Carcinoma Prostate Presenting as Multiple Cranial Nerve Palsy

Krishnan Mugundhan1, KV Arasi2, N Balamurugan3, P Chandrasekaran3, K Thiruvarutchelvan4, S Sivakumar5

Abstract
Metastatic prostatic carcinoma commonly involves bones and extra pelvic lymph nodes. CNS involvement is unusual and particularly the occurrence of leptomeningeal metastasis is extremely rare, with few cases described in the literature.1 The reported incidence at autopsy vary from 0.6 to 4.4 percent.2 We report a 65 year old male who presented with multiple cranial nerve palsies due to leptomeningeal metastasis in carcinoma prostate treated with orchidectomy and planned for radiotherapy and antiandrogen therapy.

Introduction
Brain metastasis occurs in 25% of patients with malignancies and 50 percent of neoplasms in the brain are metastatic.3 The most common sources of metastasis to the brain are carcinoma lung, breast, kidney and melanoma.4,5 Brain metastasis following carcinoma prostate is rare. We present a 65 year old male who presented with multiple cranial nerve palsies due to leptomeningeal metastasis in carcinoma prostate.

Case Report
65 year old male, non smoker, presented to us with dysphagia with nasal regurgitation, and diplopia since 1 month. On examination, the patient was conscious, oriented, afebrile. He had cervical lymph node enlargement and left lateral rectus palsy. Pupil 3 mm equally reacting to light on both sides. Fundus examination was normal. Left gag reflex was sluggish (Figure 1). Tongue wasting and weakness was noted on both sides (Figure 2). Left 6th, left 10th and bilateral 12th cranial nerves were involved. Patient did not have any meningeal signs.

There was no limb weakness. All deep tendon reflexes were normal. Plantars were flexor. Sensory and cerebellar systems were normal. On rectal examination, a hard prostatic mass was felt.

Blood investigations including blood biochemistry vasculitic profile (including ANA) and X-ray chest were normal. ENT opinion revealed no lesion in nasopharynx. Ultrasonogram of abdomen and pelvis showed enlargement of prostatic gland (4.6 x 4.7 cm). Serum prostatic specific antigen was more than 1000 ng/ml. MRI Brain (plain and contrast) was normal. MRI (T1 Sagittal) Dorsal spine showed multiple well defined hypointense sclerotic secondaries (Figure 3). Fine needle aspiration cytology (FNAC) of left cervical lymph node showed metastatic deposits (Figure 4).

Histopathological examination (HPE) of prostatic gland biopsy showed irregular small round to oval glands lined by columnar cells with mild to moderate nuclear pleomorphism and hyperchromatism. There are other foci showing sheets of tumour cells with diffuse infiltration of stroma. These features were suggestive of grade 2 adenocarcinoma of prostate (Figures 5 and 6). CSF analysis showed 40 cells/cu mm which were predominantly mononuclear cells with mildly elevated protein. CSF sugar was low at 20 mg/dl. Diagnosis of leptomeningeal metastasis (multiple cranial nerve palsies) due to carcinoma prostate was made, based on the hypercellularity, elevated protein and hypoglycorrhachia of the CSF. Patient was subjected to orchidectomy and planned for radiotherapy and antiandrogen therapy.

Discussion
The most common sites of prostate cancer metastasis include the bone, lung and liver.6 Brain metastasis is very rare in prostate cancer. The
reported incidence at autopsy vary from 0.6% to 4.4%. The intracranial sites of prostate cancer metastasis are the leptomeninges, cerebrum, and cerebellum. On the other hand, the diagnosis of metastatic prostate carcinoma to the brain has rarely been made in living patients. Brain metastasis in prostate cancer occurs late in the course of the disease. It usually represents the failure of hormone-deprivation therapy and the presence of disseminated disease. The long time between diagnosis and brain involvement strongly favors the cascade theory of tumor spread.

Metastasis to the brain can occur by way of Batson’s plexus or by direct extension from adjacent structures such as the sphenoid bone or sinuses. Other primary cancers, such as lung and breast tumors, are more likely to have intraparenchymal metastases than leptomeningeal involvement. Patients rarely present with neurologic symptoms as the first manifestation of prostate cancer. Presentation with a solitary brain metastasis as the only site of prostate cancer spread is even rarer. Leptomeningeal metastasis is usually clinically silent, although it can present with deficits in multiple anatomic sites. Gadolinium-enhanced MRI is required to exclude or confirm the presence of brain metastases. A two-week course of radiotherapy is the most common treatment for patients with multiple brain metastases or leptomeningeal involvement. Brain metastasis is associated with a poor prognosis. Once prostate cancer has spread to the brain, the one-year survival rate is 18 percent, with an average survival of 7.6 months.

Our patient presented with multiple cranial nerve palsies. The FNAC of the cervical lymphnode was a clue for metastatic lesion. Inspite of extensive metastasis, there is no evidence of cord compression. CSF analysis showed 40 cells/cumm which were predominantly mononuclear cells with elevated protein. CSF sugar was low at 20 mg percent.

The diagnosis of leptomeningeal metastasis (multiple cranial nerve palsies) due to carcinoma prostate was made based on the hypercellularity, elevated protein and hypoglycorrhachia of the CSF as per category 3 in the National Comprehensive Cancer Network (NCCN) guidelines. This is the most plausible explanation for cranial nerve involvement in our case.

Conclusion

Our emphasis is on the protean clinical presentations of carcinoma prostate with no clues to the primary site of involvement. A high index of suspicion is needed when an elderly male approaches us with such a clinical presentation.