DIVERTICULA OF THE THORACIC OESOPHAGUS

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Diverticula of the thoracic oesophagus are comparatively uncommon. Harrington1 treated 216 patients with pharyngooesophageal diverticulum, while during the same period he treated only 8 patients with thoracic oesophageal diverticula. In an analysis of 1,600 patients admitted to the Out-patient Department of the Massachusetts General Hospital because of dysphagia, Macmillan² found less than 1% due to diverticula of the thoracic oesophagus. The true incidence of thoracic diverticula is probably much higher since these diverticula may produce no symptoms. Soulas,3 states that from 1947 to 1951 the ratio of pharyngeal to thoracic pouches in his cases has been reversed, being now respectively 35 and 65%. The lower the level in the thoracic oesophagus, the greater the frequency of diverticula. In Vinson's review4 of 42 cases of diverticula of the thoracic oesophagus, 24

were located in the lower third. The majority of cases occur in persons of the 40-60 age-group.

A pure topographical description such as middle third or lower third, epibronchial or epiphrenal pouches, is to be preferred to the terms traction pouches and pulsion pouches. These terms are probably misleading and inaccurate since a sum of primary and secondary factors is usually responsible (see below).

The pulsion type denotes protrusion of the wall or part of the wall which is produced by raising the intra-luminal pressure. The traction diverticulum results from fixation of a localized area of oesophageal wall to an adjacent structure more rigid than itself. With the contraction of scar tissue or the displacement of one structure away from the other, the oesophageal wall is dragged out in a tent-like fashion.

A pulsion-traction diverticulum means a combination of both types.

Barrett⁵ gives a fascinating account of how chronic inflammation and calcification in the lymph system leads to shortening of strong bands of fibrous tissue between the anterior wall of the oesophagus and the epibronchial glands. These bands were originally described by Ribbert. Barrett also mentions that a few cases have been associated with adhesions in the vicinity of caries of the spine and pericarditis.

In view of the frequent occurrence of tuberculous adenitis, a tuberculous process can but rarely be a cause of epibronchial diverticula. Johnstone⁶ believes that attachment of the oesophagus to the aorta results in the oesophagus being pulled away from the left bronchus as the aorta unfolds. If any adhesions exist between the oesophagus and the bronchus or its adjacent structures, traction diverticula may form, causing a potential weakness in the wall.

A primary factor in the aetiology of thoracic diverticula is an area of structural weakness created by gaps in the muscle wall of the oesophagus, associated with the passage of blood vessels.⁷

It is not difficult to visualize mucosa herniating through these weak spots in the oesophageal wall. Shaw,⁸ in an excellent review of thoracic oesophageal diverticula, postulates that those pouches which do possess a muscle wall may equally well be explained as being complete congenital entities, subsequently enlarged. In support of this he cites those cases which have been reported post mortem and radiologically in the newborn infant and in older children.

Functional or false diverticula are simply areas of apparent pouching of the oesophageal wall, usually in the lower third, due to a local hypotonic state of the smooth muscle layer, associated with adjacent hypertonus or apparent stricture formation. They may often disappear at rest and are only thrown into prominence during what is usually abnormal peristalsis.

In addition to the primary factors such as areas of congenital weakness in the oesophageal wall and traction due to adherence of the oesophagus to surrounding structures, Shaw discusses the following secondary factors in his paper:

- 1. Pulsion. In the normal human oesophagus, primary and secondary peristaltic waves cause pressure sufficient to maintain a vertical column of water attached to its distal end at a height of $2\frac{1}{2}$ feet. Several authorities regard this pulsion pressure as the most important single secondary factor in most cases.
- Anatomy of the lower oesophagus. The majority of cases show pouching from the right lateral wall towards its anterior aspect, usually 2-3 inches above the diaphragm.
 In this situation the mechanical support of the peri-oesophageal tissues is least effective.
- 3. Effect of cardiospasm. This is not considered an important factor by Johnstone, who examined at least 200 cases of achalasia without finding a diverticulum. In a far greater number of cases of obstruction of the lower end of the oesophagus from other causes only one diverticulum was found. Shaw, on the other hand, quotes from 5 different papers in which cardiospasm has been an important contributing cause.
- 4. Direct pressure of diverticulum. The larger a dependent pouch becomes, the greater the tendency for its fundus to cause pressure on the subdiverticular oesophagus, thus

setting up a vicious circle which may precipitate symptoms in a patient previously unaware of trouble.

- 5. Atrophic changes. With increasing age there is an inevitable small degree of atrophy in the muscular walls of the oesophagus, together with loss of elasticity in the supporting tissues and frequently some degree of chronic oesophagitis. These changes may partly account for the greater incidence in middle-aged and older people.
- 6. Local sepsis. Changes arising in the walls from chronic oesophagitis may also contribute to pouch formation. Adhesions due to extra-mural suppuration more often produce a primary effect by traction.
- 7. Individual habits. Certain bad habits, such as air swallowing, poor mastication and bolting of food, which are not uncommon in middle-aged edentulous men, have a well known association with oesophageal disorders.

Symptoms

Diverticula of the thoracic oesophagus sometimes produce no symptoms. The occurence of symptoms depends on the size of the diverticulum, the direction in which the diverticulum points, the size of the diverticular opening into the oesophagus, and associated conditions such as chronic subdiverticular stenosis or chronic oesophagitis. Small diverticula or diverticula which are directed upwards and have a wide opening into the oesophagus rarely produce symptoms. The actual symptoms may be chronic dyspepsia, retro-sternal discomfort and variable dysphagia. There may be intermissions due to varying degrees of oesophagitis, In rare cases the contents of a large pouch may be regurgitated on lying down.

Complications are those common to all diverticula of the gastro-intestinal tract, viz. infection, perforation, haemorrhage and malignant change.

Diagnosis

Radiology and oesophagoscopy are essential in diagnosis. The use of effervescent barium milk, which will rapidly find its way into a diverticulum and produce a clearly defined gas bubble with fluid level, is recommended. Shaw states that oesophagoscopy should be carried out with 4 principal objects in view:

- 1. To find the mouth of the pouch and evacuate its contents,
- 2. to examine the interior of the pouch and exclude the presence of malignant disease,
- 3. to take smears and biopsy material from any suspicious areas, and
- to examine and explore the oesophagus in its whole length and to establish the presence and nature of any associated condition.

Treatment

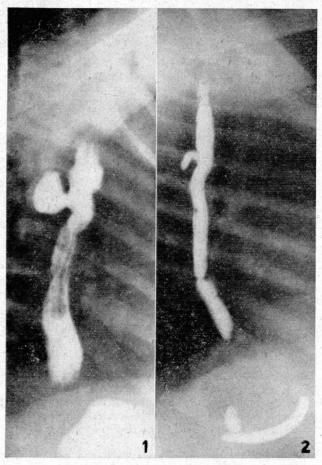
Pouches causing no symptoms require no treatment. In pouches associated with other pathological conditions the latter must be treated first. Surgical procedures employed are (1) extirpation of the sac, (2) diverticulopexy and (3) inversion of the sac. In cases of epiphrenic diverticula anastomosis between the diverticulum and the cardia of the stomach is sometimes practised.

CASE REPORT

C.A., aged $2\frac{1}{2}$ years, a Coloured female child, was admitted to the Red Cross War Memorial Children's Hospital in October

1956 with the following history. She had been quite well until July of that year, when the mother noticed that she regurgitated food she had recently eaten. Since that time the child had subsisted on a milk diet, which was straining the family budget. There was no history of having swallowed corrosives or a foreign body.

On examination no clinical abnormality was detectable. The weight was 26 lb. and the Mantoux test (1 in 1,000 old tuberculin) was negative on two occasions.



A barium swallow (Fig. 1) was X-rayed, and an oesophageal diverticulum which projected backwards and slightly to the right was demonstrated. An area of narrowing was noted below the diverticulum

On 22 October an oesophagoscopy (D.V.M.) was performed. The diverticulum was easily identified and a biopsy was taken from the thickened polypoidal mucosa around its opening. There was considerable narrowing of the oesophageal lumen immediately below the diverticulum, beyond which the only endoscope available on this occasion (a Negus child's oesophagoscope) could not be passed, so that the oesophagus distal to the diverticulum was not explored.

On histological examination of the biopsy specimen, the oesophageal mucosa was found to be infiltrated with chronic inflammatory cells. After the oesophagoscopy the patient was able to swallow without difficulty and she was accordingly discharged to be kept under out-patient observation. She did not, however, re-attend until 11 March 1957, when she was re-admitted, having experienced a recurrence of her former symptoms. The barium swallow was repeated and a similar radiological picture to that seen on her previous admission was obtained.

On 16 March the oesophagoscopy was repeated. Again with a Negus child's oesophagoscope the oesophagus below the diverticulum could not be entered. A child's bronchoscope was

then used and with that a flat plastic foreign body, $20 \times 8 \times 2$ mm., was identified below the level of the diverticulum and removed. The patient's subsequent course was uneventful and she was discharged on a normal diet.

On 10 June the barium swallow was repeated (Fig. 2) and this showed that the oesophageal diverticulum was considerably smaller than on the previous examination. The oesophageal lumen showed no abnormality.

The patient had remained free of symptoms.

Comment

This case is of interest because in the literature reviewed we have not seen a case of oesophageal foreign body associated with a diverticulum reported. It is suggested that oesophageal obstruction caused by the foreign body contributed to the periodic rise of intraluminal pressure, which resulted in a pulsion diverticulum.

The marked improvement in the patient's swallowing after the first oesophagoscopy was surprising. In retrospect a probable explanation for this was tilting of the foreign body into a less obstructing position as a result of the endoscopic manipulation.

Caffey⁹ states that in children a foreign body should be carefully looked for whenever spasm and oesophageal obstruction are demonstrated.

When a barium swallow is carried out, a foreign body may cause one or more of the following abnormalities:¹⁰

- 1. A hold up of the column of barium. The object may be wholly or partly outlined by the barium and may be recognized by its shape.
- 2. A deviation or forking of the stream as the barium passes over the object.
- 3. A spasm or abnormal contraction of the oesophageal wall, usually at the point of impaction.
- 4. A small residue of barium, often like a streak, may remain stationary within the lumen. Attempts to dislodge it with drinks of water may at first be unsuccessful, because the barium adheres to a foreign body. On the other hand, fine streaks due to barium lying in mucosal folds are readily washed down. The outlining of the object with barium is the most valuable and characteristic sign.

The considerable reduction in size of the diverticulum in the present case after 3 months is interesting. We intend repeating the barium swallow examination in 6 months' time to ascertain whether the diverticulum has become smaller still.

SUMMARY

- 1. The incidence of thoracic diverticula is discussed.
- 2. A topographical classification of thoracic diverticula is recommended. The terms traction, pulsion and functional diverticula are defined.
- 3. The aetiology is discussed and brief mention is made of the symptomatology, diagnosis and treatment of thoracic diverticula.
- 4. A case of an epibronchial diverticulum associated with an oesophageal foreign body is reported.

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REFERENCES

- 1. Harrington, S. W. (1949): Ann. Surg., 129, 606.
- 2. Macmillan, A. S. (1935): Surg. Gynec. Obstet., 60, 394.
- 3. Soulas, A. (1951): Ann. Oto-laryng. (Paris), 68, 529.

- 4. Vinson, P. P. (1934): Arch. Otolaryng. (Chicago), 19, 508.
- 5. Barrett, N. R. (1933): Lancet, 1, 1009.
- Johnstone, A. S. (1949): Brit. J. Radiol., 22, 415.
 Mosher, H. P. (1930): J. Laryng., 45, 161.
- 8. Shaw, H. J. (1954): Ibid., 68, 70.
- 9. Caffey, J. (1950): Paediatric X-ray Diagnosis, p. 487. Chicago:
- Year Book Publishers.

 10. British Authors (1950): A Textbook of X-ray Diagnosis, 2nd ed., 3, 33. London: H. K. Lewis.