

# Chapter 2

## Epidemiology and Risk Factors

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### Abbreviations

|     |                         |
|-----|-------------------------|
| CNS | Central nervous system  |
| ENT | Ear, nose, and throat   |
| SSI | Surgical site infection |

### Epidemiology

Osteomyelitis may affect any bone, with a predilection for the tubular bones of the arms and legs [1]. Osteomyelitis is primarily a disease of young children presumably because of the rich vascular supply of their rapidly growing bones [2]. The skull (neurocranium) was recognized as a rare anatomic site, but potentially serious. However, cranial osteomyelitis is regularly seen in countries with lesser sanitary capacities. Migratory flows, the ease of travelling, and number of trips have led to the possibility of the discovery and development of infectious diseases that are usually uncommon in developed countries. We must keep in mind the changing traits of cranial osteomyelitis. The presumed rarity of osteomyelitis of the skull, led us to conduct a simple search study with MEDLINE as the mean search engine. To obtain the highest number of skull osteomyelitis, the following Boolean operator combinations were used: ((“skull” and “osteomyelitis”) OR (“cranial” and “osteomyelitis”)) in English-language literature from 1955 to 2014. The search yielded



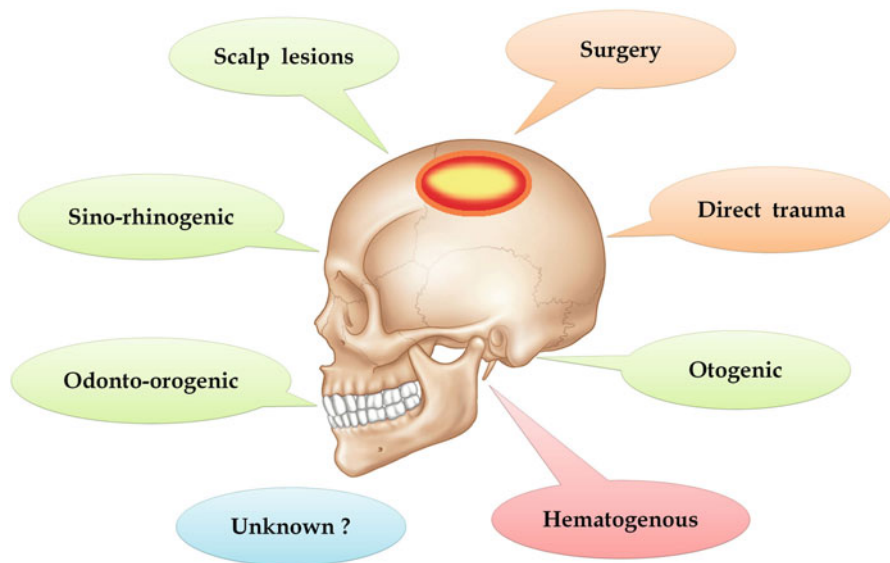
**Fig. 2.1** Bar graph showing progression of number of English language articles about *cranial osteomyelitis* found in MEDLINE database over time (from 1955 to 2014)

612 articles and we can see a significant increase in the number of articles published recently in peer-reviewed literature particularly in the first decade of the new millennium (Fig. 2.1). For many authors, this high article number seen recently suggests that this complication is far more common than supposed [3–13]. The exact reason is most likely due to either of the following:

- Firstly, a more adequate access to radiological imaging has allowed more accurate modality for the diagnosis of complications of dental, paranasal sinus and ear infections.
- Secondly, an increased awareness of physicians for cranial infections.
- Finally, a rise in the frequency and complexity of cranial neurosurgical procedures currently performed.

Many sources of osteomyelitis are described in the skull (Fig. 2.2). In developing countries, paranasal sinusitis and scalp infections remain the most common causes of cranial osteomyelitis, while in industrialized nations, postsurgical craniotomy infections have become the predominant source [8, 11, 14, 15]. However, in many developed countries, otorhinolaryngologic infections have increased the relative incidence of contiguous cranial osteomyelitis that nowadays equals that of post-operative osteomyelitis [4, 6, 12, 13, 16].

In the world literature, there are no valid data about the exact incidence of skull osteomyelitis in general. So, its prevalence was difficult to estimate from previous case series as a result from small patient numbers, the frequent inclusion of patients with acute/chronic osteomyelitis without confirmed diagnosis and variation in the diagnostic methods used. In developed countries, cranial osteomyelitis account for about 0.3–1.5 % of all skeletal localizations [1, 17, 18]. While the incidence of



**Fig. 2.2** Schematic drawing of sources of osteomyelitis formation in the skull bones

cranial vault tuberculosis is estimated to be between 0.2 and 1.3 % of all cases of skeletal tuberculosis [19]. Epidemiologic evaluation of some institutes would suggest that between one to four cases of cranial osteomyelitis would be seen per year in general neurosurgical practice. In our experience, during a 13-year-study period from 2000 to 2012, 29 cases of osteomyelitis of the skull were treated in the department of neurosurgery of Mohammed V Military teaching hospital, Rabat, Morocco, under the direction of professor Boucetta (mean of 2.4 cases per year). Nineteen patients were male (65.5 %) and the mean age at diagnosis was 41.9 years (range from 11 to 76 years). The cranial vault was involved in 25 patients (86.2 %) and 4 cases (13.8 %) had a skull base osteomyelitis. Contiguous spread of infection from paranasal sinusitis or odonto/otogenic infections was found in 13 patients (44.8 %). Also, post-craniotomy osteomyelitis was seen in 13 cases (44.8 %). One patient (3.4 %) had a penetrating head injury and a hematogenous spread infection was suspected in two patients (6.9 %). Some concurrent illnesses were found with skull osteomyelitis such as diabetes (3 cases), malignancy (3 cases), previous radiation therapy (3 cases) and renal failure (2 cases).

On the other hand, the overall incidence of skull osteomyelitis seems higher in other developing countries particularly in sub-Saharan regions of Africa, where access to health care and advanced diagnostic testing is limited [18]. Recently, 8 cases (12.7 %) of cranial osteomyelitis were reported among 63 cases of pediatric osteomyelitis in a tertiary referral centre in Tanzania (East Africa) between 2008 and 2010 [20]. During the period 1983–1997, Nathoo et al. reported 699 patients with cranial subdural empyemas in South Africa. Among them, 47 patients (6.7 %)

presented a skull osteomyelitis which was removed [21]. Infections following open traumatic injuries are less frequent.

Overall in neurosurgical practice, it seems that oro-rhino-otogenic infections account for as many as 40–50 % of cases of skull osteomyelitis. Direct surgical site infections and the use of cranioplasty materials also contribute for about 40–50 % of cases. Other major causes of cranial osteomyelitis include contiguous source following head injuries (5 %) [22] and more rarely hematogenous seeding secondary to bacteremia or fungemia [17, 23–26]. Furthermore, on extremely rare occasions, risk factors or etiology may not be ascertained.

**Risk Factors for Skull Osteomyelitis**

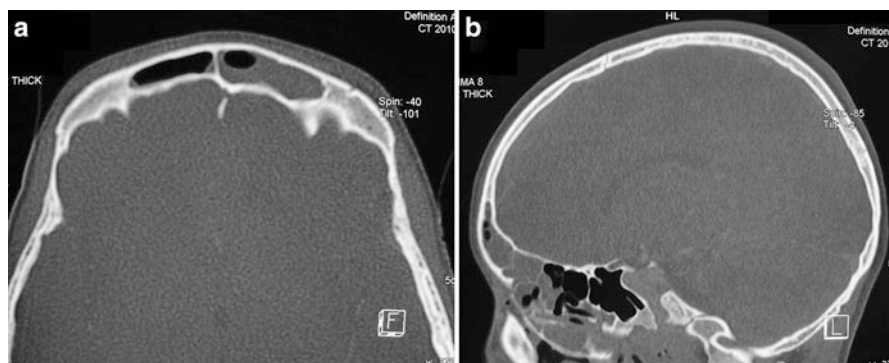
A multitude of factors have been identified that result in skull osteomyelitis. However, this disease is usually related to the following three main sources (Table 2.1):

*Direct Spread from Contiguous Sites*

Frontal sinusitis is the most important predisposing factor for osteomyelitis of the skull especially in children, adolescents and young adults than in other age-groups (Fig. 2.3). The predilection for this age-group is explained by the peculiarities of the development of frontal sinuses. Adolescents are likely to have highly vascular diploic bone, which increases valveless bidirectional flow between the frontal sinus mucosa and dural venous drainage [27]. A cranial osteomyelitis was seen in 0.5–9 % of patients with suppurative intracranial complications of paranasal

**Table 2.1** The main sources of infection in cranial osteomyelitis

| Sources of infection                                       | Causes                                             |
|------------------------------------------------------------|----------------------------------------------------|
| Direct extension from contiguous site of infection         | Paranasal sinusitis                                |
|                                                            | Otitis or otomastoiditis                           |
|                                                            | Oral or odontogenic infections                     |
|                                                            | Spontaneous scalp infections                       |
| Post-surgical or post-traumatic direct inoculation         | Neurosurgical site infections (primary)            |
|                                                            | Following cranioplasty (secondary)                 |
|                                                            | Otologic site infections                           |
|                                                            | Craniofacial injuries (scalp and/or bone fracture) |
|                                                            | Cephalhematomas                                    |
| Hematogenous dissemination from remote source of infection | Lung, spine, peripheral arthritis, meningitis      |
|                                                            | Cryptogenic                                        |



**Fig. 2.3** Axial (a) and sagittal reconstructions (b) cranial CT-scan in bone windows showing left frontal sinusitis

sinusitis in neurosurgical practice [28–31]. In head and neck, incidence of cranial osteomyelitis may be as high as 28 % [8].

As all other forms of cranial osteomyelitis, Pott's puffy tumor is reported as rare in the antibiotic therapy era, although, despite the use of broad-spectrum antibiotics, the number of cases reported has increased since 2000 [32–38]. In 2012, 141 cases of Pott's puffy tumor were reported in the world literature by Nisa et al. [7]. This form of cranial osteomyelitis is more common in male and almost always confined to the second decade of life, more rarely in adults and newborns. The prevalence in young males is attributed to their larger diploic veins, which makes it easier for infections to spread [31, 35].

Besides the frontal sinus, the other paranasal sinuses should be taken into considerations: ethmoidal sinuses (Fig. 2.4), maxillar sinuses (Fig. 2.5) if not all paranasal sinuses (pansinusitis) (Fig. 2.6). Osteomyelitis of the calvaria can also spread from orbital (Fig. 2.7), oral or dental infections [7, 16, 25, 31, 33, 37, 39, 40]. One of our patients developed an occipital osteomyelitis 3 weeks following a dental procedure [41].

The cranial vault is more commonly involved than the skull base. Skull base osteomyelitis is rare, mainly seen in ear, nose, and throat (ENT) practice following ear and mastoid infections (Fig. 2.8) or secondary to sphenoidal sinusitis [42–46] (Fig. 2.9). The patients often are elderly or immunocompromised or have micro-vascular disease such as diabetes [3, 4]. The invasive form of malignant external otitis, an otitis-associated skull base infection, remains the most frequent appearance [47]. Rothholtz and colleagues (otolaryngologists) from California, have published their experience with skull base osteomyelitis. They found a remarkable higher number of cases than previously reported articles: 820 patients over an 11-year period from 1990 to 2000. The male to female ratio was nearly 1:1. Over 90 % of the patients were over the age of 25 and 38 % of the patients were over the age of 65 years. With respect to race, the white population (69.3 %) was more likely to present with the disease than Native American (13.2 %), African American



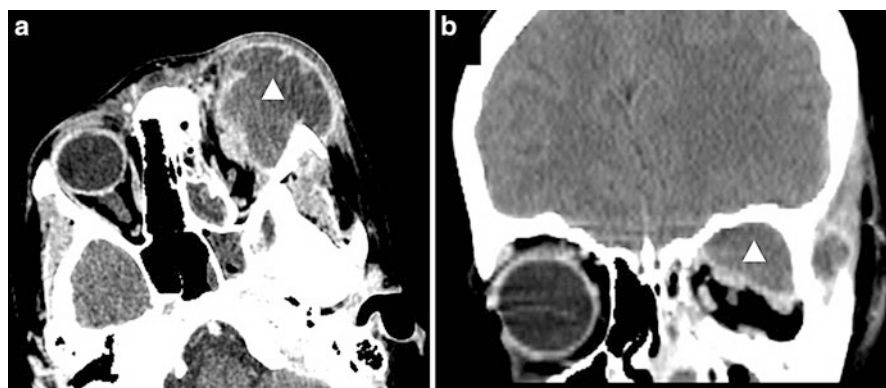
**Fig. 2.4** Axial CT-scan in bone windows with bilateral ethmoiditis



**Fig. 2.5** Bone-windows axial CT-scan showing left maxillary sinusitis



**Fig. 2.6** Coronal reconstructions CT-scan in bone windows demonstrating bilateral paranasal pansinusitis



**Fig. 2.7** Axial (a) and coronal reconstructions (b) CT-scan after contrast injection showing a left orbital abscess (*triangle*). Note adjacent sinusitis in the ethmoid cells and sphenoidal sinus

(6.5 %), or Asian (2.9 %) descent [3]. In contrast, only 12 cases were identified in a single department of ENT in New Zealand from 2004 to 2011 [6]. A prospective study in India from 2001 to 2008 found 20 patients with temporal bone osteomyelitis. The most common age-group affected was the 40–60 years (65 % of cases) without sex predilection. Interestingly, there were two sets of identical twin girls of 2 and 3 years of age respectively [5]. A systematic review of case series of central skull base osteomyelitis unrelated to otologic infection from 1946 to 2013 found 42 cases with mean age of 52 years and male female ratio of 2.2 to 1 [4].



**Fig. 2.8** Axial CT-scan in bone windows demonstrating bilateral (a, b) chronic otomastoiditis



**Fig. 2.9** Axial (a) and coronal reconstructions (b) CT-scan in bone windows showing bilateral sphenoidal sinusitis

As seen above, the epidemiology of temporal bone osteomyelitis, which is classically a part of skull base osteomyelitis, was changed in the last decade. In the past, it was predominantly seen in elderly diabetic and immunocompromised patients with history of episodic otitis externa in the months prior to presentation. In contrast with the typical features, several atypical cases have been reported more recently. Often a source of infection is not found and patients were younger and previously healthy [4, 48]. Paranasal sinuses and hematological spread from distant sites of infection are considered possible sources.

The scalp is a rare site for suppuration. The risk of its infection stems from its proximity to the skull and the possibility of spreading disease. Non healing ulcerations and chronic sinus tracts should be suspected of harboring infection. Previous scalp lesions over the skull can lead to osteomyelitis. Major causes include any spontaneous scalp infections [49, 50] (Fig. 2.10) as well as carbuncle, scalp abscess [51–53] (Fig. 2.11), scalp pressure ulcer [54] (Fig. 2.12), nasal dermal sinus tract

**Fig. 2.10** Spontaneous scalp abscess near the vertex



**Fig. 2.11** Important subperiosteal frontal extracranial abscess. Air may be seen within the collection



**Fig. 2.12** Bilateral chronic parieto-occipital non-healing scalp pressure ulcer



[55], facial cellulitis [56, 57] (Fig. 2.13), scalp necrotizing fasciitis [58] and more rarely epidermal necrolysis of the scalp [59]. Interestingly, a 5-month-old girl was previously reported with 1 cm right parietal bone osteomyelitis under a pox lesion of the scalp related to varicella infection [60].

### *Postoperative or Secondary to Direct Trauma*

According to the criteria of the Centers for Disease Control [61], a surgical site infection (SSI) was considered in the presence of one of the following cases: a purulent discharge from the wound, a serous discharge with positive bacterial culture, a deep or superficial wound abscess with or without positive bacterial culture, wound swelling and erythema with or without pyrexia, or meningitis/ventriculitis with positive bacterial culture or microorganisms seen on the Gram's stain, occurring within 30 days of the surgical procedure. More specifically, a superficial incisional SSI in the scalp was defined as having a purulent discharge or cutaneous dehiscence with positive microbiological testing. A deep incisional SSI, such as osteitis or discitis, was diagnosed on the basis of a positive computed tomographic or magnetic resonance imaging scans associated with or without positive microbiological testing; an organ/space SSI, such as a brain abscess, was determined by both magnetic resonance imaging and computed tomography parenchymal alteration, which was surgically verified and confirmed by histological or microbiological testing. Meningitis was diagnosed by a positive cerebrospinal fluid

**Fig. 2.13** Left frontal swelling with orbito-facial cellulitis on the same side (with the patient's signed permission)



stain or culture or the alteration of cytochemical parameters [62]. In 2008, these criteria were modified in a minor fashion [63].

Despite improvements in prevention, surgical site infections remain a significant clinical problem as they are associated with major source of morbidity and mortality. The primary site of inflammation after a central nervous system (CNS) infection is the choroid plexus, in which about 100,000 bacterial organisms per gram of tissue are needed to produce a postoperative CNS infection [64]. It's well known that postoperative infections represent an important contributor to the cost of care (long hospital stays and costly interventions) and a true frustrating problem for clinicians. The cost for SSI after craniotomy was estimated by O'Keeffe and colleagues to be over £9000 for each case of infection [65]. For Broex et al., healthcare costs for a patient with SSI are, on average, approximately twice the amount of costs for a patient without an SSI [66].

The incidence of SSI in cranial neurosurgery is reported between the range of 0–17.6 % (average rate of around 5 %), depending on the surgical procedure, the surveillance criteria used, and the quality of data collection [10, 62, 65, 67–75]. Interestingly, the infection rates before and after 1940 are nearly the same [75]. Generally, depth of SSI is classified as subgaleal, bone, epidural, subdural, and/or intracerebral. A given layer was considered infected if there was gross purulent material in that layer intraoperatively or if cultures taken from that layer were positive [76]. A variety of risks factors for SSI have been reported and debated in neurosurgery as well as: duration of surgery (longer than 4 h), presence of

cerebrospinal fluid leak (which provides a portal of access to deeper wound layers and increase complications from infection), reoperation (especially for glioma), type of surgery (craniofacial, emergency and dirty surgeries carry a greater risk of infection), method of surgery (craniotomy or craniectomy), type of tumor (especially meningioma) method of wound closure (staples or sutures) prior radiation therapy, use of invasive devices and immunosuppression of the patient [10, 14, 62, 69, 70, 75, 77].

The incidence of post-surgical cranial osteomyelitis is often not reported separately from other deep neurosurgical infections and may be considered together with scalp infection, extradural abscess and subdural empyema. Bone flap osteomyelitis after craniotomy is relatively unusual accounting for about a fifth of SSI (mean of 21.2 %) but this incidence can vary greatly from 0 to 60.8 % (Table 2.2). The rarity of this complication demands high case numbers to permit conclusive analysis beyond anecdotal case reports. In this setting, cranial osteomyelitis is more common in adult population than in children. The age of distribution of patients is older than that of sinusitis-related cranial osteomyelitis, reflecting the greater number of older patients requiring cranial surgery. Few recent data have been published. The incidence of postsurgical skull osteomyelitis reported in the literature ranges from 0 to 3.1 % with a mean of 1.2 % (Table 2.2). Surgery with implants like cranioplasty and bone flap fixating material has to be followed up for 12 months in order to rule out infection because of the long latency period for some chronic bone flap infection. One of our patients presented with a suppurative acrylic cranioplasty, exposure of implant, and proximate parietal cranial osteomyelitis 11 years following a craniotomy for an acute subdural hematoma (Fig. 2.14). Another 52-year-old woman was operated 25 years ago for a frontal tuberculoma with cranioplasty (methylmethacrylate). She developed a secondary suppurative denuded cranioplasty with contiguous cranial osteomyelitis (please see chap. 10 for more details about this case report). Blumenkopf et al. reported a 17-year-old man with infected cranioplasty and adjacent skull osteomyelitis revealing by purulent drainage from scalp sinus tract. These complications have occurred 10 years after cranioplasty and ventriculoperitoneal shunt following a brain abscess surgical exploration and post-meningitic hydrocephalus [78]. Even, without implants, bone flap osteomyelitis may occur several years after the first surgery. In 2001, Karabatsou et al. reported a patient with a Pott's puffy tumor manifesting itself 13 years after a hemispherectomy for intractable seizures [79]. Wilson et al. described a case of skull osteomyelitis 24 years after an intracerebral hematoma evacuation secondary to an arteriovenous malformation [80]. Recently, Levitt et al. reported a patient with an asymptomatic postoperative osteomyelitis occurring 23 years after initial craniotomy. The rarity of postoperative cranial osteomyelitis has been attributed to the vascularity of the cranial vault and the preventive efficacy of perioperative antibiotics [81]. However, for some authors, the true efficacy of prophylactic antibiotics remains controversial [62, 67, 69, 73]. Postoperative cranial wound dehiscence may induce bone flap osteomyelitis and several factors may contribute to this complication including a history of prior scalp irradiation, neoadjuvant of postoperative chemotherapy, development of a cerebrospinal fluid

**Table 2.2** Incidence of postoperative cranial osteomyelitis in more recently published studies

| Authors [reference number] | Year of publication | Country   | Time of data collection | Number of patients with CS | Incidence of SSI (number of patients)             | Incidence of cranial osteomyelitis relative to patients with CS (number of patients) | Incidence of cranial osteomyelitis relative to patients with SSI |
|----------------------------|---------------------|-----------|-------------------------|----------------------------|---------------------------------------------------|--------------------------------------------------------------------------------------|------------------------------------------------------------------|
| Rasmussen et al. [73]      | 1990                | Denmark   | 3 years                 | 202                        | n/a                                               | 6.4 % (13) <sup>a</sup>                                                              | n/a                                                              |
| Whitby et al. [67]         | 2000                | Australia | 2 years                 | 613                        | 2.4 % (15)                                        | 0                                                                                    | 0                                                                |
| Idali et al. [68]          | 2004                | Morocco   | 2003                    | 170                        | 17.6 % (30)                                       | 1.1 % (2)                                                                            | 6.6 %                                                            |
| Korinek et al. [69]        | 2005                | French    | May 1997–2001           | 4578                       | 6.6 % (303)                                       | 1.7 % (77)                                                                           | 25.4 %                                                           |
| McClelland and Hall. [70]  | 2007                | USA       | Feb 1991–2005           | 1587                       | 0.8 % (14)                                        | 0                                                                                    | 0                                                                |
| Dashti et al. [71]         | 2008                | USA       | 1997–2007               | 16,540                     | 0.3 % (50)                                        | 0.1 % (22)                                                                           | 44 %                                                             |
| Symos et al. [72]          | 2009                | Greece    | 2006–2008               | 155                        | 14.8 % (33)                                       | 2.5 % (4)                                                                            | 12.1 %                                                           |
| Abu Hamdeh et al. [10]     | 2014                | Sweden    | 2010                    | 448                        | 4.5 % at 3 months (20)<br>5.1 % at 12 months (23) | 3.1 % (14)                                                                           | 60.8 %                                                           |

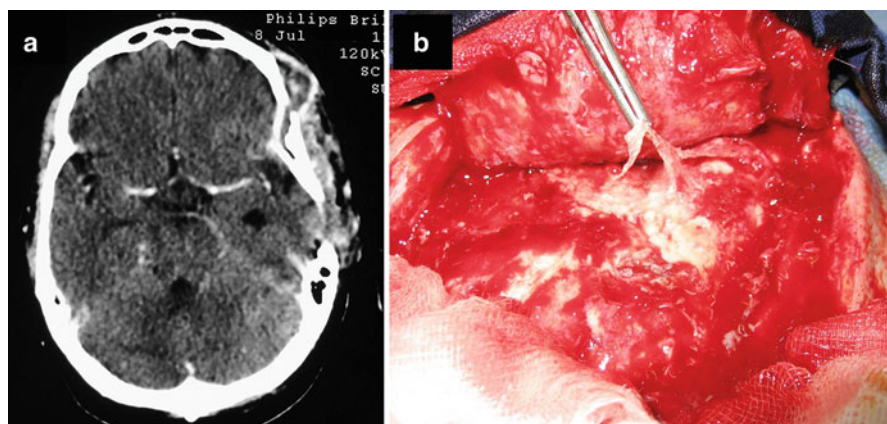
<sup>a</sup>Prophylactic antibiotics were not administered  
n/a no data available, CS cranial surgery

**Fig. 2.14** This patient was operated 11 years ago for an acute subdural hematoma with methylmethacrylate cranioplasty. Acrylic plate become exposed over time and infected with contiguous cranial osteomyelitis



leak, and/or an anterior ablative defect. Bone flaps that include part of a sinus wall (particularly the frontal sinus) are especially susceptible to developing bone flap infection [82]. Recently, published reports suggest that the incidence of wound-healing complications was higher in patients who underwent surgery after initiating bevacizumab (Avastin\*) treatment compared with those who underwent surgery alone [83, 84]. Gliadel wafers also significantly increased the risk of SSI after procedures to treat tumors [85].

The association between cranial osteomyelitis and other intracranial infections (parameningeal empyemas or brain abscess) is a well known situation [11, 14, 86, 87]. In our series of 13 patients with a postoperative osteomyelitis (4 patients had a primary craniotomy at an outside institution), bone flap osteomyelitis was seen alone (4 cases) or in combination with extradural empyema (6 cases), brain abscess (1 case) or both (2 cases). A wound purulent discharge was seen in 4 patients. Two patients had an osteomyelitis secondary to a polymethylmethacrylate cranioplasty. Interestingly, one of our patients presented with slowly progressive headache, fever, increasing tenderness, warmth and swelling at the initial surgical site 2 months after temporal craniotomy for arterio-venous fistula. Postoperative computed tomography scan and magnetic resonance imaging revealed an aspect of bone flap osteomyelitis with underlying epidural abscess. The infection was likely due to epidural cotton mistakenly left behind during operation [88] (Fig. 2.15). From 1996 to 2000, Larsson et al. treated 31 consecutive patients with postoperative bone flap infections at the Karolinska Hospital of Stockholm (Sweden). Among them, 6 had an acrylic flap [14]. Recently, Abou Hamdeh et al. identified 14 of 448 patients (3.1 %) as having bone flap infection. Postoperative osteomyelitis was seen alone in 6 cases and in conjunction with a brain abscess in 5 additional cases. Bone flap osteomyelitis and subdural empyema were seen together in 2 cases. One patient had bone flap osteomyelitis, brain abscess and meningitis. Osteomyelitis after cranial reconstruction was seen in three patients (synthetic bone substitute in one case and

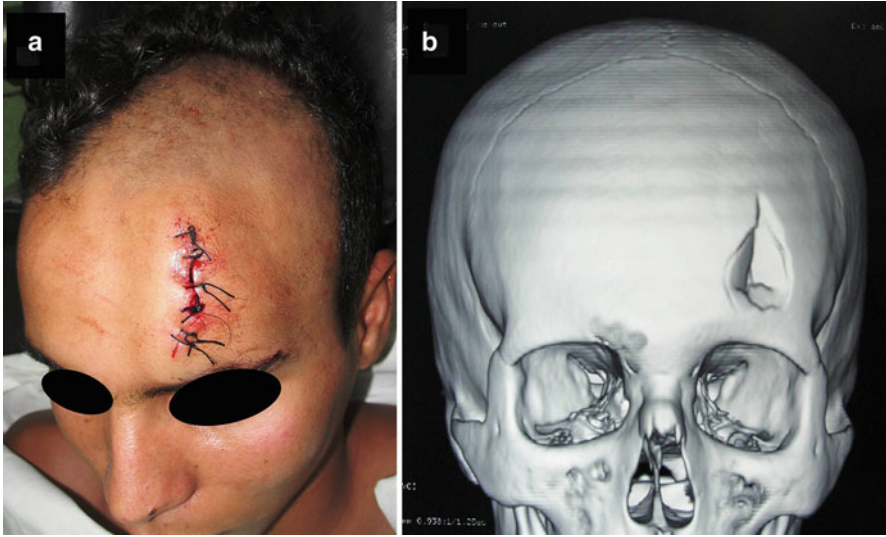


**Fig. 2.15** Postoperative computed tomography scan, axial section after contrast injection revealing left epidural abscess with frontal extracranial collection (a). Operative view after bone flap removal (b). The infection was likely due to epidural cotton left behind during surgical time

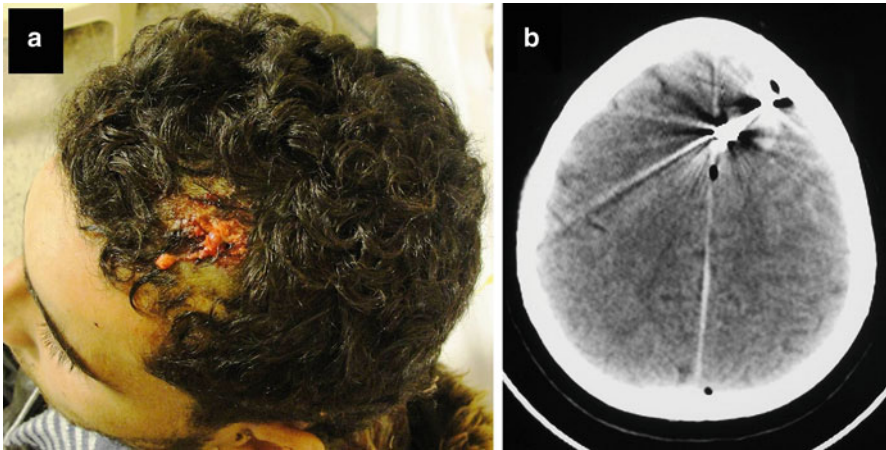
on cryo-preserved bone flap in two cases) [10]. In addition, Bhaskar et al. found 5 patients with cranial osteomyelitis among 179 autogenous bone cranioplasties (infection rate of 2.8 %) [89].

True skull base osteomyelitis has been described as occurring after surgical interventions at the site of infection [90, 91]. Ridder et al. described a series of 12 patients including middle ear surgery with mastoidectomy (58 %), operations on the external auditory canal (18 %), cochlear implant (8 %) and an otologic operation of unknown type (8 %). The mean time between the first surgery and diagnosis of skull base osteomyelitis was 44.2 months (ranged from 1 to 456 months, median 2 months) [92]. An interesting case of skull base osteomyelitis that presented 4–8 weeks after a maxillectomy for a plexiform ameloblastoma of the right posterior maxilla was previously reported [93].

Direct injuries are well recorded situations when they occur: in the newborn following infection of a cephalhematoma (particularly after vacuum extraction) [52, 94, 95], secondary to craniofacial injuries with or without fractures [22, 96–98] (Fig. 2.16), penetrating craniocerebral injury [99] (Fig. 2.17), after burns to the scalp or electrical injuries [100–102] (Fig. 2.18), following scalp wound/laceration [103–106] (Fig. 2.19) or traumatic scalp hematoma [107], in patients with sickle cell disease [108, 109], as a complication of halo traction [110–112] and secondary to hear transplantation [113]. War wounds tend to be more extensive and contaminated than civilian wounds [114]. The result from poorer wound healing leads to a propensity for infection. Unfortunately, minor trauma with small scalp wounds are usually ignored, and these may result in serious infections [115]. Many cases of skull osteomyelitis were reported secondary to minor scalp wounds such as human bite [15], scalp laceration [116], insect bite [59], acupuncture [117], needle insertion [118] or self-inflicted dermatitis [119]. Onoue et al. presented an interesting case of cranial osteomyelitis after head injury with exaggeration and remission for



**Fig. 2.16** Post-traumatic frontal wound (a) with underlying depressed bone fracture on 3-dimensional CT-scan reconstruction (reformatted volume-rendered) (b)



**Fig. 2.17** Penetrating craniocerebral injury (open head injury). Note extracranial extrusion of brain matter on the left frontal area (a). Axial CT-scan showing multiple intracranial metallic foreign bodies (b)

30 years. The mechanism by which osteomyelitis continued over a long course of time was suspected to be the formation of a focus of recurrent inflammation due to the initial head injury [120]. Also, many authors suggest that head injury contributes to the apparent increase in prevalence of Pott's puffy tumor [117, 121–123].

**Fig. 2.18** This patient was electrocuted with high tension wire. Picture showing large scalp defect during dressing change. Note frontal and parietal cranial bones exposed with burn eschar



**Fig. 2.19** Direct frontal injury with lacerated wound in the forehead



*Hematogenous Spread Secondary to Bacteremia or Fungemia*

There have been occasional case reports of hematogenous osteomyelitis of the skull [17, 23, 25, 46, 124–126], several large series of osteomyelitis reported by otolaryngologists and neurosurgeons have not mentioned such cases [3, 6, 17, 50]. This most often occurs when the patient has an infection elsewhere in the body that spreads through the blood to the skull bone. Camargo et al. reported a case of *Aspergillus spp.* osteomyelitis of the lumbar spine complicated with right orbital apex syndrome due to orbital and sphenoid wing involvement. They suspected a potential role of the Batson’s vascular plexus in disease propagation [23]. Until now, few other anecdotal cases were reported especially with salmonella, secondary syphilis and coccidioidomycosis agents [127–129]. However, the cause of infection may remain cryptogenic in spite of all the investigations [129]. To explain this phenomenon, Prasad et al. suggest that in the first stage an unrecognized infection, or a small focus of infection, heals. By the time, the secondary infection develops and spreads [130]. Interestingly, many cases manifested with septicemia and meningitis especially in children with cephalhematoma were described [95, 131]. In 1991, Yocum and Seligson reported a case of septic arthritis of the knee and osteomyelitis of the skull vault following arthroscopy. The patient had a concomitant cavitary lung lesion caused by Blastomycosis [24]. Recently, Hayashi et al. reported a patient with recurrent meningitis complicated by osteomyelitis of the clivus. They supposed that the septicemia resulted in a secondary septic pulmonary embolism and meningitis and that inflammation had extended to the base of the skull due to the meningitis [25]. In contrast to other hematogenous osteomyelitis of the skeleton, multifocal osteomyelitis is rare in the head.

Comorbidities

In addition to these three principal key sources, some comorbidities or patient-related risk factors should be taken into consideration [3, 49, 59, 70] (Table 2.3). These include vulnerability due to metabolic states (as diabetes mellitus),

**Table 2.3** Comorbidities and underlying conditions in skull osteomyelitis

| Systemic comorbidity           | Local detriment          |
|--------------------------------|--------------------------|
| Obesity and malnutrition       | Extensive scarring       |
| Diabetes mellitus              | Radiation fibrosis       |
| Renal or hepatic failure       | Small vessel disease     |
| Chronic hypoxia                | Heavy contamination      |
| Compromised immune function    | Presence of foreign body |
| Malignancy                     | Bone necrosis            |
| Long period of hospitalization |                          |
| Extremes of age                |                          |
| Tobacco abuse                  |                          |

unbalanced nutritional states (obesity and malnutrition), smoking and tobacco use, specific exposure risks (prolonged preoperative stay, colonization with *Staphylococcus aureus*), compromised immune function (defective immune system, human immunodeficiency virus infection, use of immunosuppressive medications or chronic systemic corticosteroids, irradiation of the head), malignancy, hemodialysis and age (especially newborn and advanced age).

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