DUODENAL STENOSIS IN INFANTS – CASE PRESENTATION

Cristina Singer[1], Simona Coşoveanu[1], Ovidiu Ciobanu[2], Alin Stoica[2], Ileana Puiu[3], Marinău Laura[3], Neagoe Daniela[4], Gabriela Oancea[5]

Abstract

We present the case of a 7 ½ month-old male infant who was admitted to the 2nd Pediatric Clinic of the Emergency County Hospital Craiova for vomiting. The infant was repeatedly admitted to hospital, in Romania and in Italy, ever since he was 2 weeks old, for vomiting. The diagnosis problems are presented in order to establish the causes of the vomiting. In our clinic, we performed the barium transit examination which pointed out the duodenal stenosis. The infant was transferred to the Pediatric Surgery Clinic where he was subject to a surgical intervention and we discovered the presence of an intraluminal, incomplete diaphragm, at the level of duodenal section D1. The post-operatory evolution was fair.

Key words: infant, recurrent vomiting, duodenal stenosis

Introduction

Duodenal atresia or stenosis usually occurs in the first or second part of the duodenum, most often near the papilla of Vater. Congenital duodenal obstruction may be due to intrinsic or extrinsic lesions. Duodenal obstructions usually occur in the second part of the duodenum.

The duodenal obstructions can be caused by some intrinsic and/or extrinsic lesions. The intrinsic lesions, in the form of diaphragm, atresia, or stenosis usually occur in the second part of the duodenum, between the fifth and the tenth week of gestation, because of the vacuolization failure and the recanalling of the duodenum from its solid stage [1].

Case presentation

A 7 ½ month-old male infant, admitted to the 2nd Pediatric Clinic of the Emergency County Hospital Craiova, in July 2015, for vomiting.

Heredo-collateral antecedents: young, healthy parents, no chronic diseases within family.

Physiologic personal antecedents: first born child, after a normal evolution pregnancy, on term, naturally born in a hospital in Italy, birth weight= 3000 g, height=50 cm, no sufferance, artificially fed with milk adapted formula when born, anti-reflux milk powder when he was 1 month old, diversified at 5 ½ months old, vaccinations performed in Italy. He was administered vitamin D3, prophylactically. With a corresponding psycho-motor development, he was brought in Romania by his parents when he was 5 ½ months old. He lives in a rural area, in a house with good conditions.

Pathologic personal antecedents:

- 3 admissions in Italy:
  - When he was 2 weeks old for vomiting occurred in the first days of life; he was administered an eso-gastroduodenal transit and he was diagnosed with gastro-esophageal reflux – he received anti-reflux milk powder and treatment with Gastrotuss oral suspension;
  - When he was 4 months old for vomiting and acute dehydration. There, he had a urine culture test, Doppler echocardiography, thorax and abdominal radiologic examination, biochemical tests which were normal;
  - When he was 5 months old, he was again admitted for vomiting, with an acute dehydration syndrome and metabolic alkalosis. Taking into account the recurrent vomiting episodes, the family was advised to go with their infant to a hospital specialized in metabolic diseases, but the family could not do it because of financial reasons;
- one admission in Romania, to a county hospital, when he was 6 ½ months old, for vomiting.

At admission to our clinic, the infant had an altered general state, without fever, weight=5000g, height= 69cm, PI= 0.66 (SD= -5); he was pale, with dark circles around his eyes, dry lips, abdominal skin fold with low elasticity, normal stetacustic pulmonary, rhythmic heart beats, HB=112/min, supple abdomen, normal stool, FA= 1/1 cm, slightly depressed, without meningeal symptoms.

[1]University of Medicine and Pharmacy Craiova, Emergency County Hospital Craiova, 2nd Pediatric Clinic
[2]University of Medicine and Pharmacy Craiova, Emergency County Hospital Craiova, Pediatric Surgery Clinic
[3]University of Medicine and Pharmacy Craiova, Emergency County Hospital Craiova, 1st Pediatric Clinic
[4]University of Medicine and Pharmacy Craiova, Emergency County Hospital Craiova, Medical Clinic
[5]Emergency County Hospital Craiova, 2nd Pediatric Clinic
E-mail: singercristina@gmail.com, scosoveanu@yahoo.com, ociobanu@gmail.com, alin.stoica@gmail.com, vipuiu@yahoo.com, mlaurad@yahoo.com, dananeagoe2014@gmail.com, gabrielaoancea@yahoo.com

Investigations

Hemogram: Hb= 12.7g%, L= 15400/mm^3, NS= 64.8%, Ly=30%, Mo=5.2%, Tr.=285000/mm^3, VSH= 5/10 mm, urea= 41→20 mg%, creatinine= 0.45→0,46 mg%, GOT= 47→21 U/l, GPT= 38→21 U/l, γGT= 11 U/l, alkaline deposit= 17→23 mEq/l, calcemia=9 mg%, sideremy= 89 mg%, sanguine ionogram: Na=128 mEq/l, K= 3 mEq/l, total proteins= 6.1g%, negative uroculture, F.O. eye – normal aspect, chest X-ray – no pulmonary modifications.

We proceeded to an urgent PEV with glucose and electrolytes, Quamatel i.v., then we restarted feeding him, progressively, with anti-reflux milk powder.

He went on receiving food p.o., having a fair general state; on the third day from admission he vomited again, abundantly, followed by the rapid presence of the dehydration syndrome. The abdomen got excavated, with intestinal loops, which were visible to the wall.

Because the infant presented recurrent vomiting from his first days of life, which had required repeated admissions, being diagnosed with gastro-esophageal reflux when he was 2 weeks old, fact which determined his feeding with anti-reflux milk powder and Gastrotuss p.o., and he had a non-corresponding ponderal growth, PI=0.66 (SD -5), and because of the type of vomiting he presented in our clinic, we took into consideration the possibility of a pylorus stenosis or some other digestive tract malformation.

Although the infant was performed an eso-gastroduodenal transit in Italy, at his first admission, we repeated it with barium (fig. 1), which revealed: no gastro-esophageal reflux, slightly relaxed stomach, with its inferior pole lower than normal, having a permeable pylorus with rhythmic ejection; megaduoden with significant stasis on the first segments, the inferior pole passing the line of the iliac crests; slowly transit of the barium to the small intestine, one hour after its ingestion.

The infant was transferred, being diagnosed with duodenal stenosis, to the Pediatric Surgery Clinic, where he had a surgical intervention and we noticed, at the level of the duodenal section D1, the presence of an intraluminal, incomplete diaphragm (fig.2). The post-operatory evolution was fair. Discharge diagnosis: Duodenal stenosis due to an incomplete obstacle. Dystrophy degree II.

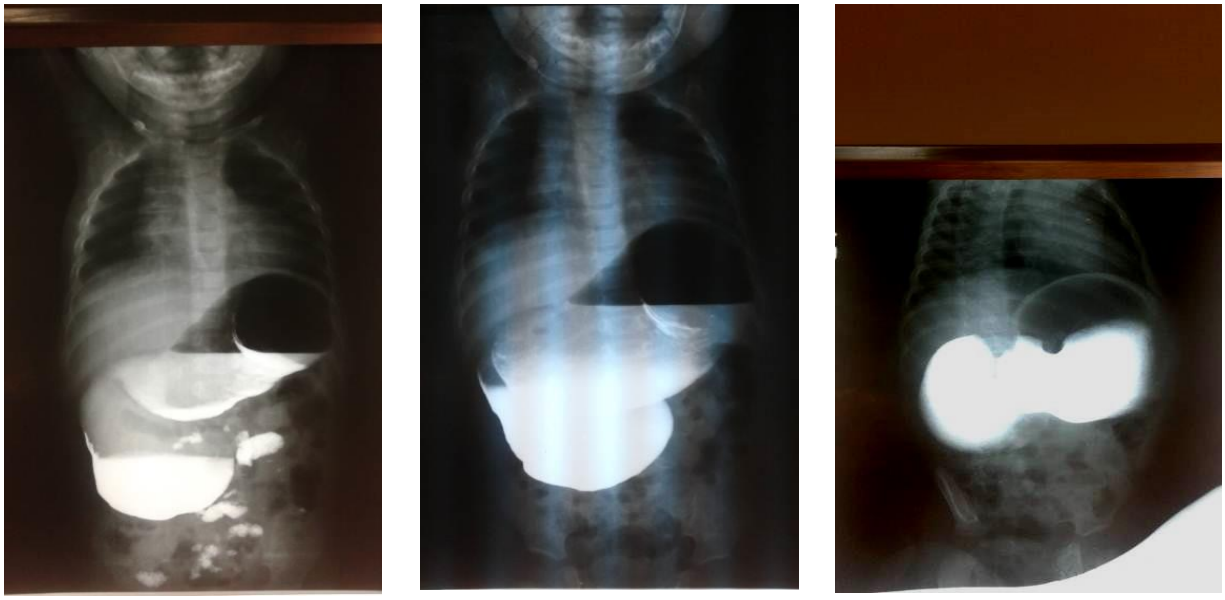

Fig. 1 Eso-gastroduodenal transit.

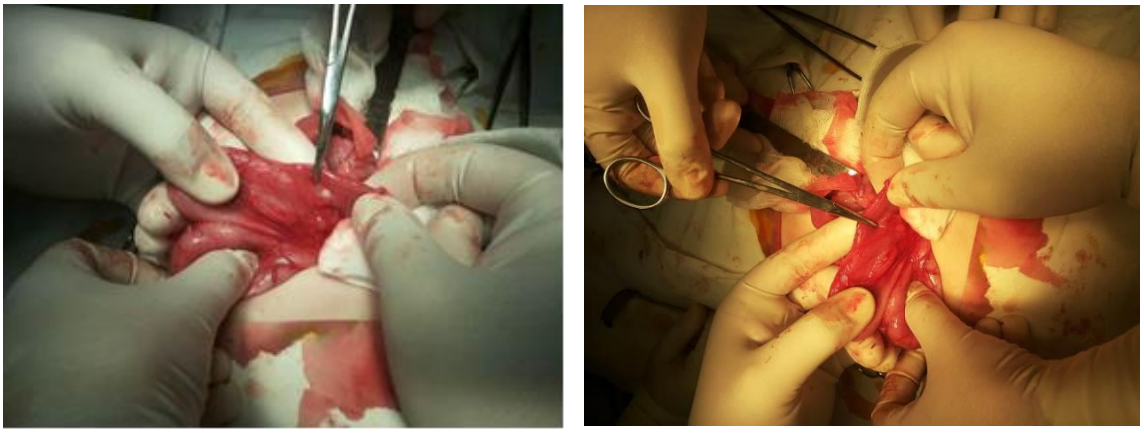

Fig. 2 Intra-operatory.

Discussions

Chronic vomiting in infants may have multiple causes, among which: organic vomiting, reflex vomiting and vomiting of neurologic sources [1]. The organic vomiting is present in: pylorus congenital atresia, duodenal or intestinal atresia/stenosis (there is a precocious onset of the vomiting, which has a bilious character; the X-ray examination tells exactly the location of the obstacle), cardia achalasia (the vomiting onset is within the first days of life and it disappears when positioning the infant in orthostatism), intestinal invagination (possible intestinal occlusion), pyloric spasm (precocious onset of vomiting even since the first days of life, frequent vomiting episodes, quantitatively reduced, during or after lunch; the X-ray examination revealed the absence of the gastric stasis liquid and a gastric plication (the vomiting has its onset several weeks after birth, it occurs during or after meals, and it is abundant; the postprandial Trendelenburg position prevents vomiting), annular pancreas (bilious vomiting) [1,2].

Vomiting represents the forceful expulsion of the gastric contents through the mouth, while the regurgitation means the effortless expulsion of the milk, usually accompanied by air. In the regurgitation caused by esophageal congenital atresia, the expelled liquid, at the first feed, does not have a gastric contents. In the atresia with tracheoesophageal fistula, the infant may present vomiting, cough, and cyanosis [3].

The reflex vomiting may be: infectious (gastroenteritis, urinary infections, ear infections, pneumonia, meningitis, septicemia, neonatal peritonitis – fever, meteorized and edematized abdomen), toxic (intoxications), metabolic (uremia, acidoketosis, ammoniemia, diabetes mellitus, congenital adrenal hyperplasia – Debre Fibiger: pseudo-hermaphroditism, vomiting starting the second week of life, hyperpigmentation, virilization, inapetence, hyperpotassemia, toxic state), food intolerance – intolerance to the cow milk proteins [4,5]. The vomiting episodes with neurologic causes are represented by: intracranial hemorrhages, tumors, abscesses, hematomas, meningitis; the vomiting may also be psychogenic [1,4]. The frequency of the duodenal stenosis: 1:20000-1:40000 births [6].

The duodenal obstructions can be caused by some intrinsic and/or extrinsic lesions. The intrinsic lesions, in the form of diaphragm, atresia, or stenosis usually occur in the second part of the duodenum, between the fifth and the tenth week of gestation, because of the vacuolization failure and the recanalling of the duodenum from its solid stage [7].

In the present case, the stenosis occurred because of an incomplete, intraluminal diaphragm which explains the intermittent character of the vomiting episodes and a diagnosis set when 7 ½ months and not during the neonatal period.

The extrinsic obstructions can be caused by an annular pancreas, malrotation or preduodenal portal vein [8]. The intrinsic lesions can appear periampullary in 20% of the cases or postampularry, and they are commonly accompanied by anomalies of the distal biliary tract and of the gallbladder. Other congenital anomalies are quite frequent, 30% of the cases being associated with Down syndrome and 50% of them with heart, genitourinary, anorectal malformations, esophageal atresia, and malrotations.

No other congenital malformations were diagnosed for this case.

Conclusions

We presented this case due to:

- its rich history, with repeated admissions for the vomiting episodes;
- the problems given by the etiologic diagnosis of recurrent vomiting episodes (gastro-esophageal reflux, metabolic disease, pylorus stenosis, other digestive tract malformations);

particularities of the duodenal stenosis.

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Correspondence to:

Senior Lecturer MD Singer Cristina, PhD
University of Medicine and Pharmacy Craiova,
2nd Pediatric Clinic, Emergency County Hospital Craiova
2-4, Petru Rares Street, Craiova, Romania
Phone: +40251502210

E-mail: singercristina@gmail.com