cerebelli evokes diffuse tumor infiltration, most often related to metastatic infiltration of cranial vault and skull base, essentially in prostate cancer [15].

Many autopsy studies affirmed the rarity of the brain metastasis emanating from prostate cancer [18]. In a large post mortem study about metastatic patterns of prostate cancer, Saitoh et al. [16], noted no cases of brain metastasis.

In our case, the brain metastasis was diagnosed while the patient was not on any treatment including chemotherapy or hormone therapy. It was revealed 24 months later from the initial radical treatment. This result approaches the result obtained by MD

Anderson's experience with brain metastasis in prostate cancer, published in 1999 by Mccutcheon and his partners that revealed that the average delay between the diagnosis of prostate cancer and the first discovery of brain metastasis was approximately 28 months [17].

Another enlarged and updated study realized in 2003 by Tremont-Lukats and partners revealed that the brain metastases represented only 0.6% [19].

The median survival of cancer prostate patient with central nervous system relapse in clinical series is poor [8]. In the MD Anderson series, the median survival was 1 month, the patients receiving palliative radiotherapy showed an improved median survival of 3.5 months (2.4 to 4.6 months). In our case, the patient is still alive after starting palliative radiotherapy for 5 months.

Conclusion

Brain metastasis of prostate cancer are rare, often discovered in postmortem. It requires close monitoring; the rise of PSA levels should alarm. Imaging is important to confirm the diagnosis and to eliminate the other etiologies. The factors underlying the selective tropism of the prostatic malignancy for the brain remain to be defined.

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Conflict of interest: none

Références

- [1] STEG A., FLAM T., ZERBIB M., DESLIGNERES S., ESCHWEGE F., WIBAULT P., CONQUY S. Cancer de la prostate pp: 230-307 in: Cancers urogénitaux: Steg A., Eschwege F. Paris, Flammarion Médecine Sciences, 1991.
- [2] Aneur A, Touiti D, EL Mostarchid B, EL Alami M, Jira H, Abbar M. Métastases cérébrales d'un cancer de la prostate : régression sous traitement hormonal. *Prog Urol* 2001 ; 11 : 1298-301.
- [3] Droz J.P., Flechon A., Terret C. Cancer métastatique de la prostate. *Rev. Prat* 2003 ; 53 : 2258-62.
- [4] Herr HW, Kornblith AB, Ofman U. Comparison of the Quality of Life of patients with metastatic prostate cancer who received or did not receive hormonal therapy. *CANCER* 1993; 71(2 suppl) : S143-50.
- [5] Conti G, La Torre G, Cicalese V et coll. Prostate cancer metas- tases to bone : observational study for the evaluation of clinical presentation, course and treatment patterns. Presentation of the Metauro protocol and of patient baseline features. *Arch Ital Urol Androl*, 2008; 80: 59-64.
- [6] Chartier E. Cancer de la prostate. *Urologie Ed. Estem et Medline*, paris, 1998, pp. 119 - 34.
- [7] Birtle AJ, Freeman A, Masters JRW, Payne HA, Harland SJ. Clinical features of patients who present with metastatic pros- tate carcinoma and serum Prostate Speci c Antigen (PSA) levels < 10 ng/ml. The « PSA negative » patients. *Cancer* 2003 ; 98: 2362-7.
- [10] Destrieux C, Becker H, Jan M. Métastases intracrâniennes in : *Neurochirurgie (P. Decq, In- Keravel)*. Ellipses , 1995 ; 145-152.
- [11] Hoang-Xuan K, Napolitano M, Cornu P, Delattre JY. Métastases cérébrales et leptoméningées des cancers solides. *Encycl Méd Chir Neurologie* 1999 ; 17-255-A 10 22 P.
- [12] Kehrl P. Biologie des métastases cérébrales. *Neurochirurgie* 1999 ; 45 : 364-368.
- [13] Takakura K, Sano K, Hojo S. Metastatic tumors of the central nervous system. Tokyo : Igaku Shoin, 1982.
- [14] Kehrl P. Épidémiologie des métastases cérébrales. *Neurochirurgie* 1999 ; 45 : 357-363.
- [15] JL Dietemann, R Correia Bernardo, A Bogorin, M Abu Eid, M Koob, Th Nogueira, MI Vargas, W Fakhoury, G Zöllner.

Les prises de contraste méningées normales et pathologiques en IRM. *Journal de radiologie*, Vol 86, N° 11 - novembre 2005, pp. 1659-1683.

[16] Saitoh H, Hida M, Shimbo T, Nakamura K, Yamagata J, Satoh T. Metastatic patterns of prostatic cancer. Correlation between sites and number of organs involved. *Cancer* 1984;54:3078–84.

[17] McCutcheon IE, Eng DY, Logothetis CJ. Brain metastasis from prostate carcinoma: antemortem recognition and outcome after treatment. *Cancer* 1999;86:2301–11.

[19] Tremont-Lukats IW, Bobustuc G, Lagos GK, Lolas K, Kyritsis AP, Puduvali VK. Brain metastasis from prostate carcinoma: the M.D. Anderson Cancer Center experience. *Cancer* 2003;98:363–8.

[8] J. Craig, J. Woulfe MD, J. Sinclair MD, and S. Malone MD. Isolated brain metastasis as first site of recurrence in prostate cancer: case report and review of the literature. *Current Oncology*, Vol. 22, No. 6, December 2015.

[9] D. N'Dri Oka, G. Varlet, N. Boni, E. Broalet, L. Boukassa, V. Ba Zeze. Métastase durale d'un adénocarcinome de la prostate simulant un hématome sous-dural aigu intracrânien. *Journal of Neuroradiology*, Vol 27, N° 4 - décembre 2000, p. 28.

[18] Baumann MA, Holoye PY, Choi H. Adenocarcinoma of prostate presenting as brain metastasis. *Cancer* 1984;54:1723–5.

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