CARDIOSPASM IN AN AFRICAN: A CASE REPORT

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Cardiospasm does occur in the African. A report is presented of a case that was recently seen at the Non-European Hospital, Johannesburg.

A suitable definition of cardiospasm is a 'condition in which without demonstrable obstructive pathological change, and usually without pain, food does not pass readily into the stomach from the oesophagus, but is held up in the oesophagus, which in the majority of cases undergoes dilatation, sometimes extreme'.

The term cardiospasm has been used for a long time for conditions of benign stenosis of the terminal portion of the oesophagus, in which peristaltic waves in the oesophageal musculature are deficient or absent. This was thought to represent a spasm at the cardia (therefore cardiospasm), but in 1930 Hurst and Rake noted a degeneration of Auerbach's plexus in the oesophageal wall and stated that 'muscular contractions of the oesophagus were inadequate, often failing to reach the cardia', and offered the name 'achalasia'. Recently (1957) further studies on the physiology of deglutition have shown that the dysphagia is due to a motor disturbance, manifested by incomplete propagation of primary peristaltic waves, with an inhibition of relaxation of the inferior oesophageal sphincter.

CASE REPORT

An African male, J.P., a kitchen servant aged 40, born in Pietersburg, was admitted on 12 May 1959, complaining of dysphagia since 1957. The dysphagia had been progressive and was treated at another hospital in 1958, where gastrostomy was performed. On admission he could only eat finely divided foods. After eating he vomited. He felt the food sticking at the level of the xiphisternum. He had lost much weight. His appetite was poor. He did not drink, and smoked 1-2 cigarettes daily.

On examination we found him very thin, underweight and malnourished (weight 94 lb.). He had a soft diffuse parotid and submandibular swelling on each side; also bilateral gynaecomastia (since puberty) and soft axillary gland swellings. Lungs and heart normal. Blood pressure 85/55 mm. Hg. Pulse rate 80 per minute. The abdomen indicated much loss of weight and showed 2 scars, viz. (a) from gastrostomy, and (b) a 2-inch vertical mid-line epigastric scar with some keloid formation. Liver, one finger enlarged. No other abdominal pathology found clinically. No oedema of limbs. The results of special examinations were as follows:

Wassermann reaction negative.
Haemoglobin 18·4 g.%. Leucocytes 16,200 per c.mm. (neutrophils 78·7%, lymphocytes 16·0%, monocytes 3·0%, eosinophils 3·0%).
Sedimentation rate 4 mm. per hour.
Serum acid phosphatase 1·9 units. Serum alkaline phosphatase 10·5 King-Armstrong units.

Faeces dark brown, solid, occult blood present (orthotolidine amidopyrin reaction +). Oesophagoscopy (J.K.) showed gross distension of the oesophagus. The mucosa was thickened and was the site of chronic oesophagitis with small areas of ulceration. The cardio-oesophageal orifice was easily visualized after aspiration of a large quantity of whitish fluid; it was dilated to size 37F oesophageal dilator, when the oesophagoscope passed easily into the stomach. A biopsy of the lower end of oesophagus was done.

Biopsy sections showed evidence of extensive ulceration with a small portion of stratified squamous epithelium remaining. There was well-marked inflammatory reaction with an infiltration of polymorphonuclear leucocytes. Structures which had the histological features of fungal hyphae and spores were observed. No evidence of malignant neoplasia was seen. A diagnosis of mycotic oesophagitis was made.

X-ray investigation (H.S.). A barium swallow showed the entire length of the oesophagus from the cardio-oesophageal junction upwards to be grossly dilated and sigmoid shaped. It contained a large quantity of fluid and undigested material. An initial hold-up took place at the cardio-oesophageal junction lasting up to approximately 30 minutes, when no barium entered the stomach. At 30 minutes a small quantity of barium was seen to trickle through the cardio-oesophageal junction, more marked during expiration (Fig. 1). The beak-shaped deformity of the cardio-oesophageal junction which is typical of cardiospasm was demonstrated in the serial radiograph. After the administration of amyl nitrate there was a dramatic release at the cardio-oesophageal junction (also characteristic of cardio-
spasm) and good emptying took place (Figs. 2 and 3). The normal mucosal pattern of the stomach was outlined.

On X-ray examination of the chest, the large oesophagus with remnants of barium still within it was seen through the heart border. There was no radiological evidence of the pulmonary parenchymal involvement that is frequently seen at the lung bases in cardiospasm.

DISCUSSION

The rarity of cardiospasm in the African may be gauged from the fact that this case is only the second diagnosed at the Non-European Hospital, Johannesburg, from 1949 to 1959, during which time 57,600 surgical and medical outpatients were seen. From large institutions for Africans such as Baragwanath and Coronation Hospitals, Johannesburg, only 7 cases were traced over a period of 5 years. Burrell, who has done much work on lesions of the oesophagus in the Transkeian African, has not seen any case of cardiospasm in the last 3-4 years. We have been unable to find any other statistical information about the incidence of this condition in the African.

Little is known of the aetiology of cardiospasm. Gammie et al. recently suggested causes such as toxic, infective, senile, and nutritional. Other authors, such as Etzel working among Brazilians of poor economic status, suggested that lack of thiamine may play a role. Weiss has suggested that there is a psychosomatic element, especially in the early stages of the malady. In our case there was certainly a high degree of malnutrition manifested by the patient's low weight and the presence of enlarged salivary glands and gynaecomastia.

Whether his cardiospasm preceded or followed the malnourishment is a moot point.

Since it is universally accepted that cardiospasm is one form of disturbed oesophageal transport a programme of investigation is recommended, comprising (1) careful history and examination, (2) barium swallow, (3) detection of occult blood in the stools, (4) oesophagoscopy, and (5) the mecholyl test (see below).

The sensation of obstruction may be referred to the lower, and occasionally to the upper, portion of the oesophagus. In the early stages it may be intermittent and warm foods are taken more easily than cold. Solids may be preferred to liquids. Regurgitation is common and may be brought about deliberately at a later stage. Bleeding from the oesophagus is rare in the early stages.

X-ray findings may show a dilated oesophagus with no mobility. In early cardiospasm a stage of compensation may exist, in which frequent and inefficient, poorly coordinated oesophageal contractions force food into the stomach. In advanced lesions a decompensated stage is reached in which only gravity and changes in the intra-thoracic pressure can overcome the resistance at the cardia.

Of particular diagnostic significance is the response of the achalasia to the injection of acetyl-beta-methylcholine chloride (mecholyl). The radiologist will see a pronounced contraction of the entire oesophagus and there is a prolonged increase in pressure. In normal patients and in patients with other conditions there is no such response. This sensitivity to mecholyl is explained by Cannon's law of autonomic denervation. Conversely the cardiac end of the oesophagus tends to dilate on the administration of amyl nitrite (see above).

Differential Diagnosis

The chief problem is to differentiate cardiospasm from organic lesions such as carcinoma. The differentiation at the lower end of the oesophagus may be very difficult and, if biopsy is inconclusive, cytologic studies of smears and secretions may be of help. If doubt still exists an actual exploration of the cardia is indicated.

Large benign tumours may simulate the X-ray picture of cardiospasm, but careful endoscopy should clarify the situation.

Hiatal hernias and oesophagitis give a long history of heartburn and regurgitation of 'acid'. By the time a stricture has formed the oesophagus is usually shortened.

Scleroderma may first manifest itself as a cardiospasm-like picture, but Raynaud phenomena are almost always present if there are no skin manifestations.

Perhaps the condition most often confused with cardiospasm is the non-organic disorder of diffuse spasm (non-sphincteric spasm, pseudo-diverticulosis or segmental spasm of the oesophagus). This condition occurs in nervous, tense people of both sexes with a slight preponderance in women. Pain is a striking feature, occurring substenally with reflex spread to arms, jaws, ears and back. Dysphagia is very variable. Radiographically the condition involves the lower half of the oesophagus. Diffuse spasm is the physiological opposite of cardiospasm; it is manifested by simultaneous contractions at various levels in response to the swallowing reflex. The mecholyl test is useful in differentiation. Diffuse spasm may be associated with other lesions of the gastrointestinal tract, such as peptic ulcer, cholelithiasis and pancreatitis.

Treatment

Briefly, treatment may be divided into the non-surgical and the surgical. The non-surgical treatment is repeated dilatation; it is best done by forceful stretching with a well-placed cylindrical bag filled with water or air. In one series' 80% were permanently relieved by this method, in which there is a 2% risk of splitting the oesophagus. Antispasmodic drugs have little or no effect.

Surgical procedures of many types have been used, for example Wendel's cardioplasty and gastro-oesophagectomy. The best results, however, may be obtained by Heller's procedure with Znaiger's modification, which consists of an oesophagomyotomy, freeing the narrowed oesophageal segment from its muscular coat; in one series the satisfactory results amounted to 85%.

SUMMARY

1. A case of cardiospasm in the African is presented.
2. The roentgenologic findings are illustrated.
3. The rarity of this condition in the African is stressed.
4. The aetiology, diagnosis and differential diagnosis are discussed.
5. The treatment is summarized.

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