

Duodenal Disruption Diagnosed 5 Days After Blunt Trauma in a 2-Year-Old Child: Report of a Case

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Abstract

Blunt duodenal injury in children is uncommon and diagnosis is often delayed because of its retroperitoneal location. Both diagnosis and treatment are difficult. We report the case of a 2-year-old boy whose trauma injury was not reported for 5 days. His vital signs were stable, but he was vomiting bile-stained fluid and his stools were white. The third portion of the duodenum was completely disrupted, and was repaired by pyloric exclusion with duodenal and bile duct drainage. The child recovered uneventfully. We discuss the diagnostic strategies and therapeutic measures for this type of injury.

Key words Duodenal rupture \cdot Blunt trauma \cdot Child \cdot Abuse

Introduction

Duodenal injuries in children usually result from blunt trauma.¹ The overall incidence of gastrointestinal tract injury resulting from blunt abdominal trauma is only 0.6%, and duodenal injuries are found in 22% of these patients.² An operative delay of more than 24h is reported to increase the complication rate from 29% to 43%, and mortality from 11% to 40%.^{3,4}

We discuss the manifestations, diagnosis, and management of complete duodenal disruption in a young child, and review the relevant literature.

Case Report

A 2-year-old boy was admitted to the emergency department of a neighboring hospital. He was pale and vomit-

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ing, and looked very unwell. His blood pressure was 70/40 mmHg, with a capillary refill time of 3s, and his hemoglobin level was 5.7 g dl⁻¹. Evidence of old and new internal bleeding was noted over the body surface, and his abdomen was flat. The attending physicians diagnosed shock. The patient was intubated and fluid resuscitation with transfusion was started, to which he responded well. Computed tomography (CT) showed peritoneal fluid without parenchymatous organ injury, free air, or retroperitoneal gas (Fig. 1). He was transferred to our hospital for operative treatment of a suspected intra-abdominal hemorrhage after receiving a total transfusion of four units of red cell concentrate and 2200 ml of normal saline. On arrival to our intensive care unit, his vital signs were stable, except for a high fever of 39.0°C. An infusion of lactated Ringer's solution was started at $50-60 \text{ ml h}^{-1}$ ($6 \text{ ml kg}^{-1} \text{ h}^{-1}$). His urine output was adequate, at more than $1 \text{ ml kg}^{-1} \text{ h}^{-1}$. On day 3, we performed puncture of the abdomen to decrease intra-abdominal pressure and to improve ventilation, and aspirated 500 ml of fluid consisting almost entirely of blood. The fluid had a red blood cell count of 105 \times 10^4 cells μl^{-1} , a white blood cell count of 275×10^2 cells μl^{-1} , and an amylase level of 1587 IU l⁻¹. His serum amylase level was 83 IU1⁻¹. On day 4, he was weaned off the ventilator and the nasogastric tube was removed. On day 5, the nasogastric tube was reinserted because he again started vomiting bile-stained fluid and started passing white stools; however, his vital signs remained stable (Fig. 2). On day 6 after admission, an oral contrast CT and an upper gastrointestinal image showed a leakage from the third portion of the duodenum with no drainage into the jejunum (Figs. 3, 4). Thus, he underwent emergency laparotomy immediately, during which his vital signs remained stable. His blood pressure was 105/70 mmHg, his body temperature was 36.8°C, his urine output was adequate, and there were no signs of infection (white blood cell count 8300 cells µl⁻¹ and Creactive protein 12.8 mg dl⁻¹). Laparotomy revealed a

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Fig. 1. Contrast computed tomography of the abdomen on admission showing peritoneal fluid without parenchymatous organ injury, free air, or retroperitoneal gas



Fig. 2. The child's vital signs were stable. Urine output was adequate and there were no signs of infection. He was given lactated Ringer's solution at 50–60 ml h⁻¹. *sBP*, systolic blood pressure; *dBP*, diastolic blood pressure; *CRP*, C-reactive protein; *WBC*, white blood cells

Grade III laceration involving 100% of the circumference of the third portion of the duodenum. The point of perforation was covered by the mesocolon, and there was no spread of inflammation into the retroperitoneal space. The stumps of the duodenum were contused and lacerated, and needed debridement. There were hematomas in the mesentery. The duodenum was repaired by duodenojejunostomy with pyloric exclusion, retrograde tube duodenostomy, and gastrojejunostomy. A biliary drain was placed in the common bile duct, and a 986







Fig. 4. Upper gastrointestinal image on day 6 showing extraluminal leakage from the third portion of the duodenum and Gastrografin pools. The perforation appeared to be localized and did not spread into the peritoneal cavity

Fig. 3. Computed tomography on day 6, after the oral administration of contrast material, showing extraluminal leakage from the third portion of the duodenum without drainage into the jejunum

jejunostomy feeding tube was inserted (Fig. 5). On postoperative day (POD) 21, the biliary drain was removed. The nasogastric output was persistent and copious, but decreased gradually. On POD 29, the nasogastric tube and jejunostomy feeding tube were removed. The patient was later transferred to another hospital to separate him from his mother whose boyfriend had pushed him into the pointed corner of a table, inflicting this injury. The child was well when he was seen 1 year after the injury.

Discussion

Blunt trauma is a leading cause of death in children,⁵ its major consequences being solid organ injury and gastrointestinal perforations. The incidence of gastrointestinal tract injuries from blunt abdominal trauma is less than 1.0%, and only 17%–30% of these are duodenal injuries.^{2,6} According to the Organ Injury Scale for the Duodenum produced by the Organ Injury Scaling Committee of the American Association for the Surgery of Trauma (Table 1), Grade III represents complete disruption of the third part of duodenum. This grade of

Table 1. Duodenal organ injury scale according to the American Association for the Surgery of Trauma⁸

Grade	Injury description
I	
Hematoma	Involving a single portion of duodenum
Laceration	Partial thickness, no perforation
Hematoma	Involving more than one portion
Laceration	Disruption of <50% of the circumference
III	
Laceration	Disruption of 50%–75% of the circumference of D2 Disruption of 50%–100% of the circumference of D1, D3, D4
IV	······································
Laceration	Disruption of >75% of the circumference of D2 Involving the ampulla or distal common bile duct
V	
Laceration Vascular	Massive disruption of the duodenopancreatic complex Devascularization of the duodenum



Fig. 5. Repair of the injury was done with primary duodenojejunostomy, pyloric closure with 3–0 polyglycolic acid sutures, gastrojejunostomy, retrograde tube duodenostomy, and the insertion of a biliary drain

injury is estimated to account for less than 25% of duodenal injuries in children.^{4,6-8}

Approximately 50% of blunt duodenal injuries result from traffic accidents, 10% from abuse, and 10% from unknown causes. However, many of the "unknown" causes could be attributed to abuse, making abuse accountable for up to 20% of cases^{4,7} and the second most frequent cause of duodenal injury in children.

Because of the retroperitoneal location of the duodenum, the rating of this injury, its nonspecific signs and symptoms, and its unreliable history, diagnosis is usually delayed.⁹ Moreover, the changes occurring in isolated duodenal injury before the development of peritonitis may be subtle. Our patient's history of sideration was unclear and his vital signs and laboratory data did not suggest peritonitis, regardless of the 5-day delay from the time of injury, making diagnosis difficult. His condition was stable because the perforated duodenum was located retroperitoneally and packed by the mesocolon, without leakage. The early insertion of the nasogastric tube drainage may also have been effective.

Because the CT findings showed peritoneal fluid without parenchymatous organ injury, we suspected intestinal injury and observed him closely. The high concentration of amylase in his peritoneal fluid may have been an indication to operate, but we decided on conservative treatment in view of his improving general condition. We checked the CT images repeatedly because initial minimal or nonexistent radiographic findings are not uncommon.¹⁰ The present recommendation for repairing most duodenal injuries is primary closure with or without pyloric exclusion, but complex injuries should be managed by pyloric exclusion with gastrojejunostomy or pancreaticoduodenectomy.¹⁰⁻¹⁶ Our patient's injury was complex, so we performed pyloric exclusion. Some authors have reported that ret-

rograde jejunostomy tube drainage is effective for duodenal rupture.^{17,18} Accordingly, retrograde jejunostomy tube drainage and biliary drainage were initially effective in our patient. Although delaying the operation increases the complication rate and mortality,^{3,4} our patient suffered no complications, such as peritonitis or leakage.

In summary, we reported delayed diagnosis of complete disruption of the duodenum in a 2-year-old boy, because of his hemodynamic stability and normal laboratory data. Considering the favorable outcome despite the delayed diagnosis, we think that the procedure we performed specifically to decrease the intraduodenal pressure may be suitable for severe duodenal injuries.

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