OPEN ACCESS Freely Available Online

Dental Hypotheses

Perspective

Reflections on Aggressive Periodontitis as a Disease Entity

Rodrigo López [a], Vibeke Baelum [b]

[a] Department of Periodontology, Faculty of Health Sciences, University of Aarhus, Aarhus, Denmark.

[b] School of Dentistry, Faculty of Health Sciences, University of Aarhus, Aarhus, Denmark.

Correspondence to:

Rodrigo López

Department of Periodontology, Faculty of Health Sciences, University of Aarhus, Vennelyst Boulevard 9, Aarhus C, 8000, Denmark.

Tel: +45 8942 4141

Fax: +45 8613 6550

rlopez@odont.au.dk

Received: May 27, 2010

Accepted: July 5, 2010

Published: July 10, 2010

Abstract

The current periodontitis paradigm embraces the idea of the existence of several particular disease entities. Among them, aggressive periodontitis has been defined as a particular form of periodontitis characterized by severe periodontal destruction usually affecting persons under 30 years of age and presenting with clearly identifiable clinical and laboratory features. Nonetheless, considerable heterogeneity exists in the criteria used to identify the disease; only scant evidence exists substantiating the suggested distinct features; and several voices have expressed concerns on the suggested advantages of considering aggressive periodontitis as a particular disease entity. The main explanation for this impasse can be found in the deep rooted belief that periodontitis is a group of particular clinical entities. This approach to periodontal disease is based on an essentialistic understanding of illness that obstructs advance in periodontal research. The use of a pragmatic nominalistic approach to periodontal diseases would provide a more rational framework for understanding periodontal health and disease and would easy the conduct of periodontal research. The implications of using a pragmatic definition of periodontitis are discussed.

Key words: Aggressive periodontitis; Chronic periodontitis; Diagnosis; Disease; Essentialism; Nominalism; Periodontal diseases; Periodontitis; Syndrome.

Dental Hypotheses_e6

doi:10.5436/j.dehy.2010.1.0006

Background

Since the 1999 Classification of Periodontal Diseases and Conditions it has been common to refer to *"Aggressive Periodontitis"* (AgP) as a particular form of periodontitis characterized by severe patterns of periodontal destruction with *"clearly identifiable clinical and laboratory features"* [1]. However, a closer inspection of the literature reveals considerable heterogeneity in the criteria used to identify cases of AgP [2,3], and evidence for distinct features of the disease is scarce [4-7]. This has recently been highlighted through a series of reviews of the *"similarities and differences between these (Chronic and Aggressive forms of periodontitis, author insert) phenotypic expressions of periodontal diseases"* [8]. The risk factors, whether genetic or environmental, seem to be the same [9,10], just as *"there appears to be no difference between aggressive and chronic periodontitis in terms of their histopathology and immunopathology"* [7,11]. The available preliminary data on the microbiology have led to the expression of contradictory views regarding possible microbial differences between chronic and aggressive periodontitis [5,12,13]. The role of neutrophil functions as contributors to the progression of aggressive and chronic forms of periodontitis *"is still open to debate"* [14]. Solid empirical evidence to assert whether patients with one or another alleged form of periodontitis would benefit from different types of intervention is currently missing [9,15], and concerns have quite rightly been expressed regarding the alleged advantages of diagnosing *"Aggressive Periodontitis"* [3,16]. Armitage et al., recently noted that *"all of the contributors to this volume of Periodontology 2000 [vol. 53 2010, author insert] expressed frustration about the inconsistent use of criteria to define cases of the different forms of periodontitis in the periodontal literature"* [8]. This raises the question how AgP can be considered a particular form of periodontitis when its clinical recognition and differential diagnosis is *"often difficult, if not impossible to make"* [10].

Reflections on aggressive periodontitis as a disease entity

The problem is that Aggressive Periodontitis has never really been properly defined as a distinct entity. What exists is a description, a broad characterization of a typical case [1] as an otherwise clinically healthy person with *"rapid attachment loss and bone destruction"* and familial aggregation of the disease. This description is not very helpful for users of the classification, because concepts such as 'clinical health', 'rapidity' and 'family' may be subject to multiple interpretations. Somewhat ironically, the alleged *"clearly identifiable clinical and laboratory findings"* were used to argue that AgP is sufficiently different from 'Chronic Periodontitis' to warrant a separate classification [1].

Notwithstanding this fundamental problem the current periodontal discourse holds that aggressive periodontitis is a real entity, a demon, or a 'natural thing' that exists over and beyond its defining characteristics and can therefore be diagnosed, treated and studied during scientific anal-

ysis and experimentation. This view reflects the essentialistic thinking [17-19], which is all-pervasive in the biomedical field where disease is usually referred to in demonic terms [18,20], i.e., as *"a thing, a horrid, hateful external thing, which invades the human organism…"* [21].

The demonic or essentialistic disease concept transpires in colloquial language when people talk about 'a disease being transmitted', of being 'attacked by cancer', or of 'having diabetes' [20,21]. The essentialistic mindset develops early in life [22,23] and probably represents normal steps in the development of cognitive reasoning. However, in many instances it remains as the only way people relate to sickness, possibly because it is easy to relate to simple essentialistic statements, which circumvent the difficult question of the defining characteristics of disease.

Unfortunately, essentialistic reasoning leads to misconstructions, with potentially undesirable implications for our attempts to understand health and disease. A person will appear to have AgP only because we project this judgment on them. This objectification of AgP is a product of the conceptualization we have developed for AgP. This is similar to what happens when we see a dog: The reason why we call the observed object 'dog' is our longstanding conceptualization of the specific set of characteristics without which the identified object would not be a dog. Therefore, when we attempt to define and classify periodontitis we should decide which are the characteristics without which the condition would not be periodontitis, or be a specific form of periodontitis.

The common belief that AgP can be dissected and investigated in the same way we investigate plants [24] fails to recognize that AgP is a conceptual expression of a judgment and not a natural thing. The fallacy arises when we confuse the idea of studying something called AgP with attempts to elucidate the concept AgP, which could be done by giving a set of explicit criteria to be fulfilled for the use of the term. This confusion reveals the difficulties in distinguishing empirical research from the investigation of concepts.

The argument that current classification systems for periodontitis originate from scientific evidence is questionable. The information about disease entities frequently accumulates due to the inductive potential of the disease categorizations. We can learn a classification system before we internalize the theory which is supposed to substantiate it [23]. This can make us search for underlying differences between the categories, which may or may not be relevant. However, what is certain is that such observed differences will be used to substantiate the value of the categorization, whereby a vicious cycle of circular reasoning is set in motion. The mere existence of a categorization containing at least two entities will automatically influence the ways in which new knowledge is acquired and interpreted, and this may lead us to generate knowledge of definitions (i.e., periodontal nosology) rather than to generate knowledge about features that are relevant for the conceptuali-

zation of a definition of periodontitis. Believing that in investigating AgP we are drawing information from sources outside our intentions is wrong; we are not drawing information from nature, because definitions of syndromes such as periodontitis [25,26] will necessarily be arbitrary as the signs & symptoms follow a continuum of disease extent and severity without clear-cut and natural thresholds [27-29]. We will not generate progress by making the empirical evidence fit our AgP definition but from making periodontitis definitions fit the empirical evidence available.

At this point it is relevant to ask the question why we should at all bother about periodontal definitions and classifications. The traditional arguments hold that periodontal definitions are useful, capture contemporary understandings of the disease and provide a frame for communication and further inquiries. However, this view underestimates the incompleteness of the data upon which the definitions and classification are based, and hence the volatility of definitions and classifications that are not motivated by distinct practical consequences. The many changes of the periodontal disease classifications over the past few decades amply testify to this volatility and the deleterious consequences of not basing classifications on distinct practical consequences. Our understanding changes because of the acquisition of new knowledge, and our conceptualization of periodontitis therefore also changes. The process cannot go the other way around [30], and no new knowledge can be gained from continuing the seemingly endless cycle of periodontal classifications and reclassifications.

Paralleling essentialistic reasoning, some philosophers consider 'natural kinds' as entities possessing essential properties given by natural law. Cited examples usually include plants and animals [31]. However, Zachar [29,32] discourages the 'natural kinds' approaches to disease definition and suggests the use of disease models based on what he calls 'practical kinds' to represent a non-essentialistic theory of disease. By 'practical kinds' he understands stable constructs justified by their usefulness for specific purposes as opposed to 'natural kinds', which are supposed to be naturally occurring entities defined by essential properties. Whereas 'natural kinds' are supposed to have perfect reliability, 'practical kinds' allow for imperfection and can be understood of as existing on a continuum, with some having higher reliability than others [29,33].

Those who believe in periodontal diseases as 'natural kinds' claim that the goal of the scientist is to isolate the 'real' categories [33]. This kind of essentialistic thinking is scientifically malignant because it promotes stereotyping and inflexible thinking [23,33]. A scientific approach to the study of periodontitis is inconsistent with thinking about it in terms of 'natural kinds' and 'real' entities, such as AgP or Chronic Periodontitis [29]. By definition, practical kinds are human constructs based on pragmatic considerations, such as the need for clear case definitions for the assessment of the effect of clinical

interventions, or for etiological research purposes. In our view, the case definitions should be relevant for both clinical practice and research purposes, since this would ease the translation of the results of etiologic and therapeutic research into the clinic for the benefit of the periodontal patients. Armitage & Cullinan [34] have recently stated, somewhat at variance with earlier statements [35], that the current periodontal classification system describing AgP is *"ill-suited for direct and rigid application to individuals"* and that *"clinicians should avoid rigid application of disease-category definitions of classifications in arriving at a diagnosis"* [34]. When Armitage & Cullinan [34] state that *"arguments among clinicians about whether the patient has chronic or aggressive periodontitis are pointless, especially if the proposed treatment is going to be the same"*, they point to the central problem with the current classification that it has no clinical relevance. Since research efforts regarding periodontitis are all fundamentally motivated in periodontitis being a clinical problem for real patients, we suggest that classification of disease entities should be based primarily on clinical considerations. Otherwise, their only purpose may be for third-party payment purposes to *"fit the diagnosis assigned to a specific patient somewhere into a currently recognized …. classification system"*. The use of the practical kinds approach is intended to be pluralistic and compatible with both the existence of a continuum of disease distribution [27] and the heterogeneity of the disease under consideration [28,29,32]. A practical kind model *"…does not deny that things have internal structures; it only denies that internal structure by itself determines category membership"* [32].

Conclusion

In summary, periodontitis has been historically conceptualized as encompassing a group of different diseases. However, much of the research based on this assumption may inadvertently be biased towards confirmation of the model because the design of the studies has been determined by the assumption. This has obstructed comparisons of descriptive epidemiological studies and hampered etiological research and inference. The fundamental essentialistic mistakes made when defining and classifying periodontitis lie in the confusion of a definition of something with the thing itself [21]; and in the belief that periodontal disease categories are discovered rather than invented [23;29].

List of abbreviations

AgP: Aggressive periodontitis.

Conflicts of interests

The authors declare no conflicts of interest related to this manuscript.

Acknowledgments

The development of this manuscript was partially supported by a grant from the Danish Medical Research Council.

Authors' contributions

- ***Main idea:*** by RL.

- ***Literature search:*** by RL.
- ***Data collection:*** by RL and VB.
- ***Data interpretation:*** by RL and VB.
- ***Manuscript preparation:*** by RL.
- ***Funds Collection:*** by RL.

References

1. Lang N, Bartold PM, Cullinan M, Jeffcoat M, Mombelli A, Murakami S, et al. 1999 International Workshop for a Classification of Periodontal Diseases and Conditions. Papers. Oak Brook, Illinois, October 30-November 2, 1999. Consensus report: Aggressive periodontitis. Ann Periodontol 1999;4:53.
2. Merchant AT, Pitiphat W. Researching periodontitis: challenges and opportunities. J Clin Periodontol 2007;34:1007-15.
3. Meyer J, Lallam-Laroye C, Dridi M. Aggressive periodontitis - what exactly is it? J Clin Periodontol 2004;31:586-7.
4. Loos BG, John RP, Laine ML. Identification of genetic risk factors for periodontitis and possible mechanisms of action. J Clin Periodontol 2005;32 (suppl 6):159-79.
5. Mombelli A, Casagni F, Madianos PN. Can presence or absence of periodontal pathogens distinguish between subjects with chronic and aggressive periodontitis? A systematic review. J Clin Periodontol 2002;29 (Suppl 3):10-21.
6. Lopez R, Frydenberg M, Baelum V. Clinical features of early periodontitis. J Periodontol 2009;80:749-58.
7. Ford PJ, Gamonal J, Seymour GJ. Immunological differences and similarities between chronic periodontitis and aggressive periodontitis. Periodontol 2000 2010;53:111-23.
8. Armitage GC, Cullinan MP, Seymour GJ. Comparative biology of chronic and aggressive periodontitis: introduction. Periodontol 2000 2010;53:7-11.
9. Deas DE, Mealey BL. Response of chronic and aggressive periodontitis to treatment. Periodontol 2000 2010;53:154-66.
10. Stabholz A, Soskolne WA, Shapira L. Genetic and environmental risk factors for chronic periodontitis and aggressive periodontitis. Periodontol 2000 2010;53:138-53.
11. Smith M, Seymour GJ, Cullinan MP. Histopathological features of chronic and aggressive periodontitis. Periodontol 2000 2010;53:45-54.
12. Armitage GC. Comparison of the microbiological features of chronic and aggressive periodontitis. Periodontol 2000 2010;53:70-88.
13. Picolos DK, Lerche-Sehm J, Abron A, Fine JB, Papapanou PN. Infection patterns in chronic and aggressive periodontitis. J Clin Periodontol 2005;32:1055-61.
14. Ryder MI. Comparison of neutrophil functions in aggressive and chronic periodontitis. Periodontol 2000 2010;53:124-37.
15. Adams DA, Barrington EP, Caton J, Genco RJ, Goodman SF, Hildebrand CN, et al. Parameters of care. J Periodontol 2000;71 (Suppl):847-83.
16. Van der Velden U. Diagnosis of periodontitis. J Clin Periodontol 2000;27:960-1.
17. Baelum V, Lopez R. Defining and classifying periodontitis: need for a paradigm shift? Eur J Oral Sci 2003;111:2-6.
18. Wulff HR. What is understood by a disease entity? J R Coll Physicians Lond 1979;13:219-20.
19. Wulff HR, Gøtzsche PC. The disease classification. Rational diagnosis and treatment. Evidence-based clinical decision making. 3rd edition. Oxford: Blackwell Science, 2000: 39-62.
20. Seguin CA. Concept of disease. Psychosom Med 1946;8:252-7.

21. Menninger K. Changing concepts of disease. Ann Intern Med 1948;29:318-25.
22. Gelman SA. Psychological essentialism in children. Trends Cogn Sci 2004;8:404-9.
23. Gelman SA, Coley JD, Gottfried GM. Essentialist belief in children: The acquisition of concepts and theories. In: Hirschfeld LA, Gelman SA, editors. Mapping the mind. 1st edition. New York: Cambridge University Press, 1994; 341-65.
24. Wulff HR, Gøtzsche PC. Rational diagnosis and treatment: Evidence-based clinical decision-making. 3rd edition. Oxford: Blackwell Science, 2000:1-221.
25. Tonetti MS, Mombelli A. Early onset periodontitis. Ann Periodontol 1999;4:39-52.
26. Papapanou PN. Epidemiology and natural history of periodontal disease. In: Lang NP, Karring T, editors. Proceedings of the 1st European Workshop on Periodontology. Quintessence Publishing Co; 1994: 23-41.
27. Lopez R, Fernández O, Jara G, Baelum V. Epidemiology of clinical attachment loss in adolescents. J Periodontol 2001;72:1666-74.
28. Rose G. The strategy of preventive medicine. 1st edition. Oxford: Oxford University Press, 1992:1-381.
29. Zachar P, Kendler KS. Psychiatric disorders: a conceptual taxonomy. Am J Psychiatry 2007;164:557-65.
30. Scadding JG. The clinician and the computer. Lancet 1968;1:139-40.
31. Gelman SA. The essential child. Origins of essentialism in everyday thought. 1st edition. New York: Oxford University Press Inc, 2003:1-382.
32. Zachar P. The practical kinds model as a pragmatist theory of classification. Philos Psychiatry Psychol 2005;9:219-27.
33. Zachar P. Psychiatric disorders are not natural kinds. Philos Psychiatry Psychology 2000;7:168-82.
34. Armitage GC, Cullinan MP. Comparison of the clinical features of chronic and aggressive periodontitis. Periodontol 2000 2010;53:12-27.
35. Armitage GC. Periodontal diagnoses and classification of periodontal diseases. Periodontol 2000 2004;34:9-21.

Citation: López R, Baelum V. Reflections on aggressive periodontitis as a disease entity. Dent Hypotheses 2010; 1:31-38. doi:10.5436/j.dehy.2010.1.0006.