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Case Report

Sickle cell disease and jawbone osteomyelitis: case report and literature review

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Abstract – Introduction: Sickle cell disease (SCD) can manifest with vaso-occlusive crises (VOC) in various anatomical locations. In this case, a mandibular osteomyelitis developed following a VOC but was initially misdiagnosed due to a lack of specific clinical and radiological indicators. This case report is accompanied by a brief review that highlights potential warning signs shared among similar cases, with the aim of aiding early identification and diagnosis. **Observation:** A 22-year-old male with SCD of Congolese origin was initially referred to the dental service at Henri Mondor Hospital for facial cellulitis during his hospitalization for a hip vaso-occlusive crisis (VOC). Due to the absence of specific clinical and radiological findings, a dental origin was initially suspected. However, as the patient's condition deteriorated, a diagnosis of mandibular osteomyelitis was eventually established. In addition to antibiotic therapy, a sequestrectomy was performed under general anaesthesia. Delayed healing was observed. **Conclusion:** Diagnosis osteomyelitis of jawbone in SCD patients can be challenging, as clinical and radiological signs may not always be evident. Dental practitioners and oral surgeons should be vigilant when managing these patients, as complications like mandibular osteomyelitis may present similarly to dental conditions but require prompt investigation and intervention to prevent serious health consequences.

Introduction

Sickle cell disease (SCD) is one of the most common genetic disorders, predominantly affecting individuals of Afro-Caribbean descent. In France, it stands as the most prevalent genetic disorder, currently affecting approximately 26,000 individuals, with around 600 children born with SCD each year [1]. This disorder, inherited in an autosomal recessive manner, is characterised by the production of an abnormal form of hemoglobin known as hemoglobin S (HbS) [2]. In environments with low oxygen levels, HbS molecules undergo polymerization into fibres, causing deformation of red blood cells, rendering them rigid and adhesive. This aggregation leads to hemolysis, resulting in hemolytic anemia [3]. Additionally, it can cause vascular occlusion in small blood vessels, reducing oxygen supply to affected organs and triggering painful vaso-occlusive crises (VOC).

These crises frequently manifest in bone marrow, likely due to marrow hypercellularity, which impedes blood flow, leading to regional hypoxia and subsequent tissue infarction. The affected bone marrow becomes more susceptible to infection, which can lead to osteomyelitis. Although osteomyelitis can affect any hematopoietically active bone marrow, the long bones of the extremities, such as the humerus, tibia, and femur, are most commonly affected with SCD patients facing a lifetime risk of 29–31% [4]. However, jaw osteomyelitis is rare among SCD patients, with an incidence rate of 3–5% [5].

This article presents a case of mandibular osteomyelitis in a patient with SCD, initially misdiagnosed due to a lack of specific clinical and radiological evidence. The case report is accompanied by a brief literature review aiming to identify potential warning signs shared among various cases reported.

Observation

A 22-year-old male of Congolese origin with G6PD-deficient sickle cell disease (SS type) was admitted to Henri Mondor Hospital (Créteil, France) with acute left mandibular cellulitis, two weeks after treatment for a vaso-occlusive hip crisis that required exchange transfusions.

His medical history included frequent hospitalizations for vaso-occlusive crises, episodes of splenic sequestration leading to a splenectomy in 2013, and a cholecystectomy in 2010. His regular treatment included folic acid and hydroxyurea.

In the emergency department, treatment with amoxicillin/clavulanic acid (6g/750mg per day) was initiated following a positive bacteriological sample for *Staphylococcus aureus*.

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The patient was then referred to the oral medicine department at Henri Mondor Hospital.

Extraoral examination revealed diffuse, soft swelling in left submandibular and buccal spaces with concomitant numbness along the distribution of the inferior alveolar nerve (IAN). Pericoronitis of a partially erupted and decayed lower third molar was identified as the cause of the cellulitis. This hypothesis was supported by findings of lesions at the apex of tooth 38 on the panoramic radiograph and cervico-facial scanner performed in the emergency department (Fig. 1). Antibiotic therapy was changed to amoxicillin 3g/day with Metronidazole 1.5g/day, leading to an improvement in infectious symptoms and a reduction in swelling at the 7-day check-up. Given the initial symptoms (cellulitis, sensitivity disturbance), a CBCT could have been indicated; however, the avulsion of the 38 was carried out with radiological support from the panoramic radiograph and cervico-facial scanner alone.

During extraction of tooth 38 under local anaesthetic, unexpected mobility of tooth 37 was detected, unobserved during the initial clinical and radiological examination. This necessitated its simultaneous extraction. Curettage of the alveolar bone revealed avascular necrotic tissue with loss of gingival attachment. An exploratory flap and curettage of the necrotic bone were performed at the surgical site. A specimen sent for histopathological analysis confirmed osteonecrosis with *Staphylococcus aureus* present. Antibiotic therapy was therefore continued to cover the mucosal healing period.

At the 7-day follow-up, a significant delay in healing was noted, with bone exposure and paleness of the covering tissues. Despite the absence of radiological evidence, left mandibular osteomyelitis was suspected, potentially related to vascular occlusion following the recent vaso-occlusive hip crisis.

One month follow-up revealed delayed healing with necrotic bone exposure from the retro-molar trigone to the lingual cortex of tooth 36, as well as significant mobility of teeth 36 and 35 (Fig. 2). Cone beam computed tomography (CBCT) revealed an ill-defined radiolucency extending horizontally from the extraction site of tooth 38 to the mesial root of tooth 36 and vertically to the upper wall of the mandibular canal, with loss of bone trabeculation (Fig. 3). Due to the risk of bone fracture, a sequestrectomy and extraction of non-conservable teeth (36/27) was performed, in collaboration with the maxillofacial surgery department, under general anaesthesia with exchange transfusion prior to the surgery.

Delayed mucosal healing was observed at both 15 day and 1.5 months post-operatively. At the 6 months follow-up, complete mucosal healing was observed (Fig. 2) but CBCT showed the persistence of a significant vertical bone defect after healing (Fig. 4). The restorative treatment of teeth 17, 16, 47, 46, 25 and the endondontic treatment of tooth 35 were carried out under nitrous oxide inhalation and local anaesthesia without vasoconstrictor to minimise the risk of triggering a new local vaso-occlusive crisis.

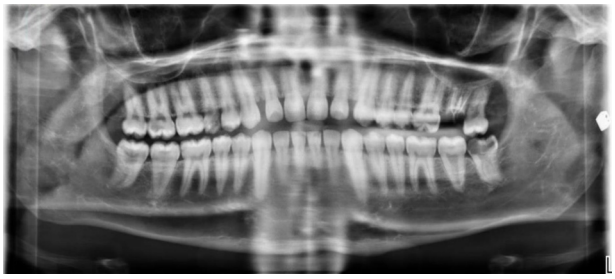

Fig. 1. Initial orthopantomogram showing no bone changes in the left molar mandibular region. However, tooth 38 showed deep decay and periapical radiolucency.

Discussion

The literature has documented only 43 cases of jawbone involvement in SCD patients since 1971 (Tab. I). Most cases involved male patients, with a sex ratio of 36:7, indicating a higher male prevalence (84%). These These infections commonly affect young individuals, as in the present case (aged 22), with most patients occurring in the third decade (ages range between 6-40 years). These patients were primarily affected by homozygous S/S sickle cell anemia, which is the most common and severe form of SCD. Moreover, most cases were reported after a recent vaso-occlusive crisis (within the subsequent month) in patients who regularly suffer from VOC.

Chekroun *et al.* [25] reported that during a VOC episode, the entanglement of sickled cells within the inferior alveolar artery can lead to blockage, infarction, and aseptic pulpal necrosis. These ischemic regions may subsequently become necrotic and infected, either via hematogenous spread or local invasion along the periodontal ligaments. Among documented cases, the mandible is the most frequently affected part of the jaw, particularly in the molar and angle region. Olaitan *et al.* [5] suggested that this may be because the mandibular molar region is supplied solely by the ipsilateral inferior alveolar artery and periosteum. The anterior region is less susceptible to ischemia during a VOC episode involving the inferior alveolar artery, as it receives additional blood supply from contralateral vessels. Only three cases of maxillary involving the molar region have been reported in the literature because of its richer vascularization and its ability to easily supply occluded vessels [13,15–22].

Shroyer *et al.* [14] presented two separate hypotheses to elucidate the pathophysiology of osteomyelitis in sickle cell patients. The first hypothesis posits that a sickling crisis might induce bony infarctions, subsequently leading to bacterial colonization via hematogenous spread or local dissemination from the periodontal region. The second hypothesis suggests that a systemic infection could trigger a VOC, consequently causing bony infarction.

Initial symptoms may include facial pain and swelling, pus discharge, necrosis, periodontal issues such as tooth mobility, or numbness in the perioral region (predominantly situated in the lower lip in instances of inferior alveolar nerve

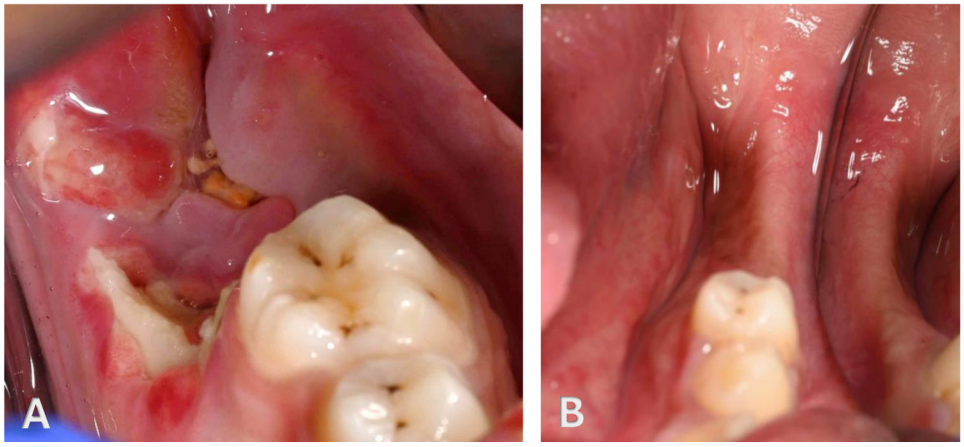

Fig. 2. Intraoral photographs of affected site A) Pre-sequestrectomy (tooth 36) B) 6 months post-op.

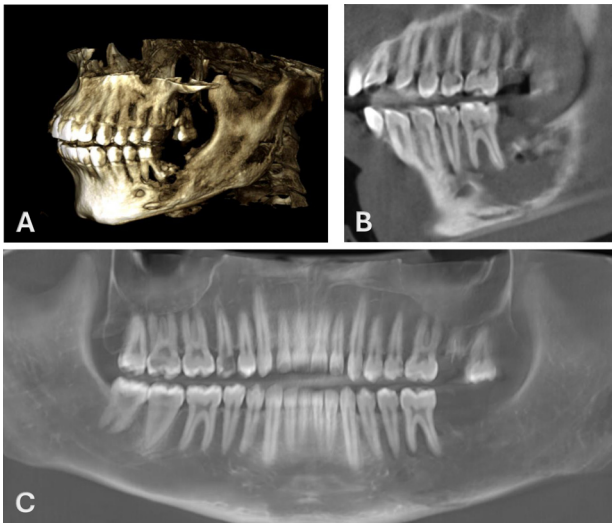

Fig. 3. Cone Beam Computed Tomography (CBCT) post-extraction (38, 37) and minimal curettage of necrotic alveolar bone at 1 month post-operative. A) 3D reconstruction of mandibular defect B) Sagittal section showing radiolucent necrotic bone, loss of trabecular structure distal to tooth 36C) Panoramic reconstruction showing "PDL thickening (35, 36), evidence of mobility, as well as the mottled, poorly defined necrotic zone distal to these teeth.

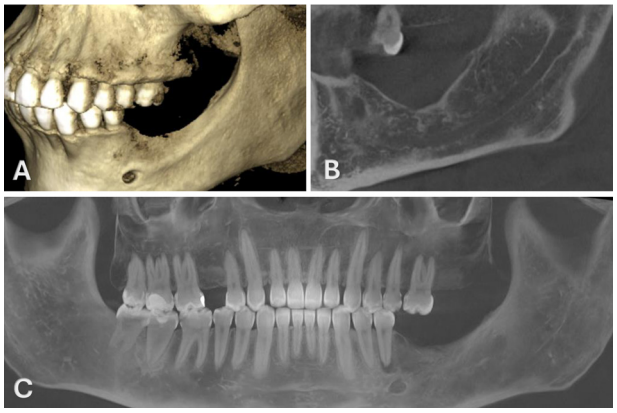

Fig. 4. CBCT at 6 month post-operative. A) 3D reconstruction showing the healed bone defect B) Sagittal section showing satisfactory reossification, with a residual vertical defect C) Panoramic reconstruction showing healing in sector 3, extraction sites 15, 27, 28, 36. Endo (35), restorations (25, 46) were subsequently carried out.

involvement) (Tab. I). In the current case study, the patient initially experienced paresthesia of the lower lip and a dental infection in the molar region, associated with pericoronitis of the decayed lower third molar. Olaitan *et al.* [5] and Shroyer *et al.* [14] have also documented pericoronitis as a common cause of osteomyelitis among SCA patients with mandibular osteomyelitis.

The challenge in our case was the absence of radiological signs suggestive of bone involvement. Although numerous authors describe recognizable radiolucent or radiopaque lesions in affected areas, the initial CT scan and panoramic radiograph showed no abnormalities, except for minor lesions at the apices of tooth 38. Kavadia-Tsatala *et al.* [18] described radiopaque lesions in regions of vaso-occlusive events and infarctions. The lesions are characterised by decalcification surrounded by reactive sclerosis, followed by the formation of sclerotic bone that may be separated from the cortex by a thin radiolucent area, resulting in a bone-within-bone appearance. Conversely, osteomyelitis lesions may present as periosteal thickening, lytic lesions, osteoporosis, loss of trabecular architecture, sequestration, or new bone formation. Some authors as Shroyer *et al.* [14] or Watanabe *et al.* [19] have recommended the use of advanced radiological techniques, such as MRI, positron emission tomography or technetium Tc 99m scintigraphy to identify necrosis/ bone infract in cases of suspicion.

Regarding the pathogens involved, Al-Ismaili *et al.* [22] indicated that Salmonella is frequently responsible for long bone osteomyelitis among SCD patients, while *Staphylococcus aureus* is predominantly associated with jaw osteomyelitis, as seen in the present case. Other germs, such as *Streptococcus spp.*, *Pseudomonas spp.*, *Actinomyces spp.*, and various mixed species, have also been identified as potential causative agents [5,11–13,14,16,22].

Table I. Literature review of jawbone osteomyelitis in patient with sickle cell disease.

Author and year of case report	Number of cases	Age/gender	Sickle cell disease type/ Recent VOC of other localization	Affected jaw	Initial symptoms	Radiographic manifestation	Pathogen involved	Treatment
Ryan *et al.* (1971) [6]	1	36/M	N/A N/A	Mandible	Abscess with mandibular third molar.	Loss of supporting alveolar bone	Normal flora plus Staphylococcus aureus	Antibiotherapy, Drainage, sequestrectomy
Walker *et al.* (1973) [7]	1	21/M	Homozygous SS On admission, pain in the elbow region, lower back and hips (repeated admission for treatment of sickle cell crises)	Mandible	Tooth-ache, Tooth infection, Jaw pain, unilateral facial swelling, Trismus	No apparent change of inferior border or angle of mandible, osteoporosis of left mandible, Periodontal disease	N/A	Antibiotherapy, Drainage, Tooth extraction, Sequestrectomy
Girasole *et al.* (1977) [8]	3	22/M 34/F 33/M		Mandible Mandible Mandible			Staphylococcus aureus Streptococcus viridans Pseudomonas spp.	Sequestrectomy and curetage Debridement and corticotomy Sequestrectomy and curetage
Daramola *et al.* (1982) [9]	1	28/M	N/A N/A	Mandible	Painful facial swelling, purulent periodontal pockets, trismus	Patchy areas of radiolucency and radiopacity suggesting bone destruction and sequestrum formation, pathological fracture	Salmonella spp.	Antibiotherapy, sequestrectomy with loose teeth removal, bone cavity was saucerised and the wound packed open with Eusol on ribbon gauze
Hammersley (1984) [10]	1	29/W	Homozygous SS During an hospitalisation for a severe limbs crisis (≈ 2 VOC/year)	Mandible	Mandibular pain, Mental nerve anaesthesia	Alveolar bone only showed classic osteoporosis caused by extramedullary haemopoiesis as a result of severe haemolytic anaemia	N/A	Antibiotherapy, Pain relief in 8 days, Partial resolution of the anesthesia in 8 months (residual paraesthesia)
Grodecki *et al.* (1985) [11]	1	19/W	N/A N/A	Mandible	Bilateral buccal and submandibular facial swelling, Trismus, lower lip hypoaesthesia, Periodontal infection of the 1st molar	Diffuse moth-eaten radiolucent areas in the area of the molars, and a bilateral proliferative periostitis on the buccal surfaces of the involved areas increasing the mandibular breadth.	Mixed oral flora / Salmonella tiphy	Drainage, Antibiotherapy, Sequestrectomy with teeth extraction under general anaesthesia (with preoperative exchange transfusion)
Iwu (1989) [12]	3							

Table I. (continued).

Author and year of case report	Number of cases	Age/gender	Sickle cell disease type/ Recent VOC of other localization	Affected jaw	Initial symptoms	Radiographic manifestation	Pathogen involved	Treatment
		16/W 20/M 18/W	Homozygous SS (repeated admission for treatment of sickle cell crises) Homozygous SS Homozygous SS	Mandible Mandible Mandible	Facial pain and swelling, tooth mobility with whitish alveolar bone exposed	N/A Buccal expansion of the right ascending ramus and bilateral radiolucency of the body of the mandible. N/A	Negative N/A Negative	Antibiotherapy, teeth extraction with sequestrectomy of necrotic bone
Patton *et al.* (1990) [13]	1	34/M	N/A 2 days after the onset of a sickle cell crisis	Mandible	Jaw pain, Facial swelling, Paraesthesia of inferior alveolar and mental nerve, teeth mobility and periodontal infection	Mandible lytic lesion in the molar area, Coarse trabeculation and osteoporotic bone character	Normal flora	Antibiotherapy, teeth extraction with debridement of necrotic bone
Shroyer *et al.* (1991) [14]	1	22/M	Homozygous SS sickle cell crisis 7 days before his admission	Mandible	Facial pain and swelling, trismus, purulent pericoronitis of 3rd molar	N/A	Eikenella corrodens, Bacteroides melaninogenicus, Peptostreptococcus spp., mixed Streptococcus spp. and Staphylococcus spp.	Antibiotherapy, drainage, teeth extraction,resection of devitalized bone and muscles, hemimandibulectomy in 2nd intention (infected relapse)
Bishop *et al.* (1995) [15]	1	41/M	N/A During a recent stay in hospital for a sickle cell crisis (SCC) / Patient was not aware of any oral problems prior to the SCC	Mandible	Jaw pain, unilateral lower lip anesthesia, teeth mobility and periodontal infection, pericoronitis of 3rd molar	Well-defined multi-locular radiolucency in the molar area of the mandible	N/A	Antibiotherapy, Teeth extraction, multiple root canal treatments
Podlesh *et al.* (1996) [16]	1	21/M	N/A Pain and fever consistent with sickle cell crisis (multiple admissions for painful crisis)	Mandible	Jaw pain, profound anesthesia of the mental nerve	Radiodensity associated with the apecies of the left mandibular first molar	Negative	Antibiotherapy, spontaneous improvement, no dental treatment because no symptoms
Olaitan *et al.* (1997) [5]	16	12-30 13 M 3 F	11 Homozygous SS, 5 Heterozygous SC N/A	Mandible	Pericoronitis with impacted molar either bilaterally or unilaterally (n=12), carbuncle on the skin (n=1), uncertain aetiology (n=3).	N/A	Mixed (n = 5), Staphylococcus aureus (n = 4), Microaerophyllic streptococcus (n=1), Non-haemolytic streptococcus (n=1), Klebsiella species (n=1). none (n = 4)	Antibiotherapy, Sequestrectomy alone or in combination with extraction of wisdom teeth (n=12), Medical treatment only (complete healing in n=1/4)

Table I. (continued).

Author and year of case report	Number of cases	Age/gender	Sickle cell disease type/ Recent VOC of other localization	Affected jaw	Initial symptoms	Radiographic manifestation	Pathogen involved	Treatment
Borle *et al.* (2001) [17]	1	25/W	N/A N/A	Maxilla	Facial pain and swelling, congestion of the right maxillary gingiva, discharge of pus, and exposure of the bone from first premolar to third molar region	Large sequestrum of bone extending from right maxillary first premolar to the maxillary tuberosity	Pseudomonas aerugenosa	Antibiotherapy, Sequestrectomy
Kavadia-Tsatala *et al.* (2004) [18]	6	22-34 3M 3F	N/A N/A	Mandible	Pain in maxillofacial region, no evidence of dental pathology or other infection	Intraosseous, radiopaque, quite homogeneous, adequately well-defined, multilobular lesions	N/A	N/A
Watanabe *et al.* (2013) [19]	4	6-22 3M 1F	3 Homozygous SS, 1 Heterozygous SC N/A	Mandible	Jaw and facial pain, facial swelling	N/A	N/A	N/A
DeBlieux *et al.* (2014) [20]	1	18/F	N/A During hospitalisation for a sickle cell crisis of her left leg. (monthly admissions to the hospital for sickle cell crisis)	Maxilla, Mandible	Facial swelling, trismus, paresthesia of the upper lip, dental carie, impacted 3rd molar	Numerous nonenhancing subperiosteal collections over the right maxilla, right body of the mandible, and left subcondyle	Negative	Intraoral decompression of the infarcts
Araújo *et al.* (2015) [21]	1	28/M	Homozygous SS Hospitalization 1 month-prior for chest and femoral pain	Mandible	Pain, bilateral teeth mobility, gingiva recession of the lower molars	2 radiolucent symmetrical periapical lesions evolving both the first and the second lower molars, bilaterally	Streptococcus viridans	Antibiotherapy, Teeth extractions with sequestrectomy
Al-Ismaili *et al.* (2016) [22]	3	23/M 16/M 25/M	N/A 3 weeks prior the patient had been admitted due to a sickle cell crisis VOC episode two weeks prior to the start of his symptoms. following his admission to a local hospital due to a severe VOC. (history of frequent hospital admissions)	Mandible Mandible Maxilla	Suppurative alveolitis of molar site, lower lip hypoaesthesia Pericoronitis	Area of radiolucency related to molar root	Actinomyces spp., Klebsiella pneumoniae and Moraxella catarrhalis Streptococcus anginosus Actinomyces spp.	Antibiotherapy, surgical curettage/ sequestrectomy of the mandible with removal of the involved teeth, bismuth iodine paraffin paste (BIPP) packing of surgical site

Table I. (continued).

Author and year of case report	Number of cases	Age/gender	Sickle cell disease type/ Recent VOC of other localization	Affected jaw	Initial symptoms	Radiographic manifestation	Pathogen involved	Treatment
Mahendran *et al.* (2020) [23]	2	34/M 38/F	N/A N/A Recent hospital admission for sickle crisis Three days prior, she was admitted for management of an acute painful vaso-occlusive crisis.	Mandible Mandible	Bilateral facial swelling and altered sensation of the lower lip, trismus	Irregular radiolucency in the mandible, buccal plate cortical pitting and alveolar bone destruction	N/A	Antibiotherapy, Oral bisphosphonates (risendronate/ oral alendronic acid)
Chang *et al.* (2021) [24]	1	37/M	Hemoglobin S-Beta-Thalassemia N/A	Mandible	Facial pain and swelling	N/A	N/A	Antibiotherapy, hemimandibulectomy and reconstruction with a titanium prosthesis + radial forearm and adipofascial free flap
Present case (2024)	1	22/M	Homozygous SS 2 weeks after management of a vaso-occlusive hip crisis	Mandible	Facial pain and swelling, Paresthesia of the lower lip, pericoronitis of decayed 3rd molar, secondary abnormal teeth mobility	No suggestive radiological signs were found on the panoramic radiograph and CT scan	Staphylococcus aureus	Antibiotherapy, surgical curettage/ sequestrectomy of the necrotic bone with removal of the involved teeth

Early detection and timely referral to a specialised centre for expert management are crucial for these patients. Therefore, dental practitioners and oral surgeons should maintain a high level of suspicion for this condition when treating SCD patients.

As indicated in Table I, primary treatment typically includes antibiotic therapy, guided by microbiological and histopathological analyses of samples obtained from the necrotic area. However, Daramola *et al.* [9] suggested that antibiotics should be used alongside surgical interventions, as using them alone may not effectively manage the condition. The antibiotics most frequently employed are penicillin, metronidazole, and lincomycin.

The surgical approach varied depending on the extent of sequestration, ranging from simple curettage of the necrotic bone with tooth extraction to more extensive procedures like hemimandibulectomy with microvascular free flap reconstruction, as detailed in the study by Chang *et al.* [24].

Regarding anaesthesia management, some authors such as Al-Ismaili *et al.* [22] or Olaitan *et al.* [5] recommended general anaesthesia with a prior transfusion exchange to ensure optimal resection access. However, other researchers like Chekroun *et al.* [25] suggested using local anaesthesia with conscious sedation to avoid systemic complications while minimising procedural stress.

General anaesthesia is not indicated in patients with sickle cell disease and should be discussed with the patient's medical team on a case-by-case basis [26]. In our case, the recent vaso-occlusive crisis and the need to perform a thorough curettage with a possible risk of fracture given the extensive limits of the necrosis led us to perform the operation under general anaesthesia in consultation with our colleagues in the maxillofacial surgery department.

In consultation with the haematologist and considering the high risk in our patient, the other dental procedures were carried out under conscious sedation to limit stress and with local anaesthetic without vasoconstrictor to avoid triggering a new VOC in an area that has already been affected.

However, patients with sickle cell disease are not considered to be at risk of infection (SFCO 2012 [27]), they may receive conservative care from general dentists without special precautions for conservative care. Depending on the severity of the sickle cell disease, only procedures involving a risk of haemorrhage require special advice from the haematologist (transfusions if necessary), as it is the vascular problems that can lead to the 'infectious' risk, and hospital treatment for these procedures is therefore indicated.

Futhermore, vasoconstrictors are not totally contraindicated in sickle cell patients for procedures outside the affected areas (oral surgery and conservative care) [26]. If used carefully, they can be used to achieve a more effective surgical silence, thus guaranteeing the best possible 'anti-stress' for these patients at risk of VOC. However, intra-septal and intra-ligamentary injections are not discouraged [28].

Additionally, Mahendran *et al.* [23] proposed a non-surgical approach involving the administration of oral bisphosphonates for chronic osteomyelitis. Although their use remains controversial within the dental community, the authors argued that the risk of developing medication-related osteonecrosis of the jaw from oral bisphosphonates typically occurs only after prolonged use exceeding four years, with an incidence remaining below 1%. In contrast, the average duration of bisphosphonate therapy in osteomyelitis cases is around 12 months. Moreover, clinical improvement is often observed shortly after initiation of treatment after starting treatment, suggesting that it may be advisable to stop treatment if symptoms persist beyond eight weeks.

Conclusion

Dental practitioners and oral surgeons must remain vigilant when providing care to SCD patients. This case report highlights the importance of recognising facial cellulitis in sickle cell patients who frequently experience vaso-occlusive crisis, as they may be misdiagnosed as odontogenic infections when they may in fact represent jawbone infarction. Prompt investigation and treatment are crucial for these patients, s delayed intervention can result in serious complications.

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Conflicts of interest

The authors declare that they have no conflicts of interest in relation to this article.

Data availability statement

The data used for this case can be accessed by contacting the corresponding author, D.R..

Ethics approval

Ethical approval was not required for this study.

Informed consent

Written informed consent was obtained from the patient.

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