Prostate Cancer Presenting as Collet-Sicard syndrome

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60 year old male, non smoker, non alcoholic, not a known diabetic or hypertensive presented to us with dysphagia with nasal regurgitation, 6 months duration. On examination, the patient was conscious, oriented, Fundus examination was normal. Right side tongue atrophy was noted (Figure 1). Right side palatal palsy was present (Figure 2). wasting of sternomastoid (Rt) and Wasting of trapezius (Rt) was also present (Figures 3 and 4). There was no limb weakness. All deep tendon jerks were normal. Plantars were flexor. Sensory and cerebellar systems were normal. On rectal examination, hard prostatic mass was felt.

Blood investigations including blood biochemistry were normal. Serum alkaline phosphatase was 98U/l. Ultrasonogram of abdomen and pelvis showed enlargement of prostatic gland (4.3 x 4.6 cm). Serum prostatic specific antigen was 650 ng/ml. MRI Brain showed secondaries in clivus (Figure 5). MRI Dorsal spine showed multiple well defined hypointense sclerotic secondaries (Figure 6). 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 adenocarcinoma of prostate.
Fig. 7: 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 adenocarcinoma of prostate (Figure 7). Diagnosis of prostate cancer presenting as Collet-Sicard syndrome was made. Patient was subjected to orchidectomy and planned for radiotherapy and antiandrogen therapy. Patient is on follow up.

Collet-Sicard syndrome is caused by lesions at the base of the skull affecting the lower cranial nerves. It is associated with various etiologies of tumoral and other origin. This case is unusual because of the diagnosis of bone metastasis (Collet-Sicard syndrome) secondary to prostate cancer. The clinical presentation of metastasis to the temporal bone is uncommon, and few cases have been reported. Vázquez et al. described 21 cases, of which 71% were secondary to neoplasia. The potential mechanisms responsible for metastatic dissemination to the temporal bone include hematogenous dissemination, direct invasion by carcinoma of the head and neck, and leptomeningeal carcinomatosis. The management of Collet-Sicard syndrome consists of treatment of the etiology. A high index of suspicion is needed when an elderly male approaches us with such a clinical presentation. This case is being presented for its rarity.

References