Oesophageal perforation at fibreoptic gastroscopy

Perforation is the most serious complication of fibreoptic endoscopy.[1] The commonest site of perforation during upper gastrointestinal endoscopy is the oesophagus,[2] but predisposing factors have not been fully documented. We report a retrospective survey of major endoscopy units in the United Kingdom and attempt to identify the factors associated with oesophageal perforation.

Methods and results

In a survey of 173 endoscopy units conducted through the British Society for Digestive Endoscopy (which merged with the British Society of Gastroenterology in 1980) 31 of the 101 units who replied reported serious complications (R Cockel, unpublished data). A supplementary questionnaire was sent to these centres requesting further details, and replies were received from 24. The table records the information concerning oesophageal perforation at oesophagogastroduodenoscopy and related procedures.

Perforation at diagnostic oesophagogastroduodenoscopy was rare (one in 5474 examinations; seven cases altogether). These seven patients were relatively elderly (mean age 72 years) compared with others undergoing the procedure (mean age 53 years). Barium swallow had been performed in four of the seven patients before endoscopy. In five cases the perforations were high and associated with difficult intubation; in four of these cases intubation was performed by inexperienced operators. The remaining two patients had radiologically identified strictures (one peptic, one carcinoma). No particular type of fibrescope was associated with perforation and no obvious medical factors contributed. Perforation was recognised almost immediately in six patients, but one outpatient died two days later at home. Thoracotomy was performed in two patients, one of whom died.

Perforation during oesophageal dilatation was more common (one in 109 procedures; 11 patients) and occurred despite previous radiological visualisation of the stricture in nine patients. Eight patients had peptic strictures, and perforation occurred at first dilatation in seven and at second dilatation in one. On three occasions perforation occurred during the first stage of a planned two-stage procedure, and defective dilators contributed in one case. Two oesophageal cancers, both misinterpreted as benign strictures, and one invading bronchial carcinoma were perforated. *X*-ray screening was used in only three of the 11 patients. Four patients died, two after thoracotomy, which was performed in only four patients. Heavy sedation was used in many of the patients with perforation. Perforation occurred at oesophageal dilatation in two additional patients not recorded in the table. One patient died after perforation of the stomach during dilatation of a peptic oesophageal stricture when the metal flexible finger end of the Eder-Puestow dilator became unwound. Another patient died from jejunal perforation after dilatation of a benign oesophagojejunal stricture.

Perforation during palliative oesophageal intubation for carcinoma was common (one in 13 procedures; 18 cases). Barium swallow had been performed in 15 of the 18 patients before the procedure. Except for one bronchial carcinoma, all were lesions of the mid or lower third of the oesophagus. Eight of the strictures were so tight that the guidewire passed only with difficulty. Despite perforation intubation was successful in 16 of the patients. The remaining two died soon after the procedure. Screening was used in 13 of the patients. All were managed conservatively, cardiorespiratory problems contributing to death in five.

Comment

These data indicate that perforation at diagnostic oesophagogastroduodenoscopy is rare and when it does occur is commonly associated with difficult intubation, inexperienced operators, and elderly patients. Conversely, therapeutic endoscopy is associated with a higher incidence of perforation, palliative intubation being particularly hazardous. Sedation was heavy in many cases when perforation occurred during dilatation. Staging of the procedure did not prevent complications. The presence of a tight stricture was the major risk factor for perforation at intubation. Nevertheless, morbidity and mortality were considerably lower than in series in which dilatation[3] and intubation[4] were performed using rigid endoscopes. Finally, we emphasise that, contrary to recent alarmist views,[5] prior radiology does not prevent perforation at endoscopy.

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Oesophageal perforation during fibreoptic oesophagogastroduodenoscopy

	No of perforations	No of examinations	Incidence of perforation (%)	No of deaths	Mean age (years) (range)	Heavy sedation*	Radiology before procedure	Other factors
Diagnostic oesophagogastroduodenoscopy	7	38 315	0·018	3	69 (55-80)	2 (0)	4	Difficult intubation/ inexperience
Oesophageal dilatation	11	1 203	0·9	4	72 (58-92)	7 (3)	9	Unsuspected carcinoma
Oesophageal intubation	18	229	7·9	12	70 (58-84)	11 (0)	15	Tight stricture
Other procedure	2			1	34, 45	0	2	Balloon dilatation for achalasia/variceal sclerosis
Total	38	39 747	0·096	20	69	20 (3)	30	

*Large doses of opiates or (in parentheses) a general anaesthetic.

Actinobacillus lignieresii infection after a horse bite

Infected animal-bite wounds are not uncommon. Most reports concern cat and dog bites infected by *Pasteurella multocida*. We present a case of serious facial infection after a horse bite, from which the related but far more unusual small Gram-negative rod *Actinobacillus lignieresii* was isolated.

Case report

A 13-year-old boy was bitten on the face by a horse. He sustained a 10-cm long gash stretching from the tip of the nose to the lateral part of the left cheek. The wound was primarily sutured, and no antibiotics were given. On the next day considerable swelling, which gradually became worse, was noticed around the wound. Two days after the injury he was admitted to hospital because of massive infection in the wound. There was a pronounced rubor and oedema of the surrounding tissues, and the left eye was almost occluded by periocular oedema. The patient's temperature was slightly raised but his general condition was good. Several sutures were removed, and a large amount of bloody, foul-smelling pus was drained from the wound. Initially the patient was given intravenous penicillin but this was subsequently changed to peroral tetracycline because of an allergic reaction. The infection resolved rapidly, and he was discharged after one week. The wound healed well, but the scar required plastic surgery.

Large numbers of polymorphonuclear leucocytes and pleomorphic Gram-negative rods, many of which were coccoid, were seen in a Gram-stained smear of the pus. Initial culture showed a heavy growth of viscous, smooth, greyish-white colonies on human-blood agar incubated overnight in air. There was also a moderate growth of *Escherichia coli* and a few bacteroides colonies after anaerobic incubation. The viscous colonies were oxidase- and catalase-positive and grew on bromothymol blue agar. The isolate was immotile, indole-negative, and urease-positive. Mannitol was fermented, nitrate was reduced, and ornithine decarboxylase activity was absent. The strain was sensitive to penicillin and tetracycline when tested by the disc diffusion method. The organism was initially classified as a variant of *Pasteurella pneumotropica* but was later reclassified as *A lignieresii*.

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Comment

Infected animal-bite wounds usually involve bacteria originating from the oropharyngeal flora of the animal. *A lignieresii* has been isolated from the mouths of normal cattle and sheep but may cause disease—for example, "wooden tongue" of cattle—in these animals. Surprisingly, it is not a microbe primarily associated with horses, but the closely related species, *Actinobacillus equuli*, may be found in the oral flora of normal horses.[1]

The pathogenic role of *A lignieresii* in this infection seems certain. Human infections are extremely rare and to our knowledge this is the first report from Scandinavia and one of the few confirmed cases in the world. We know of only one other isolate from an animal (horse) bite wound.[1] It is possible that *A lignieresii* may be more common than reports suggest because it may easily be confused with *Pasteurella* sp as it was by us[2] and others[3] initially. Differentiation of these organisms is not easy but may be achieved by using a fairly wide range of biochemical reactions.[1]

The risk of infection must always be considered when treating an animal-bite wound. *A lignieresii* should be recognised as a potential cause of wound infection after bites by larger domestic mammals.

We thank Dr R E Weaver, Special Bacteriology Section, Center for Disease Control, Atlanta, USA for examining our isolate and confirming the identification.

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Further observations on serum free thyroxine concentrations during pregnancy

In a previous communication[1] we reported that serum free thyroxine (FT4) concentrations fall appreciably during pregnancy. The commercial kit which we used then was an indirect method of measuring FT4, though Kurtz *et al*[2] using a direct method published results which gave some support to our original data. Two new direct methods for measuring FT4 have recently become available commercially. We have used them to repeat our study.

Patients, methods, and results

Blood was taken from clinically euthyroid pregnant women undergoing routine check-ups. About 20 women in each trimester of pregnancy were selected, and 20 euthyroid non-pregnant female members of staff of similar age range volunteered as controls for the study. No one in the control group was taking a contraceptive pill. We measured FT4 using Amerlex and LiquiSol FT4 kits, total T4 by our in-house and Tetrak-Peg kits, and thyroxine binding globulin (TBG) by RIA-gnost TBG kits. The T4:TBG ratio was calculated. The results are shown in the table.

Results from the two FT4 kits showed no correlation in any of the four groups. With the Amerlex kit the mean for the control group was not significantly different from the mean of the first-trimester group but was highly significantly different from the mean of the second- and third-trimester groups. Twenty per cent of the results lay outside the manufacturer's reference range. With the LiquiSol kit none of the means were significantly different from the mean of the control group, though the mean of the third-trimester group was significantly different from that of the first- and second-trimester groups. All values lay within the manufacturer's reference range. Changes in T4 and TBG were similar to those found in our original study, in which we used different methods for measuring these variables. In particular TBG rose more rapidly than either T4 or FT4 during the first trimester. Furthermore, the T4:TBG ratio again showed a pronounced downward trend during pregnancy, and in this study 56% of values in the third trimester were lower than those in the control range (previous study 58%). A recent paper[3] suggests that a modified version of this ratio is clinically useful in patients who do not have TBG concentrations outside the normal range. Our data suggest that when TBG concentrations are raised the validity of this simple ratio must surely be in doubt.

Comment

This limited comparative study emphasises the unresolved contradictions even when FT4 is measured directly. It has generally been assumed that FT4 concentrations remain constant during pregnancy despite the changes in TBG and T4 concentrations, but several observers have shown a fall in FT4 with gestational age.[4] In this study the mean value of the third-trimester samples when assayed by the LiquiSol kit was not significantly different from the mean value of the control group samples, whereas the opposite was true with the Amerlex kit. We cannot yet be certain whether the FT4 concentration changes during pregnancy, and we cannot therefore say which of the two kits reflects the true physiological state. If the observed fall in FT4 is true then an additional reference range for pregnancy might be helpful to the clinician. If not, then some explanation must be found by way of method or artefact.

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FT4, T4, and TBG concentrations and T4:TBG ratios. Results are means (and 95% confidence ranges)

	FT4 (Amerlex kit) (pmol/l)	FT4 (LiquiSol kit) (pmol/l)	T4 (nmol/l)	TBG (mg/l)	T4:TBG
Controls	20·5 (29·9–11·1)	18·7 (23·9–13·5)	90 (118–63)	20 (25–16)	4·5 (5·9–3·1)
First-trimester group	19·1 (29·3–8·7)	20·6 (27·4–13·8)	122 (168–76)	35 (50–19)	4·0 (5·2–2·8)
Second-trimester group	16·4 (28·0–4·8)	20·2 (27·0–13·4)	148 (210–85)	45 (60–30)	3·3 (4·7–1·9)
Third-trimester group	13·4 (18·4–8·4)	17·6 (22·2–13·0)	131 (177–86)	48 (63–32)	2·8 (3·8–1·8)

Conversion: SI to traditional units—FT4: 1 pmol/l ≈ 0·0008 μg/100 ml. T4: 1 nmol/l ≈ 0·08 μg/100 ml.

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