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Review article

Metastatic prostate cancer mimicking a subdural hematoma: A case report and literature review

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ABSTRACT

Occurrences of metastatic prostate cancer imitating a subdural hematoma are limited to a small number of case reports, even though prostate cancer spreads to the dura more than other types of cancer. Here, we present the case of a 64 year-old male whose prostate carcinoma's metastasis mimicked a subdural hematoma, and he suffered a middle cerebral artery stroke. Prostate cancer's high rate of progression to the dura is disproportionate to its relatively low rate of brain metastasis. Furthermore, we explore the potential molecular implications of prostate cancer's propensity to spread to the dura.

1. Introduction

Although prostate carcinoma is the most common malignancy amongst U.S. men only about 1.6% of all incidents of metastasis of prostate carcinoma were found to progress to the brain which is a much lower rate than other cancers [1]. Furthermore, only 0.1% of all cases of prostate carcinoma spread to the dura [1]. Yet, prostate cancer makes up the most dural metastases at 19.2% followed by breast (16.5%), and lung (11%) [2].

It is not uncommon for a dural lesion to mimic a meningioma, but it is very rare for brain metastasis of prostate carcinoma to mimic a subdural hematoma (SDH), with only 12 case reports in the literature [3]. A review of some of these case reports done by Nzokou [4] revealed three main patterns in the CT scans which were a nodule in the SDH, multinodular metastasis surrounded by an SDH, and an extensive en plaque subdural tumor.

We present a case of a 64-year-old male who presented with what appeared to be a SDH which upon surgery was found to be an en plaque metastasis of his prostate carcinoma. Furthermore, we summarize the results of the other 12 case reports in the literature.

2. Case description

2.1. History and examination

Patient JG was a 64-year-old male with hypertension, past history of alcohol abuse, current smoker, remote rheumatic fever, and acute kidney injury with dialysis who was transferred to Strong Memorial Hospital (SMH) ED from an outside hospital (OSH) due to altered mental status, restlessness, headache, nausea, and vomiting. Before coming to the hospital, the patient had a headache with emesis, then had gait unsteadiness and may have hit his head on a wall, or fell, with no loss of consciousness. The next morning, he was found unresponsive, and mumbling incomprehensible words leading to his admission to OSH. Upon further evaluation, a CT scan revealed bilateral hyperdensity, which along with his recent injury, was consistent with a bilateral SDH. He was then transferred to SMH where a physical examination found him to be moderately obese, hypertensive, combative, and in severe distress. Neurological examination found him to be encephalopathic, combative, and not following commands. He also had a mild right

Abbreviations: CT, computed tomography; MCA, middle cerebral artery; ND, not determined; NP, not performed; OSH, outside hospital; SDH, subdural hematoma; SMH, Strong Memorial Hospital; STR, subtotal resection.

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facial droop with conjugate gaze and decreased right upper extremity movement indicating possible stroke although other motor functions were deemed normal.

2.2. Radiographic evaluation

Computed tomography scan was obtained, finding bilateral hyperdensity in the frontal and temporal lobes suggestive of a bilateral SDH (Fig. 1). A small area of subarachnoid hemorrhage was seen at the anterior left temporal lobe with ulcerated plaque of the proximal left middle cerebral artery (MCA) consistent with a MCA stroke (Fig. 1).

2.3. Operative description

Due to the patient's acute neurological decline and CT scan revealing hyperdensity consistent with a SDH the patient was taken to the OR for a decompressive craniotomy for SDH evacuation. A perforator was used to make three Bur holes, one paramedian at the frontal area, one paramedian at the posterior occipital area, and one above the temporalis area about the tragus. A large fronto-parieto-temporal bone flap was removed. The dura was found intact, but under high pressure. We found a thick layer of the organizing granulation/reaction tissue with about 1 to 2 cm thickness underneath the dura which was firmly attached to the dura and did not invade the cortex (Fig. 1). During the removal of the lesion the tumor did bleed with the blood coming from the extracranial supply to the dura. Most of the lesion was removed during surgery.

2.4. Pathologic findings

We dissected the dura with organizing tissues and sent it for pathology. The frozen report sent was consistent with carcinoma metastasis with organized SDH. We further removed the thick dural organization with the tumor, and hemostasis was obtained.

The sections revealed a variably differentiated adenocarcinoma with diffuse growth, large ducts and acini as well as multifocal necrosis and brisk mitotic activity (Fig. 1). Tumor cells were present in unlined channels in the dura. Also present is remote hemorrhage and fibrosis. Tumor cells show extensive PSA and PAP, but no TTF-1, napsyn cytokeratin7, cytokeratin 20, CDX2, Pax-8, cytokeratin 5, P63, chromogranin or CD56 immunoreactivity. Positive controls for IHC/Special Stains and negative tissue elements were both evaluated and are adequate for diagnosis.

3. Discussion

Brain metastasis occurs in over 40% of cancer patients and if left untreated after diagnosis patients have a median survival time of 1–2 months which increases to 6 months with treatment [5]. In terms of incidence, the most prevalent of brain metastases are lung (19.9%), melanoma (6.9%), renal (6.5%), breast (5.1%), and colorectal (1.8%) [5]. Yet for cancer spreading to the dura, prostate cancer is most prevalent at 19.2% followed by breast (16.5%) and lung (11%) even though only 1.6% of all prostate carcinoma travels to the brain [2,1]. Therefore, the dura seems to be more appealing on a molecular level to the prostate than other types of cancer.

Brain metastasis begins with epithelial cells undergoing a change into more invasive mesenchymal cells (EMT process) and then intravasate into the blood stream. Once inside the brain, the epithelial cell must settle in as a "seed", induce angiogenesis, and undergo a mesenchymal to epithelial transition (MET) to activate the growth of the tumor [5]. It is important for the particular brain tissue to support the circulating tumor cells (CTCs) for implantation and the MET process.

Considering the origin of prostate cancer is epithelial basal cells there may be a receptor or cytokine attraction to the dural

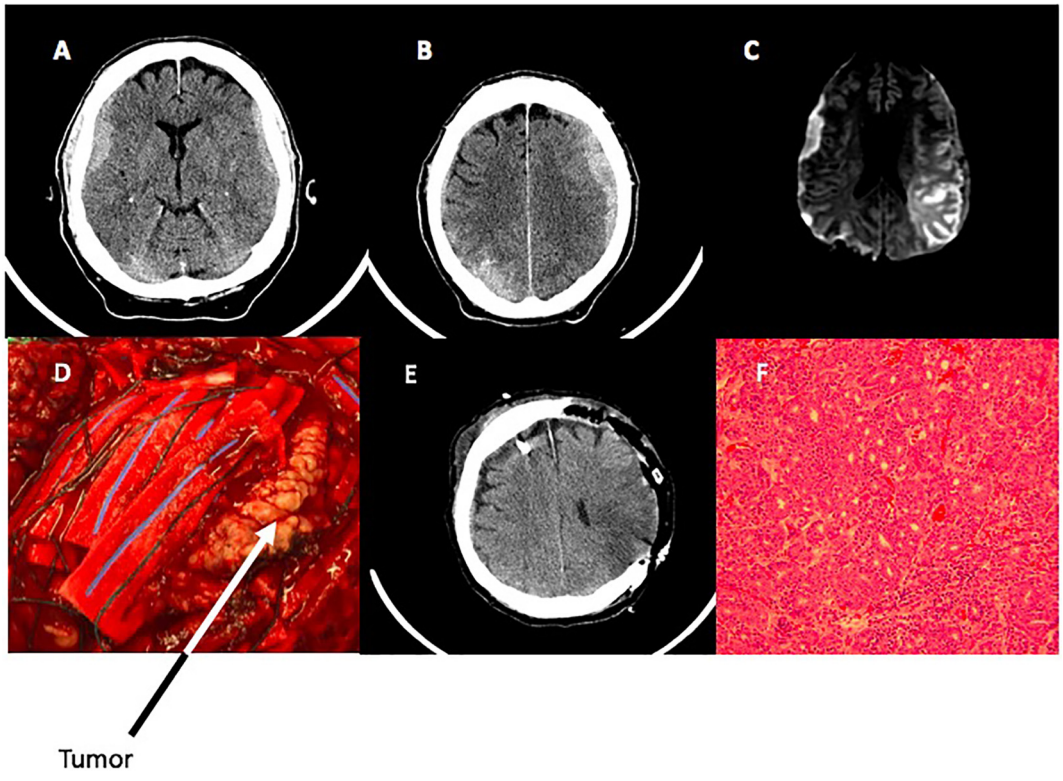

Fig. 1. Imaging: Images A&B are pre-operative CT scans whose axial view shows bilateral hyperdensity suggestive of bilateral subdural hematomas. (C) Pre-operative MRI scan whose axial view illustrates what appears to be a MCA stroke. (D) Intra-operative photo of the brain with arrow pointing to the tumor. (E) Post-operative CT scan in axial view. (F) Characteristic glandular pattern of prostatic adenocarcinoma with focal chronic inflammation. Hematocylin and Eosin, original magnification × 200.

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