Chapter 6
Acute Frontal Sinusitis

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Contents

Core Messages

- Uncomplicated acute frontal sinusitis (AFS) is most often associated with an antecedent viral upper respiratory tract infection. Bacterial infection is suspected if symptoms are persistent for at least 10 days.
- The diagnosis of AFS is considered in patients who meet the diagnostic criteria for acute sinusitis and have symptoms referable to the forehead region.
- The predominant organisms cultured from patients with uncomplicated AFS are *Hemophilus influenzae*, *Streptococcus pneumoniae* and *Moraxella catarrhalis*.

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- When oral antibiotics are indicated, uncomplicated AFS should be treated for 10–14 days with amoxicillin-clavulanate (in patients without penicillin allergy).
- Although uncomplicated AFS is a self-limited disease, complicated acute frontal sinusitis can progress rapidly with catastrophic sequelae.
- Complicated AFS is suspected when symptoms are protracted and severe or when neurological deficits, frontal headache and fever are present.
- Work up of complicated AFS should include CT scan with IV contrast and MRI for inconclusive cases.
- Epidural and subdural abscesses are the most common intracranial complications of AFS.
- Patients with complicated AFS should be admitted for intravenous antibiotic therapy and intravenous hydration. Endoscopic sinus surgery or frontal trephination may be necessary to drain the frontal sinus. Craniotomy may be indicated for management of intracranial abscess.

Introduction

The reported prevalence rates of acute rhinosinusitis (ARS) observed in primary care practice varies between 6 and 12 % [1]. Between 2000 and 2009 there was an average of 4.3 million outpatient visits annually for ARS. Antibiotics were prescribed in 83 % of these visits [2]. The National Ambulatory Medical Care Survey indicates that sinusitis (acute and chronic) is the fifth most common disease for which antibiotics are prescribed [3].

The primary predisposing factor for ARS is an antecedent upper respiratory viral infection. Approximately 0.5–2 % of viral upper respiratory tract infections are complicated by acute bacterial infection. The incidence of ARS is higher in winter months, in damp climates, and in cities with significant air pollution.

Acute frontal sinusitis (AFS), a subset of ARS, occurs most commonly in adolescent males and young men. While the reasons for the male predilection are unknown, the age predilection appears likely due to the peak vascularity and peak development of the frontal sinuses between the ages of 7 and 20. Although acute frontal sinusitis is largely a self-limited disease, complications of acute frontal sinusitis can have catastrophic clinical consequences if not detected promptly.

Etiology and Pathophysiology of Acute Frontal Sinusitis

- Acute frontal sinusitis is most commonly preceded by a viral upper respiratory tract infection.
- Human rhinovirus is implicated in 50 % of cases, but other viruses may include coronavirus, influenza, parainfluenza, respiratory syncytial virus, adenovirus, and enterovirus.

The peak prevalence of these viruses occurs in early fall and spring, which parallels the peak incidence of acute bacterial rhinosinusitis (ABRS). Viral infection leads to an inflammatory cascade in which T-helper type 1 cytokine polarization is associated with a high level of tumor necrosis factor-β and interferon-γ. There is also an associated release of proinflammatory cytokines such as interleukin (IL)-1β, IL-6, and IL-8. These cytokines are considered very potent chemoattractants for neutrophils [4]. The viral induction of the inflammatory cascade results in acute mucosal edema, occlusion of sinus ostia, and impairment of mucociliary clearance. The resulting mucus stasis can contribute to a milieu that favors the proliferation of pathogenic micro-organisms, resulting in acute bacterial sinusitis.

Risk factors for acute sinusitis may include a variety of host factors, including anatomic, inflammatory, immunologic, and environmental. Structural concerns, such as concha bullosa or septal deviation, may be clinically significant. Inflammatory conditions such as nasal polyposis may predispose to acute sinusitis by gross obstruction of sinus drainage by polyps, as well as by generalized mucosal edema. Environmental exposures should be considered, although the evidence for their associations can be variable. For example smoking is thought to be a risk factor for ARS by disrupting ciliary function [1], but the evidence for passive smoke exposure as a significant risk factor is less compelling [5]. Host immune factors such immunodeficiency or immunosuppression can be important risk factors, whereas the role of allergy in ARS is the subject of considerable debate, with studies both supporting and challenging its role [6, 7].

While acute sinusitis typically affects the ethmoid and maxillary sinuses, progression of disease to involve the frontal sinus may be influenced by anatomic variations of the superior aspect of the ethmoid sinus that may affect frontal sinus drainage. Because the frontal sinus is embryologically derived from pneumatization of the ethmoid, frontal sinus outflow is thus influenced and defined by the degree of pneumatization of the ethmoid labyrinth. A variety of ethmoid-derived structures that comprise the frontal recess can thus narrow the outflow tract and predispose to acute frontal sinusitis. These structures may include agger nasi cells anteriorly; the bulla lamella and suprabullar/frontal bullar cells posteriorly; supraorbital ethmoid cells laterally; and type I–IV frontal cells comprising variable spatial orientations within the frontal recess [8]. A recent study found that the presence of frontoethmoid cells in the posterior and posterolateral aspects of the frontal recess (suprabullar cells, frontal bullar cells, and supraorbital ethmoid cells) may have a more significant association with the development of frontal sinusitis than those cells in the anterior aspect of the frontal recess [9].

Uncomplicated Acute Frontal Sinusitis

Diagnosis

Historically recommended diagnostic algorithms based on combinations of major and minor symptoms have been abandoned in favor of more recent literature which focuses on three cardinal symptoms: purulent nasal discharge, nasal obstruction, and facial pain/

pressure/fullness [10]. According to the most recent guidelines from the American Academy of Otolaryngology (AAO) [10], ABRS is defined by cardinal symptoms of purulent nasal discharge, nasal obstruction and facial pain/pressure/fullness that are present 10 days or more beyond the onset of upper respiratory symptoms, or that worsen after initial improvement within the first 10 days (double worsening). The 10 day time point is selected in part because of the difficulty in discerning viral versus bacterial etiologies in the first 7–10 days of an acute upper respiratory tract infection [11].

The Infectious Diseases Society of America (IDSA) guidelines [12] define ABRS as either persistent symptoms or signs compatible with acute rhinosinusitis, lasting for 10 days without any evidence of clinical improvement; or onset with severe symptoms or signs of high fever 39 °C (102 °F) and purulent nasal discharge or facial pain lasting for at least three to four consecutive days at the beginning of illness; or onset with worsening symptoms or signs characterized by the new onset of fever, headache, or increase in nasal discharge following a typical viral upper respiratory infection (URI) that lasted 5–6 days and were initially improving ("double sickening").

The European Position Paper on Rhinosinusitis (EPOS) guidelines from 2012 define ARS in adults as sudden onset of two or more symptoms, one of which should be either nasal blockage/obstruction/congestion or nasal discharge (anterior/posterior nasal drip) and the other being facial pain/pressure or reduction or loss of smell [1]. ABRS is suggested by the presence of at least three of any of the following symptoms and signs- discolored nasal discharge, severe local pain, fever >38 °C, elevated ESR/CRP or double sickening. Endoscopic evidence of middle meatal purulence supports the diagnosis.

Both the AAO and EPOS recommend against plain x-rays for patients already meeting the clinical diagnostic criteria. CT scan or MRI of the sinuses is recommended only when a complication is suspected or when the patient is immunocompromised.

There are no site-specific criteria for the diagnosis of acute frontal sinusitis. Generally acute frontal sinus symptoms are referable to the brow, temple, and frontal bone region. Frontal headache is the most prevalent symptom of acute frontal sinusitis [13].

- Thus, a diagnosis of acute frontal sinusitis should be considered in patients who meet the diagnostic criteria for acute sinusitis, in whom symptoms localize to the forehead region.

In some cases, the acute onset of frontal headache, even in the absence of more classic symptoms such as nasal congestion and rhinorrhea, should prompt the physician to consider a diagnosis of acute frontal sinusitis. This is especially true in those patients without a prior history of chronic headache.

Bacteriology

The most common bacteria isolated from patients with ABRS are Streptococcus pneumoniae (20–43 %), Haemophilus influenzae (22–35 %) and Moraxella catarrhalis (2–10 %). Staphylococcus aureus, Streptococcus pyogenes, and anaerobic

bacteria may also be involved to a lesser extent, with anaerobic bacteria being classically associated with odontogenic infections. Pseudomonas aeruginosa and other gram-negative rods may be recovered in patients with nosocomial sinusitis (e.g., associated with nasal tubes or catheters), immunocompromised patients, and those with cystic fibrosis [14]. Although regional geographic variations exist, about 15–20 % of Strep. pneumoniae are resistant to penicillin, and about 80 % of M. catarrhalis and 30 % of H influenza are beta-lactamase producing [10].

In children, the pathogen profile of acute sinusitis in the US has undergone significant shifts since the introduction of the seven valent pneumococcal vaccine. The incidence of Strep pneumonia isolates has dropped from 44 to 27 %, along with reported increases in H influenzae from 37 to 44 %, Strep pyogenes from 7 to 12 %, and Staph aureus from 4 to 8 %, with no change in Moraxella catarrhalis.

Changing patterns of resistance rates deserve attention and should be taken in consideration in patients not responding to first line treatment. Endoscopic cultures of the middle meatus may be appropriate in these cases.

- Middle meatal cultures correlate well with maxillary sinus puncture cultures, with an of 87 % concordance rate [15].

Culture data specific to acute frontal sinusitis are scarce owing to the difficulty of obtaining frontal sinus cultures. Given that acute frontal sinusitis typically occurs in conjunction with acute maxillary and ethmoid sinusitis, it would be reasonable to expect that the same pathogens observed in acute maxillary and ethmoid sinusitis would also be found in acute frontal sinusitis. Although the literature is sparse, the few studies that have examined this indeed support this notion [16–18].

Treatment

In light of the fact that some cases of acute bacterial sinusitis may spontaneously resolve without antibiotic therapy, the AAO recognizes that observation is an option for selected patients with uncomplicated ABRS who have mild pain and temperature <38.3 °C. Patients who are observed without antibiotic therapy must be reliable and compliant with follow up examination.

- Antibiotics should be started if the patient's condition fails to improve within 7 days or worsens at any time.

Conversely, in patients with more severe symptoms or multiple comorbidities, or in those that cannot be followed up, antibiotics should be prescribed at the outset. Antibiotic therapy should be selected for coverage of the primary organisms associated with acute rhinosinusitis: *Strep pneumoniae*, *H. influenzae*, and *M. catarrhalis*. Resistance patterns as indicated above should be taken in consideration as well. Risk factors for antibiotic resistance include: age <2 or age >65, prior antibiotics received within the previous month, prior hospitalization in the past 5 days, multiple co-morbidities, or immunocompromised status.

IDSA 2012 guidelines for antibiotics in acute sinusitis:

- Amoxicillin-clavulanate as empirical first line therapy in adults and children with severe or worsening symptom of acute sinusitis.
- Macrolides are not recommended due to high rates of resistance among S. pneumonia (30 %).
- TMP/SMX is also not recommended due to high rates of resistance among both S pneumonia and H influenza (30–40 %).
- Second generation oral cephalosporins are not recommended for monotherapy due to variable rates of resistance among S pneumoniae [12].
- In adult patients allergic to penicillin, either doxycycline or a respiratory fluoroquinolone (levofloxacin or moxifloxacin) may be used.
- In children, combination therapy of oral third generation cephalosporin (cefixime or cefpodoxime) and clindamycin is recommended.
- Routine coverage of MRSA is not recommended.
- Recommended treatment duration in uncomplicated ABRS is 5–7 days in adults and 10–14 days in children.

In patients who fail to improve with antimicrobial treatment within 3–5 days or whose symptoms actually worsen after 48–72 h, antimicrobial coverage should be broadened. Endoscopic culture should be pursued to direct more specific antibiotic coverage. Depending on the severity of symptoms and level of clinical suspicion, radiologic imaging should also be considered to rule out suppurative complications.

Additional Therapies

There is level Ia evidence to support treatment of acute rhinosinusitis with intranasal corticosteroids as monotherapy in moderate disease, and as an adjunct to oral antibiotics in severe disease [19]. A recent Cochrane analysis suggests that oral corticosteroids are effective for short term relief of symptoms as an adjunct therapy to oral antibiotics in ARS [20]. A recent Cochrane review found that nasal irrigation with saline has limited benefit in shortening the duration of illness in adults with ARS, although it may be considered for symptomatic relief (level 1a) [21]. There is no evidence to support the use of antihistamines, either oral or intra-nasal, in the treatment ABRS, except in patients with co-existing allergic rhinitis. Also, there is no evidence that the use of nasal or oral decongestants alters the course of ARS, although they may be indicated for alleviating acute symptoms [1, 10].

Surgery

There is a limited role for surgery in uncomplicated acute frontal sinusitis. It should be considered only in those patients with severe symptoms not responding to aggressive oral or IV antibiotic therapy, or in whom there is concern for an imminent

complication. Endoscopic frontal sinusotomy can be considered, either by traditional frontal recess dissection, or balloon dilation [22]. Frontal recess dissection in the face of acute infection may be especially challenging with extensive mucosal edema, inflammation and bleeding, necessitating advanced skills and experience in these procedures. External drainage via frontal sinus trephination is an alternative option and may be more facile for the less experienced surgeon. Trephination, however, only evacuates the frontal sinus and does not directly address restoration or widening of the natural drainage path of the frontal sinus.

Complicated Acute Frontal Sinusitis

Extrasinus complications from acute bacterial rhinosinusitis are uncommon. The estimated incidence of complications, per one study from the Netherlands, is 1:12,000 for pediatric ABRS and 1:32,000 for adult ABRS [23]. Adolescent and young adult males are significantly more affected than females [24], with a seasonal pattern favoring the winter months [1, 25]. Whereas orbital complications are the most common complications from all forms of ABRS, the vast majority of intracranial complications result from acute frontal sinusitis [23, 26–35]. An epidemiologic study of intracranial complications of ABRS in US children recorded between 2.7 and 4.3 cases per million per year.

Infections can spread from the frontal sinus to intracranial structures, or less commonly to the orbits, by hematogenous or direct routes.

- The frontal sinus is susceptible to extrasinus spread of infection in part because its venous drainage occurs through diploic veins that traverse the posterior table and communicate with the venous supply of the meninges, cavernous sinus and dural sinuses.

Septic thrombophlebitis of the sinus submucosal venous net spreads through the valveless veins into the frontal bone dipole and then to the meningeal veins. These venous channels may be more porous in the developing sinus, and thus adolescents and young adults (especially male) are at increased risk for complications of acute frontal sinusitis. Alternatively, infection can reach the intracranial or orbital structures by erosion of the frontal sinus posterior table or floor, respectively, or through congenital or acquired bony dehiscences.

The workup of the patient with a suspected complication of acute frontal sinusitis includes carefully directed history and exam with specific attention to neurologic and ophthalmologic symptoms and signs. Nasal endoscopy should be performed to culture purulent material that can guide antimicrobial therapy. Lumbar puncture may also be indicated to obtain CSF cultures and to rule out meningitis, but only after exclusion of an abscess using imaging. Consultations with an ophthalmologist, neurosurgeon, neurologist, or infectious disease specialist should be considered.

Whereas radiologic imaging is usually unnecessary in uncomplicated acute frontal sinusitis, radiologic studies play an important role in confirming and characterizing

Table 6.1 Intracranial complications

Epidural abscess
Subdural abscesses
Intraparenchymal brain abscess
Meningitis
Encephalitis
Superior sagittal thrombosis
Cerebral infarcts
Cavernous sinus thrombosis

the extent of disease in patients with extrasinus complications. CT scan with intravenous contrast is the imaging modality of choice in evaluating intracranial or orbital complications of acute frontal sinusitis. CT scans can characterize bony erosions of the frontal sinus as well as phlegmons or rim-enhancing fluid collections in adjacent orbital and intracranial soft tissue. Serial imaging studies should be considered in patients who appear clinically unresponsive to initial treatment. MRI may also be useful, being more sensitive than CT in evaluating intracranial pathology, particularly when CT scans are negative or inconclusive in the setting of high suspicion for intracranial complication [36].

Intracranial Complications

The most common intracranial complications caused by acute frontal sinusitis are epidural and subdural abscesses [23, 26–35]. Table 6.1 lists the range of intracranial complications from acute frontal sinusitis. Figure 6.1 depicts CT and MRI scans of a patient with frontal sinusitis complicated with intracerebral abscess.

Intracranial complications should be suspected when symptoms are protracted or more severe than would be expected for a typical case of acute sinusitis. The most common symptoms are severe frontal headache and fever. Other common warning signs are depicted in Table 6.2. Surprisingly, however, only 50 % of patients who manifest with complicated acute frontal sinusitis experience symptoms of acute sinusitis during the 1–2 weeks prior to presentation. Thirty to 40 % of patients with complicated AFS receive antibiotics in the weeks prior to presentation. The majority do not have a history of previous sinus problems.

Sinus cultures in patients with intracranial complications of acute frontal sinusitis may reveal no growth in up to 25 %. Nonetheless the most common cultured bacteria reported in these cases are Streptococcal species, Staphylococcal species and anaerobes. Gram-negative infections occur less frequently [23, 27, 28, 30, 33, 34]. Table 6.3 lists the most common pathogens.

Because complicated frontal sinusitis can progress rapidly with high morbidity, a high degree of clinical suspicion for potential complications should be maintained during the workup of patients with severe or persistent presentations of acute

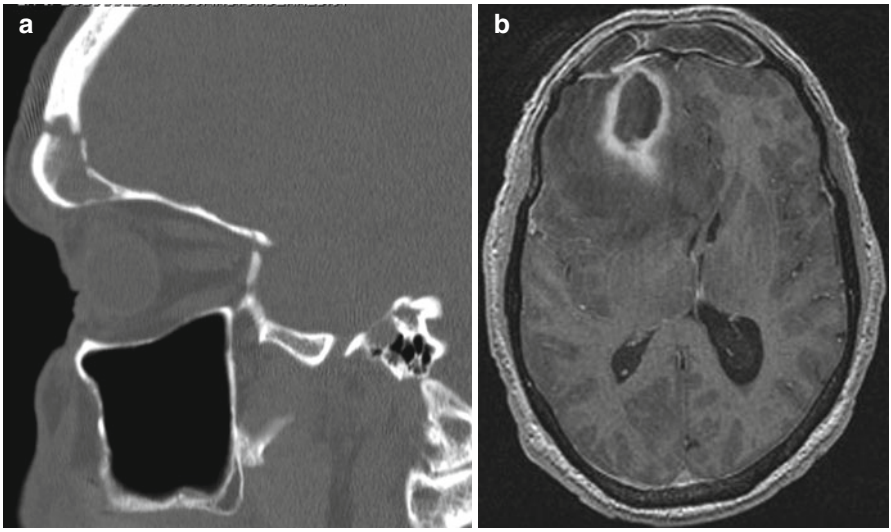

Fig. 6.1 (**a**) Non contrast CT scan of a patient with complicated frontal sinusitis showing erosion of both anterior and posterior tables of the frontal sinus. (**b**) MRI brain T 1 post contrast of the same patient showing right frontal lobe intraparenchymal abscess associated with right frontal lobe epidural enhancement and bilateral frontal sinus mucosal thickening

Table 6.2 Warning signs for intracranial complication

Severe frontal headache
Altered mental status
Fever >39 °C
Cranial nerve palsy
Hemiparesis
Seizures
Nausea, vomiting
Photophobia
Nuchal rigidity
Forehead swelling
Focal neurologic signs
New onset seizures

rhinosinusitis. Those patients with a confirmed diagnosis of complicated acute frontal sinusitis should be admitted emergently for intravenous antibiotic therapy, intravenous hydration, serial neurologic examination, and consideration for surgical treatment. If cultures can be obtained, these should be performed expeditiously so as to not interfere with the initiation of intravenous antibiotics. If cultures are not possible, empiric antibiotic therapy should be initiated immediately, choosing broad spectrum agents that have favorable penetration of the blood-brain barrier. As mentioned previously, a significant percentage of cultures from patients with intracranial

Table 6.3 Common pathogens cultured in intracranial complications of acute frontal sinusitis

Aerobic bacteria
Strep pneumoniae
Strep milleri/anginosus
Strep intermedius
Staphylococcus aureus
Staph coagulase negative
Anaerobic bacteria
Fusobacterium sp.
Peptostreptococcus
Prevotella
Porphyromonas sp.
Bacteroides sp.
Propionibacterium acnes

complications are negative. This may perhaps occur because antibiotic therapy is often initiated emergently before cultures can be obtained. The duration of antimicrobial treatment varies with the nature and severity of the complication, as well as the response to initial therapy. Depending on the degree of morbidity, many patients with complicated acute frontal sinusitis will require continuation of intravenous antibiotic therapy as an outpatient after resolution of the acute phase of illness. Oral antibiotic therapy may be appropriate in selected patients.

The use of intravenous corticosteroids in patients with complicated AFS is controversial. Some studies have advocated their use in patients with cerebral edema and clinical deterioration [23] while others argue that they may interfere with antibiotic penetration and immune response [37]. No prospective studies or animal models have conclusively shown that steroids improve mortality or morbidity associated with cerebral edema; thus the use of corticosteroids should be considered on an individual basis.

Surgical treatment should include craniotomy to evacuate any intracranial abscess, and concurrent drainage of the frontal sinus. Methods of draining the frontal sinus include trephination and endoscopic frontal sinusotomy (Draf 2a/Draf 2b). The advantages of trephination include technical simplicity, good efficacy of decompressing and draining the sinus, and provision of a portal to the sinus lumen for irrigation. Disadvantages of trephination include potential scar from the external incision, potential injury to the supraorbital nerve, and failure to address the critical area of impaired outflow of the sinus.

In experienced hands, endoscopic frontal sinusotomy is a satisfactory alternative technique for surgical management of complicated AFS. The endoscopic approach provides a minimally invasive means of improving frontal sinus drainage through its natural outflow tract. Disadvantages of the endoscopic approach include its technical complexity as well as the potential difficulty of obtaining adequate visualization in the acutely infected milieu. In addition, there is a higher risk of post-operative synechia and stenosis of the frontal sinus ostium. Use of silicone stents and creation of Draf 2b cavities has been reported in one study [28] to achieve a low rate of re-stenosis. Balloon dilation techniques may be an appropriate alternative for surgical enhancement of frontal sinus drainage.

- In recent series, the mortality rate from intracranial complications of frontal sinusitis has been found to have decreased from earlier reports, but remains a notable 5 %.

Furthermore, 15–40 % of patients are reported to have residual neurological sequelae. These include cognitive defects in visual and verbal memory, new onset seizure disorder, cranial nerve palsies, hemiparesis, frontal syndrome and blindness. Patients with neurological deficits at the time of clinical presentation are at much higher risk for late or persistent sequelae compared to patients presenting without neurological symptoms.

Orbital Complications

- Isolated acute frontal sinusitis infrequently causes orbital complications. However, acute frontal sinusitis in the context of pansinusitis is associated with 60–80 % of orbital complications [38, 39].

Although direct spread to the orbits from the frontal sinus is possible, the ethmoid sinuses are more commonly implicated in the development of orbital complications. Potential orbital complications include orbital cellulitis, subperiosteal abscess, orbital abscess and cavernous sinus thrombosis. A subperiosteal abscess that is directly associated with frontal sinusitis is typically located supero-laterally within the orbit, displacing the globe medially and inferiorly.

Signs of an orbital complication include periorbital edema/erythema, chemosis, proptosis/globe displacement, double vision and ophthalmoplegia. Diminished visual acuity is a sign of advanced disease. Cranial neuropathies involving 3, 4, V1 and V2 and/or 6 may be associated with cavernous sinus thrombosis. Ophthalmological consultation is a critical part of the workup. CT scan of the sinus and orbits with IV contrast should be obtained to make the diagnosis.

Surgical treatment is indicated in patients not responding to 24–48 h of IV antibiotics or in patients with evidence of reduced visual acuity. Surgical drainage may be performed endoscopically in experienced hands [40], or through an external approach via Lynch incision with or without frontal trephination.

Frontal Bone Osteomyelitis

Osseous complications of AFS occur in 5–10 % of the cases. Osteomyelitis of the frontal sinus may be caused by direct extension of infection or by thrombophlebitis of the diploic veins. The resulting vascular necrosis caused by frontal sinus osteitis leads to erosion of the anterior table of the frontal sinus, with possible progression to osteomyelitis.

- Of the paranasal sinuses, the frontal sinus is most commonly associated with osteomyelitis.

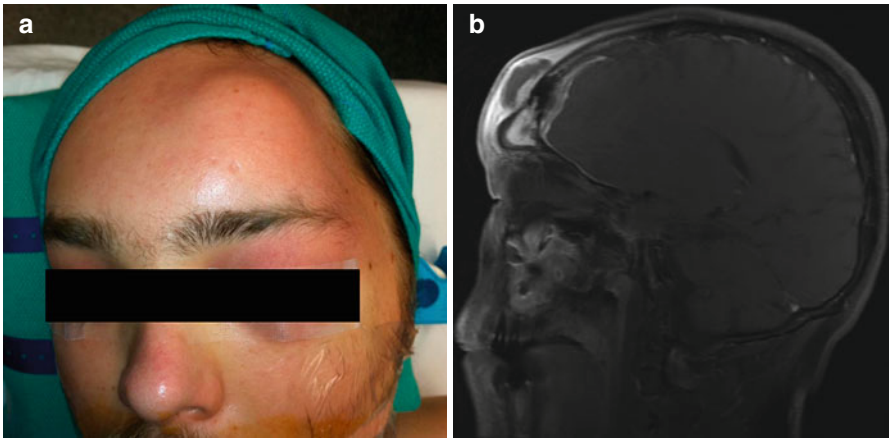

Fig. 6.2 (**a**) Patient with left forehead Pott's Puffy tumor. (**b**) MRI brain T 1 post contrast of the same patient showing a subgaleal abscess, left frontal sinus mucosal thickening and inflammation and left frontal lobe epidural enhancement

When osteomyelitis involves the anterior table, a subperiosteal abscess may develop, presenting as a subcutaneous fluctuant protuberance over the brow or forehead (Fig. 6.2). This abscess is known as Pott's Puffy tumor, which was first described by Sir Percival Pott in 1775. Strictly an infectious complication and not neoplastic in any way, Pott's Puffy Tumor may present with severe headache, fever, and photophobia.

Frontal bone osteomyelitis is predominantly observed in adolescents and young adults and is a risk factor for intracranial complications such as subdural empyema and brain abscess, which have been observed in 60–100 % of cases [41]. The most common organisms are streptococci, staphylococci and anaerobic bacteria.

Treatment should include administration of broad spectrum IV antibiotics and early surgical drainage. At a minimum, surgical drainage should include percutaneous drainage of the subperiosteal abscess, as well as drainage of the frontal sinus by either trephination or endoscopic frontal sinusotomy. Debridement of the infected bone may be indicated as well, although studies have shown that percutaneous drainage and repeated antibiotic irrigations through an externally placed drain may be effective and may substitute debridement [42]. In general, intravenous antibiotics are recommended for 4–6 weeks.

References

1. Fokkens WJ, Lund VJ, Mullol J, Bachert C, Alobid I, Baroody F, et al. European position paper on rhinosinusitis and nasal polyps 2012. Rhinol Suppl. 2012;50:1–12, 3 p preceding table of contents, 1–298.
2. Fairlie T, Shapiro DJ, Hersh AL, Hicks LA. National trends in visit rates and antibiotic prescribing for adults with acute sinusitis. Arch Intern Med. 2012;172(19):1513–4.

3. Mattos JL, Woodard CR, Payne SC. Trends in common rhinologic illnesses: analysis of U.S. healthcare surveys 1995–2007. Int Forum Allergy Rhinol. 2011;1(1):3–12.
4. Eloy P, Poirrier AL, De Dorlodot C, Van Zele T, Watelet JB, Bertrand B. Actual concepts in rhinosinusitis: a review of clinical presentations, inflammatory pathways, cytokine profiles, remodeling, and management. Curr Allergy Asthma Rep. 2011;11(2):146–62.
5. Lieu JE, Feinstein AR. Confirmations and surprises in the association of tobacco use with sinusitis. Arch Otolaryngol Head Neck Surg. 2000;126(8):940–6.
6. Savolainen S. Allergy in patients with acute maxillary sinusitis. Allergy. 1989;44(2):116–22.
7. Schatz M, Zeiger RS, Chen W, Yang SJ, Corrao MA, Quinn VP. The burden of rhinitis in a managed care organization. Ann Allergy Asthma Immunol. 2008;101(3):240–7.
8. Kuhn FA. Surgery of the frontal sinus. In: Kennedy DW, Bolger WE, Zinreich SJ, editors. Diseases of the sinuses: Diagnosis and Management. Hamilton, Ontario: BC Decker Inc.; 2001. p. 281–301.
9. Lien CF, Weng HH, Chang YC, Lin YC, Wang WH. Computed tomographic analysis of frontal recess anatomy and its effect on the development of frontal sinusitis. Laryngoscope. 2010;120(12):2521–7.
10. Rosenfeld RM, Andes D, Bhattacharyya N, Cheung D, Eisenberg S, Ganiats TG, et al. Clinical practice guideline: adult sinusitis. Otolaryngol Head Neck Surg. 2007;137(3 Suppl):S1–31.
11. Hwang PH. A 51-year-old woman with acute onset of facial pressure, rhinorrhea, and tooth pain: review of acute rhinosinusitis. JAMA. 2009;301(17):1798–807.
12. Chow AW, Benninger MS, Brook I, Brozek JL, Goldstein EJ, Hicks LA, et al. IDSA clinical practice guideline for acute bacterial rhinosinusitis in children and adults. Clin Infect Dis. 2012;54(8):e72–112.
13. Seiden AM, Martin VT. Headache and the frontal sinus. Otolaryngol Clin North Am. 2001;34(1):227–41.
14. Brook I. Microbiology of sinusitis. Proc Am Thorac Soc. 2011;8(1):90–100.
15. Benninger MS, Payne SC, Ferguson BJ, Hadley JA, Ahmad N. Endoscopically directed middle meatal cultures versus maxillary sinus taps in acute bacterial maxillary rhinosinusitis: a meta-analysis. Otolaryngol Head Neck Surg. 2006;134(1):3–9.
16. Antila J, Suonpaa J, Lehtonen OP. Bacteriological evaluation of 194 adult patients with acute frontal sinusitis and findings of simultaneous maxillary sinusitis. Acta Otolaryngol Suppl. 1997;529:162–4.
17. Brook I. Bacteriology of acute and chronic frontal sinusitis. Arch Otolaryngol Head Neck Surg. 2002;128(5):583–5.
18. Brook I. Acute and chronic frontal sinusitis. Curr Opin Pulm Med. 2003;9(3):171–4.
19. Zalmanovici A, Yaphe J. Intranasal steroids for acute sinusitis. Cochrane Database Syst Rev. 2009;4:CD005149.
20. Venekamp RP, Thompson MJ, Hayward G, Heneghan CJ, Del Mar CB, Perera R, et al. Systemic corticosteroids for acute sinusitis. Cochrane Database Syst Rev. 2011;12:CD008115.
21. Kassel JC, King D, Spurling GK. Saline nasal irrigation for acute upper respiratory tract infections. Cochrane Database Syst Rev. 2010;3:CD006821.
22. Hopkins C, Noon E, Roberts D. Balloon sinuplasty in acute frontal sinusitis. Rhinology. 2009;47(4):375–8.
23. Hansen FS, Hoffmans R, Georgalas C, Fokkens WJ. Complications of acute rhinosinusitis in The Netherlands. Fam Pract. 2012;29(2):147–53.
24. Piatt Jr JH. Intracranial suppuration complicating sinusitis among children: an epidemiological and clinical study. J Neurosurg Pediatr. 2011;7(6):567–74.
25. Oxford LE, McClay J. Complications of acute sinusitis in children. Otolaryngol Head Neck Surg. 2005;133(1):32–7.
26. Bair-Merritt MH, Shah SS, Zaoutis TE, Bell LM, Feudtner C. Suppurative intracranial complications of sinusitis in previously healthy children. Pediatr Infect Dis J. 2005;24(4):384–6.
27. Bayonne E, Kania R, Tran P, Huy B, Herman P. Intracranial complications of rhinosinusitis. A review, typical imaging data and algorithm of management. Rhinology. 2009;47(1):59–65.

28. Betz CS, Issing W, Matschke J, Kremer A, Uhl E, Leunig A. Complications of acute frontal sinusitis: a retrospective study. Eur Arch Otorhinolaryngol. 2008;265(1):63–72.
29. DelGaudio JM, Evans SH, Sobol SE, Parikh SL. Intracranial complications of sinusitis: what is the role of endoscopic sinus surgery in the acute setting. Am J Otolaryngol. 2010; 31(1):25–8.
30. Germiller JA, Monin DL, Sparano AM, Tom LW. Intracranial complications of sinusitis in children and adolescents and their outcomes. Arch Otolaryngol Head Neck Surg. 2006; 132(9):969–76.
31. Glickstein JS, Chandra RK, Thompson JW. Intracranial complications of pediatric sinusitis. Otolaryngol Head Neck Surg. 2006;134(5):733–6.
32. Hakim HE, Malik AC, Aronyk K, Ledi E, Bhargava R. The prevalence of intracranial complications in pediatric frontal sinusitis. Int J Pediatr Otorhinolaryngol. 2006;70(8):1383–7.
33. Hicks CW, Weber JG, Reid JR, Moodley M. Identifying and managing intracranial complications of sinusitis in children: a retrospective series. Pediatr Infect Dis J. 2011;30(3):222–6.
34. Kombogiorgas D, Seth R, Athwal R, Modha J, Singh J. Suppurative intracranial complications of sinusitis in adolescence. Single institute experience and review of literature. Br J Neurosurg. 2007;21(6):603–9.
35. Leotta N, Chaseling R, Duncan G, Isaacs D. Intracranial suppuration. J Paediatr Child Health. 2005;41(9–10):508–12.
36. Blumfield E, Misra M. Pott's puffy tumor, intracranial, and orbital complications as the initial presentation of sinusitis in healthy adolescents, a case series. Emerg Radiol. 2011; 18(3):203–10.
37. Clayman GL, Adams GL, Paugh DR, Koopmann Jr CF. Intracranial complications of paranasal sinusitis: a combined institutional review. Laryngoscope. 1991;101(3):234–9.
38. Jackson K, Baker SR. Clinical implications of orbital cellulitis. Laryngoscope. 1986; 96(5):568–74.
39. Schramm Jr VL, Curtin HD, Kennerdell JS. Evaluation of orbital cellulitis and results of treatment. Laryngoscope. 1982;92(7 Pt 1):732–8.
40. Roithmann R, Uren B, Pater J, Wormald PJ. Endoscopic drainage of a superiorly based subperiosteal orbital abscess. Laryngoscope. 2008;118(1):162–4.
41. Ketenci I, Unlu Y, Tucer B, Vural A. The Pott's puffy tumor: a dangerous sign for intracranial complications. Eur Arch Otorhinolaryngol. 2011;268(12):1755–63.
42. Akiyama K, Karaki M, Mori N. Evaluation of adult Pott's puffy tumor: our five cases and 27 literature cases. Laryngoscope. 2012;122(11):2382–8.