Surgical Neurology International

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Case Report

Prostate dural metastasis presenting as chronic subdural hematoma. A case report and review of the literature

Alexandros Boukas, Geraint J. Sunderland, Nicholas Ross

Department of Neurosurgery, Regional Neurosciences Centre, Royal Victoria Infirmary, The Newcastle Upon Tyne Hospitals NHS Foundation Trust, Queen Victoria Road, Newcastle Upon Tyne, NE1 4LP, UK

E-mail: *Alexandros Boukas - Alexaderboukas@gmail.com; Geraint J. Sunderland - geraint_s@hotmail.com; Nicholas Ross - Nicholas.Ross@nuth.nhs.uk
*Corresponding Author:

Received: 24 August 14 Accepted: 26 October 14 Published: 19 February 15

This article may be cited as:
Boukas A, Sunderland GJ, Ross N. Prostate dural metastasis presenting as chronic subdural hematoma. A case report and review of the literature. Surg Neurol Int 2015;6:30.
Available FREE in open access from: http://www.surgicalneurologyint.com/text.asp?2015/6/1/30/151713

Abstract

Background: Malignant disease metastasising to the cranial dura is rare. Dural metastases manifesting as a subdural fluid collection and presenting as a chronic subdural hematoma is an uncommon entity with unknown pathophysiology.

Case Description: We present a patient with known prostate cancer metastasising to the cranial dura masquerading as a chronic subdural hematoma. The patient presented with bilateral subdural collections manifesting with confusion and dysphasia. Initial drainage of the larger, symptomatic left side improved only temporarily patient's symptoms. A second drainage of the collection was performed on the same side 5 days later and dural biopsies taken during the same procedure revealed prostate metastases. The patient improved slowly and was discharged to a hospice for palliative care management.

Conclusions: Prostate dural metastases should be suspected in patients with known prostate cancer presenting with a subdural collection in the absence of cranial trauma. If decision to drain the subdural collection is taken, then biopsies can be taken the same time as they can pose a diagnostic challenge.

Key Words: Chronic subdural hematoma, dural metastasis, prostate cancer

Access this article online

Website: www.surgicalneurologyint.com

DOI: 10.4103/2152-7806.151713

Quick Response Code:

INTRODUCTION

Prostate adenocarcinoma is the most common cancer in men, approximately 40,000 cases are diagnosed each year in the UK and it accounts for around 7% of all cancer deaths. The clinical course is very variable and tumors can be low grade and indolent or high grade, aggressive and advanced at the time of diagnosis. Grading is done as per the Gleason grading system, which scores the tumor on the basis of biopsy tissue architecture criteria giving a maximum possible score of 10 for the most aggressive and worst prognostic tumors.

Prostate cancer has a propensity to metastasise to pelvic and retroperitoneal lymph nodes as well as to the liver, bones, lung, and less frequently the central nervous system [CNS].[9] Most characteristic is the affinity of metastases to establish in bones of the vertebral column and pelvis via a retrograde venous route as described by Batson in 1940.[1] While spinal metastatic disease and subsequent spinal cord compression is common, metastatic disease affecting the rest of the CNS is less so. Brain metastases are rare occurring in between 0.83% and 4% of all prostate cancer cases and dural involvement is even rarer and has been previously reported.

We report a case of prostate cancer metastasising to the cranial dura mater presenting as a spontaneous chronic subdural hematoma.

CASE REPORT

A 75-year-old male was referred to our unit with unsteadiness, falls, and slurred speech. His past medical history included a hormone escaped prostate adenocarcinoma with known bony metastases to the ribs and pelvis as well as intrabdominal lymph node involvement. He was originally managed with single androgen suppression therapy (LHRH analog) and external beam radiotherapy for the primary disease but not for the bony metastases. At the time of presentation no calvarian or intracranial disease was present. His Eastern Cooperative Oncology Group (ECOG) performance status was 2.

Computed tomography (CT) head performed at the referring hospital revealed bilateral chronic subdural collections, larger on left side, with 18 mm midline shift to the right. These had the typical radiological appearance of liquefied hematomas [Figure 1].

On admission his Glasgow Coma Scale (GCS) was 15/15 with no lateralizing neurology except mild expressive dysphasia. He was not taking any antiplatelet or anticoagulant medication and his hematological profile revealed unimpaired coagulation and normal biochemical profile.

The patient underwent evacuation of his left sided subdural collection (the prominently symptomatic side) via two burr holes under general anesthetic. The operating surgeon described the procedure uneventful, the subdural collection as "typical light brown color," under high pressure. A Jacques catheter was left in the subdural space for 48 h, which is standard practice in our unit. Postoperatively the patient improved only marginally and the fifth postoperative day, it was clear that he was again expressively dysphasic. Repeat CT of his head revealed re-accumulation of the subdural collection, with moderate mass effect [Figure 2]. He returned to theatre where again the left sided subdural collection was re-opened, found to be under high pressure, re-drained and send for cytology. In addition a specimen of dura was biopsied.

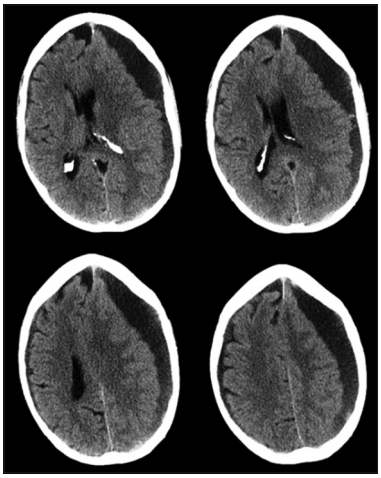

Figure 1: CT head from referring hospital. Left sided subdural collection causing midline shift, effacement of the ipsilateral ventricle, sulci and guri. Small collection is evident on the right side as well

Histopathology revealed connective tissue containing occasional small, cohesive epithelial cell lobules. The cells had pale cytoplasm and round or oval nuclei with medium-sized nucleoli. Immunohistochemistry revealed positive staining for AE1/AE3 (cytokeratin cocktail), prostate specific antigen and prostatic specific acid phosphatase, but not for CK7, CK20, and TTF-1. All these findings were consistent with a metastatic prostate adenocarcinoma with similar histopathological features with the original biopsy. Cytology did not reveal any malignant cells.

The patient had a slow and fluctuating neurological recovery. Further CT of his head revealed re-accumulation of the subdural fluid [Figure 3]. He was discussed in the regional neuro-oncology multidisciplinary meeting (MDM) and it was decided to be refereed for whole brain radiotherapy (WBR). Four weeks later, and while still inpatient, he received 20 Gy in 4 fractions of 5 Gy, delivered in five consecutive days. Despite WBR, the patients ECOG performance status deteriorated from 2 to 3. He was discussed again in the neuro-oncology MDT meeting and the decision was made for best supportive treatment and palliative care. After a total inpatient stay of 5 weeks, he was discharged to a hospice for palliative care management where he succumbed to his disease one month later.

DISCUSSION

While not an uncommon finding at postmortem examination, the first case of prostate dural metastasis was reported in 1981 and since then there have been 16 cases plus a case series of 10 patients published. Similarly, cases of dural and leptomeningeal metastatic prostate disease masquerading as chronic subdural hematoma are sparse within the literature.

The incidence of dural metastases in patients with advanced systemic has been reported as frequently as 8–9% in postmortem series.[5] The proposed mechanisms of seeding include direct hematogenous spread, extension from bony skull metastases and rarely even extension from a cerebral metastasis. Direct hematogenous spread in other tumors is thought to be predominantly via the arterial route while somewhat unique to prostate cancer is via the cerebrospinal venous system as described by

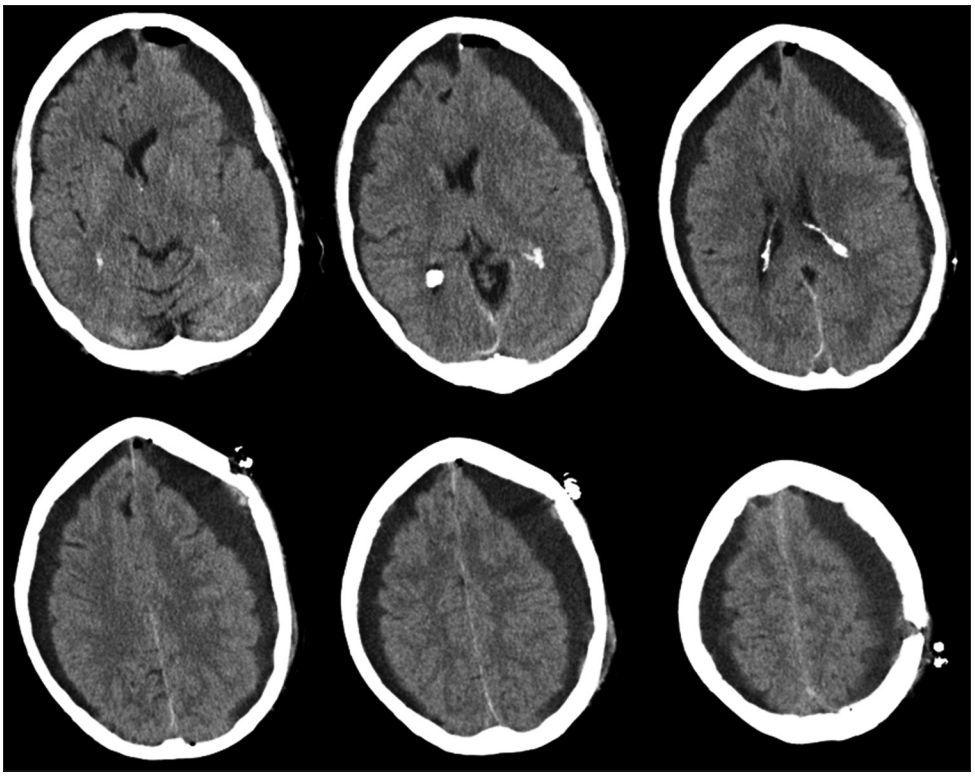

Figure 2: CT head, day 5 postoperative. Evident re-accumulation of the previously drained left sided subdural collection with frontal and parietal burr-holes causing mass effect. The right side collection is bigger causing mass effect and effacement of cerebral surface as well

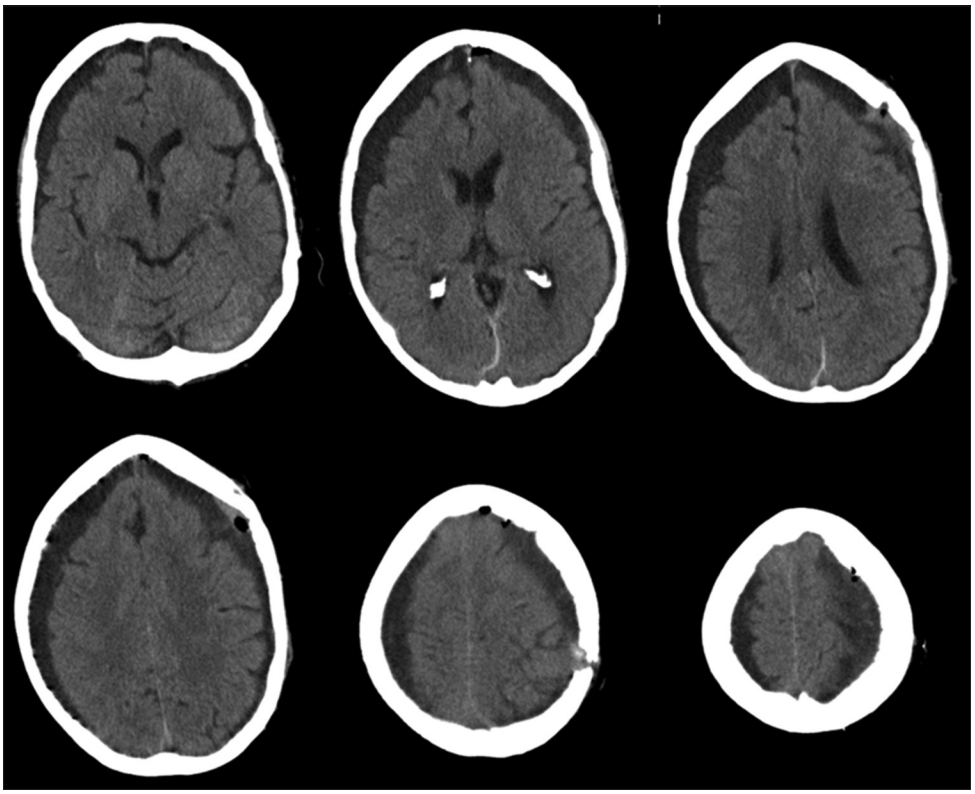

Figure 3: CT head postsecond drainage. Reaccumulation of subdural collection on left hand side and stable collection on the right side

Batson.[1] Within this system there is bidirectional flow facilitated by the absence of valves, thus providing a theoretical route for the cranial progression of metastatic disease seen in these patients. This theory

has gained credence over the years due to the presence of concomitant vertebral metastases, often multiple and extensive in the cases reported.[6]

In a comprehensive review of the literature pertaining to dural metastases from all cancers by Laigle-Donadey *et al.*, the authors evaluate 198 reported cases of dural metastases from the past 100 years.[5] They found that the most common primary lesion to metastasise to the dura is prostate adenocarcinoma (19.5% of cases), followed by breast (16.5%), lung (11%), and stomach (7.5%). Despite this, the condition still remains very rare in patients with prostate cancer with an incidence of 0.1%.[10]

Subdural hematoma, either chronic or acute, is estimated to occur in conjunction with dural metastases in 15–40% of cases.[5] In addition, a separate but related condition "pachymeningitis hemorrhagica interna" in which the dura is diffusely thickened and hemorrhagic on its internal surface can also mimick CT appearances of subdural hematoma, but is much rarer.[7] In a review published in 2012, George *et al.* found only 55 cases reported in the literature.[4] A relatively high proportion of these (45%) were attributed to cancer-related coagulopathy and thus a mechanism by which these hematomas occur was surmised. In 2011, Reichman *et al.* published a series documenting the experience of a single large oncology unit of subdural hematomas related in patients with cancer.[8] In the 90 patients identified, they found that coagulopathy was the leading etiology. The rate reported, however, was considerably lower at 27%. It should be noted that these data sets included all cancers, including hematological. While it is known that metastatic prostate cancer and some of the treatments employed to control it can cause coagulopathy, it is likely that it has less of a role than previously thought.

It has been suggested that the bleeding that precipitates the subdural hematoma could occur as a result of either: (i) Rupture of fragile neo-vasculature within the metastatic deposits, (ii) Mechanical obstruction of dural veins and consequent dilation of upstream capillary beds, or (iii) Hemorrhagic effusion from metastatic lesions due to an angiodesmoplastic reaction to the metastatic invasion. Other reviewers have questioned this cause and effect sequence and have proffered a mechanism by which the subdural hematoma occurring secondary to brain atrophy and mild trauma, or spontaneously due to coagulopathy, produces a route by which the dural barrier to cancer spread is compromised.[2] The metastatic disease is thus permitted to establish. This route may also help to explain the presence of leptomeningeal carcinomatosis (i.e. involving the pia and arachnoid mater) in a number of the reports to date, though less common still than dural metastases.[3]

Most commonly, subdural hematoma is diagnosed on the basis of an unenhanced CT head scan. In the presence of a history of trauma or known coagulopathy, it would be reasonable to treat patients on this basis. Indeed many of the cases reported, including our own, discovered the presence of dural metastases only upon operation or re-operation. Contrast-enhanced CT has proven beneficial in identifying dural lesions although in the presence of subdural hematoma, especially acute, this can prove difficult.[5,7] An advantage that CT can present, however, is the ability to detect bony involvement. The gold standard investigation is contrast-enhanced magnetic resonance imaging (MRI), which is able to identify dural lesions, inform surgeons regarding their morphology, extent and resectability and can even differentiate leptomeningeal involvement as this characteristically follows the gyral convolutions of the brain. Despite these advantages, dural metastases can be difficult to differentiate from meningiomas using MRI. The advent of new techniques such as MR spectroscopy may improve its application in this regard, although that is yet to be proven.[11] The definitive diagnostic investigation therefore remains histological examination and thus it is vital to biopsy tissue at the time of operation.

A poor prognosis is predicted for patients presenting with dural metastases based on the literature available, although this likely more a reflection of the disseminated nature of the underlying disease than a consequence directly of dural involvement. In their review of cases presenting with subdural hematoma in the presence of dural metastasis, George *et al.* conclude that concurrent coagulopathy results in a dismal prognosis.[4] Compared with similar patients without coagulopathy they report worse outcomes, 88% of coagulopathic patients having a poor outcome compared with 20% in those without coagulopathy. Mortality rates differed similarly (68% vs. 6.7%) as did reoperation rates (63% vs. 25%). In a small series (n = 10) reported by Lawton *et al.*, the median survival from diagnosis of dural metastases was just 6.7 months.[6] The authors of this series hypothesize that the improved survival of patients with aggressive, castrate-resistant prostate cancer due to improved medical therapies, for example, docetaxel-based chemotherapy, is leading to an increase in rare manifestations such as dural metastases and as such we can expect to see a rise in the incidence.

The optimal management of dural metastases is yet to be outlined. There does, however, appear to be a clear consensus that surgical evacuation of related subdural collections is advantageous. This intervention can be lifesaving, is minimally invasive, and can even be performed under local anesthetic in selective patients. Some authors advocate resection of the dural metastasis with the aim of preventing recollection and thus re-operation. While clearly a noble aim, this requires the lesion to be small and discrete when in many circumstances the dural involvement is multiple

or diffuse. The benefit to be gained must be balanced against the risk of surgical morbidity considering the patients in question have advanced malignant disease and a limited life expectancy. Whole brain radiotherapy is another treatment strategy that has been employed to control the dural disease either as an alternative to surgery for unfit patients or as an adjunct. The effectiveness of this is yet to be properly evaluated, however.

CONCLUSION

Dural prostate metastasis mimicking chronic subdural hematoma is rare. It reasonable to suspect it, however, in cases of disseminated prostate malignancy, especially when there is no convincing history of cranial trauma. Biopsy of the dural tissue with cytological examination of the fluid collection is an essential part of the diagnostic workup for such patients, which can markedly change the subsequent management plan.

In the stable patient without gross or rapidly progressive neurological deficit, a contrast-enhanced MRI scan can be used to assess the extent of meningeal involvement and therefore the plausibility of complete resection along with evacuation of the collection.

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