Imaging Characterization of scalp and skull lesions

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Learning objectives

- Describe the normal anatomy and the differential diagnosis for skull and scalp lesions presenting palpable lumps and bumps on the head.
- Discuss the importance of complete and appropriate imaging for preoperative assessment of these skull and scalp lesions.
- Know characteristic imaging findings for diagnosing these lesions.
Background

Skull is formed by hard bones and encases the brain. Scalp covers the skull and acts as protective cushion from trauma. A wide spectrum of congenital lesions (eg, encephaloceles, dermoid and epidermoid cysts, and benign tumors) and acquired lesions (eg, sarcoma, Langerhans cell histiocytosis [LCH], metastatic neuroblastoma, infectious or traumatic lesions) are commonly encountered in skull and scalp. Lumps and bumps of the scalp are a common presenting complaint and often offer a diagnostic dilemma. These lesions can be difficult to image and the evaluation confounded by their small size. However, the accuracy in diagnosis is critical because the diagnostic and therapeutic implications can vary significantly.
Normal anatomy

The brain is encased in several protective layers that cushion it from trauma. Beneath the skin and subcutaneous tissues lie the hard bones that form the skull. Skull is formed by hard bones and encases the brain. Scalp covers the skull and acts as protective cushion from trauma.

The scalp has five layers; (Fig. 1)

1) Skin

2) Subcutaneous Connective tissue

3) Gales Aponeurotica

4) Loose areolar connective tissue

5) Pericranium (peiosteum)

The skull is consisted with 28 separated bones, mostly connected by fibrous sutures. Cranium has several parts; clavarial vault, cranial base, facial skeleton.

Congenital Lesions

1. Encephaloceles / Meningocele (Fig. 2-4)

Intracranial tissue that herniates through a defect in the cranium results in an encephalocele. Such lesions are called meningoceles when they contain only meninges and meningoencephaloceles when brain tissue is included in the herniated tissue. They occur in one of every 4,000 live births and are most commonly occipital in location (75% of cases); lesions are frontoethmoidal in 15% of cases and basal in 10%. There are often significant associated intracranial anomalies. Occipital encephaloceles may be associated with Chiari or Dandy-Walker malformations and callosal or migrational anomalies. Frontoethmoidal lesions are not typically associated with these types of anomalies. Frontothmoidal encephaloceles are also known as sincipital encephaloceles and are subdivided into nasofrontal, nasoethmoidal, and naso-orbital types. These encephaloceles are more common in South and Southeast Asian populations. They are found projecting along the nasal bridge between the nasofrontal sutures into the glabella (nasofrontal region), under the nasal bones and above the nasal septum (nasoethmoidal region), or along the medial orbit at the level of the frontal
process of the maxilla and the ethmoid-lacrimal bone junction (naso-orbital region). Frontoethmoidal encephaloceles manifest as a clinically visible mass along the nose. The intracranial root of most frontoethmoidal encephaloceles lies at the foramen cecum, a small ostium located at the bottom of a small depression anterior to the crista galli and formed by the closure of the frontal and ethmoid bones.

Atretic encephaloceles should be mentioned because they are included in the differential diagnosis of skin-covered midline scalp masses in childhood. They are typically parietal in location and contain meninges and neural rests. A vertically positioned straight sinus is commonly associated with these malformations, and anomalies of the tentorial incisura and superior sagittal sinus have also been reported. These malformations are also seen occasionally in the occipital region. Atretic encephaloceles contain a fibrous stalk at their base that connects to the dura mater. Association with intracranial anomalies is variable, and some children may have normal clinical outcomes with no associated intracranial anomalies.

2. Nasal Gliomas (Fig. 5)

Nasal gliomas occur near the root of the nose (where the cranial portion of the nose joins the forehead), are composed of dysplastic glial tissue, and are congenital nonneoplastic lesions best categorized as heterotopia. A nasal glioma may be connected to the brain by a stalk of tissue in up to 15% of cases, but the stalk does not contain a direct fluid-filled tract that communicates with the subarachnoid spaces; therefore, a nasal glioma is distinct from an encephalocele, which does contain such a connection to the intracranial subarachnoid spaces. Nasal gliomas are intranasal in 30% of cases, extranasal in 60%, and mixed in 10%. Extranasal gliomas are usually seen in a paramedian location at the bridge of the nose external to the nasal passage, whereas intranasal lesions are usually located within the nasal passage medial to the middle turbinate bone. Surgical resection is used to treat these lesions. Nasal gliomas are often isointense relative to normal brain at MR imaging, which is the imaging modality of choice. High-resolution surface coil MR imaging is often useful in demonstrating the intracranial stalk.

3. Dermoid & Epidermoid Cysts (Fig. 6)

Dermoid and epidermoid cysts are found in a variety of locations around the skull and midface. They are thought to occur as a result of the persistence of ectodermal elements at sites of suture closure, neural tube closure, and diverticulation of the cerebral hemispheres as discussed earlier. Dermoid cysts contain ectoderm and skin elements, whereas epidermoid cysts contain ectoderm but no skin elements. Dermoid and epidermoid cysts are most commonly seen in midline and frontotemporal locations, followed by parietal locations. Midline locations include the anterior fontanelle, glabella, nasion, vertex, and subocciput. Sutures that are commonly affected include the
frontozygomatic, sphenofrontal, sphenosquamosal, squamosal, coronal, lambdoid, and parietomastoid sutures. Nasal dermal sinuses and dermoid and epidermoid cysts occur at multiple locations from the glabella to the columella. These lesions may be associated with external skin ostia or deep sinus tracts, which may potentially extend intracranially. CT attenuation varies depending on content (eg, fat attenuation with dermoid cysts, fluid attenuation with epidermoid cysts). Similarly, the signal intensity at MR imaging depends on the contents of the cyst and may range from pure fluid signal intensity (hypointense on T1-weighted images, hyperintense on T2-weighted images) in an epidermoid cyst to a more complex signal intensity (hyperintense on T1-weighted images, hypointense on T2-weighted images) in a dermoid cyst. Epidermoid cysts typically have bright signal intensity on isotropic diffusion-weighted MR images. Nasal dermoid cysts may be associated with a sinus tract that extends for variable distances in the prenasal space to the foramen cecum. High-resolution surface coil MR imaging is very useful in determining if there is a connecting tract to the foramen cecum. In such cases, sagittal T1-weighted or heavily T2-weighted images may best delineate the tract running beneath the nasal bones to the foramen. If the fluid is hyperintense on T1-weighted images, the tract will often be seen on both unenhanced T1-weighted images and T2-weighted images. Nasal dermoid and epidermoid cysts are typically surgically resected due to the potential for an intracranial connection and the risk of CNS infection, as well as for cosmetic reasons.

4. Craniosynostosis (Fig 7)

Craniosynostosis is a condition in which one or more of the fibrous sutures in an infant skull prematurely fuses by ossification, thereby changing the growth pattern of the skull. Because the skull cannot expand perpendicular to the fused suture, it compensates by growing more in the direction perpendicular to the open sutures. The resulting growth pattern provides the necessary space for the growing brain, but results in an abnormal head shape and sometimes abnormal facial features. In cases in which the compensation does not effectively provide enough space for the growing brain, craniosynostosis results in increased intracranial pressure leading possibly to visual impairment or an impairment of mental development combined with a significant reduction in IQ.

Craniosynostosis is part of a syndrome in 15 to 40% of the patients, but it usually occurs as an isolated condition.

# Sutures

1) Scaphocephaly - the premature closure of sagittal suture

2) Trigonocephaly - the premature closure of the metopic suture

3) Plagiocephaly - ant. (unilateral coronal synostosis)
- post. (Unilateral lambdoid synostosis)

4) **Brachycephaly** - a closure of both the coronal sutures

(short head)

5) **Pansynostosis** - all of the sutures are closed

(primary microcephaly ; cloverleaf skull )

### 5. Benign Tumors

#### (1) Vascular Lesions

Vascular anomalies of the face and scalp in childhood have generally been classified as either hemangiomas or vascular malformations. Hemangiomas are benign endothelial tumors that undergo cellular proliferation and accompanying enlargement in the 1st year of life, followed by gradual involution during childhood. Vascular malformations, on the other hand, consist of some combination of congenitally abnormal veins, lymphatic vessels, capillaries, or arteries. They tend to enlarge proportionately and progressively with the growth of the child, unless acutely complicated by trauma, hemorrhage, infection, or the hormonal influences of puberty.

1) **Hemangioma** *(Fig 8)*

Hemangiomas are the most common tumor of infancy. It is estimated that they occur in 1%-2% of the population in general and in up to 10% of white persons. They occur more frequently in girls than in boys (3:1 ratio), and their prevalence is higher in premature infants. Hemangiomas are present at birth in 30%-40% of cases, with the remainder generally being appreciated in the first months of life. More than one-half are located in the head and neck, with the most common sites of involvement being the midcheek, upper lip, and upper eyelid. Hemangiomas can be classified as focal, localized to a particular region, or diffuse and segmental. They may be superficial (red in appearance) or deep (flesh colored or blue in appearance). The vast majority are managed clinically without imaging. However, hemangiomas that might affect the airway, disturb vision, or be associated with other anomalies may be imaged. These hemangiomas include cervicofacial or "beard distribution" hemangiomas, which are associated with subglottic hemangiomas, and deep bilateral parotid hemangiomas, which may directly impinge on the airway. Periorbital hemangiomas are often imaged to assess the extent of retro-orbital involvement and the potential for compromise of orbital movement and vision. Diffuse and segmental hemangiomas of the face often trigger neuroimaging for assessment of features of PHACE(S) syndrome. Multiplanar MR imaging provides an operator-independent method of demonstrating the deep and superficial extent of these masses. Typically, proliferating hemangiomas are isointense relative to muscle on T1-weighted
images, have high signal intensity on T2-weighted images, demonstrate homogeneous enhancement, and have internal flow voids. The flow voids are highly suggestive but not a specific feature of hemangiomas.

2) Venous malformation (Fig 9)

Venous malformations include a wide spectrum of dysmorphic and congenital venous lesions and are characterized clinically by a soft and nonpulsatile mass, often of bluish color. Up to 40% of venous malformations occur in the head and neck. MR imaging demonstrates a hyperintense mass on T2-weighted images, with occasional septation and variable enhancement. Phleboliths, which appear as a focal signal void, are a relatively specific characteristic. As a low-flow lesion, the flow voids demonstrated with proliferating hemangiomas or high-flow arteriovenous malformations are not seen in venous malformations. Treatment typically involves some combination of sclerotherapy and surgical removal.

3) Sinus pericranii (Fig 10)

A collection of nonmuscular scalp veins that communicate with the intracranial venous sinuses is described as sinus pericranii. These abnormal veins are often congenital in origin, although some investigators have postulated trauma as the cause in at least some cases. These veins are commonly appreciated in the frontal and parietal regions. Imaging studies show a soft tissue mass that enhances and is accompanied by scalloping of the outer table of the skull. Sagittal images may show the relationship to the underlying dural venous sinus. Treatment usually consists of ligation of the communicating veins and surgical removal of the sinus itself.

4) Lymphatic malformation (Fig 11)

Lymphatic malformations of the head and neck develop from lymphatic sacs that fail to communicate with the remainder of the lymphatic system. They are most commonly appreciated in the posterior triangle of the neck and axilla in the first 2 years of life. Macrocystic lymphatic malformations, sometimes referred to as cystic hygromas, have characteristic MR imaging features, manifesting as multiseptate cystic masses, often with intracystic hemorrhage or fluid levels. Treatment typically involves some combination of sclerotherapy and surgical resection.

5) PHACE syndrome (Fig 7)

PHACE(S) syndrome, which consists of posterior fossa malformations, hemangiomas, arterial anomalies related to the intracranial circulation, coarctation of the aorta or cardiac anomalies, eye abnormalities, and, occasionally, sternal clefting or supraumbilical raphe (a fibrous band or cleft in the midline above the umbilicus).
Acquired Lesions

1. Sarcomas of the Head and Neck

According to the Agency for Research on Cancer, soft-tissue sarcomas account for 4%-8% of cancers in patients up to 14 years of age. Whereas head and neck primary sarcomas represent only 5%-15% of all adult sarcomas, 35% of all sarcomas in the pediatric population manifest in the head and neck.

(1) Rhabdomyosarcoma

Rhabdomyosarcoma is the most common soft-tissue sarcoma of childhood (60% of cases). Rhabdomyosarcomas of the head and neck generally have a better prognosis than those in the extremities. In most studies, approximately one-third of pediatric rhabdomyosarcomas occur in the head and neck region, the most common location. Rhabdomyosarcomas are slightly more common in males, and two-thirds of tumors occur in children less than 6 years of age. The most common histologic type is embryonal rhabdomyosarcoma, which accounts for 70%-80% of cases and is considered to have a more favorable prognosis.

The presenting symptoms of rhabdomyosarcoma are variable and depend on tumor location, patient age, and the stage of the disease at diagnosis. In the head and neck, rhabdomyosarcomas are classified as (a) orbital, (b) parameningeal (including the pterygopalatine fossae, paranasal sinuses, middle ear, and mastoid process), or (c) superficial. At clinical examination, rhabdomyosarcoma may be difficult to differentiate from other soft-tissue lesions. The four cases of rhabdomyosarcoma reported
by Chigurupati et al. were initially misdiagnosed as hemangioma, lymphangioma, lymphoma, and lymphadenopathy, respectively.

At CT, most rhabdomyosarcomas are poorly defined and relatively homogeneous lesions with bone destruction. At MR imaging, they are homogeneous masses that are isointense to minimally hyperintense relative to muscle on T1-weighted images and hyperintense on T2-weighted images. Rhabdomyosarcomas manifest with mild to moderate enhancement at both CT and MR imaging. The bone destruction typically manifests as areas of signal loss in cortical bone with or without bone marrow infiltration. These findings may be seen with both unenhanced T1-weighted MR imaging sequences (which is probably the most useful sequence) and T2-weighted sequences. MR imaging is also the technique of choice in assessing the therapeutic response.

(2) Infantile myofibroma (Fig 12)

Infantile myofibromatosis is rare disease and fibrous tumor that occurs the most commonly in the infancy and early childhood. It is characterized with single or multiple myofibroblastic tumors in the subcutaneous tissue, muscle, bone, occasionally internal organ. Nearly all cases occur under 2 year-old (88%). Myofibromatosis is divided by the number of lesion, into solitary and multicentric type. The prognosis is related with the involvement of internal organ.

The solitary type is mostly benign and has self-limited course. The recurrence rate after operation is about 10 %. CT findings are isodense or hypodense with brain parenchyma. The lesion presents as osteolytic lesion and occurs in the temporal bone and parietal bone. The MRI findings are low SI on T1WI and high or low SI on T2WI, depending on the amount of collagen and necrosis, and strong contrast enhancement.

(3) Cranial fasciitis (Fig 13)

Cranial fasciitis is a rare fibroblastic tumor, which shows a predilection for the scalp of young children. It usually present as a lytic lesion in the skull with associated soft tissue mass. Histologically it shows a loose proliferation of spindle-to-stellate shaped fibroblasts in a myxoid matrix. Cranial fasciitis is commonly presented as a rapidly growing soft tissue mass. The lesion is firm and non-tender or may be soft or tender. The mass is usually under 3.5 cm in the greatest dimension, although it may attain a size as large as 15 cm. The lesion is usually single.

The cranial fasciitis shows mild inhomogeneity, rapid growth in size, marked enhancement, and multiple calcifications within mass on serial CT and MR images. The enhancing soft tissue mass makes saucer-like erosion with intact inner table that don't
appear lytic skull lesion in temporal region. The sequential change is related with it rapid growth, increased myxoid matrix, and calcifications.

(4) Plexiform neurofibroma (Neurofibromatosis) (Fig 14)

The excessive growth of craniofacial plexiform neurofibromas is another cause of cranial complications in NF 1. Plexiform neurofibromas are locally aggressive congenital lesions composed of tortuous cords of Schwann cells, neurons, and collagen in an unorganized intercellular matrix. They tend to progress along the nerve of origin into the intracranial space. Most commonly, plexiform neurofibromas develop in the orbit. The neck is another common location for neurofibromas, with an estimated occurrence of 25% to 30% in patients with NF1.

Solitary neurofibromas have slightly greater signal intensity than skeletal muscle on T1-weighted sequences. On T2-weighted sequences, the lesions have variable signal intensity. Most commonly, the periphery of the lesions tends to be of high signal intensity with respect to muscle, whereas the center of the lesions is often of low signal intensity; this has been referred to as the "target sign". The central area of decreased intensity is probably related to the known dense central core of collagen within these lesions. Collagen has a low mobile proton density and therefore is of low signal intensity on T2-weighted images. The enhancement is variable, although at least a portion of the tumor usually enhances.

(5) Osteoblastoma (Fig 15)

Osteoblastoma is uncommon, 1% of bone tumor and occurs under 30 years and M:F=2:1. It is uncommon primary neoplasm of bone that is composed of a well-vascularized connective tissue stroma in which active production of osteoid and primitive woven bone occurs. It is presented with dull aching pain, soft tissue swelling in tumor site, scoliosis. Pain does not worsen at night and is not relieved by aspirin. The osteoblastoma is distinguished by nidus size (>2cm) with osteoid osteoma.

The Axial skeleton is involved m/c (post. Spinal involvement 35%), long bone (femur, tibia and medulla eccentrically) and diaphysis, skull and facial bone (15% temporal, occipital, ethmoid, frontal, and sphenoid bones), hands and feet (14%) and ribs (4%) respectively.

Radiographic appearance are 1) a circumscribed oval radiolucent defect, with varying degrees of central calcification, involving both the inner and the outer tables of the skull, 2) similar to osteoid osteoma (but less reactive sclerosis and more prominent periostal reaction), 3) Blow-out expansile behavior with multiple central small calcifications, a thin shell of peripheral periosteal bone and well-defined margin -spine, 3) More aggressive with bone expansion and destruction, adjacent soft tissue infiltration, intermixed matrix
calcification -humerus. MR finding is non-specific and low to intermediate signal intensity on T1 WI and intermediate to high on T2 WI.

(6) Lipoma (Fig 16)

Lipomas of the face and scalp are reportedly rare, comprising less than 2% of lipomas. Three such cases have recently been diagnosed in our clinic. Among 110 lipomas in our files, a total of 16 (14.5%) involved the face and scalp. The clinician rarely considered lipoma as the primary preoperative diagnosis. Often mistaken clinically for epidermal cysts, face and scalp lipomas appear more common than the literature would indicate.

2. Langerhans Cell Histiocytosis (Fig 17)

The term histiocytosis refers to a group of disorders that have in common the proliferation of pathologic Langerhans cells, a type of histiocyte from the monocyte-macrophage cell line. LCH has been divided into three forms on the basis of the organs involved, patient age at onset, and clinical course: localized, chronic disseminated, and fulminant-disseminated. Localized LCH is the mildest and most common form (70%-75% of cases) and involves either bone or lung. Lung involvement is uncommon in children, whereas bone involvement is typical and visceral involvement is occasionally seen. LCH corresponds to the so-called eosinophilic granuloma. Peak prevalence of LCH occurs between 1 and 4 years of age, and the disorder has a slight male predilection. Because the localized lesions in this age group are frequently painful lumps misdiagnosed as local trauma or seborrhoeic skin lesions similar to cradle cap, the disease may go undiagnosed. Head and neck manifestations of LCH occur in up to 73%-82% of children during the course of the disease. The skull and the skin are frequently involved and were the most common sites of involvement in the series reported by DiNardo and Wetmore and Irving et al. The calvaria is the most common location of osseous LCH. Other commonly involved sites include the orbit, maxilla, mandible, and temporal bone. At radiography, bone lesions in LCH usually appear lytic. Their borders may be either well or poorly defined, and they are classically described as "punched-out" lesions without reactive sclerosis or periosteal reaction. In the skull, the lesion edges typically have a beveled appearance due to asymmetric destruction of the inner and outer tables of the skull. At CT, LCH appears as an enhancing soft-tissue mass with bone erosion. At MR imaging, lesions have low to intermediate signal intensity on T1-weighted images, are hyperintense on T2-weighted images, and enhance diffusely on contrast-enhanced images. Extensive osseous destruction is often seen at MR imaging as well. A combination of contrast-enhanced CT and MR imaging is often required for accurate long-term follow-up of repair of bone destructive lesions and of resolution of soft-tissue masses.

3. Metastasis
(1) Neuroblastoma

Neuroblastoma is usually a secondary metastatic lesion rather than a primary lesion of the head and neck. It may manifest with Horner syndrome and a metastatic mass at the skull base involving the cervical sympathetic ganglion. Clinical findings related to tumor metastases may be investigated with various imaging modalities. Traditionally, the radiologic evaluation of neuroblastoma has included intravenous urography, US, CT, bone scintigraphy, and metaiodobenzylguanidine scintigraphy. Neuroblastoma commonly metastasizes to the lateral orbital walls, manifesting clinically as two "black eyes" ("raccoon sign"), and to the skull base and calvaria.

Recently, MR imaging has been demonstrated to be superior to CT in staging and determining the extent of these lesions. There is involvement of the bone marrow in up to 56% of patients as reported by Siegel et al. At MR imaging, neuroblastoma lesions appear as focal areas of high signal intensity on T2-weighted images and enhancing lesions on fat-suppressed T1-weighted images. There is a growing body of data supporting MR imaging as the imaging modality of choice for the evaluation of local and regional disease.

(2) SCC / Melanoma / Breast cancer / RCC (Fig 18)

Skull metastases are malignant bone tumors which are increasing in incidence. Primary sites were breast cancer (55%), lung cancer (14%), prostate cancer (6%), malignant lymphoma (5%), and others (20%). The mean time from primary diagnosis to skull metastasis diagnosis was 71 months for cases of breast cancer, 26 months for prostate cancer, 9 months for lung cancer, and 4 months for malignant lymphoma. Calvarial circumscribed intraosseous metastases were found most frequently (27%). The patients were mainly asymptomatic. However, some patients suffered from local pain or cranial nerve palsies that harmed their quality of life. Treatment, mainly for symptomatic cases, was by local or whole-skull irradiation. Metastatic skull tumors are not rare, and most are calvarial circumscribed intraosseous tumors. MR images contribute to understanding their type, location, and multiplicity, and their relationship to the brain, cranial nerves, and dural sinuses.

4. Infectious Lesions

(1) Cellulitis & Abscess (Fig 19)

Frontal sinus infection can spread directly through the thin bone wall of this sinus or through the network of small veins that drain its mucosa. Today, this is a rare complication given the widespread use of antibiotics. Trauma and frontal sinusitis are the most common causes of this condition. The most common causal organisms are streptococci, staphylococci, and anaerobic bacteria. Cultures frequently reveal
polymicrobial involvement. The infection may spread as a thrombophlebitis from the frontal sinus through the diploic veins, involving the intracranial space with consequent epidural or subdural empyema, meningitis, brain abscess, and venous sinus thrombosis.

Once the diagnosis of abscess is suspected, contrast-enhanced CT (bone window) or MR imaging is needed to evaluate for possible intracranial complications. Subtle intracranial involvement is more easily seen at MR imaging. With the injection of gadolinium-based contrast material, one may see early linear enhancement of the dura mater, an extraaxial fluid collection, or an area of cerebritis. In the scalp, peripheral or rim contrast enhancement may be seen when an organized fluid collection is present. Surgical drainage remains the mainstay of therapy. Careful resection of the granulation tissue in the scalp and of infected bone up to the margins of normal bone is important, along with prolonged antibiotic therapy.

(2) Skull Tuberculosis (Fig 20)

Skull tuberculosis is found in approximately 1/10,000 of all tuberculosis cases, and it concerns 0.2-1.37 % of tuberculosis cases affecting the skeletal system. About 50% of the cases reported in the literature were in patients younger than 10 years, and 70-90% were younger than 20 years. The disease is rarely seen in infants.

Tuberculosis of the skull is commonly associated with tuberculosis elsewhere in the body. It is believed that calvarial tuberculosis occurs by hematogenous seeding of bacilli to the diploe. Lymphatic dissemination of tuberculosis, common in other bones, is not though to occur in the skull. Tuberculosis of the skull base usually results from direct extension of paranasal sinus or mastoid disease and only rarely from meningitis.

Three types of lesions are recognized i.e. circumscribed lytic lesions, spreading type (progressive infiltrating type) and circumscribed sclerotic type. "circumscribed lytic lesions" is the most common type that small punched-out lesions with granulation tissue covering both the inner and outer tables of the calvaria. It is associated with little tendency to spread and hence is not associated with a periosteal reaction. The frontal and parietal bones are the most common sites of involvement. The typical radiographic appearance is that of a single or multiple well-demarcated destructive osteolytic lesions.

CT showed lesions involving the entire thickness of the calvarium (involving both inner and outer tables) and accompanying contrast-enhancing soft tissue. A bony sequestrum may also be seen. Several study revealed 90% had subgaleal soft tissue swelling, 30-52% had extradural soft tissue, 85% had calvarial destruction, 5-11.9% had parenchymal involvement, 52% had sinus formation. MRI shows a high signal intensity soft tissue mass within the defect in bone on proton density-and T2-weighted images. Peripheral capsular enhancement on the contrast-enhanced image was shown, indication the presence of capsule.
5. **Traumatic Lesions** (Fig 21-23)

1) Cephalohematoma

- traumatic subperiosteal accumulation of blood confined by the cranial sutures

2) Caput succedaneum

- a hemorrhage within the skin that crosses suture lines and is usually located at the vertex

3) Subgaleal hematoma

- a hemorrhage subjacent to the aponeurosis covering the scalp beneath the occipitofrontalis muscle and is also not confined by the cranial sutures

The prevalence of cephalohematomas is close to 1%-2% in spontaneous vaginal deliveries and 3%-4% in forceps- or vacuum-assisted deliveries. In cases of prolonged resorption, cephalohematomas begin to calcify and have a characteristic clinical and radiologic appearance. They tend to increase in size after birth and manifest as a tense and firm mass, occasionally with an underlying skull fracture. Resolution usually occurs without treatment by a few weeks to 3-4 months of age. Although cephalohematomas are not usually of clinical significance, they may present a challenge for the clinician because they occasionally become infected, requiring drainage and antibiotic therapy. At CT and MR imaging, acute cephalohematomas appear as crescent-shaped lesions adjacent to the outer table of the skull. Chronic cephalohematomas may calcify and appear hyperattenuating at CT. During the evolution of a cephalohematoma, a mixed picture of erosive changes and periosteal reaction can be worrisome, especially in the absence of a good clinical history. At MR imaging, signal intensity typically follows that of subacute hemorrhage (ie, hyperintensity on T1- and T2-weighted images) but may vary depending on the stage of the hemorrhage.

6. **Regional and Focal skull thickening**

(1) **Morgagni Stewart Morel syndrome** (Fig 24)

It is a condition with a wide range of associated endocrine problems including; diabetes mellitus, diabetes insipidus, and hyperparathyroidism. Thickening of the inner table of the
frontal part of the skull is a usually benign condition known as "hyperostosis frontalis interna".
Fig. 0: Fig 1. Scalp Layers of Protection for the brain

© Diagnostic and Surgical imaging anatomy
Fig. 0: Fig. Occipital meningocele. Axial CT of bone window setting (a) shows linear bone defect at bony protuberance. Axial T1-weighted (c) and axial and Sagittal T2-weighted (b,d) MR images reveal an occipital meningocele with no evidence of involvement of venous structures. The lesion consists predominantly of herniated meninges, but no brain tissue was found at surgery.

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Fig. 0: Nasoethmoidal encephalocele. Axial and Coronal CT scans (a,b) demonstrates a nasoethmoidal encephalocele (arrow). The axial and coronal MR images (c, d) show frontal lobe tissue and the meninges extending under the nasal bones and above the septal cartilage.

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Fig. 0: Fig. Vertically positioned straight sinus with persistent fetal anatomy. The sagittal US scan (a) shows echogenic mass with connection into skull. Axial T2-weighted MR image (b) and axial and sagittal (c,d) CE T1WI demonstrate a vertically positioned straight sinus and a fenestrated superior sagittal sinus resulting from deflection around the tract of a histologically proved atretic parietal menigoencephalocele.

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Fig. 0: Fig. Drawings illustrate aberrant development of the nasofrontal region leading to the formation of various midface masses. Schematic A shows that frontonasal encephaloceles form when the fonticulus nasofrontalis remains patent. Schematic B shows that nasal gliomas form when the dural diverticulum involutes late or only proximally through the foramen cecum, leaving sequestered neurogenic tissue that may be connected to the intracranial content by a fibrous stalk. As shown in schematic C, dermal sinuses result from a lack of involution of the dural diverticulum through the foramen cecum. Dermoid or epidermoid cysts may form along the dermal sinus tract due to desquamation of tissue lining the tract.

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**Fig. 0:** Periorbital dermoid cyst. Axial T2-weighted (a), FLAIR (b), T1-weighted (c), and enhanced (d) MR images show a well-defined cyst at the left periorbital area. There was no intracranial connection. The hyperintensity of the lesion on the T1-weighted image reflects the lipid contents of the cyst.
**Fig. 0:** Fig. Metopic synostosis. The sequential axial CT images (a-c) show the keel-shaped forehead, consistent with trigonocephaly. The coronal sutures are open. 3D-CT, oblique view demonstrates typical prominence and ridging along the fused metopic suture.

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**Fig. 0:** Fig. PHACE syndrome. Axial T2-weighted, FLAIR, enhanced T1-weighted MR images (a-c) reveal a periorbital left soft-tissue mass that is isointense relative to brain and an intensely enhancement of left periorbital mass, a finding that is consistent with a hemangioma. There is left cerebellar hypoplasia in FLAIR image (b). The MR angiogram (d) shows left ICA stenosis and the tortuous dilatation of basilar artery.

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Fig. 0: Fig. Scalp venous malformation. Axial (a,b) T2-weighted MR images show a markedly hyperintense mass containing septation and tortuous venous structure in left parietal area. Axial CE images (c,d) reveal variable contrast enhancement, as septa and vascular structures.

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Fig. 0: Fig. Sinus pericranii. Sequential axial enhanced CT images (a-c)) shows a enhancing serpiginous scalp mass, connecting with right transverse sinus. (d) Sagittal venogram multiple, small, transosseous vessels that supply the scalp venous malformation from right transverse sinus.

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**Fig. 0:** Fig. Lymphatic malformation. Axial (a) and coronal (b) T2-weighted and axial T1-weighted (c) MR images demonstrate a macrocystic, multiseptate mass with layered intracystic hemorrhage in the right temporal area. There is the contrast enhancement of multiple septum in the lesion on CE-T1WI (d).

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**Fig. 0:** Fig. solitary infantile myofibroma. The brain US and enhanced CT (a,b) scans reveal enhancing soft tissue mass in the scalp of right temporo-occipital area. The axial T1- and T2-weighted (c,d) images show focal bulging, scalp mass of low SI in right T-O area.

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**Fig. 0:** Fig. Radiography of the skull (a) shows soft tissue mass in right temporal area and saucerization of underlying temporal bone. Follow-up MR image of soft tissue mass in right temporal area (b-d) reveal homogeneous low signal intensity on axial T1-weighted image (b), heterogeneous high signal intensity on T2-weighted image (c), and relatively homogenous dense enhancement with non-enhancing necrotic portion on contrast enhanced image (d).

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**Fig. 0:** Multiple scalp plexiform neuromas in NF1 patient. The axial T2-weighted MR images (a,b) show multiple nodules of high SI with central core of low SI (Target Sign). The axial and sagittal CE-T1WIs (c,d) reveal the contrast enhancement of central core in the multiple scalp nodules.

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Fig. 0: Fig. Skull osteoblastoma. The axial T1-weighted and axial and sagittal T2-weighted MR images reveal round mass of intermediate SI on T1WI and low SI on T2WIs. This mass is well enhanced on sagittal CE-T1WI.

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Fig. 0: Scalp lipoma. The axial CT scans (with bone window) reveal low attenuated mass in left occipital area (HU = -99).

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Fig. 0: Fig. LCH of the skull. (a) Radiograph of the skull shows a typical punched-out lesion of the right parietal bone. (b-d) Axial T1-weighted, T2-weighted, and enhanced MR images demonstrate focal nodule of low SI on T1WI and high SI on T2WI, and strong contrast enhancement of nodule within skull bone defect in right parietal area.

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**Fig. 0**: Fig. Chronic cephalohematoma. Axial unenhanced CT scan (with bone window) shows a partially calcified mass with fluid-fluid level in the periosteum of the right parietal bone. The mass originated from a cephalohematoma.

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Fig. 0: Fig. Acute cephalohematoma. Axial unenhanced and enhanced CT scans show a crescent-shaped soft-tissue mass in the periosteum of the left frontal bone.

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Fig. 0: Fig. Subgaleal hematoma in a newborn. Axial and Coronal T1-weighted and T2-weighted MR image shows a heterogeneous mass in the both parietal region beneath the aponeurosis.

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**Fig. 0:** Fig. Skull Tuberculosis. Skull radiography and axial unenhanced CT scan (bone window) show multiple, mottled osteolytic lesions in skull bone. The axial T2-weighted and enhanced T1-weighted images reveal well-enhancing soft tissue mass within the skull bone defect.

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Fig. 0: Fig. Scalp abscess after operation of brain abscess. The axial unenhanced and enhanced CT scans show focal mass with peripheral contrast enhancement in right parietal area.

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**Fig. 0:** Fig. Single skull metastasis from lung cancer (SCC). The axial T-weighted and T2-weighted MR images (a,b) show a nodule of low SI on T1WI and intermediate SI on T2WI. This nodule is well enhanced on axial sagittal CE-T1WI.

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Fig. 0: Fig. Morgagni Stewart Morel syndrome The axial CT scans (with bone window) (a,b) show diffuse skull thickening in the frontal bone and calcifications in both basal ganglia, presenting the manifestation of hyperparathyroidism. The axial T-weighted and coronal enhanced MR images reveal thickening of the inner table of the frontal bone.

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Conclusion

There are a wide variety of lesions that may occur in the head and neck, leading to the visible lumps and bumps. These lesions range from malformative encephaloceles, nasal gliomas, and dermoid cysts to acute, life-threatening lesions such as Pott puffy tumors and more aggressive neoplasms.

The use of proper imaging techniques and the knowledge of many features of the lesions are important and available to help characterize the many scalp and skull lesions.
References