Intussusception—the forgotten postoperative obstruction

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SUMMARY Ten children developed intussusception after laparotomy. The bowel obstruction caused by the intussusception was evident within 8 days of major laparotomy in 8 children and within 39 days in the other two. It developed after the usual postoperative laparotomy ileus, and did not respond to normal non-operative treatment not even to a barium enema. There was one palpable abdominal mass and no rectal bleeding. Only at surgery was the correct diagnosis made. Eight of 10 intussusceptions affected only the small-bowel; most of them were ileoileal, but 2 were ileocolic. Seven of the 10 intussusceptions needed only manual reduction. Postoperative recovery was uneventful.

During the 20-year period 1961–1981, more than 700 children with intussusception have been treated at this hospital. In 10 patients intussusception had occurred as a complication after major abdominal surgery for conditions other than intussusception. This is a postoperative problem that is often forgotten.

Patients

The average age was 30 months. Each patient had started some type of postoperative feeding. The signs and symptoms were those of small-bowel obstruction, but only one abdominal mass was palpated and blood per rectum was not seen (Table).

Follow-up of these infants and children with postoperative intussusceptions has been from a few months to 20 years, and there has been no recurrent bowel obstruction or intussusception.

Discussion

In reviewing the paediatric journals1–7 on postoperative intussusceptions, a clear definition of what is a 'true' postoperative intussusception must be made. We have included every intussusception that has occurred after a laparotomy, but have excluded any intussusception that has presented itself after surgery for a previous intussusception, or any intussusception seen after surgery outside the abdomen.1,3,5

At our hospital an intussusception occurs after 0.08% of all laparotomies. Hays and Gwinn8 reported that postoperative intussusceptions represented 3% of all intussusceptions they treated; they represent 1.4% of all intussusceptions we treat.

The sex ratio and age range of our patients are

Table Postoperative intussusception

<table>
<thead>
<tr>
<th>Case</th>
<th>Sex</th>
<th>Age</th>
<th>Year</th>
<th>Original operation</th>
<th>Start of symptoms</th>
<th>X-ray films</th>
<th>Duration of treatment (days)</th>
<th>Second operation</th>
<th>Type of intussusception</th>
<th>Lead point</th>
<th>Treatment</th>
<th>Complications</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>F</td>
<td>23 months</td>
<td>1961</td>
<td>Gastrointestinal bleeding</td>
<td>PO 33</td>
<td>SBO, No BE</td>
<td>2</td>
<td>PO 38</td>
<td>Ileoileal</td>
<td>0</td>
<td>Resection</td>
<td>None</td>
</tr>
<tr>
<td>2</td>
<td>F</td>
<td>2 months</td>
<td>1961</td>
<td>Pin from duodenum</td>
<td>PO 39</td>
<td>SBO, No BE</td>
<td>1</td>
<td>PO 6</td>
<td>Ileoileal</td>
<td>0</td>
<td>Manual reduction</td>
<td>Wound infection</td>
</tr>
<tr>
<td>3</td>
<td>M</td>
<td>17 months</td>
<td>1961</td>
<td>Gastrointestinal bleeding</td>
<td>PO 2</td>
<td>SBO, No BE</td>
<td>4</td>
<td>PO 10</td>
<td>Ileoileal</td>
<td>0</td>
<td>Manual reduction</td>
<td>Wound infection</td>
</tr>
<tr>
<td>4</td>
<td>M</td>
<td>6 months</td>
<td>1965</td>
<td>Huelman</td>
<td>PO 2</td>
<td>SBO, No BE</td>
<td>8</td>
<td>PO 10</td>
<td>Jejunai</td>
<td>0</td>
<td>Manual reduction</td>
<td>None</td>
</tr>
<tr>
<td>5</td>
<td>F</td>
<td>15 years</td>
<td>1965</td>
<td>Ileal bladder Wilms</td>
<td>PO 5</td>
<td>SBO, No BE</td>
<td>2</td>
<td>PO 41</td>
<td>Ileoileal</td>
<td>0</td>
<td>Manual reduction</td>
<td>None</td>
</tr>
<tr>
<td>6</td>
<td>F</td>
<td>8 months</td>
<td>1967</td>
<td>Ileal bladder Wilms</td>
<td>PO 5</td>
<td>SBO, No BE</td>
<td>11</td>
<td>PO 17</td>
<td>Jejunai</td>
<td>0</td>
<td>Manual reduction</td>
<td>None</td>
</tr>
<tr>
<td>7</td>
<td>M</td>
<td>12 months</td>
<td>1971</td>
<td>Swenson</td>
<td>PO 6</td>
<td>SBO, No BE</td>
<td>6</td>
<td>PO 11</td>
<td>Ileoileal</td>
<td>0</td>
<td>Manual reduction</td>
<td>None</td>
</tr>
<tr>
<td>8</td>
<td>F</td>
<td>13 months</td>
<td>1973</td>
<td>Diaphragmatic hernias</td>
<td>PO 5</td>
<td>SBO, No BE</td>
<td>6</td>
<td>PO 14</td>
<td>Ileoileal</td>
<td>0</td>
<td>Manual reduction</td>
<td>None</td>
</tr>
<tr>
<td>9</td>
<td>F</td>
<td>2 years</td>
<td>1977</td>
<td>Hiatal hernia Wilms</td>
<td>PO 8</td>
<td>SBO, No BE</td>
<td>6</td>
<td>PO 14</td>
<td>Ileoileal</td>
<td>0</td>
<td>Manual reduction</td>
<td>Wound infection</td>
</tr>
<tr>
<td>10</td>
<td>M</td>
<td>2 years</td>
<td>1980</td>
<td>Hiatal hernia Wilms</td>
<td>PO 8</td>
<td>SBO, No BE</td>
<td>6</td>
<td>PO 14</td>
<td>Ileoileal</td>
<td>0</td>
<td>Manual reduction</td>
<td>Wound infection</td>
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</tbody>
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BE = barium enema, SBO = small-bowel obstruction, PO = postoperative.
not very different from those reported in other papers. Moreover, the interesting common factor throughout most series is the major abdominal procedure.

It is hard to propose the same aetiological theories for the development of both primary and postoperative intussusceptions, because postoperative intussusceptions are thought to be caused by altered peristalsis due to prolonged and excessive manipulation with drying and bruising of the bowel, extensive retroperitoneal dissection, abnormal serum electrolyte levels, local hypoxia, chemotherapy, radiotherapy, anaesthetic agents, postoperatively administered drugs, or neurogenic factors. However, it is our impression that the multiple small and insignificant paediatric intussusception at laparotomy is much more common than the clinically significant postoperative intussusception. Most postoperative intussusceptions seem to develop within the time span of the immediate postoperative course. On close review, even though feedings had started, many of these infants and children had never really recovered from their postlaparotomy ileus which either blended clinically into the intussusception, or the intussusception had been there shortly after the abdomen was closed. Mollitt et al. gave this point strong support by showing that 75% of their paediatric postoperative laparotomy intestinal obstructions due to intestinal adhesions developed symptoms after 2 weeks, while 90% of their infants and children with postoperative laparotomy intussusception demonstrated their obstruction within 2 weeks of the laparotomy, with 64% occurring within the first week. We certainly found these facts and figures were similar in our patients. Obviously this is not the uniform picture, because 2 children had postoperative intussusceptions within 39 days of the first operation and in both the gastrointestinal tract was functioning between the two operations.

It is easy to appreciate why a postoperative intussusception is often overlooked, because apart from its rarity (0.08% of all laparotomies) signs and symptoms (pain, vomiting, palpable abdominal mass, rectal bleeding) are for the most part absent. The most common symptom (pain) is generally less obvious because the child is receiving postoperative pain medication. The vomiting may not be too obvious because of the usual nasogastric suction tube after laparotomy, although in many of these patients initial attempts to clamp or remove the tube are unsuccessful, or soon after oral feeds are started they are not well tolerated. The 'traditional' abdominal mass is almost impossible to find (if it is in fact looked for) because of the laparotomy incision and the difficulty in examining the still tender abdomen. Rectal bleeding from the more usual ileocolic intussusception occurs in only 60% of our patients, while bleeding from a small-bowel intussusception is even less common. Therefore, it seems that the best way to recognise such a postoperative intussusception is to remember that an ileus which is prolonged and unremitting immediately after a major abdominal operation (or returns within a few days after feeds have started) must be considered a postoperative intussusception until proved otherwise.

The only way to make the diagnosis, short of reoperation, is with barium studies and these are not always helpful because the intussusception almost always exclusively affects the small-bowel. In only 2 of our 10 infants and children was there an ileocolic intussusception and one was correctly diagnosed with a barium enema but hydrostatic reduction was unsuccessful. An interesting clinical fact that we had not appreciated was reported by Hayes when he stated that 'a granulocytic leucocytosis of major proportions is usually produced by intussusception'.

Once the diagnosis is suspected and the barium enema is normal indicating that the intussusception (if present) is in the small-bowel, the surgeon must decide how long to carry on with nonoperative treatment of the postoperative ileus or obstruction. A keen clinical observer may note that a postoperative paralytic ileus has few bowel sounds, while a postoperative mechanical obstruction has hyperactive bowel sounds. If the symptoms persist despite nasogastric suction, operation will of necessity be sooner. If the signs and symptoms and radiological picture are those of a continuing and unremitting small-bowel obstruction (which should resolve within a few days if it is due to early postoperative fibrinous adhesions) then reoperation should be considered within a week. This is a bedside decision left to the child’s surgeon.

It should be remembered that Mollitt et al. showed that post-laparotomy intussusception occurs generally within 2 weeks of surgery while postlaparotomy adhesive obstruction generally develops after 2 weeks. In our series, non-operative treatment continued for as long as 11 days, but on the average, surgery was performed about 5 days after the signs and symptoms had been noted and appropriate treatment either instituted or continued. All authors indicate that the longer one waits, the higher the morbidity and mortality rate.

At operation, as is usually the case, most of the intussusceptions can be manually reduced, but 3 of our children required resections because of irreducible intussusception (n=2) and because the small-bowel anatomy resembled a Meckel’s diverticulum
Orocraniodigital (Juberg-Hayward) syndrome with growth hormone deficiency

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SUMMARY A boy is described with Juberg-Hayward syndrome. In addition to unilateral cleft lip and palate, bilateral absent thumbs, anomalous carpal bones, and deformity of the radial heads, there were other skeletal abnormalities. The patient showed clinical and biochemical evidence of growth hormone deficiency which appears to be an integral part of the syndrome.

The orocraniodigital syndrome was first described by Juberg and Hayward in 1969, affecting 5 children from a sibship of six. The expression of the syndrome was variable in these individuals, the 2 brothers being more severely affected than the 3 sisters. The main features described were unilateral or bilateral cleft lip and palate, hypoplasia and distal displacement of the thumbs, bilateral forearm deformities with anterior displacement of the radial head, anomalous toes, and mild microcephaly. The inheritance appeared to be autosomal recessive with variable expression. The possible sex limitation suggested originally now seems unlikely since the case reported by Nevin et al. in 1981, in which the patient was a female with full expression of the syndrome. That patient also had additional skeletal abnormalities, notably absence of the pituitary fossa and flattened vertebral bodies. No evidence of endocrine dysfunction was found to account for her short stature.

In this report we describe a boy with features of the orocraniodigital syndrome, who had growth hormone deficiency as well.

Case report

The patient, a 17-year-old boy, was the only child of healthy unrelated parents. At the time of conception the mother was aged 43 and the father 38 years, neither had oral, cranial, or digital abnormalities and each was of normal intelligence. Their heights were 155 cm (−2 SD) and 160 cm (−2 SD). There were no congenital abnormalities in more distant family members. The patient was born at term after a normal pregnancy, with a birthweight of 1.24 kg. Unilateral cleft lip and palate and bilateral absent thumbs were noted at birth. Surgical correction of the cleft lip was carried out at 9 months and of the palate at age 2 years. His progress was satisfactory, apart from short stature and hearing impairment. At age 17 years his height was 143.3 cm (−4.7 SD), weight 49.4 kg (−2 SD), and head circumference 54.4 cm (−1 SD). Upper to lower segment ratio was 1:1. The facies (Fig. 1) showed a prominent forehead with hypoplasia of the mid-face, micrognathia, and repaired unilateral cleft lip and palate. He had a short neck, broad chest, and micropenis. Testes measured 12 ml in volume bilaterally and pubic

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References


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