Anatomy and common pathology of the parotid space: The imaging appearance

Poster No.: C-2059
Congress: ECR 2015
Type: Educational Exhibit
Keywords: Abdomen, Head and neck, CT, MR, Diagnostic procedure, Pathology, Neoplasia, Inflammation
DOI: 10.1594/ecr2015/C-2059

Any information contained in this pdf file is automatically generated from digital material submitted to EPOS by third parties in the form of scientific presentations. References to any names, marks, products, or services of third parties or hypertext links to third-party sites or information are provided solely as a convenience to you and do not in any way constitute or imply ECR's endorsement, sponsorship or recommendation of the third party, information, product or service. ECR is not responsible for the content of these pages and does not make any representations regarding the content or accuracy of material in this file.
As per copyright regulations, any unauthorised use of the material or parts thereof as well as commercial reproduction or multiple distribution by any traditional or electronically based reproduction/publication method ist strictly prohibited.
You agree to defend, indemnify, and hold ECR harmless from and against any and all claims, damages, costs, and expenses, including attorneys' fees, arising from or related to your use of these pages.
Please note: Links to movies, ppt slideshows and any other multimedia files are not available in the pdf version of presentations.
www.myESR.org
Learning objectives

The current exhibit aims to:

- To illustrate the imaging anatomy of the parotid space (PS) and its content.

- To illustrate a variety of lesions affecting the PS and their appearance on imaging, using CT and MR techniques.

Background

The PS is the most lateral of the major spaces of the upper suprahyoid neck and as the name suggests is mostly filled with the parotid gland. The parotid gland is the largest salivary gland in humans and is divided into a superficial and a deep portion, which are frequently involved in disease processes (1). Clinical findings have limitations in diagnosing PS pathologies. Therefore, imaging assumes a major role for assessing the precise location and, where possible, indicating the nature of the lesion.

Findings and procedure details

I- Anatomy of the parotid space (2, 3, 4):

The parotid space is one of the seven deep compartments of the head and neck and as the name suggests is mostly filled with the parotid gland. It is the most lateral of the major spaces of the upper suprahyoid neck.

1-Gross anatomy:

The parotid space is a roughly pyramidal space, the broad elongated base facing laterally, formed by cervical fascia overlying the superficial lobe of the parotid gland, and its apex pointing medially (Figure 1). It is traversed by the external carotid artery (ECA), retro-mandibular vein and facial nerve (Figure 2).

2-Contents:
The parotid space contains the parotid glands, intraparotid lymph nodes, the intraparotid facial nerve, the external carotid artery (ECA) and the retro(mandibular vein (Figure 2).

3-Anatomic relationships:

The parotid space comes into contact anteriorly with masticator space, posteromedially with the carotid space, medially with the parapharyngeal space, superiorly abuts the external auditory canal and the mastoid tip and inferiorly with the posterior sub-mandibular space (Figure 1).

4-Boundaries:

The space is circumscribed by the superficial layer of the deep cervical fascia:

The superior margin is represented by the external auditory canal; apex of the mastoid process. The inferior margin is the inferior mandibular margin (although the parotid tail can extend further inferiorly below the angle of the mandible). The anterior margin is the masticator space.

5-Radiographic appearance (2, 3, 4):

* Ultrasound:

With the parotid gland filling around two-thirds of the parotid space, ultrasound findings will reflect those of the intraparotid lymph nodes (may number up into the twenties). These lymph nodes are also evident as rounded/bean-shaped hypoechoic structures with an echogenic central fatty hilum within the hyperechoic parotid gland.

The external carotid artey and retro-mandibular vein will be found posteriorly.

The retro-mandibular vein crosses the superficial lobe of the parotid gland and continues longitudinally until it reaches the inferior margin of the parotid gland to join with the external jugular vein. The external carotid artery travels in the same route; however, it is a larger structure and is found in a deeper plane to the retro-mandibular vein.

The facial nerve is not visualized on ultrasound, but is inferred to be located lateral to the retro-mandibular vein.
* Computed tomography (CT):  

The lower attenuation in comparison to muscle -due to the combination of fat and glandular tissue- allows for good visualization of deep lesions as well as reliable assessment of size, location, margins, extra-capsular extension, involvement of adjacent structures, and areas of necrosis, hemorrhage or calcification of cysts (Figure 3).

* Magnetic resonance imaging (MRI):  

The parotid space has a higher signal than muscle on T1-weighted images and lower signal than muscle on T2-weighted images (Figure 4).

MRI is preferred for imaging the parotid space in children. In fact, neonates and young children have limited amounts of fat within the parotid gland which decreases the amount of natural contrast available on CT, increasing the difficulty to perceive a mass lesion and define its margins.

The MRI is significantly less affected by the relative lack of native fat.

6-Division of the parotid space (2):  

The parotid space is divided into superficial and deep compartments. The true dividing line between these compartments is the facial nerve (CN7), but the nerve is not visible radiologically, so an imaginary line between the stylo-mastoide foramen and the lateral margin of the retro-mandibular vein serves as a radiologic surrogate.

The deep parotid space compartment lies anterior to the styloid process and lateral to the parapharyngeal fat.

II- Lesions of the parotid space (2, 5):  

Most of the pathologies related to parotid space arise from the parotid gland, which may be involved by various neoplastic or non-neoplastic pathologies.

In the setting of a parotid space mass, the key findings are:
* Benign versus aggressive margins,
* Unifocal versus multifocal,
* Homogeneity versus heterogeneity,
* Involvement of facial nerve (2).

The most important consideration is multiplicity:

- For multiple bilateral lesions, the diagnosis should be discussed are: the Sjögren syndrome, the Warthin tumor, the non-Hodgkin lymphoma (NHL), and the systemic metastasis.

- For the multifocal unilateral lesions, the diagnosis should be discussed are: the primary parotid lymphoma and the regional metastasis.

- For the solitary intraparotid lesion, the diagnosis should be discussed are: the Benign mixed tumor (BMT) and the Warthin tumor (2).

1-Malignant tumors:

a-Parotid muco-epidermoid carcinoma (2, 5, 6):

Parotid muco-epidermoid carcinoma is the most common salivary gland malignancy. More than 80% of these occur in the parotid gland. It is most frequently seen in fifth decade of life. It is also the most common salivary malignant tumor in children. The most commonly implicated etiologic factor is radiation (44%).

Histologically, it is composed of varying proportions of epidermoid and mucus-secreting cells. Clear cells, many of which contain glycogen and/or mucin, are present in most parotid muco-epidermoid carcinoma and are a prominent feature. Pathologic grades include low, intermediate, and high.

Recurrence and survival rates depend heavily on histological grade. Late local recurrence after (5years) is possible.
Imaging appearance varies depending on grade of the tumor. On CT, low-grade muco-epidermoid carcinoma may mimic appearance of benign tumor. They have well-delineated, smooth margins with cystic areas and rarely focal calcification.

On MRI, the high grade lesions have indistinct infiltrating margins with invasion of salivary ducts. These cellular tumors tend to have low to intermediate signal intensities on both T1- and T2-weighted images. T2-weighted images may show cystic high signal foci (Figure 5).

b-Parotid adenoid cystic carcinoma (2, 5):

Adenoid cystic carcinoma (AdCC) arises from peripheral parotid ducts and is the second most common major salivary gland malignancy. AdCC is a slow-growing, widely infiltrative tumor with a tendency for perineural spread. It accounts for 2-6% of parotid gland and 30% of minor salivary gland tumors. It occurs predominately in the fourth to seventh decades of life.

Clinically, it presents as a slow growing mass with symptoms of pain. Among all the head and neck cancers, AdCC has the greatest propensity for the perineural spread. Due to its proximity, the facial nerve is most commonly involved with parotid gland.

The WHO classifies AdCC into three microscopic patterns: tubular, cribriform (most frequent), and solid (least frequent).

The imaging appearance widely depends on the site of the origin of tumor. The parotid lesions tend to appear as benign, well-delineated tumors, whereas the minor salivary gland neoplasms usually have malignant infiltrative margins. Lesions show high signal intensity on T2-weighted images due to higher water contents. Retrograde tumor extension (perineural invasion) to the skull base often occurs via the facial nerve or the mandibular nerve (Figure 6).

CT is less sensitive than MRI in diagnosis of perineural invasion. CT findings include nerve thickening, widening of the bony neural canal, and obliteration of the fat in the stylomastoid foramen.

On MRI, fat-saturated contrast-enhanced T1-weighted images are most sensitive to diagnose perineural invasion. This sequence demonstrates enhancement of the affected nerve (facial, trigeminal) with mild thickening of the nerve (Figure 6). Skip lesions may be seen at considerable distances away from the main mass. MRI is equally sensitive in
initial diagnosis of perineural invasion or recurrence following total parotidectomy despite anatomic distortion.

c-Parotid malignant mixed tumor (2, 5):

Parotid malignant mixed tumor is a malignant tumor arising within parotid benign mixed tumor (BMT). There are two types of malignant mixed tumor: the first type is the carcinoma, ex pleomorphic adenoma which is the common type and consists of single malignant cell type, the second type is carcino-sarcoma which is very rare and consists of multiple malignant cell types.

The early malignant mixed tumor (MMT) appears as an encapsulated mass with gross appearance of MBT. The late MMT appears as an extensive aggressive parotid mass with erosion of surrounding structures. MRI is the best imaging technique for extent of lesion, invasion and characterizing tumor regions.

d-Parotid lymphoma (5, 7):

Primary lymphoma of the salivary glands is rare. The diagnosis of the primary lymphoma is considered only if there is histologic proof of involvement of the parotid gland without involvement of the extraparotid lymph node. The prognosis is usually poor because of the high-stage and high-grade tumor.

The most common primary lymphoma is MALT (mucosa-associated lymphoid tissue) type (MALTO). MALTOs show infiltration of small centrocytes and monocytoid B cells in the parotid ductal and acinar tissue. MALT is more common in patients with autoimmune disease (such as Sjögren syndrome and rheumatoid arthritis) and on immunosuppressant therapy. Due to the indolent nature of the disease, the overall prognosis of salivary MALTO is favorable.

Secondary lymphomas of the parotid gland (parotid involvement by systemic lymphoma) are more common than primary.

The CT appearance of secondary lymphoma of the parotid gland varies with the pathologic distribution of the disease. Usually, each lymph node is homogeneous and may enhance slightly on post-contrast CT scans.

Primary parotid lymphoma usually has a nodular, diffuse infiltrative pattern with inhomogenous signal intensity on MRI. As such, it is nonspecific and can mimic chronic
sialadenitis. Diagnosis is established by fine needle aspiration. Systemic workup and bone marrow examination are necessary for staging.

2-Metastatic disease of parotid nodes (5, 8, 9):

Metastases to the major salivary glands comprise around 4% of salivary gland neoplasms. Parotid and periparotid nodes are the first order nodal station for skin squamous cell carcinoma and melanoma from scalp, auricle and face.

On imaging, it is very difficult to differentiate benign enlarged lymph nodes from metastatic. Therefore persistently enlarged lymph nodes require biopsy. If a metastasis to the parotid gland is solitary, it is indistinguishable from a primary high-grade salivary tumor.

Nodes are usually well defined, but may be infiltrative if extranodal spread occurs; they may be heterogenous with central necrosis (Figure 7).

PET and CT are sensitive for identification of small nodes. MRI is more sensitive for extranodal spread. MRI may show single or multiple intraparotid masses with or without central nodal inhomogeneous signal.

3-Benign tumors:

a-Parotid benign mixed tumor: Pleomorphic adenoma (5, 10):

Pleomorphic adenomas (PAs) are the most common benign neoplasms in salivary glands. PAs occur at any age but occur more commonly in patients between 30 to 60 years old and are slightly more frequent in women. Of all PAs, 84% occur in the parotid gland, 90% of which arise lateral to the plane of the facial nerve.

Histologically, this tumor contains epithelial elements, which may be glandular, ductal, or solid, hence the name pleomorphic. Sites of necrosis, hemorrhage, hyalinization, calcification, and rarely ossification may be present.

Clinically, PA is a very slow growing and painless mass tumor. Treatment of the choice is parotidectomy, as the recurrence rate is much higher with enucleation.
On CT, PA is a smoothly marginated, spherical tumor that has a higher attenuation than the surrounding parotid parenchyma. Tumor contour is often lobulated. PA may be totally cystic due to mucoid contents mimicking a cyst on CT. Almost all the PAs enhance on contrast-enhanced CT; the smaller lesions show homogeneous enhancement, while larger masses may show nonhomogeneous appearance, with sites of lower attenuation representing areas of necrosis, old hemorrhage, and cystic changes. Lobulated margin and dystrophic calcifications or ossifications within a parotid mass are highly suggestive of PA.

On MRI, these lesions are low on T1-weighted images and high on T2-weighted images. A low signal intensity capsule is often seen on T2-weighted images (Figure 8, 9, 10). Larger masses show heterogeneous signal intensity due to calcification, hemorrhage, necrosis, and ossification (Figure 11).

b-Adenolymphoma (Warthin's tumor)(5):

Adenolymphoma (Warthin's tumor) is a common salivary gland benign tumor with poor diagnosis and ranking second after the parotid tumors. Presently, a rising tendency of the incidence of adenolymphoma has been noted.

This tumor has characteristic histopathologic appearance: papillary structures, mature lymphocytic infiltrate and cystic changes. They are slow growing, often cystic neoplasms that arise frequently in the lower portion of the parotid gland over the angle of the mandible. They are the most common multifocal or bilateral parotid gland tumor; 25% of them are synchronous, and 75% are metachronous.

On CT, Warthin's tumors are ovoid, smoothly marginated masses, most commonly in the posterior aspect of the superficial lobe of the parotid gland. Tumors often show cystic component. Exophytic Warthin's tumor with cystic component may sometimes mimic branchial cleft cyst or a necrotic node.

On MRI, solid Warthin's tumor may be difficult to differentiate from pleomorphic adenomas. They are well defined and show low signal on T1-weighted images and are hyperintense on T2-weighted images (Figure 12). Cystic component is very hyperintense on T2-weighted images.

c-Parotid schwannoma (2, 5):
Neurogenic tumors (schwannomas or neurofibromas) are the second most common benign mesenchymal neoplasm of the parotid space. Schwannomas are solitary, whereas neurofibromas often are multiple and are seen in association with NF1 (Neurofibromatosis 1). Plexiform neurofibromatosis can also involve the parotid space diffusely. Neurofibroma arises from actual neuronal and perineuronal tissue and shows uniform distribution of spindle cells. Schwannoma arises from supporting neural elements and is located eccentric to the nerve.

Differentiating neurofibroma and schwannoma may be difficult on imaging. However, this distinction is vital, since excision of neurofibroma requires a sacrifice of the nerve; schwannoma can be resected with minimal damage to the nerve.

On CT, these tumors are isodense to muscle with cystic changes and show enhancement on post-contrast scan. The cystic changes are usually small and multiple. Neurofibromas may have a low, almost fatty attenuation that may simulate a lipoma.

On MRI, these tumors have usually low-to-intermediate T1-weighted and high T2-weighted signal intensity. Nonhomogeneous regions of higher and lower signal intensity can occasionally occur, thus making these lesions indistinguishable from a pleomorphic adenoma on MRI.

d-Parotid lipoma (5, 11):

Lipoma is one of the most common forms of benign neoplasm. In the head and neck, it arises mainly in the posterior cervical triangle and forehead. On rare occasions, however, it may occur in either the parotid gland or the parapharyngeal space.

On CT, simple lipomas are discrete lesions with homogeneous low attenuation (65 to 125 HU). Hemorrhage and fibrotic changes are not uncommon, but if present alter their classical attenuation value on CT.

On MRI, lipoma has strong signals on T1 and T2-weighted images and a weak signal on fat-suppressed images; the margin of a lipoma is clearly defined as a "black rim," enabling one to distinguish lipomas from the surrounding adipose tissue (Figure 13, 14).

4-Infectious-Inflammatory lesions:

a-Acute parotitis (2, 5):
There are 4 types of acute parotitis; bacterial, viral, calculus inducted and autoimmune. Acute parotitis is most commonly due to bacterial or viral infections. The bacterial parotitis is a localized infection which may become suppurative, with central abscess, the viral parotitis is usually from systemic viral infection, the calculus inducted parotitis is due to the ducted obstruction by sialolith, and the autoimmune parotitis is an acute episode of chronic disease.

Clinically, patients present with prodromal symptoms followed by an acute, painful swelling of the bilateral glands that may persist for one week or more. Clinical presentation is sufficient for definitive diagnosis, and imaging findings are nonspecific. Imaging is therefore rarely obtained. Treatment is predominantly symptomatic. Parotids appear enlarged with surrounding fat standing. The location is different depending on the type. Bacterial, calculus-induced and autoimmune parotitis are usually unilateral while viral parotitis is usually bilateral.

On CT, parotids appear usually hyperdense and enlarged, with parotid duct calculus usually evident when sought in calculus-induced parotitis and less involvement of surrounding fat in autoimmune parotitis.

On MRI, parotids appear hypointense on T1, hyperintense on T2 and enhance moderately diffusely with sometimes focal areas of high signal on T2 (abscess or dilated ducts) or rim-enhancing fluid lesions (abcess) (Figure 15).

b-Chronic parotitis (2, 5):

Chronic sialadenitis of the parotid gland may be either due to obstructive or nonobstructive diseases. Imaging is most commonly performed to differentiate between these two etiologies. Chronic sialadenitis may be due to infection (eg, bacteria, mycobacteria, syphilis, toxoplasmosis, and actinomycosis) or noninfectious processes like sarcoidosis, prior irradiation, or autoimmune disease. Chronic inflammation results in shrinkage of the gland, which is inhomogeneous on ultrasound. Ductal ectasia may occasionally be seen. Sialadenitis is a common complication of radiotherapy, a common treatment modality for head and neck cancers as well as neck lymphoma. The serous acini of the glands are particularly sensitive to radiation injury, leading to atrophy with xerostomia. Sarcoidosis, a noncaseating granulomatous infection, involves the parotid glands in 10% to 30% of the cases.

Clinically it presents as nontender, nonpainful, chronic enlargement of the gland. On palpation, the gland is often multinodular and may mimic a malignancy. Parotid gland enlargement with sarcoid uveitis and facial nerve paralysis (Heerfordt syndrome) should not be confused with a parotid gland malignancy.
On CT or MRI, sarcoid may either present as a solitary parotid mass (indistinguishable from malignancy) or multiple, benign-appearing, noncavitating masses (Figure 16). There may be associated cervical lymphadenopathy and/or associated pulmonary and mediastinal findings.

c-Parotid Sjögren syndrome (2, 5):

Sjögren's syndrome (SS) is a systemic autoimmune disorder of the exocrine glands that occurs either as a solitary finding (primary SS) or as a conglomerate of connective tissue diseases (secondary SS). SS is characterized by periductal lymphocytic and plasma cell infiltration, leading to destruction of exocrine glands, which causes decreased secretions and dryness. When SS involves the parotid gland, the lymphoid infiltrate produces a localized parenchymal mass referred to as a benign Lympho-epithelial lesion (BLEL) or Godwin tumor. SS is most commonly seen between 40 to 60 years of age, with a striking female predominance (90% to 95%).

Clinically, SS presents with exocrinopathy involving the lacrimal and salivary glands, leading to kerato-conjunctivitis sicca and xerostomia. There may be associated extrasalivary involvement with articular, neurologic, pulmonary, and hepatic manifestations. Although the gland is usually diffusely affected, in some patients a localized area may be more involved, clinically simulating a solitary mass. This finding needs to be further evaluated with biopsy to exclude lymphoma, especially since the risk of developing non-Hodgkin lymphoma in the setting of SS is estimated to be about 44 times greater than in the general population. The primary site of disease in SS is the most peripheral intraglandular ducts and acini.

Imaging appearance depends on stage of disease and presence or absence of lymphocyte aggregates within parotid. On CT and MRI, the gland appears normal in early stages. As the disease progresses to acinar destruction, sialogram may show dilatation of the central ducts and changes of sialoadenitis.

In the later stages, CT demonstrates glandular enlargement with diffuse high attenuation value. As the stages progress, a honeycomb glandular appearance develops on CT, and MRI shows globular enlargement of the parotid ducts with speckled high T2-weighted collection reflecting saliva collections within the dilated ducts (Figure 17). In the advanced stages, the parotid gland shows low signal on T2-weighted images due to focal accumulation of lymphocytes and fibrous tissue.
MRI sialogram (heavily T2-weighted, fast spin echo sequence with spectral fat suppression) has been shown to be useful in the diagnosis of SS, which shows punctate, globular, cavitary, or destructive appearance within the parotid glands (Figure 18). The staging criteria are based on conventional sialography or MR sialography:

- **Stage I**: Punctate contrast/high signal ≤1mm,
- **Stage II**: Globular contrast/high signal 1-2mm,
- **Stage III**: Cavitary contrast/high signal >2mm,
- **Stage IV**: Completed destruction of parotid gland.

### 4-Congenital lesions:

**a-Parotid lymphangioma (2, 5):**

Lymphangiomas probably represent benign proliferative developmental abnormalities involving the lymphatic system. Most lymphangiomas are present at birth, and by the age of 2 years 80% to 90% are present. Generally, the neck is the host site. Occurrence of a lymphangioma in an adult is infrequent. Even more uncommon in the adult is the finding of a lymphangioma in the area of the parotid gland.

Sudden onset of a lymphangiomatous swelling has frequently been reported and may be precipitated by trauma or the inflammation associated with an upper respiratory infection. Spontaneous permanent remission in infants can occasionally occur, but it is not likely in adults. Lymphangiomas are often treated either surgically or with sclerotherapy.

On ultrasound, they are predominantly cystic with septae of variable thickness. When hemorrhage or infection is present, the cysts contain floating debris. On color Doppler, lymphangiomas appear avascular or hypovascularized.

On CT, parotid lymphangioma appears as a nonenhancing homogeneous cystic mass filled with material approaching the density of water. Good definition of the mass and its relation to surrounding anatomy can be obtained, particularly if contrast enhances the wall.

MRI shows fluid levels within the cyst. The multiple, intercommunicating nature of the cysts is better appreciated on T2-weighted images (Figure 19).
**b-Parotid hemangioma (2, 5):**

Hemangiomas are the most frequent salivary gland neoplasm in children. These lesions are classified according to their pathology. Capillary hemangiomas are composed of small capillary-sized vessels with plump endothelial cells, and cavernous hemangiomas are made of large, thin-walled vessels with flattened endothelial cells. The diagnosis of capillary hemangiomas is usually made clinically at or immediately after birth. They represent 90% of parotid gland tumors in this age. Clinically they are nonencapsulated, lobulated, compressible, bluish-colored, soft masses. Most regress spontaneously.

On ultrasound, hemangiomas usually appear as ill-defined, hyperechoic, compressible lesions, or as hypoechoic lesions with a typical lobular pattern. Color Doppler shows hypervascularity.

Contrast-enhanced CT demonstrates an intensely enhancing lobulated mass, enlarging most or all of the visualized parotid gland and extends to the overlying skin. Speckled calcifications (phleboliths) are seen within the mass.

On MRI, the lesion is low to intermediate on T1-weighted images and is hyperintense on T2-weighted images (**Figure 20**). There may be areas of high signal intensity on T1- and T2-weighted images, due to prior hemorrhage and slow flow.

**5-Other lesions:**

**a-Fibromatosis of the parotid gland (2) (figure 21):**

The term fibromatosis refers to a group of fibrous tumors or tumorlike lesions of soft tissue that share similar microscopic characteristics and possess an intermediate biologic potential between benign and malignant lesions. These nonencapsulated lesions of unknown cause take their origin from fascia, musculo-aponeuroses or periosteum, and are locally aggressive in behavior with a tendency for recurrence. They do not metastasize but may kill as a result of local infiltration and extension into vital structures.

The CT findings are nonspecific. Most tumors are of attenuation similar to that of muscle or slightly increased, although some may be hypodense. Lesions usually become more conspicuous after the injection of iodinated contrast material, but the degree of enhancement varies. Most lesions show heterogeneous uptake of contrast material,
whereas others show diffuse homogeneous enhancement and some show relatively little change.

On MRI, lesions may be well defined and nodular, or infiltrative and ill-defined. Most desmoid tumors are heterogeneous soft-tissue lesions of intermediate signal intensity. It may be hypointense, isointense, or occasionally marginally hyperintense with respect to muscle signal on T1 and of mixed predominantly high signal on T2 (Figure 21).

After injection of gadolinium, tumors may show homogeneous, inhomogeneous, or no significant enhancement. No relationship has been shown between the pattern of enhancement and tumor recurrence.

**b-Benign lympho-epithelial lesions-HIV: BLEL (2, 5):**

Benign lymphoepithelial lesions-HIV is bilateral and is shown by painless enlargement of both parotid glands.

In BLEL, CT or MR show multiple, bilateral, well circumscribed, cystic and solid masses within enlarged parotid glands, associated with reactive cervical adenopathy, adenoidal, palatine and lingual tonsillar hypertrophy.

**Images for this section:**
**Fig. 1:** Graphic of the suprahypoid neck soft tissues shows the relationships between the parotid space (green) and the surrounding axial spaces. Notice the masticator space is anterior, while the parapharyngeal space is medial and the carotid space is postero-medial. On the left, the superficial fascia (yellow line) is seen to suscram both the masticator and the parotid spaces.
**Fig. 2:** Axial graphic at the level of C1 body, shows the content of the the parotide space. The intraparotide course of the facial nerve (not seen with imaging) extend from just medial to the mastoid tip to a position just lateral to the retro-mandibular vein. Within the superficial lobe, only parotide tissue and and nodes are present. Within the deep lobe, notice the medial external carotid artery and retro-mandibular vein. The parapharyngeal space fat lies just medial to the deep lobe of the gland.
Fig. 3: Contrast-enhanced axial CT image showing the outline of the right parotid space (marked by red line).
**Fig. 4**: Cervical MRI: axial T2-weighted image: Parotide space.
**Fig. 5:** Parotid mucoepidermoid carcinoma. Cervical MRI: axial (a) T2-weighted and (b) contrast-enhanced fat-saturated T1-weighted images: ovoid and well-circumscribed mass of the right parotid gland. This lesion has an heterogeneous signal with predominantly low signal on T1-weighted images and high signal on T2-weighted images, and enhances heterogeneously after injection of Gadolinium (b).
Fig. 6: Parotid adenoid cystic carcinoma. Cervical MRI: sagittal contrast-enhanced fat-saturated T1-weighted images demonstrate high grade adenoid cystic carcinoma as an ill-defined enhancement in parotid gland infiltrating the cavernous sinus (arrowhead), meninges of the anterior cranial fossa (black arrow) and the tentorium (white arrow). Note the thickening and intense contrast enhancement of maxillary nerve (V2) and the mandibular nerve (V3).
**Fig. 7:** Contrast-enhanced cervico-thoracic CT: (a) coronal and (b) axial images: Right small cell lung carcinoma with costal lysis (red arrow) and metastatic necrotic lymph node in the ipsilateral parotid gland (yellow arrow).
Fig. 8: Pleomorphic adenoma of the superficial lobe of the parotid gland. Cervical MRI: coronal (a) T1-weighted and (b) fat-saturated T2-weighted images, (c) axial contrast-enhanced fat-saturated T1-weighted images and (d) ADC map: lobulated intraparotid mass with low signal on T1-weighted image, uniform intermediate signal on T2-weighted image, intense enhancement and high ADC.
**Fig. 9:** Pleomorphic adenoma of the superficial lobe of the left parotid gland. Cerial MRI: Coronal (a) T1-weighted and (b) fat-supressed T2-weighted images: well circumscribed intra parotid mass with low and heterogenous intensity on T1 and a very high signal on T2 (specific for BMT).
**Fig. 10:** Pleomorphic adenoma in the left parotid gland. Cervical MRI: (a) axial contrast-enhanced fat-saturated T1-weighted images, (b) Diffusion sequence and (c) dynamic enhancement curve: Ovoid and smoothly marginated intra parotid mass with high signal intensity on Diffusion sequence (ADC=1.56), intense enhancement with type A on the dynamic enhancement curve.
**Fig. 11**: Pleomorphic adenoma of the left parotid. Cervical MRI: (a) coronal fat-saturated T2-weighted images, and (b) axial, (c) coronal and (d) sagittal contrast-enhanced fat-saturated T1-weighted images: large and lobulated intraparotid mass with very high signal on T2-weighted images and heterogeneous enhancement.
Fig. 12: Bilateral parotid Wartin tumor. Cervical MRI: axial (a, b) unenhanced T1-weighted, (c) T2-weighted and (d) contrast-enhanced fat-saturated T1-weighted images: tow intraparotid masses. The right mass is ovoid and well circumscribed with an intermediate signal on T1 and intermediate and homogeneous signal on T2. The left mass is lobuled and heterogenous with high signal areas on T1, low signal intensity on T2 and a moderate contrast enhancement.
**Fig. 13:** Lipoma in the superficial lobe of the parotid gland. Cervical MRI: axial (a) unenhanced T1-weighted, (b) T2-weighted and (c) contrast-enhanced fat-saturated T1-weighted images: note the strong signal produced by the tumour on T1 (a)- and T2 (b)-weighted images (yellow arrow), the low signal on fat-suppressed images (c) and the clearly-defined margin of the tumor.
**Fig. 14:** Right parotid lipoma. Cervical MRI: axial (a) T2-weighted and (b) contrast-enhanced fat-saturated T1-weighted images: a well circumscribed hyperintense lesion with attenuation of the signal on fat-suppressed images.
Fig. 15: Acute suppurative parotitis. Cervical MRI: axial fat-saturated (a) unenhanced T1-weighted, (b) T2-weighted and (c) contrast-enhanced T1-weighted images: Left parotid is diffusely enlarged with multiples abcess whose size does not exceed 5mm. They present typically high signal intensity on T2-weighted images and low signal on T1-weighted images with a continuous rim of enhancement.
Fig. 16: Chronic parotitis of the right parotid. axial (a) unenhanced T1-weighted, (b) T2-weighted and (c) contrast-enhanced fat-saturated T1-weighted images:.Note the relatively small volume of the right parotid with heterogeneous and hyperintense signal on T2 without enhancement (yellow arrow).
**Fig. 17:** Parotid Sjogren syndrome. Cervical MRI: (a) axial unenhanced T1-weighted, (b) axial and (c) fat-saturated T2-weighted images: bilateral parotid enlargement with diffuse and bilateral discrete collections, with low signal intensity on T1 and high signal intensity on T2.
Fig. 18: MR Sialography: sialoadenitis classification in Sjogren syndrome. Stage I (A), stage II (B), stage III (C), stage IV (D).
Fig. 19: Parotid lymphangioma of the left parotid gland. Cervical MRI: (a) axial unenhanced T1-weighted, coronal fat-saturated (b) T2-weighted and (c) contrast-enhanced T1-weighted images, (d) Diffusion sequence and (e) ADC map: presence of a multiloculated cystic mass with a diameter of 35 mm in the left parotid gland, with multiple thin septas. This lesion is well-defined, hypointense on T1, hyperintense on T2 and on Diffusion sequence. It has a low ADC, and only septas are enhanced (yellow arrow).
**Fig. 20:** Parotid Hemangioma. Cervical MRI: axial (a) unenhanced T1-weighted, (b) T1-weighted images, axial (c) and coronal (d) contrast-enhanced fat-saturated T1-weighted images: well-defined and lobulated mass (yellow arrow), with intermediate signal on T1 weighted image, high signal intensity on T2 weighted and intense and homogeneous enhancement.
Fig. 21: Fibromatosis of the right parotid gland. Cervical MRI: coronal (a) unenhanced T1-weighted and (b) contrast-enhanced fat-saturated T1-weighted images, axial fat-saturated (c) T2-weighted and (d) contrast-enhanced fat-saturated T1-weighted images: an invasive and poorly marginated mass, with low signal intensity compared to the muscle on T1, heterogeneous signal intensity on T2, and a moderate contrast enhancement. Note the involvement of the masseter muscle (blue arrow) and the right vertical branch of the mandible (yellow arrow).
Conclusion

Understanding the anatomy of the PS and how it links up with the other deep facial spaces helps the radiologist to recognize the different lesions of this space and to avoid unnecessary surgery, or any other than less optimal treatment.

Personal information

References


