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ACHALASIA OF CARDIA, CARCINOMA OF OESOPHAGUS, AND HYPERTROPHIC PULMONARY OSTEOARTHROPATHY

BY

M. A. PEYMAN, M.A., D.M., M.R.C.P.*

Late Senior Medical Registrar, Charing Cross Hospital, London

It is generally considered that carcinoma of the oesophagus is an unusual complication of achalasia of the cardia. A case of achalasia is described below in which carcinoma of the middle third of the oesophagus developed together with hypertrophic pulmonary osteoarthropathy.

Case Report

A man aged 26 first attended the out-patient department of Charing Cross Hospital in 1931 with a history of difficulty in swallowing for the previous six months. No abnormalities were found on physical examination. A diagnosis of achalasia of the cardia was made, and he was given a mercury bougie to use regularly.

During the next 23 years he remained well and did not attend the out-patient department again until November 16, 1954, when he stated that the bougie had broken about two months previously. Since then he had developed increasingly severe dysphagia, especially for solid food, and he had lost about 14 lb. (6.4 kg.) in weight. During this period he had also experienced pain and swelling in the ankles, knees, wrists, and finger-joints. He was adamant that daily use of the bougie in the past had ensured normal swallowing.

Examination showed a thin ill-looking man with marked clubbing of the fingers and toes as well as some swelling and tenderness and slight limitation of movements of the ankles, knees, wrists, and finger-joints. The chest showed generalized emphysema and bronchospasm. There were no palpable lymph nodes and examination of the other systems was essentially negative.

A fortnight later (December 2, 1954) he was admitted to hospital for further investigation. The following results were obtained: Hb, 12.4 g./100 ml.; red cells, 4,900,000/c.mm.; white cells, 16,900/c.mm. (polymorphs 68%); E.S.R., 27 mm. in one hour; serum uric acid, 3 mg./100 ml.; serum

W.R., negative. A chest x-ray examination showed generalized emphysema with pleural thickening in the region of the oesophagus on the right side. The oesophagus was filled with food up to the level of the thoracic inlet and bulged to the right of the midline. A barium swallow showed that the oesophagus was dilated and tortuous, with evidence of oesophagitis. It was concluded that this appearance was due to achalasia of the cardia. Thin barium passed into the stomach without undue delay, but a mercury bougie could not be manoeuvred below the lower third of the oesophagus even after the inhalation of octyl nitrite. There appeared to be a filling defect in the middle third of the oesophagus (Fig. 1). A series of x-ray films of the limbs showed evidence of new bone formation in the subperiosteal regions of the bones of the hands and feet as well as of the lower parts of the radius, ulna, tibia, and fibula. The changes were those of hypertrophic pulmonary osteoarthropathy (Fig. 2).

During the next two weeks the patient received daily oesophageal lavages. At the end of this period screening examination of the chest showed that, although the oesophagus was still dilated and tortuous, a bougie could be passed through the cardia. The patient was using a bougie daily and finding that swallowing was becoming easier. On January 4, 1955, after a further fortnight of oesophageal lavages, oesophagoscopy revealed a large friable papilliferous growth involving the middle third of the oesophagus. A subsequent thoracotomy showed that the growth had spread into both hilar regions and was involving the pleura as well as becoming firmly attached to the aorta and vertebral bodies. No metastases were seen. The oesophagus was dilated and

FIG. 1.—Barium swallow showing dilated oesophagus with appearance of filling defect in middle third of oesophagus.

FIG. 2.—Radiograph showing new bone formation at lower ends of radius and ulna.

*Now at New England Center Hospital, Boston, Massachusetts, U.S.A.

hypertrophied below the lower part of the growth and there was marked narrowing of the cardia, thus confirming the presence of achalasia. In view of these findings it was decided that resection of the growth was not possible, and surgical treatment was confined to a Heller operation. Histological examination of a biopsy of the oesophageal growth revealed a well-differentiated Grade I squamous-cell carcinoma.

After operation, swallowing continued to improve and a bougie was no longer necessary. However, the general condition of the patient remained rather unsatisfactory, and the clubbing as well as the pain and swelling in the distal joints persisted. A barium swallow three weeks after operation showed that gross oesophageal dilatation was still present, and, although there was no appreciable retention of barium, a very narrow area was still present immediately below the dilatation.

The patient was discharged from hospital on February 13. During the next two months he gradually lost strength, although he still swallowed without difficulty. He died at home on April 14. A post-mortem examination was not made.

Discussion

In 1872 Hilton Fagge described the development of carcinoma within a dilated oesophagus in a man of 84 who had experienced difficulty in swallowing for over 40 years. Since then several case records of a similar nature have been published (Rake, 1931; Baer and Sicher, 1947; Gore and Lam, 1952; Kastl, 1953; Klayman, 1955; Bohn, 1957). Although most of these authors concluded that carcinoma of the oesophagus was a definite complication of long-standing achalasia of the cardia, Groves (1956) felt that the risk of carcinoma developing in patients with achalasia could not be regarded as being greater than in the general population until further case reports were available. Indeed, Vinson (1942) believed that when carcinoma of the lower end of the oesophagus was found in patients with achalasia it could be assumed that the carcinoma was in fact the original lesion. However, this seems unlikely, since in most of the reported cases, including the one described here, there had been a long history of preceding difficulty in swallowing. Thus Baer and Sicher in a review of nine cases found that the shortest period from the onset of dysphagia until the discovery of malignancy was 10 years, while the average duration of symptoms was 19 years. Admittedly there was only a five-year history of dysphagia in one of the cases described by Klayman, but it is extremely unlikely that an oesophageal carcinoma existed throughout this period. As Baer and Sicher suggest, the apparent infrequency of an association between achalasia and carcinoma of the oesophagus may well be explained by the difficulties that beset the diagnosis of this combination.

Rake has emphasized the effects of stagnation of food and saliva in the oesophagus of patients with achalasia. He pointed out that the development of acute and chronic oesophagitis with ulceration is likely to be followed by attempted healing, which may in turn lead to papillomatous overgrowth of the epithelium. That author regarded the continual irritation of the oesophageal mucosa as a dangerous process which predisposed to carcinoma. In the majority of cases in which carcinoma has followed upon achalasia of long duration, the neoplasm has arisen in the middle third of the oesophagus. This site of predilection may be related to the fact that in patients with achalasia the

column of food and fluid often extends as far up as the mid-oesophagus. It is conceivable that the fluid level causes a particularly marked degree of irritation of the mucosa. Although in the above case the patient had apparently remained free of dysphagia during the 23-year period of daily bougienage, it is quite possible that there was in fact a certain degree of intermittent retention of fluid in the lower half of the oesophagus. Thus it is well recognized that achalasia is sometimes asymptomatic, and occasionally a grossly dilated oesophagus is first discovered during the course of a mass radiography survey (Garland and Phillips, 1953). It is likely that the risk of carcinoma is negligible only in those patients in whom permanent dilatation of the cardia is achieved during the early stages of the disorder. Another factor which may have contributed to the development of chronic irritation of the oesophageal mucosa in this patient was the daily passage of a mercury bougie for a total period of 23 years. Indeed, such a possibility may constitute an additional reason for the early adoption of permanent dilatation in these patients.

If it is accepted that patients with achalasia occasionally develop carcinoma in the oesophagus, then it is important to appreciate the early manifestations of this complication. According to Baer and Sicher, suspicious symptoms include recurrence of dysphagia, more marked for solids than fluids, as well as blood-stained vomit, retrosternal pain, and excessive loss of weight. However, as Groves explained, the presence of the enlarged oesophagus may prevent aggravation of the dysphagia appearing as an early feature, swallowing not becoming really difficult until the cervical part of the oesophagus or the recurrent laryngeal nerves are involved. In the above case dysphagia developed after the breakage of the bougie but responded well to subsequent medical and surgical treatment, suggesting that this particular disability was due to the achalasia. Groves stated that an unexplained deterioration in the general condition of the patient may be the only indication that carcinoma has developed. Unfortunately, as Baer and Sicher stressed, even radiological examination of the oesophagus does not always exclude a carcinoma in the presence of well-established achalasia. In practice it is now generally accepted that oesophagoscopy is a more valuable diagnostic aid in these patients. Indeed, some authors, such as Rake, Gore and Lam, and Kastl, have suggested that oesophagoscopy should be carried out at regular intervals in all patients with achalasia, while Klayman has discussed the value of exfoliative cytological examination in the early diagnosis of oesophageal carcinoma. Klayman actually considered that this special technique should be employed annually as well as in the event of any exacerbation of symptoms in every patient with achalasia, in addition to radioscopy and oesophagoscopy. Certainly there seems no doubt that these patients should be seen at regular intervals, probably at least once every six months, and, if any suspicious features have developed, radioscopy and oesophagoscopy should be immediately undertaken. An important point emphasized by several authorities is the necessity for carrying out oesophageal lavage before either of these diagnostic procedures is performed, as otherwise a carcinoma may be missed.

It was probably a coincidence that the bougie of this patient broke less than four months before the diagnosis

of oesophageal carcinoma was established, although it is conceivable that the carcinoma offered additional obstruction to the passage of the instrument. Another possibility is that the carcinoma actually developed after the bougie had broken during the period when the achalasia was no longer under control. However, this sequence of events seems very unlikely, since examination of the chest contents at thoracotomy less than four months later revealed that the carcinoma had already extensively invaded the structures around the oesophagus.

It is generally accepted that hypertrophic pulmonary osteoarthropathy is rarely associated with diseases of the oesophagus. However, Brooks (1913) observed that hypertrophic osteoarthropathy was sometimes a feature in patients with mediastinal neoplasms which had invaded pulmonary tissue. Although in the above patient it is possible that clubbing had existed for a considerable period, the symptoms of hypertrophic osteoarthropathy were first noticed less than three months before his admission to hospital. For this reason it seems likely that the specific changes which developed in his distal bones and joints were associated with the spreading oesophageal carcinoma rather than with the achalasia. If this was so, the development of hypertrophic osteoarthropathy or even of simple clubbing should be regarded as highly significant in any patient with achalasia. Unfortunately, however, if the suggestion made in this case is correct, the appearance of these physical signs is likely to imply that the carcinoma has already spread to the pleura and pulmonary tissue. According to Flavell (1956) and Brea (1957), the severe joint pain of hypertrophic pulmonary osteoarthropathy may be relieved by vagotomy or hilar denervation. If this neurogenic theory is correct, it is not surprising that the pain in the limbs of this patient persisted after thoracotomy, since, although a Heller operation was performed, the hilar regions and the vagi were left intact.

Summary and Conclusions

A case of achalasia of the cardia of 23 years' duration is described in which carcinoma of the middle third of the oesophagus developed in association with hypertrophic pulmonary osteoarthropathy.

The occasional development of carcinoma of the oesophagus in patients with long-standing achalasia is emphasized. Although the early diagnosis of this complication is often difficult, regular observation of patients with achalasia is important. If a superimposed oesophageal carcinoma is suspected, oesophagoscopy as well as radioscopy, preceded by adequate lavage, should be promptly carried out. Exfoliative cytological examination may also be of value in these patients.

Probably the risk of carcinoma in patients with achalasia is negligible only in those in whom permanent dilatation of the cardia is achieved during the early stages of the disorder. It is possible that the daily passage of a bougie over many years further contributes to the development of chronic irritation of the oesophageal mucosa.

The development of hypertrophic pulmonary osteoarthropathy in patients with achalasia may be an indication that an oesophageal carcinoma has developed, with secondary spread to the pleura and pulmonary tissue.

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NOSE-BLEEDING AND HIGH BLOOD PRESSURE

BY

J. R. A. MITCHELL, M.B., B.Sc., M.R.C.P.

Registrar to the Regius Professor of Medicine,
Radcliffe Infirmary, Oxford.

Nose-bleeding is often said to be associated with high blood pressure. This belief is perpetuated by the standard textbooks of nasal disease and general medicine, in statements based on clinical impressions unsupported by objective quantitative data. One reads that epistaxis frequently accompanies high blood pressure (McLaggan and Collier, 1952); that hypertension is a common cause of nose-bleeding (Reading, 1953); that arterial hypertension often gives rise to epistaxis (Garland and Phillips, 1953); and that hypertension is the commonest systemic cause of epistaxis (Hunter, 1956).

This survey attempts to assess the validity of these statements by determining the frequency of nose bleeding in patients with high blood pressure, and the blood-pressure levels in patients presenting with nose bleeding.

Material

The hospital notes of two groups of patients were reviewed.

Series I: Patients Known to Have High Blood Pressure.—This group consisted of (a) 173 patients (86 males, 87 females) currently attending the hypertension clinic at Radcliffe Infirmary, and (b) 201 patients (89 males, 112 females) with diastolic pressures of more than 100 mm. Hg, culled from the patients admitted to the Radcliffe Infirmary in the 12-months period January–December, 1956. Of this subgroup, 143 (71%) were admitted because of high blood pressure, cardiac or cerebral vascular disease; 24 (12%) were admitted for surgical treatment; 20 (10%) for gynaecological

TABLE I.—Age Incidence of 374 Patients with High Blood Pressure (Mean Age, 57.4 Years)

Age (Years)	No. of Patients	Age (Years)	No. of Patients
Under 30	8 (2.1%)	60–69	99 (26.5%)
30–39	20 (5.4%)	70–79	47 (12.6%)
40–49	62 (16.5%)	80+	7 (1.9%)
50–59	131 (35.0%)		

procedures; 7 (3.5%) on account of diabetes mellitus, and 7 (3.5%) with skin conditions. The age structure of the whole series, comprising 374 patients (175 males, 199 females) is shown in Table I.

Series II: Patients Presenting with Nose-bleeding.—In this group were 162 patients (86 males, 76 females) admitted to the E.N.T. department of the Radcliffe