CASE REPORT

Regional Odontodysplasia with Actinomycosis Infection: A Rare Case Report

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Abstract

A rare dental developmental anomaly known as regional odontodysplasia (RO) manifests itself in hypoplastic and hypomineralized teeth with discolored yellow to brown enamel. Radiographically, the affected tooth appears as a shell tooth with a large pulp chamber and a thin layer of teeth structure. The treatment plan is not well-established, and a multidisciplinary approach is often needed. However, a literature review revealed that most of the studies regarding RO are case reports, and more research, including epidemiological, genetic, and experimental studies, is needed to understand this condition better. Additionally, the cause of this anomaly is currently unknown. Removing the affected tooth is a controversial matter, and the treatment options should be chosen on an individual basis, considering the patient's goals for treatment.

This report describes an uncommon case of an 8-year-old girl who has RO affecting the front teeth in the upper jaw and a long-standing infection caused by actinomycosis.

Keywords: Actinomycosis, Infection, Regional odontodysplasia.

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Introduction and Literature Review

Regional odontodysplasia (RO) is a rare, noninherited, and localized developmental disorder that affects dental tissues that are derived from both ectoderm and mesoderm.[1] Hitchin[2] originally recognized odontodysplasia in 1934, and McCall and Wald first documented it in 1947, referring to it as "arrested tooth development." After observing their radiographic characteristics,[3] Rushton developed the term "shell teeth" in 1954.[4] The disorder has been referred to by several different names, including "ghost teeth," "nonhereditary segmental amelogenesis imperfecta," and "unilateral dental deformity." Odontodysplasia was first identified by Zegarelli et al. in 1963.[5] Pindborg subsequently included the word "regional" to stress the fact that only specific parts of the jaw are impacted by the disorder.[6] There is no racial discrimination in the prevalence of unilateral RO, which affects the maxilla more than the mandible (2:1) and typically affects the anterior teeth.[7] Pulpal pathosis, periodontal symptoms, and abnormalities in the eruption pattern of afflicted teeth are the clinical hallmarks of this condition. Teeth affected by this condition tend to be tiny and abnormal in shape and color (yellow or brown).[8] It is simple to diagnose this disorder because of its distinctive symptoms, but its treatment is challenging. There are different perspectives on the treatment.[9]

The cause of RO is not entirely clear, but it is thought to be due to local factors that influence the development of tooth tissues. The causes of RO include issues, such as neural crest cells not moving properly, problems with blood flow (localized lack of blood flow), viral infection, medication, somatic mutation, and localized trauma. However, the exact cause of the condition is still unknown.[10]

Although RO has been recognized for over 60 years, only case reports exist in the literature. Therefore, more epidemiological, experimental, and genetic research is needed. Caries is a possible diagnosis for RO. The condition needs to be managed over the long-term, so an early diagnosis is crucial for both primary and permanent teeth.[9] Roughly 176 cases were published in 2015.[11] From 1953 to 2017, 161 cases were examined in a recent study.[12] Then another with 44 cases in 2020.[13] Due in part to there not being a national or international registry for patients with RO, there are not a lot of cases reported in the literature. Typically, RO affects just one side of the body and does not radiate outward from the midline. To date, 12 cases involving both sides and multiple body parts have been documented.[10,14] Diagnosis occurred between the ages of 1 and 23, with two distinct peaks at 4–10-year-old; the condition typically presents itself when the child is in the midst of their formative years and still has their baby teeth.[7]

Etiology and Pathogenesis

In the literature, multiple possible causes have been proposed for RO, including local trauma and infection, ischemia and vascular pathogenesis, activation of a dormant viral infection that affects the development of tooth germs, metabolic and nutritional disturbances, medications taken during pregnancy, irradiation, and systemic disease.[10]

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Ischemia and Vascular Pathogenesis

The most accepted explanation for RO's origins is that it originates in the cardiovascular system. Several cases have been linked to abnormalities in the circulation just above the site of the problem. There have been three cases documented of RO where a vascularity abnormality manifested itself on the skin overlaying the teeth. Similar symptoms of RO have been observed in animal trials in which arteries in the head and neck were either ligated or otherwise disrupted. Odontogenesis can be negatively impacted by localized ischemia due to defects in blood vessel formation or other regional disturbances.[7]

Trauma and Infection

Although trauma and infection have been linked to the ailment in some reports, the actual reason will always rely on the patient's history.[15]

Viral Infection

When the polyomavirus was produced in mice tooth germs that had been transplanted from humans, Nagai et al.[15] observed abnormalities in tooth development. Thus, it has been hypothesized that viruses may be responsible for infecting the odontogenic epithelium and causing tooth developmental anomaly. Despite this, published case reports did not indicate any signs of viral infection.

Malnutrition

Patients with RO have been reported to have phosphatemia, hypocalcemia, and an imbalance of essential proteins, such as metalloproteinases, all of which may contribute to the condition's structural disarray.[16] The failure to account for some data has led to the elimination of many of the earlier genetic or geographical factors.

Clinical Features

One of the most commonly reported signs of RO is a delay in the emergence of teeth or an enlargement of the gingival tissue surrounding them that is not due to inflammation. Even in the absence of visible caries, some patients may nevertheless experience pain and abscesses in the afflicted area. Bacterial infection of the pulp, brought on by cracks in the enamel and dentine, is thought to be the root cause of these symptoms.

The teeth affected by the condition are smaller than normal, have less calcium, and are discolored (yellow or brown); also, the eruption of teeth is delayed or does not happen at all. As a result, the alveolar crest in that area of the mouth is often expanded and coated in fibrous tissue. Teeth affected either do not erupt at all or later than expected.[1]

Radiographic Features

A lack of enamel and dentin causes the affected teeth to seem ethereal ("ghost teeth") in X-rays. Large pulp chambers and short, open-ended roots are also symptoms of teeth that have stopped developing.

Those teeth have hypoplastic crowns and aberrant morphology. In rare cases, calcification can be found in the pulp chambers or root canals.[17] The presence of pericoronal radiolucency around an impacted tooth indicates an enlarged dental follicle.[17]

Histological Features

Hyperplastic connective tissue in the dental follicles contain odontogenic epithelial rests, which are distributed foci of calcification within the odontogenic epithelium. When compared to typical teeth, the predentin region is typically larger. There is prominent interglobular dentine and broad open apexes on these small, thin-walled roots.[15]

In light of the consensus among researchers, it is fitting to use this term to describe all tooth germ offspring. Histological findings are unique, despite radiographic similarities with other disorders (such as amelogenesis imperfecta type III, or "shell teeth"). Dentin and enamel defects can be caused by improper odontoblast and ameloblast development. Hypoplasia, hypomineralization, and degraded globular calcifications characterize the enamel organ. Hypoplastic dentin is characterized by the presence of clefts, a type of dentin that forms between the globules, a widened area before dentin, and fewer tubules in the dentin. There have also been reports of a scalloped dentinoenamel junction and osseous metaplasia of the dentin.[16] Fibrosis and calcifications are examples of alterations that can occur in the pulpa. In some instances, people may see microscopic, calcified formations adhering to the tooth germ or hypoplastic enamel.[18]

Differential Diagnosis

Hereditary disorders, such as amelogenesis imperfecta, dentin dysplasia, and dentinogenesis imperfecta, should be considered in the differential diagnosis of RO. While enamel, dentin, and pulp changes can be seen in both hereditary disorders, RO differs in that it affects the entire dentition rather than just one area.[19]

Management

Regional odontodysplasia (RO) treatment is complex and often calls for the participation of experts from other fields. It is not uncommon for specialists in pediatrics, orthodontics, prosthodontics, and surgery to need to consult with one another. When considering treatment options for a lesion on the teeth, various factors must be considered, such as the patient's overall health, age, the extent of the lesion, the timing of tooth emergence, and the appearance of the treatment.[5]

There is some controversy over the best way to treat odontodysplasia. Most dentists would rather have the affected teeth pulled and replaced with prosthetics right away (e.g., acrylic partial denture). Other medical professionals have stressed the importance of restorative operations to safeguard the impacted teeth that have already erupted.[4,18] The most important aspect is timing. Unrestorable abscessed teeth should be taken from young children; however, it is preferable to save as many of the impacted teeth as possible, both for the sake of the child's jaw growth and their mental health. If a child is older, they likely have one or more abscessed permanent teeth that need to be pulled along with any others that need to be kept until a prosthesis can be made.

The treatment's objectives include enhancing chewing performance, encouraging the eruption of unerupted teeth, improving esthetics, reducing psychological discomfort, and ensuring the safety of teeth that are emerging at an angle.[20,21] Many dentists decide to remove these teeth straight away and then restore the patient with a removable partial acrylic denture because these teeth, even if they do erupt, are misshapen and have an unattractive appearance.

Some dentists have suggested that extracting teeth too early can have negative psychological repercussions, while others point out that improper jaw growth and facial asymmetry can be caused by a significant decrease in the height of the jaw ridge and a loss of the vertical measurement on the side that is affected.[2,5,15]

There is a lot of research on using osseointegrated implants to treat hypodontia in growing kids.[16,22] As RO does not affect the

overall bone quality; implants may be useful in some circumstances. However, because there have been reports of the decreased density of the bone surrounding the affected teeth, extra care should be taken when performing these types of treatments.[16] Mandibular implants must be carefully positioned anterior to the mental foramen,[20] and it is common for there to be some level of difficulty in the positioning of dental implants in the upper jaw because of changes that occur in the sinus and nasal cavity during growth and development.

Currently, there is no agreed upon standard for treating RO. The dentist must think about the child's age, dental experience, teeth affected, medical history, the presence or lack of disease, and the children and parents' attitudes and preferences. Treatment targets should include facilitating mastication and speaking, enhancing esthetics, allowing for proper growth and development of the jaw, minimizing psychological damage, and protecting any erupted problematic teeth, if possible.[23]

Case Description

An 8-year-old girl who appeared to be in good health visited a pediatric specialist clinic at King Abdul Aziz University in Jeddah, Saudi Arabia. The patient was experiencing a delay in the emergence of one of their upper front teeth, and another was only partially visible. Additionally, the gums around both teeth had become enlarged, as depicted in Figure 1. The patient has one older brother and two younger sisters. No definitive information was uncovered on the patient's prenatal or natal problems. Neither mother nor father could recall a family history of teeth or genetic problems. Her medical and familial histories were completely normal; she had no infectious infections as a child and had never been exposed to radiation, except for a single incident involving a broken front tooth when she was 3.

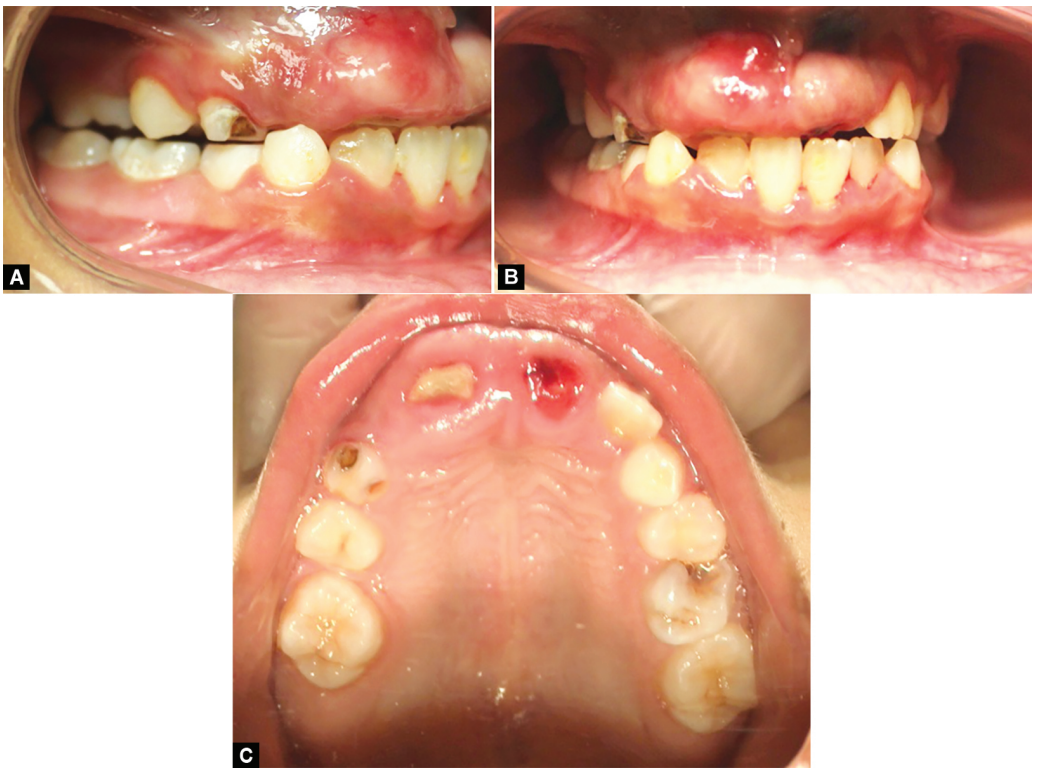
Dental history was insignificant and showed multiple restorations. The patient had a poor protein and vegetable intake, according to the examination of her diet. The patient had normal facial symmetry and healthy skin, hair, and nails, according to the extraoral examination. Results were clinically nonabnormal, and no lymphadenopathy was seen. An examination of the patient's mouth revealed a mixed dentition that was both complete and unrestored, as well as evidence of extensive caries and subpar restorations. Based on the results of the caries risk assessment, the patient was classified as a high-risk individual. All of the teeth seemed healthy except for the permanent maxillary front ones—the right central incisor was only partially erupted, hypomineralized and hypoplastic, unusually shaped with a rough surface, and yellow discoloration, and the two upper front teeth, the left central incisor, and the right lateral incisor were not visible as they had not yet emerged from the gums.

An abscess connected to the upper central tooth and fibrous growth of the upper jaw was noted (Fig. 1).

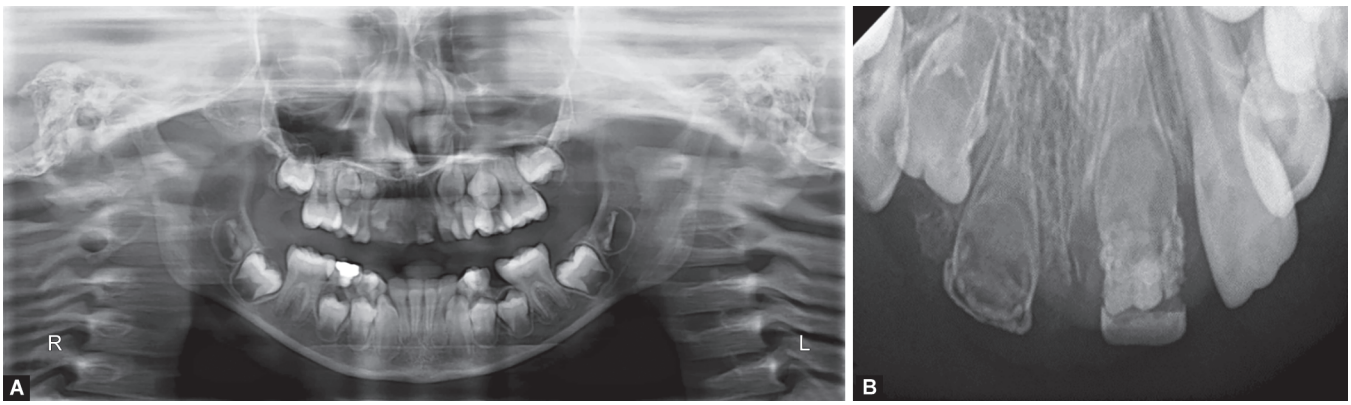
Same-day panoramic and upper occlusal radiographs (Fig. 2) confirmed the presence and normal development of all permanent and deciduous teeth except the right lateral incisor, upper central incisors, and canine. Hypomineralization and decreased radiodensity were also noted. These teeth seemed to be "ghost-like," with a faint radiopaque contour but no discernible crown or root structure. The damaged teeth had extensive radiolucent regions surrounding the crowns.

Differential Diagnosis

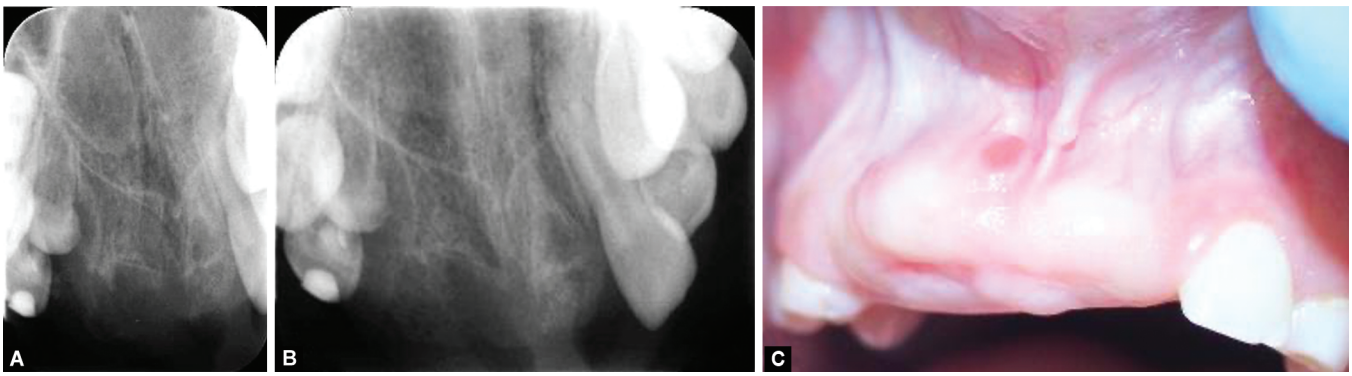
The consultation was performed with multiple teams in the dental hospital, including the teams from the periodontics, endodontics, orthodontic, and pediatric dental departments. A consensus was obtained on extracting the teeth. The team of orthodontics decided to start the orthodontic treatment after the removal of



Figs 1A to C: (A) Lateral view; (B) Anterior view; (C) Occlusal view. The figure shows fibrotic gingival enlargement surrounding the affected teeth with signs of infection



Figs 2A and B: (A) Panoramic X-ray; (B) Upper occlusal X-ray shows ghost tooth appearance of the affected tooth



Figs 3A to C: (A) Upper periapical; (B) Upper occlusal show remaining ghost teeth roots; (C) Clinical picture of fistula and abscess

the front teeth and providing a fixed partial prosthesis with the fixed appliance to raise the bite and avoid further trauma to the region of the odontodysplasia. The pediatric team planned on improving the oral hygiene, scaling, and smoothening of the rough surface of the tooth and following up on the eruption of the tooth every 3 months.

After 1 year had passed since the removal of the problematic teeth in a private clinic, the patient presented to the pediatric dental specialty clinic complaining of pain and an abscess at the surgery site. Upper occlusal and periapical radiographs confirmed the diagnosis and pinpointed the specific site of the damaged teeth, showing a shadow of the surviving root of the upper left central incisor and discontinuity of the labial cortical plate (Fig. 3). Cone-beam computed tomography analysis revealed the tooth follicle and root of the upper right canine, as well as a break in the bone's cortical plate near the upper left incisor (Figs 4 and 5). So, we opted to conduct a more extensive sort of surgery on the patient, called exploratory.

During exploratory surgery at a dentist's clinic, a maxillofacial surgeon removed the remaining root of the upper left central incisor, the tooth follicle, and granulation tissue of the upper right canine using nitrous oxide as anesthesia. Histopathology was performed on both hard and soft tissues, and the patient was given an antibiotic (amoxicillin 250 mg, three times a day for 5 days) and a mouthwash (chlorhexidine twice a day for 10 days).

Even after 5 days of antibiotic treatment, the patient's inflammatory surgical site had not improved, and radiography showed an irregular appearance of the bone (Fig. 6).

Upon examination, the hard tissue appeared to resemble teeth in structure but was thinner than typical teeth and had a smaller number of dentinal tubules that were not arranged in a regular pattern. Additionally, the pulp chamber was larger than usual and contained many small stones. The dentinal tubules were interrupted by globular masses, and interglobular dentine was present in abundant quantities. Near the enamel–dentine interface, the dentine calcified more consistently. Tooth roots were small and thin-walled, and interglobular dentine was more obvious than in teeth with normal development, characteristics that point to RO (Fig. 7). Granulation tissue had cells that were causing inflammation, and the presence of certain types of bacterial colonies that have a blue center and a red outer ring (as seen in Fig. 8) suggested the presence of a condition called actinomycosis.

The doctor recommended not to touch the patient until the actinomycosis was treated and sent the patient to the pediatric clinic at King Abdul Aziz University Hospital for further examination. Antibiotic treatment for the infection lasted for 6 months and was successful (amoxicillin, 500 mg twice/day).

The medical doctor gave his or her okay for the orthodontic and restorative treatment to begin. After finishing up with antibiotics, the patient recovered enough to move on to the next stages of orthodontics and prosthodontics.

The patient was fitted with an interim fixed appliance (Figs 9 and 10). The daughter gained confidence and resumed her previous activities, much to the delight of her mother. So, 6 months after finishing antibiotic treatment, the patient reported feeling better intraorally and had stopped complaining about the site. The patient's doctor has given her the all clear to commence orthodontic therapy. Orthodontic therapy was undertaken to preserve dental health, enhance facial beauty, and promote healthy jaw development.

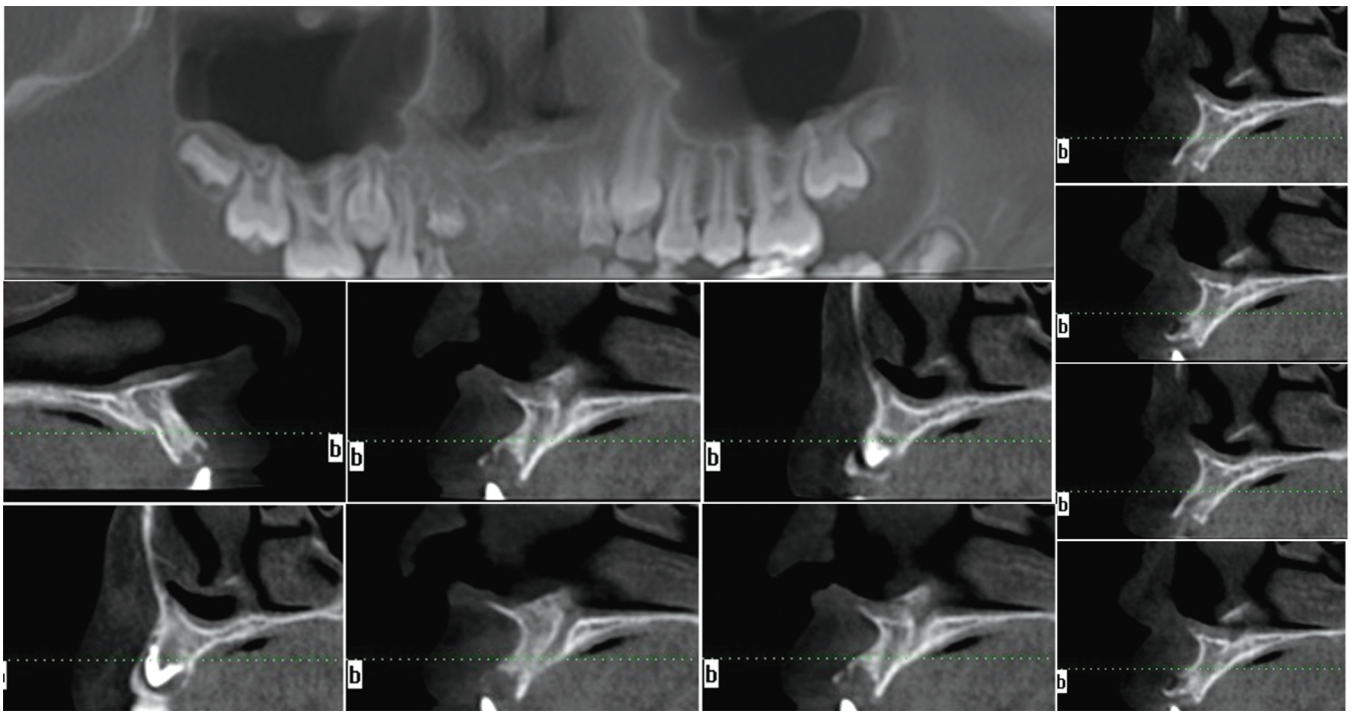


Fig. 4: CBCT shows destruction of the labial cortical plate and remaining root

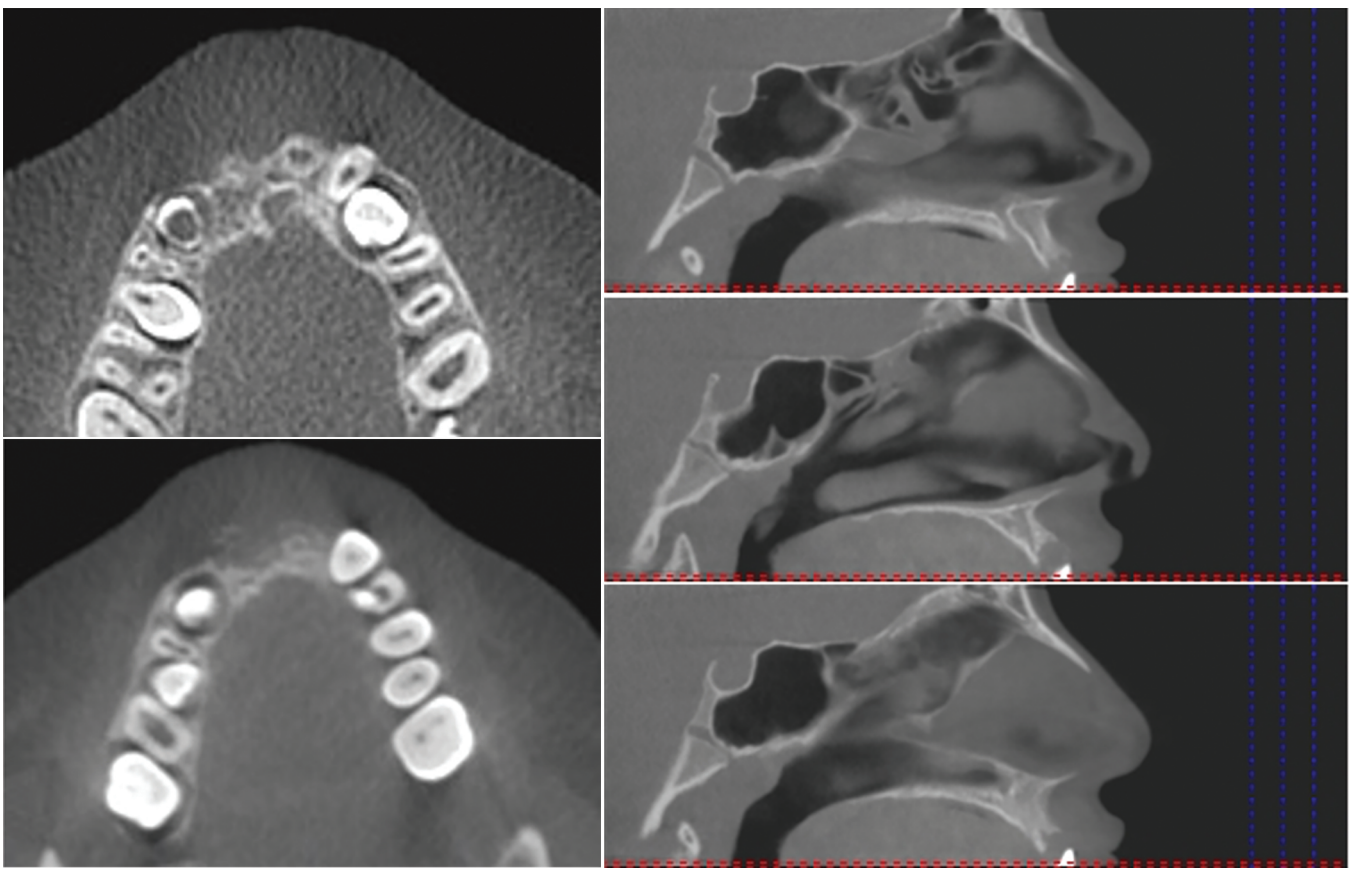
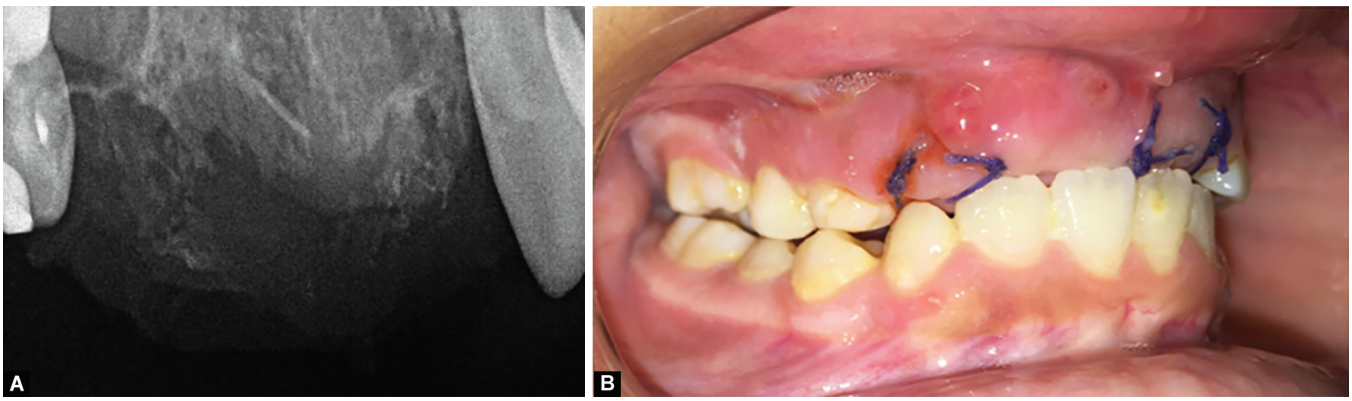


Fig. 5: CBCT axial view shows severe destruction of the labial cortical plate and the remaining root of tooth 21



Figs 6A and B: (A) Osteoclastic activity on periapical X-rays; (B) Signs of infection still present

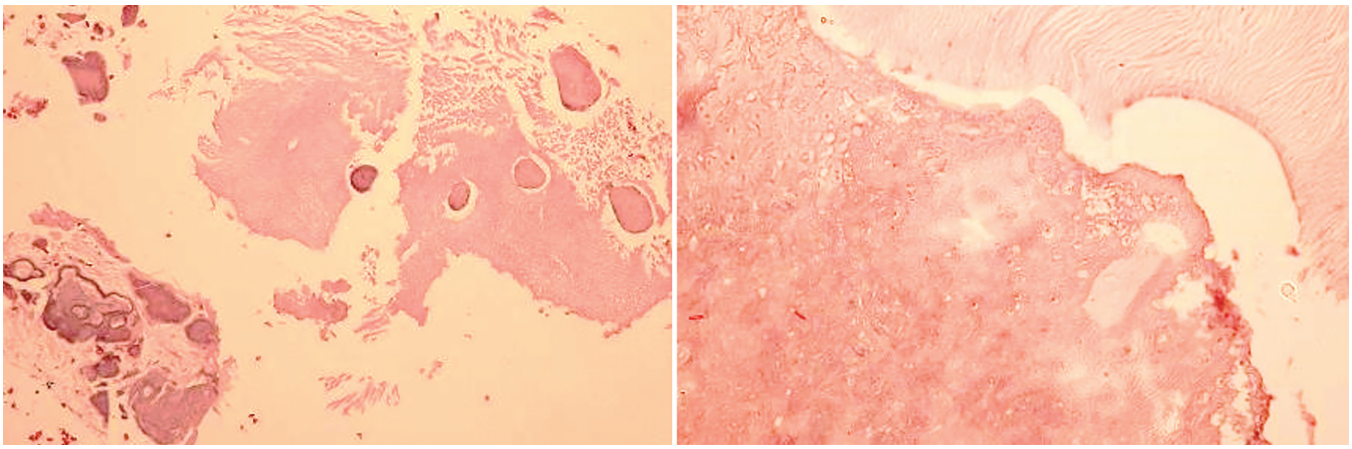


Fig. 7: Microscopic view of hard tissue. Tooth-like structure with large pulp cavity and many pulp stones

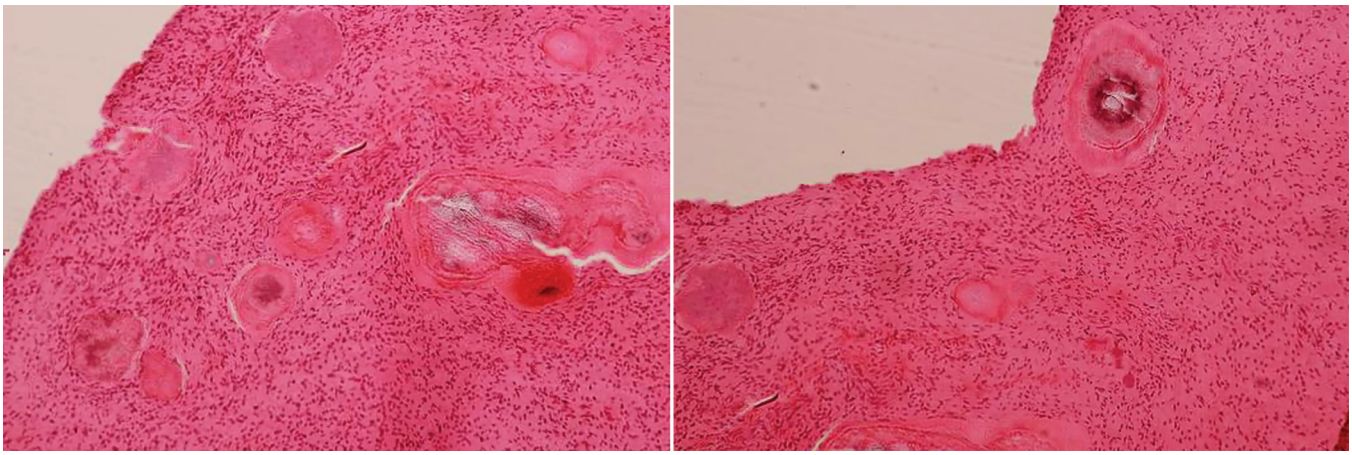


Fig. 8: Microscopic view of soft tissue. Collection of granulation tissue infiltrated with inflammatory cells. Bacterial colonies with basophilic stain in the center of the colony and eosinophilic stain at the periphery. Areas of dense collagen with minimum vascularity

Once orthodontic therapy is finished, the implant would be placed in the region of the missing tooth, and the patient would use the new fixed appliance until they turned 18-year-old.

Discussion

Regional odontodysplasia (RO) is an uncommon, localized, and noninherited developmental disorder that affects the dental hard tissues that are derived from both the ectoderm and mesoderm. The maxillary anterior tooth is more prone to the disease than the corresponding male molar. Both the baby teeth and the adult teeth are affected by this problem. This case report presents a textbook example of RO.

Several potential causes have been proposed in the scientific literature, including ischemia and vascular pathogenesis,

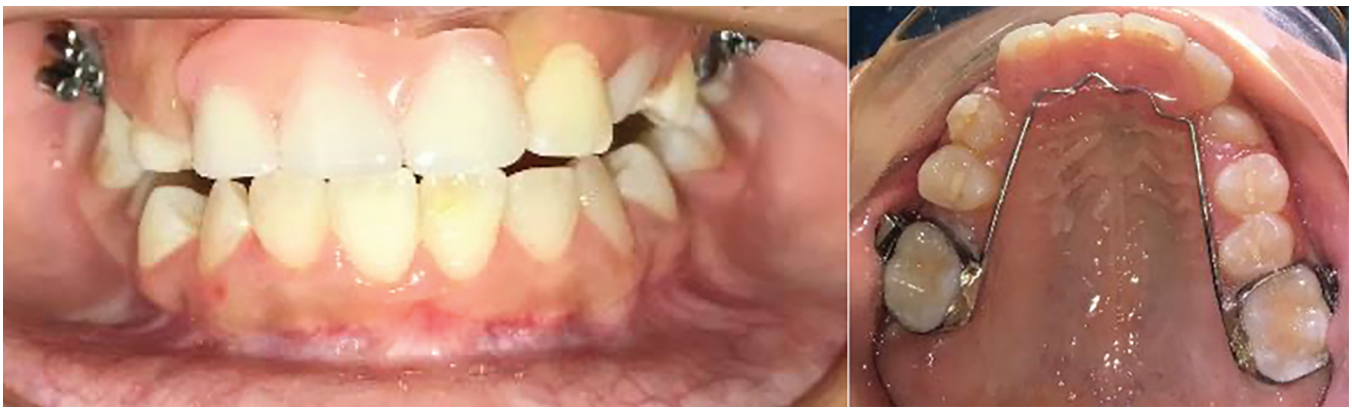
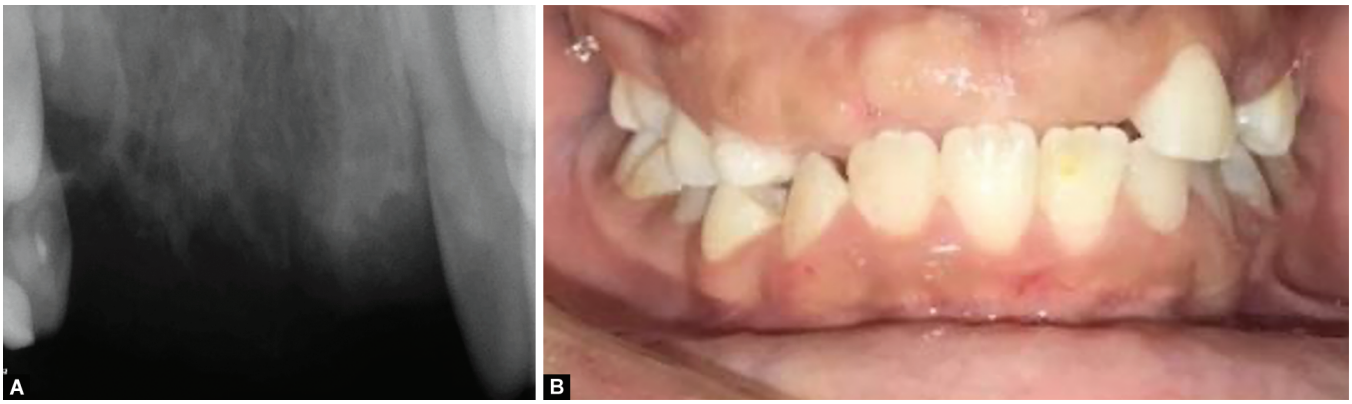


Fig. 9: Interim intraoral appliance



Figs 10A and B: 6 months follow-up. (A) Periapical X-ray shows signs of healing and bone fill; (B) Normal soft tissue appearance with no signs of fistula or infection.

medications taken during pregnancy, metabolic and nutritional disturbances, the activation of a viral infection that was previously dormant in the tooth germ during the process of tooth development, local trauma and infection, irradiation, and systemic disease.[10]

The most widely held idea attributes RO's origins to a vascular hypothesis. In multiple recorded occurrences, abnormalities in the region's local circulation were found to be to blame. There are three reported examples of RO, a condition characterized by a vascularity abnormality of the skin covering the teeth.[7]

Similar characteristics of RO have been seen in animal tests in which arteries in the head and neck have been ligated or otherwise disrupted. Furthermore, localized ischemia might disrupt odontogenesis due to a malfunction in blood vessels or other small disturbances in blood flow.[7]

The reports that attribute the disease to trauma and infection depend entirely on the patient's history.[14] This case study features a patient who, at the age of 3, experienced trauma. When the polyomavirus was produced in mice tooth germs that had been transplanted from humans, Nagai et al.[15] observed abnormalities in tooth development. Therefore, odontogenic epithelial infection by viruses has also been proposed as an explanation. In this case study, an actinomycosis infection was found in the patient. More research is needed to determine if there is a connection between actinomycosis and RO, despite the fact that there was no association between the two in this patient.

Patients with RO have been reported to have hypocalcemia, phosphatemia, and an imbalance of essential proteins, such as metalloproteinases, all of which may contribute to the condition's deterioration in the structural organization.[16] Because of their inability to account for certain observations, many of the earlier genetic or geographical causes have been discounted.

Clinical Features

Local trauma could be the cause of this. The current situation can be characterized as a typical RO case. According to the literature, the condition is more prevalent in the left maxillary arch compared to the right mandibular arch, with a 2:1 ratio of involvement. Additionally, it is four times more likely to occur in females. It is also noteworthy that the condition does not cross the midline.[2,8,15,22]

This accords with the current case's findings; however, it also reveals evidence of bilateral involvement.[16] All of the teeth in the affected arch were damaged to the same degree, even though the problem typically strikes the front teeth first. The abnormality typically "skips" a tooth or set of teeth along a line of teeth; however, this is not always the case.[23]

Classically, patients with hypoplasia present with partially erupted teeth that seem yellowish-brown and pitted.[2,5,8,20] Due to hypomineralization, the tooth has become fragile and easily broken. Consistent with the current report, gingival enlargement is seen even in the absence of caries.[14,24]

The radiography of the teeth affected by the condition displayed a characteristic "ghost-like" appearance, with enamel and dentin that were less mineralized and no clear demarcation line between the two.[2,15] The teeth seemed shorter than normal,

and their apices were extremely wide open, revealing a huge pulp chamber. This matches up with what has been reported in the literature about this issue.[15,18,24] In addition, we noticed that the permanent teeth or the next generations of tooth germs were also affected.

The way to treat the teeth affected by RO (the condition) is still a topic of debate among professionals.[8] There must be a consistent and interdisciplinary effort for these cases. Some recommend extraction of a tooth with periapical diseases, followed by placement of a prosthetic that restores the tooth's function and appearance, while others advocate for restorative care wherever possible. Although autotransplantation was offered as a treatment for patients with RO by Cahuana et al.,[9] access to an eligible donor makes this approach unfeasible in most cases. For patients with odontodysplasia, oral rehabilitation using endosseous implants was proposed by Bulut et al.[25]; however, this treatment can be done at a later age. As there is no particular treatment for odontodysplasia, the care must be customized to the specific patient considering factors such as age, willingness to comply with treatment, attitude towards dental care, and the severity of the condition.

Conclusion

Although RO was described >6 decades ago, most studies related to this condition are case reports; therefore, more epidemiological studies regarding the condition and the etiology of the disease are needed. This case illustrates the importance of thorough examination and a multidisciplinary approach to managing difficult cases of RO. Further research is needed to confirm the association between RO and actinomycosis.

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