Stagnant Loop Syndrome: A Rare Cause of Severe Malabsorption

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Keywords
Stagnant loop syndrome · Small bowel overgrowth · Malnutrition · Anaemia

Summary

Background: Intestinal bacterial overgrowth as a consequence of postsurgical anatomical abnormalities as well as other small bowel diseases can lead to malabsorption.

Case Report: A female patient had several abdominal operations due to recurrent intestinal obstructions. Initially, she presented with severe megaloblastic anaemia. Subsequently, she suffered from weight loss, diarrhoea, oedema, recurrent anaemia (despite vitamin B12 substitution), and severe malabsorption of proteins, lipids, iron, and vitamins. Vague information about the performed surgeries, an anatomy of the bowel that was difficult to interpret, and an unusual cholestasis made it difficult to reach the diagnosis of bacterial overgrowth due to a stagnant loop syndrome. Treatment with antibiotics only temporarily improved the condition of the patient. After restoring bowel continuity and after the resection of an enteroenteric fistula as well as of a bowel conglomerate, the patient did not show any further symptoms.

Conclusion: The history of this patient indicates that the diagnosis of a stagnant loop syndrome may be difficult. The primary goal regarding surgically created small intestinal bacterial overgrowth should be the correction of the underlying small intestinal abnormality, whenever possible.

Schlüsselwörter
Syndrom der blinden Schlinge · Bakterielle Überbesiedlung des Dünndarms · Mangelernährung · Anämie

Zusammenfassung


Schlussfolgerung: Der Verlauf der Erkrankung dieser Patientin zeigt, dass die Diagnose eines «stagnant loop syndroms» schwierig sein kann und dass die beste Therapie einer chirurgisch hervorgerufenen bakteriellen Fehlbesiedlung des Dünndarms in einer chirurgischen Korrektur der anatomischen Abnormalität besteht, wann immer dies möglich ist.
**Introduction**

Several diseases of the small bowel can lead to malabsorption, such as coeliac disease, inflammatory bowel diseases, and small intestinal bacterial overgrowth (SIBO). SIBO can be a consequence of reduced intestinal clearance due to impaired peristalsis and/or anatomical abnormalities altering the luminal flow [1–7]. The main reasons for anatomical abnormalities are postoperative syndromes such as short bowel syndrome, blind loop syndrome, or stagnant loop syndrome. SIBO is characterised by one or more of the following symptoms: chronic diarrhoea, diffuse abdominal pain, flatulence, meteorism, malabsorption, and weight loss. In the following case, the clinical symptoms of the patient were unusual. Therefore, it took several years until the diagnosis was finally established and the patient could be cured by surgical correction of the stasis caused by a stagnant loop syndrome.

**Case Report**

The patient, a woman born in 1950, had an appendectomy in 1958. Between 1959 and 1988 she suffered from recurrent obstructions of the bowels and therefore underwent three strictureplastic surgeries and, as reported by the patient, resection of the terminal ileum. Details of the operations were not known as the surgery protocols were not available. In 1992 she developed anaemia, with a haemoglobin of 10.3 g/dl and a MCH (mean corpuscular haemoglobin) of 39.6 pg. Vitamin B12 serum level was low with <35 pmol/l (normal level 120–700 pmol/l), and vitamin B12 excretion into urine amounted to only 1.4% of the administered dose. Autoantibodies directed against intrinsic factor could not be detected. Serum folate concentration was within the normal range, and the bone marrow showed a normal iron content. Therefore, the diagnosis of megaloblastic anaemia due to vitamin B12 deficiency, presumably as a result of a short bowel syndrome, was made. A resection of the terminal ileum was described by an X-ray of the small bowel. In contrast, only a mild gastritis and duodenitis were considered to be normal. D-xylose absorption was reduced. Subsequently, vitamin B12 was substituted intramuscularly. A myelodysplastic syndrome was suspected but could not be proven.

In May 1995, the patient was admitted for the first time to our gastroenterology unit because of diarrhoea, diffuse abdominal pain, flatulence, meteorism, malabsorption, and weight loss. In the following case, the clinical symptoms of the patient were unusual. Therefore, it took several years until the diagnosis was finally established and the patient could be cured by surgical correction of the stasis caused by a stagnant loop syndrome.

**Table 1.** Selected laboratory values of the reported case at different time points

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<tbody>
<tr>
<td>Erythrocytes (3.8–5.2 million/µl)</td>
<td>3.69</td>
<td>3.4</td>
<td>3.95</td>
<td>2.70</td>
<td>4.27</td>
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<tr>
<td>Haemoglobin (11.8–15.5 g/dl)</td>
<td>10.9</td>
<td>9.8</td>
<td>12.1</td>
<td>8.3</td>
<td>12.7</td>
</tr>
<tr>
<td>Thrombocytes (140–440/µl)</td>
<td>575</td>
<td>669</td>
<td>531</td>
<td>719</td>
<td>349</td>
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<tr>
<td>Alkaline phosphatase (40–190 U/l)</td>
<td>192</td>
<td>173</td>
<td>146</td>
<td>139</td>
<td>74</td>
</tr>
<tr>
<td>CRP (&lt;5 mg/l)</td>
<td>39</td>
<td>18</td>
<td>21</td>
<td>110</td>
<td>22</td>
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<tr>
<td>Total serum protein (6.7–8.7 g/dl)</td>
<td>6.5</td>
<td>5.3</td>
<td>6.4</td>
<td>5.2</td>
<td>6.2</td>
</tr>
<tr>
<td>Albumin (3–5 g/dl)</td>
<td>3.8</td>
<td>3.1</td>
<td>4.2</td>
<td>2.7</td>
<td>3.6</td>
</tr>
<tr>
<td>Serum iron (49–151 µg/dl)</td>
<td>52</td>
<td>nd</td>
<td>58</td>
<td>28</td>
<td>nd</td>
</tr>
<tr>
<td>Serum copper (65–165 µg/dl)</td>
<td>250</td>
<td>146</td>
<td>123</td>
<td>147</td>
<td>nd</td>
</tr>
<tr>
<td>Ferritin (30–150 ng/ml)</td>
<td>27</td>
<td>27</td>
<td>54</td>
<td>444</td>
<td>73.8</td>
</tr>
<tr>
<td>Vitamin D3 (25-OH) (18.5–94 nmol/l)</td>
<td>&lt;12.5</td>
<td>50.1</td>
<td>nd</td>
<td>56.5</td>
<td>nd</td>
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nd = Not determined.

Severe Malabsorption: Stagnant Loop Syndrome
Weigand/Herfarth/Weigand

The following operations were performed:

1) Stagnant loop due to a side-to-side anastomosis between jejunum and terminal ileum. A bowel conglomerate including a fistula, which was resected. 2) A bowel conglomerate of 30 cm of small bowel led to a stasis due to interenteric circulation of the bowel content. Intraoperatively, the following situation was found (fig. 1): Beginning at the flexura duodenojejunalis, the jejunum was normal for 170 cm. Within the adjacent 10 cm an enteroenteric fistula was seen. At 200 cm, a wide side-to-side anastomosis between the jejunum and the terminal ileum, 20 cm proximal of Bauhin’s valve, was found. Bauhin’s valve was narrow, strongly suggesting a relevant interenteric circulation with reflux from the lower to the upper jejunum. From 200 to 300 cm, the small bowel was significantly dilated and scarred, showing the typical picture of a stagnant loop syndrome. At 300–330 cm, a small bowel conglomerate was found showing extensive inflamed and clotted changes. Within the next 30 cm, another stagnant loop syndrome with dilated intestinal loops was found. The last 20 cm of the ileum and Bauhin’s valve showed no pathological findings.

The following operations were performed:

- Resection of the convolute, including the enterocenteric and enterocutaneous fistulas, restoring the continuity between the proximal and distal loop.
- Resection of the jejunum at 170–180 cm, skeletonising the mesentery and performing a typical end-to-end anastomosis using a single-suture technique (Vicryl 4.0, backstitch).
- Removal of the side-to-side anastomosis between the jejunum and the terminal ileum. The side-to-side anastomosis was opened exactly at the site of the old anastomosis. The oval openings in the jejunum and in the ileum were closed by plastic occlusions using a single-suture technique.

Without any stenosis, complete continuity of the small bowel was restored by surgery. The histological workup of the resected bowel showed no signs of Crohn’s disease but inflammatory and ischaemic changes. 4 weeks after the operation the patient developed fever, abdominal pain, leukocytosis, and swelling of the abdominal wall. An interenteric abscess was found by means of a CT scan. By minilaparotomy, the abscess was located in the region of the median laparotomy scar, reaching deep into and between the small bowel loops. The abscess, with a volume of about 100 ml, was rinsed. Subsequently, each of the three cavities was drained by a separate Robinson drainage system. Microbiological examination verified *Escherichia coli*, *haemolytic streptococci*, and anaerobic organisms. The antibiotics ceftriaxone and metronidazole were given for 12 days.

After drainage of the abscess and antibiotic treatment, the patient recovered completely and felt good for the following years. She was seen annually in the hospital, the last time in January 2005. At that time her weight was constant at 53.5 kg, while she had formed stools, had no diarrhoea, and showed almost normal laboratory values (table 1). On the occasion of an interview in 2010, she was still in good health and felt fine.

Discussion

Our patient had two reasons for SIBO: failure of the gastric barrier due to *Helicobacter*-positive gastritis and reduced intestinal clearance. The main line of defence against bacterial colonisation of the small bowel is intestinal peristalsis [4]. Anatomical abnormalities can lead to reduced intestinal peristalsis and impaired intestinal clearance, which may result in bacterial overgrowth in the small intestine with colon-like bacteria, particularly with anaerobic gram-negative bacilli [2]. The surgically created anastomosis between jejunum and terminal ileum and the conglomerate of 30 cm of small bowel led to a stasis due to interenteric circulation of the bowel content. In addition, the enterocenteric fistula contributed to SIBO [5]. Furthermore, exocrine pancreatic insufficiency can also contribute to SIBO [3, 8]. However, the pancreatic function of the patient was normal.

The diagnosis of stagnant loop syndrome as the underlying cause for SIBO could have been made earlier if the patient and/or the local physician had had the correct information regarding the performed operations. Unfortunately, the protocols of the previous operations, some of which had been performed long ago, were not available. In contrast to the information given by the patient, the terminal ileum had not been resected, as was shown by colonoscopy. Nevertheless, a short bowel syndrome could have been possible. The anatomy of the small bowel as seen by X-ray was confusing, probably due to the unusual operations performed in the past. Two different radiologists did not detect a side-to-side anastomosis. The final diagnosis could only be made by an operation. It remains unclear why a side-to-side anastomosis was chosen as surgical treatment for an adhesive ileus in the patient. This unusual operation and the lack of correct information, combined with a rare presentation of severe malabsorption and cholestasis, led to a delayed diagnosis and therefore to a late effective treatment.

The gold standard for detecting SIBO is a culture of intestinal content. This diagnostic tool is costly and difficult for clini-
and iron. The very low concentration of cholesterol and tri-

liposomal bacteria [1, 7]. Maldigestion and malabsorption in
of mucosal inflammation and of metabolic action of the intra-
sorption and weight loss. Malabsorption can be a consequence
SIBO is usually not of clinical relevance [5]. The main fea-
leukocytes hepatis, probably due to the contraceptive agent, no
opsy showed normal portal tracts and bile ducts. Except for
the diagnosis can be obtained by liver biopsy [11]. Liver bi-
cult. In more than 90% of the patients with unclear cholestasis
serum copper. These symptoms, which are usually not related
as a reason for the anaemia.

The reason for transferring the patient to the gastrointesti-
nal unit was elevated alkaline phosphatase and elevated
secret copper. These symptoms, which are usually not related
to bacterial overgrowth, made the diagnosis even more diffi-
cult. In more than 90% of the patients with unclear cholestasis
the diagnosis can be obtained by liver biopsy [11]. Liver bi-
opsy showed normal portal tracts and bile ducts. Except for
peliosis hepatis, probably due to the contraceptive agent, no
liver disease was found histologically.

The degree to which nutrient absorption is impaired by
SIBO is usually not of clinical relevance [5]. The main fea-
tures of SIBO in this patient, however, were severe malab-
sorption and weight loss. Malabsorption can be a consequence
of mucosal inflammation and of metabolic action of the intra-
luminal bacteria [1, 7]. Maladjustment and malabsorption in
SIBO are responsible for impaired absorption of the compo-
nents of fat, carbohydrates, and proteins as well as of vitamins
and iron. The very low concentration of cholesterol and tri-
glycerides in the serum is a result of a malabsorption of fatty
acids and chylomicrons [1] and of a deconjugation of bile
acids by small bowel bacteria, making them unavailable for
micellar solubilisation. In addition, deconjugated bile acids in-
hibit the absorption and esterification of fatty acid and are
toxic to the enterocytes [1, 5–7].

A low serum albumin level in blind loop syndrome has been
previously described [12, 13]. Our patient had severe
protein malnutrition, probably as a combined result of pro-
tein-losing enteropathy and reduced protein synthesis in the
liver. A consequence of the obstruction and stasis of the small
bowel is an inflammation of the epithelium, leading to in-
creased exudation of plasma proteins. The ulcerations found
in the resected small bowel led to a loss of plasma proteins
and explain the observed blood loss in the stools. In addition,
protein synthesis in the liver was most likely diminished due
to bacterial deamination of amino acids [12, 13] as well as di-
minished brush border peptidases, followed by reduced amino
acid uptake [1]. The consequence of hypoalbuminaemia is a
diminished colloid osmotic pressure, followed by hyperaldos-
teronism, both leading to the observed oedema [14].

Diarrhoea, bloating, and distension of the abdomen, of
which the patient complained, are a consequence of impaired
monosaccharide absorption and subsequent bacterial degra-
dation of sugars [1, 5]. D-xylose absorption is often reduced
[4], as seen in this patient. Carbohydrate malabsorption and
malabsorption is due to gut damage and diminished entero-
cyte brush border disaccharidases [1]. The presence of
hydroxylated fatty acids and deconjugated bile acids may also
contribute to the abdominal symptoms [5].

Each time after treatment with antibiotics directed against
anaerobic bacteria (particularly with metronidazole; rifaximin
was not available at that time) the condition of the patient im-
proved, i.e. stools, laboratory tests, and body weight normal-
ised. However, the symptoms always recurred at varying inter-
vals. Only the elimination of the fistulas and the anastomosis,
and thus of the stagnant loop, in addition to restoring the
bowel continuity through surgery, finally led to permanent im-
provement, as was monitored by a gain in body weight and a
normalisation of laboratory values such as haemoglobin, albu-
min, iron, and vitamin B12. This may indicate that the primary
goal in surgically created SIBO should be the correction of the
underlying small intestinal abnormality, whenever possible [4].

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acknowledged.

Disclosure Statement

The authors have nothing to disclose regarding this case report and
declare that they have no competing interests.
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