Periodontal disease and leucopenia

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Journal of Small Animal Practice (1995) **36**, 542-546

ABSTRACT

Periodontal disease is rare in nature but widespread in domestic dog and cat populations. Unnatural diets are known to facilitate the build-up of oral microbial communities which then interact with host-immune defences giving rise to periodontitis. Eight of 14 animals undergoing dental treatment and dietary change at a suburban veterinary practice were investigated and found to have low leucocyte counts. Follow-up testing revealed changes averaging a 77·7 per cent increase with concomitant 'subjective good health'. These findings serve to cast doubt on the commonly used haematological reference ranges where the subject animals may have suffered from periodontal disease. The demonstrated reversibility of white cell depression associated with periodontal disease should provide a focus for further research.

INTRODUCTION

Periodontal disease is an inflammatory process involving the supporting structures of the teeth. Whilst plaque organisms are known to be closely involved in the initiation of disease, emphasis is now shifting to investigations of the factors that influence host/plaque interaction (Harvey 1993b).

Removal of plaque, either by physical or chemical means (Sanz and Newman 1994) or by the abrasive forces of feeding, exerts control over the development of periodontal disease. Various authors have commented that the natural diet of wild carnivora has a plaque-retarding effect (Page and Schroeder 1982, Higgins 1987, Harvey 1993a, Watson 1994). The human dentist and comparative periodontist, Sir Frank Colyer (1947) concluded: 'Paradontal disease is always associated with an alteration in the physical or chemical character of the diet of the animal – in other words with a departure from natural diet and conditions.'

There is accumulating evidence of the systemic consequences of periodontal disease – in its simplest view, pain, local to the lesion, is registered in the brain with concomitant lack of appetite and evidence of malaise. Various authors (Beard 1991, Hamlin 1991, Lonsdale 1993a, DeBowes 1994) cite a range of systemic conditions, eg, bacterial endocarditis, glomerulonephritis, polyarthritis and polyvasculitis, believed to be consequences of chronic oral inflammation.

It is thought that systemic conditions may actively exacerbate periodontal disease (Harvey and Emily 1993, Liebana and Castillo 1994). One theory postulates that there is an interactive cascade of events involving a progressive, two-way worsening of periodontal disease and body sytems giving rise to a range of degenerative diseases (Lonsdale 1993a). Due to the reserve capacity of the various organ systems (eg, liver, cardiovascular system and kidneys) it is believed that the interactive spiral decline may remain clinically undetectable until end-stage developments. The same could be said for circulating leucocytes which may exhibit minimal effects on circulating numbers but a profound alteration in the function of the cells (Latimer and Meyer 1989b). Due to a wide reference range, leucocyte numbers can increase or decrease markedly whilst still disguising underlying disease. In the event of reduced circulating numbers this can be explained by a decreased production of the leucocytes, redistribution between the circulating and marginal pool and emigration into the tissues (Latimer and Meyer 1989a).

This paper details the outcome of eight cases presented to a suburban clinic showing advanced periodontal disease and reduced leucocyte and

erythrocyte numbers. Surgical treatment of the presenting condition and change of diet resulted in a marked increase in circulating leucocytes with a return to 'subjective good health'.

MATERIALS AND METHODS

Eight animals presented at a suburban practice for a number of reasons were selected. These comprised six dogs and two cats. In each instance clinical examination revealed advanced stage periodontal disease. Treatment consisted of radical dentistry including removal of diseased teeth, and the institution of a five-day course of amoxycillin (Amoxil; SmithKline Beecham Animal Health) or clindamycin (Antirobe; Upjohn).

From day 1 after surgery the animals were fed a raw meaty bone diet with occasional supplemental table scraps. The small dogs and cats received chicken wings, rabbit legs and whole raw fish whilst the larger dogs received lamb flaps, ox brisket, kangaroo tails, etc. The size of pieces was important as a regulator of chewing function. Pieces too small would permit swallowing whole with the risk of obstruction. Large bones without meat have lesser nutrient value whilst risking the wear or breakage of teeth. At the time of dental treatment, five of the cases had full blood counts performed; of the remaining three cases a white cell count plus differential cell count were performed. Blood tests were repeated at the time of the animals' check up.

RESULTS

The results of the present study are summarised in Table 1.

Cases 1 and 2, a seven-year-old male and nine-year-old female silky terrier, were assessed first. During the previous six years the dogs had been presented to the surgery on numerous occasions with non-specific illness, lethargy or dermatitis.

Table 1. Clinical details and haematology profiles for eight animals undergoing radical dental treatment and diet change

Case	1	2	3	4	5	6	7	8
Breed	Silky terrier	Silky terrier	Terrier cross	Chihuahua	DLH	Golden retriever cross	DSH	Maltese terrier
Age (years)	10	12	11	8	7	13	12	12
Sex	M(E)	F(S)	F(S)	M(E)	F(S)	F(S)	F(S)	F(E)
Presenting complaint/signs	Routine vaccination	Routine vaccination	Routine vaccination	Routine vaccination	Prolapsed third eyelids, temperature 39°C	Halitosis, lipoma, arthritis, fleas	Inappetence, lethargy, dehydration	Lame right hind leg, severe heart murmur, mammary lump, dermatitis, otitis, fleas
Treatment	Radical dentistry, flea treatment, dietary change	Radical dentistry, flea treatment, dietary change	GA, multiple extractions, dietary change	Dentistry, blood tests, dietary change	Liquid paraffin and dentistry, dietary change	Dentistry, lipoma removal, dietary change	Dentistry, dietary change	Dentistry, ear and flea treatment, dietary change
Blood results at presentation	12.8.91	12.8.91	12.1.93	5.2.93	27.12.92	19.12.92	27.11.92	28.10.92
RCC ($\times 10^{12}$/litre)	6·0	5·6	5·27	6·03	Not done	6·34	7·16	7·21
WBC ($\times 10^{9}$/litre)	5·1	5·2	4·9	5·7	2·6	7·0	6·8	6·8
Neutrophils ($\times 10^{9}$/litre)	3·4	4·0	3·8	4·0	1·0	4·6	4·8	4·9
Lymphocytes ($\times 10^{9}$/litre)	1·5	0·8	1·0	1·3	1·4	1·4	1·2	1·0
Monocytes ($\times 10^{9}$/litre)	0·1	0·2	0·1	0·3	0·1	0·4	0·1	0·5
Eosinophils ($\times 10^{9}$/litre)	0·2	0·3	0·0	0·1	0·2	0·5	0·7	0·3
Repeat blood results	17.6.92	17.6.92	6.3.93	11.3.93	17.3.93	13.1.93	12.1.93	17.12.92
RCC ($\times 10^{12}$/litre)	7·1	7·4	7·68	8·83	9·2	Not done	7·29	6·98
WBC ($\times 10^{9}$/litre)	8·5	8·2	7·8	8·0	6·5	14·4	9·3	14·0
Neutrophils ($\times 10^{9}$/litre)	4·8	5·9	5·0	4·1	2·7	11·7	7·4	11·1
Lymphocytes ($\times 10^{9}$/litre)	2·7	1·7	2·2	2·2	3·3	2·0	1·3	1·3
Monocytes ($\times 10^{9}$/litre)	0·4	0·4	0·6	0·6	0·1	0·3	0·2	0·4
Eosinophils ($\times 10^{9}$/litre)	0·5	0·2	0·0	1·1	0·4	0·4	0·4	1·3
% change								
RCC ($\times 10^{12}$/litre)	18	32	46	46	NA	NA	2	-3
WBC ($\times 10^{9}$/litre)	67	58	59	40	150	106	37	105
Neutrophils ($\times 10^{9}$/litre)	40	47	31	2	170	154	54	126
Lymphocytes ($\times 10^{9}$/litre)	80	112	120	69	136	42	8	30

DLH Domestic longhaired cat, DSH Domestic shorhaired cat, M(E) Entire male, F(S) Neutered female, F(E) Entire female, GA General anaesthesia, RCC Red cell count, WBC White blood cell count, NA Not applicable

A recurrent complaint was that the male had 'anxiety attacks'. Several theories and treatments had been propounded by different clinicians in the practice but all to no avail. At the time of the animals' annual vaccinations the owners were not aware of any unusual features concerning their pets, but a clinical examination revealed a sparse hair coat and marked generalised periodontitis in both animals. Up until that time dietary advice had been to feed dry dog food, table scraps and the occasional raw bone. The raw bone component had been progressively overlooked by the owners and their pets' halitosis had been accepted as normal.

The owners were persuaded that radical dentistry involving tooth extractions followed by a change of diet to raw meaty bones would benefit their pets. In an attempt to obtain objective information about the animals, a haemogram was performed. The dogs were discharged postoperatively with a five-day course of amoxycillin and instructions for the feeding of raw meaty bones.

After 10 months the owners reported how delighted they were with the change in health and wellbeing of their animals. The initial change of diet had created some difficulties but a regimen had become well established. Clinical assessment revealed a much-reduced halitosis, although there was evidence of calculus on those teeth which lacked an opposing member in the opposite jaw. The hair coat of both animals was generally more lustrous and the non-specific dermatitis of past years was absent. Comparison blood tests were commissioned and are recorded in Table 1.

The percentage change in cell counts in these two cases prompted further investigation in other cases. Blood tests were undertaken in the next 12 cases presenting at the clinic for radical dentistry. In six cases the cell counts were considered low or at the low end of 'normal' and in these cases the owners were asked to bring their pets in for follow-up screening at a later date. In each instance treatment involved radical dentistry with teeth scaling and, or, extractions together with a postoperative course of antibiotics and a change in diet.

Owner assessment of all the test animals was of a markedly increased vitality and wellbeing; four of the cases (1,2,3 and 4) were only considered to be healthier in hindsight. These cases had been presented for routine vaccination by their owners who were unaware that their animals were unwell.

Fuller clinical findings were recorded in case 8, a 12-year-old female Maltese terrier. The owner's presenting complaint was of a sudden onset lameness of the right hind leg. Clinical examination revealed a generalised dermatitis, bilateral otitis, flea infestation, pansystolic murmur, mammary lump, marked dehydration and severe oral disease. The weight was recorded as 3·8 kg. Postoperative clindamycin was prescribed together with eardrops and a splint applied to the presumed sprained hock. The owners were asked to institute a raw chicken wing diet immediately with the addition of a few table scraps from time to time commencing one week later. Although the majority of teeth had been removed from the maxilla and pre-maxilla, case 8's bodyweight had increased to 4·55 kg within 50 days.

Table 2. Normal values utilised by Macquarie Vetnostics Services (MVS), Veterinary Pathology Services (VPS) and Jacobs and others 1992 (Guelph)

	MVS		VPS		Guelph	
	Dogs	Cats	Dogs	Cats	Dogs	Cats
RCC ($\times 10^{12}$/litre)	5·0-8·0	5·5-10·0	5·5-8·5	5·5-10·0	5·6-8·5	5·0-10·0
WBC ($\times 10^{9}$/litre)	6·0-14·0	6·0-16·0	6·0-17·0	5·5-19·0	6·1-17·4	5·5-15·4
Neutrophils ($\times 10^{9}$/litre)	4·1-9·4	3·8-10·1	4·0-12·0	2·0-13·0	3·9-12·0	2·5-12·5
Lymphocytes ($\times 10^{9}$/litre)	0·9-3·6	1·6-7·0	0·9-5·0	0·9-7·0	0·8-3·6	1·5-7·0
Monocytes ($\times 10^{9}$/litre)	0·2-1·0	0·1-0·6	0·1-0·6	0·1-0·4	0·1-1·8	0·0-0·85
Eosinophils ($\times 10^{9}$/litre)	0·1-1·2	0·2-1·4	0·1-0·5	0·1-0·8	0·0-1·9	0·0-0·75

RCC Red cell count, WBC White blood cell count

Case 8's red cell count was found to have decreased by 3 per cent. However, it should be borne in mind that the initial reading was taken from a markedly underweight, dehydrated animal. By the second reading the animal's bodyweight had increased by 20 per cent and as such it can be assumed that the total circulating erythrocytes would have increased significantly.

DISCUSSION

The eight cases studied provided a strong subjective correlation between increase in health and increase in blood cell counts – this result being demonstrated by a simple percentage change. Whilst correlation does not imply causation, the fact that all blood cell counts moved in the same direction (with one small exception) provides persuasive evidence that either the periodontal disease or diet or both were continuing to influence the overall health of the patients.

Dental treatment and dietary change represent generic concepts that incorporate numerous elements in numerous interrelationships. In the light of the known presence of endotoxins and exotoxins and the immune interaction at the gingival sulcus (Liebana and Castillo 1994), these factors might be considered in the lowering of white cell counts. Cooked, macerated processed food has no cleansing effect on teeth and gingiva,

so can be expected to potentiate periodontal disease. Page and Schroeder (1982) state that in order to produce periodontal disease 'somewhat extreme measures such as placement of ligatures in a subgingival position, mono-infection of the oral cavity, or feeding of grossly abnormal diets are required'. Adverse reactions to ingesta are well known and can affect many systems (Wills and Harvey 1994) which, in turn, can be expected to directly or indirectly affect white cell counts.

This study made no attempt to provide a qualitative assessment of the circulating red and white blood cells. It should not be overlooked that the health of an immune system involves a balance of number and function of immune components. Valid concerns arise that dogs and cats suffering from severe periodontal disease can show cell counts within the standard reference range. In our study, four animals were alleged to be normal by their owners but then showed average red blood cell increases of 35 per cent and white blood cell increases of 55 per cent following dentistry. Since these increases coincided with an increase in the apparent wellbeing of the animals then it would be reasonable to assume that the higher values were closer to the true 'normals' for the subject animals.

It is recognised that the use of reference values is in itself a controversial topic (Lumsden and Mullen 1978). Previously, debate has centred on the statistical treatment of results and the method of population selection for the gathering of data. To the author's knowledge consideration as to periodontal health and diet of subject groups has not been well considered. Details were obtained of three published reference ranges from two large commercial pathology laboratories operating in NSW, Australia. Their data was obtained from animals presented to suburban veterinary clinics and variously assumed to be healthy by their owners and or veterinarians (Macquarie Vetnostics Services 1986, Veterinary Pathology Services 1989 to 1991). Questions regarding unnatural artificial diets were seldom raised and the appreciation of periodontal disease problems of domestic pets was at that time in its infancy. The reference ranges published by Jacobs and others were derived from the University of Guelph between 1989 and 1991. These were colony dogs and cats fed a named commercial diet, it is widely reported that colony dogs and cats suffer from periodontal disease (Brown and Park 1968). It is for these reasons that the reference ranges of laboratories may be suffering distortion.

In a broader context, reference ranges represent an establishment of values from a 'normal control group'. If these values can be so sharply affected, then one must have concerns for the validity of any control group where the subject animals can suffer periodontal disease and at the same time be fed processed food (AAFCO 1993). This sentiment was expressed in another way by the authors of the diet and periodontal disease literature search in the Australian Veterinary Association News: 'Those investigating small animal health problems should also take diet and diet consistency into account when researching systemic diseases – possible confounding effects of diet and poor oral health must be considered in such studies.' (Australian Veterinary Association News 1994).

In our general practice, no further comparative blood testing is performed due to the inconvenience suffered by clients and the costs involved. Standard care does involve, regardless of presenting conditions, the provision of dental care and advice to return to a natural diet (Lonsdale 1993b). The results more than justify our confidence with the rapid resolution and reduction in recurrence of medical complaints.

The research potential of this area remains enormous. Extensive trials can be performed to examine the importance of periodontal disease and diet in both the initiation and maintenance of ill health. The standard range of laboratory parameters can be re-examined as can other less familiar haematological and biochemical systems. The apparent reversibility of immune cell depression in carnivores by correcting periodontal disease and diet suggests a potential model in which to explore immune deficiency syndromes.

ACKNOWLEDGEMENTS

This study depended on long-suffering patients, understanding owners and supportive colleagues. Drs Bruce Duff and David Snow of Macquarie Vetnostics, Sydney, kindly donated the laboratory testing. Susan Rutter processed the various drafts.

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BOOK REVIEW

Manual of Small Animal Diagnostic Imaging. Edited by R. Lee. Published by the British Small Animal Veterinary Association, Cheltenham. Paperback. Price £29.00 (members), £39.50 (non-members), £3.75 postage and packing. 200 pages. 1995.

THE renaming of this manual reflects the rapid evolution of veterinary imaging. These days, as well as conventional radiography many practitioners will be utilising ultrasound; they will also be familiar with what computed tomography (CT) and magnetic resonance imaging (MRI) can offer their patients. It is therefore somewhat disappointing that the changes from the 1989 edition of 'Radiography and Radiology in Small Animal Practice' are more of format than content.

While the introduction does indeed include a brief description of CT and MRI, these imaging techniques are not mentioned in any of the subsequent chapters even when relevant to the structures or conditions being described. Indeed these chapters have had little or no revision from the previous edition. The main change is that the layout of the text has been altered to a two-column format. This is justified as when it is combined with the line diagrams being limited to the width of a column, it does seem to improve the assimilation of the subject matter.

The addition of a chapter on diagnostic ultrasound is welcome but suffers from having only limited accompanying illustrations.

Probably the most significant observation that can be made about this manual of diagnostic imaging is that it contains no radiographs or ultrasonographs. Other BSAVA titles now routinely include numerous radiographs and their absence from this manual does seem rather incongruous. The accompanying line diagrams are of high quality and certainly are more than adequate for the positioning diagrams. However I feel their usual role in this sort of publication should be to aid the readers interpretation of the accompanying radiographs rather than a stylised representation of a lesion or condition.

My worry is that a neophyte in veterinary imaging will sometimes struggle to relate the written descriptions and line diagrams to the images obtained by themselves without supporting high quality radiographic and ultrasonographic illustrations.

This manual does contain a wealth of information on imaging from knowledgeable authors in the field. However, I feel that a more comprehensive revision of the text and illustrations is required before one could justify purchasing it to replace the previous edition.

GRAHAM PECK