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Empyema of Odontogenic Origin: A Case Report

Abstract: A subdural empyema of odontogenic origin is an uncommon, but significant sequela of dental disease with associated morbidity. This article discusses the case of a 15-year-old boy who presented with a subdural empyema of odontogenic origin. The case highlights the multidisciplinary care required to manage such cases, including the involvement of the dental team. It discusses the complexities in its diagnosis and management as well as the associated morbidity and mortality.

CPD/Clinical Relevance: Subdural empyema of odontogenic origin has implications for the dental team in terms of prevention and management of dental disease.

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An intracranial empyema is a pus collection in the subdural or extradural space. This results from the spread of infection from one anatomical space to another with otitis, frontal sinusitis or a post-surgical/post-traumatic direct inoculation providing the source.¹ The clinical presentation of a subdural empyema can vary, but timely management is required to ensure the best outcome for the patient.

Case report

A 15-year-old boy attended accident and emergency (A&E) reporting a 3-day history of fever, myalgia, vomiting, photophobia, and headaches. He had an otherwise unremarkable medical history. The initial medical diagnosis was of a viral upper respiratory tract infection for which he was

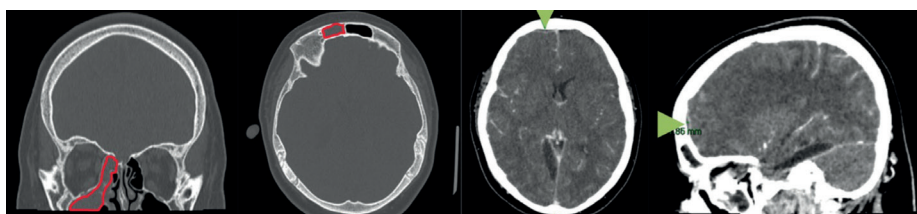


Figure 1. CT scan illustrating the opacification of the right maxillary and frontal sinuses (marked in red) and the subdural empyema and subdural effusion (green arrows).

prescribed a course of oral antibiotics and advised to arrange a COVID-19 swab.

He re-presented to A&E 2 days later with a worsening fever and myalgia, urinary incontinence, and reduced mobility. On examination, he had an evolving neurological deficit with left leg weakness, a convergent squint, and a visual field

defect –all suggestive of raised intracranial pressure. He also demonstrated pyrexia and an increased respiratory rate. He was admitted for further investigations and the empirical treatment of sepsis, with intravenous antibiotics, antivirals and dexamethasone, was initiated.

The blood results showed a raised white cell count (WCC: $23 \times 10^9/l$; normal range $4.5\text{--}11.0 \times 10^9/l$) and C-reactive protein (CRP: 198mg/l ; below 10mg/l is normal). An urgent CT scan revealed a suspected intracranial infection with subdural effusion and empyema, as well as fluid opacification of the right maxillary, ethmoid and frontal sinuses (Figure 1). The radiologist suggested

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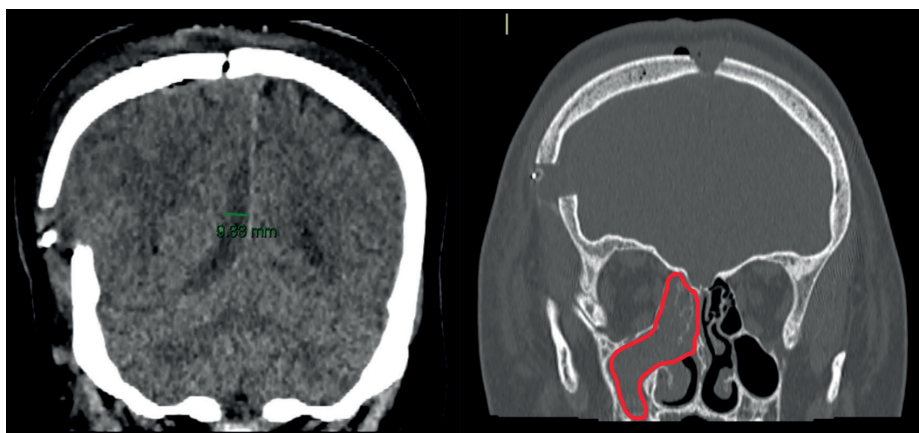


Figure 2. CT Scan prior to the 3rd operation showing the 'floating' cranial bone flap and herniation of the brain. Continued frontal and maxillary sinus infection illustrated in red.

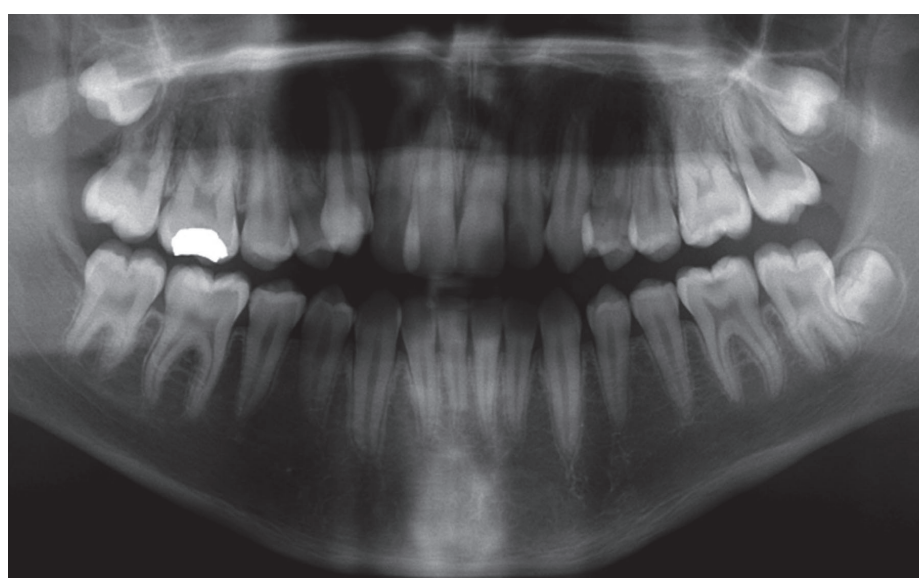


Figure 3. Panoramic radiograph. Note the peri-apical pathology associated with the UR6.

that the maxillary sinus may be the source of infection.

The patient was taken to theatre within 24 hours of admission for a right-sided craniotomy and washout of the subdural empyema by the neurosurgery team. Additionally, functional endoscopic sinus surgery (FESS) and a washout of the maxillary and ethmoidal sinuses were carried out by the ear, nose and throat surgical team. During surgery, foul-smelling pus was liberated on opening the dura of the brain and from the right maxillary antrum. The patient returned to the paediatric intensive care unit (PICU) where he began to stabilize and regain anti-gravity arm control, indicative of some neurological recovery.

The patient returned to theatre 3 days after the initial surgery owing to a marked loss of arm power. An MRI scan

gave evidence of a further collection of pus (Figure 2). The craniotomy site was re-opened and washed out revealing no gross collection of pus, but showed brain tissue herniating from the posterior dural incision. The ENT team carried out additional FESS to open the maxillary sinus further, identifying a recollection of pus. The patient returned to the paediatric neurosurgery ward.

Three days following the second surgery, the patient experienced an increased frequency of focal seizures and a reduction in his Glasgow Coma Scale (GCS) to 13 (normal value: 15). This was attributed to raised intracranial pressure (ICP) and therefore the patient returned to theatre for a third procedure. To reduce the ICP, the cranial bone flap was removed, sectioned into four pieces, and stored in the abdominal wall. The patient

continued to be given anticonvulsants and antibiotics in PICU, and subsequently stepped down to the neurosurgery ward for ongoing management.

The team were still uncertain of the source of infection. When looking through the patient's historical records they revealed that he had been referred to the hospital dental service in 2014 regarding grossly carious primary teeth; however, the patient was not brought for the appointment. At that point, the surgical team sought an opinion from the paediatric dentistry team.

An initial dental assessment was carried out on the ward. The patient was still significantly unwell, and obtaining an accurate history was challenging. The patient was fluent in English, but his parents' understanding was limited. Clinical examination revealed a heavily restored UR6 and some caries in permanent molars, but no obvious signs of dental infection. A panoramic radiograph was requested to be taken as soon as the patient had regained sufficient core control to independently support himself.

At a subsequent review, the patient's condition had improved, and his family were able to give a more comprehensive history with the help of translation services. He had attended an emergency dental appointment with his mother 2 weeks prior to presenting at A&E, following an episode of pain and palatal swelling in the upper right quadrant. This was managed by an emergency dentist with local measures only.

Further oral examination confirmed there was no apparent extra- or intra-oral swelling. Of note was an enlargement of the right maxillary tuberosity and a large occlusal amalgam restoration with buccal caries in the upper right first permanent molar (UR6). There was no tenderness to percussion or pain on biting. The panoramic radiograph revealed peri-apical radiolucencies associated with the palatal and distobuccal roots of the carious UR6 (Figures 3 and 4). The culture reports from the brain pus samples had isolated *Streptococcus anginosus* and *Prevotella nigrescens*.

Multidisciplinary input was sought from infectious diseases, neurosurgery and oral and maxillofacial radiology, and considering the microbiology and the clinical and dental history, it was felt that the foci of infection was the UR6.

The patient became pyrexial again with an elevated CRP. This was later attributed to a hospital-acquired rhinovirus. He returned

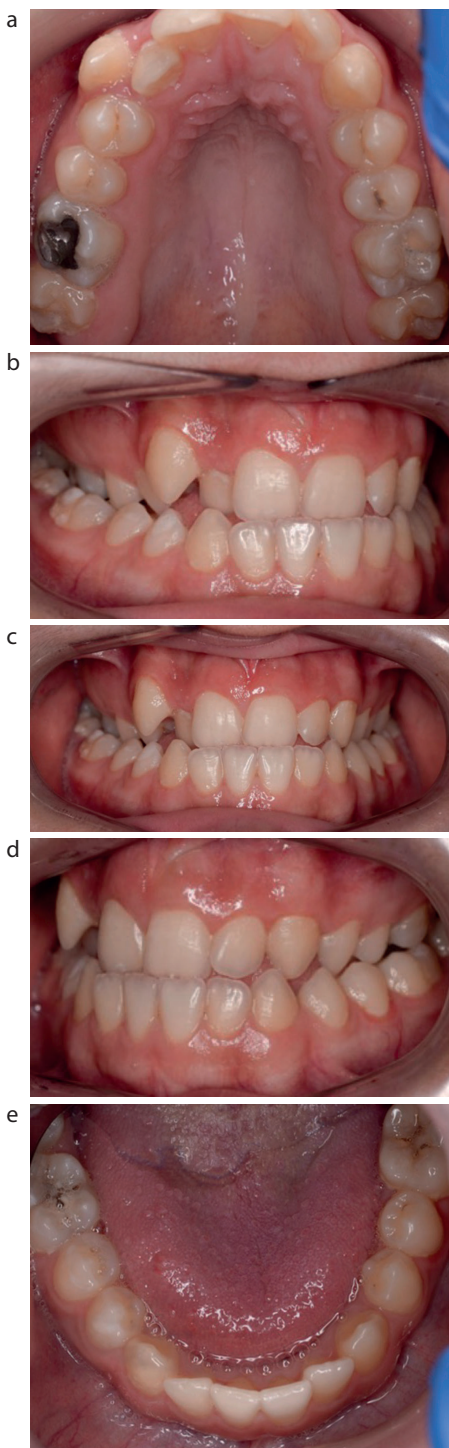


Figure 4. (a-e) Intra-oral photographs showing the UR6 in question.

to theatre for the extraction of the carious UR6 and LR6.

Subsequent MRI scans 1 and 3 weeks following the dental extractions showed a reduction in the thickening of the right sinus lining. At 54 days post-admission, and with the ability to now stand independently, the bone flap was replaced,

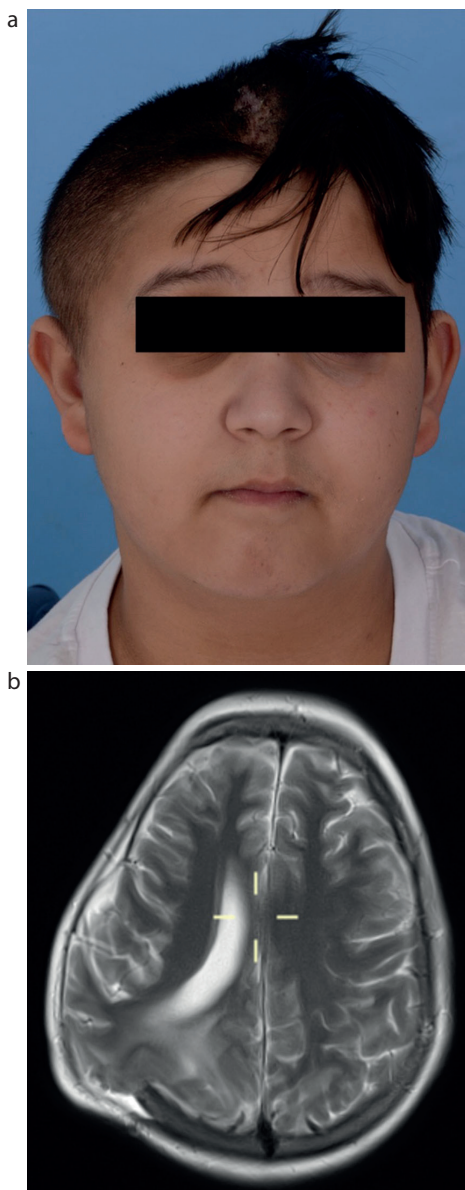


Figure 5. (a) Photo and **(b)** MRI scan showing the herniation of the brain through the skull once the bone flap was placed in the abdomen.

and the remaining poor prognosis teeth were extracted under general anaesthetic. The patient was deemed fit for discharge the following week. His social circumstances remained challenging – his family were living in homeless accommodation and the involvement of social services, occupational health and physiotherapy remained key in facilitating his full recovery (Figure 5).

Discussion

Sinusitis of odontogenic origin

Odontogenic sinusitis (ODS) is a well-recognized condition and accounts for approximately 25–40% of chronic

maxillary sinusitis.^{2–4} The maxillary sinus is susceptible to invasion by pathogenic bacteria through the nasal ostium and oral cavity. ODS can occur from several dental causes, such as peri-apical infection, and incorrectly placed implant fixtures when they disrupt the membranous lining of the maxillary antrum.⁵ Most often, concomitant management of the dental origin and the associated sinusitis will ensure complete resolution of the infection, and may prevent recurrences and complications. A combination of a medical and surgical approach is generally required for the treatment of odontogenic sinusitis.⁶

ODS generally has symptoms consistent with rhinosinusitis, but is more likely to have purulence and a foul smell. It can often be asymptomatic and dental pain is infrequently encountered. A history of prior dental procedures may increase the likelihood of a patient's sinusitis being odontogenic in origin.⁴

The diagnosis of ODS is based on thorough dental and medical examination, correlation of the patient's symptoms and medical/dental history, sensibility testing and appropriate radiological imaging. Multidisciplinary collaboration with otolaryngologists may be beneficial to ensure correct diagnosis, and therefore, correct management.

From sinusitis to empyema

In the case described, peri-apical periodontitis led to odontogenic sinusitis that spread to pansinusitis. From the frontal sinus, the infection was transmitted into the subdural space causing an empyema.

Intracranial extension of acute or chronic sinusitis is a known complication, and has a low reported incidence of 1 per 100,000 per year¹ and 3.7–11% in hospitalized patients.⁷ Commonly, intracranial complications are seen in the first two decades of life because younger patients are more prone to sinus disease,⁸ and male subjects are 1.5–3 times more likely to be affected.¹

The characteristic signs of a subdural empyema can be described by dividing them into four groups: those due to pansinusitis (mild headache, pyrexia); raised ICP (worsening headache, intractable vomiting, deteriorating consciousness); meningeal irritation (neck stiffness); and focal neurological deficits.⁹

Skelton *et al* described a case series of 10 children who were managed with sinus-induced empyema. Most children

had symptoms for 1 or 2 weeks prior to presentation, with fever, mild headache, and malaise, which were usually treated with oral antibiotics. Symptoms then progressed over the second week as signs of raised ICP developed – worsening headache, vomiting, seizures and lethargy. Neurological signs usually precipitate hospital admission and appropriate diagnosis.¹⁰ This clinical presentation is remarkably like the case report described.

The mortality rate for subdural empyema is around 4%, while the morbidity is higher with residual neurological deficits reaching up to 50%, hemiparesis in 15–35% and persistent seizures in 12–37.5%.¹¹

Microbiology

Both *Streptococcus anginosus* and *Prevotella nigrescens* were isolated in the brain pus swab. The *Streptococcus anginosus* (*milleri*) group comprises the normal microbiota of the gastrointestinal tract and oropharynx, and when pathogenic, is often associated with abscess formation and endocarditis. These micro-organisms are micro-aerophilic, catalase-negative, Gram-positive cocci, and are generally susceptible to beta-lactam antibiotics.¹² *Prevotella nigrescens* is a member of the *Prevotella* species found in the normal oral flora and plays a role in the pathogenesis of periodontal disease.¹³

S. anginosus is one of the most prevalent species in the microbiota of primary endodontic infections with necrotic pulp, with *P. nigrescens* also found in high proportions.¹⁴ *S. anginosus* has been reported as the most common organism of subdural empyema due to sinusitis.¹⁵ The microbiology results indicate a likely odontogenic cause, which reinforces the clinical presentation.

Implications for the dental team

Owing to the morbidity and mortality of subdural empyema, the dental team must ensure that odontogenic infection is managed appropriately, and in a timely manner. This is an unusual case, but it highlights the importance of oral health and its role in general wellbeing.

The delayed request for input from the paediatric dentistry team may have had an impact on the patient's recovery, therefore, education of our medical colleagues is imperative to ensure timely and appropriate management of such cases.

This case highlights the importance of having appropriate 'Was Not Brought' and child protection protocols in place to ensure

that children's missed appointments are followed up, and their treatment needs are met. If this patient's social circumstances were known at the time of referral, this may have allowed for appropriate support to be made available to assist the family in accessing dental services and preventing this disease escalation.

Conclusion

This case demonstrates the morbidity that is associated with a subdural empyema of odontogenic origin. This is very uncommon; however, dentists should be aware of varied clinical presentations of odontogenic sinusitis, and the importance of timely and appropriate prevention and management of dental disease. The added complications of language barriers, concerns around poor housing and social circumstances all contributed to the complexity of managing this case.

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Compliance with Ethical Standards

Conflict of Interest: The authors declare that they have no conflict of interest.

Informed Consent: Informed consent was obtained from all individual participants included in the article.

References

- Bartt RE. Cranial epidural abscess and subdural empyema. *Handb Clin Neurol* 2010; **96**: 75–89. [https://doi.org/10.1016/S0072-9752\(09\)96006-7](https://doi.org/10.1016/S0072-9752(09)96006-7)
- Albu S, Baciut M. Failures in endoscopic surgery of the maxillary sinus. *Otolaryngol Head Neck Surg* 2010; **142**: 196–201. <https://doi.org/10.1016/j.otohns.2009.10.038>
- Melén I, Lindahl L, Andréasson L, Rundcrantz H. Chronic maxillary sinusitis. Definition, diagnosis and relation to dental infections and nasal polyposis. *Acta Otolaryngol* 1986; **101**: 320–327. <https://doi.org/10.3109/00016488609132845>
- Craig JR, Poetker DM, Aksoy U *et al*. Diagnosing odontogenic sinusitis: an international multidisciplinary consensus statement. *Int Forum Allergy Rhinol* 2021; **11**: 1235–1248. <https://doi.org/10.1002/alr.22777>
- Kretzschmar DP, Kretzschmar JL. Rhinosinusitis: review from a dental perspective. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 2003; **96**: 128–35. [https://doi.org/10.1016/s1079-2104\(03\)00306-8](https://doi.org/10.1016/s1079-2104(03)00306-8)
- Brook I. Sinusitis of odontogenic origin. *Otolaryngol Head Neck Surg* 2006; **135**: 349–355. <https://doi.org/10.1016/j.otohns.2005.10.059>
- Dolan RW, Chowdhury K. Diagnosis and treatment of intracranial complications of paranasal sinus infections. *J Oral Maxillofac Surg* 1995; **53**: 1080–1087. [https://doi.org/10.1016/0278-2391\(95\)90128-0](https://doi.org/10.1016/0278-2391(95)90128-0)
- Roche M, Humphreys H, Smyth E *et al*. A twelve-year review of central nervous system bacterial abscesses; presentation and aetiology. *Clin Microbiol Infect* 2003; **9**: 803–809. <https://doi.org/10.1046/j.1469-0691.2003.00651.x>
- Kaufman DM, Miller MH, Steigbigel NH. Subdural empyema: analysis of 17 recent cases and review of the literature. *Medicine (Baltimore)* 1975; **54**: 485–498. <https://doi.org/10.1097/00005792-197511000-00003>
- Skelton R, Maixner W, Isaacs D. Sinusitis-induced subdural empyema. *Arch Dis Child* 1992; **67**: 1478–1480. <https://doi.org/10.1136/adc.67.12.1478>
- 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**: 969–976. <https://doi.org/10.1001/archotol.132.9.969>
- Petti CA, Stratton CW. 205: Streptococcus anginosus group. In: Bennett JE, Dolin R, Blaser MJ (eds) *Mandell, Douglas and Bennett's Principles and Practice of Infectious Diseases*. 8th edn. Saunders: 2015.
- Yakob M, Söder B, Meurman JH *et al*. *Prevotella nigrescens* and *Porphyromonas gingivalis* are associated with signs of carotid atherosclerosis in subjects with and without periodontitis. *J Periodontol Res* 2011; **46**: 749–755. <https://doi.org/10.1111/j.1600-0765.2011.01398.x>
- Sassone L, Fidel R, Figueiredo L *et al*. Evaluation of the microbiota of primary endodontic infections using checkerboard DNA-DNA hybridization. *Oral Microbiol Immunol* 2007; **22**: 390–397. <https://doi.org/10.1111/j.1399-302X.2007.00376.x>
- Waseem M, Khan S, Bomann S. Subdural empyema complicating sinusitis. *J Emerg Med* 2008; **35**: 277–281. <https://doi.org/10.1016/j.jemermed.2007.07.019>
- Mehra P, Murad H. Maxillary sinus disease of odontogenic origin. *Otolaryngol Clin North Am* 2004; **37**: 347–364. [https://doi.org/10.1016/S0030-6665\(03\)00171-3](https://doi.org/10.1016/S0030-6665(03)00171-3)
- Niehaus MT, Krape KN, Quinn SM, Kane BG. Frontal sinusitis complicated by a brain abscess and subdural empyema. *Radiol Case Rep* 2018; **13**: 456–459. <https://doi.org/10.1016/j.radcr.2018.02.003>