# Is there any surgical cause of non-bilious vomiting?

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Madam, Pyloric atresia is a rare disorder - less than 1% incidence is reported in the literature.[1] It usually occurs as an isolated anomaly but it may arise in association with other congenital abnormalities mostly with epidermolysis bullosa/congenita aplasia cutis.[2] Other, less commonly described associated anomalies include atresia in oesophagus, duodenum, jejunum, rectum, colon and gallbladder agenesis.[3] This condition was first described by Calder in 1749. Moore searched the literature in 1989 and reported 125 cases of congenital gastric outlet obstruction worldwide. Aetiology of pyloric atresia is unknown. There are several embryological theories available; some advocate a developmental arrest between the 5th and 12th week of intrauterine life. Mechanical and chemical injury of the pylorus leads to denudation of the mucosa and results in the development of synechiae and subsequent fibrous cicatrisation.[4] According to Tandler in 1900, this anomaly

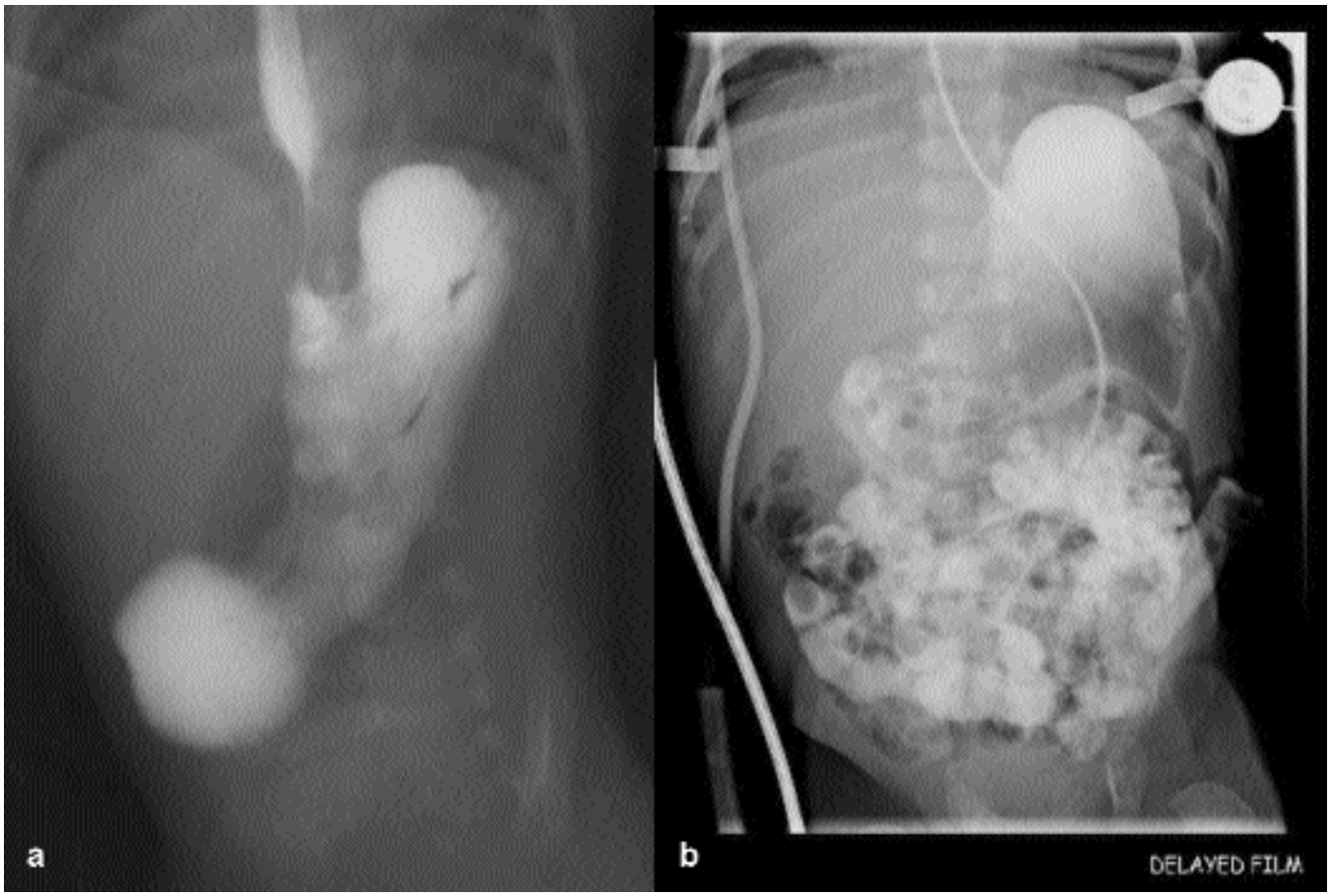


**Figure-1:** (a) Preoperative contrast study showing contrast in stomach and a single air bubble in the pylorus suggesting pyloric atresia. (b) Postoperative contrast study on the 7th postoperative day showing free passage of contrast in the distal bowel loops.

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is a consequence of the failure of the tube to canalise during development, in contrast to a lot of other intestinal anomalies that are the result of vascular disabilities.[4] There is a lack of awareness among the health workers

and the diagnosis is usually delayed due to non-bilious vomiting. This may lead to pulmonary aspiration, severe metabolic derangement, and gastric perforation which can be fatal.[5] We discuss herein a rare cause of congenital malformation with persistent neonatal non-bilious vomiting for several weeks before surgical intervention was done.

A full term baby boy, presented at 30th day of life with persistent non-bilious vomiting. He was delivered by Caesarean section due to polyhydramnios and non-progression of labour with good Apgar score and normal passage of meconium. He had persistent vomiting since then and required multiple hospital admissions for correction of dehydration. On arrival, he was ill-looking with severe dehydration and emaciated with visible peristalsis at the epigastrium. There were no other associated congenital anomalies present. After initial resuscitation, an abdominal x-ray was planned, which revealed a single air bubble in the upper abdomen with no gas in the distal pylorus that raised the suspicion of pyloric atresia. An upper gastrointestinal contrast study confirmed that there was no passage of contrast beyond the pylorus (Figure). After correction of electrolytes and appropriate resuscitation, an exploratory laparotomy was performed. There was type 1 pyloric atresia with no distal obstruction. We performed Heineke-Mikulicz pyloroplasty with transgastric tube jejunostomy and inserted a Hickman line for postoperative intravenous nutrition. Tube feeding was started on 6th post-operative day and a contrast study was performed on the 7th post-operative day which showed free passage of contrast beyond the pylorus (Figure). He had initial feeding difficulties with intermittent vomiting which responded to prokinetic agents. He was discharged on full feed and is currently on regular follow-up in our clinic.

This case enlightens the fact that all children with persistent non-bilious vomiting should be evaluated for pyloric atresia. There should be high index of suspicion for a timely diagnosis. Initial management should be proper optimisation of electrolytes followed by definitive correction.

## Reference

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