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Case Report

Isolated congenital microgastria in 4 years old child: a case report

Pramod S.*, Ravikiran K.

Department of Paediatric Surgery, Kempegowda Institute of Medical Sciences and Research Institute, Bangalore, Karnataka, India

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***Correspondence:**
Dr. Pramod S.,
E-mail: pramodbmc76@gmail.com

ABSTRACT

Microgastria is a rare congenital anamoly. It was first reported in 1800s. It occurs in association with other anamolies. Common associated anomalies include asplenia, malrotation of intestine, upper limb anomalies, situs inversus. Isolated cases of Microgastria are very few in literature. Most cases of microgastria present during infancy. This is a report of a four year old child, eldest in litreature presenting with isolated microgastria. Child presented with vomiting, failure to thrive. Child underwent excision of stomach and oesophago-jejunostomy with pouch after initial stabilization. The child had an uneventful post-operative recovery.

Keywords: Congenital microgastria, Oesophago-jejunostomy, Stomach

INTRODUCTION

Microgastria is a rare congenital anomaly of caudal part of foregut.[1] It was reported in 1800s by Dide.[2] It is usually associated with asplenia, situs inversus, upper limb anomalies, malrotation of intestine. The exact etiology of microgastria is unknown. Defective mesodermal development during fourth and fifth week of intrauterine life is a probable etiology.[3] Various treatment options include conservative management with nasogastric feeds, gastric augmentation and gastric dissociation with Roux-en-Y esophagojejunostomy.[4] Less than 4 cases of isolated microgastria have been reported so far. The cases reported in literature are during infancy. The following case is 4 years old, being the eldest reported till date.

CASE REPORT

A four years old male child presented with history of non-bilious vomiting, feed intolerance since the neonatal period. Vomitus contained ingested food particles. Child was born to 25 years old primigravida by normal delivery at 31 weeks. His birth weight was 1.5kg. Child required neonatal intensive care admission in view of prematurity in the immediate post-natal period. Initially diagnosed as GER and received prokinetics following which the child was apparently normal till 8 months of life. Later symptoms recurred, with vomiting both to solid and liquid food. The frequency of vomiting had increased from last 6 months.

In the past 3 years child had three admissions for the evaluation of vomiting elsewhere. Presently child was on liquid diet which he was tolerating. But above a certain amount of liquids he used to vomit.

On examination child was malnourished and dehydrated. His weight and height were less than 3rd percentile of the normal. Endoscopy done in the past showed narrowing at 22cm from incisor. The narrowing was such that even a guide wire could not be negotiated beyond it.

A differential diagnosis of congenital oesophageal stenosis and achalasia was thought of. A contrast study showed dilated oesophagus with a small stomach and delayed emptying across the pylorus (Figure 1). The above investigations were in favor of microgastria. Child was further investigated to rule out associated VACTERL anomalies. No other anomalies were detected. It was a case of isolated microgastria.

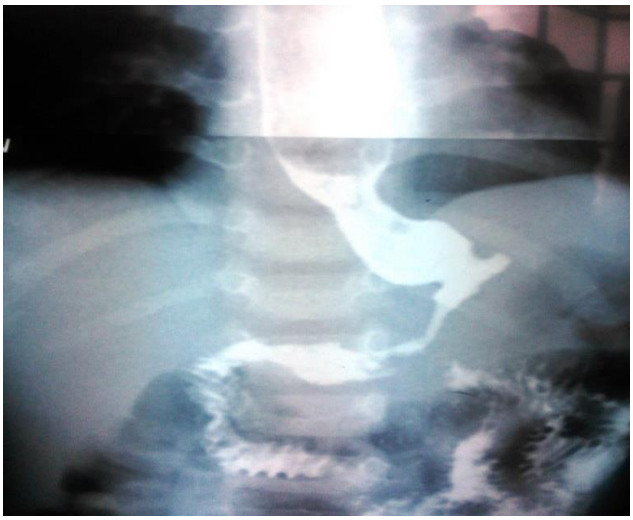

Figure 1: Upper GI contrast showing microgastria.

Child underwent laparotomy after stabilization. On laparotomy with upper midline incision the following findings were noted. The oesophagus was dilated, stomach was small tubular with capacity of 10ml (Figure 2). The child underwent excision of stomach (Figure 4) with Roux en Y Oesophago- Jejunostomy with Pouch (Hunt Lawrence jejunal pouch; Figure 3). There was no other associated malrotation or splenic abnormality.

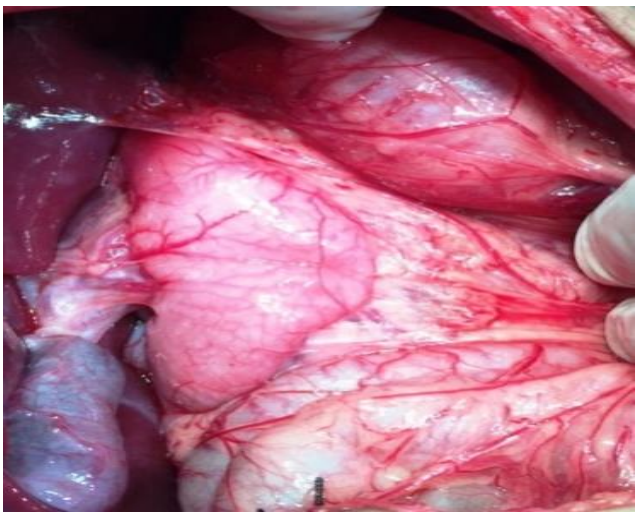

Figure 2: Intraoperative picture of microgastria.

Post-operative recovery was uneventful. Dye study was done on post-operative day 7 which showed no evidence of leak. Child was initially started on liquids, gradually upgraded to semisolid diet. At the time of discharge child was tolerating semisolid diet with no dysphagia. Child was put on vitamin B12 and multivitamin supplementation and advised regular follow up. At one year follow up child is doing well with improvement in the weight.

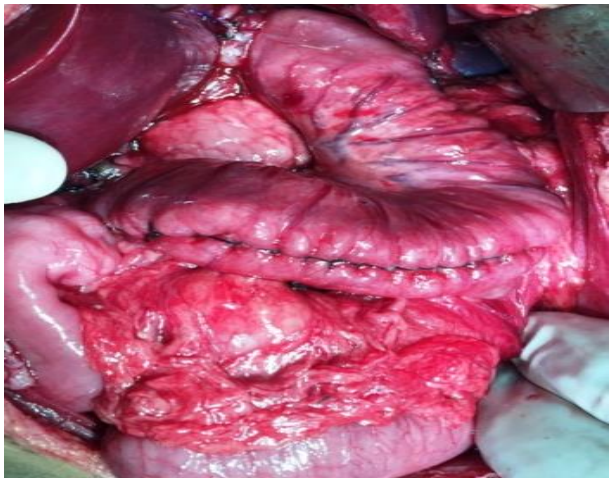

Figure 3: Roux en Y oesophago-jejunostomy.

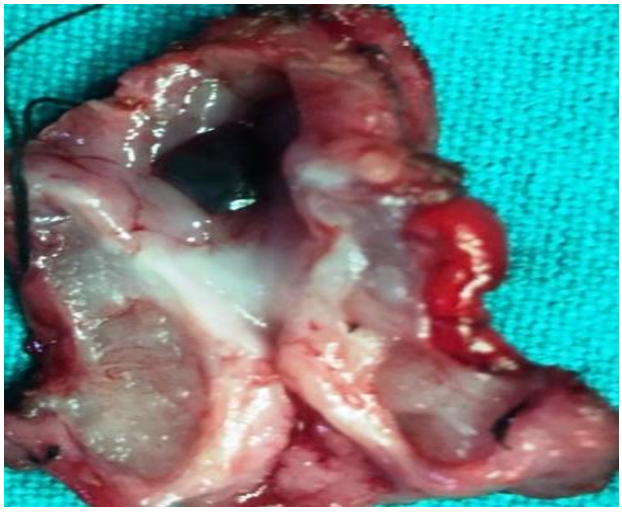

Figure 4: Excised stomach.

DISCUSSION

Congenital microgastria is an extremely rare anomaly. It was first described by Dide in 18942. The exact aetiology of congenital microgastria is unknown. It may be secondary to defective mesodermal development at fourth to fifth week of intrauterine life during rotation of stomach.[3] Migration of the pancreatic buds and the differentiation of dorsal mesogastrium into splenic anlage occur at the same time. As the development of spleen and stomach occur from dorsal mesogastrium associated splenic anomalies may be seen. Other associated anomalies include asplenia, congenital heart disease, lung sequestration, laryngotracheal cleft, oesophageal atresia,

midgut malrotation, renal, limb, and central nervous system malformations.

Prenatally microgastria mimics oesophageal atresia with non-visualization of stomach and polyhydramnios.[5] But most of the cases present during infancy with vomiting, failure to thrive.

The diagnostic investigation of choice would be an upper GI contrast study which reveals a dilated oesophagus with a small tubular stomach.[6,7] Though the present case presented during infancy the child survived with such a small capacity stomach till four years of life. This is so far the eldest reported case with isolated microgastria.

The treatment modalities were adopted from the surgeries in relation to carcinoma stomach.

Various treatment options for microgastria include conservative management with frequent feeding of small amounts, gastric augmentation with a jejunal loop and total gastric dissociation with a Roux en-Y esophagojejunostomy, with or without pouch.[4,8]

Conservative management with frequent feeding of small quantity is said to enlarge the stomach. This conservative approach can be tried in less severe form of microgastria. But Jones and Cohen found that conservative treatment resulted in poor somatic growth, sexual underdevelopment, delay in cognitive milestones and recommended early reconstructive surgery.[9]

Excision of stomach will result in rapid emptying of food, alkaline reflux into oesophagus, decreased absorption of vitamin B12, bacterial colonization. This is because of loss of pyloric sphincter, loss of lower oesophageal sphincter, loss of parietal and chief cells and loss of hydrochloric acid respectively. In order to decrease the incidence of dumping, Hunt and Lawrence described creation of a food pouch from a segment of jejunum.[10] Food pouch acts a reservoir, slow down the transit and prevents alkaline reflux.

All children with gastrectomy require vitamin B12 supplementation on monthly basis to prevent megaloblastic anaemia. Long term follow up of these children include assessment by endoscopy with biopsies of oesophagus and jejunum, barium meal follow through to assess jejunal pouch size, peristalsis, presence of stasis, pouch dilatation and reflux.

CONCLUSION

Microgastria is rare anomaly. The treatment of microgastria should be indivualised. In patients with severe gastro oesophageal reflux and failure to thrive gastric exclusion with esophago- jejunal anastomosis has got good results.

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