Hyperplastic Gastric Polyps in an Infant, Mimicking Infantile Hypertrophic Pyloric Stenosis - A Case Report and Review of the Literature

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INTRODUCTION

The primary cause of Gastric Outlet Obstruction (GOO) in infancy is Infantile Hypertrophic Pyloric Stenosis (IHPS). Less common causes include gastric hamartoma, teratoma, hyperplastic polyp, pre-pyloric web and primary acquired GOO(Jodhpur disease)[1,2]. Treatment for IHPS with pyloromyotomy, webs and primary acquired GOO with pyloroplasty, and wide excision of gastric tumours are well established. Polyps either single or multiple may be excised endoscopically or surgically. We would like to present the rare case of a 6 month old infant with projectile vomiting of a non bilious character, with visible gastric peristalsis and a palpable mass per abdomen which happened to be secondary to gigantic hyperplastic polyps of the stomach.

CASE REPORT

A 6 month old infant presented with forceful projectile vomiting immediately after feeds. The symptoms had progressed over the last two months and had gone from posseting to forceful vomiting. The vomitus was non - bilious, and contained curdled milk and undigested food with streaks of blood and mucus. The child had also lost considerable weight in the last few weeks.

On physical examination he was found to be pink, moderately dehydrated and inspection of his abdomen revealed visible gastric peristalsis. On palpation a mass was appreciable in the right hypochondrium which was not the classical olive like tumour of IHPS despite the other clinical signs and symptoms.

His blood gases did not show evidence of the classical hypokalemic hypochloremic metabolic alkalosis of Infantile Hypertrophic Pyloric Stenosis. His hemoglobin was within the normal range.

An ultrasonogram of the abdomen did not reveal thickening of the pylorus with its elongated channel to suggest IHPS. There was gross intra luminal debris in the antrum and body of the stomach, which was thickened, with failure of dextrose water to pass through the pyloric channel on real time ultrasonography. In view of the unusual ultrasonographic findings, a water soluble upper GI contrast study was performed. It revealed a dilated stomach with multiple filling defects in the body and antrum (fig 1), and the contrast medium did not flow past the pylorus.

A Laparotomy revealed a dilated stomach with a large intra-luminal masses arising from almost the entire anterior wall of the infant's stomach (fig2). The mass was extensive, with multiple islands of sessile masses with polypoid, frond like outgrowths with minimal intervening normal gastric mucosa, and it was seen extending all the way into the antrum, some of the protruding polyps functioning like a stop valve at the pyloric canal, causing the GOO. There were no other discernible abnormalities.

The entire mass of polyps along with the anterior wall of the stomach was excised. The antrum and pylorus were preserved. The remaining cylinder of the stomach that was less than half its original size was primarily reconstructed. A gastrostomy tube was left in situ.

Histopathological examination of the specimen revealed that masses consisted of multiple Hyperplastic polyps with no discernible malignancy.

The child made an uneventful recovery post-operatively, and was fed on the 5th post-operative day. The gastrostomy tube was removed 1 week later and full feeds were established over a period of two weeks. There were no clinical symptoms or signs of increased intra-gastric pressure leading to gastro-esophageal reflux or signs of post-prandial hypoglycemia or dumping syndrome.

The child was subsequently discharged from the hospital and continued monthly follow up for a whole year during which time he progressed well developmentally and nutritionally. At the end of the year he was eating normally, growing well and had normal physical and developmental milestones comparable to children of his age.

A gastroscopy done a year after the surgery showed a capacious stomach, open pylorus and no polyps. He will require annual gastroscopy surveillance.

DISCUSSION

Our patient presented at 6 months of age, with symptoms suggestive of IHPS, which had started 2 months before albeit mildly.

He had non-bilious, projectile vomiting which was suggestive

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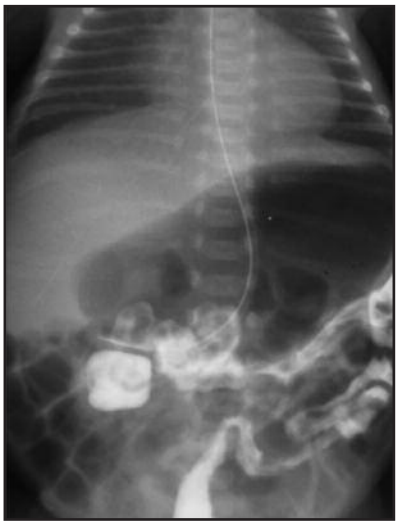

Fig. 1 : Upper GI study showing a persistent filling defect, with no passage of contrast past the pylorus.

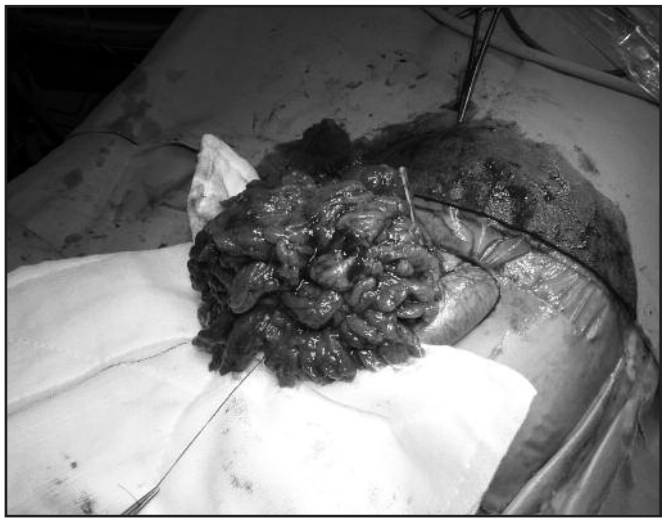

Fig. 2 : Polyps prolapsing out of the gastrotomy.

of a GOO. Haemetemesis is also often noted in IHPS due to gastritis.

The usual biochemical scenario in IHPS is hypochloremic, hypokalaemic metabloic alkalosis.

Presence of a palpable pylorus ('pyloric tumor') is considered diagnostic of IHPS. This feature alone together with the signs and symptoms of IHPS and the classical biochemical milieu is sufficient indication for surgery. In our department a routine ultrasonogram is not performed and is reserved for atypical/equivocal cases. Upper GI studies are also not routine in infants suspected to have IHPS. Given the inconclusive ultrasound findings, the contrast study was done as a diagnostic adjunct. The classic 'shouldering' sign caused by the thickened pylorus was absent and there were multiple filling defects seen in the grossly dilated stomach.

Gastric polyps are rare in children and are recognized either incidentally or on endoscopy. The commonest variety are hyperplastic polyps which may be solitary or multiple, the other forms being adenomatous and hamartomatous. Whilst the former are neoplastic in nature the latter's malignant potential is unclear.

The neoplastic potential of hyperplastic polyps has been investigated in adults and some of the polyps have been found to harbour areas of carcinoma or have an intrinsic neoplastic potential[3,4].

The adaptation of the stomach to loss of volume and digestive surface area took a period of 3 weeks, by which time he was able to consume full feeds of formula milk via a bottle. The appearance of the stomach on Gastroscopy a year later was normal with no evidence of volume contraction despite our extensive resection. The mucosa was normal, and there were no evidence of polyps/gastritis or raised areas of gastric mucosa.

CONCLUSION

IHPS is still the commonest cause of GOO in infants. However a high index of suspicion is required when dealing with an older child with symptoms suggestive of IHPS. Gastric polyps causing GOO are rare and such large and extensive polyps are even rarer. Ultrasonography and contrast studies used appropriately shed more light on the possible diagnosis and aid surgical planning. To the best of our knowledge this is perhaps the first case report of such extensive polyposis of the stomach causing GOO with signs and symptoms mimicking IHPS. Given the possible malignant potential, patients diagnosed with Hyperplastic polyps should remain on long term follow up with surveillance endoscopy.

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