

Imaging of the paediatric paranasal sinuses

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Sinus development is a dynamic process in children, and appropriate imaging is necessary to adequately demonstrate pathology in our vulnerable paediatric population. The paediatric paranasal sinuses are affected by a wide spectrum of conditions including congenital abnormalities and inflammatory, traumatic and neoplastic diseases.

The purpose of this review is to illustrate the normal pattern of development and the complex anatomy of the sinuses, which should guide imaging protocols. Common anatomical variations and their clinical relevance are described. The paper also describes the various conditions affecting the paediatric paranasal sinuses, with emphasis on imaging features. The diagnostic algorithm for sinus disease continues to evolve along with advances in imaging modalities, and this review discusses suggested imaging guidelines for sinus imaging in children.

S Afr J Rad 2013;17(3):91-97. DOI:10.7196/SAJR.778



Anatomy and development

The development of the paranasal sinuses begins as evaginations of the nasal mucous membranes during the second and fourth months of pregnancy. Further development takes place after birth. The process of development is completed after puberty. As sinus development progresses, asymmetry in size and shape is common, and opacification or mucous membrane thickening continues to be physiologic until approximately age 6 years.^[1]

The **maxillary sinus** is the first paranasal sinus to develop from the ethmoid sinuses. These sinuses show a biphasic growth pattern until the adult form at about the age of 12 years. They are rudimentary at birth and expand by pneumatization into the developing alveolar process. Their growth rate matches that of the maxilla and development of the dentition. Inferior growth usually reaches the hard palate by age 9, although the timing of these various stages of development is highly variable (Fig. 1a).^[2]

The maxillary sinus resembles a four-sided pyramid. The base lies vertically on the medial surface and forms the lateral nasal wall. The apex extends laterally into the zygomatic process of the maxilla. The roof of the sinus is also the floor of the orbit. The posterior wall extends the length of the maxilla.

The nasolacrimal duct drains the lacrimal sac and runs from the lacrimal fossa down the posterior aspect of the maxillary vertical buttress and empties into the inferior meatus. The duct lies very close to the maxillary ostium.^[3]

The **ethmoid air cells** are present at birth and continue to grow until late puberty or until they reach compact bone (Fig. 1b). The ethmoid sinuses are divided into groups of cells by bony basal lamellae of the middle turbinate, which separate the ethmoid into anterior and posterior groups with different drainage patterns.

Pneumatization progresses in a posterior direction. The final phase includes medial and inferior air cell extension. As a result, the ethmoid

air cells are rarely limited to the lateral ethmoid masses; they often extend into the turbinates, crista galli and the neighbouring frontal, maxillary, sphenoid and palate bones.^[4] This extension into the adjacent structures creates the commonly encountered normal variants. Inferomedial extension into the middle turbinate creates the concha bullosa variant, best seen on computed tomography (CT) scans (Fig. 2a).

Anteroinferior extension results in formation of the agger nasi cells. The agger nasi cells serve as the anterior floor of the frontal sinus (Fig. 2b).

Haller's cells are created when pneumatization progresses inferolaterally into an infraorbital location.^[2] Haller's cells can be clinically significant because of their location along the course of the infundibulum. At birth, the **sphenoid sinus** is undeveloped, with the sphenoid bone containing erythropoietic marrow. Usually at about age 3 years, aeration begins anteriorly and progresses in an inferior posterolateral direction. The sinus attains its mature size by the age of 14 years.^[1] The degree of pneumatization is highly variable. Aplasia is extremely rare. Pneumatization can extend into the greater wing of sphenoid, creating lateral recesses. Other variants include aeration of the medial pterygoid process (44%), anterior clinoid process (13%) and posterior orbital wall (Fig. 2c).

Sphenoethmoidal/Onodi cells represent contiguous extension of the posterior ethmoid air cells laterally and superiorly to the sphenoid sinus, and are closely associated with the optic nerve. In the presence of this anatomical variation, there is an increased risk of optic nerve damage during sphenoid surgery (Fig. 2d).

The last paranasal sinus to develop between the inner and outer table of the frontal bone is the **frontal sinus**. The frontal sinus is formed by pneumatization of the frontal recess into the frontal bone. The earliest pneumatization occurs at, or shortly after, 2 years of age. By 4, the cranial extent reaches half the height of the orbit.^[2] Usually at about 6 years of age, the cranial extent of the frontal sinus is at the superior orbital rim. Growth continues throughout childhood, and full size is reached after puberty.^[4]

The frontal sinuses are exceedingly variable in extent and form. Not infrequently, one sinus is entirely lacking. Aplasia or overly pneumatized frontal sinuses may occur.

The formation of additional cells in the frontal recess and the frontal infundibulum, apart from the agger nasi cells, is very individual. They are described according to their anatomic orientation. A cell that pneumatizes into the frontal bone is named a frontal cell of the anterior ethmoid or bulla frontalis, and lies above the ethmoid bulla. A supraorbital cell is a variant that develops as an extension, from the posterior aspect of the frontal or suprabullar cells.^[5] Other variants include a pneumatized crista galli or cells of the interfrontal septum.

Sinus drainage

Sinus drainage involves complex anatomical structures and pathways. The ostiomeatal complex (OMC) is a functional entity of the anterior ethmoid complex that represents the final common pathway for drainage and ventilation of the frontal, maxillary and anterior ethmoid cells.^[5] The maxillary sinus ostium drains into the infundibulum, which joins the hiatus semilunaris and drains into the middle meatus. The uncinate process is a thin bony leaflet that resembles a hook. It is orientated almost sagittally and runs from anterosuperior to posteroinferior and forms the medial border of the ethmoid infundibulum. The lateral border of the infundibulum is the lamina papyracea (Fig. 3a).^[5]

The anterior OMC comprises the frontal sinus ostium, frontal sinus drainage pathway, maxillary sinus ostium, infundibulum and middle meatus. These structures connect the frontal, anterior ethmoid and maxillary sinuses.

A second possible route of drainage from the maxillary sinus is via an accessory ostium along the medial maxillary wall, into the middle meatus. An accessory ostium exists in 15 - 30% of individuals (Fig. 3b). Accessory ostia are over-reported in the literature owing to incorrect window settings on CT.

The infundibulum frontale is a funnel-shaped narrowing at the inferior aspect of the frontal sinus, extending toward the floor of the frontal sinus ostium. Both frontal sinuses have their ostia at the most dependent portion of the cavity (posteromedial).^[6] In the sagittal section, the frontal recess, when taken together with the infundibulum frontale, resembles an hourglass, with the constricted portion being at the level of the natural ostium (Fig. 3c). This drainage system ends in the middle meatus. Anatomical obstruction to this pathway can result when the frontal recess is narrowed posteriorly by an ethmoid bulla. Anterior obstruction occurs when an enlarged agger nasi cell attenuates the infundibulum frontale.^[5]

The **anterior ethmoid** drains into the hiatus semilunaris and middle meatus. **Posterior ethmoid** drainage is into the sphenoethmoidal recess and superior meatus.

The **sphenoid sinus** drains via the ostium into the sphenoethmoidal recess, which opens into the nasal cavity inferiorly.^[3]

Any of the surrounding cells, recesses and ostia, with their dependent sinuses, may become diseased, thereby contributing to the symptoms and pathophysiology of sinusitis.

Anatomical variations can affect normal drainage, efficiency of mucociliary clearance and the morphology of the sinuses, predisposing the patient to sinus disease. This is particularly possible if occurring at the level of the frontal recess and the OMC.^[7] This relationship between anatomical variations and the development of chronic sinusitis is less significant in children than in adults.^[8]

At the level of the OMC, there can be variations to the intrinsic structures, such as an abnormal uncinate process orientation or a large ethmoid bulla.^[9] The ethmoid bulla is one of the largest anterior ethmoid cells and, if enlarged, can encroach on the OMC (Fig. 3a).^[10] Common variants of the uncinate process include pneumatization, or an uncinate bulla. The uncinate process can have variations in its insertion. The uncinate process may deviate medially and fuse with the middle turbinate, affecting the middle meatus, or deviate laterally and attach to the lamina papyracea; this will obstruct the hiatus semilunaris and/or infundibulum.

The uncinate process tip may fuse with the orbital floor or the inferior portion of the lamina papyracea, which is known as an atelectatic uncinate process. This variation is usually associated with a hypoplastic maxillary sinus.^[11] This variant is important to note for surgical planning because the ipsilateral orbit will be low-lying.^[12]

Extrinsic variations that could result in obstruction are a large concha bullosa, Haller's cells or a paradoxical middle turbinate.

Congenital abnormalities

Aplasia or hypoplasia are uncommon conditions, but are occasionally encountered. In the paediatric population, maxillary hypoplasia may be misdiagnosed as chronic sinusitis.^[13] The maxillary sinus is most frequently affected. There are other nonheritable congenital bony conditions that arise from the sinuses or affect them as a result of their anatomic location.

Fibrous dysplasia is a developmental disease of bone typically seen in childhood and adolescence. The maxilla is the most frequent facial bone affected.^[14] The medullary cavity is filled by abnormal fibrous tissue, resulting in progressive swelling and expansion of the affected bone (Fig. 4). The adjacent sinuses are thus secondarily involved. Sequelae include obstruction to the drainage pathway or sinuses of small volume. The sphenoid and frontal bones are also involved.

Fibrous dysplasia can be monostotic or polyostotic. Most cases of monostotic fibrous dysplasia are incidental findings on a cranial CT exam.^[12] Characteristic radiological features described include overgrowth of the bone with outward expansion of the outer table, and a 'ground-glass' texture to the involved bone. This is well demonstrated on CT.^[12] The craniofacial form of fibrous dysplasia has been named leontiasis ossea. An additional variant with autosomal dominant inheritance is familial fibrous dysplasia or cherubism.^[15]

The eventual size of the sinuses is influenced by brain growth. Owing to impaired brain growth in Sturge-Weber syndrome, there is enlargement of the ipsilateral sinuses.^[15] In other malformation syndromes (e.g. neurofibromatosis type 1 and Gardner syndrome), there may be a sinus abnormality secondary to local bony malformation.^[1] In beta thalassaemia, there may be osseous abnormalities caused by marrow hyperplasia. This can result in delayed pneumatization and expansion of the maxilla.^[12,16]

Cystic fibrosis (CF) is a heritable systemic condition where patients have chronic sinus disease owing to an abnormality of the mucus-producing exocrine glands. The paranasal sinuses are ultimately involved in almost all patients. The abnormal viscous secretions result in chronic pan-sinusitis, often with associated polyposis (Fig. 5).^[17] Nasal polyposis is otherwise rare in children without CF.

Kartagener's syndrome is another uncommon familial disorder in which patients have polyposis and chronic sinusitis secondary to abnormal mucociliary function.

Inflammatory conditions

The spectrum of inflammatory conditions affecting the paranasal sinuses, collectively represent the most frequently encountered paranasal sinus diseases in both the adult and paediatric patients. Sinusitis may be secondary to infection, allergy, altered immunity or a combination of these factors. Pathogens implicated include bacteria (more commonly) and a variety of fungi.

Sinusitis may be classified as acute, subacute, and chronic. The American Academy of Pediatrics (AAP) defines acute bacterial sinusitis as infection lasting less than 30 days. Subacute sinusitis is infection lasting between 30 and 90 days, and inflammation of the sinuses lasting more than 90 days as chronic.^[18]

The imaging findings are nonspecific, more so in children, and should be correlated clinically and with age. The radiographic hallmark of acute sinusitis is sinus opacification. However, an opacified sinus in childhood does not equate to sinusitis unless there is clinical corroborative evidence. Air-fluid levels in a sinus usually correlate with acute sinusitis (Fig. 6).

Chronic sinusitis usually results from repeated episodes of sinusitis. Mucosal thickening is not uncommonly interpreted as chronic sinusitis. The degree of mucosal thickening is important, as up to 2 - 3 mm may be seen normally as part of the nasal cycle. The degree of mucosal thickening can be described as mild (<5 mm), moderate (5 - 10 mm), or severe (>10 mm), and the location is important to mention.^[19] Mucosal thickening is, however, a non-specific finding. Other important CT findings in chronic sinusitis are a small-volume maxillary sinus, sclerosis, and thickening of the bone surrounding the affected sinus.^[14]

Allergic sinusitis is a local manifestation of an allergic reaction in the respiratory tract resulting in oedema and increased secretions. Infection may co-exist and radiological differentiation is usually impossible. In the majority of patients, the turbinates are thickened and nasal and/or sinus polyps can co-exist.^[1]

Fungal sinusitis occurs in a variety of forms depending on the patients' immune system, and can be divided into non-invasive and invasive subgroups. The non-invasive group can be further divided into allergic fungal sinusitis and fungal balls (mycetomas). The allergic form may mimic sinonasal polyposis radiographically and is more commonly seen in children. The pathogenesis is similar to allergic bronchopulmonary aspergillosis. *Aspergillus* is the most commonly encountered fungal pathogen. There is usually involvement of several sinuses, and the disease tends to be bilateral.^[20] Mycetomas are also usually encountered in immune-competent people.

A differentiating radiological feature of fungal sinusitis that is commonly demonstrated is foci of high attenuation in the centre of the affected sinus. Punctate calcifications can be seen on CT, particularly within mycetomas (Fig. 7a).^[20] On T2-weighted (T2w) MRI, there may be significant signal loss, particularly centrally within the involved sinus. This is thought to be secondary to the high fungal mycelial iron, magnesium and manganese content (Fig. 7b). On T1w sequences, the signal is usually elevated in allergic fungal sinusitis.^[15,20]

Invasive fungal sinusitis can be classified as acute, chronic and chronic granulomatous, and tends to be encountered more in the immune-compromised population, particularly the acute form, which is rapidly progressive and may demonstrate bone erosion, vascular invasion and intracranial extension. Whereas CT is better for assessing bony changes, MRI is superior in evaluating intraorbital and intracranial extension. In

the invasive form of fungal sinusitis, the T1w signal can be decreased, unlike in the allergic form.^[12]

Chronic invasive fungal sinusitis usually affects immune-competent individuals, but those with diabetes mellitus or a low level of immune compromise are also susceptible. On uncontrasted CT, mass-like hyperattenuated soft-tissue collections may mimic malignancy. There can also be invasion of adjacent structures. Differentiation between the chronic invasive form and malignancy may not be possible on imaging.^[20]

Mucormycosis is a rare fungal opportunistic infection that usually affects the sinuses and brain. The infection can be very aggressive, with high mortality rates, and commonly affects individuals with diabetes and those in immune-compromised states.

Dental-related pathology

Owing to the close relationship with the dentition, dental disease can extend into the maxillary sinus or cause maxillary infection. Cysts of dental origin arising from the maxilla may result in bone remodelling and extension into the maxillary sinus. **Apical or radicular cysts** are by far the most common; they are located at the apex of the root of a carious tooth. The lamina dura in this region is usually absorbed (Fig. 8). A **dentigerous cyst** is related to the crown of an unerupted tooth. This is the most common type of noninflammatory odontogenic cyst and is primarily found in adolescents and young adults.^[16] It is important to determine if the sinus pathology is odontogenic in origin, as this may influence the surgical management.

Complications of sinusitis

There are local changes that occur secondary to chronic inflammation, and complications that arise from contiguous extension of infection/inflammation. These can affect the orbits and bones, as well as cause potentially life-threatening intracranial complications. These are best assessed with cross-sectional imaging. Other complications, including pulmonary abscess and sepsis, may also occur infrequently:

1. **Mucous retention cysts** are asymptomatic and result from obstruction of the seromucinous glands. They can form after a sinus infection and are commonly found at the inferior aspect of the maxillary antrum. Radiologically, they are seen as dome-shaped structures, and cannot be differentiated from a solitary polyp on imaging (Fig. 9a).^[14,19]
2. **Polyps.** Polyposis is an inflammatory condition of the mucosa of the nose and paranasal sinuses that assumes a characteristic polypoid appearance. Sinonasal polyposis is encountered in all sinuses, but commonly in the maxillary antrum. Polyps form secondary to folding and hypertrophy of the mucosa, with accumulation of fluid in the submucosal space. They frequently have an allergic association, but are also seen following inflammation, infection and vasomotor rhinitis. Polyposis is more common in adults. In children, predisposing conditions such as cystic fibrosis and Kartagener's syndrome may need to be considered. Imaging features include polypoid nasal and sinus masses, and expansion of the nasal fossa, sinus and ostium. Partial or complete pansinus opacification can also be seen frequently. Occasionally, polyps may cause bone thinning that mimics carcinoma (Fig. 5). The polyps may show high density centrally and have a peripheral rim of low attenuation on CT. A CT finding that allows differentiation from tumours is the presence of a thin low-attenuation zone of mucoid material between the polypoid sinus masses.^[12] MR imaging demonstrates these polyps (polyps can have mixed signal

intensities, depending on their composition), mucosal hypertrophy and/or sinonasal secretions.^[17,19] An **antrochoanal polyp** refers to a solitary lesion that arises in the maxillary antrum of a non-atopic person, increases in size to widen the sinus ostium, and then extends into the nasal cavity. It may eventually extend into the nasopharynx (Fig. 9b).^[1]

3. **Mucocoeles** are expansile mucosa-lined sinus cysts that form secondary to an obstructed paranasal sinus ostium. They are most commonly post inflammatory, but any other cause of ostial obstruction could result in their formation. The frontal sinuses are most frequently involved (60%). On imaging, an expanded sinus cavity is seen. CT reveals a low attenuation mass. There may be bone thinning and remodelling secondary to pressure, but no destruction (Fig. 9c). In severe cases, there may be encroachment on adjacent structures, as seen with expansion into the subarachnoid space, resulting in a CSF leak. The signal intensity on MRI depends on the state of hydration, protein content and viscosity of the mucocoeles. They characteristically demonstrate a regular, peripheral rim of enhancement.^[15] This feature, together with the lack of bony destruction, is helpful in differentiating mucocoeles from neoplasms. If infection is superimposed, a pyocoele results.

4. **Orbital complications.** The most common complication of acute sinusitis in young children is extension of the infection into the periorbital region. The majority of cases are unilateral, but bilateral orbital cellulitis may occur. When confined to the eyelids, the condition is termed preseptal cellulitis. Imaging features include eyelid swelling, thickening of the preseptal tissues, and posterolateral extension of the inflammation into the temporal fossa.^[21] The process may extend postseptally, resulting in intra-orbital cellulitis, orbital abscess, subperiosteal abscess and cavernous sinus thrombosis.^[1] Postseptal complications of sinusitis mandate a CT scan to differentiate abscess from orbital cellulitis. Postseptal disease is treated with intravenous antibiotics, with surgery necessary in selected cases.^[22] The ethmoid sinuses are the most frequent source of infection. The infection can spread directly through the thin lamina papyracea into the orbits. Infection may also extend through the neurovascular foramina or ethmoidal veins.^[19]

Retrobulbar infection is most commonly confined to the extraconal space, with the subperiosteal space on the medial wall the most frequent location. This may progress to a frank subperiosteal abscess with pus that will require surgical drainage. This is the most common intraorbital complication in the paediatric population.^[23] CT and MRI demonstrate this subperiosteal fluid collection that may be rim-enhancing, with displacement of the adjacent fat and extraocular muscles (Fig. 10a).

Orbital cellulitis or abscess may lead to thrombosis of the superior ophthalmic vein, which may lead to cavernous sinus thrombosis. The clinical signs of ophthalmoplegia and proptosis may be present in orbital infection without cavernous sinus thrombosis. The presence of cranial nerve palsy in the setting of an orbital or sinus infection should raise the suspicion of cavernous sinus thrombosis.^[23] Contrast-enhanced CT imaging will show a non-enhancing 'filling' defect within the thrombosed cavernous sinus. The enhancing cavernous carotid artery will stand out in this enlarged cavernous sinus. MRI will show loss of the normal low-signal intensity flow void owing to the thrombosis (Fig. 10b). An engorged superior ophthalmic vein is also seen on both CT and MRI.

5. **Intracranial complications** of sinusitis are the most feared, and include meningitis, epidural empyema, subdural empyema and cerebral abscess formation. Brain infarction and mycotic aneurysm formation are

rare complications of sinusitis and can have devastating consequences. These occur most often after frontal sinusitis, with sphenoid sinusitis being next in frequency. Sphenoid sinusitis has a relatively high rate of associated neural and ophthalmic complications.^[22] MRI is superior to CT in the detection of intracranial complications of sinusitis.^[19,23]

The first site of infection following direct extension is the epidural space. Once there is penetration of the dura, a subdural collection can form. On imaging, it may be difficult to differentiate a subdural from an epidural empyema. An epidural empyema should not cross suture lines. Multiplanar contrast enhanced imaging is essential in imaging intracranial complications of sinusitis. These infected extra-axial fluid collections usually have enhancing rims and may show mass effect. Meningitis is often seen in conjunction with empyema, and is more frequently associated with sphenoid or ethmoid sinusitis. Meningeal enhancement is seen with contrast enhanced imaging (Fig. 10b).

Intracerebral abscesses are an uncommon complication of sinusitis. When they do occur, they usually involve the frontal and frontoparietal lobes. CT and MRI demonstrate surrounding vasogenic oedema, mass effect and ring enhancement of the abscess. Spectroscopy may be helpful in differentiating abscess from cystic tumour. The morbidity and mortality for a brain abscess remain high, despite treatment advances.^[23,24]

6. **Osteomyelitis** can affect the bony wall of any sinus, but most often involves the frontal sinus. Pott's puffy tumour is an entity that occurs predominantly in the paediatric and adolescent population. This is a subperiosteal abscess of the frontal bone where the precipitating infection originates in the frontal sinus and causes a progressive osteomyelitis of the bone. In the absence of osteomyelitis, it is thought that the frontal sinus infection spreads by transvenous spread through the frontal bone. If this is suspected, contrast-enhanced CT or MRI should be performed to look for frontal sinusitis, signs of osteomyelitis and any intracranial complications (Fig. 11).^[1,24]

Neoplastic lesions

Paranasal sinus neoplasms are uncommonly encountered in the paediatric patient. A variety of benign and malignant tumours affect the sinuses (Table 1). Neoplasms may clinically present as chronic or recurrent sinusitis. Unilateral involvement of a sinus with extensive opacification or extension into the OMC, with or without bony changes, should always raise the question of a tumour. A large number in fact do not arise from the sinuses but from adjacent structures, and secondarily affect the sinuses. The diagnosis of a soft-tissue tumour within the sinus cavities can be challenging on imaging. On CT imaging, bone involvement and changes are important clues. The presence of bone destruction would indicate an aggressive malignancy with rhabdomyosarcoma the most common in the paediatric patient.

Table 1. Benign and malignant tumours that affect the sinuses

Benign	Malignant
Osteoma	Rhabdomyosarcoma
Juvenile angiofibroma	Lymphoma
Haemangioma	Olfactory neuroblastoma
Ossifying fibroma	Germ cell tumour
Inverted papilloma	Leukaemia
Skull base meningioma	Nasal osteosarcoma

Benign tumours and inflammatory processes tend to remodel bone and cause thinning.^[12] Fungal infections and Wegener's granulomatosis can, however, also present with an aggressive appearance. T2w MR imaging can also occasionally provide a clue. Benign tumours as well as inflammatory diseases can have increased T2w signal owing to the high water content. Sinonasal tumours are highly cellular and most have an intermediate T2w signal.^[12] The enhancement characteristics of tumours also help to differentiate tumours from other entities. The enhancement patterns of some tumours are mentioned below.

Benign tumours

1. **Osteoma** is the most common benign tumour of the paranasal sinuses. It is a well-defined, bone-forming tumour arising from the wall of the sinus that extends into the sinus lumen.^[25] It may become large, deforming and obstruct the sinus cavity. This tumour is usually an incidental finding and most commonly arises from the frontal sinus.^[1] Multiple osteomas are associated with Gardner's syndrome. CT and plain radiography show these tumours as well delineated sclerotic lesions with smooth borders (Fig. 12).

2. **Juvenile angiofibroma**. Typically, this benign, vascular, locally invasive neoplasm occurs in pubescent males, presenting with nasal obstruction and epistaxis. Juvenile angiofibromas are the most common benign nasopharyngeal tumour, and can grow to enormous size.^[15] This neoplasm predominantly comprises angiomatous tissue, which accounts for the increased vascularity and intense enhancement seen on contrast enhanced CT and MRI. Biopsy of these masses is contraindicated. The mass is usually centred in the posterior nasal cavity, originating at the sphenopalatine foramen, extending into the pterygopalatine fossa (in 90% of cases). The mass can extend into the sinuses, orbits and middle cranial fossa.^[15,25] Intracranial extension occurs uncommonly in about 5 - 20% of cases. Superior orbital fissure widening is seen as an indication of intracranial extension.^[21] Embolisation of the tumour prior to surgery decreases intraoperative blood loss.

3. **Haemangioma**. These benign lesions uncommonly arise from the sinonasal cavity. They are divided into two types: capillary (seen in infancy) and cavernous (childhood or adolescence). Most nasal haemangiomas arise from the nasal septum or vestibule and are of the capillary type.^[26] These tumours are usually small and non-aggressive. They are best imaged with contrast-enhanced multiplanar imaging and appear as a well-defined, lobular, diffusely enhancing mass. There may be adjacent bone changes in chronic tumours or due to increasing size of the mass.^[25,26]

4. **Ossifying fibroma** is an uncommon, benign fibro-osseous tumour that can occasionally arise from a sinus. The mass is well demarcated and expansile, generally asymptomatic, but may be found if there is obstruction to a sinus drainage pathway or owing to facial deformity.^[25] CT demonstrates a mass of central low attenuation and a peripheral ossified rim. On imaging, it may be impossible to differentiate from fibrous dysplasia. It is usually unilateral and monostotic.^[15]

5. **Inverted papilloma**. This benign nasal tumour is usually seen in older males, but can occur in children and adolescents. This epithelial tumour is almost always unilateral and characteristically arises from the lateral nasal wall. It can extend into the ethmoid and maxillary sinuses.^[19] The homogenous enhancement seen with CT and MRI can help to differentiate it from an antrochoanal polyp, with which it can be confused. Although histologically benign, it can be locally aggressive,

and a small percentage can either degenerate into, or co-exist with, squamous cell carcinoma.^[15,25]

Malignant tumours

1. **Rhabdomyosarcoma (RMS)**. Also called parameningeal RMS, this is the most common soft tissue tumour in children. Up to 40% arise in the head and neck region. Sites include the orbit, nasopharynx, paranasal sinuses and the middle ear. RMS occurs primarily in patients from age 2 - 5 years of age. Embryonal RMS is the most common histological subtype, particularly in the head and neck region, and is considered the most treatable form of the disease.^[15,27] The risk of childhood RMS increases with certain inherited diseases, e.g. Li-Fraumeni syndrome, neurofibromatosis type 1 and Beckwith-Wiedemann syndrome.^[4,15] RMS is an aggressive malignancy, with intracranial extension not uncommon and distant metastases also seen. Multiplanar imaging with contrast is essential. MRI is the modality of choice, particularly for demonstrating the presence of intracranial and perineural spread. CT displays the extent of bony destruction (Fig. 13). Cross-sectional imaging and serial scans should be done to objectively monitor tumour regression or residual and recurrent disease.

2. **Lymphoma** is a common neoplasm of childhood, accounting for approximately 50% of all head and neck malignancies.^[27] Lymphomas arising primarily in the nose and paranasal sinuses are of the non-Hodgkin's type, but are uncommon. Non-Hodgkin's lymphoma presenting with extranodal disease in the head and neck region most frequently involves Waldeyer's ring. On CT and MRI, lymphoma of the sinonasal cavity may mimic the more common entities of chronic sinusitis, polyposis, granulomatous diseases and other neoplasms. MRI with contrast is the best imaging tool. The imaging features are non-specific, demonstrating a bulky, locally destructive soft tissue mass. Enlarged regional lymph nodes may assist in the diagnosis.^[19]

3. **Olfactory neuroblastoma**. Also known as esthesioneuroblastoma, this neuroendocrine malignancy arises from the olfactory epithelium of the superior nasal cavity.^[16] It may present in the adolescent patient with nasal blockage and mild epistaxis. The tumour demonstrates avid enhancement on both CT and MRI.^[19] Occasionally, the classical imaging appearance of a dumb-bell shaped mass with an intracranial portion and a portion within the nasal cavity is seen. Long-term follow-up is recommended as they have a tendency to recur late.^[25]

Trauma

The patterns of facial injury in children differ from adults because of anatomical and physiological differences, as well the extent of pneumatisation of the sinuses. The diagnosis is more difficult in children, and paediatric plain X-ray reporting is more challenging than in adults. CT is necessary to confirm the diagnosis, and for detailed evaluation. The overall frequency of paranasal sinus fractures is much lower than in adults. The radiologist should always bear in mind the possibility of child abuse. Nasal fractures are the most common, followed by mandibular fractures.^[28] The maxillary sinus is the sinus most frequently injured in direct trauma.

The 'blow-out' fracture of the orbital floor is the most common fracture involving the paranasal sinuses in childhood. The frequency of such fractures increases with increasing pneumatisation of the maxillary sinus. There may be orbital injury, enophthalmos as well as opacification of the maxillary sinus. Peri-orbital fat and/or the inferior rectus muscle can become entrapped on the maxillary sinus side of the orbital floor (Fig. 14).

Medial wall fractures also result from direct trauma. Fracture of the lamina papyracea is usually best demonstrated on CT. This fracture is usually clinically insignificant and heals spontaneously.^[1,28]

Complex mid-facial fractures result from severe trauma and are the least common facial fractures in children. The Le Fort classification is generally used to describe these complex fractures. There are three types, all having certain common features: (i) all bilateral; (ii) in all, some part of the face is mobile; and (iii) all involve the pterygoid processes.^[1]

Miscellaneous

1. Foreign bodies. A variety of foreign bodies (organic and inorganic) have been reported in the sinus cavities. They are more frequently seen in the paediatric patient, and more commonly within the nasal cavity. If the foreign body is present for a long time, it may act as a nidus for infection and may also calcify.^[12] Complications related to foreign bodies include sinusitis, nasal perforation, cellulitis and meningitis. CT may localise a radio-opaque foreign body and identify related complications.

2. Wegener's granulomatosis is typically a disease of adults but rarely can affect the paediatric population. Wegener's is an aseptic, necrotising vasculitis that usually involves the kidneys and respiratory tracts. Within the upper respiratory tract, most start in the nasal cavity as soft tissue masses with, or without, septal and bone destruction. There may be an associated, chronic, non-specific pan-sinusitis.^[16,24] CT is recommended to assess extent of disease and degree of bone destruction.

3. Langerhans cell histiocytosis is an idiopathic group of disorders, characterised by abnormal clonal proliferation of the Langerhans cell, that can manifest as local or systemic disease.^[15] The mildest expression of histiocytosis is eosinophilic granuloma with predominantly bony lesions. The skull is involved in a quarter of cases. In approximately two-thirds of patients, the lesions are solitary. The radiographic differential diagnosis in these cases may be extremely difficult, and ranges from osteomyelitis to fibrous dysplasia.^[25] Isolated bony involvement is associated with the best prognosis. Rarely, the bones of the sinus cavities are involved and there may be a soft tissue mass overlying the lytic bony lesion (Fig. 15).

Imaging recommendations

When considering imaging in a child with sinus disease, important factors that need to be considered are age, radiation exposure and the possibility of an underlying clinical condition. Most of the sinuses are rudimentary with very small volumes until age 5 or 6 years. This normal process of pneumatization has to be considered when planning imaging of the sinuses. In developing countries, availability and cost-effectiveness of imaging protocols are other important factors. In view of this, the AAP and American College of Radiology recommend that the diagnosis of a sinus infection in a child be made clinically. It would not be necessary to get a sinus X-ray, especially in children <6 years old.^[29] Although controversial, imaging studies may be necessary to confirm a diagnosis of acute bacterial sinusitis in children >6 years old.^[18] A suggested guide to imaging the sinuses is given in Fig. 16.

Plain X-ray interpretation of the findings in the paranasal sinuses is less accurate in infants and children than in adults. In the former, the cavities are smaller and the margins of normal sinuses are often indistinct. The sinus cavities are commonly cloudy and opaque when the infant is in good health. This physiologic cloudiness is caused by the

relatively redundant normal mucous membranes of early infancy.^[4] They can also become opacified during and after crying. Sinus opacification does not equate to sinus disease in young children (Fig. 17).

The diagnostic algorithm for sinus disease continues to evolve along with advances in imaging modalities. Plain radiographs were once the mainstay in the diagnosis of sinus disease; however, CT has become a major diagnostic tool in both children and adults for the adequate evaluation and diagnosis of sinus disease. Standard radiography lacks sensitivity and specificity, and radiography-based evaluations alone can either over- or underestimate soft tissue changes in the paranasal cavities.^[30] Plain radiographs are not adequate in evaluating the osteomeatal complex or the sphenoid and ethmoid sinuses because of overlapping anatomic structures. Thickening of the extra sinus soft tissues superimposed on the paranasal air spaces also causes haziness. An example is facial cellulitis, which may be associated with maxillary and/or ethmoid sinusitis. Asymmetric bone wall thickening of one of the paired sinuses may also cause it to appear more opaque.^[4] Lateral sinus radiographs in children <3 years old have been found to be of little value. Submentovertex projections also contribute little diagnostic information in patients with sinus disease.^[31]

CT is an important diagnostic tool in children when used in the appropriate clinical setting. The issue of increased radiation dose always needs to be remembered with CT. Children are more sensitive to radiation than are adults by a factor of 10, and girls are more sensitive than boys.^[18,30] However, when CT is properly performed for appropriate indications, the benefits far exceed this very small individual risk; this is the basis of the ALARA (as low as reasonably achievable) principle in paediatric CT. The use of radiography v. CT for imaging of sinusitis in children continues to be controversial, owing to the radiation dose and costs involved. Such precautions are needed because there seems to be an increased lifetime cancer mortality risk for children exposed to CT radiation.^[32]

The AAP recommends that CT scans of the paranasal sinuses be reserved for patients in whom surgery is being considered as a management strategy. This policy includes children who present with complications of acute bacterial sinus infection or those who have very persistent or recurrent infections that are not responsive to medical management.^[18]

The improved anatomical detail seen with CT is important for assessing the OMC in patients with recurrent sinusitis and for assessing complications of sinus inflammatory disease. The goal of CT in chronic sinusitis is to provide objective information to support diagnosis and detailed anatomy for surgery, and to predict which patients will benefit from surgery.^[33] CT scanning of the sinuses involves imaging of tissues with three markedly different densities: air, bone and soft tissue. This results in an inherent high image contrast and allows the possibility of using low-dose CT of adequate diagnostic quality in children. With the widespread introduction of multidetector CT technology, the use of low-dose CT has become the method of choice, delivering images of good quality.^[30] Improved multiplanar reconstructions are also obtained, affording optimal visualisation and assessment of the complex anatomy and pathological conditions affecting the sinuses.

CT is also used in assessing sinonasal tumours; however, in this regard, MRI is superior to CT for mapping the extent of the tumour. Its advantages include multiplanar capability, superior soft tissue contrast, absence of ionising radiation, and the potential to differentiate neoplasms from adjacent inflammation.^[1] A more recent development

in CT technology is a miniaturised CT scanner called a cone beam CT (CBCT), providing sufficient resolution to outline the facial bony architecture. The spectrum of its clinical usage remains to be determined. Some advantages include decreased cost and radiation exposure; however, an important disadvantage is the poor soft tissue contrast resolution. There are many other factors that may limit its widespread clinical use.^[34]

There have been studies evaluating the clinical use of ultrasound in the evaluation of maxillary sinusitis. Studies of ultrasound in paediatric maxillary sinusitis revealed conflicting results, with wide variations in sensitivity and specificity. Ultrasound may come to represent an accessible, non-invasive imaging alternative to assess the presence of fluid in the maxillary sinus in children.^[35]

Conclusion

The air-filled spaces of the facial and skull bones are dynamic and evolving structures in growing children. Normal variations are well demonstrated with imaging. The importance of these normal variants increases with increasing numbers of children undergoing imaging and endoscopic surgery. This review has covered a spectrum of conditions affecting the paranasal sinuses in the paediatric patient. Salient features regarding their imaging appearances have been mentioned. Cross-sectional imaging has become the mainstay in imaging of the sinuses, and provides exceptional detail of the anatomy and variations seen in the paranasal sinuses. It is also an essential tool for assessing disease extent, and assists in determining diagnosis and planning surgery. The diagnostic algorithm is developing and needs to be continuously and individually adapted in the paediatric population.

Acknowledgements. I thank Professors Martin Mack and Savvas Andronikou for their help and advice in preparing this review.

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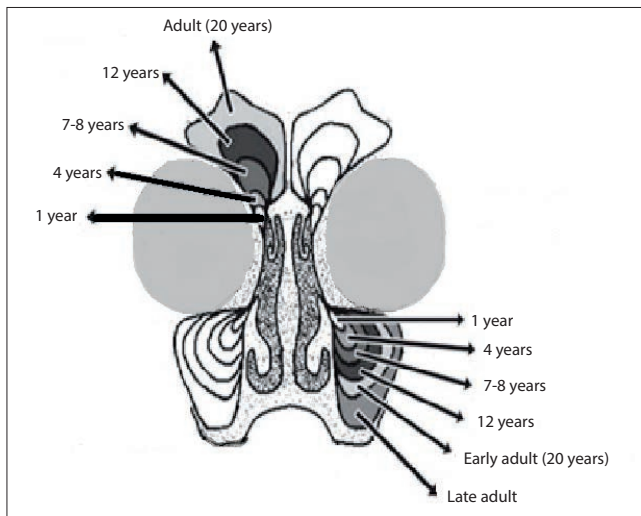


Fig. 1a. Coronal drawing showing changes in size and shape during normal pneumatization of the maxillary and frontal sinuses.

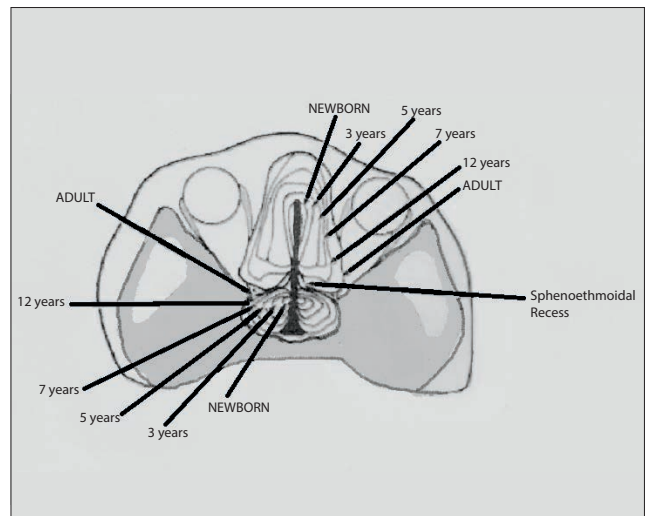


Fig. 1b. Composite axial drawing showing changes in size and shape of the ethmoid and sphenoid sinuses during normal development.

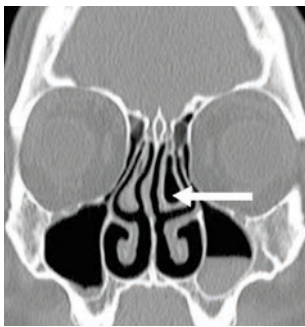


Fig. 2a. Coronal CT demonstrating concha bullosa variant of the left middle turbinate (arrow).



Fig. 2b. Occipitofrontal X-ray demonstrating bilateral agger nasi cells.

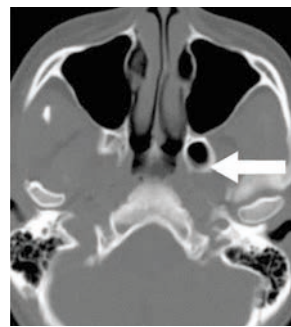


Fig. 2c. Axial CT. Sphenoid pneumatization extends into the left pterygoid process.

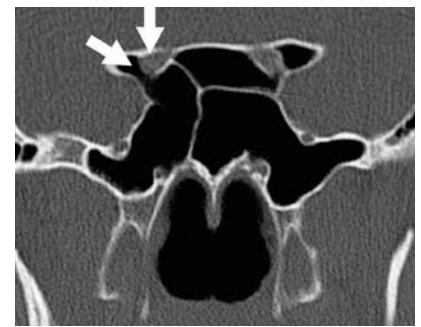


Fig. 2d. Coronal CT demonstrates posterior ethmoid pneumatization extending into the sphenoid sinus creating Onodi cell variant (thin solid arrow). There is aeration of the anterior clinoid processes, with the aerated cavities lying lateral to the optic canal (broad hollow arrow).

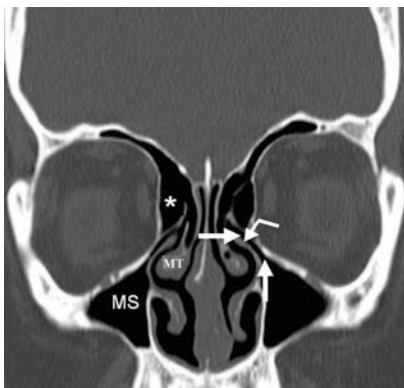


Fig. 3a. Coronal CT of normal ostiomeatal complex (OMC) showing maxillary sinus (MS), middle turbinate (MT), right ethmoid bulla (*), uncinate process (solid arrow), ethmoid infundibulum (curved arrow) and ostium (hollow arrow). The right ethmoid bulla encroaches on the infundibulum.

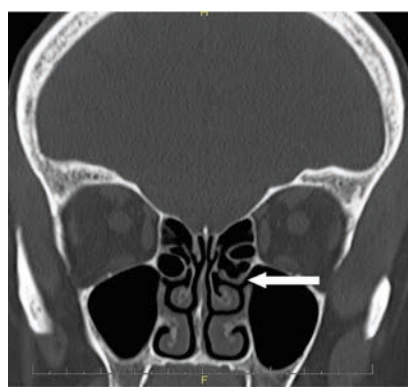


Fig. 3b. Coronal CT demonstrating the left accessory maxillary ostium (arrow).

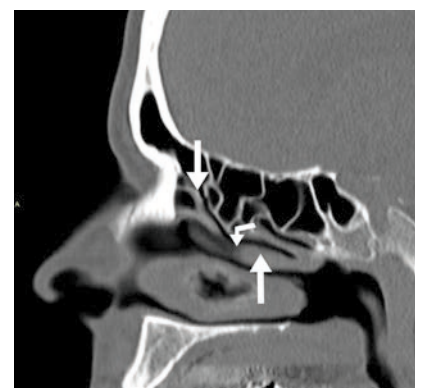


Fig. 3c. Sagittal CT depicting normal frontal sinus drainage pathway. The frontal sinuses drain via the frontal ostium (short solid arrow) into the frontal recess. The frontal recess communicates with the middle meatus via the hiatus semilunaris (curved arrow). Note the middle turbinate (hollow arrow).

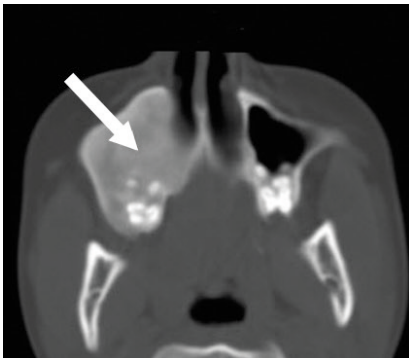


Fig. 4. Axial CT demonstrating fibrous dysplasia of the right maxilla (arrow), and bony expansion of the maxilla with obliteration of the sinus cavity.

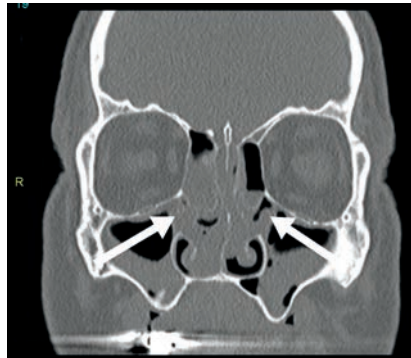


Fig. 5. Coronal CT demonstrating extensive sinonasal polyposis in a patient with cystic fibrosis. Lobulated, mucosal thickening involving the sinuses and nasal cavity with widening of the osteomeatal complexes (arrows) is present.

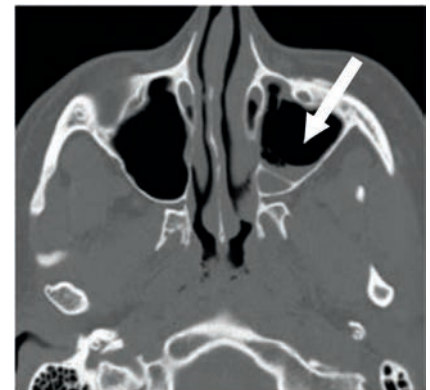


Fig. 6. Acute maxillary sinusitis. CT demonstrates air-fluid level in the left maxillary sinus (arrow).

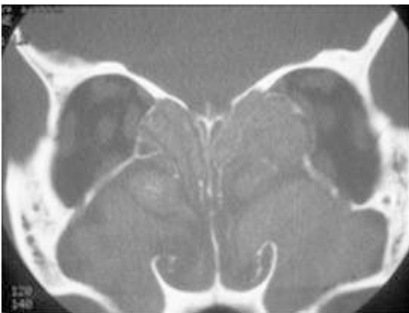


Fig. 7a. Chronic allergic non-invasive fungal sinusitis. Coronal uncontrasted CT depicts the high-attenuation, expansile opacification of the maxillary and ethmoid sinuses. The CT demonstrates the amorphous calcifications, sinus expansion and bony thinning.

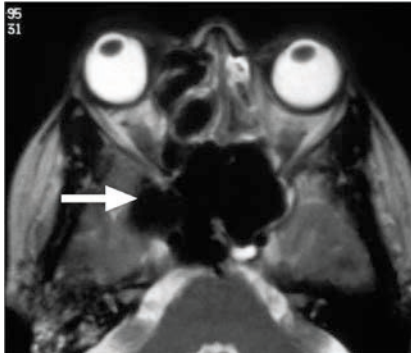


Fig. 7b. Axial T2w MR of fungal sinusitis. There is significant signal loss seen centrally within the opacified sphenoid sinus. The intracranial extension of the fungal sinusitis is demonstrated by the low signal extending into the right middle cranial fossa (arrow).

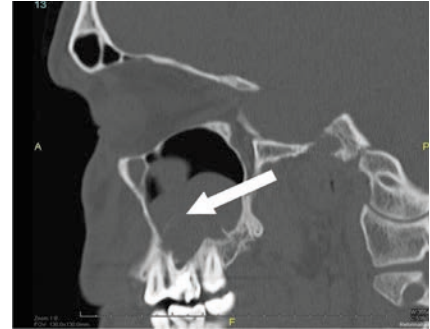


Fig. 8. Sagittal uncontrasted CT reveals radicular cyst with bony remodelling and extension into the maxillary sinus (arrow). The lamina dura of the affected tooth is destroyed. There is sinus opacification secondary to the underlying inflammatory process.

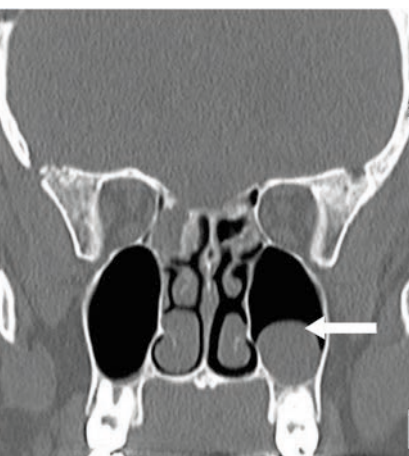


Fig. 9a. Coronal CT demonstrating a mucus retention cyst. A dome-shaped low-attenuation cyst (arrow) is present within the dependant portion of the left maxillary sinus.

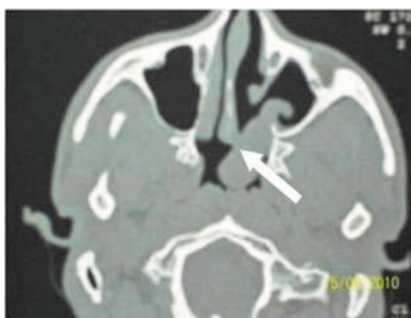


Fig. 9b. Antrochoanal polyp. Axial CT demonstrates a solitary polypoid lesion (arrow) arising from the maxillary antrum widening the sinus ostium and extending posteriorly into the nasopharynx.

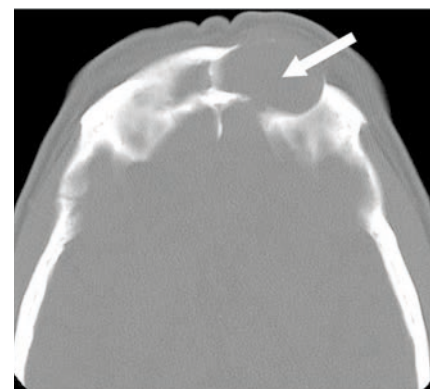


Fig. 9c. Left frontal mucocoele. Axial CT demonstrates a low-attenuation mass expanding the frontal sinus. Bone thinning and remodelling is seen secondary to pressure.



Fig. 10a. Axial enhanced CT of orbits. A subperiosteal abscess is present within right medial extraconal space (arrow). The rim-enhancing abscess displaces the medial rectus muscle.

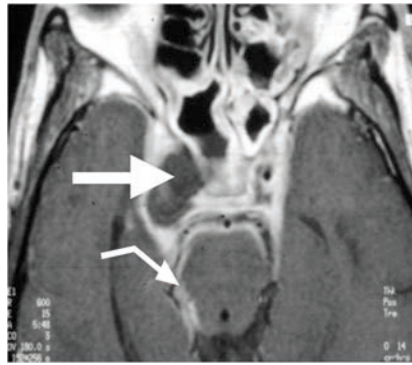


Fig. 10b. Contrast-enhanced axial T1w MRI. Non-enhancing filling defect is seen within expanded right cavernous sinus (arrow). There is loss of flow void within the right internal carotid artery, indicating thrombosis. There is prominent leptomeningeal enhancement surrounding the brainstem, indicating meningitis (curved arrow). An air-fluid level is seen within the right sphenoid sinus.

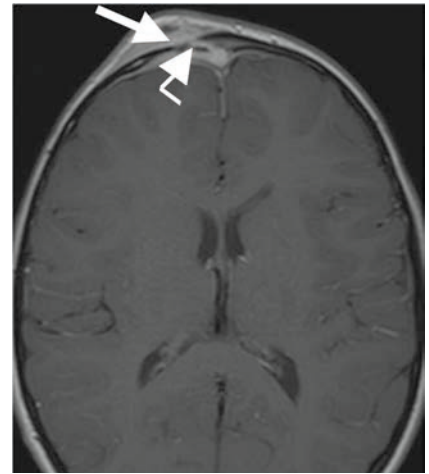


Fig. 11. Pott's puffy tumour. Contrast-enhanced axial T1w MRI demonstrates a rim-enhancing subcutaneous frontal scalp collection (arrow) communicating with osteomyelitis of the frontal bone secondary to frontal sinusitis. There is also a small right frontal empyema (curved arrow).

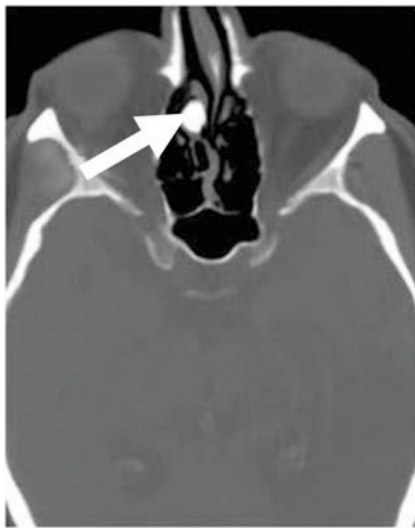


Fig. 12. Right ethmoid osteoma. Axial uncontrasted CT demonstrates well-delineated sclerotic lesion with smooth border (arrow).



Fig. 13. Embryonal rhabdomyosarcoma. Axial contrast-enhanced T1w MRI demonstrates enhancing, aggressive mass arising from sinuses. There is intracranial extension, into the middle cranial fossa (curved arrow). There is also invasion of the right cavernous sinus and right orbit, closely related to the right optic nerve (arrow).

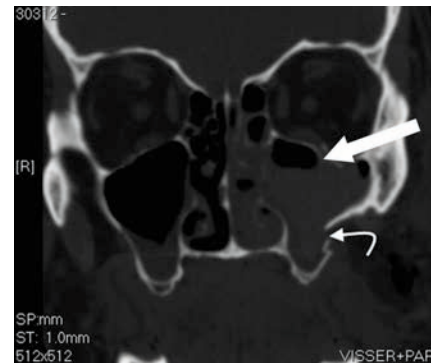


Fig. 14. Blowout fracture. Coronal CT shows fracture of the floor of the left orbit (arrow). There is opacification of the right maxillary sinus and also a fracture of the lateral wall of the sinus (curved arrow).



Fig. 15. Coronal contrast-enhanced T1w MR. Eosinophilic granuloma of the right greater wing of sphenoid (arrow). The adjacent enhancing soft tissue mass encroaches on the maxillary sinus and orbit.



Fig. 17. Axial T2w MRI demonstrating incidental finding of opacified ethmoid air cells in an asymptomatic 4-year-old.

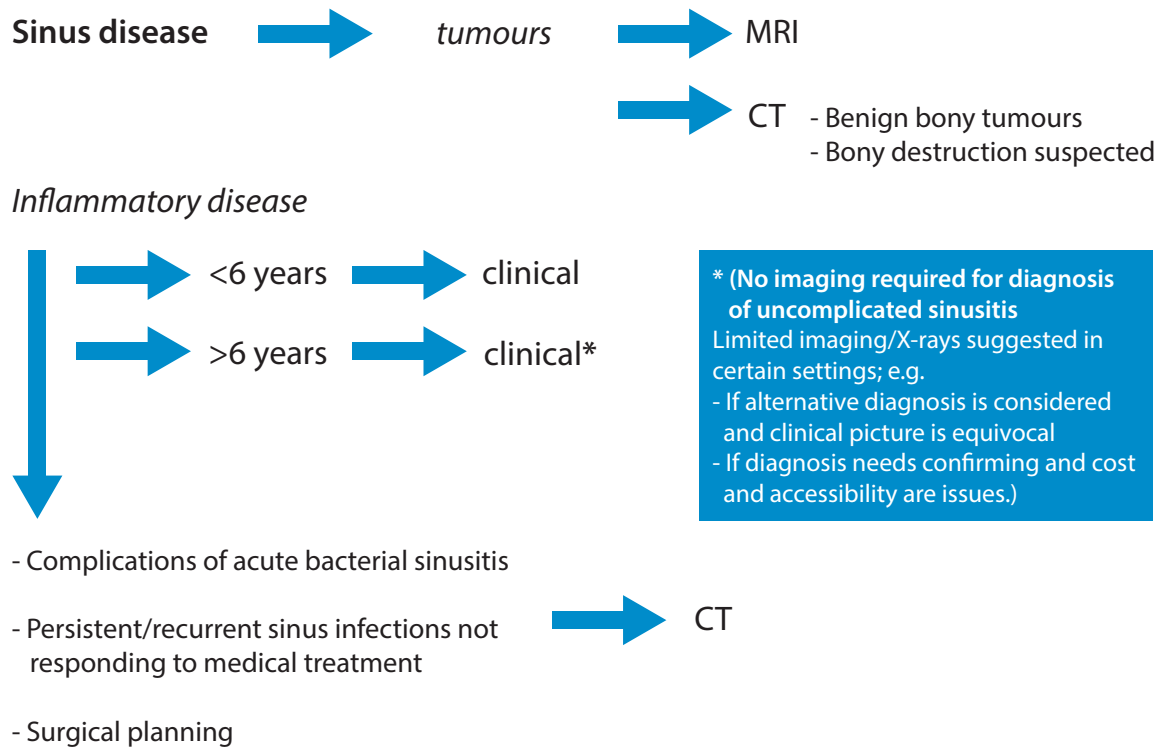


Fig. 16. Proposed algorithm for imaging children with sinus disease.