Review Article

Intestinal pseudo-obstruction – a review

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Introduction

Pseudo-obstruction is an abnormality of intestinal motility severe enough to produce the clinical features of intestinal obstruction.

Normal intestinal motility depends upon the modulation by nerves and hormones of the intrinsic rhythmical contractility of the smooth muscle syncytium. Contraction occurs when hyperpolarising action potentials (spikes or electrical response activity, ERA) coincide with the peaks of a continuously fluctuating potential difference across the cell membrane (slow wave or electrical control activity, ECA) (Brading, 1979). Spike potentials are triggered by neurotransmitters but because of the fixed rate of the slow wave, the rhythmic contractile response also has a fixed maximum rate (3 c.p.m. in stomach, 12 c.p.m. in duodenum and 8 c.p.m. in ileum of man; Duthie, 1979). The contraction of the intestinal muscle coats is integrated by the myenteric plexus whose neurones lack specialized neuro-effector junctions but have varicosities containing vesicles which release a wide variety of neurotransmitters including acetylcholine, noradrenaline, 5HT, peptides and purines (Burnstock, 1982). Nicotinic cholinergic fibres mediate the rapid inhibitory descending reflex and purinergic fibres mediate the slower excitatory descending pathway (Hirst, 1979). The myenteric plexus also propagates the inter-digestive myoelectric complex (IMC) (Sarna *et al.*, 1981) which, in fasting man, sweeps aborally the length of the small intestine at intervals of 15 to 195 min, clearing the intestinal lumen. An abnormal bacterial population supervenes when IMC are abolished or absent. IMCs are stimulated by vagal activity (Wingate, 1982), motilin (Itoh *et al.*, 1981) and suppressed by the ingestion of meals (Heppell *et al.*, 1983).

After meals, mixing and an aboral movement of the contents is produced by segmenting contractions of the small bowel. Gastric emptying and jejunal motility are inhibited by the presence of nutrients in the distal small bowel (Spiller *et al.*, 1984).

Abnormalities of gut motility may be produced by disorders which affect the gut nerves, muscle layers, both of these or abnormal gut collagen and interstitium (See Table I).

Table I Aetiological classification of intestinal pseudo-obstruction

Gut nerve dysfunction

- Toxic – drugs, heavy metals, insecticides.
- Metabolic – amyloidosis, diabetes mellitus, porphyria, paraneoplastic
- Inflammatory – Chagas' disease, varicella, Kawasaki disease,
- Genetic – congenital and familial visceral neuropathy.

Gut muscle dysfunction

- Familial visceral myopathy.
- Dystrophia myotonica.
- Polymyositis.

Disorders affecting gut muscle and nerve function

- Myxoedema.
- Phaeochromocytoma.
- Hypoparathyroidism.
- Pregnancy.
- Enteroglucagonoma.
- Trauma.
- Jejuno-ileal bypass.

Disorders of gut collagen and interstitium

- Scleroderma.
 - Radiation.
 - Strongyloidosis.
 - Ehlers-Danlos syndrome.
 - Mesenteric panniculitis.
 - Ceroidosis.
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Accepted: 14 May 1985

Disorders of gut nerves

Drugs which inhibit intestinal motility are the most common cause of acute pseudo-obstruction, particularly atropine-like drugs (Faulk *et al.*, 1978), phenothiazines (Sriram *et al.*, 1979), clonidine (Bear & Steer, 1976), tricyclic antidepressants and vincristine (Rosenberg & Caridi, 1983). A wide variety of diseases may damage the myenteric plexus but the morphological changes have to be extensive before symptoms are produced (Smith, 1982). Congenital abnormalities of the myenteric plexus are usually evident soon after birth because of vomiting or obstruction and are sometimes associated with pyloric hypertrophy and malrotation of the gut (Tanner *et al.*, 1976), patent ductus arteriosus (Harris *et al.*, 1976) or urogenital tract plexus degeneration (megacystis-microcolon-intestinal hypoperistalsis) (Jountz, 1981), which is probably due to cholinergic neurone loss.

Inherited generalized disease of the autonomic nervous system (familial dysautonomia, Riley-Day syndrome) and congenital ganglioneuromatosis (Carnegie *et al.*, 1976) lead to small bowel pseudo-obstruction, pyloric hypertrophy and megacolon probably because both adrenergic and cholinergic nerves are involved.

Familial myenteric plexus degeneration (Schuffler *et al.*, 1978) is associated with generalized evidence of autonomic dysfunction, ataxic gait and hypotonia. The pupils are hyper-reactive to pilocarpine suggesting a denervation hypersensitivity due to cholinergic neurone degeneration. Other families exhibit an achalasia-like abnormality of the oesophagus and abnormal duodenal motility which responded normally, to cholinergic agents suggesting deficiency of non-adrenergic inhibitory mechanisms (Lewis *et al.*, 1978).

In diabetes mellitus, autonomic neuropathy is common, gastric emptying is frequently impaired (Loo *et al.*, 1984) but small bowel pseudo-obstruction is rare. Acute colonic dilatation may occur as a terminal event in severe diabetes (Paley *et al.*, 1961).

Intestinal pseudo-obstruction is also usually a terminal event in secondary amyloidosis (Legge *et al.*, 1970) but in familial amyloidosis with polyneuropathy, episodic diarrhoea and constipation are typical. The amyloid deposition in gut nerve is responsible for the abnormal gut response to cholinergic agents (Battle *et al.*, 1979). Rarer degenerative disease of the myenteric plexus such as glycolipid deposition in Fabry's disease (Friedman *et al.*, 1984) and porphyria (Gorchein *et al.*, 1982) may present as pseudo-obstruction syndromes.

In Europe and North America infective neuropathies are rare but have been reported following varicella (Walsh, 1982) and in some sporadic cases of myenteric plexus damage, immune reactions to an infective agent similar to that seen in mucocutaneous lymph node syndrome (Kawasaki disease) is possible (Franken *et*

al., 1979). In endemic areas Chagas' disease produces widespread abnormality of gut function (Oliveira *et al.*, 1983) due to the inflammatory reaction in the myenteric plexus (Smith, 1980).

Disorders of gut muscle

Failure of development of duodenal musculature has been reported (Handelsman *et al.*, 1965) but inherited metabolic abnormalities of gut and muscle usually present later. Hereditary visceral myopathy is a degeneration of the longitudinal muscle coat inherited as an autosomal dominant (Schuffler & Pope, 1977). Sub-clinical achalasia-like abnormality of the oesophagus or intestinal pseudo-obstruction may occur in association with urogenital tract smooth muscle degeneration (Faulk, 1978). An autosomal recessive-type associated with ophthalmoplegia has been described (Anuras *et al.*, 1983). Investigations of relatives may help in making the diagnosis and in genetic counselling.

Skeletal muscle disorders such as dystrophias myotonia (Lewis & Daniel, 1981) and polymyositis (Patterson & Rios, 1959) may produce abnormal gastric and duodenal motility.

Disorders affecting gut muscle and nerve

A wide variety of metabolic abnormalities may affect both gut nerve and muscle function. Myxoedema slows small bowel transit (Shafer *et al.*, 1984) sometimes producing acute pseudo-obstruction (Salerno & Grey, 1978). Congenital hypoparathyroidism may also produce intestinal pseudo-obstruction (Cockell *et al.*, 1973).

Pregnancy slows intestinal transit and renders IMCs less frequent (Scott *et al.*, 1983) and pseudo-obstruction is particularly frequent following Caesarian section (Ravo *et al.*, 1983).

Abdominal pain, vomiting, constipation or pseudo-obstruction may be presenting symptoms of pheochromocytoma (Turner, 1983) and the risks of laparotomy in both this condition and in myxoedema are particularly high.

A unique patient with an enteroglucagonoma had chronic constipation and villous hyperplasia (Gleeson *et al.*, 1971) but no other peptide hormone secreting tumours have been reported as causing pseudo-obstruction.

The mechanism of acute pseudo-obstruction of the colon after blunt abdominal trauma with sepsis (Addison, 1983) may be akin to pseudo-obstruction of the colon after jejunal-ileal bypass which is improved by antibiotic therapy, suggesting that the bacterial production of substances toxic to gut nerve and muscle is responsible.

Abnormalities of gut collagen and interstitium

In comparison with skeletal muscle, smooth muscle contains much more collagen and disordered collagen metabolism produces intestinal dysmotility.

The excess collagen in scleroderma leads to bowel symptoms ranging from pseudo-obstruction, volvulus, perforation and diverticulosis (Krishnamurthy *et al.*, 1983). Steatorrhoea appears to be due to bacterial overgrowth occurring in the absence of IMCs (Rees *et al.*, 1982).

Irradiation-induced recurrent pseudo-obstruction is also related to gut fibrosis and is particularly likely after pelvic irradiation (Lopez *et al.*, 1981) and similar fibrosis may result in severe strongyloidiasis (Bartholomew, 1977). Mesenteric panniculitis (Tytgat *et al.*, 1980), malignant disease of the mesentery or rare infiltrative disorders, such as ceroidosis (Boller *et al.*, 1976), may produce intestinal pseudo-obstruction mainly by reducing compliance of the bowel wall, but some involvement of the nerve supply may occur.

Investigation and management of intestinal pseudo-obstruction

A carefully taken history will frequently detect clues to the presence of intestinal dysmotility. Previous laparotomies with negative findings, oesophageal or intestinal dysfunction in relatives (Schuffler & Pope, 1976) or clinical features suggesting abnormal motility in more than one area of the gut should suggest chronic idiopathic intestinal pseudo-obstruction. A history or clinical features of scleroderma, thyroid disease, diabetes or skeletal myopathy should be sought and a careful drug and radiotherapy history obtained.

The investigation of dysmotility could require the elimination of a wide variety of metabolic and endocrine causes (Snape, 1981) but clearly the investigation plan should be tailored to the clinical problem. Assessment of oesophageal motility by manometry, cineradiography or scintigraphy is of value in assessing patients with presumed visceral myopathy. Radiological examination of the small intestine, preferably by barium infusion, is essential to eliminate sub-acute small bowel obstruction (Schuffler *et al.*, 1976). Jejunal manometry detects pseudo-obstructive motility but because intubation in itself will accelerate intestinal motility (Read *et al.*, 1983), non-invasive transit measurements using breath hydrogen monitoring after ingestion of non-absorbed carbohydrates may be preferred as a first assessment.

If a laparotomy is carried out and no obstructing lesion is found, a full thickness biopsy of the intestine should be considered, particularly when a previous laparotomy has also been negative. Silver stains of

horizontal sections through the gut wall fixed as a sheet (Smith, 1982) (Figure 1) will show abnormalities not seen by routine histological techniques (Krishnamurthy & Schuffler, 1983) and special stains to show fibrous tissue and muscle will help differentiate systemic sclerosis from chronic idiopathic intestinal pseudo-obstruction (Schuffler & Beegle, 1979), although even electron microscopic appearances may be normal in some cases. Suction biopsy of the jejunal mucosa is useful in excluding coeliac disease but the nerve fibres visible in these specimens are not known to reliably reflect changes in the myenteric plexus.

If a treatable cause for pseudo-obstruction cannot be found, management is directed to reducing gaseous distension, combatting bacterial overgrowth and stimulating intestinal motility. Schuffler (1981) finds

Figure 1 Examples of myenteric plexus pathology. (top) Myenteric plexus from the small intestine of a child of 7 months dying from total peristaltic failure. The extrinsic trunks are normal and well formed but do not appear to communicate at all with the ganglia which contain neurones without processes. (lower) A myenteric ganglion from a patient dying from carcinoma of the bronchus. The ganglion is invaded by lymphocytes and there is severe neuronal damage. Reproduced from Smith (1982) by kind permission of the author.

that patients with idiopathic pseudo-obstruction are most comfortable on a low-lactose and low-fibre diet which minimizes gas production. Treatment of bacterial overgrowth with intermittent antibiotic therapy can produce periods of prolonged improvement especially in scleroderma, possibly because the abnormal bacterial population itself is contributing to the abnormal motility (Justus *et al.*, 1983). A wide variety of drugs has been used to stimulate gut motility, including combinations of cholinergic and adrenolytic agents. Neely & Catchpole (1971) improved post-operative ileus with a combination of guanethidine 20 mg intravenously followed by repeated doses of bethanechol 2.5 mg subcutaneously or prostigmine 0.05 mg intravenously and this regime was also used with benefit in patients with chronic idiopathic intestinal pseudo-obstruction by Ballet *et al.* (1982). There have been no double-blind trials of drugs in pseudo-obstruction other than one of metoclopramide, which proved ineffective (Lipton & Knauer, 1980). Non-steroidal anti-inflammatory drugs appeared to be useful in rare patients with excessive circulating prostaglandins (Luderer *et al.*, 1983) although the response may not be sustained. For an acutely dilated gut, intubation and aspiration followed by a gradual reintroduction of elemental diet has been successful (Gibbons & Sullivan, 1978).

Unfortunately, idiopathic intestinal pseudo-obstruction is often diagnosed only after a number of negative exploratory laparotomies. Resection is best avoided (Schuffler & Deitch 1980) but may be beneficial to patients with severe localized abnormalities. Radical resection of a megaduodenum may be necessary if there is no improvement after a duodenojejunostomy (Newton, 1968). Because bacterial overgrowth in the dilated gut leads to high septic complication rate, antibiotic preparation of the bowel as for colon surgery is recommended (Schuffler & Deitch, 1980). In some patients with severe small bowel distension temporary ileostomy is of value (Keshavarzian *et al.*, 1983) although we have observed one patient with idiopathic intestinal pseudo-obstruction in whom the

ileostomy became obstructed with impacted hard faeces. Small bowel pseudo-obstruction complicating jejunio-ileal bypass is an indication that the bypass should be reversed (Wills, 1978) but colonic dilatation can temporarily be controlled by antibiotics active against obligate anaerobes (Barry *et al.*, 1977). Chronic symptoms and severe malabsorption may necessitate parenteral nutrition (Greenall, 1983) and some patients on total parenteral nutrition long-term may be enabled to take some social meals without pain and distension if a subtotal resection of their small intestine is carried out (Anuras, 1978).

Colonic pseudo-obstruction may be distinguishable from luminal obstruction by the presence of normal faecal consistency and haustral pattern on plain abdominal X-ray (Byrne *et al.*, 1981). Persisting dilatation of the transverse colon or the caecum to more than 12 cm indicates a risk of perforation (Melzig & Terz, 1978) which is usually caecal (Bern-ton, 1983). A barium enema given without bowel preparation can sometimes relieve colonic pseudo-obstruction (Dudley, 1958) in addition to giving diagnostic information, but preferably the colon is decompressed by colonoscopy, either directly (Nivatvongs *et al.*, 1982) or by leaving a tube in the caecum (Groff, 1983). Colonoscopy also detects mucosal necrosis which is a strong indication for colectomy. Colostomy in this condition carries a high mortality and probably should not be performed (Addison, 1983).

The syndromes of intestinal pseudo-obstruction are a continuing challenge to the clinician and our increasing understanding of abnormalities of gut muscle and nerve renders it likely that more forms of intestinal dysmotility will be detected as investigative techniques improve.

Acknowledgements

My thanks to Catherine Weeks and Joan Hodkinson for their patient secretarial assistance.

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