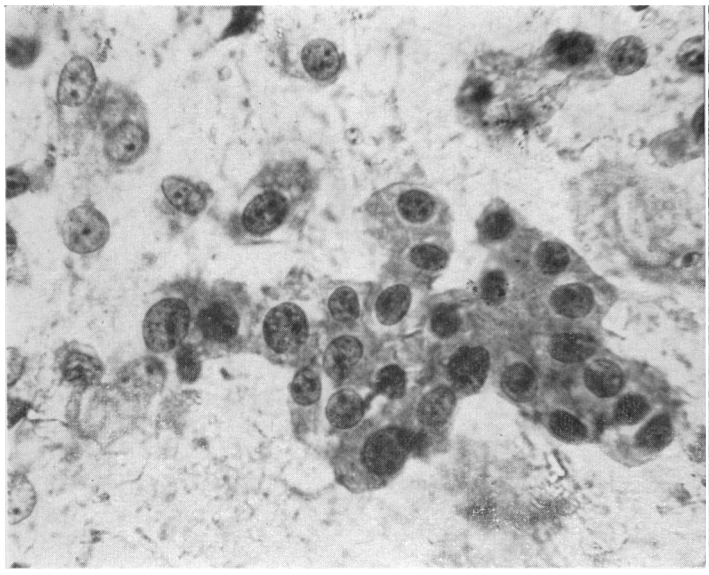

FIG. 1

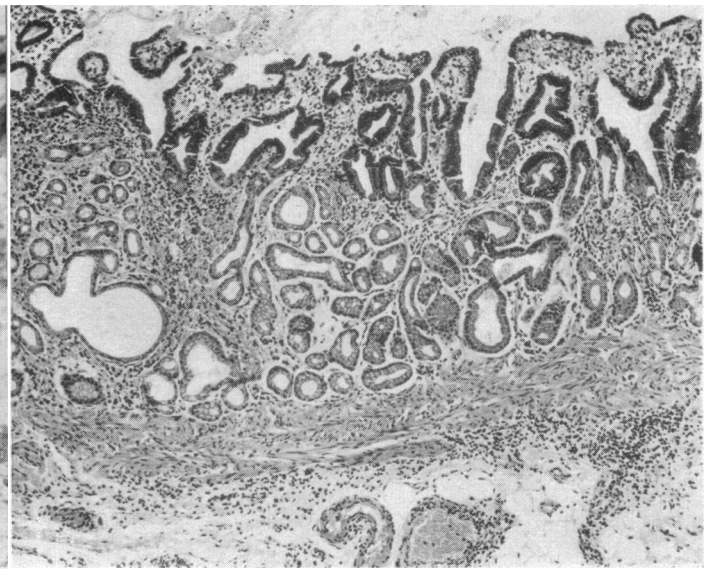

FIG. 3

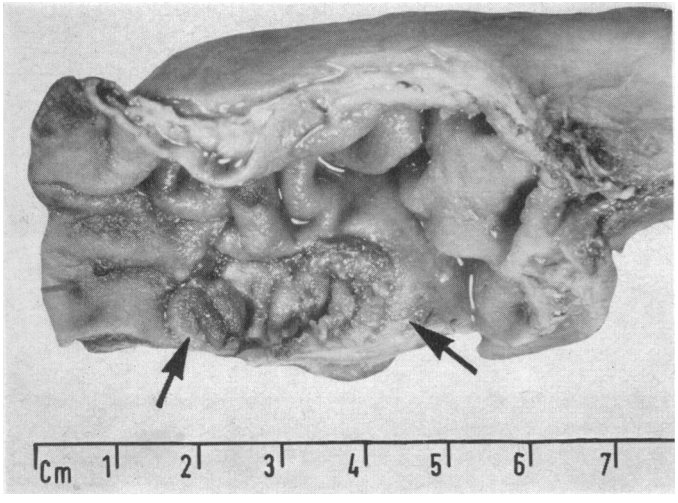

FIG. 2

FIG. 1.—Group of darkly staining cells which contrast with the lightly staining gastritic cells. (Papanicolaou. ×600.)

FIG. 2.—Resected stomach showing oval lesion, marked with arrows.

FIG. 3.—Irregular and darkly staining superficial mucosa. Note absence of fibrosis and intact muscularis mucosae. (H. and E. ×70.)

FIG. 4.—Papillary proliferative glandular epithelium. (H. and E. ×300.)

FIG. 5.—Mitotic figure, marked with arrow, in atypical acinus. (H. and E. ×600.)

FIG. 4

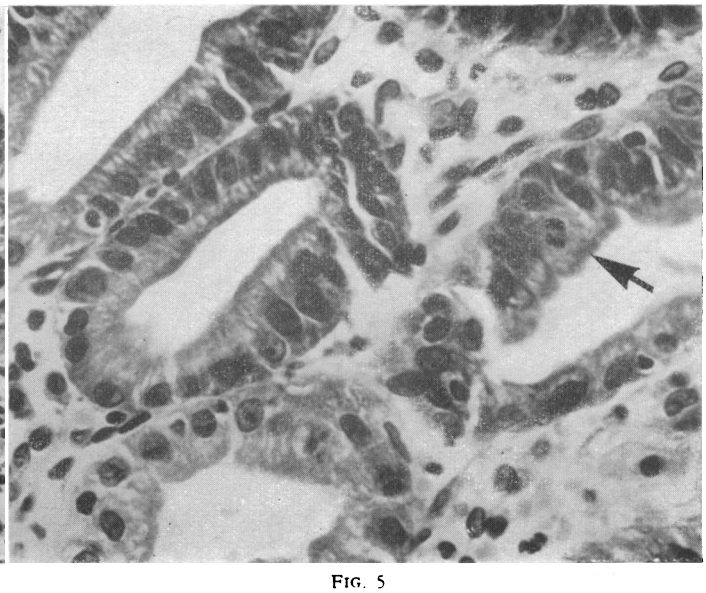

FIG. 5

and there was early vascularization and band-shaped keratopathy. His present visual acuity in the right eye is counting fingers; projection to light is accurate.

It is of interest to note that the visual acuity of the left eye when first seen was 6/24 unaided, the patient stating that this eye had been poor all his life. In August 1966 the refraction for the left eye was +3·50 D.C. at 120 degrees, giving a visual acuity of 6/12. By February 1968 the visual acuity of the left eye had improved to 6/5 with the above correction.

Comment

Ammonia is a colourless gas with a perceptible odour in a concentration of 50 p.p.m. in air. It is very soluble and combines with water to form ammonium hydroxide. The strongest solution available is between 28 and 29%.

Ocular injury following exposure to ammonia is basically similar to that from other alkalis, with special features attributable to its high solubility in water and lipids and consequent rapid penetration. The effect of this solubility has been investigated by many workers; Siegrist (1920) notably found ammonium ions in the aqueous humour of rabbits' eyes five seconds after the local application of ammonium hydroxide.

Grant (1950) showed that it is the *p*H and not the ammonium ion itself that is significant to the degree of injury. Depending on the concentration and duration of contact, all degrees of damage may be seen from mild corneal ulceration to severe damage of the whole anterior segment—loss of corneal epithelium especially in the lower half, stromal oedema, endotheliosis, iritis, lens damage, and corneal anaesthesia with a tendency to sloughing. Grant (1962) mentioned that a rise in the intraocular pressure had been recorded one month after the initial insult.

These two cases illustrate that a rise in the intraocular pressure may occur sooner than we previously thought, since they showed such a rise within four hours of exposure to ammonia.

The other presenting signs in addition to the early rise in the intraocular pressure mimicked in many respects those seen in acute-angle closure glaucoma—namely, the oval semi-dilated non-reacting pupil and corneal oedema. Closure of the angle of the anterior chamber was considered as a likely cause, but in the first case gonioscopy of the affected eye and of its fellow did not confirm this. Gonioscopy in the second case was not performed at the time of the injury, but the anterior chamber of the affected eye was at no time noted to be shallow, and later examination of the unaffected eye showed a medium angle. Considering the speed of penetration of ammonia through the tissues of the eye, inflammatory swelling in the region of the trabecular meshwork could explain this rise in the intraocular pressure. The semi-dilated fixed pupil, however, is more difficult to explain, but considering the speed of penetration of ammonia simultaneous paralysis of both the sphincter and dilator muscles of the iris are distinct possibilities, especially since in both cases there was no return of iris tone. It is interesting to note that Grant (1962) stated that iris damage may not become apparent until the second week after exposure to ammonia.

I wish to thank Mr. Redmond Smith and Mr. R. A. Burn for their kind help and advice, and for allowing me to make use of their cases.

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Medical Memoranda

Detection by Cytology of Carcinoma-in-situ after Gastroenterostomy

[WITH SPECIAL PLATE FACING PAGE 355]

Brit. med. J., 1969, **1**, 360–361

Though gastric cytology has been in use for a long time its value in many situations is only now becoming apparent. It is accepted that the diagnosis of carcinoma of the stomach is more accurate when gastroscopy, radiology, and cytology are used in combination (Blendis *et al.*, 1967). A study of survival rates for the disease shows that in most cases cure by means of surgery would necessitate diagnosis at the carcinoma-in-situ stage. In the intact stomach surface carcinoma diagnosed by cytology has been reported in six cases by Graham (1954), in three cases by Klayman *et al.* (1955), and in 16 cases by Schade (1960). The diagnosis of carcinoma after partial gastrectomy or gastroenterostomy is more difficult, but cytology has been used in three cases after partial gastrectomy (Gibbs, 1962); all were found to be extensive at operation. Passarelli *et al.* (1963) reported a carcinoma-in-situ which developed in the gastric remnant three years after partial gastrectomy and gastroenterostomy for an adenocarcinoma of the greater curvature. Here we report the diagnosis of carcinoma-in-situ by cytology 23 years after gastroenterostomy.

CASE REPORT

The patient, a man aged 48, developed symptoms of duodenal ulceration in 1940. Haematemesis occurred in 1941, 1942, and 1943. The ulcer then perforated and was treated by simple closure. In 1944 pyloric stenosis developed, and a retrocolic gastroenterostomy was performed. Upper abdominal pain related to meals continued, and there were several further haematemeses, before vagotomy was performed in 1963. Gastric secretory studies were not performed. At the age of 47 he was admitted to the Edinburgh Royal Infirmary with complaints of tiredness and nausea. He was anaemic, with haemoglobin 9·8 g./100 ml., M.C.H.C. 29%, serum iron 29 μg./100 ml., and total iron-binding capacity 570 μg./100 ml. Stool occult blood was negative. A barium-meal examination showed minor irregularity of the greater curvature of the antrum. The patient was achlorhydric to pentagastrin (Peptavlon 6 μg./kg.), the minimum *p*H being 7·3. Intrinsic factor output was 1,056 mμg. in the post-pentagastrin hour. Tests for parietal cell and intrinsic factor antibodies were negative. Gastric lavage carried out with two 300-ml. aliquots of normal saline gave recoveries of 160 and 120 ml. Malignant cells were found (Fig. 1). At laparotomy no abnormality of the stomach was seen, but a Billroth I antrectomy was carried out. A further gastric lavage performed six months after operation showed active epithelial cells but no malignant cells.

PATHOLOGY

Exfoliative cytology revealed clumps of cells which were clearly different from the palely staining gastric epithelial cells (Fig. 1). The former showed hyperchromatic nuclei with large and prominent nucleoli and darkly staining basophilic cytoplasm.

The lesion itself consisted of a velvety patch of rather irregular but moderately well circumscribed mucosa measuring 2·5 to 1·5 cm.

(Fig. 2). There were no signs of ulceration, though the mucosal pattern had been lost. Microscopical examination showed an almost intact mucosa where the superficial glandular elements were darkly staining and grossly irregular (Fig. 3). The glands were dilated; some were microcystic and contained papillary ingrowths of proliferative epithelium (Fig. 4). In some areas the acini were highly irregular and hyperchromatic. The nuclei were no longer basal, were much larger than usual, and contained prominent nucleoli.

Some glands were lined with relatively normal columnar epithelium, but, interspersed, there were large bizarre basophilic cells, clearly epithelial but containing large nuclei with several nucleoli. Mitotic figures were readily found (Fig. 5). The muscularis mucosae was intact, and there were no signs of either fibrosis or endarteritis. A minor degree of round-cell infiltration was present. Though the atypical glands were situated mainly in the superficial part of the mucosa, careful search revealed their presence close to or just within the muscularis. These appearances were interpreted as pre-invasive carcinoma.

Discussion

The postoperative stomach is difficult to evaluate radiologically because of similarities between the appearances of malignancy and the distorted anatomy following surgery (Taebel and Kirsner, 1967). In patients who develop gastric symptoms more than 10 years after partial gastrectomy or gastroenterostomy radiological examination will provide the diagnosis in only half of those with gastric carcinoma (Pygott and Shah, 1968). Furthermore, gastric surgery greatly increases the difficulty of gastroscopic examination. Under these circumstances gastric cytology may play an important part in diagnosis. However, the similarities between active epithelial cells and well-differentiated carcinoma cells can provide difficulties. In the present case our interpretation of the cells, some of which are illustrated in Fig. 1, was that they represented well-differentiated adenocarcinoma cells. It is of note that such cells cannot be found in the gastric washings now that the lesion has been removed.

With regard to the histology of the specimen, the differential diagnosis lay between pre-invasive carcinoma and regenerative hyperplasia. It is well known that regenerating gastric mucosa may be irregular in pattern and the individual cells large and hyperchromatic. Such appearances are often seen at the margin of a healing ulcer, but in the present case there were no signs of previous ulceration as judged by fibrosis or endarteritis. While we hesitate to lay much emphasis simply on irregular and hyperchromatic acini, the finding of abrupt transition of columnar cells to large and highly atypical epithelial cells with mitotic figures is a strong pointer to a malignant basis for this lesion.

The extent to which gastric mucosal lesions, such as chronic gastritis and carcinoma-in-situ, can cause symptoms is unknown. However, in a nation-wide survey in the United States of America and Canada of the value of gastric cytology in the hospital diagnosis of gastric carcinoma, only 37 small cancers were diagnosed, not all of which were of the in-situ type (Ackerman, 1967). This suggests that cancers are usually relatively advanced once symptoms occur. These studies for the early detection of carcinoma should be directed towards those persons at special risk, so that screening methods can be instituted for specific groups of patients (Shearman and Finlayson, 1967). The existence of achlorhydria in the postoperative stomach, as in the case of the intact stomach (Shearman *et al.*, 1967), should arouse suspicion, and cytological examination should be made.

We wish to thank Dr. J. G. M. Hamilton, who referred his patient for cytological examination, for kind permission to publish this case. We also thank our cytology technicians, Miss Isobel Fraser and Miss Norma Reilly. We are in receipt of a grant for technical assistance and apparatus from the Scottish Hospitals Endowment Research Trust.

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Ischaemic Colitis

Brit. med. J., 1969, **1**, 361–362

It has been suggested that ischaemia of the colon occurring in the same age group and from the same causes as myocardial infarction accounts for certain cases of "segmental" colitis, particularly those involving the splenic flexure (Marston, Pheils, Thomas, and Morson, 1966).

Infarcts of the colon are not uncommon (Thomson, 1948; McCort, 1960; Marshak, Maklansky, and Calem, 1965; Varga and Currie, 1965; Sturdy, 1968), and the patient thus affected may present as an acute abdominal emergency (Brownlee, 1951; Kellock, 1957; Killingback and Williams, 1961; Marston, 1962; McGovern and Goulston, 1965). In previously reported cases the evidence for the production of an ischaemic colitis with subsequent stricture formation has been clinical, radiological, and pathological. In no instance has an obvious infarct of the large bowel been found at laparotomy and left in situ subsequently to produce an ischaemic stricture. The following case report describes such a sequence of events.

Case Report

A woman aged 73 was admitted to hospital as an emergency case on 13 June 1966 with acute colicky abdominal pain of sudden onset. There was no previous history of abdominal pain or bowel symptoms. The only relevant medical history was that of three years' angina treated with pentaerythritol tetranitrate tablets. On examination she was shocked and had signs of generalized peritonitis. Rectal examination was normal. A provisional diagnosis of mesenteric vascular occlusion was made, and after two hours of preliminary resuscitation with intravenous fluid a laparotomy was performed.