Childhood Obesity: A Risk Factor for Omental Torsion

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ABSTRACT. Purpose. To determine the risk factors and clinical presentation of primary omental torsion (POT) in children.

Methods. Histopathology records of a pediatric hospital from January 1993 to March 2003 were reviewed to identify cases of POT. Hospital charts of patients diagnosed with POT were reviewed for demographic data and clinical presentation.

Results. A diagnosis of POT was recorded in 12 of 41,987 pathology records reviewed. Most of the patients were white (92%), male (75%), and 9 to 16 years old (75%). Weight percentiles were ≥95th in 11 (92%) of 12 patients. Body mass index was calculated in 9 of the 12 cases with 8 >95th percentile. Clinical presentation including right-sided abdominal pain, tenderness, and anorexia closely mimics acute appendicitis.

Conclusions. Obesity seems to be an important risk factor for POT in children. The presentation for POT seems to be less acute than with other causes of surgical abdomen. Pediatrics 2003;112:e460–e462. URL: http://www.pediatrics.org/cgi/content/full/112/6/e460; obesity, BMI, children, torsion, abdominal pain.

ABBREVIATIONS. POT, primary omental torsion; BMI, body mass index; CT, computed tomography.

P

Primary omental torsion (POT) unrelated to any other intraabdominal lesion was first reported by Eitel1 in 1899. By 1981, 223 additional cases had been reported,2 mostly in adults. Many more cases have been reported since, including those occurring in children.3–9 Historically, ~0.1% of children undergoing laparotomy for suspected appendicitis have POT.3 Although it is a rare cause of abdominal pain in children, it has no distinguishing features to separate it from other causes of a surgical abdomen.3 Problematically, its clinical presentation can closely mimic that of acute appendicitis.4,5,7,8,10,11

Obesity has been recognized as a predisposing factor of POT and explains its greater frequency in adults.12 In the past 2 decades, the prevalence of obesity in children and adolescents has doubled.13 It has been suggested that the increased rate of pediatric obesity may result in an increase of POT in younger age groups.9

At our institution, we perceived an increase in cases of POT in the last 5 years. This observation prompted us to review the medical records of children with POT to determine what predisposing factors may have led to its rise and to delineate its clinical presentation in children.

METHODS

We reviewed the pathology specimen records designated “omentum” from January 1, 1993, to March 31, 2003, at the Kosair Children’s Hospital. All cases with primary omental infarction were identified, and their hospital charts were reviewed. Demographic data, history, and physical, laboratory, and histopathology findings were recorded. Cases with secondary omental torsion or other omental pathology such as omental cysts, hernias, tumors, and adhesions were excluded.

RESULTS

Among 41,987 pathology records reviewed, 136 pathology specimens designated “omentum” were recorded during a 10-year period. POT was identified in 12 cases. The histopathology was reviewed, and all 12 cases were confirmed to have foci of hemorrhage and necrosis. During the same period, there were ~3300 appendectomies, a rate of POT of 1:275 appendectomies. The diagnosis in 8 (67%) of the 12 cases were made in the past 5 years. The majority of the patients were white (92%) males (75%). The children affected were predominantly (75%) in the 9- to 16-year age group; 3 were 3 to 4.5 years old. Weight percentiles were ≥95th in 11 (92%) of 12 patients, with 9 greatly >95th percentile. The body mass index (BMI) was calculated in 9 of 12 patients, and all were >85th percentile for age with 8 of the 9 >95th percentile. We were unable to calculate the BMI in 3 of the patients because the height was not documented in the medical record.

All children presented with abdominal pain of 1 to 5 days duration (mean of 3). It was localized in the right lower quadrant in 6, right upper quadrant in 5, and both right upper and right lower quadrant pain in 1 child. Only half of the children complained of anorexia. Nausea with vomiting was noted in only 2 patients, and nausea or vomiting were each observed in 1 child. No precipitating event was identified with the exception of 2 9-year-old children, 1 of whom was kicked in the abdomen 3 days before hospital admission and the other who ran into a wall, striking the right side of the abdomen. Body temperature on admission was normal in 7 children, 100.1 to 100.9°F in 3, and 101°F in 2. Abdominal tenderness localized to the right side was noted in all the patients. The mean peripheral white blood cell count was 13.2/m3 (range of 8.8–19.5; see Table 1).

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Computed tomography (CT) scan of the abdomen was performed in 8 patients. All of the scans showed evidence of inflammation in the omental fat on the right side of the abdomen.

**DISCUSSION**

The greater omentum is a sheet of peritoneum that hangs from the greater curvature of the stomach to the adjacent organs. It is attached more firmly to the greater curvature of the stomach to the diaphragm. The greater omentum grows freely from the greater curvature of the stomach and attaches itself to the diaphragm. The greater omentum is a sheet of peritoneum that hangs from the greater curvature of the stomach to the adjacent organs. It is attached more firmly to the greater curvature of the stomach to the diaphragm. The greater omentum grows freely from the greater curvature of the stomach and attaches itself to the diaphragm. The greater omentum is a sheet of peritoneum that hangs from the greater curvature of the stomach to the adjacent organs. It is attached more firmly to the greater curvature of the stomach to the diaphragm. The greater omentum grows freely from the greater curvature of the stomach and attaches itself to the diaphragm. The greater omentum is a sheet of peritoneum that hangs from the greater curvature of the stomach to the adjacent organs. It is attached more firmly to the greater curvature of the stomach to the diaphragm. The greater omentum grows freely from the greater curvature of the stomach and attaches itself to the diaphragm. The greater omentum is a sheet of peritoneum that hangs from the greater curvature of the stomach to the adjacent organs. It is attached more firmly to the greater curvature of the stomach to the diaphragm. The greater omentum grows freely from the greater curvature of the stomach and attaches itself to the diaphragm. The greater omentum is a sheet of peritoneum that hangs from the greater curvature of the stomach to the adjacent organs. It is attached more firmly to the greater curvature of the stomach to the diaphragm. The greater omentum grows freely from the greater curvature of the stomach and attaches itself to the diaphragm. The greater omentum is a sheet of peritoneum that hangs from the greater curvature of the stomach to the adjacent organs. It is attached more firmly to the greater curvature of the stomach to the diaphragm. The greater omentum grows freely from the greater curvature of the stomach and attaches itself to the diaphragm.

TABLE 1. Clinical Presentation of Primary Omental Torsion at Kosair Children’s Hospital (University of Louisville), 1993–2003

| Year of Report | Race/Sex | Age (Years) | Weight (kg) | Weight (Percentile) | BMI (Percentile) | Temp. (°F) | Pain | Duration (Days) | Anorexia | NVD* | WBC
|---------------|----------|-------------|-------------|---------------------|-----------------|-----------|------|----------------|----------|------|-----
| 1993 W/M      | 4.5      | 22          | >95th       | >95th               | 100.1           | RLQ       | 2    | Yes            | No       | 8.8  |
| 1995 W/M      | 9        | 53          | >95th       | >95th               | 100.9           | RUQ       | 5    | Yes            | No       | 11.5 |
| 1995 W/F      | 3        | 28          | >95th       | >95th               | 100.8           | RLQ       | 2    | Yes, V         | 16.6     |
| 1996 W/F      | 4        | 15          | >75th       | >95th               | 99.6            | RLQ       | 1    | No             | No       | 12.1 |
| 2000 W/M      | 9        | 52.6        | >95th       | >95th               | 99.4            | RLQ       | 2–3  | Yes            | N        | 14.8 |
| 2000 AA/M     | 16       | 100         | >95th       | >95th               | 98.7            | RUQ       | 4    | No             | No       | 15.3 |
| 2000 W/M      | 9        | 46          | >95th       | >95th               | 101             | RLQ       | 2    | Yes            | No       | 16.0 |
| 2001 W/M      | 12       | 70.8        | >95th       | >95th               | 97              | RUQ       | 3    | No             | No       | 12.4 |
| 2001 W/M      | 9        | 54          | >95th       | >95th               | 98.8            | RUQ       | 3    | No             | Yes, NV  | 11.1 |
| 2002 W/M      | 10       | 54.4        | >95th       | >95th               | 96.8            | RLQ       | 5    | No             | Yes, NV  | 9.4  |
| 2003 W/M      | 10       | 47.8        | >95th       | >95th               | 101             | RLQ       | 3    | Yes            | No       | 19.5 |
| 2003 W/M      | 9        | 48.9        | >95th       | >95th               | 97.8            | RUQ/RLQ   | 2    | No             | No       | 10.8 |

NVD indicates nausea, vomiting, diarrhea; WBC, white blood count; W, white; AA, African American; RLQk, right lower quadrant; RUQ, right upper quadrant.

Computed tomography (CT) scan of the abdomen was performed in 8 patients. All of the scans showed evidence of inflammation in the omental fat on the right side of the abdomen.

DISCUSSION

The greater omentum is a sheet of peritoneum that hangs from the greater curvature of the stomach to the adjacent organs. It is attached more firmly to the abdominal organs on the left side of the abdomen and attaches itself to the diaphragm. The greater omentum grows freely from the greater curvature of the stomach and forms the 4-layered, fat-laden omental apron. Initially transparent and thin in the neonate, progressive deposition of fat and inferior extension of this membrane occur throughout childhood and adult life. The amount of fat present in the omentum is reflected in the body habitus.

POT occurs when the omentum twists around its long axis, causing venous obstruction, edema, and vascular compromise. In most cases of omental torsion, the omentum is found twisted around the distal right epiploic artery, resulting in right-sided abdominal pain in 90% of cases.4 When no predisposing abnormality is found, omental torsion is considered primary.

The exact etiology of POT is not known. Previous reports suggested several predisposing factors including anatomic variations and excess omental fat associated with obesity5,10,12,15–18. Trauma, straining, overeating, overexertion, and sudden positional change were also implicated.10,14

Data derived from many sources show a remarkable increase in the prevalence of children with excess body fat in the United States.13 The BMI is the preferred method of expressing body fat percentile from clinical measurements. Children with a BMI >85th percentile for age are considered at risk for being an overweight adult, and those >95th percentile are overweight as defined by the Center for Disease Control. Adults with a BMI >95th percentile are obese. The prevalence of American children with a BMI >95th percentile has doubled between 1963 and 1991, along with an increase of approximately 50% in the prevalence of children with a BMI of >85%.13

Obesity causing irregularly distributed accumulations of excess omental fat has been cited as a predisposing cause of POT.5,10,12,15–18 A recent report by Varjavandi et al9 presented 4 children with POT who had BMI >95th percentile for age. They postulated that increased fat deposit in obese children outstrips the blood supply to the developing omentum, leading to either relative ischemia as the inciting event, increased omental weight leading to torsion, or traction to the most distal parts of the omentum. The heavily fat-laden omentum in these obese children predisposes the omentum to twist around its long axis, leading to vascular compromise, infarction, and gangrene.9

Similar to previous observations, we found that obesity was a strong predisposing factor in all our cases of POT. The preponderance of male children with POT in our series lends further support of obesity as a predisposing factor in that males have a higher accumulation of omental fat than females at the same total body weight.19 The children in our series were all overweight and frankly obese (>95th percentile BMI) in 89% of those whose BMI we were able to calculate. They commonly presented with pain on the right upper or lower abdomen. The occurrence of pain in this location and the higher incidence of right-sided omental torsion has been attributed to an omentum that is longer and more mobile on the right side.3 The location of the pain mimics the presentation of acute appendicitis and acute cholecystitis. Our findings of minimal or no fever, anorexia in only half the cases, and peripheral white blood cell counts that were elevated only mildly are similar to the findings of previous reports.5,6,9 The children appeared to be less ill-appearing than other children undergoing evaluation of an acute abdomen. A review of the literature on POT in children also showed that the children had fewer constitutional symptoms than those who had an inflamed appendix. The overall duration of the abdominal pain ranged from 1 to 8 days, with an average of 3 days. Neither the symptoms nor the physical findings present any characteristic pattern to suggest the diagnosis, because any or all of these features are common to several other acute abdominal diseases.12

Our cases occurred in the last decade: 4 in the first 5 years (1993–1998) and 8 in the last 5 years (1999–2003). This is a doubling of POT occurrence coincident with the increasing prevalence of obesity in...
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The increase in childhood obesity in our nation has reached epidemic proportions. POT is just one of the disorders that add to the morbidity of the obese child. As child advocates, the onus on obesity prevention, identification, and intervention rests on the primary care physician.

REFERENCES

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