Clinical Radiology xxx (2015) e1–e10

Contents lists available at ScienceDirect

Clinical Radiology

journal homepage: www.clinicalradiologyonline.net

Pictorial Review

Patterns of spread of head and neck adenoid cystic carcinoma

F.M. Singh [a,b,*], S.Y. Mak [a], S.C. Bonington [a]

[a] The Christie NHS Foundation Trust, Manchester, UK
[b] North Western Deanery School of Radiology, Manchester, UK

ARTICLE INFORMATION

Article history:
Received 2 September 2014
Received in revised form
14 January 2015
Accepted 23 January 2015

We present a review of head and neck adenoid cystic carcinoma (ACC). Imaging features of the primary tumour, patterns of locoregional spread, and distant metastasis with emphasis on perineural extension and imaging pitfalls are discussed with illustrated examples.

Introduction

Adenoid cystic carcinoma (ACC) was first described by three French authors, Robin, Lorain and Laboulbene in the 1850s. They used the term "tumeur heteradenique" and noted that it was able to spread along nerves.[1] ACC accounts for 1% of all head and neck malignancy and arises in secretory glands,[2] most commonly the major and minor salivary glands of the head and neck. ACC arises most frequently (60%) in the minor salivary glands distributed throughout the head and neck mucosa, the most common locations being the palate, tongue, paranasal sinuses, and nasal cavity.[3–6] The parotid gland is the most common single site of origin (25%).[5] It has also been reported to arise from the external auditory canal,[7] lacrimal gland,[8,9] and orbit.[10] Although primary ACC arises most commonly within the head and neck, it has rarely been described in the trachea and central airways, breast, female reproductive tract, thymus, prostate, oesophagus, and skin.[11]

Characteristically, ACC has a slow but relentless growth rate with overall treatment failure, locoregional recurrence, and distant metastatic rates of 62%, 51%, and 38%, respectively.[12] Recurrence typically occurs late, with disease-specific survival of 89% at 5 years and 40% at 15 years.[13] Metastases are rare at presentation but occur in over 50% of patients over the course of the disease.[5,14,15]

Perineural invasion is defined as tumour cell invasion in, around, and through the nerves.[16] Some authors use the terms perineural invasion and spread interchangeably, and others refer to invasion as the microscopic process and spread as the macroscopic result seen on imaging such as MRI.[17] Perineural invasion is common in head and neck ACC, seen in over 50% of cases on histopathological examination.[18,19] Some studies have shown perineural spread to be under-reported on imaging and detection of its extent is variable when comparing MRI to histology as the reference standard. It ranges from 10 to 100% and appears to depend, at least in part, on whether the actual preoperative report was used or whether the preoperative images were later reported by a head and neck imaging specialist.[19–22]

Tumour reaches distant locations by travelling along the neural sheath in the absence of lymphatic or vascular

* Guarantor and correspondent: F. M. Singh, Wrightington Wigan and Leigh NHS Foundation Trust, Radiology Department, Royal Albert Edward Infirmary, Wigan Lane, Wigan WN1 2NN, UK. Tel.: +1942 778627; fax: +1942 822402.
E-mail address: fionasingh@hotmail.co.uk (F.M. Singh).

http://dx.doi.org/10.1016/j.crad.2015.01.013
0009-9260/

Please cite this article in press as: Singh FM, et al., Patterns of spread of head and neck adenoid cystic carcinoma, Clinical Radiology (2015), http://dx.doi.org/10.1016/j.crad.2015.01.013

invasion. This is thought to be facilitated by the expression of neural adhesion molecules.[16,23,24] Extension most frequently occurs centripetally, towards the central nervous system, but can also occur peripherally. Perineural spread is also seen in other malignancies such as squamous cell carcinoma[20,25,26] and melanoma, particularly the desmoplastic subtype.[14,23,27] The radiologist plays a crucial role in detecting perineural spread as it is frequently asymptomatic.[17,25]

The mainstay of treatment is surgery and postoperative radiotherapy.[28] Perineural invasion of major nerves, positive margins at surgery, and solid histological subtype are associated with treatment failure.[13]

Imaging the primary tumour

MRI is the optimum imaging technique due to its high soft-tissue contrast, although it is not specific for distinguishing ACC from other head and neck primary tumours[14,29] (Fig. 1).

Suggested MRI sequences

Routine use of intravenous gadolinium and fat-suppression sequences aids detection of locoregional spread. Axial and coronal planes are most useful for assessing skull base involvement. Unenhanced T1-weighted (T1W) images are important for assessing fat planes and fat-containing structures such as the pterygopalatine fossa and bone marrow for low signal tumour infiltration.[14,30]

Our regional guidelines recommend the routine use of thin-section axial T1 and T2, coronal T2 STIR (short tau inversion recovery), and axial and coronal T1 post-gadolinium with fat saturation with additional sagittal sequences for midline disease.

Imaging features of primary acc

Primary ACC tumour can have a variable appearance at MRI and CT. It can present as a well-defined mass or an ill-defined mass with diffuse infiltration of surrounding structures.[31] The latter has a tendency to invade fat and bone with a permeative pattern rather than present as an expanding mass[14] (Fig. 2). ACC typically enhances homogeneously, although heterogeneous enhancement and necrosis can also be a feature of this tumour.[31] Three histological subtypes have been described: cribriform, solid, and tubular.[3,5] The more cellular, solid form has lower signal on T2W imaging and carries a worse prognosis.[29]

Although ultrasound is frequently used in the initial detection of ACC within the major salivary glands, there are no specific ultrasound features to distinguish ACC from other head and neck tumours (Fig. 3).

Patterns of perineural extension

The trigeminal (V) and facial (VII) nerves are commonly involved due to their distribution at sites where primary head and neck ACC frequently arises. The maxillary (V2) and mandibular (V3) branches of the trigeminal nerve are most frequently involved in perineural spread and allow a route of tumour infiltration into the

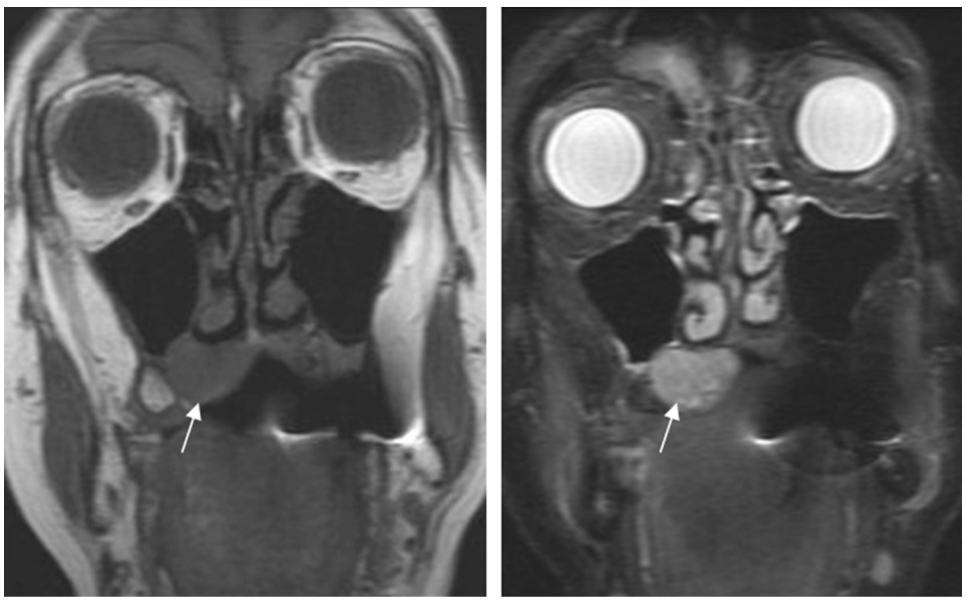

Figure 1 Coronal MRI images of a patient with primary ACC of the hard palate. (a) T1W and (b) STIR images from the same examination demonstrate a well-defined mass within the right hard palate, which has low signal on T1W and intermediate to high signal on STIR images (arrows).

Please cite this article in press as: Singh FM, et al., Patterns of spread of head and neck adenoid cystic carcinoma, Clinical Radiology (2015), http://dx.doi.org/10.1016/j.crad.2015.01.013