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CASE REPORT

Pneumatosis intestinalis associated with enteral tube feeding

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The Netherlands**Correspondence to**
Marcel Zorgdrager,
marcelzorgdrager@online.nl**SUMMARY**

A 49-year-old man presented with a Hinchey II perforated diverticulitis and underwent laparoscopic peritoneal lavage. During the postoperative course the patient received enteral tube feeding which was followed by a bowel obstruction accompanied with pneumatosis intestinalis (PI). Explorative laparotomy showed an omental band adhesion without signs of ischaemia. After a short period of total parenteral nutrition PI resolved almost completely and enteral tube feeding could be continued once again. In the weeks that followed the patient developed atypical bowel symptoms and recurrent PI which resolved each time the drip feeding was discontinued. Despite the mild clinical course, a CT scan showed massive PI on day 21 after the laparotomy. After excluding life-threatening conditions conservative management was instituted and the patient recovered completely after discontinuing the drip feeding. We present one of the few cases of subclinical PI associated with enteral tube feeding that could be managed conservatively.

BACKGROUND

Pneumatosis intestinalis (PI) is a rare radiological finding and is defined as the presence of extraluminal gas within the intestinal wall. It is first described in the early 1700s by Du Vernoy, who recognised it during cadaver dissections.¹ Nowadays, PI is frequently discovered during routine radiological examination and may be caused by a number of conditions such as sarcoidosis and *Clostridium* infection (table 1).

The clinical presentation varies from asymptomatic to life-threatening conditions, depending on the underlying cause. Although the exact aetiology remains unclear, prompt surgical evaluation is usually necessary to exclude potential fatal causes such as bowel ischaemia. A subclinical presentation of PI is very rare and leads to a surgical dilemma. We present one of the first case reports of PI after jejunal tube feeding, without acute abdominal symptoms.

CASE PRESENTATION

A 49-year-old man was admitted to a neighbouring hospital because of a Hinchey II perforated diverticulitis. Laparoscopic drainage was performed and two drains were positioned, one in the rectovesical pouch and the other paracolic next to the affected sigmoid. After initial good recovery the patient developed an ileus and conservative management which was started by intestinal intubation and gastric decompression, combined with aggressive

intravenous rehydration and a central venous catheter was placed for parenteral feeding.

After 3 days clinical improvement occurred and a gastroduodenoscopy was performed with the aim of placing a jejunal feeding tube. During endoscopy a reflux esophagitis grade B and an erosive duodenitis were seen but nonetheless a jejunal feeding tube was successfully placed and carefully enteral feeding was started. The next day the patient developed a small bowel obstruction and a CT scan showed a massive distension of jejunum with a narrowed jejunal segment and PI (figure 1). Because of a breach of trust, the patient was transferred to our university hospital for further treatment. Based on the calibre difference, an explorative laparotomy was performed on the same day. This revealed an internal jejunal herniation with dilated loops due to an omental band adhesion. There were no macroscopic signs of intestinal ischaemia or thrombosis. The omental band adhesion was divided and intestinal decompression through the jejunal and nasogastric tubes was performed. In accordance with current literature on diverticulitis no additional sigmoid resection was performed.³ The following days enteral feeding was carefully continued as discomfort and bloating continued. On the 16th day the patient developed fever and leukocytosis of $20.8 \times 10^9/L$, lactate level was 0.7 mmol/L. A new CT scan excluded an abscess and as PI had improved significantly, enteral drip feeding was continued (figure 2). In the following 2 weeks the patient developed an intermittent ileus as the enteral drip feeding was expanded. The patient showed no signs of a mechanical small bowel obstruction. The patient received a balanced formula of 250 mOsm/L at a rate of 10 mL/h that could be expanded to 1000 mL/day. Because of the bowel symptoms this was changed to 455 mOsm/L at a rate of 10–60 mL/h on day 21. A new CT scan was made which again showed massive jejunal PI (figure 3). No PI of the colon or portal venous gas was seen. Remarkably, after discontinuation of the jejunal drip, feeding the patient recovered quickly and no surgical intervention was necessary.

OUTCOME AND FOLLOW-UP

On the 41st day after the initial diagnosis the patient was discharged from our hospital in good clinical condition. An outpatient check afterwards showed a complete recovery of gastrointestinal function.

DISCUSSION

We present one of the first cases in which subclinical PI is associated with enteral tube feeding. This



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Table 1 Underlying conditions associated with pneumatosis intestinalis described in the literature²

Traumatic and mechanical	Inflammatory and autoimmune	Infectious	Pulmonary	Drug induced	Other
Blunt abdominal trauma	Ulcerative colitis	<i>Clostridium difficile</i>	Astma	Cytotoxic agents	Intestinal infarction
Endoscopy	Crohn disease	HIV and AIDS	COPD	Immunosuppression	Necrotising enterocolitis
Jejunioileal bypass	Appendicitis	Cryptosporidium	Cystic fibrosis	Corticosteroids	Scleroderma
Pyloric stenosis	Diverticular disease	Cytomegalovirus		Lactulose	Sarcoidosis
Gastroduodenal ulcer	Cholelithiasis	Rotavirus		α -Glucosidase inhibitor	Multiple sclerosis
Duodenal stenosis	Lupus enteritis	Adenovirus		Sorbitol	Hirschsprung disease
Malrotation	Celiac sprue	Varicella-zoster virus		Practolol	Primary immunodeficiency
Volvulus	Polymyositis	<i>Candida albicans</i>		Nitrous oxide	Graft-versus-host disease
Surgical anastomosis	Dermatomyositis	<i>Klebsiella</i>		Trichloroethylene	Quadriplegia
Enteric tube placement	Mixed connective tissue disease	<i>Lactobacillus</i>		Chloral hydrate	Pseudo-obstruction
Intussusception	Polyarteritis nodosa	<i>Mycobacterium tuberculosis</i>			Haemodialysis
Carcinoma		Whipple disease			Cholangiocarcinoma
Barium enema					Idiopathic (primary)

COPD, chronic obstructive pulmonary disease.

case report demonstrates that mild variants of PI exist and a conservative therapy is sometimes warranted. Usually subclinical PI is an uncommon radiological finding in which only intramural gas is present. The incidence is estimated at 3–10 000 in autopsy studies⁴ but is increasingly reported due to improvements and innovations in radiological imaging techniques. Through the last decades over more than 60 different diseases associated with PI have been described² (table 1). The exact pathological cause of PI remains unclear and various theories exist.⁵ First, intraluminal gas can diffuse across the mucosa due to increased intraluminal pressure or increased mucosal permeability. This increased pressure is seen in patients after blunt abdominal trauma and bowel obstruction, increased permeability in mucosal damage or necrosis and in patients with immunodeficiency or steroid therapies. The second theory beholds diffusion of bacterial gas due to direct mucosal invasion or indirect by gas accumulation, leading to an increase of intraluminal pressure. Finally, migration of pulmonary gas from mediastinal vessels to the mesenteric region due to alveolar damage could lead to PI. In patients with obstructive lung disease a

combination can occur in which an increased intra-abdominal pressure may be present together with an increased mucosal permeability due to steroid therapy.

The cause of PI in this case is difficult to determine and most likely several factors may have contributed. First, our patient could have experienced iatrogenic mucosal injury after two endoscopic procedures and placement of the jejunal feeding tube. During the second endoscopy an erosive duodenitis was seen which could be an indication of iatrogenous injury. Second, intraluminal pressure could have been raised because of the accumulation of drip feed, iatrogenic due to endoscopic air inflation, or bowel distension as result of the small bowel obstruction and omental band adhesion. Conventional X-rays and CT scans showed persistent bowel distension with signs of accumulation of intestinal fluids. Third, diffusion of bacterial gas due to bacterial overgrowth during antimicrobial therapy and enteral drip feeding may have been a possible cause.

PI is primarily a radiological sign which consist of cystic or curvilinear distribution of gas inside the intestinal wall.^{6,7} Other radiological findings include wall thickening, free intra-abdominal air and the presence of portal venous gas. The combination of a curvilinear PI and portal venous gas is associated with bowel ischaemia in 70% of cases, a life-threatening condition with 80% mortality.² Previous reports showed no significant correlation between the extent of PI and the severity of



Figure 1 A coronary plain which shows pneumatosis intestinalis in the jejunal area. The arrow points to the presence intramural gas. Notice the bright appearance of the jejunal feeding tube.



Figure 2 Control CT after 5 days shows minimal pneumatosis intestinalis on the left

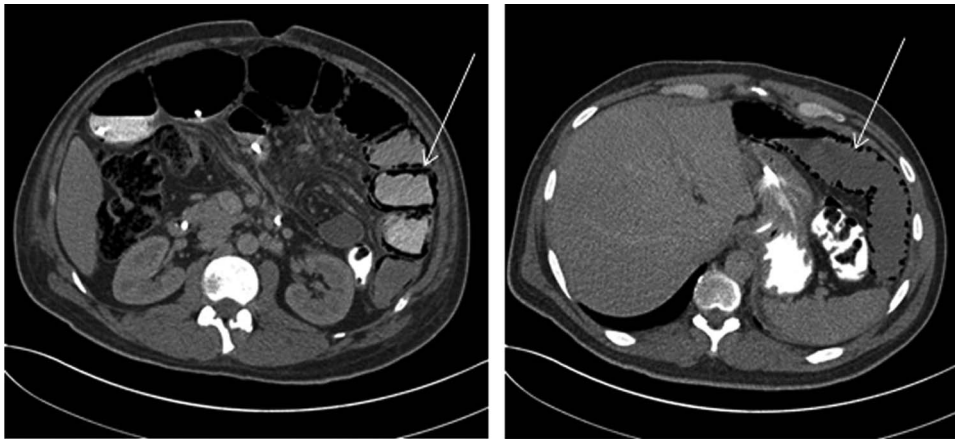


Figure 3 One month after initial presentation of perforated diverticulitis. Massive pneumatosis intestinalis is seen in the jejunal area. The arrows point to a typical curvilinear distribution of gas within the bowel wall.

the symptoms and the clinical presentation varies depending on the underlying cause. Life-threatening PI usually presents with signs of acute abdomen and shock, whereas benign PI most commonly presents with diarrhoea, abdominal pain, constipation, weight loss or tenesmus.

The extent of PI is in 42% of cases limited to the small intestines, followed by the colon in 36% of the cases and both are involved in 22% of cases.⁸ Radiological evaluation showed no PI in the colon of our patient. However, the patient suffered from diverticulosis which made it difficult to fully differentiate between cystic PI and diverticula. Furthermore, the fermentation of nutritional supplements varies between the small intestines and the colon, allowing segmental PI to develop. Finally, precipitating factors such as iatrogenous mucosal damage due to tube or bowel distension were only present in the proximal intestines of which the colon has been spared. In current literature several reports have described an association between an enteral tube feeding and PI.^{9–14} The pathophysiological mechanism for this is complex and multifactorial and appears to consist of a combination of mucosal injury due to a jejunostomy or enteric tube and increased intraluminal pressure. Mucosal damage leads a decrease of absorption of nutritional elements, which in turn leads to decreased bowel motility due to osmotic changes. This precipitates bacterial overgrowth and fermentation of unabsorbed elements and further increases intraluminal pressure next to a postoperative ileus. This mechanism fits well with PI in our patient and offers an excellent explanation for the subclinical course. Important to emphasise here is that PI is therefore not associated to substances in the tube feeding itself and is considered completely safe.

Our patient presented mainly with abdominal discomfort and constipation which resolved completely each time the drip feeding was discontinued. The patient received a relatively hyperosmolar feeding solution and the tube was not removed until the last CT scan. The clear relationship in terms of symptoms and jejunal drip feeding, in combination with the apparent explanation for the fever due to a central venous catheter infection, make that the tube feeding the most likely cause of PI. On the last CT scan no portal venous air or free intra-abdominal air was seen and lactate level was normal.

The management of PI remains controversial and different reports have proposed different algorithms.^{2 15 16} In cases of high suspicion of bowel ischaemia and acute abdomen immediate surgical exploration is advocated. Subclinical or benign PI

should be treated based on radiological imaging, clinical and physical examination in which the underlying cause can be found and managed properly. Most of these patients can be treated conservatively. Our patient recovered completely after stopping the drip feeding and similar results are described by other reports.^{11 13 14}

In conclusion, we present one of the few cases of a subclinical presentation of PI with an apparent association with enteral tube feeding and iatrogenous mucosal damage. Although the aetiology can be multifactorial, our report further strengthens the relationship with enteral tube feeding. When such an association exists, a conservative therapy seems justified. Other causes of PI should always be considered carefully to avoid unnecessary and potentially hazardous delay in treatment.

Learning points

- ▶ Enteral tube feeding can be associated with subclinical pneumatosis intestinalis (PI).
- ▶ Prompt surgical consultation is essential in PI to rule out life-threatening underlying conditions.
- ▶ Conservative management is justified in subclinical PI without symptoms of an acute abdomen.

Contributors MZ analysed the literature and was involved in drafting the manuscript. RP was involved in drafting and revising the manuscript.

Competing interests None.

Patient consent Obtained.

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