

Chapter 4

The Diagnosis of Periodontal Diseases in the Periodontal Clinic, Dental Hospital, University of Indonesia

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Introduction

The Periodontal Clinic is part of the Dental Hospital, Faculty of Dentistry, University of Indonesia. As a public hospital we provide specialist dental treatment as well as manage referrals from other dental services. It is one of the specialist dental care units in Jakarta that provides public services to a population of over 10 million people.

Periodontal treatment in our clinic is performed by students under the supervision of nine staff from the Department of Periodontology. Undergraduate students in the 7th to 10th semesters of their program are responsible for treatment in the clinic. Treatment consists of basic to complex procedures which have been selected for undergraduate or clinical postgraduate students.

The diagnosis of periodontal lesions is determined by clinical and radiographic examination and classified based on the Classification of American Academy of Periodontology International Workshop 1999. Until the year 2002 we referred to the classification of the AAP 1993 to diagnose periodontal lesions, where early onset periodontitis was termed rapidly progressive periodontitis & localized juvenile

periodontitis. However, commencing in 2003 we modified the clinical diagnosis of periodontal lesions to follow the classification of AAP 1999, with some modification based on clinically evidenced conditions.

The average social status of most patients is low to middle class level, predominantly from the low class. Statistically it would appear very difficult to treat periodontal problems in an ideal manner.

Diagnosis of periodontal lesions

To determine the diagnosis of periodontal lesions, clinical examinations and conventional radiographs are utilized. In cases where periodontal problems correlated to systemic diseases or aggressive periodontitis, laboratory tests were performed. The diagnosis and disease recognition are divided into two groups: gingivitis and periodontitis.

We classify periodontal diseases based on a modification of the Classification of the American Academy of Periodontology (AAP) 1999 (Armitage 1999), as follows:

- Gingivitis
 - Puberty gingivitis
 - Gingival overgrowth
 - Necrotizing ulcerative gingivitis

- Chronic periodontitis
 - Localized
 - Generalized
- Aggressive periodontitis
 - Generalized aggressive periodontitis
 - Localized aggressive periodontitis
- Periodontitis as a manifestation of systemic diseases, such as periodontitis associated with Diabetes Mellitus
- Periodontitis associated with endodontic lesions
- Development or acquired deformities and conditions such as occlusal trauma

The periodontal diagnosis should be first determined by the presence of disease; the type, the extent, the distribution, the severity and the pathologic processes (Newman *et al* 2002). With very limited facilities in our clinic, the examination commences with analysis of the case history and identification of clinical signs and symptoms using conventional dental, bitewing and panoramic radiographs. Clinical indications such as, probing pocket depth, tooth mobility, loss of attachment and recession are measured to define the amount of periodontal destruction.

To diagnose periodontal lesions we classify chronic periodontitis as localized and generalized forms, which were formerly known as adult periodontitis or chronic adult periodontitis. It is generally considered to be a slowly progressing disease (Kinane *et al* 2001). We still use the term “generalized form of chronic periodontitis” to refer to slowly progressing periodontitis.

The distribution of periodontal lesions in our clinic is shown in Table 1. The data were

taken from 274 patients in the Periodontal Clinic in the year 2002.

It is shown that the most prevalent form is localized chronic adult periodontitis (61.68 %) a result similar to the US National survey showing that the highest prevalent form of periodontitis in the adult population among individuals 30 years and older was chronic periodontitis (Kinane *et al* 2001).

Based on clinical evidence, the progression of chronic periodontitis can be modified by local factors such as overhanging dental restorations, food impaction caused by tooth form and position, environmental factors such as smoking habits and stress. This is supported by Kinane (1999), stating that the localized form of periodontitis was found to be related to local factors, and site involvement was less than 30% (Kinane 1999). The attachment loss may vary from slight (1 to 2 mm) to moderate (3 to 4 mm) or severe (≥ 5 mm). The amount of destruction was consistent with local factors and supra- and sub-gingival calculus was frequently found. With increased age the distribution of the periodontal disease also increased. This finding is supported by Axelsson (2002), in that the prevalence of periodontal disease is strongly related to age and the susceptibility to periodontitis was found to increase with age (Axelsson 2002).

Referring to the previous classification, that early onset periodontitis includes Rapidly Progressive Periodontitis (RPP), in our experience and observation of clinical evidence we have grouped RPP into 2 categories/types.

Type 1: when patients aged 30–35 years visit the clinic with moderate to severe periodontal inflammation, with some tooth mobility or loss of one or two teeth, with no evidence of other local factors such as malposition, traumatic occlusion, food impaction and with conditions unlikely related to systemic disease

Type 2: when patients aged 20–30 years

Age group	CAP		Aggressive Periodontitis		Juvenile Periodontitis	Periodontitis + DM
	Generalized SPP	Localized	RPP I	RPP II		
15-29	2	37	3	2	1	-
30-44	14	71	8	8	-	1
45-64	56	49	3	-	-	3
≥ 64	4	12	-	-	-	-
	76 (27.74%)	169 (61.68%)	14 (5.11%)	10 (3.65%)	1 (0.46%)	4 (1.46%)

Table 1. Periodontal lesions distribution per age group in Periodontal Clinic, Faculty of Dentistry, University of Indonesia (2002)

visit the clinic with severe periodontal inflammation, generalized bone destruction with severe tooth mobility and significant tooth loss.

In both categories the amount of microbial deposits is usually inconsistent with the disease activity. Due to limited facilities and the high expense in doing so we do not identify the bacterial pathogen. However, in our experience prescribing Amoxicilline and Metronidazole gives a very good response before further treatment is needed.

Several clinical signs such as increase in pocket depth and clinical attachment loss, loss of alveolar crest and the presence of bleeding on probing are important for prognosis of the disease.

By assaying levels of alkaline phosphatase in gingival crevicular fluid (GCF ALP), Dewi Nurul (2003) found that RPP type I and II can be differentiated.

The distribution of localized Juvenile Periodontitis in our clinic is 0.36%, a figure nearly equal to the study in Saudi Arabia where the prevalence of LJP was reported to be 0.42% (Albandar & Rams 2002). We found

however, that if the LJP affected patient was under the age of 30 the onset of localized aggressive periodontitis usually happens around puberty (Hormand 1979).

We classify LJP as an aggressive type of periodontitis which is different from the aggressive periodontitis type I and type II. Clinical evidence showed that the inflammation in LJP patients is less than seen in RPP or aggressive periodontitis. The color and texture of gingiva look normal, but there is increased severity in attachment and alveolar bone loss. Bone destruction is found more around the incisors and first molars than other areas. Occasionally it becomes unclear as to whether the disease is Aggressive Periodontitis or LJP. More recently, diseases with characteristics of LJP have been renamed as localized aggressive periodontitis (Armitage 1999).

The distribution of periodontitis associated with systemic disease is 1.46%. Periodontitis associated with Diabetes Mellitus (DM) is the most common form found in our clinic. To distinguish between generalized aggressive periodontitis and periodontitis associated with

systemic disease such as DM, blood tests have been performed to analyze blood glucose levels. We found that the synergistic effects of plaque accumulation and host response in DM patients may increase periodontal destruction. According to our study of 30 DM type II patients, there is no significant relationship between blood glucose levels and periodontal disease status.

Other studies related to periodontal diseases in our clinic

A requirement for clinical postgraduate education students, besides certain skill capacities, is to carry out studies relating to clinical therapy. Unfortunately, we have very few students in the Periodontics Program compared to other programs such as orthodontics, oral surgery and prosthodontics (Prayitno 2001).

Examples of studies which have been carried out in 2002:

- The analysis of correlation of the Antigen HLA – class I (A, B, C) with Rapidly Progressive Periodontitis type I. This study found that a genetic immune factor had played an important role in the pathogenesis of rapidly progressive periodontitis. It showed a high frequency of distribution of HLA – A9, A11 and A24 in 10 RPP patients.

- The effectiveness of diluted chlorhexidine 0.2 % 1:1 for Gingivitis patients and evaluation of its discoloration of teeth. It was found that diluted CHX 1:1 was effective on gingivitis and there was no significant difference in tooth discoloration between rinsing regimens on seventh days (Rosemlita 2003).

- The clinical effect of subgingival application of metronidazole gel 25% mixture and providone–iodine 10 % as an adjunct to scaling and root planing in chronic periodontitis patients. This study showed a

significant difference in reduction of pocket depth and attachment gain in each group before and after application (Suwandi 2003).

- The effect of irrigation Tetracycline HCl 10 % solution after scaling and root planing in chronic periodontitis patients with pocket depths of 4–6 mm. The results showed that there were significant reductions in probing pocket depth and loss of attachment at test sites compared to control sites (Natalina 2003).

Conclusion

Clinical examination, conventional radiographs and blood tests are standard examinations we performed to determine the diagnosis of periodontal lesions. The most prevalent type periodontal disease in our clinic is Chronic Adult Periodontitis, therefore attention should be paid to prevent the progression of periodontal disease as early as possible which is in line with the government's policies to concentrate on prevention and promotion programs. Based on our clinical evidence we are hesitant to rename LJP as localized aggressive periodontitis. We still subclassify aggressive periodontitis into type I and type II, where in type I the destruction and progression is less than in type II and is related also to age group.

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