

RADIO “LOGICAL” REFLECTIONS

Of all stripes and colors

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PRESENTING THE CASE

Salomé B., 10 years old, arrives in the office with her parents who want us to take charge of her comprehensive dental care. She is generally healthy, with no special medical history, but is presenting acute pain from a temporary molar (64) and a permanent molar (36) as well as generalized dyschromia.

- The intra-oral exam reveals discolored incisors (brown/gray) and slightly asymmetric open-bite stretching from canine

to canine (fig. 1 and 2). The lateral incisor in the upper jaw on the right side (12) is significantly rotated and tilted buccally, the upper frenum maintains a medial diastema.

- The deciduous teeth are dark brown and the coronal portions of the teeth have been reduced to a few millimeters (fig. 3).
- The tip of a cusp appears to be erupting in buccal ectopia, between the upper temporary molars 54 and 55.

DESCRIPTION OF THE RADIOGRAPHIC RECORDS

It consists of a panoramic view (fig. 4) and a periapical radiograph centered on permanent molars 26 (fig. 5) and 36 (fig. 6a and 6b). We see:

to canine (fig. 1 and 2). The lateral incisor in the upper jaw on the right side (12) is significantly rotated and tilted buccally, the upper frenum maintains a medial diastema.



Figure 1
Frontal intraoral view of the arches.



Figure 2
Intraoral view of the right sector in occlusion.

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Figure 3

Maxillary occlusal view.

Note the morphology and color of the primary teeth.

- A complete dental formula, with patient's dental age corresponding approximately to that of a 10-11 years old, the second mandibular molars have erupted into the arch despite the persistence of almost all the primary teeth in the lateral sector;

- A radiolucent centrimetric lesion that is oval, and well-defined on the apex of 36;
- The crowns look bulbous and squat; roots are short, with almost total obliteration of the pulp chamber (no visibility of pulp chamber or canal);
- The superimposition of the intra-alveolar image of the premolars 14, 15 and the cervical portion of 16;
- The transposition of 13/12; 12 has buccal intrusion in a quasi-horizontal position;
- The intra-alveolar malposition of 24-25 whose crowns are superimposed, and who face each other obliquely, whereas 23 is in a raised position.

Since the deciduous molars were interfering with the eruption of the premolars, they were extracted.

An endodontist specialist (Dr. Hervé Uzan, Paris) began a root canal treatment for 36.

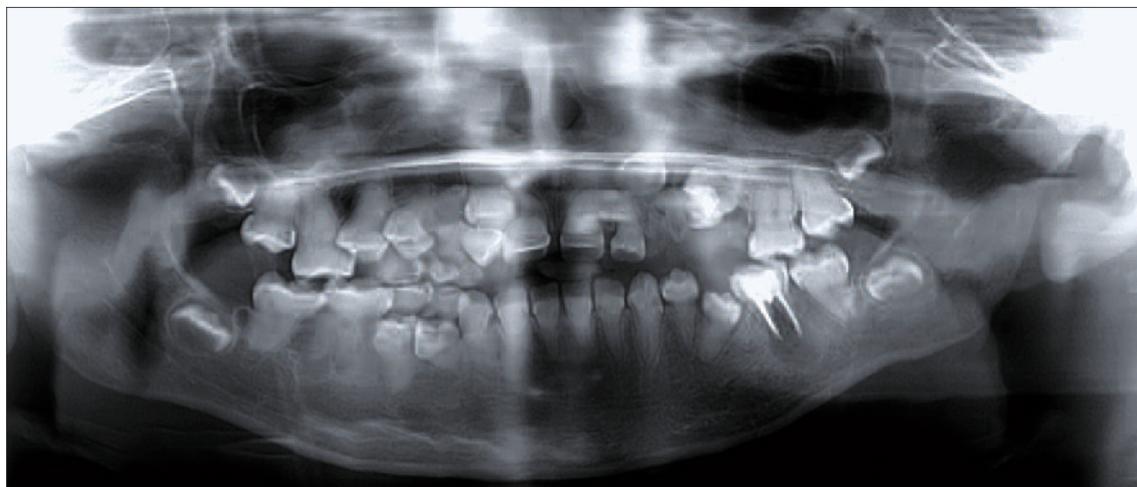


Figure 4

Panoramic view after extraction of 64 and 65.

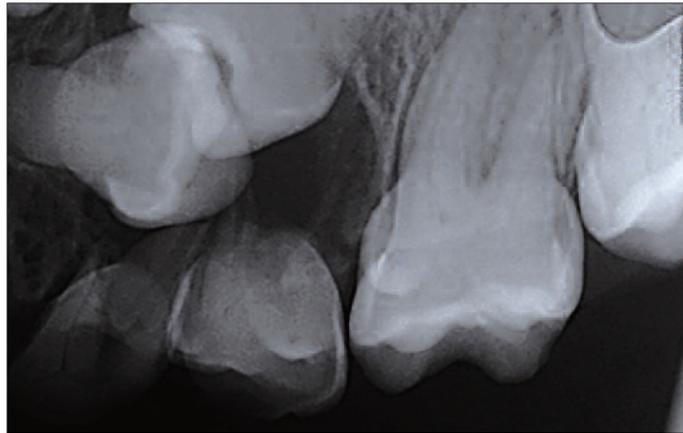


Figure 5
Periapical radiograph centered on 65.

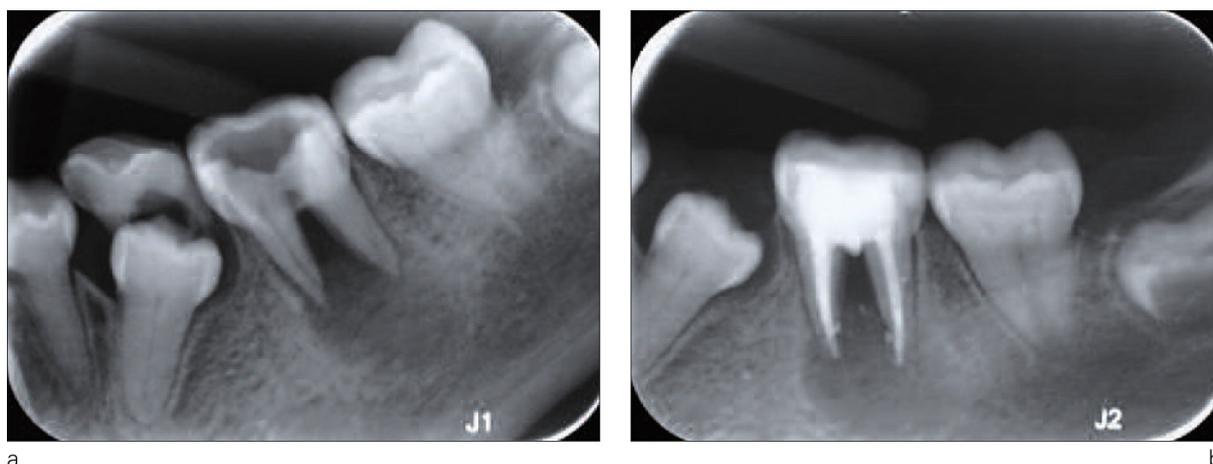
WHAT WAS THE DIAGNOSIS?

Dentinogenesis imperfecta (type II) is a hereditary disorder that is inherited in an autosomal dominant pattern. This structural anomaly attacks both primary and permanent teeth and has a clinically variable expressivity. This disability is rare and affects approximately 1 in 6000 to 8000 people. From a genetic perspective, the genes responsible for dentinogenesis imperfecta type II are linked to the long arm of chromosome 4. Mutations in the gene encoding dentine sialophosphoprotein have been implicated; the products derived from this protein are non-collagen components of the extracellular dentin matrix involved in the initiation and control of the calcification of collagen fibers and also involved in the growth of both osseous and dentinal hydroxyapatite crystals. The undermining of dentinal formation and the disorganization of its calcification process can occur in varying degrees of severity depending

upon the type of mutation. In the case dealt with here, dentinogenesis imperfecta is not associated with osteogenesis imperfecta, and is isolated.

Intra-orally, dental dyschromia varies from blue gray to yellow brown with a characteristic amber “opalescent” color. After the teeth have appeared, the enamel tends to crack away from the dentin that is soft and vulnerable. The crown appears globular and bulbous and pronounced attrition may occur secondarily to the dentinal reduction.

Radiographically, the crowns of the teeth are bulbous, because of constriction at the cemento-enamel junction. The roots are short and dysmorphic. Pulp chamber obliteration is constant: chambers seem to have disappeared and canals are partially or even totally calcified. Periapical lesions are frequent, perhaps as a result of necrosis associated with pulp chamber obliteration and/or bacterial



a

b

Figures 6a and 6b
Periapical views centered on 36 :

- a) before endodontic treatment ;
b) after endodontic treatment and extraction of 75, 35 erupted and straightened spontaneously.

penetration of the enamel through dentinal tubules that open directly into the oral cavity.

Before treating the affected dentition, the endodontist has the difficult task of finding the canal inlets and locating the numerous calcifications that are present. Dentinal hypomineralization affects the resistance of the teeth to dental instruments that can lead to lateral perforations. Chelating

agents are not recommended because the microhardness of dentin is similar to that of cement. The prognosis for endodontic treatment of dentinogenesis imperfecta should be guarded because of the highly irregular and decalcified dentin present.

Routine radiographic screening will be necessary in order to reevaluate the long-term retention of 36.

WHAT STEPS SHOULD WE TAKE AND HOW WILL THEY POSSIBLY AFFECT ORTHODONTIC TREATMENT?

The severity of this dental affliction with its impacted teeth and malocclusion requires, in this case, a multidisciplinary management approach which includes: restorative dental work, follow-up with an endodontist, consultation with a periodontist (about the labial frenum before possibly freeing it), functional re-education of the

tongue (primarily swallowing, or even thumb sucking if there is reason to suspect its involvement in the asymmetry), orthodontics... before possibly installing a prosthetic device.

It is advisable to take a computed tomography (or CbCt) at 6 months, in this case, to evaluate the morphology, the exact interdental relations and

decide the axis of traction of the impacted teeth.

We might also suggest the use of osseous anchorage in order to decrease dental anchorage. Radiographic screening and pulp vitality tests should be routinely scheduled.

The treatment objectives might have to be revised down following this reevaluation.

The parents of Salomé should also be asked to sign a highly detailed informed consent form indicating that they have been informed of the different proposed solutions and risks incurred: risks of coronary fracture, root resorption (short and weak roots), increased risk of periapical lesions or necrosis and, risk of aggravating the dyschromia. Orthodontic movements should be limited and the exerted forces should be applied with particularly light pressure.

The placement of orthodontic brackets on the weak enamel covering of these teeth could be somewhat difficult. In order to simultaneously avoid repeated bracket debonding with accompanying loss of enamel, the anchorage should be strong enough to achieve traction without however, running the risk of causing dental fractures. To achieve this goal, we suggested that the orthodontist etch the enamel with 30% orthophosphoric acid for 30 seconds and bond the attachments with glass ionomer cement amended by adding resin (Cvimar).

Another method involves the application of a hydro-gel adhesive such as Orthosolo[®] (Ormco) and a photopolymerizable composite cement such as Transbond[®] (3M Unitek).

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