

REFERENCES

1. Levy GH. Allergic reactions during anesthesia: diagnosis and treatment. In: Hersley SG, ed. *American Society of Anesthesiologists' Refresher Courses in Anesthesiology*, vol 11. Philadelphia: J B Lippincott, 1983: 109.
2. Beaven MA. Anaphylactoid reactions to anesthetic drugs. *Anesthesiology* 1981; **55**: 3-5.
3. Watkins J. Anaphylactoid reactions to IV substances. *Br J Anaesth* 1979; **51**: 51-60.
4. Stoelting RK. Allergic reactions during anesthesia. *Anesth Analg (Cleve)* 1983; **62**: 341-356.
5. Clarke RSJ. Adverse effects of intravenously administered drugs used in anaesthetic practice. *Drugs* 1981; **22**: 26-41.
6. Assem ESK, Frost PG, Lewis RD. Anaphylactic-like reaction to suxamethonium. *Anaesthesia* 1981; **36**: 405-410.
7. Fisher MMCD. Reaginic antibodies to drugs used in anesthesia. *Anesthesiology* 1980; **52**: 310-320.
8. Baldwin AC, Churcher MD. Anaphylactoid response to intravenous tubocurarine. *Anaesthesia* 1979; **34**: 339-340.
9. Farmer BC, Sivarajan M. An anaphylactoid response to a small dose of *d*-tubocurarine. *Anesthesiology* 1979; **51**: 358-359.
10. Fisher MMCD. Anaphylactic reactions to gallamine triethiodide. *Anaesth Intensive Care* 1978; **6**: 62-65.
11. Hirshman CA, Peters J, Cartwright-Lee I. Leukocyte histamine release to thiopental. *Anesthesiology* 1982; **56**: 64-67.
12. Mathieu A, Goudsouzian N, Snider MT. Reaction to ketamine: anaphylactoid or anaphylactic? *Br J Anaesth* 1975; **47**: 624-627.
13. Beamish D, Brown DT. Adverse responses to intravenous anaesthetics. *Br J Anaesth* 1981; **53**: 55-57.
14. Lascenaire MC, Moneret-Vautrin DA, Watkins J. Diagnosis of the causes of anaphylactoid anaesthetic reactions. *Anaesthesia* 1983; **38**: 147-148.

Bolus obstruction of the intestine

Case reports

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Summary

Two cases of intestinal obstruction caused by peaches are reported. In the first case steamed dried peaches were eaten by a 56-year-old woman who had undergone a Billroth I gastrectomy 18 years previously, while in the second case canned peach halves were swallowed whole by a 75-year-old edentulous man. The cases both typify the usual clinical setting of bolus obstruction, certain aspects of which are discussed. The responsibility of the attending practitioner to advise his high-risk patients with regard to their diets is emphasized.

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Intestinal obstruction from impaction of a food bolus is a well-documented though unusual phenomenon associated with a significant morbidity and an operative mortality of up to 5%.^{1,2} In some reports it accounts for 4% of simple small-bowel obstruction.^{3,4} By 1966 a list of 62 foods incriminated in bolus obstruction had been compiled,⁵ varying from grasshoppers to gooseberries and from popcorn to poppy-seeds. Three foods seem to predominate in different geographical areas: persimmons or 'date plums' in North America, peaches in South America, and citrus fruit, particularly in Britain.³ Peaches

seem to feature prominently in South Africa too, and 5 cases were reported in the *SAMJ* in 1983.² This stimulated us to report 2 further cases which were recently treated at Tygerberg Hospital.

Case reports

Case 1

A 56-year-old white woman presented with a 12-hour history of acute onset of severe abdominal cramps associated with vomiting. The symptoms had gradually worsened until the abdominal pain had become constant. The patient had undergone a Billroth I gastrectomy for peptic ulcer disease 18 years previously. She had also had an appendectomy as a child. On examination the patient was acutely ill; the abdomen was distended and diffusely tender, and there were no bowel sounds audible. The blood pressure was 150/90 mmHg, pulse rate 102/min and oral temperature 35°C. An abdominal radiograph revealed several small-bowel fluid levels, supporting a clinical diagnosis of small-bowel obstruction due to adhesions. At surgery a bolus obstruction of the mid-ileum caused by two dried peach halves was found — these were evacuated via an enterotomy. No intraperitoneal adhesions were present and no other lesion was evident. The patient's postoperative course was complicated by ileus and prerenal uraemia which responded rapidly to appropriate therapy. She was discharged in good health 9 days after admission. The patient recalled eating a bowl of steamed dried peaches a few hours before the onset of her symptoms.

Case 2

A 75-year-old edentulous coloured man presented with a 7-hour history of severe cramping abdominal pain localized mainly in the left iliac fossa and radiating towards the umbilicus. The pain was associated with nausea and vomiting, and by the time of presentation was constant. He had never had an

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abdominal operation, and had been quite healthy up to this point. Clinically, the patient was acutely ill; he had a markedly distended abdomen characterized by diffuse tenderness and board-like rigidity, absent bowel sounds and an empty rectum. The temperature was 35°C, blood pressure 110/80 mmHg, pulse rate 112/min, haemoglobin 16,0 g/dl, white blood cell count $14,7 \times 10^9/l$. Abdominal radiographs showed multiple small-bowel fluid levels. A diagnosis of small-bowel obstruction of uncertain origin was made. At surgery a bolus obstruction was identified in the terminal ileum approximately 30 cm from the ileocaecal valve. Another smaller bolus was identified in the stomach. No intrinsic bowel lesions could be identified. The bolus could not be easily broken by palpation, and was therefore evacuated via an enterotomy. It consisted of three canned-peach halves. Another peach half was evacuated from the stomach. Several more similar boluses were palpated in the large bowel but these were not removed. The patient made an uneventful recovery and was discharged in good health 9 days later. He subsequently recalled eating a tin of canned peaches, which he had consumed with unusual alacrity — and with an unusual quantity of local wine.

Discussion

There are certain clinically identifiable groups of patients at particular risk for developing a food bolus obstruction: (i) the elderly, often senile, patient with no teeth or ill-fitting dentures which are frequently removed while eating,^{1,3,6,7} and (ii) the patient who has previously undergone a partial gastrectomy.^{1,8-10} There are also certain pathological conditions which have been associated with obstructions due to food boluses and bezoars, such as diverticula of the duodenum and small bowel,¹¹ and strictures of the small bowel, e.g. after tuberculosis.³ Interestingly enough, intraperitoneal adhesions have rarely been implicated in bolus obstructions — in the 84 cases described by Schlang and McHenry¹ adhesions definitively contributed to the obstruction in only 1 case.

Many anecdotal cases have been reported where a 'normal' person with an adequate complement of teeth and no previous abdominal surgery bolts his food and subsequently develops a bolus obstruction — the youngster competing in a cherry-eating competition⁶ is an example.

The clinical picture of bolus obstruction varies considerably from the child with transient cramps due to 'green apple colic' to the patient presenting with severe but transient migrating abdominal cramps to the acutely ill patient who presents with complete intestinal obstruction.

The site of impaction in bolus obstruction is usually the distal ileum approximately 100 cm from the ileocaecal valve;⁶ at this site the bowel lumen is narrowest and peristaltic activity is most sluggish.¹² The obstruction may, however, occur anywhere in the bowel, including the sigmoid colon and rectum.^{1,7}

The manner in which the bolus obstruction is relieved depends largely on the circumstances prevailing at surgery; if the bolus cannot easily be broken up by palpation and milked down through the ileocaecal valve, an enterotomy must be performed. It is extremely important to palpate the rest of the bowel to exclude a second bolus obstruction; cases have been

reported where a patient required a second laparotomy to relieve a further obstruction which had been missed during the first operation.¹³

Bolus obstruction following partial gastrectomy was first described by Seifert¹⁴ in 1930. Many cases have subsequently been reported;^{1,8-10} in one report 84 such cases caused by oranges are described.¹

The reason for the apparent increase in bolus obstruction after gastrectomy remains unclear. Norberg¹⁰ believes that the loss of the normal physiological pylorus allows larger than usual food boluses to enter the small bowel. The size of the anastomosis may also be important, since bolus obstruction is more common after a Billroth II than a Billroth I gastrectomy.^{1,10} Davenport¹⁵ thought that the loss of the stomach antrum was largely responsible for the loss of fragmentation of the ingested food. Equally inexplicable is the fact that the interval between partial gastrectomy and obstruction has usually been over 5 years.^{1,4,8,10} All that is certain is that a patient who has previously undergone a partial gastrectomy is at increased risk of developing a food bolus obstruction, and that this is related in part to his eating habits — particularly in relation to fresh oranges and other dried fruit.

Conclusion

Food obstruction is an avoidable entity in most cases. It is incumbent upon the surgeon to warn his patients after a gastrectomy to avoid eating citrus fruits and to chew carefully whatever food they do eat; it is equally the responsibility of the general practitioner to warn their geriatric and particularly their edentulous patients against the same high-risk foods.

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REFERENCES

- Schlang HA, McHenry LE. Obstruction of the small bowel by orange in the postgastrectomy patient. *Ann Surg* 1964; **159**: 611-622.
- Van Eeden A, Retief P. Intestinal obstruction and dried peaches. *S Afr Med J* 1983; **64**: 377.
- Davies DGL, Lewis RH. Food obstruction of the small intestine. *Br Med J* 1959; **2**: 545-548.
- Bevan P. Acute intestinal obstruction in the adult. *Br J Hosp Med* 1982; **28**: 258-265.
- Stephens FO. Intestinal colic caused by food. *Gut* 1966; **7**: 581-582.
- Ward-McQuaid N. Intestinal obstruction due to food. *Br Med J* 1950; **1**: 1106-1109.
- Faircloth DE, Robinson WJ. Obstruction of the sigmoid colon by grape seeds. *JAMA* 1981; **246**: 2430.
- Butler MF. Orange-pith ileus after partial gastrectomy. *Br Med J* 1959; **2**: 549-550.
- Kott I, Urca I. Intestinal obstruction after partial gastrectomy due to orange pith. *Arch Surg* 1970; **100**: 79-81.
- Norberg PB. Intestinal obstruction due to food. *Surg Gynecol Obstet* 1961; **113**: 149-152.
- Shocket E, Simon SA. Small bowel obstruction due to enterolith (bezoar) formed in a duodenal diverticulum: a case report and review of the literature. *Am J Gastroenterol* 1982; **77**: 621-624.
- Editorial. Bolus colic. *Lancet* 1940; **i**: 1170.
- Ebner E. Darmverschluss durch Nahrungsmittel. *Wien Klin Wochenschr* 1959; **71**: 743-745.
- Seifert E. Über Krautileus. *Dtsch Z Chir* 1930; **224**: 96.
- Davenport HW. *Physiology of the Digestive Tract*. Chicago: Yearbook Medical Publishers, 1961: 221.