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A role of PSMA PET/CT in multimodality imaging approach in adenoid cystic carcinoma

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Adenoid cystic carcinoma is a rare disease and characterised by slow but unrelenting local progression and risk of haematogenous metastases. We present a case of locally unresectable disease where PSMA PET/CT provided complementary staging and early treatment response assessment.

Introduction

Adenoid cystic carcinoma (ACC) is rare, but accounts for approximately 30% of all salivary gland tumours.\(^1\) We present a case of an unresectable hard palate ACC with intracranial peri-neural spread (PNS) where serial PSMA PET/CT studies provided early treatment response assessment.

Case report

A 56-year-old female presented with a 6-month history of right-sided V2 paraesthesia and eyelid heaviness. Biopsy at the greater palatine foramen revealed ACC.

A pre-treatment MRI confirmed an enhancing mass in the right hard palate, adjacent to the greater palatine foramen, with evidence of PNS in the right infraorbital nerve, pterygopalatine fossa, vidian nerve, foramen rotundum, trigeminal ganglion and pre ganglionic trigeminal nerve. At the right foramen ovale there was mild asymmetrical enhancement surrounding V3, but the nerve was similar in size to the left and was not considered conclusive for disease involvement.
Staging PSMA PET/CT demonstrated asymmetrical increased PSMA uptake in the right palate, pterygopalatine fossa, superior and inferior orbital fissures, cavernous sinus, trigeminal ganglion and right foramen ovale. A small area of increased PSMA uptake posteriorly in Meckel’s cave adjacent to right internal carotid artery was suspicious for small volume disease when correlating with a second look of the MRI.

Given the intracranial involvement, definitive radiotherapy was recommended and she completed 66Gy in 33 fractions. In the absence of histological confirmation of V3 involvement, the suspicious areas identified on PSMA PET were incorporated into the radiotherapy target volumes (Figure 1).

Post-treatment PSMA PET/CT studies demonstrated a partial reduction in the abnormal PSMA uptake at 3 months, normalization of the abnormal uptake at 6 months and sustained response with no disease recurrence at 18 months (Figure 2). Post treatment MRI at 3 months demonstrated complete response at the right palate and subtle reduction of abnormal enhancement along V2. Subsequent MRI studies up to 12 months demonstrated stable enhancement at the sites of known abnormality.

Discussion

ACC is characterised by slow but unrelenting local progression, peri-neural extension and risk of haematogenous metastases. Surgery followed by adjuvant radiotherapy is considered a standard of care. Where unresectable, accurate staging, including identification of PNS is crucial to optimise local control with targeted radiotherapy.

Prostate-specific membrane antigen (PSMA) is expressed in normal salivary and lacrimal epithelium as well as in the majority of epithelial tumour cells in ACC. A prognostic value of PSMA expression remains to be explored, with a recent immunohistological analysis suggesting low expression of PSMA may be associated with worsened recurrence-free survival.

Small series have shown a high sensitivity of PSMA PET/CT in ACC. MRI remains the gold standard for imaging PNS, but our case suggests that PSMA PET imaging can have an important complementary role both in staging and assessing early treatment response, where MRI changes can be subtle. Imaging slice thickness on the low dose CT component of PET/CT and physiological PSMA uptake of normal tissue may reduce the diagnostic accuracy. The use of PSMA PET/CT in ACC is currently explorative and confirmatory studies are needed. While this case has demonstrated a promising complementary role of PSMA PET/CT in a patient with ACC, longer follow up will determine if the PSMA molecular response on imaging translates to durable local control.
Other potential explorative areas include screening for metastatic ACC and the use of PSMA-based radiolabelled therapy.\textsuperscript{5, 6}

References


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