## *Original Article*

# Analysis of Postoperative Reoperation for Congenital Duodenal Obstruction

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**OBJECTIVE:** To analyse the risk factors for reoperation after initial surgical repair of congenital duodenal obstruction and demonstrate that they can be decreased with more careful attention and more advanced techniques during surgery.

**METHODS:** The records of newborns and infants (aged 0–2 months) who had surgical therapy for congenital duodenal obstruction in the past 30 years were reviewed and analysed. Of the 298 patients, 132 (44%) were boys and 166 (56%) were girls. All patients who underwent repeat surgery postoperatively were evaluated by the reasons for surgery. The number of patients with various combination lesions of congenital duodenal obstruction was also calculated and the relationship to postoperative reoperation was analysed.

**RESULTS:** Twenty patients (6.7%) had congenital duodenal obstruction with combination lesions including duodenal web, malrotation, annular pancreas and multiple duodenal web. Twelve patients required further operation 5 days to 2 years postoperatively for complications ($n = 5$) and other duodenal atresias that were not discovered initially ($n = 7$).

**CONCLUSION:** More than half of reoperated patients (7/12) had multiple lesions of duodenal obstruction that were missed during the primary operation. The postoperative reoperation rate for congenital duodenal obstruction could be decreased with more careful attention to operative details and more preoperative and intraoperative evaluation of the gastrointestinal tract. [*Asian J Surg* 2005;28(1):38–40]

**Key Words:** congenital duodenal obstruction, multiple duodenal web, postoperative reoperation

## Introduction

Congenital duodenal obstruction is one of the most common anomalies in newborns and infants. Several embryological defects in foregut development, canalization or rotation lead to congenital duodenal obstruction such as duodenal atresia, duodenal web and malrotation. In addition, anomalies of the pancreas can cause duodenal obstruction. Although advances in management in neonatal intensive care, respiratory support and nutritional therapy have dramatically increased the survival rate, the relatively high postoperative reoperation rate remains a challenge in the treatment of congenital duodenal obstruction.[1] We report 12 patients who required reoperation after initial repair among 298 patients with congenital duodenal obstruction. The dangers and preventive strategies were compared with those in other studies.

## Patients and methods

Two hundred and ninety-eight newborns and infants (aged 0–2 months) with congenital duodenal obstruction were treated at Beijing Children's Hospital between 1972 and 2002.

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Of these, 132 were boys and 166 were girls. The mean birth weight was 2.43 kg (range, 1.32–3.56 kg) and the mean gestational age was 38.6 weeks (range, 34–42 weeks). Patients were divided into two groups. Group 1 consisted of 121 cases treated in the first 15 years (1972–1986) and Group 2 included 177 cases treated in the last 15 years (1987–2002).

The diagnosis was mainly made from presenting signs and symptoms including bilious vomiting at birth or during first feeding and upright plain film abdominal roentgenogram. Upper and/or lower gastrointestinal contrast studies were performed to confirm the diagnosis in 42% of patients.

Initial treatment of patients with congenital duodenal obstruction was gastric decompression by nasogastric tube, intravenous fluids to correct dehydration and maintain electrolyte balance and appropriate respiratory support. First surgical repair was performed as quickly as preoperative preparation allowed. The operations performed to repair congenital duodenal obstruction included Ladd's procedure for malrotation, duodenoduodenostomy or duodenoplasty for annular pancreas or duodenal atresia, and web excision for duodenal web. Combination procedures were performed for those who had multiple gastrointestinal anomalies. All surgery was achieved as quickly as possible to minimize the risk to patients, especially newborns and premature babies.

Postoperative care included intravenous nutritional support until the gastric volume decreased appropriately and the patient began to have stools. If the bilious gastric fluid did not obviously decrease, there were no stools or the patient vomited severely on eating by postoperative day 5–7, plain film abdominal roentgenograms in combination with barium meals were used to confirm the existence of gastrointestinal obstruction and evaluate the possibility of further surgical therapy.

## Results

Of the 27 patients (9%) who died of sepsis, pneumonia, short-bowel syndrome or severe cardiac anomalies, 17 were in Group 1 and 10 in Group 2. Twenty patients (6.7%) had various combination abnormalities: 12 patients had lesions consisting of duodenal web plus malrotation, three had duodenal web plus annular pancreas, three had malrotation plus annular pancreas, and two had multiple duodenal web.

Reoperation was required in 12 patients (6 in each group) 5 days to 2 years after initial repair. Of these, 58% had combination lesions: malrotation plus duodenal web ($n = 5$) and multiple duodenal web ($n = 2$). The remaining five patients underwent repeat surgery within a few days after the initial

operation due to anastomotic leak ($n = 1$), adhesive obstruction ($n = 3$) and necrotizing enterocolitis (NEC) plus wound dehiscence ($n = 1$). Among the reoperations in Group 1, 67% (4/6) were due to common postoperative complications, while in Group 2, 83% (5/6) of reoperations were due to combination lesions.

Half of the reoperated patients (6/12) died from severe systemic complications such as sepsis or malnutrition. Four cases (33%) were in Group 1 and two (17%) in Group 2. Three were cases of missed duodenal webs including one premature baby. There was only one baby with severe cardiac anomalies who died from NEC plus wound dehiscence after reoperation.

## Discussion

The embryological anomalies that cause duodenal obstruction can be classified as either intrinsic or extrinsic lesions.[2] Intrinsic lesions are formed by failure of recanalization or arrest in duodenal growth at the 12th week *in utero*, leading to the development of atresia, stenosis or web.[3,4] Extrinsic compression of the duodenum can be caused by an annular pancreas or congenital duodenal band.

The incidence of combinations of anatomical abnormalities in our study is relatively low (6.7%) compared with other studies.[1,2,5] Most combination lesions in our patients were duodenal web with malrotation caused by duodenal bands (60%).

Although the number of patients who underwent further surgical therapy in our report was quite low (4%, 12/298) compared with other reports,[1] more than half of reoperations were needed for lesions missed during initial surgery. The significant differences between the major causes of reoperations in Groups 1 and 2 demonstrated improvements in surgical techniques and further recognition of congenital duodenal obstruction. All reoperated patients with combinations of anatomical abnormalities in the duodenum had duodenal web plus malrotation or multiple webs. Both duodenal web and malrotation-induced bands lead to incomplete duodenal obstruction. They are not usually found by gastrointestinal contrast preoperatively.[6,7] This kind of combination obstruction is liable to be released with distal bowel bulge by Ladd's procedure without discovering the web. Also, obvious release of obstruction by proximal web resection may lead to failure to find other webs. Most of these patients underwent repeat surgery for permanent bilious vomiting on initial feeding within a week. The symptoms of some of these patients were improved for a few months or years until the progression of

malnutrition prompted physicians to re-evaluate the gastrointestinal tract. We report one patient who had undergone Ladd's procedure and had a duodenal web resected 2 years later and another one who underwent a second duodenal web resection 3 months later.

As improved surgical techniques, materials and operative time have decreased surgical stress on patients, repeat surgery for surgical complications was rare in our group, especially in the last 15 years, compared with other reports.[5]

The 50% death rate in reoperated patients compared with the 9% total death rate demonstrated that a second operation increases stress on the patient. Obviously, this additional stress could be decreased with more careful intraoperative exploration. We think that routine examination of the distal gastrointestinal tract is important to find more obstructions, and this should be performed by injecting normal saline instead of air through the gastrointestinal tube. We injected normal saline into the stomach through a nasogastric tube and compressed normal saline into the duodenum and jejunum to exclude intrinsic duodenal lesions. This is a simple, minimally invasive and effective technique compared with other catheterizations. Using this method, we successfully detected duodenal webs that had not been revealed by preoperative gastrointestinal tract contrast during operation in three patients in the last 2 years.

In conclusion, although multiple combinations of the various anatomical abnormalities producing congenital duodenal obstruction are not common, they remain a challenge to survival rates for total congenital duodenal obstruction because of their relatively high reoperation rate. However, with more careful attention and improved surgical technique during the primary operation, the survival rate for congenital duodenal obstruction can be increased.

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