Clin Oral Invest (2004) 8:45–51
DOI 10.1007/s00784-003-0245-0

REVIEW

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Regional odontodysplasia: a review of the literature and report of four cases

Received: 27 August 2003 / Accepted: 12 November 2003 / Published online: 9 January 2004

Abstract Regional odontodysplasia (RO) is a rare dental anomaly involving both dentitions, mostly teeth of one quadrant. The characteristic findings are discolored soft teeth accompanied by gingivitis, swelling or abscess. Enamel and dentin are hypomineralised and hypoplastic, so that the 'ghost teeth' appear shadowy in radiographs with wide pulp chambers. The etiology is unknown. Epidemiological data is rare; 138 cases of RO have been published to date and reports on ultrastructure are few. An analysis of published cases of RO in the international literature is presented. The sex ratio of females to males was 1.7:1. The age at the time of diagnosis ranged between 4 and 23 years. The maxilla was more often affected (maxilla to mandible ratio 1.6:1). In 67 patients the deciduous and permanent dentitions were affected (47.1%). In 129 cases, affected teeth lay side by side. Missing tooth development was observed in 10.7%. Failure of tooth eruption of RO teeth occurred in 39.7%. In addition, four cases with RO which were collected over a period of more than 25 years are presented. Ultrastructural findings of one specimen are demonstrated.

Key words Regional odontodysplasia · 'Ghost teeth' · Ultrastructure · Epidemiology

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Introduction

Regional odontodysplasia (RO) was probably first described by Hitchin in 1934 [42]. It is an uncommon dental developmental anomaly affecting primary and/or permanent teeth and is usually apparent in one jaw and mostly in one quadrant. Both the maxilla and the mandible may be affected. Six cases described in the literature displayed generalised odontodysplasia with involvement of all teeth [37, 38, 39, 58, 85, 102]; however, some cases appear doubtful due to poor radiographs and presentation of histological findings [37, 58, 85].

Clinically, teeth show a brown or yellowish discoloration and are soft on probing. The most frequent clinical symptoms after eruption of teeth with RO are swelling of the gingiva, gingivitis, periapical infection and abscess formation. Failure of tooth eruption may occur. In three cases, fever attacks were reported, which disappeared when the affected teeth were removed [21, 34, 92]. Nine cases were associated with haemangioma [3, 20, 35, 44, 62, 89, 93, 102, 109] and six cases with facial haemiatrophy [8, 29, 73, 89, 107].

Radiographically, the affected teeth show a ghostlike appearance ('ghost teeth') due to their reduced radiodensity. A demarcation between hypomineralised dentin and hypomineralised enamel is not visible and pulp chambers, as well as the apical foramen, appear wide.

The morphological findings in RO are distinctive. A typical finding is hypoplastic, hypomineralised and matrix-enriched enamel of variable thickness. Often globular calcifications or apatitic needles have been found. The prism structure appears atypical.

The dentin layer is reduced in thickness and expanded areas of irregular interglobular dentin are found. A layer of extended predentin is frequently observed. In severe cases the tubules are reduced in number and their processes are disturbed. In less severe cases the dentin and in particular the mantle dentin seems to be normally structured. However, interglobular dentin and clefts between the tubules have often been observed [1, 25, 26, 61]. Clefts predominantly occur in proximity to the

amelodentinal junction and partly extend into the pulp and can be an entrance for bacteria and a cause of pulpitis [1, 14, 25]. Often the pulp contains denticles and amorphous calcified material. There are few investigations of the cement and follicle. However, foci of calcification have also been found here [61, 45].

The etiology of RO is not yet clarified and different theories including local circulatory disorders [14, 20], infection [29], teratogenic drugs, neural crest cell defects or trauma [20] have been discussed [20, 60, 61]. Regional odontodysplasia seems to be non-hereditary, since no cases with affection of family members have been reported. Likewise, data on a specific ethnic distribution are not available.

The aim of the present study was 1) to evaluate cases of RO published in the international literature and to compile epidemiological data; and 2) to report four of our own cases of RO and supplement reports on the ultrastructure of RO [13, 20, 25, 27, 40, 45, 46, 47, 48, 49, 54, 61, 69, 82, 88] with findings in one of these four cases.

Materials and methods

A review of the literature revealed 138 cases of RO that have been published from 1934 up to the end of the year 2002 [1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 11, 12, 13, 14, 15, 16, 17, 18, 19, 20, 21, 22, 23, 24, 26, 28, 29, 30, 31, 32, 33, 34, 35, 36, 37, 38, 39, 40, 41, 42, 43, 44, 45, 46, 47, 48, 49, 50, 51, 52, 53; 55, 56, 57, 58, 59, 62, 63, 64, 65, 66, 67, 68, 69, 70, 71, 72, 73, 74, 75, 76, 77, 78, 79, 80, 81, 82, 83, 84, 85, 86, 87, 89, 90, 91, 92, 93, 94, 95, 96, 97, 98, 99, 100, 101, 102, 103, 104, 105, 106, 107, 108, 109]. Including our four own cases, a total of 142 cases could be analysed statistically using the Computer programme SPSS 8.0. Data were analysed according to the criteria of age, sex, number and type of affected teeth, history and clinical symptoms.

Four cases of RO were diagnosed over a period of more than 25 years. In the first patient, a 7-year-old boy, the second quadrant was affected with involvement of teeth 22–25. The second patient was a 10-year-old male with involvement of teeth 21–26. The third case was a 5-year-old girl with involvement of teeth 54, 55 and 16. In the fourth case of a 10-year-old girl, teeth 31–35 were revealed to be odontodysplastic radiographically (Fig. 1a–f). Tooth 31 was removed surgically and tooth 73 was extracted. Tooth 31 was immersed in glutaraldehyde (2.5% in phosphate buffer, pH 7.4), dehydrated and embedded in epoxy resin (Epon 812) for light and transmission electron microscopy without decalcification. The specimen was sawed longitudinally and segmented in 15 pieces from which semi-thin sections and successive ultrathin sections of 70 nm were obtained. The specimens were examined by transmission electron microscopy (TEM, Philips CM 100) at an acceleration voltage of 80 kV.

Fig. 1 **a** RO involvement of teeth 22–25 in a 7-year-old boy. Tooth 65 is impacted. **b** RO involvement of teeth 21–26 in a 10-year-old boy. **c** RO involvement of teeth 54, 55 and 16 in a 5-year-old girl. **d** RO involvement of teeth 31–35 in a 10-year-old girl. **e** Periapical radiograph showing details of d. **f** Clinical photograph of tooth 31 (case 3) showing hypoplastic enamel

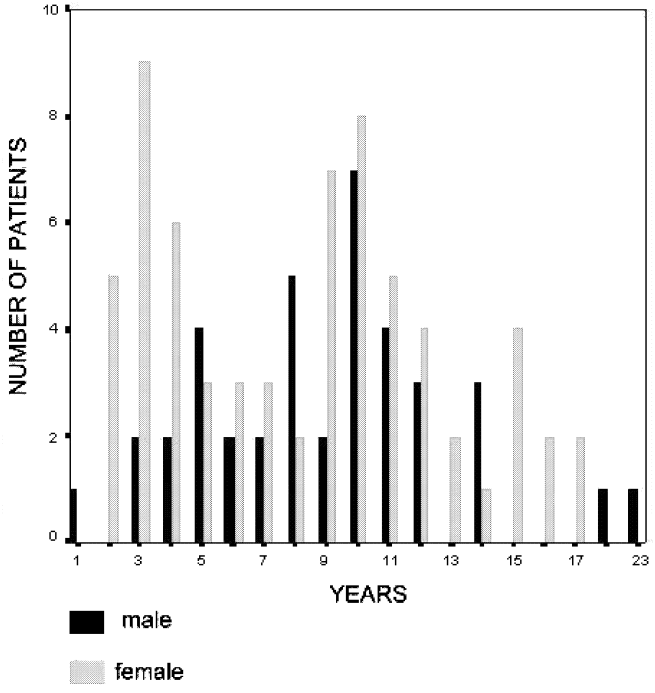

Fig. 2 Age of patients at time of diagnosis

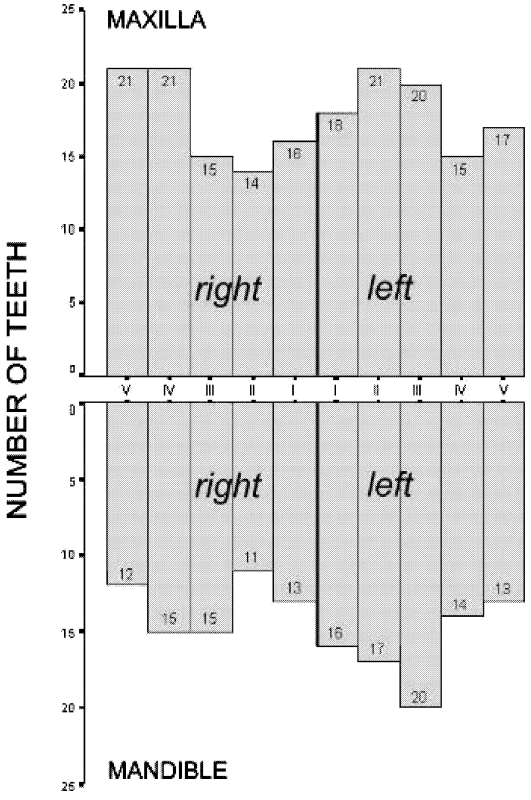

Fig. 3 Distribution of deciduous teeth affected by RO

Results

Epidemiology

At 63.1% (n=89), females were more frequently affected than males (ratio 1.7:1). At the time of diagnosis, the age of all recorded patients ranged between 1 and 23 years. The age distribution was bimodal with two peaks at 4 and 10 years in relation to the expected time of eruption of either primary or permanent teeth (Fig. 2).

The maxilla was more frequently involved than the mandible (1.6:1). In detail, the first quadrant was affected in 31.9%, the second quadrant in 35.5%, the third quadrant in 22.0% and the fourth quadrant in 23.4%. In each single patient, either one quadrant or two quadrants of the same side or jaw were affected with the exception of six cases with generalised odontodysplasia [37, 38, 39, 58, 85, 102]. Comparing males and females, a significant difference in affected quadrants was not detectable.

In 67 cases, both dentitions were affected. In 52.9% of the patients in whom RO of the deciduous dentition was diagnosed, 42.9% of the permanent teeth were also affected.

In 129 cases all teeth affected of a quadrant lay side by side, whereas in the remaining 13 cases healthy teeth adjacent to RO-positive teeth were found [19, 20, 22, 33, 37, 38, 43, 68, 71, 84, 90, 93, 95]. Missing teeth (missing tooth anlage) were observed in 10. 7% [8, 11, 19, 32, 35, 57, 59, 65, 70, 75, 79, 99, 106, 107]. The distribution of affected individual teeth is shown in Figs. 3 and 4. In the deciduous maxillary dentition ($n_{maxilla}$=178), the most frequently affected teeth were the left lateral incisor and

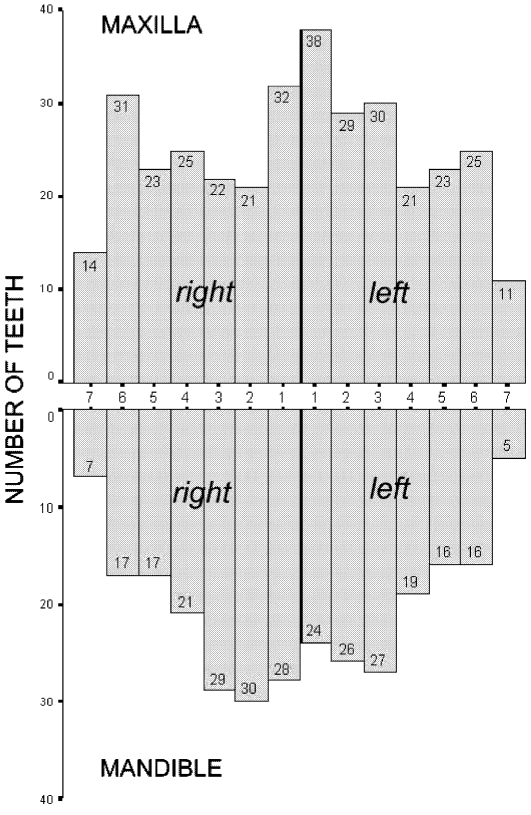

Fig. 4 Distribution of permanent teeth affected by RO

the right first and second molars, each with 15% (Fig. 3). In the deciduous mandibular dentition ($n_{mandible}$=146), the most frequently affected tooth was the left canine (14.3%) followed by the left first (11.4%) and second incisors (12.1%).

In the permanent maxillary dentition (Fig. 4) ($n_{maxilla}$= 345), both first incisors were most frequently affected, with the highest percentage of 11% for tooth 21. The right first incisor and the right first molar were affected in 9.3 and 9.0%, respectively. In the permanent mandibular dentition ($n_{mandible}$=282) the most frequently affected teeth were the right and left second incisors and canines (9.9–10.6%).

A most characteristic clinical symptom was failure of tooth eruption of RO-positive teeth in both dentitions (39.7%). Frequently, hyperplastic tissue was observed overlying the unerupted or partly erupted teeth. Inflammation of the gingiva and periodontium was found in 50.4% and was occasionally accompanied by periapical infection and fistulas [10, 18, 28, 34, 44, 84, 94, 100, 105]. As a consequence, local pain was frequently reported by the patients. Usually, RO-positive teeth were not decayed but were very fragile due to hypomineralisation. History of trauma was recorded only in 3.3% of the cases [7, 9, 75, 108]. RO was associated with facial haemangioma [3, 20, 35, 44, 62, 89, 93, 102, 109] in 7.4% and with facial asymmetry or hypoplasia [8, 29, 73, 89, 107] in 5% of patients. In two cases RO was an incidental finding [55, 79]. Three cases of RO were diagnosed in adults, with one case due to aesthetic problems [78], while the majority was diagnosed in adolescents.

Morphological findings

Macroscopically, the crown of the permanent mandibular incisor (31, case 4) was dysmorphic and dysplastic. Light microscopically, a smooth membrane coating of dentin was observed as a narrow band of an intensely stainable material. Due to its high affinity for staining agents, this material was supposed to contain a high organic and a low or even no mineral portion. TEM revealed the enameloid substance not to be composed of a homogenous material but rather of polymorphous bodies with a granular content that were embedded in a less-electrondense mass and demarcated from it by a clearly visible border line (Fig. 5). Both the polymorphous bodies and the enveloping mass were completely void of mineral deposits, i.e. apatitic needles or crystals. Furthermore, ameloblasts, a reduced enamel epithelium or remnants of the enamel organ at the surface of the enameloid substance were not observed.

Dentin presented as a regularly structured hard tissue apart from extended areas of interglobular dentin comprising larger spaces than physiological crown interglobular dentin. These numerous concave-shaped spaces of interglobular dentin were enriched by an unmineralised collagenous matrix. Some of the dentinal tubules traversing the circumpulpal dentin appeared as empty canals and were considered to have lost their odontoblastic processes as a result of preparation artefacts (Fig. 6).

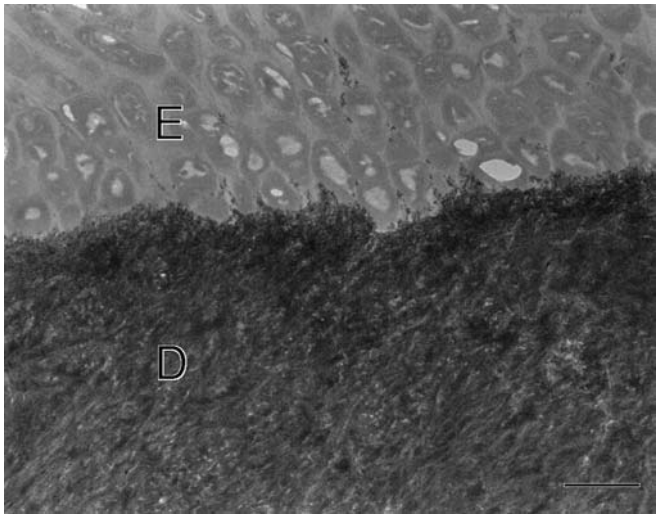

Fig. 5 Transmission electron micrograph of junction between dentin (*D*) and enameloid substance (*E*). Original magnification ×5000 (bar=2 μm)

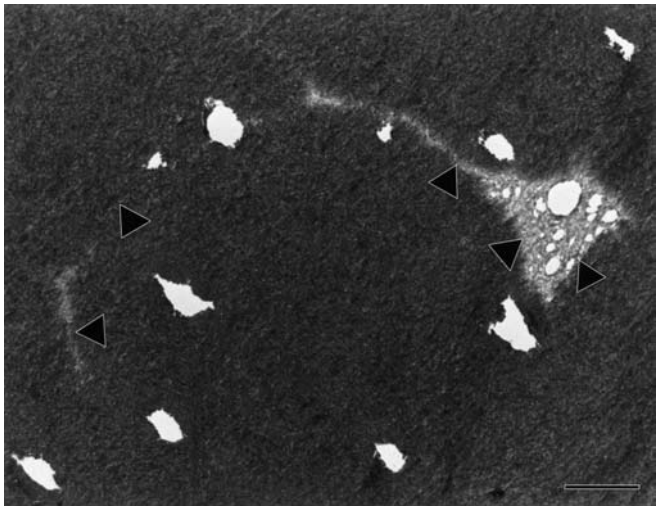

Fig. 6 Transmission electron micrograph of dentin displaying a region of interglobular dentin (*arrowheads*). Note cross-section of empty dentin tubules. Original magnification ×5000 (bar=2 μm)

Discussion

No prevalence data for RO are presently available, possibly as a result of false diagnosis, missing documentation and publication. Only Crawford and Aldred [14] presented a statistic evaluation in 1989. One problem of the statistic evaluation was the large number of insufficiently documented case reports. Details of the history were not always available and poor radiographs were presented frequently. Often no histological investigations were accomplished or these were only insufficiently

described and documented. Also, the status of the dentition was not documented in some cases.

The present study showed a ratio of females to males of 1.7:1, indicating a somewhat higher prevalence in females when compared with the findings of Crawford and Aldred [14]. The involvement of the maxilla compared with the mandible (1.6:1) was slightly reduced when compared with the study of Crawford and Aldred (2.5:1) [14]. In 47.1% of cases both dentitions were involved.

In contrast to other ultrastructural studies, we did not find crystallites in enamel, but rather only in organic matrix. Dentin did reveal spaces with interglobular dentin with unmineralised collagenous matrix but was nearly normal otherwise. Herold et al. [40] assumed that hypoplastic enamel results from incorrect differentiation or degeneration of ameloblasts. Kerebel and Kerebel [46] considered enamel formation of odontodysplasia to probably depend on the following processes: while normally formed enamel would result from an initial normal differentiation of ameloblasts, a decreased number of ameloblasts would lead to hypomineralised enamel. Finally, missing enamel would be associated with a loss of ameloblasts.

Generally, dysplasia of enamel does not seem to have a defined chronology since enamel defects do not run along the growth lines and the thickness varies from normal to completely missing [7, 61, 79, 96]. Rohanizadeh et al. [82] supposed the hypomineralisation of the enamel to be due to errors with the absorption of the enamel matrix by proteases. These could have undergone changes, e.g. by an altered chemistry of the enamel, which was exhibited as a higher Mg/Ca and Na/Ca quotient. Courson et al. [13] showed a possible connection between odontodysplastic changes and metalloproteinases (MMPs) as well as their inhibitors (TIMPs). MMPs are a family of proteases which can split components of the extracellular matrix. Some are able to split collagen or amelogenin. It is assumed that inflammatory processes stimulate the tissue cells to produce more MMPs, whereby resorption of the enamel and dentinal matrix may be induced. Quantitative measurements in one case showed an imbalance between MMP-1 and MMP-2 and the suitable inhibitors TIMP-1 and TIMP-2, so that enzymatic activities cannot be stopped due to a surplus of MMPs. Inclusions of vessels and erythrocytes could refer to haemorrhages, which lead to Mg- and Na-variations of the fluid surrounding the enamel [82]. A further theory is based on hyperaemia, which may lead to entry of mineral-binding-proteins during later stages of the enamel maturing so that incomplete maturing of the crystalline structure may result [82]. Where enamel was missing, Kerebel and Kerebel [46] found no mantle dentin and concluded that due to the loss of ameloblasts, the odontoblasts do not receive induction to form the first dentin layer. Since nearly always large areas of interglobular dentin are to be found under the mantle dentin representing a failure of mineralisation which is associated with the terminal differentiation of the odontoblasts, it was assumed that this is disturbed in RO [96].

General suggestions for therapy of regional odontodysplasia cannot be given, since treatment has to be individual. Sometimes restorative treatment can be done with composite-fillings or crowns. Cases accompanied by inflammation should be treated by extraction [42, 55, 56, 63, 85, 95]. To prevent functional disorders and orthodontic problems, it could be necessary to insert a denture [42, 56, 63, 85]. Two cases with autotransplantation have been published [95, 97].

The etiology of RO remains unknown. None of the mentioned etiologic factors were revealed in the medical histories of our patients. In order to confirm possible etiologic factors, further detailed investigations of cases with RO must be reported.

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