

Duodenal Obstruction

- Duodenal obstruction results from intrinsic or extrinsic causes:

-Intrinsic:

1. Atresia
2. Stenosis
3. Duodenal web (“wind sock” defect) > membranous ring within the duodenum --> proximal peristalsis causes formation of “wind sock,” with radiologic appearance of the obstruction distal to the actual annulus of the wind sock
4. Duplication

-Extrinsic:

4. Peritoneal (Ladd’s) bands
5. Annular pancreas
6. Anterior portal vein

Duodenal Obstruction

- Pathogenesis:

Intrinsic>

1. Weeks 4-6 gestation, duodenal lumen is obliterated by rapidly growing epithelium, which then recanalizes at the 12th week; failure of recanalization may lead to **atresia, stenosis, or webs.**
2. **Duodenal duplication** is the result of dual formation of the organ, which may or may not communicate with the normal (“real”) duodenal lumen

Duodenal Obstruction

•Pathogenesis:

Extrinsic>

1. Congenital peritoneal bands form with abnormal rotation-->during 10th week gestation, as the embryonic foregut and hindgut return to the abdomen, rotation occurs to cause the gut to lie to the left and posterior to the SMA-->the distal ileum, cecum, and colon move anteriorly and to the right; failure of these rotation results in abnormal cecal position and peritoneal bands which extend from the cecum to the RUQ, across the duodenum. (Also, at risk for midgut volvulus).
2. Annular pancreas caused by dorsal and ventral migration of pancreas to create a concentric constriction of the duodenum

Duodenal Obstruction

- Clinical presentation:

- Depends on degree of obstruction.
- Majority are distal to ampulla of Vater-->bilious emesis without abdominal distension is the cardinal sign for duodenal obstruction.
- High grade obstructions present within the first few days of life.
- Some may go undiagnosed for months or years.
- Weight loss, dehydration, and hypochloremic metabolic alkalosis

Duodenal Obstruction

- Diagnosis:

- Double bubble sign on abdominal X-ray (diagnostic in 58%)
- Upper GI contrast study
- If delay in operative therapy contemplated, a contrast enema will demonstrate position of cecum-->if normal position, then malrotation is highly unlikely and delay is probably safe.

Duodenal Obstruction

Presenting Symptoms

(N = 138, 1956-1988, 47% male, birth to 30 days)

Emesis	90%
Bilious emesis	66%
Abdominal distension	25%
Dehydration	24%
Weight loss	17%
Prematurity	45%

**Reference: Congenital Duodenal Obstruction: A 32-Year Review
Bailey et al J. Pediatric Surgery 1993**

Duodenal Obstruction

Associated Anomalies in 51 Patients (38%)

Down syndrome	15 (11%)
Cardiac (with Down's)	6 (4%)
Imperforate anus	6 (4%)
Cornelia de Lange syndrome	3 (2%)
Vertebral anomalies	2 (2%)
Esophageal atresia	2 (2%)
Ileal atresia	2 (2%)
2+ VACTERL	5 (4%)
>2 Total anomalies	15 (11%)

**Reference: Congenital Duodenal Obstruction: A 32-Year Review
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Duodenal Obstruction

•Results:

-128/138 (93% survived); 8/10 had other serious, life-threatening anomalies

-33% (N=46) intrinsic lesions; 46% (N=64) extrinsic; 21% mixed

-Of the 46 intrinsic lesions> 24 atresias, 14 webs, 5 stenoses, 1 duplication, (2 not noted)

-Of the 64 extrinsic> 53 peritoneal bands, 11 annular pancreas

-Of the mixed lesions> duodenal webs associated with stenosis in 2 patients, and with annular pancreas in one.

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Duodenal Obstruction

- Results:

- Reoperation required in 20 (14%) patients; 5d-4 yrs post-op>

- Anastomotic leak (N = 6)

- Obstruction (N=5)

- Wound dehiscence (N=3)

**Reference: Congenital Duodenal Obstruction: A 32-Year Review
Bailey et al J. Pediatric Surgery 1993**