Squamous Cell Carcinoma of External Auditory Canal Metastasis to CSF: A Diagnostic Conundrum

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Summary

Squamous cell carcinoma (SCC) metastatizing to leptomeninges is rare. We report a case of immunocytochemistry proven cerebrospinal fluid (CSF) involvement in a diagnosed case of SCC of external auditory canal (EAC). This report discusses the diagnostic challenges in cyto-morphological diagnosis, major differential diagnosis and underscores the need to look for leptomeningeal involvement at the earliest due to its prognostic importance despite remote clinical possibility.

Keywords: Squamous cell carcinoma, external auditory canal, CSF

Introduction

CSF cytology is a routinely practised test due to its simplicity, diagnostic ability and prognostic significance. In oncology practise, CSF examination is commonly performed to rule out Central Nervous System (CNS) infection in view of chronic immunosuppression, in dwelling vascular catheter, head and neck surgeries and CNS metastasis which affects the therapeutic management and clinical outcome.[1, 2] Presence of tumor cells in leptomeninges or CSF away from primary tumor is defined as leptomeningeal metastasis (LM).

Around 5-15% of haematological malignancies and 5-8% of solid tumors involve leptomeninges as late complication.[3,4]

Metastatic carcinomas from lung, breast, gastrointestinal tract are commonly reported.[3] SCC involving CSF is extremely rare with few case reports mentioning different sites of head and neck region as primaries.[4] Incidence of SCC of EAC is rare with a reported incidence of between 1 to 6 cases per million population per year and metastasis to CSF is unreported.[5]

Case Detail

A 40-year male patient presented to emergency department (ED) with complains of left sided facial weakness and hoarseness of voice. MRI brain revealed peripherally enhancing hypo intense lesion with absence of perilesional edema measuring 15x8mm in left temporal lobe suggesting possibility of brain abscess. (Figure 1) Patient was diagnosed case of SCC of left EAC a year back and on chemotherapy with regular follow up showing signs of improvement. Tumor did not show any direct extension into the brain parenchyma (Figure 1).

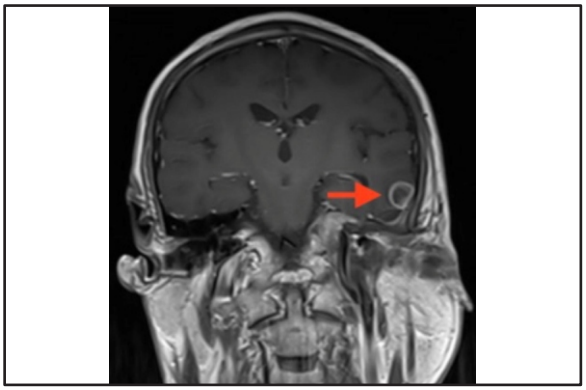

Figure 1: MRI Coronal view shows ring enhancing lesion and absence of perilesional edema suggestive of brain abscess

Presently patient had no complains of fever and on examination signs of meningitis were not elicited. Microbial etiology was suspected as the patient had already completed 5 cycles of carboplatin and paclitaxel, lumbar puncture was performed and CSF tapped was subjected for biochemical, microbiological and cytological examination.

CSF Examination

CSF sample collected by lumbar puncture was around 6ml and clear in appearance. Biochemical examination revealed hypoglycorrhachia (15.34 mg/dl), raised protein (176.11 mg/dl), chloride of 121.6 mmol/dl. Wet mount preparation suggested hyaline round structures similar to size of RBCs and considered a possibility of cryptococcal infection. However, CSF culture was sterile after 14 days of incubation.

CSF Cytology

Cytoprep and papanicolaou stained smears

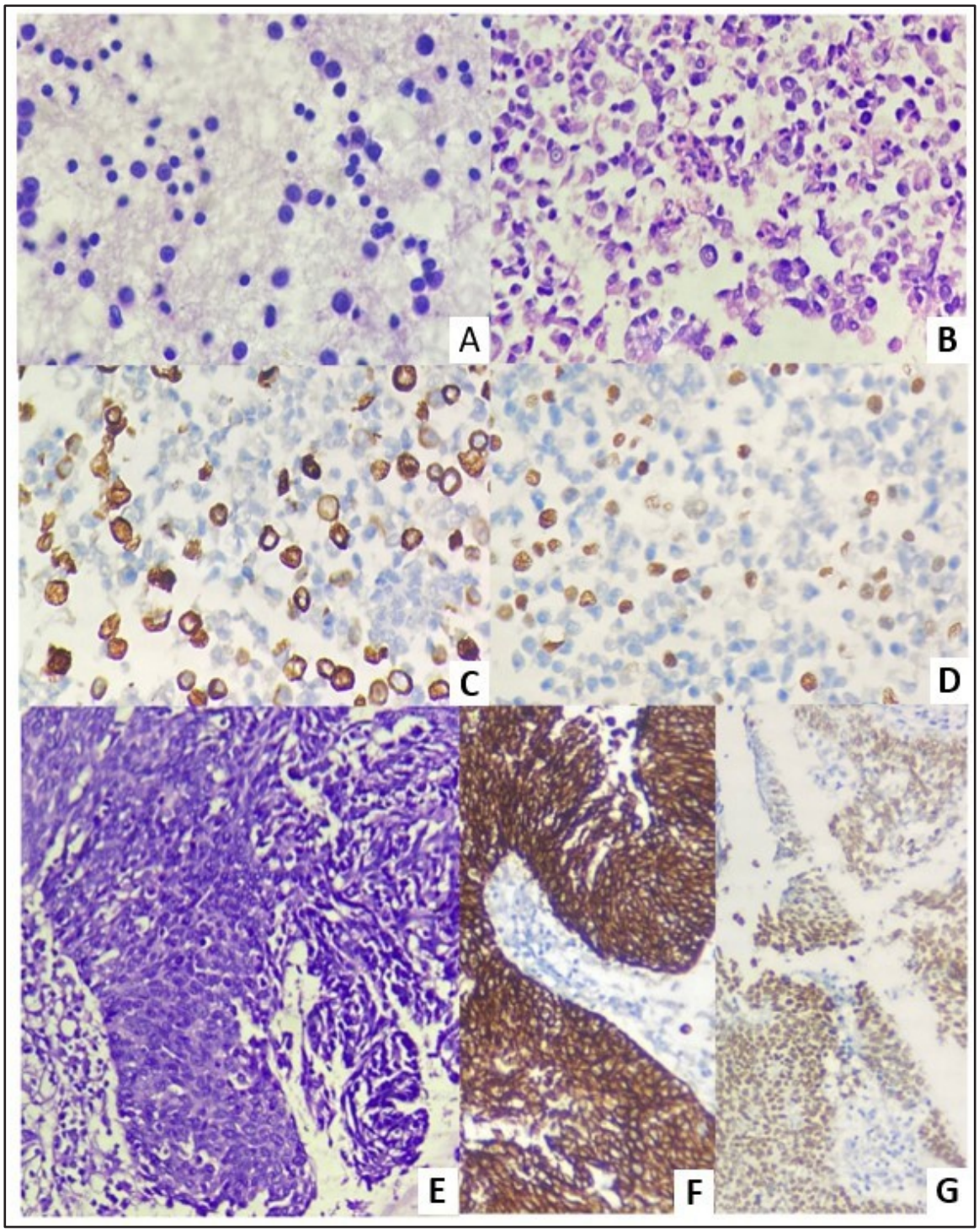

Figure 2: Pap stained cytospin smear shows tumor cells in loose aggregates and scattered singly with uniform round to oval hyperchromatic nuclear and minimal to moderate amount of cytoplasm with scattered lymphocytes in background (A) x20. H&E stained cellblock section is cellular and shows tumor cells with hyperchromatic and vesicular nuclei and moderate amount of cytoplasm (B) x20. H&E stained biopsy from EAC show tumor cell in cord like fashion with basaloid morphology and intercellular bridges are appreciated (E) x20. AE1/AE3 shows membranous staining pattern and p63 shows nuclear staining in (C) and (D) respectively which corresponds to the staining pattern on the biopsy slide from EAC in (F) and (G) respectively.

examined were highly cellular with malignant tumor cells arranged in clusters and scattered singly. Tumor cells had variable nuclear size, regular nuclear contour, round to oval nuclei, fine chromatin and mild to moderate amount of cytoplasm. (Figure 2A) Tumor giant cells and few bizzare cells were noted amidst scattered lymphocytes and macrophages. Structures resembling microorganisms was not identified. Cell

block prepared by fixed sediment method and stained by hematoxylin and eosin revealed pleomorphic cells having fine to vesicular nuclei and moderate amount of cytoplasm with plenty of artifactual cytoplasmic vacoulations. (Figure 2B)

Discussion

Carcinomas metastatizing to CSF is a complication in the natural course of disease and is seen in 5-8% of solid tumors.[3] LM spread from SCC is exceedingly rare with case reports from various sites of head and neck (lip, oropharynx, larynnx, nasopharynx), lung, genitourinary and skin. Occurance of SCC in EAC is quite uncommon and metastasis to CSF is unreported based on Pubmed search.

In this case, clinical and radiologic findings had a very minimal suspicion of metastasis to CSF. Cyto-morphologically also the tumor cell sin CSF failed to show any squamous features such as thick, dense cytoplasm with well-defined cell borders or keratin pearls. The common primaries such as of adenocarcinoma from lung or GIT was considered but glandular morphology of tumor cells and supporting radiological findings were absent. No evidence of moulding was seen to suspect small cell carcinoma lung. Possibility of melanoma was ruled out due to absence of large cells with macronucleoli or melanin pigment.

Since the tumor morphology at the primary site was predominantly basaloid (Figure 2E), the metastatic spread was confirmed by the same immunopanel applied on the biopsy. AE1/AE3 and p63 confirmed the squamous nature of the tumor and reinforced the fact that primary was from EAC. (Figure 2C-D, 2F-G). Generally, identification of malignant squamous cells in CSF is limited and require repeated tapping in addition it is uncommon to encounter the characteristic cytology which adds to the diagnostic difficulty.[3] Despite immunocytochemistry aids in diagnostic accuracy, is limited if the cellularity is low.

Several mechanisms of CSF involvement have been postulated with perineural mode commonly described in SCC metastasis.[2] The mode of CSF involvement in our case has occurred as direct extension through temporal bone.

The incidence of leptomeningeal involvement is increasing as a price paid for longer survival in cancer patients and improvised neuroimaging studies has led to increased detection frequency.[3] LM of SCC carries dismayed outcome and is generally less sampled due to its varied clinical presentation and delayed clinical suspicion. Nevertheless, diagnosis at the earliest warrants a palliative care which carries a benefit of prolonged survival.[6]

Even in the era of remarkable advances in diagnostic field with MRI, DNA amplification, flow cytometry, mere cytological examination of CSF continues to remain gold standard for its simplicity and diagnostic ability.[7] In our case, simple and cost effective CSF examination with immunocytochemistry led to the diagnosis of this exceedingly unusual and probably the first reported work of basaloid SCC of EAC metastasizing to CSF.

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