

Oesophageal obstruction following hyperemesis gravidarum

R. ABBEY SMITH AND C. S. NELSON

From the Thoracic Surgical Unit, King Edward VII Chest Hospital, Hertford Hill, near Warwick

Although since 1921 (Vinson, 1921) severe obstruction of the oesophagus has been known to follow vomiting in pregnancy, little is known about the subject, and the significance of a history of vomiting in pregnancy is not always recognized. We record five patients with severe oesophageal obstruction thought to be caused by hyperemesis gravidarum. The poor outlook for spontaneous and complete cure of the oesophageal obstruction is apparent.

It is generally believed that post-pregnancy oesophageal obstruction is associated with a hiatus hernia and is due to oesophagitis from vomiting or reflux of gastric juice, producing a fibrous stricture in the oesophagus (Allison, Johnstone, and Royce, 1943; Rennie, Land, and Park, 1949; and Cranmer, 1955). In one of our patients a hiatus hernia could not be demonstrated on repeated examination. A number of points regarding the cause of the obstruction will be discussed.

Dutton and Bland (1953) observed nine patients in a maternity unit between 1950 and 1952. Each of these nine patients had a sliding hiatus hernia. One patient suffered transient dysphagia; another developed severe dysphagia within three months of delivery. This patient vomited excessively from the twentieth week of pregnancy. The oesophageal stricture, which was the cause of the obstruction, was subsequently resected. These authors suggest the need for a large-scale investigation to ascertain the role of hiatus hernia in the vomiting of pregnancy; they state that a patient with a hiatus hernia may be symptom-free.

Marchand (1955) examined 250 women within one week of delivery; 180 (70%) experienced heartburn during pregnancy, sufficiently severe in 93 to require a regular antidote. Of these, only three had symptoms before pregnancy. Of 14 patients examined radiologically, none was found to have a hiatus hernia and no patient with dysphagia was recorded. These figures suggest that reflux occurs in pregnancy where no hiatus hernia exists. The apparent anomalies between Marchand's findings and those of Dutton and Bland are presumably explained by the higher inci-

dence of abnormal pregnancy in the latter's patients. It seems certain that dysphagia is more likely to occur after pregnancy complicated by hyperemesis gravidarum, and is more commonly encountered therefore in a maternity unit treating complicated pregnancy. The presence of a gastro-duodenal (Ryle) tube in the oesophagus and stomach may be important in the genesis of oesophageal stricture. The subject has been discussed by Douglas (1956). He regarded the Ryle tube as a factor of secondary importance to the excessive vomiting; he stated that 'the combination of these three factors, regurgitation, the gastro-duodenal tube, and debilitation, may result in a stricture being formed'. Hussain (1964) described a 22-year-old primigravida suffering from hyperemesis gravidarum who complained of dysphagia one week after the removal of a Ryle tube which had been passed to treat excessive vomiting. A stricture of the oesophagus developed and was resected eight months later; a hiatus hernia was present.

Intubation of the oesophagus cannot seriously be considered as a cause in our patients, nor is there evidence that relaxation of the controlling mechanism at the cardia, due to anaesthesia or the ingestion of irritant anaesthetic agents (suggested by Hara (1949) as a possible cause), was a factor of any importance in the production of the oesophageal obstruction.

To avoid repetition in the case histories of our patients a number of features common to each must be mentioned. It was ascertained from the parents of each of these patients that none of the patients ever suffered from feeding difficulties, excessive vomiting, or difficulty in swallowing during infancy. The importance of this will be discussed later. Each suffered from hyperemesis with excessive vomiting during her pregnancy and was told, when the dysphagia began, that the condition was of functional origin. Indeed, in two patients psychiatric treatment was given. No patient complained of dysphagia whilst carrying the baby. Fear of exacerbating symptoms was given as the reason for four of the patients

deciding against further pregnancy, even though three were without living offspring. None has swallowed normally since the onset of the troubles and all stress the fact that they are worse when under emotional strain. These symptoms, the gross dietary restrictions, and the long periods in hospital have completely altered each of these young patients' way of life in a most frustrating manner.

SUMMARY OF CASE REPORTS

CASE 1 Mrs. M. at the age of 22 developed hyperemesis in the twenty-eighth week of her first pregnancy in 1952. Two weeks later she was delivered of a normal, premature baby, which died within 24 hours. The vomiting ceased. Her post-partum progress was uneventful except that she developed dysphagia within one week of delivery. Six months later oesophagoscopy confirmed the presence of a stricture in the oesophagus at 38 cm. No oesophagitis or ulceration was present and no biopsy was taken. A barium swallow examination confirmed the presence of a hiatus hernia with reflux, with a stricture at the lower end of the oesophagus. The patient continued to complain of severe dysphagia for solid food, and six months later a further oesophagoscopy was carried out. Because of continued dysphagia the hiatus hernia was repaired by a standard method in April 1953. After this operation her dysphagia and vomiting continued, and on the fourth post-operative day a further dilatation of the stricture was performed. There was some improvement after this operation, but within three months of the repair her total dysphagia recurred.

During the next five years the patient had periods of remissions alternating with periods of total dysphagia and regurgitation. She lived principally on a milk diet. Her symptoms remained so severe and her mode of life was so changed by these symptoms that some further procedure became necessary. In 1957 the stricture was resected and a free jejunal graft was placed between the lower end of the oesophagus and the stomach. She obtained dramatic relief from this operation and had no dysphagia for the next seven years. In August 1964 a further barium swallow showed no evidence of obstruction in the reconstructed oesophagus, but the presence of two large gastric ulcers was noted. These gastric ulcers are being treated by a medical regime. Throughout the course of the illness, dating back to 1952, the patient never had a Ryle's tube passed, was never anaesthetized, and was never jaundiced. She lost a total of 39 lb. in weight. Her blood group is A, rhesus positive.

CASE 2 Mrs. H., aged 30, developed hyperemesis during the last few weeks of her second pregnancy in 1950. At full term she was delivered of a normal male baby. Her vomiting ceased immediately after delivery, but on the night of the baby's birth she

complained of dysphagia. This continued intermittently, and within two weeks of delivery a barium swallow examination was carried out which showed no hiatus hernia but a stricture just below the level of the aortic arch. Eight months after delivery an oesophagoscopy was carried out, which confirmed the presence of a stricture at 29 cm. from the lower incisor teeth. The patient continued to have intermittent dysphagia for solid food, and during the next 10 years she had four dilatations of the oesophagus at oesophagoscopy, with symptomatic relief on each occasion. Her present state is that she can swallow food fairly well except when under some form of emotional stress; she finds 'eating in company' particularly difficult. A barium swallow carried out in October 1964 is reported on as follows: 'No intrinsic lesion has been demonstrated in the oesophagus at this examination. There is no delay in swallowing thick or thin barium; there is no evidence of any stricture. There is no evidence of any disorder of the swallowing mechanism; there is no hiatus hernia or reflux. No intrinsic lesion has been demonstrated in the stomach.' Throughout the course of the illness, dating from 1950, the patient never had a Ryle's tube passed and had no anaesthetic. She was jaundiced before delivery of the child. She lost a total of 37 lb. in weight. Her blood group is O, rhesus positive.

CASE 3 Mrs. C., aged 24, started to vomit at the beginning of her first pregnancy in 1958. At the thirty-second week she began to vomit profusely, and was admitted to a maternity unit in a state of collapse. She was delivered of a female baby at full term, and her vomiting immediately ceased. From the day of delivery she noticed dysphagia for solid food; this continued during the first three weeks of the post-partum period, and she was admitted to a psychiatric hospital for treatment of a supposed nervous disorder of swallowing. Her dysphagia became total and the patient was then admitted to this hospital, where she was found on barium swallow examination to have a hiatus hernia with reflux and a stricture at the junction of the lower and middle thirds of the oesophagus. She was unwilling to undergo any form of surgery, and over the next six years had six dilatations of the stricture at oesophagoscopy, without lasting benefit. The first oesophagoscopy carried out in March 1959 confirmed the presence of a stricture at 24 cm. from the lower incisor teeth. The patient had lived for the past six years predominantly on a milk diet, and during periods of abnormal difficulty may be unable to take more than one pint of milk daily in amounts of about 1 oz. or so during each of her waking hours. At present she is unable to swallow any solid food, and even on a soft diet food is held up at the level of the stricture. Her present barium swallow demonstrates the presence of a stricture in the mid-oesophagus. During the course of her illness the patient had a Ryle's tube passed for 24 hours three days before delivery; she has never been jaundiced, had gas and oxygen anaesthesia for

delivery of her baby, and lost a total of 82 lb. in weight during the first six months of her illness. Her blood group is A. rhesus positive.

CASE 4 Mrs. B., aged 27, developed hyperemesis during the thirty-fourth week of her first pregnancy in 1950. All her symptoms disappeared after the normal delivery of a premature baby. She first complained of dysphagia at the time of her first post-natal visit. Six weeks after delivery she was admitted to hospital as an emergency owing to vomiting and total dysphagia. A barium meal examination showed a hiatus hernia and the presence of a stricture at the lower end of the oesophagus. She was first oesophagoscoped in 1951, and the presence of a stricture in the oesophagus at 37 cm. from the incisor teeth was confirmed. Because her symptoms continued her hiatus hernia was repaired in May 1953 with dilatation of the stricture. After this operation she remained almost symptom free for nearly 18 months; she has, however, had four separate periods of impaction of solid food over the last 10 years. She was oesophagoscoped on three occasions in 1955 because of food impaction, and on one occasion in 1962, when a portion of meat was removed from the lower end of the oesophagus, impacted above the level of a mild stricture. Her present state is similar to that of case 1. She can eat a normal diet fairly well, but on occasions solid food obstructs and she finds this particularly likely to happen when she is eating in company or when she feels 'her nerves are bad'. A barium meal examination carried out in November 1964 is reported on as follows: 'There is a small hiatus hernia present, but I was unable to induce reflux by any manoeuvre at this examination. There was no delay to the passage of the barium from the oesophagus into the stomach and there is no stenosis. There is a constant deformity of the cardia, which I imagine is the result of her previous operation; this does not appear to affect the ability of this segment to expand and contract. No other abnormality has been demonstrated in the oesophagus, stomach, or duodenum.' Throughout the course of her illness, dating from 1950, the patient never had a Ryle's tube passed, had gas and oxygen anaesthesia for delivery of her baby, was jaundiced for two weeks before delivery, and lost a total of 40 lb. in weight. Her blood group is A. rhesus positive.

CASE 5 Mrs. G., aged 22, developed hyperemesis during the course of her second pregnancy in 1948. She complained of dysphagia shortly after the delivery of her baby, and this mild dysphagia has continued intermittently ever since. A barium meal examination carried out in 1951 showed a hiatus hernia but no reflux or stricture. In March of 1953 oesophagoscopy confirmed the absence of any stricture in the oesophagus. She has the sensation of food sticking and will not eat anything for several hours and is then 'all right again'. At one time she refused to eat because she felt that if she did the food would stick. She has been pregnant on three occasions since

1948, and with the fourth pregnancy her dysphagia was exacerbated. At that time she underwent a period of psychiatric treatment for a supposed nervous disorder of swallowing. The patient was sterilized in 1955 because of the severity of her symptoms with pregnancy. At the present time she still has occasional difficulty with solid food; she knows of no reason why this difficulty recurs and can never tell when she is going to have difficulty in swallowing her food. A barium swallow examination carried out in October 1964 shows the presence of a hiatus hernia, but there is no reflux or stricture of the oesophagus. During the course of her illness she has never had a Ryle's tube passed and has never been jaundiced. Her weight loss has not been significant. Her blood group is A. rhesus positive.

DISCUSSION

Transient dysphagia after pregnancy is not uncommon. A number of patients, who in late middle age develop oesophageal stricture in association with a hiatus hernia, give a history of difficulty in swallowing for the first few months after delivery, which spontaneously resolves. The difference between this and the progressive condition of the patients described may be simply one of degree. Several features of this condition are of interest. First is the fact that, although vomiting may be severe and continuous from a very early date in the pregnancy, dysphagia does not occur until after the baby is delivered and may then start from the day of delivery. This is in contrast to the patient who develops dysphagia after a gastric operation in which a Ryle tube has been used. Although vomiting may be trivial, the dysphagia may occur within the first week and the barium swallow demonstrate an obstruction. The means by which the mucosa of the lower oesophagus seems to be protected against the results of regurgitation during the pregnancy, yet so liable to change when delivery is complete, seems worth further investigation. A hormonal cause initiating the change seems possible. We have not been able to find any report in the literature of serious dysphagia occurring during the pregnancy, even where vomiting was copious and prolonged over six months. In each of Vinson's (1921) six patients, the pattern of symptomatic development was excessive vomiting before delivery of the baby and delivery followed by the onset of dysphagia. One reservation must concern exactly what the patient means by the term 'dysphagia' and the different history obtained by separate observers from the same patient. The possibility of a latent obstruction in the oesophagus being present during the pregnancy remains. Insufficient information is available on

the appearances at barium swallow in patients suffering from hyperemesis who are examined during pregnancy.

Another interesting feature is that these are the only patients we have observed with progressive dysphagia from a localized oesophageal obstruction developing between the ages of 20 and 35 years. In a number of other patients between these age limits who have complained of dysphagia due to a benign oesophageal stricture, it has been established that these patients' symptoms are a relapse of a condition established in infancy. None of our post-pregnancy patients has given such a history. In those who at first sight appear to develop obstruction *de novo* between 20 and 35 years of age, radiological evidence of a stricture in the first three years of life or a history of persistent vomiting requiring drip feeding in infancy has been obtained from their parents. Within the limits of our experience we regard pregnancy complicated by hyperemesis as the only circumstance in which dysphagia supposedly from benign oesophageal stricture starts for the first time between the ages stated. The reason for this is not clear. It may be a measure of a limited experience, but we have been impressed by the striking difference between the two groups within the same age limits and apparently suffering from a similar condition. In one there is dysphagia, no previous pregnancy, but a history of severe feeding difficulty in infancy; in the other group there is dysphagia, pregnancy complicated by hyperemesis, but no history whatsoever of infantile feeding difficulty. A problem complicating any all-embracing explanation is a patient known to have had severe dysphagia from a stricture of the oesophagus before the third year of life whose symptoms spontaneously diminished and who went through a pregnancy at the age of 21 without any relapse after the pregnancy or subsequently. It seems certain that for some reason benign oesophageal stricture is a disease predominantly of the extremes of life, or, more precisely, first causes symptoms before the fifth year of life or after the fiftieth year, except in the post-pregnancy group.

A firm opinion on the management of this condition from an experience of five patients is not justifiable. Prophylaxis would seem to be paramount, but how to separate those patients with hyperemesis who after delivery do not develop dysphagia from the small minority who do is difficult. Examination of all patients with toxæmia by routine barium meal examination during pregnancy is not practicable for a number

of reasons. It seems reasonable, however, to insist on this examination immediately post-partum dysphagia is complained of, and if hyperemesis has been severe and a hiatus hernia or reflux is demonstrated, the hernia should immediately be reduced and the crus of the diaphragm repaired. Although not curative in two of the patients described, an obvious criticism that the procedure was too long delayed could be made. One patient (case 2) had neither a hiatus hernia nor demonstrable oesophageal reflux. In spite of this it is suggested that reduction of the hernia and repair of the crus (Allison, 1951) should be carried out in any patient complaining of dysphagia as soon in the post-partum period as this symptom arises, if a barium meal examination confirms the presence of either a hiatus hernia or reflux into the oesophagus. Furthermore, all other methods of diminishing the effects of regurgitation of acid gastric juice on the lower oesophagus should forthwith be instituted. Prophylaxis by these means may well be urgent enough to be given emergency priority. The case reports demonstrate the problems of treating the established condition.

SUMMARY

Five patients with severe oesophageal obstruction following hyperemesis gravidarum are described. The condition is believed to originate in a similar way to stricture of the oesophagus associated with hiatus hernia.

The need for proper investigation of a history of dysphagia early in the puerperium is emphasized. Difficulties in the treatment of the established condition are described.

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