FOOD INDUCED CHRONIC HEPATIC DISEASE

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The role of foods as causative agents of disease is becoming increasingly more common. It is apparent that food induced diseases are being diagnosed more accurately and frequently.

We define the term Chronic Hepatic Disease (CHD) as a chronic liver disease which, if undiagnosed and not treated, persists for four months or longer, and which may, after an extended period, terminate in liver destruction and morphological disorganization. Due to the insidious nature of CHD it may manifest itself in many different forms and have varied etiology. This article is concerned with CHD due to food antigen hypersensitivity.

The disease most typically reflects a chronically elevated SGPT level. The SGOT level may be elevated, depending on the chronicity of the disease. Alkaline phosphatase levels are often elevated. The white blood cell count ranges from normal to low normal. There may be an elevated peripheral eosinophil count. Often, long term steroid therapy may be effective, while long term antibiotic therapy tends to be ineffective. A multisystem disorder may ensue if the disease continues undiagnosed for long periods of time. Transitional clotting defect, chronic pancreatitis, chronic nephritis, epilepsy or encephalitis-like syndromes may accompany food induced CHD.

Those suspected cases of food induced CHD were placed on a non-meat diet.[1] Significant blood tests (white blood cell count, eosinophil count, SGPT, SGOT and alkaline phosphatase) were performed at weekly intervals. Once diagnosis and treatment had been completed, the patients were re-exposed to their particular offending food allergens. Once again the CHD panel was performed. Reactivation of CHD ensued, as exemplified by elevated hepato-blood chemistry levels.

Case Report No. 1:

A male, six-year-old Afghan was referred to the hospital for a chronic liver, chronic kidney disorder. Over the past three years the patient suffered from CHD, sporadic proteinuria and sporadic hematuria. Lab tests were performed with the following results: eosinophil count – 19%, white blood cell count – 8,800, SGPT level – 780 I.U., SGOT level – 102 I.U. and an alkaline phosphatase level of 1015 I.U. All other tests were normal. The patient had a history of flatulence and hyperperistalsis. At this time

the patient was placed on bottled water and non-meat diet.[2] The first week the hyperperistalsis and flatulence disappeared. One month later all the patient's blood levels were normal.

Lamb and chicken were each added daily for 7 day periods without recurrence of the clinical disease. Beef, wheat and corn antigens all elicited exacerbation of CHD.

Case Report No. 2:

A three-year-old, female red Doberman Pinscher was referred to the hospital with chronic dermatitis, otitis and weight loss. A total body function was performed at that time; the SGPT level was 246 I.U. All other results were normal. The patient was then placed on a diet of lamb, rice and bottled water. After two weeks on this diet the SGPT level returned to normal. Within this same period of time the patient gained four pounds and the dermatitis and otitis were greatly diminished. Interestingly enough, eleven months later an exacerbation of the original clinical signs occurred. Blood levels indicated that the patient was now reactant to lamb, with a consequential increase in the SGPT level. The patient was then placed on a non-meat diet and the SGPT level returned to normal within two weeks.

Case Report No. 3:

A 2½-year-old, male Springer Spaniel entered the hospital with a tense abdomen, gastro-enteritis and general malaise. Total body function reflected SGOT level of 102 I.U. and a SGPT level of 143 I.U. All other tests were normal. The patient was placed on lamb, rice and bottled water. Within two weeks all blood levels returned to normal. The patient, however, was losing weight on a proportional amount of this new diet. Increasing the volume of food did nothing to arrest this weight loss. Further tests, including fecal trypsin and parasites, revealed no abnormalities. The patient was taken off lamb and rice and placed on non-meat diet. Within one week the patient gained two pounds and has proceeded to normal weight maintenance.

Case Report No. 4:

A ten-year-old, male Manchester Terrier was referred to the hospital with CHD. For the last eight years of the patient's life he was unable to tolerate dry food products. His diet consisted of Kal Kan MPS Chunks. On this diet he had relatively soft, and sometimes chronically loose stools. Total body function revealed a SGPT level of 166 I.U., SGOT level of 102 I.U. and an alkaline phosphatase level of 300 I.U. The patient was placed on lamb, rice and bottled water; stools immediately became normal.

Case Report No. 5:

We examined a female eleven-year-old Standard Poodle with a history of chronic diarrhea and vomiting. The patient's blood tests revealed no abnormalities. Fecal analysis also revealed no abnormalities. The patient was placed on I/D at this time, and clinical disease disappeared. Upon re-exposure to beef antigen in the form of fresh meat, canned beef and/or kibble, exacerbation of clinical signs occurred. The patient remained normal on I/D for three years. At the end of this period chronic gastro-enteritis occurred. Blood and fecal analysis revealed no abnormalities. The patient was then placed on a less allergenic diet. The new diet, lamb and rice, brought about remission of the clinical disease.

This normal state remained for four months, at which time the clinical disease again recurred. At this time blood analysis revealed a SGOT level of 102 I.U., a SGPT level of 780 I.U. and an alkaline phosphatase level of 399 I.U. The patient was switched to non-meat diet and blood levels returned to normal, with no reactivation of clinical disease.

SUMMARY

It is interesting to consider the effects of food mediated disorders. We have found substantial evidence that food antigens can cause severe clinical disease. It has been well documented that the liver retains antigen for long periods of time.[3] This may account for the extended period for total remission after non-allergenic diet is substituted.

Since beef appears to be the most antigenic of meats, and lamb the least, it is interesting to note the incidence of hyper-reactivity to lamb antigen after a given period of exposure. With modern day pet foods having multiple antigens, preservatives and other modifying agents, it has been extremely difficult to isolate any one or more allergens from these diets.

We have developed a less allergenic diet as a means of incriminating food in food-induced hypersensitivity disorders. This tends to be a more dependable diet than lamb and rice, and it appears to decrease the possibility of future food hypersensitivity.

Apparently there are a number of etiological agents in CHD. We feel that one segment of CHD is food induced.

References

1. Now produced by Breeder's Choice: Pet's — Naturally Yours.
2. For diet formulation and a complete discussion of Food Induced Hypersensitivities see *Modern Veterinary Practice* 3/77.
3. John J. Miller, III, "Localization and Fate of Foreign Antigens in Tissues." *Immunological Diseases*, 2nd Ed. 1 (Boston: Little, Brown and Co., 1971) pp. 24-25.