Parotid gland tumours: CT, MRI features and histopathologic correlation

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Authors: H. Tie¹, S. Sim², S. Bhuta³; ¹UPPER MOUNT GRAVATT/AU, ²BRISBANE/AU, ³SOUTHPORT/AU
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Learning objectives

1. To describe anatomy and embryology of the parotid gland and its relationship to the facial nerve.
2. To discuss multimodality imaging modalities in the characterization of parotid gland tumours.
3. To illustrate common benign and malignant parotid tumours with correlation of imaging findings with histopathology.

Background

6% of all head and neck tumours arise within the salivary gland and up to 80% of these tumours are found in the parotid gland. 70-85% of parotid gland tumours are benign in nature.

Despite their frequently benign nature, surgical excision is the treatment of choice in these tumours. The differentiation between a benign and a malignant tumour and the extent of the lesion is therefore critical in treatment planning. Surgeons rely on this information to decide between performing a partial paratidectomy or extracapsular dissection for a benign lesion; or a more aggressive total or radical parotidectomy, with or without a facial nerve resection, block dissection of lymph nodes and bony resections in a malignant tumour.

Although clinical features like pain, overlying skin, facial nerve and lymph node involvement suggest malignancy, it is often difficult to distinguish a benign lesion from a malignant tumour clinically. Obtaining a histopathological diagnosis before surgery is also often challenging. Even in the most experienced hands, it is arduous to perform a biopsy of parotid gland tumours as sampling frequently has to occur deep to normal mucosa, where the tumour may occur and it carries an inherent risk of tumour dissemination. After a successful biopsy, due to the complex histopathological features of the parotid gland, a definitive diagnosis may still not be achieved. Even in the best case scenario where a histopathologic diagnosis has been achieved, there are other features of the tumour which are critical in treatment planning and can only be ascertained with medical imaging.
Critical features in the assessment of a parotid tumour:

1. Intraglandular vs. extraglandular tumour
2. Relationship to the facial nerve
3. Benign vs. malignant tumour
4. Invasion of surrounding structures

Due to the large variety of pathology and multiple available modalities, imaging of the parotid gland tumour is challenging.

**The parotid gland**

**Early Development**

The salivary glands arise from the epithelial lining of the oral cavity in weeks 4-6 of embryonic life and the parotid gland is the largest of the major salivary glands. The secretory and parenchymal tissues of the parotid gland arise from the ectoderm whilst those of the rest of the salivary glands arise from the endoderm. The parotid glands develop first, followed by the submandibular and sublingual glands and epithelial cords branch from the enlarging epithelial buds before becoming excretory ducts whilst the terminal buds differentiate into acini.

The mesenchyme surrounding the epithelium then forms a capsule between the lobes and lobules. Even through the parotid anlagen are the first to be developed, they become unsheathed in the loose condensation of connective tissue. The lymphatic system's embryogenesis occurs after the encapsulation of the submandibular and sublingual glands but prior to that of the parotid gland - thus the parotid gland is the only salivary gland that incorporates lymphatic tissue.

During the encapsulation of the parotid gland, salivary epithelial cells may also be included within the intraparotid and periparotid lymph nodes and thus salivary gland tumours may be found in lymph nodes without being found in the major salivary glands.

During the embryogenesis of the parotid gland, the parotid duct anlagen also extends between the divisions of the developing facial nerve, resulting in the extension of the facial nerve branches into parotid gland tissue [Figure 1]

**Normal Anatomy**
The parotid is a pair of lobulated glands located between the mandibular ramus, the mastoid and styloid processes of the temporal bone, extending to the oropharynx. It is invested with a fibrous capsule derived from the deep cervical fascia and has superficial and deep lobes which are divided by a plane created by the facial nerve, which exits the skull base from the stylo mastoid foramen, enters the gland between the mastoid and styloid processes and branches to the face [Figures 2, 3, 4].

Once the facial nerve enters the parotid gland, it divides into temporofacial and cervicofacial divisions. The temporofacial division then further divides into the temporal branches - innervating the auricular muscles and most of the orbicularis oculi, the zygomatic branches - supplying the rest of the orbicularis oculi and nasal muscles, and the buccal branch - supplying the buccinator and lip muscles. The cervicofacial division branches into a marginal mandibular branch - innervating the muscles of the lower lip and a cervical branch. The nerve branches eventually communicate in a plexiform arrangement.

The zygomatic arch, mandibular ramus and styloid process are the three main surgical landmarks used in the intra-operative identification and therefore, preservation of the facial nerve. The stylomastoid foramen is identified to locate the main trunk of the facial nerve and which is then followed distally.

Images for this section:

**Fig. 1**: The development of the parotid duct around the facial nerve divisions.
Fig. 2: Anatomy of the salivary glands.
Fig. 3: CT scan showing the distinction between the superficial and deep lobes of the parotid gland. The image on the left shows the boundary of the two lobes as a line extending from the stylomastoid foramen to the posterior mandible. The image on the right showed a change of the boundary to the sternocleidomastoid muscle to the anterior mandible.

Fig. 4: Facial nerve branches.
Currently, Computer Tomography (CT) and Magnetic Resonance Imaging (MRI) are commonly used as they have a >99% sensitivity in detecting parotid gland tumours.

Imaging of the salivary gland tumors often includes a baseline CT study. Dual phase contrast technique is ideal in achieving contrast enhancement of the solid tumors as well as lymph nodal involvement. Ultrasound has a limited role and can help to distinguish solid from cystic tumors and is useful in image guided biopsies for preoperative planning. PET-CT has a role in demonstrating distant metastatic disease.

MRI of the suprahoid neck in evaluation of the parotid neoplasms is the modality of the choice. MR has superior contrast resolution and true multiplanar capability provides critical information for Head and Neck surgeon in planning the surgical approach. MR scores over CT in detecting early perineural spread along facial nerve and auriculo-temporal nerve. The extent of facial nerve involvement, extracranial or intracranial can be seen with high resolution 3.0 T MR Neurography protocol utilising T1 W fat saturated images. Surgeons often decide there operative technique on MR findings like, perineural spread, skin infiltration, extension to the carotid space and external auditory canal.

MRI is superior than CT in identifying small tumours, differentiating parapharyngeal space lesions from deep parotid lobes and enables better tissue characterization - which has been further improved with the utilization of dynamic contrast enhanced MRI (DCE-MRI), diffusion-weighted MRI (DW-MRI) and apparent diffusion coefficients (ADC) values. Its greatest advantage is its ability to demonstrate perineural dissemination, which is critical in prognosis and outcome. The basic MRI sequences consist of:

1. T1 weighted sequence - useful in the demonstration of parotid gland anatomy and tissue characterization.
2. T2 weighted sequence with fat saturation - useful in the detection of cystic lesions, infiltration into surrounding structures and inflammation.
3. T1 weighted sequence enhanced with gadolinium - useful in the evaluation of tumour margin and extent and perineural spread

**Parotid Tumours**

The World Health Organization classifies salivary gland tumours into benign epithelial tumours, malignant tumours, soft tissue tumours and haematolymphoid tumours. 80% of salivary gland tumours occur in the parotid gland and of these, 80% are found in the superficial lobe and 70-85% are benign in nature. [Figure 5]
In general, benign tumours tend to be well encapsulated, uniformly enhancing focal lesions whilst malignant tumours tend to have poorly defined margins and have inhomogeneous enhancement.

The following parotid tumours are some of the most commonly encountered in clinical practice and will be further discussed:

1. Pleomorphic Adenoma
2. Warthin's Tumour
3. Mucoepidermoid Carcinoma
4. Adenoid Cystic Carcinoma
5. Acinic Cell Carcinoma
6. Metastatic Squamous Cell Carcinoma

**Benign Tumours**

Benign parotid tumours are generally well defined and have a lobulated surface and intratumoral vascularity. Cystic or haemorrhagic changes may also be found in tumours that are 3cm or larger. Calcification may be present in long standing tumours.

**1. Pleomorphic adenoma**

Pleomorphic adenoma, a mixed tumour of salivary gland origin, is the most commonly occurring benign parotid tumour and originate from myoepithelial cells. They comprise of epithelial components within a myxoid stroma. They are usually found in the middle aged population and present as a slow growing, asymptomatic parotid mass, usually confined to the superficial lobe. As it has a local recurrence rate of up to 50% and 15-20% undergo malignant changes when left untreated, wide surgical excision - a partial or total parotidectomy, is the treatment of choice.

On CT, they have well defined margins and tend to demonstrate high, homogeneous enhancement following the administration of contrast. Dystrophic internal calcifications may sometimes occur and are well demonstrated on CT. [Figure 6]

On T1 weighted MRI, pleomorphic adenomas tend to have intermediate signal intensity and an inhomogeneous high signal intensity within a hypointense capsule on T2 weighted MRI. The areas with high signal intensity on T2 weighted imaging were found to correspond to myxoid areas on histology. They enhance following gadolinium administration - a useful feature to differentiate them from cystic spaces in benign tumours. [Figure 7]
Pleomorphic adenomas are also known to seed during surgical resection and recurrence may occur after surgery. [Figure 8]

There is a diverse range of histopathologic features of pleomorphic adenoma. They are comprised of a capsule, epithelial and myoepithelial elements, mesenchymal and stromal components. Some pleomorphic adenomas may be mucoid in nature - with a predominantly mucoid mesenchymal component whilst others may be more cellular - with the bulk of the lesion being composed of the epithelial element, or lipomatous in nature - with a largely lipomatous stroma. [Figure 9]

2. Warthin's Tumour (Cystadenolymphoma)

Warthin's tumours are the second most common benign epithelial parotid gland tumours and are the most common salivary gland tumour to present bilaterally with synchronous metachronous foci (10-15%). They arise from heterotopic parotid tissue occurring within parotid lymph nodes. They present as small (<4cm), asymptomatic, slow growing masses and are most common in elderly men.

On CT, they commonly appear as smoothly bordered heterogeneous lesions due to cystic components. [Figure 10]

On MRI, Warthin's tumours often have a heterogeneous signal intensity MRI - a low to intermediate signal intensity on T1 weighted imaging and intermediate to high signal intensity on T2 weighted imaging. They show none or only slight enhancement post gadolinium administration and this property has been regarded as a useful diagnostic feature. [Figure 11]

They do not have the potential for malignant degeneration but can recur. [Figure 12]

Warthin's tumour are comprised of cystic and solid epithelial and lymphoid elements within a capsule. The papillary structures may have lymphoid stromal cores and extend into the lumina. [Figure 13]

Malignant Tumours

The overall incidence of salivary gland malignancies is estimated to be 4-26 cases per million and mostly affect the parotid gland. The most common primary tumour is the mucoepidermoid carcinoma and metastatic tumours account for around 5% of salivary gland malignancies - majority in the parotid gland and of that, 80% originating from head
and neck squamous cell carcinoma or melanoma. Haematogenous spread from lung or breast carcinoma are less commonly encountered.

Poorly defined, heterogeneous tumours are often attributed to a malignant or metastatic disease. Any infiltration into the parapharyngeal space, vascular encasement, perineural spread, lymph node and bony involvement found on imaging is also indicative of malignancy.

**Primary malignancy**

### 3. Mucoepidermoid carcinoma

Mucoepidermoid carcinoma, a mixed epidermoid and mucus secreting tumour, is the most common primary parotid gland malignancy (80-90%). They tend to present in the middle aged population as a firm, fixed and asymptomatic swelling. These tumours are frequently cystic with a solid component and are categorised into low and high grade lesions.

CT and MRI characteristics depend on the histological grade of the tumour - low grade tumours are usually demonstrated as well defined, homogeneous lesions - similar to benign parotid tumours. High grade tumours appear to be poorly defined and demonstrate heterogeneity, sometimes with an irregular focal necrosis. They have variable enhancement characteristics on CT and MRI but generally low grade lesions display high signal intensity on T2 weighted images and high grade lesions display low signal intensity on both T1 and T2 weighted images. Even though mucoepidermoid tumours produce mucin, high grade tumours do not show a high intensity on T2 weighted images due to the high cellularity of myxoid areas in these tumours. [Figure 14, 15, 16]

Histopathologically, mucoepidermoid carcinomas present with a range of cell types and arrangements but are usually distinguished from other tumours by their mucous producing squamoid cells. They often present with cystic and solid components with mucous cells lining cystic spaces. [Figure 17]

### 4. Adenoid cystic carcinoma

Adenoid cystic carcinoma is the second most common parotid malignancy and have variable morphologic configuration - tubular, cribriform and solid patterns. They typically present as an infiltrating, slow growing mass in the middle aged and elderly population and patients gradually experience pain due to perineural and perivascular invasion. It is
associated with a poor prognosis due to a 50-60% risk of perineural and perivascular invasion as well as a high rate of recurrence.

On CT, well defined, low grade tumours can usually be differentiated from infiltrative, high grade tumours although homogeneous enhancement following contrast is common in both groups. [Figure 18]

MRI is especially useful for these tumours, due to their tendency for perineural spread. They generally have a hypointense appearance on T1 weighted imaging, a slightly hyperintense appearance on T2 weighted imaging and, similar to CT, enhance homogeneously. [Figure 19]

On histopathology, adenoid cystic carcinoma are classified according to their cell types - modified myoepithelial cells or ductal cells. They also display one of three predominant pattern - cribriform, tubular and solid/basoloid. The most common is the cribiform - where groups of cells have microcystic spaces comprising of mucoid components, whilst the tubular type tumour has epithelial and myoepithelial cell lined tubules. In the solid form, basaloid cells are observed to be lined up in sheets. Composite tumours made also be comprised of 2 or more these patterns. [Figure 20]

5. Acinic cell carcinoma

Acinic cell carcinoma is the second most common malignant parotid tumour in the paediatric population. However, it usually presents between the ages of 20-70 and is slightly more common in females. Patients usually present with a slow growing, solitary, mobile parotid mass and >30% of patients complain of pain. However, the tumour may also be multinodular, ill defined, with irregular borders and fixed. The tumour cells have serous acinar cell differentiation.

On imaging, they may present as a solid tumour, cystic lesion or mixed, depending on their histological features and may sometimes resemble pleomorphic adenoma. [Figure 21]

The histopathology of acinic cell carcinomas are characterised by serous acinar cell differentiation but may also demonstrate a range of cell types and growth patterns - cystic, follicular, acinar, vacuolated, clear, solid, glandular or intercalated ductal. A common finding in these tumours is lymphoid infiltration of the stoma. [Figure 22]

Metastatic Malignancy
Less than 10% of parotid gland malignancies comprise of metastatic cancer and of these, 40% are metastatic squamous cell carcinoma - predominantly from skin cancers of the head and neck.

6. Metastatic Squamous Cell Carcinoma

Primary parotid squamous cell carcinoma (SCC), with an incidence of 0.3-1.5%, is rarely encountered. Therefore metastatic disease must be considered when a diagnosis of parotid SCC is made. Most patients present with a history of cutaneous SCC of the face or head.

On imaging, metastatic SCC may appear as a well defined, homogeneous lesion, a cystic lesion or an invasive mass as imaging findings are dependent on the degree of differentiation of the tumour. CT and MRI important in the assessment of local invasion, perineural invasion and for staging. [Figure 23, 24]

Histopathological features of metastatic squamous cell carcinoma in the parotid gland and its lymph nodes is dependent on the primary tumour. [Figure 25]

Images for this section:
### Malignant Epithelial Tumours
- Mucoepidermoid carcinoma
- Acinic cell carcinoma
- Adenoid cystic carcinoma
- Carcinoma ex pleomorphic adenoma
- Squamous cell carcinoma
- Clear cell carcinoma, not otherwise specified
- Basal cell adenocarcinoma
- Sebaceous carcinoma
- Sebaceous lymphadenocarcinoma
- Cystadenocarcinoma
- Low grade cribiform cystadenocarcinoma
- Mucinous adenocarcinoma
- Oncocytic carcinoma
- Salivary duct carcinoma
- Adenocarcinoma, not otherwise specified
- Myoepithelial carcinoma
- Polymorphous low-grade adenocarcinoma
- Carcinosarcoma
- Epithelial-myoeplithelial carcinoma
- Small cell carcinoma
- Large cell carcinoma
- Lymphoepithelial carcinoma
- Sialobastoma

### Benign Epithelial Tumours
- Pleomorphic adenoma
- Warthin’s Tumour
- Oncocytoma
- Myoepithelioma
- Basal cell adenoma
- Canalicular adenoma
- Sebaceous adenoma
- Lymphadenoma
- - Sebaceous
- - Non-sebaceous
- Ductal papilloma
- - Inverted ductal papilloma
- - Intraductal papilloma
- Cystadenoma

### Soft Tissue Tumours
- Haemangioma
- Lipoma

### Haematolymphoid Tumours
- Diffuse Large B-cell lymphoma
- Hodgkin Lymphoma
- Extranodal marginal zone B-cell lymphoma

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**Fig. 5:** WHO classification of salivary gland tumours
**Fig. 6:** Axial and coronal CT showing a well circumscribed, homogeneous pleomorphic adenoma with peripheral enhancement around the right parotid tumour.

**Fig. 7:** MRI of right parotid pleomorphic adenoma - Focal lesion exhibiting intermediate signal on T1 weighted imaging, heterogeneous signal of T2 weighted imaging with areas of high signal and intermediate signal, with a hypodense periphery. There is further enhancement of the high signal regions after the administration of contrast.

**Fig. 8:** MRI of extensive recurrent left parotid pleomorphic adenoma 1 year post left parotidectomy - Multiple hyperintense nodules in left parapharyngeal space and left parotid bed, some demonstrating mild enhancement.
Fig. 9: Pleomorphic adenoma with abundant chondromyxoid stroma and benign epithelial and myoepithelial components. There is adjacent parotid parenchyma present.
**Fig. 10:** Axial and coronal CT demonstrating a right Warthin's tumour - well circumscribed, slightly heterogeneous, no evidence of infiltration into surrounding tissues.

**Fig. 11:** MRI of Warthin's tumour - Complex lesion in left superficial parotid gland: T1 weighted imaging exhibiting intermediate and hypointense signal. T2 weighted imaging exhibiting hypointense, intermediate and hyperintense signals. There is mild enhancement after the administration of gadolinium, but with some non-enhancing regions.
**Fig. 12:** MRI of recurrent right parotid Warthin’s tumour 5 months after surgical excision of initial Warthin’s tumour - demonstrating intermediate signal on T1 weighted imaging, moderately hyperintense signal on T2 weighted imaging and post contrast enhancement.
**Fig. 13:** Warthin’s tumour composed of oncocytic cells and arranged in trabeculae, papillae and cystic spaces supported by reactive lymphoid tissue.

**Fig. 14:** Axial and coronal CT of left parotid mucoepidermoid carcinoma - Left parotid lesion with a large low density centre and a thin peripheral area of soft tissue density.

**Fig. 15:** MRI of mucoepidermoid carcinoma - Left superficial parotid gland lesion with a central, non enhancing area and extensive surrounding enhancement.
Fig. 16: MRI of a mucoepidermoid carcinoma - Poorly defined tumour involving both superficial and deep lobes of right parotid gland with a similar appearance to parotid gland tissue on T1 weighted imaging - intermediate signal. On T2 weighted imaging, it demonstrates a mildly hyperintense signal. There is moderate post contrast enhancement.
**Fig. 17:** High grade mucoepidermoid carcinoma with extensive squamous differentiation. There is adjacent overlying skeletal muscle.

**Fig. 18:** CT adenoid cystic carcinoma - well defined lesion in the left deep parotid lobe.

**Fig. 19:** MRI adenoid cystic carcinoma: Well defined, lobulated and calcified mass lesion arising from the deep lobe of the parotid on the left with extension into the left para pharyngeal space. Low signal intensity on T1 weighted images, heterogeneous hyperintensity on T2 and post contrast heterogeneous enhancement.
Fig. 20: Adenoid cystic carcinoma with both cribriform and solid patterns. The tumour has an infiltrative edge and tumour is seen within adipose tissue of parotid parenchyma.
Fig. 21: MRI of acinic cell carcinoma - An encapsulated, well defined lesion in the right superficial parotid gland demonstrating a mixed signal on all sequences, with focal areas of low signal consistent with calcification.

Fig. 22: Acinic cell carcinoma with a cystic and follicular pattern. There is adjacent parotid parenchyma.
**Fig. 23:** CT of metastatic SCC of a patient with a history of skin SCC on the head - Well defined focal lesion with homogeneous moderate post contrast enhancement in the right parotid gland.

**Fig. 24:** MRI of metastatic SCC of a patient with a history of skin SCC of the head - well defined, homogeneous, focal lesion with a smooth outline in right superficial parotid gland with intermediate signal on T1 weighted imaging, moderately intense signal on T2 and moderate enhancement following contrast administration. No perineurral infiltration in the mandibular or facial nerve.
Fig. 25: Semi-encapsulated metastatic moderately differentiated squamous cell carcinoma within an intraparotid lymph node, with focal extranodal spread into adjacent parotid gland parenchyma.
Conclusion

CT and MRI are important in the evaluation of parotid tumours. They provide vital information, for treatment planning, extent of the tumour, bone invasion etc. MR adds diagnostic specificity and can aid in distinction of benign and malignant masses. Perineural spread along facial nerve, overlying skin infiltration and spread to the external auditory canal are better characterised on MRI.

With development of new techniques especially, high resolution diffusion-weighted imaging and ADC values can assist in predicting tumor biology and may change the clinical practice and patient outcome in near future.

Personal information

Han Tie
Medical Officer
Dept of Medical Imaging
Gold Coast University Hospital
The University of Queensland School of Medicine,
Brisbane
Contact: Han.Tie@health.qld.gov.au

Sarah Sim
Pathology Registrar
Pathology Queensland
Royal Brisbane and Women's Hospital
The University of Queensland School of Medicine,
Brisbane

Corresponding Author
Associate Professor. Sandeep Bhuta

Staff Neuroradiologist

Dept. of Medical Imaging

Gold Coast University Hospital

Griffith University School of Medicine,

Gold Coast

Email - sandeepbhuta@gmail.com

References


