LACK OF THERAPEUTIC EFFECT OF CIMETIDINE IN NON-ULCER DYSPEPSIA

Pages with reference to book, From 168 To 169

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Abstract

Therapeutic effect of cimetidine in non ulcer dyspepsia was studied in a placebo controlled trial. The reduction of pain was statistically significant in both groups during the first week of treatment but no change in subsequent three weeks. It was concluded that cimetidine has no therapeutic advantage over the placebo in dyspeptic patients (JPMA 38: 168,1988).

INTRODUCTION

Non ulcer dyspepsia is a common clinical problem associated with upper abdominal pain of varying severity, fullness, discomfort, belching, nausea, vomiting and acid regurgitation. Exact diagnosis necessitates extensive investigations to exclude organic diseases like peptic ulcer, cholecystitis, pancreatitis and irritable bowel syndrome. As the pathogenesis of non ulcer dyspepsia is not known, no rational therapy has yet been advocated for this condition. The purpose of this trial was to assess the efficacy of cimetidine in the treatment of dyspepsia.

PATIENTS AND METHODS

Thirty patients of both sexes, aged 18-50 years presenting with upper abdominal pain of varying severity and a negative endoscopy for ulcer, erosions, reflux oesophagitis or atrophic gastritis were included in this trial. Investigations prior to treatment included abdominal ultrasound, haemoglobin, total leucocyte and platelet counts, serum transaminases and creatinine. Patients meeting the entry criteria were allocated to cimetidine (200mg) or a matching placebo three times a day with meals and at bed time for 4 weeks. A diary card was given for recording the intensity of pain. Intensity was scored as for no pain, 1 for mild, 2 for moderate and 3 for severe pain. No concurrent medication was allowed during the study. Patients were followed up every week for four weeks. Endoscopy and laboratory investigations were repeated at the end of the trial.

RESULTS

Of 30 patients accepted for trial according to entry criteria only 20 (14 males and 6 females) completed the trial. The patients characteristics are shown in table 1.

Table I. Characteristics of Patients assigned to Placebo and Cimetidine Groups.

Characteristics	Placebo	Cimetidine
No : of Patients	10	10
Mean Age	30 ±10	33 ±10
Males/Females	8 / 2	6 / 4
Length of history		
<1 year	3	5
1 – 4 years	4	5
5 – 9 years	2	–
>9 years	1	–

There was a significant improvement in pain in both groups during the first week but in subsequent three weeks there was no change in symptoms (Table II).

Table II. Effect of Placebo and Cimetidine on Symptoms.

Duration	Placebo (9)		Cimetidine (9)	
	Percentage of change in Symptoms	Significance	Percentage of change in symptoms	Significance
First week	61.5	<0.001	46	<0.05
Second week	7.3	0.43	10.4	0.59
Third week	9.2	0.67	No change	0.05
Fourth week	5.0	0.22	No change	0.05

Incomplete pain intensity charts of one patient each in two groups were not analysed. The mean reduction in pain intensity with placebo was 50% ahd with cimetidine 56.4%. Changes in the weekly pain intensity scores are shown in the accompanying figure.

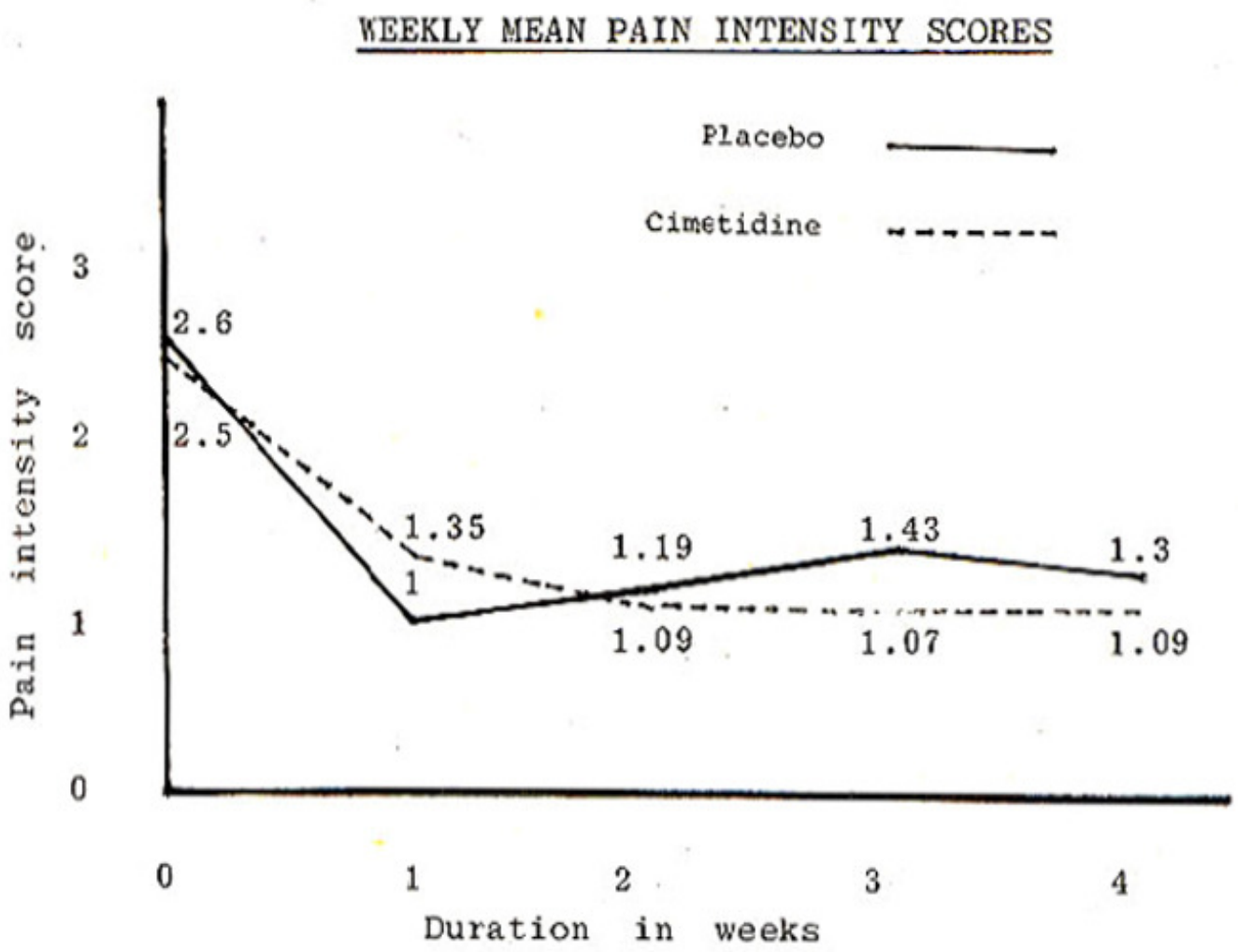

Figure 1. Weekly mean pain intensity scores.

There was no difference between two groups at any stage of treatment.

DISCUSSION

This controlled clinical trial shows no significant difference in pain scores in both groups and the active drug cimetidine, does not have any therapeutic advantage over placebo. Comparative trials of cimetidine and antacids show variable results. Panijel[1] found cimetidine significantly superior to the antacid while Nyren et al[2] found no difference between placebo, antacid and cimetidine. Similar observations are made in this trial. The tendency for spontaneous or placebo induced relief of pain in non ulcer dyspepsia and difficulty in clinically distinguishing between this clinical syndrome and acid peptic disease, with positive pain relief with acid suppressing and neutralizing drugs, may explain why these drugs are being prescribed as a standard therapy even in functional disorders. More studies on psychological and physiological mechanisms producing the symptoms complex of non ulcer dyspepsia may form the basis for alternative therapeutk strategies.

ACKNOWLEDGEMENT

Financial help by Smith Kline and French Pakistan is gratefully acknowledged.

REFERENCES

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