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Gastric Antral Membrane in Childhood: A Case Report

OA Awojobi,* AO Okubanjo** and NA Akingbehin*†

Summary

Awojobi OA, Okubanjo AO and Akingbehin NA. Gastric Antral Membrane in Childhood: A Case Report. *Nigerian Journal of Paediatrics* **1982; 9 : 115.** Gastric antral membrane is a rare cause of pyloric obstruction in infants and children. A case of gastric antral membrane associated with malrotation of the gut occurring in a Nigerian child is described. The literature on this disorder is also reviewed briefly.

Introduction

Congenital malformations of the stomach are rare.[1 2] Congenital hypertrophy of the pylorus is a more common anatomic cause of gastric outlet obstruction than pyloric atresia or membrane[1 3 4] and fibrosis resulting from peptic ulceration.[5] Atresia involving the gastrointestinal tract has been estimated to occur in 1 out of 10,000 births.[2] It affects the ileum and duodenum more commonly. A review of the English language literature reveals that about 60 cases of incomplete pyloric membrane have been reported in the paediatric age group.[1 3] Most of the patients reported are caucasians and, as far as we are aware, no case has been reported in an African child. In the present communication, we report a case of incomplete gastric antral membrane in a Nigerian child who also had intestinal malrotation.

University College Hospital, Ibadan

Department of Surgery
*Senior Registrar
*†Consultant Paediatric Surgeon

Department of Radiology
**Consultant Radiologist

Correspondence to: Dr OA Awojobi
Department of Surgery
University College Hospital
Ibadan, Nigeria.

Case Report

LM, a five-year old girl, presented in November, 1977, at the age of eleven months, with repeated attacks of vomiting since birth. Vomiting was usually projectile and sometimes contained bile. She was the sixth child in the family. Pregnancy and delivery were uneventful. Physical examination at that time, revealed a lethargic and dehydrated infant weighing 7.3kg. No other abnormality was found. The clinical diagnosis was congenital pyloric stenosis or duodenal stenosis. Barium meal and follow-through did not show any obstructive feature. She was managed conservatively and followed-up in the clinic until March, 1978 when she weighed 9.3 kg.

She presented again in July, 1981, with attacks of vomiting which had been occasional since March, 1978, but had occurred daily in the previous month before presentation. Vomiting was again projectile and bilious. When examined, she was small for age, weighing 14.6 kg and was moderately dehydrated. The abdomen was full

with visible gastric peristalsis; succussion splash was positive 5 hours after the last meal. The clinical diagnosis was gastric outlet obstruction due to duodenal stenosis. Barium meal revealed a translucent filling defect across the pyloric antrum (Fig. 1). During fluoroscopy, a temporary hold-up was observed before the antrum was filled. In view of this, a diagnosis of gastric antral membrane was made.

Through an epigastric midline incision, pyloroduodenotomy was performed and this revealed a thin antral membrane, 2cm from the pylorus with an eccentric orifice about 5mm in diameter. The stomach was moderately dilated and oedematous. The membrane was excised and a Heineke-Mikulicz pyloroplasty was done. The post-operative course was uneventful and the patient was discharged on the eighth post-operative day. Histologically, the membrane showed two gastric mucosae supported by muscularis mucosae and oedematous submucosa but no true gastric muscular layer was present (Fig. 2).

The patient was readmitted one week after discharge, with persistent bilious vomiting. Repeat barium meal and follow-through this time, revealed malrotation of the gut and gastric distension (Fig. 3). A review of the films of previous barium studies did not show malrotation. A second laparotomy confirmed malrotation of the small and large bowel and a band of Ladd constricting the jejunum. The band was divided and appendicectomy performed. Post-operatively, she developed right lower and middle lobe pneumonia that responded to antibiotic therapy. She was discharged on the twenty-first post-operative day. She has been followed-up for 8 months and has remained asymptomatic.

Fig. 1 Barium meal studies showing antral membrane (arrow). Note also the characteristic 'hair-line' appearance.

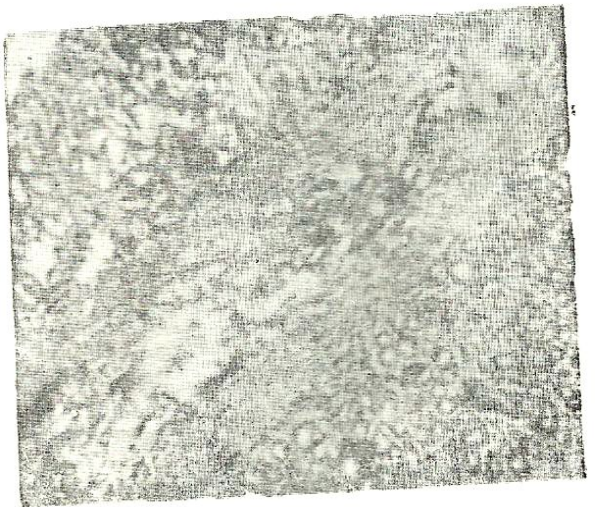

Fig. 2 Photomicrograph of antral membrane. Note the two mucosal surfaces supported by muscularis mucosae and a narrow submucosae. (Haematoxylin and eosin ×95).

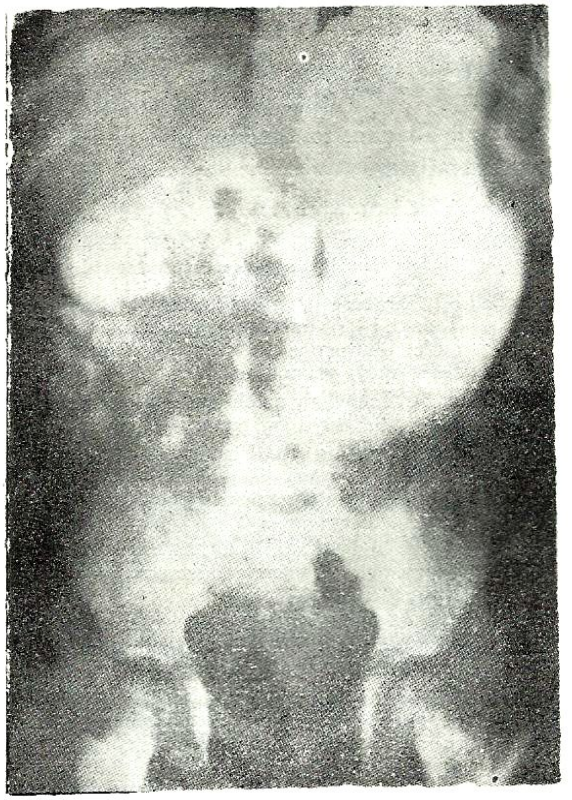

Fig. 3 Barium meal and follow-through studies demonstrating a dilated stomach and malrotation of the gut with the proximal jejunum in the right flank.

Discussion

The stomach enjoys an unusual freedom from congenital malformations.[2] Among the few malformations seen, gastric antral membrane is a rare entity.[1-3]

Gerber,[2] in a review of the literature, put forward a widely accepted classification of congenital gastric membranes based on the gross anatomy:

(*i*) Pyloric: (*a*) membrane, (*b*) atresia

(*ii*) Antral: (1cm or more proximal to pylorus): (*a*) membrane, (*b*) atresia

The membrane can be complete or incomplete. Complete and atretic malformation will present soon after birth with incessant vomiting with or without respiratory distress and epigastric distension.[2] It is the incomplete membrane that has a variable onset of symptomatology. It may present in adulthood.[2 6] It has a biphasic peak age incidence, namely: childhood and the sixth decade.[2] The symptoms are those of gastric outlet obstruction. Vomiting is occasionally projectile and bilious as in our patient. Older children may complain of upper abdominal pain, feeling of fullness after small meals and frequent foul eructations.[1 3] Thus, such diagnosis as gastroesophageal reflux or peptic ulcer is usually entertained in these children and treatment considered appropriate instituted, without relief of symptoms. However, incomplete gastric membranes may be asymptomatic.

Diagnosis depends on a high index of suspicion. Definitive dignosis is made on finding the characteristic lucent hairline lying perpendicularly, across the pyloric antrum on a barium meal radiograph. Anterior or posterior bowing of the membrane or concentrated barium sulphate may prevent radiographic identification of the membrane. During fluoroscopy, an initial hold-up of the contrast before antral filling or submaximal antral distension should increase the suspicion of the presence of a membrane. Delay in gastric emptying is a common feature.[1] Several barium meal studies may be needed before the diagnosis is made. In older children, gastroscopy is a useful diagnostic procedure. The membrane and the small orifice are visualized. It is usually not possible to advance the gastroscope through the orifice.[1 3] This procedure will also exclude inflammatory causes such as peptic ulceration which has been reported in children.[5]

The histology of antral membrane usually reveals a double layer of normal gastric mucosa separated by submucosa and muscularis mucosae. The muscle layer is absent and there is no inflammation or fibrosis to suggest an acquired lesion.

Other structural congenital malformations found in association with antral membrane include hypertrophic pyloric stenosis, duodenal

stenosis, malrotation of the gut, coarctation of the aorta, ventricular septal defect, patent ductus arteriosus and hypospadias.[3] This association has been the main evidence supporting the congenital nature of the membrane although the pathogenetic mechanism still remains conjectural.[1 2]

Our patient presented with symptomatic antral membrane as evidenced by the dilated stomach and symptomatic malrotation of the gut which was unmasked by the excision of the membrane. It explained the persistent bilious vomiting with which the child presented.

The treatment of antral membrane is operative. Segmental gastrectomy, gastroenterostomy and various forms of pyloroplasty combined with excision or incision of the membrane have been done.[1] However, pyloroplasty with excision of the membrane if it is thick, or incision if it is thin, is an effective procedure that carries minimal morbidity and mortality. It is envisaged that gastroscopic incision of the membrane would be feasible in future and so, obviate the morbidity and possible mortality of open surgery.[6]

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