

Effect of dietary restrictions on disease activity in rheumatoid arthritis

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SUMMARY Additions in five steps were made, as a possible therapeutic measure, to the diet of 27 patients with rheumatoid arthritis (RA) after a period of two weeks of a basal isocaloric diet free from pulses, cereals, milk, and non-vegetarian protein foods. Fourteen patients finally took part in the trial, 10 (71%) of whom showed significant clinical improvement. Only three patients (11%) adhered to the diet for a period of 10 months. The others discontinued the diet and were then treated with conventional disease modifying drugs. The study indicates that dietary factors may influence inflammatory response in RA.

Key phrases: food intolerance in RA, individual susceptibility to food in RA, dietary manipulation in RA, response assessment in RA.

Recently there has been a resurgence of interest in the relation between diet and RA.¹⁻³ Some reports have shown definite and objective correlation between certain dietary factors and the inflammation of RA.⁴⁻⁸ The present study was undertaken to analyse systematically the effect of various dietary components on the inflammatory activity of this disease.

Patients and methods

PATIENTS

Twenty seven patients with definite RA⁹ were included in the study. None of the patients had previously received any disease modifying anti-rheumatoid drugs.

DIET

The details of the study were explained to the patients and their informed consent obtained before the study began. Before the start of the dietary elimination therapy, patients who were receiving non-steroidal anti-inflammatory drugs (NSAIDs) began a wash out period of two weeks. During this time patients were allowed a normal diet and 500 mg of paracetamol as required for pain relief.

A complete clinical and laboratory assessment was carried out at the beginning and at the end of the wash out period. Patients not requiring a wash out period were immediately placed on the dietary therapy after the initial assessment.

For the first two weeks of the dietary therapy, patients were allowed an isocaloric diet consisting of fruit, vegetables, sugar, and refined oil (diet I). This was followed by a repeat estimation of the erythrocyte sedimentation rate (ESR) (Westergren) and clinical variables, after which the patient was given diet II, consisting of diet I plus all pulses, for another period of two weeks. The clinical and laboratory assessments were repeated as before. Two further diets, diet IIIA, consisting of diet I plus wheat and wheat products, and diet IIIB, including rice and rice products in addition to diet I, were again given for a period of two weeks each and were each followed by assessments. These diets were followed by stepwise two weekly additions of milk and milk products (diet IV) and non-vegetarian food (diet V), consisting of eggs, meat, fish, and chicken, with regular clinical and laboratory estimations as before.

If any increase in symptoms was observed after a particular diet the patients were returned to diet I until improvement was observed before attempting the next diet. Thus groups of foods causing exacerbation of the disease activity were determined and eliminated from the diet, with long term

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assessment every three months. During the entire period of dietary therapy patients were allowed to take paracetamol tablets if required.

The criteria for clinical assessment included morning stiffness, joint pain score, articular index, and patients' global assessment. Morning stiffness was assessed arbitrarily, i.e., grade I=15 min, grade II=up to 1 hour, grade III=up to 2½ hours, grade IV=2½ hours or more. Joint pain score was marked according to the criteria of Deodhar *et al.*,¹⁰ and the articular index was determined by Ritchie's method.¹¹ Patients' global assessment was graded as follows: grade I=much better, grade II=better, grade III=unchanged, grade IV=worse, and grade V=much worse. Initial laboratory estimations included haemoglobin, total leucocyte estimation, and ESR by standard methods. Latex fixation titre (Hoechst (India) Pvt Ltd) was also estimated at the beginning of the trial. The ESR was repeated every two weeks, i.e., at the initiation/completion of each diet step.

A record of the patient's weight was also maintained in order to ensure that no excessive weight loss took place (i.e., >5%).

Results

In the present study only 14 (52%) of the 27 patients could take the diet as advised, the rest being unable to complete the first step.

Of the 14 patients who carried out the dietary instructions, 10 (71%) showed significant clinical improvement. The remaining four patients did not show any benefit from the first and second diets and were changed to standard drug therapy.

Of the 10 responsive patients, one responded to

elimination of pulses alone, four to the elimination of both the cereals (wheat and rice), and two to the exclusion of rice and rice products. For the remaining three patients improvement was observed only on the elimination of multiple food items, i.e., pulses, wheat and milk, milk and non-vegetarian food, and pulses and non-vegetarian food.

The pattern of response for these 10 patients was as follows: after diet I these patients showed a mean percentage improvement in the clinical variables ranging from 25 to 54%, as compared with their baseline levels, while improvement in their ESR was 33%. Introduction of diet II led to deterioration of the condition of three patients (Table 1), one of whom showed a rapid flare of symptoms. The remaining two patients complained of exacerbation of their disease, particularly with one pulse (Bengal Gram). The other seven patients showed clinical improvement and a mean percentage improvement of 45% in their ESR. One patient dropped out of the trial at this stage and could not be contacted again.

Diet III (A and B) led to significant deterioration in the condition of the remaining six patients, of whom four worsened with both the cereals and two with rice and rice products (Tables 1 and 2). Drug therapy was initiated for the first group as it was difficult to prevent their consuming cereals entirely. Four patients entered diet IV, of whom two deteriorated and two improved. Two patients reached the last stage, i.e., diet V, and both deteriorated.

Considerable weight loss (9%) was observed in five patients, but there was no difference between the weight loss of good and poor responders.

At the time of reporting, three patients (11%)

Table 1 Variation of the erythrocyte sedimentation rate (ESR) in response to various food groups

Food groups	Serial No of susceptible patients	ESR (mm/1st h)		
		Baseline	Elimination	Challenge
Pulses	1	84	22	60
	2	78	55	45
	3	28	16	16
Wheat and rice	1	80	45	80
	2	88	67	107
	3	82	40	72
	4	66	25	45
Wheat	1	78	45	60
Rice	1	56	15	51
	2	73	30	41
Milk	1	78	45	60
	2	43	8	15
Non-vegetarian food	1	28	8	14
	2	43	8	15

Table 2 Variation of the global assessment in response to various food groups

Food groups	Serial No of susceptible patients	Global assessment		
		Baseline	Elimination	Challenge
Pulses	1	4	1	4
	2	4	2	4
	3	4	1	4
Wheat and rice	1	4	1	3
	2	4	1	3
	3	2	1	1
	4	3	1	4
Wheat	1	4	1	3
Rice	1	4	1	4
	2	4	2	2
Milk	1	4	1	3
	2	3	1	4
Non-vegetarian food	1	4	1	4
	2	3	1	4

were still adhering to dietary restrictions and continue to do well without taking any drugs.

Discussion

Patients with rheumatoid arthritis frequently complain of an increase in their disease activity on consuming certain food items.^{1 2} The role of food as a cause of inflammation in rheumatoid arthritis is still controversial, however. A relation between ingested food and fluctuation in disease activity has yet to be determined unequivocally.

Most studies are based on dietary elimination, i.e., a certain food item is eliminated, a challenge is then made with the same dietary item, and observations are recorded.^{1 2 4} With this method it is usually difficult to establish multiple food 'allergies' in a given patient. In the present study, therefore, the method consisted of 'diet additions' after a period of wash out, rather than elimination. Although this method was very cumbersome, one of the main advantages was that the responders could be identified immediately. The results showed that a high proportion of patients improved on dietary manipulations, and that there was marked individual variation in response to the elimination of different dietary items. Some patients improved on elimination of only one of the food items, whereas others were susceptible to more than one dietary component. The study also highlighted the practical difficulty involved in instituting dietary treatment as a major mode of treatment in RA as the follow up was difficult and the drop out rate was high. The present work indicates, however, that individual

susceptibility (allergy?, unusual immune response?) may be an important area for future investigations of the pathogenesis of RA.

One possible mode of action of dietary therapy in RA has been considered to be non-specific suppression of inflammation by suppression of synthesis of proinflammatory prostaglandins.¹² An alternative possibility could be abnormal handling of the food antigens by the host. A possible future line of work may be to identify the food allergies (intolerance?) in patients with RA and to try to correlate their humoral and cell mediated immune response against these food antigens in the blood and joint fluid.

As mentioned earlier the dietary addition procedure used in this study was complicated and cumbersome, yet, in contrast with the usual elimination-challenge methods had the advantage that the responders could be identified immediately. Possibly an easier approach would be to give a basal isocaloric diet free from all the major food groups followed by a rotation diet with elimination alternating with challenge at shorter intervals. Such a study is in progress.

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References

- 1 Panush R S, Carter R L, Katz P, Kowsari B, Longley S, Finnie S. Diet therapy for rheumatoid arthritis. *Arthritis Rheum* 1983; **26**: 462-71.
- 2 Panush R S, Stround R M, Webster E M. Food-induced (allergic) arthritis. *Arthritis Rheum* 1986; **29**: 220-6.

- 3 Ziff M. Diet in the treatment of rheumatoid arthritis. *Arthritis Rheum* 1983; **26**: 457-61.
- 4 Darlington L G, Ramsey N W, Mansfield J R. Placebo-controlled, blind study of dietary manipulation therapy in rheumatoid arthritis. *Lancet* 1986; **i**: 236-8.
- 5 Sköldstam L, Larson L, Lindström F D. Effect of fasting and lactovegetarian diet on rheumatoid arthritis. *Scand J Rheumatol* 1979; **8**: 249-56.
- 6 Lucus C P, Power L. Dietary fat aggravates active rheumatoid arthritis [Abstract]. *Clin Res* 1981; **29**: 754A.
- 7 Parke A L, Hughes G R V. Rheumatoid arthritis and food: a case study. *Br Med J* 1981; **282**: 2027-9.
- 8 Sundqvist T, Lindström F, Magnusson K E, Sköldstam L, Stjernström I, Tagesson C. Influence of fasting on intestinal permeability and disease activity in patients with rheumatoid arthritis. *Scand J Rheumatol* 1982; **11**: 33-8.
- 9 Ropes M W, Bennett G A, Cobb S, Jacox R, Jessar R A. Revision of diagnostic criteria for rheumatoid arthritis. *Bull Rheum Dis* 1958; **9**: 175-6.
- 10 Deodhar S D, Dick W C, Hodgkinson R, Buchanan W W. Measurement of clinical response to anti-inflammatory drug therapy in rheumatoid arthritis. *Q J Med* 1973; **42**: 387-401.
- 11 Ritchie D M. Clinical studies with articular index for the assessment of joint tenderness in patients with rheumatoid arthritis. *Q J Med* 1968; **37**: 393-406.
- 12 Kremer J M, Michalek A V, Lininger L, *et al*. Effects of manipulation of dietary fatty acids on clinical manifestations of rheumatoid arthritis. *Lancet* 1985; **i**: 184-7.