Risk factors of oesophageal perforation during pneumatic dilatation for achalasia

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Abstract

Background/Aims—Pneumatic dilatation of the oesophagus is a well established treatment for achalasia. Oesophageal perforation is the most serious complication that occurs in 2% to 6% of cases. The aim of this retrospective survey was to identify predictive risk factors for perforation in a consecutive series of 218 patients with achalasia.

Methods—Between 1983 and 1993, 270 pneumatic dilatations were performed in 218 patients. A Witzel dilator was used in 58 cases and a Rigiflex dilator in 212. Eight oesophageal perforations occurred (3%). The clinical, radiological, endoscopic, manometric, and technical data for the eight perforated patients were compared with those of 30 patients randomly sampled among those without perforation.

Results—All perforations occurred during the first dilatation. Perforations were fewer during dilatations with the Rigiflex dilator than with the Witzel dilator (2.4% v 5.2%). Perforations were all located above the cardia, on the left side of the oesophagus. In a multivariate analysis, a small weight loss and a high amplitude of oesophageal contractions in the group of patients with perforations were predictive of complications (respectively, $p=0.001$ and $p=0.026$). A contraction amplitude higher than 70 cm H₂O in the lower part of the oesophagus was observed in three of eight patients with perforations but was not seen in any of the 30 patients without perforation ($p<0.01$).

Conclusions—This identification of risk factors should facilitate the choice between pneumatic dilatation or a surgical approach.

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Keywords: achalasia, pneumatic dilatation, oesophageal motility disorders, oesophageal perforation.

Achalasia is a frequent primary oesophageal motor disorder characterised by loss of oesophageal peristaltic waves and failure of the lower oesophageal sphincter (LOS) to relax completely in response to swallowing. Dysphagia and regurgitation are prominent symptoms of the disease and lead to gradual weight loss. The primary cause remains unknown. Treatment is directed towards symptom relief by reducing LOS pressure, to allow the passage of food at the oesophago-

gastric junction, but this cannot restore oesophageal peristalsis and LOS relaxation. Two forms of treatment are routinely available: surgery and pneumatic dilatation. The surgical treatment consists of an extra mucosal oesophagomyotomy. Its main complication is gastro-oesophageal reflux; excellent to good results are observed in 70% to 90% of cases.¹ Pneumatic dilatation is supposed to be effective by disrupting the circular muscle fibres of the LOS; it yields good to excellent results in 65% to 80% of patients. The most serious complication of pneumatic dilatation is oesophageal perforation, which occurs in 2% to 6% of cases.¹ This complication may be treated medically or surgically, but it can lead to serious morbidity and even death. Some authors consider that the perforation risk is unacceptable in the light of the low risk of surgery. Only a few studies have dealt with the search for risk factors predictive of this complication.²⁻⁴ Botulinum toxin injection is a new alternative treatment. Few patients have been treated with good clinical improvement and no adverse effects but longterm follow up data are still lacking.⁵

The aim of this retrospective survey was to identify risk factors of oesophageal perforation during pneumatic dilatation performed in a single centre, in a consecutive retrospective cohort of patients. Identification of these factors should facilitate the choice between dilatation or a surgical approach.

Methods

PATIENT SELECTION

From January 1983 to July 1993, 270 consecutive pneumatic dilatations were performed in 218 patients with achalasia (once in 170 patients, twice in 44, and three times in four others). Achalasia was diagnosed from the association of dysphagia or thoracic pain and typical motor disorders, including the absence of peristaltic waves in the oesophageal body and a defective or incomplete LOS relaxation.⁶ Patients with oesophageal tumours, Chagas disease, and intestinal pseudo-obstruction were excluded. Epiphrenic diverticulum was considered to be a contraindication to pneumatic dilatation.

Each patient had a clinical evaluation, upper gastrointestinal endoscopy, a barium oesophagram, and oesophageal manometry. The largest diameter of the oesophagus was measured on upright series. Manometry was performed with pneumohydraulically infused catheters with three distal radially oriented orifices. Side holes were 5 cm from each other.

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LOS pressure was measured using the station withdrawal method, and considered as the mean of the three radial measures ($n=14$ to 61 cm H₂O). LOS relaxation was evaluated by the mean relaxation after three wet swallows ($n>80\%$), and contraction amplitude, by the mean amplitude of 10 wet swallows measured 5 cm above the LOS.

Two groups of patients were reviewed for comparison: group 1, which comprised eight patients who had an oesophageal perforation, and group 2, comprising 30 patients randomly sampled among the 210 patients without perforation.

STUDY DESIGN

Pneumatic dilatation technique

All patients fasted for at least eight hours before the procedure. The last meal given the day before dilatation was liquid. Dilatation was performed without analgesia from January 1983 to April 1987, and under anaesthesia (Midazolam and Propofol) as from May 1987. Before dilatation, gastroscopy was performed to suck up the residual fluid and food, and to assess the absence of oesophageal ulcer or epiphrenic diverticulum. Two types of dilatator were used: a Witzel balloon in 58 cases, and a Rigiflex balloon in 212 cases.

The Witzel pneumatic dilatator (ABS, Parc d'Activité Saint Michel, BP 234, 88 106 Saint Dié Cedex, France) consists of a polyurethane balloon, 15 cm long and 40 mm in diameter, mounted on a polyvinyl tube with an internal diameter of 10 mm and an external diameter of 11 mm. It is connected to an external inflation device. The polyvinyl tube encased by the balloon is fitted over a paediatric gastroscope and secured to the shaft of the endoscope. The endoscope is passed through the oesophagogastric junction and retroflexed to allow the operator to make sure that the balloon is correctly positioned.⁷ Rigiflex polyethylene achalasia dilator (Microvasive, Boston Scientific, BP 32, 78 184 Saint Quentin en Yvelines Cedex, France) is 30, 35, or 40 mm in diameter, 10 cm long, and is mounted on a 100 cm, 7 Fr catheter. It slides along a metal guidewire set up during the endoscopy. Two radio-opaque tantalum markers at each end of the balloon help to position the balloon under fluoroscopy.⁸ The 35 mm balloon was usually used for the first dilatation, and the 40 mm balloon for subsequent dilatations. Both types of dilator are expansion limited. In each of the present cases, the balloon was inflated with air to a pressure of 300 mm Hg (5.9 psi) for one minute, three times at each session, at intervals of one minute. All dilatations were performed by the same operator (MG).

After dilatation, patients fasted until the next morning and, in the absence of complications, were discharged after a normal meal. Routine contrast study was not the rule after dilatation, but every clinical finding such as chest or abdominal pain, even minor, or fever constituted an indication for a prompt water soluble oesophagram, to detect any

complication. Perforation was defined as the extravasation of the water soluble contrast into the mediastinum.

Analysis of the results

The perforation group was compared with the control group for the following characteristics: clinical data (age, sex, symptoms and their duration, previous bougienage, weight and weight loss), endoscopic data (residual fluid or food, considerable resistance to passage through the oesophagogastric junction and oesophageal or gastric biopsy during the two days before the procedure), manometric data (LOS and oesophageal pressure, LOS relaxation, and the amplitude of the contractions in the oesophageal body), radiological data (largest oesophageal diameter on the barium oesophagram), pharmacological data (ingestion of nitrates or calcium channel antagonists during the two previous days and anaesthesia), and the characteristics of the balloon.

Statistical analysis

Results are expressed as either percentages or means (SD). The relation between the presence of an oesophageal perforation and various risk factors was first studied by univariate analysis in which variables were compared by parametric tests. The χ^2 test with Yates's correction was used to compare qualitative variables in the perforated and the non-perforated groups and Student's t test to compare quantitative variables. In the multivariate analysis, stepwise logistic regression was used. The aim of this multivariate analysis was to identify variables that best discriminated between the two groups (dependent variables: presence or absence of perforation) when all variables included in the model were considered together (independent variables). Five per cent was taken as the level of significance.

Results

Eight oesophageal perforations (3%) occurred in the course of 270 pneumatic dilatations. Four patients were treated by surgery and four others medically, mortality was zero. Two other complications were observed: isolated fever in one case and chest pain in another one; the water soluble oesophagram was normal for both these patients and both had a rapid and uneventful recovery. No patient had gastrointestinal haemorrhage.

All eight perforations occurred in patients undergoing their initial dilatation. All these perforations were longitudinal gaps 1 to 4 cm long, located above the cardia on the left side of the oesophagus. The incidence of perforation was twice as frequent with the Witzel balloon (three of 58, 5.2%) as with the Rigiflex balloon (five of 212, 2.4%), but the difference was not significant ($p=0.5$). The clinical, endoscopic, manometric, radiographical, and pharmacological data, and the associated treatment in the two groups of patients, are

TABLE I Clinical characteristics of the two groups of patients (univariate analysis)

	Perforation (n=8)	Control (n=30)	p Value
Age (y)	62 (17)*	55 (21)*	NS
Sex M/W (%)	50/50	50/50	NS
Weight (kg)	62 (8)*	64.7 (16.5)*	NS
Previous bougienage (%)	25	6.7	NS
Duration of symptoms (y)	15 (13)*	4.2 (4.4)*	<0.001
Weight loss (kg)	0.25 (0.7)*	7 (8)	<0.01
Pain (%)	25	53	NS
Dysphagia (%)	100	100	NS
Aphagia (%)	12.5	16.7	NS
Regurgitations (%)	63	63	NS
Nocturnal cough (%)	25	20	NS

*Mean (SD).

TABLE II Endoscopic and therapeutic characteristics of the two groups of patients (univariate analysis)

	Perforation (n=8)	Control (n=30)	p Value
Gastroscopy:			
Resistance at the passage through the cardia (%)	75	67	NS
Residual fluid (%)	63	83	NS
Residual food (%)	50	27	NS
Recent biopsy	0	0	NS
Recent ingestion of nitrates or calcium channel antagonists (<J-2)	25	23.3	NS
Anaesthesia	37.5	40	NS

TABLE III Manometric and radiological characteristics of the two groups of patients (univariate analysis)

	Perforation (n=8)	Control (n=30)	p Value
Oesophageal manometry			
Contractions amplitude (cm H ₂ O)	52.6 (22.9)*	33.8 (16.7)*	<0.02
Contractions >70 (cm H ₂ O)	37.5	0	<0.01
Intraoesophageal pressure (cm H ₂ O)	8.6 (2.4)*	9.9 (9.9)*	NS
LOS pressure (cm H ₂ O)	40.1 (16.8)*	48.3 (21)*	NS
LOS relaxation (%)	45 (32)*	37 (27)	NS
Barium oesophagram			
Oesophageal diameter (mm)	43 (8)	49.4 (16)	NS

*Mean (SD).

listed in Tables I to III. Patients' age and sex were comparable in the two groups. Barium oesophagrams showed that the oesophagus tended to be less enlarged in patients with perforation. According to univariate analysis, patients with perforation had a longer duration of symptoms than those without (15 (13) years *v* 4.2 (4.4) years, $p = <0.001$), a smaller weight loss (0.25 (0.7) kg *v* 7 (8) kg, $p < 0.01$), and a greater amplitude of the oesophageal contractions (mean amplitude = 52.6 (22.9) cm H₂O *v* 33.8 (16.8) cm H₂O, $p < 0.02$). Stepwise logistic regression showed that weight loss ($p = 0.001$) and oesophageal contraction amplitude ($p = 0.026$) were positively correlated with the presence of perforation, when the above three variables (symptom duration, weight loss, and contraction amplitude) were considered together. A contraction amplitude higher than 70 cm H₂O in the lower part of the oesophagus was observed in three of eight patients who had been perforated but not in any of the 30 patients without perforation ($p < 0.01$).

Discussion

Oesophageal perforation is the most serious complication of pneumatic dilatation and may result in pronounced morbidity, which can, exceptionally, lead to death.^{9 10} The perforation rate we observed is consistent with that reported in other studies – that is, from 2 to

6%. Gastric perforation is a rare occurrence only observed in a patient with previous gastric resection and a small remnant.¹¹ Other less serious complications have been reported, such as gastrointestinal haemorrhage, fever, oesophageal tears, and haematoma.^{1 2} In one case we observed isolated fever, and in another chest pain. The water soluble oesophagram was normal for both these patients and both had a rapid and uneventful recovery. No patient had gastrointestinal haemorrhage.

Several risk factors for oesophageal perforation have been suggested: malnutrition, recent oesophageal biopsy, epiphrenic diverticulum, low LOS pressure, complete LOS relaxation, and anaesthesia.^{1 3} None of these factors was seen in our series, from which epiphrenic diverticulum had been excluded. Recently, Nair *et al* searched retrospectively for risk factors in a series of 178 patients who had 236 pneumatic dilatations.² They used a Browne Mac Hardy dilator 38 mm in diameter. The pressure (from 7 to 17 psi, 350 to 860 mm Hg), duration (10 to 75 sec), and number of inflations at each session (one to three) were chosen at the operator's discretion. Sixteen patients experienced complications (four of them had perforations). An inflation pressure of more than 11 psi (560 mm Hg) and previous dilatations were risk factors for complications. Our results cannot be compared with those of Nair, because in our study, duration, pressure, and number of inflations were always the same for each procedure. Furthermore, we used low compliance balloons instead of the Browne Mac Hardy dilator, which is a high compliance balloon. It has been suggested that inelastic balloons like the one we used are probably safer and should be preferred.¹²

In our series, perforation always occurred during the first dilatation, suggesting the presence of individual risk factors. In case of achalasia, the oesophageal body is usually hypotonic. In some instances, the preservation of the ability of the oesophagus to contract may favour the occurrence of a perforation. Note that perforation does not occur at the level of the LOS, where the maximal pressure is applied, but above it, on the left side of the oesophageal body. In multivariate analysis, only one clinical risk factor was identified here – that is, low weight loss, despite longstanding dysphagia. Furthermore, the oesophagus was less enlarged in the patients with perforation, but their symptoms had been present for longer. Nair *et al* also observed that symptoms tended to last longer for the four patients with perforation than for the other 118 control patients (11.3 (4.3) years *v* 6.3 (6) years, non-significant difference). These characteristics suggest that the oesophagus may be less compliant in cases of perforation. However, Fennerty *et al* considered poor nutritional status as an important risk factor of perforation, as they observed a perforation in three patients with pronounced weight loss and severe hypoalbuminaemia. However, they had no control group, and they used a higher pressure level of dilatation than ours. In our series, the mean contraction amplitude in the

lower part of the oesophagus in the perforation group was higher than that seen in the control group. In achalasia, oesophageal contractions are usually of low amplitude. Inflation of a balloon in the oesophagus may induce high amplitude contractions of prolonged duration, and it is conceivable that these contractions may be higher when spontaneous contractions are preserved. The combined effects of the inflation pressure of the balloon and of these contractions might cause oesophageal disruption in a frail anatomic site. In our series, this site was located a few centimetres above the cardia on the left side of the oesophagus according to radiological and surgical assessments. At this site, the longitudinal layer of the smooth muscle fibres is asymmetric and thinner on the lateral sides of the oesophagus than on their dorsal or ventral sides.^{13 14-16} Comparable findings were reported in spontaneous rupture of the oesophagus that occurs at the same site.¹⁷ Its aetiopathogeny is unknown but the most commonly proposed mechanism is the sudden appearance of high intraluminal pressure after an uncoordinated act of vomiting against a closed cricopharyngeal sphincter. Here we observed twice as many perforations with the Witzel balloon as with the Rigiflex balloon. The retroflexed position maintained during dilatation requires persistent inflation, which increases the intra-gastric pressure. This sustained inflation might help to increase the pressure of the oesophageal contractions, and thus to explain the larger number of perforations with the Witzel balloon.

The presence of two factors – that is, persistence of high amplitude contractions and quick distension of a frail anatomic site might favour perforation of the oesophagus during pneumatic dilatation. The disappearance of contractions in an enlarged oesophagus might explain the smaller rate of complications in the patients with more advanced achalasia.

In conclusion, the occurrence of contractions with an amplitude higher than 70 cm

H₂O in the lower part of the oesophagus is a risk factor of oesophageal perforation, which was identified in one third of the patients perforated during pneumatic dilatation for achalasia. As preliminary results suggest that better response are observed in patients with a vigorous pattern of achalasia,^{18 19} a controlled clinical trial would be useful to determine the best choice between pneumatic dilatation or a surgical approach.

- 1 Reynolds JC, Parkman HP. Achalasia. *Gastroenterol Clin North Am* 1989; 18: 223–55.
- 2 Nair LA, Reynolds JC, Parkman HP, Ouyang A, Strom BL, Rosato EF, et al. Complications during pneumatic dilatation for achalasia or diffuse esophageal spasm. Analysis of risk factors, early clinical characteristics, and outcome. *Dig Dis Sci* 1993; 38: 1893–904.
- 3 Fennerty B. Esophageal perforation during pneumatic dilatation for achalasia: a possible association with malnutrition. *Dysphagia* 1990; 5: 227–8.
- 4 Tubman AB. Complications of esophageal dilation and guideless for their prevention. *Gastrointest Endosc* 1981; 27: 229–34.
- 5 Pasricha PJ, Ravich WJ, Hendrix TR, Sostre S, Jones B, Kalloo N. Intraspincteric botulinum toxin for the treatment of achalasia. *N Engl J Med* 1995; 322: 774–8.
- 6 Castell DO. Achalasia and diffuse esophageal spasm. *Arch Intern Med* 1976; 136: 571–9.
- 7 Witzel L. Treatment of achalasia with a pneumatic dilator attached to a gastroscope. *Endoscopy* 1981; 13: 176–7.
- 8 Kozarek RA, Gelfand MD, Christie DL, Ball TJ. Balloon dilation in achalasia. A promising technique. *Gastroenterology* 1986; 90: 1502.
- 9 Miller ER, Tiskenkel HI. Esophageal perforation due to pneumatic dilation for achalasia. *Surg Gynecol Obstet* 1988; 166: 458–60.
- 10 Vantrappen G, Hellemans J. Treatment of achalasia and related motor disorders. *Gastroenterology* 1980; 79: 144–54.
- 11 Brown FC, Johnson RB, Castell DO. Gastric perforation during pneumatic dilation for achalasia. *Gastroenterology* 1977; 72: 893–8.
- 12 Rabinovici R, Katz E, Goldin E, Kluger Y, Ayalon A. The danger of high compliance balloons for esophageal dilatation in achalasia. *Endoscopy* 1990; 22: 63–4.
- 13 Winans CS. Manometric asymmetry of the lower esophageal high pressure zone. *Am J Dig Dis* 1977; 22: 348–54.
- 14 Mackler SA. Spontaneous rupture of the esophagus. *Surg Gynecol Obstet* 1952; 95: 345–56.
- 15 Derbes VJ, Mitchell RE. Rupture of the esophagus. *Surgery* 1956; 39: 688–709, 865–88.
- 16 Tidman MK, John HT. Spontaneous rupture of the esophagus. *Br J Surg* 1967; 54: 286–92.
- 17 Henderson JAM, Peloquin AJM. Boerhave revisited: spontaneous esophageal perforation as a diagnostic masquerader (reviews). *Am J Med* 1989; 86: 559–67.
- 18 Pasricha PJ, Rai R, Ravich WJ, Hendrix TR, Kalloo N. Botulinum toxin for achalasia: long term follow up and predictors of outcome. *Gastroenterology* 1995; 108: A187.
- 19 Jin HO, Potter GD, Shah AN, Ona FV, Malkin M, Chey NM. Effect of intraspincteric injection of botulinum toxin on esophageal motility in achalasia. *Gastroenterology* 1995; 108: A123.