ALIMENTARY TRACT

Prospective clinical and manometric study comparing pneumatic dilatation and sublingual nifedipine in the treatment of oesophageal achalasia

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Abstract

A study was carried out in 30 patients affected by a mild or moderate degree of oesophageal achalasia to compare the clinical and manometric effects of sublingual nifedipine and pneumatic dilatation. Sixteen patients were dilated twice with Rider-Moeller dilators and 14 were treated with sublingual nifedipine 10-20 mg 30 minutes before meals. A manometric evaluation was performed before and six months after starting treatment. The clinical evaluation (according to Vantrappen's criteria) was performed every three months for a mean follow up of 21 months. In both groups of patients a significant ($p < 0.001$) fall in lower oesophageal sphincter pressure was observed after treatment and excellent or good clinical results were observed in 75% of dilated patients and in 77% of patients treated with nifedipine. One patient could not tolerate nifedipine. No complications were observed after dilatation. It is concluded that longterm treatment with sublingual nifedipine and pneumatic dilatation are equally effective in the treatment of oesophageal achalasia of mild or moderate degree.

The traditional approach to the treatment of patients with achalasia is based on stretching or cutting the muscle fibres at the oesophagogastric junction by pneumatic dilatation¹⁻⁶ or surgical myotomy.⁷⁻¹² More recently, nitro derivatives and calcium antagonists, particularly nifedipine, have been proved to be effective in reducing lower oesophageal pressure and improving oesophageal emptying in patients with achalasia.¹³⁻¹⁷ The results obtained both with surgical or mechanical procedures, such as myotomy and dilatation, and with drug treatment are well known.¹⁸⁻²⁰ No prospective study, however, has been reported comparing drug treatment with nifedipine and pneumatic dilatation.

The aim of our study was to perform such a prospective trial to determine whether there is a significant difference in the results of both treatments.

Methods

Thirty consecutive patients with clinical, radiological, endoscopic, and manometric evidence of oesophageal achalasia, stage I or II

using the classification of Adams *et al.*,²¹ were randomly assigned to two groups. Patients with sigmoid dilatation of the oesophagus or those who had had treatment for achalasia were excluded. Group A had 16 patients assigned to undergo pneumatic dilatation (seven men, nine women; mean age 49.5 years, range 18-82 years). The mean duration of symptoms was 4.1 years (range 6 months-8 years) and the mean oesophageal diameter 3.8 cm (range 2-6 cm). Group B had 14 patients who were treated with sublingual nifedipine (six men, eight women; mean age 51.3 years, range 17-83 years). The mean duration of symptoms was 3.5 years (range 6 months-7 years) and the mean oesophageal diameter 3.9 cm (range 2.5-6 cm).

CLINICAL STUDY

The patients were examined and details of the severity of dysphagia, regurgitation, retrosternal pain, and weight loss were taken before the study and every three months for a mean follow up of 21.4 months (range 12-36, median 21 months) according to the criteria of Vantrappen and Hellemans.² Patients were classified on the basis of the results of each treatment: excellent, good, moderate, and poor.

MANOMETRIC STUDY

Manometry was performed with a polyvinyl probe with five orifices, three of which, radially orientated, were positioned at the level of the lower oesophageal sphincter and two in the oesophageal body 5 and 10 cm above the sphincter. Each catheter was perfused with distilled water by a pneumohydraulic pump and connected to Statham P 23 Db transducers and a multichannel polygraph. The compliance of our system showed a rise in pressure of 150 mmHg/s on occlusion. After the patients had fasted overnight the probe was passed through the nose into the stomach and the lower oesophageal sphincter pressure was evaluated with a slow gastro-oesophageal pullthrough. Pressure was expressed as the mid-inspiratory pressure using intragastric pressure as zero reference. In group B patients, after a recording period of at least 30 minutes nifedipine 20 mg was administered sublingually and pressure recorded for another 45 minutes. In all patients a

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manometric control was performed six months after dilatation in group A and after six months of treatment in group B.

TREATMENT

In patients in group A, after an overnight fast and premedication with intravenous benzodiazepines a Rider-Moeller pneumatic dilator (35 mm diameter) was positioned at the oesophago-gastric junction over a guide wire and rapidly inflated to 200 mmHg for one minute and then to 300 mmHg for another minute.² The procedure was repeated 48 hours later with a larger dilator (40 mm diameter). After each dilatation an endoscopic control was performed to exclude complications such as perforation or haemorrhage. All patients were treated as outpatients.

In patients in group B nifedipine 10–20 mg was administered sublingually 30 minutes before each meal up to a maximum dose of 80 mg/day (mean 55 mg/day). The dose was chosen after a trial lasting a week.

Manometric results were expressed as mean (SD). Student's *t* test for paired and unpaired data and Wilcoxon's rank sum test were used for statistical evaluation.

Results

CLINICAL STUDY

Before treatment all patients had had dysphagia and regurgitation daily. In group A excellent or good results were obtained in 12 patients, moderate results in two, and poor results in two. No complications were observed in this group. In group B excellent or good results were obtained in 10 patients, moderate results in two, and poor results in one. One patient could not tolerate nifedipine because of headache. No patient developed a tolerance for nifedipine during follow up.

MANOMETRIC RESULTS

In group A patients the mean (SD) lower oesophageal sphincter pressure before treatment was 40.9 (15) mmHg and after treatment the pressure fell to 20.5 (7) mmHg ($p < 0.001$). In group B the mean lower oesophageal sphincter pressure before treatment was 40.8 (11) mmHg and 30 minutes after nifedipine administration it fell to 22.5 (8) mmHg ($p < 0.001$). After six months of treatment it was 25.7 (6) mmHg ($p < 0.001$).

Discussion

In our study both pneumatic dilatation and treatment with sublingual nifedipine reduced lower oesophageal sphincter pressure in patients with achalasia by 50% and 48%, respectively, from the pretreatment value. This result is in agreement with previous reports which showed a decrease in lower oesophageal sphincter pressure ranging from 42% to 51% in association with clinical improvement.^{19–22, 25} Clinical follow up, even when short, showed that both treatments are effective in reducing symptoms and that

excellent or good results are obtained in nearly the same proportions of patients. Our results confirm that pneumatic dilatation is a rapid procedure requiring no hospitalisation and in our patients no immediate complications were observed. There is, however, some risk of perforation, haemorrhage, or subsequent gastro-oesophageal reflux.²⁶ Moreover, some rupture of the mucosa often occurs, with fibrosis causing problems if surgery is subsequently performed.²³

On the other hand, sublingual nifedipine is unpleasant²⁷ and in some patients can cause excessive vasodilatation with peripheral oedema, headache, and hypotension. But only one of our patients was unable to tolerate nifedipine because of headaches and no hypotension occurred, even at doses up to 80 mg/day. Finally, in no case did tolerance to the drug develop.

Our manometric and clinical results are in agreement with previous studies. In fact Traube *et al* obtained a significant decrease in lower oesophageal sphincter pressure after 10 mg of sublingual nifedipine,¹⁹ and Gelfond *et al*, who administered oral nifedipine, with a consequent delay and decrease in drug adsorption, obtained satisfactory results.²⁷ Our results, however, contrast with a recent report from Robertson *et al*, who obtained no appreciable decrease in lower oesophageal sphincter pressure after nifedipine administration.²⁵ The methods of that trial are questionable because the lower oesophageal sphincter pressure was measured only 15 minutes after nifedipine was given and was compared with a value measured in a previous manometric examination. It is known that nifedipine has its maximum effect on lower oesophageal sphincter pressure 30 minutes after sublingual administration.²⁸ Finally, regarding the clinical part of that study, unfortunately nifedipine was given at too low a dose (10 mg), at too short intervals before meals (15 minutes), and to a population of unselected patients, including those with sigmoid dilatation of the oesophagus.

The results of our study show that, provided that nifedipine is administered sublingually to patients with stage I or II achalasia at least 30 minutes before meals and in an individualised dose, sublingual nifedipine is as effective as pneumatic dilatation in the treatment of oesophageal achalasia.

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