Wernicke-Korsakoff Encephalopathy and Polyneuropathy After Gastroplasty for Morbid Obesity

Report of a Case

Fabio Cirignotta, MD; Mauro Manconi, MD; Susanna Mondini, MD; Giorgio Buzzi, MD; Paolo Ambrosetto, MD

Background: Gastric partitioning is a surgical procedure for the treatment of morbid obesity that may engender neurological complications, such as Wernicke encephalopathy and polyneuropathy.

Setting: A specialist hospital.

Patient: A 36-year-old woman developed Wernicke-Korsakoff encephalopathy and polyneuropathy 3 months after gastroplasty for morbid obesity. A magnetic resonance scan documented the diagnosis, and a clear improvement occurred after parenteral thiamine treatment. In our patient and in previously described cases of the literature, postsurgical vomiting is a constant finding that seems to be the precipitating factor of neurological complications of gastric partitioning.

Conclusion: Persistent vomiting after gastroplasty for morbid obesity should be considered an alarming symptom to treat immediately with appropriate measures.

Arch Neurol. 2000;57:1356-1359

GASTRIC PARTITIONING is used to control body weight by reducing the size of the stomach in patients with morbid obesity. This procedure may engender neurological complications due to starvation or vitamin deficiency. We report a case of Wernicke-Korsakoff encephalopathy and polyneuropathy after gastroplasty for morbid obesity and review the few previously described cases.

REPORT OF A CASE

A 36-year-old woman (weight, 115 kg; body mass index, 42.8; blood pressure, 165/95 mm Hg), underwent vertical banded gastroplasty with a Silastic ring according to the method of Eckhout. During the year before surgery, the patient had presented with repeated episodes of bulimia, with a 30-kg weight increase.

After surgery she consumed a liquid diet for 7 weeks, during which time she vomited frequently, with repeated syncope. Seven weeks after surgery she began consuming a semiliquid diet, but frequent vomiting persisted. She subsequently developed muscle weakness, gait impairment, and occasional diplopia. Three months after surgery, after a 23-kg weight loss, the patient was hospitalized again because of worsening asthenia and gait difficulties and the onset of confusion.

On admission, she was stuporous, with lateral gaze paralysis and a flaccid tetraparesis. Her pupils were equal and reactive, but deep tendon reflexes and plantar response were absent. Blood pressure was 140/80 mm Hg. Laboratory studies, including electrolyte, serum B12, hormone, serum albumin, and blood glucose levels, and cerebrospinal fluid analysis, yielded normal results. Erythrocyte transketolase and thiamine levels were not evaluated.

A cerebral magnetic resonance scan showed a marked increase in signal intensity in the periaqueductal gray matter, mammillary bodies, and dorsomedial thalamic nucleus (Figure). An electromyogram revealed diffuse sensorimotor neuropathy.

These findings suggested a diagnosis of Wernicke encephalopathy with peripheral neuropathy, and treatment was started with parenteral vitamins, including thiamine, 100 mg/d.

Following treatment, the patient had a rapid improvement in consciousness with disappearance of the lateral gaze paralysis, which was replaced for some days by
A nystagmus present in all directions; flaccid tetraparesis persisted, with a mild, predominantly proprioceptive sensory deficit and mild dysarthria.

Neuropsychologic testing performed on the following days disclosed a severe impairment in short-term memory.

During the following weeks the patient underwent rehabilitation, with slow and progressive improvement in her muscular strength. The patient refused to undergo a biopsy.

After 4 months the Silastic prosthesis was removed because of persistent vomiting.

Eight months later, the patient weighed 72 kg. Her memory had improved, and her motor deficit was limited to the anterior tibial muscles. She resumed her usual activities as a homemaker.

**COMMENT**

In 1881, Carl Wernicke termed **polioencephalitis hemorrhagica superioris** a syndrome with a fatal course characterized by ophthalmoplegia, ataxia, and confusion in two alcoholics and in a young woman with persistent vomiting after sulfuric acid ingestion. In all cases, an au-

---

Magnetic resonance images of the brain. Axial proton density–weighted images (A and C) and T2-weighted images (B and D) show markedly increased signal intensity in the periaqueductal gray matter, mammillary body (arrows, A and B), and dorsomedial thalami (arrows, C and D).
Topsy showed punctuate hemorrhages in the periventricular and periaqueductal gray matter and in the mammillary bodies.1

Some years later, Bonhoeffer conjectured that Wernicke encephalopathy (WE) and the “psychosis polyneuritica” described by Korsakoff represented two clinical manifestations of the same pathogenic process.2 Though WE is most frequently encountered in alcoholics, any disease causing malnutrition and thiamine deficiency can give rise to the syndrome, which has been described as a consequence of hyperemesis gravidarum,3-5 prolonged parenteral feeding,6 gastric carcinoma,7 pyloric obstruction,7 hunger strike,8 and long-term hemodialysis.9

Gastroplasty for morbid obesity is listed among the causes of WE. First reports of this complication were published between 1982 and 1984 by Haid et al,10 MacLean,11 Feit et al,14 and Fawcett et al.13 These authors described 6 young women who had not taken vitamin supplementation and who presented 2 to 4 months after surgery with persistent postoperative vomiting.

In 1982, Feit et al14 reported the development of severe polyneuropathy in two patients who underwent gastric partitioning; an autopsy revealed an extensive accumulation of lipids in nerve cell bodies and Schwann cells, which suggested the production of a toxic derivative of marked fat catabolism or loss of carnitine, essential for fat metabolism, as a result of starvation.

Some years later, Paulson et al15 described 6 patients who were exhibiting signs of confusion, inappropriate behavior, and peripheral neuropathy after gastric partitioning. They concluded that, although vitamin deficiency could have contributed to the neurologic complications, the basic physiopathologic process was uncertain, particularly in regard to the rapid metabolism of fat. In the case described by Oczkowski and Kertesz,16 ocular gaze palsy, ataxia, and sensorimotor neuropathy were associated with a normal mental state.

Seehra et al17 described two cases and stressed that thiamine deficiency is the physiopathogenetic factor of WE but not of other neurological sequelae of gastroplasty, such as peripheral neuropathies and psychosis, the causes of which are probably multifactorial.

In 1987, Abarbanel et al18 reported the occurrence of neurological complications in 500 patients who underwent gastric restriction surgery for morbid obesity, and described 1 patient with acute polyneuropathy and mild WE as well as 2 patients with Wernicke-Korsakoff encephalopathy (WKE) and mild polyneuropathy.

For technical reasons we could not measure the blood thiamine levels in our patient; however, the clinical findings, magnetic resonance imaging evidence, and clinical response to treatment seem sufficient to indicate WKE as a result of thiamine deficiency. The case was characterized by an association of encephalopathy and polyneuropathy, both severe.

The development of WKE after long intervals (2-20 years) following gastroplasty seems to be unusual19 and is probably due to mechanisms other than those underlying the subacute forms described above.

From the data reported in literature, it appears that both cerebral and peripheral injury in patients undergoing gastric partitioning may occur with a wide range of severity. The table below summarizes the cases of Wernicke-Korsakoff encephalopathy and polyneuropathy after gastroplasty reported in the literature:

<table>
<thead>
<tr>
<th>Source, y/</th>
<th>Age, y/Sex</th>
<th>Weight Before Surgery, kg</th>
<th>Weight Lost After Surgery at Onset of Illness, kg</th>
<th>Time From Surgery Until Onset of Illness, d</th>
<th>Rate of Weight Loss, kg/d</th>
<th>Frequent Vomiting</th>
<th>Clinical Features</th>
</tr>
</thead>
<tbody>
<tr>
<td>Haid et al,10 1982</td>
<td>33/F</td>
<td>130</td>
<td>23</td>
<td>105</td>
<td>0.22</td>
<td>Yes</td>
<td>WKE, PN?</td>
</tr>
<tr>
<td>Haid et al, 1982</td>
<td>32/F</td>
<td>120</td>
<td>30</td>
<td>70</td>
<td>0.43</td>
<td>Yes</td>
<td>WKE, PN?</td>
</tr>
<tr>
<td>MacLean,11 1982</td>
<td>24/F</td>
<td>NR</td>
<td>NR</td>
<td>90</td>
<td>NR</td>
<td>Yes</td>
<td>WE</td>
</tr>
<tr>
<td>Feit et al,14 1982</td>
<td>25/F</td>
<td>206</td>
<td>45</td>
<td>103</td>
<td>0.44</td>
<td>Yes</td>
<td>PN, PCH</td>
</tr>
<tr>
<td>Feit et al,14 1982</td>
<td>29/F</td>
<td>147</td>
<td>35</td>
<td>116</td>
<td>0.30</td>
<td>Yes</td>
<td>PN, PCH</td>
</tr>
<tr>
<td>McComas et al,12 1983</td>
<td>33/F</td>
<td>NR</td>
<td>NR</td>
<td>120</td>
<td>NR</td>
<td>Yes</td>
<td>WE</td>
</tr>
<tr>
<td>Fawcett et al,13 1984</td>
<td>45/F</td>
<td>NR</td>
<td>30</td>
<td>45</td>
<td>0.66</td>
<td>Yes</td>
<td>WE, PN</td>
</tr>
<tr>
<td>Oczkowski and Kertesz,15 1985</td>
<td>25/F</td>
<td>34</td>
<td>NR</td>
<td>90</td>
<td>NR</td>
<td>Yes</td>
<td>WE, PN</td>
</tr>
<tr>
<td>Paulson et al,15 1985</td>
<td>37/F</td>
<td>158</td>
<td>31</td>
<td>60</td>
<td>0.53</td>
<td>Yes</td>
<td>EN, PN</td>
</tr>
<tr>
<td>Paulson et al,15 1985</td>
<td>30/M</td>
<td>171</td>
<td>22</td>
<td>90</td>
<td>0.25</td>
<td>Yes</td>
<td>EN, PN</td>
</tr>
<tr>
<td>Paulson et al,15 1985</td>
<td>53/F</td>
<td>155</td>
<td>22</td>
<td>60</td>
<td>0.38</td>
<td>Yes</td>
<td>EN, PN, CH</td>
</tr>
<tr>
<td>Paulson et al,15 1985</td>
<td>42/F</td>
<td>126</td>
<td>13</td>
<td>120</td>
<td>0.11</td>
<td>Yes</td>
<td>EN, PN</td>
</tr>
<tr>
<td>Paulson et al,15 1985</td>
<td>20/F</td>
<td>130</td>
<td>NR</td>
<td>21</td>
<td>NR</td>
<td>Yes</td>
<td>EN, PN</td>
</tr>
<tr>
<td>Paulson et al,15 1985</td>
<td>25/F</td>
<td>148</td>
<td>22</td>
<td>42</td>
<td>0.54</td>
<td>Yes</td>
<td>EN, PN</td>
</tr>
<tr>
<td>Abarbanel et al,14 1987</td>
<td>27/M</td>
<td>NR</td>
<td>45</td>
<td>90</td>
<td>0.5</td>
<td>Yes</td>
<td>PN, WE</td>
</tr>
<tr>
<td>Abarbanel et al,14 1987</td>
<td>39/F</td>
<td>NR</td>
<td>47</td>
<td>365</td>
<td>0.12</td>
<td>Yes</td>
<td>WKE</td>
</tr>
<tr>
<td>Abarbanel et al,14 1987</td>
<td>26/F</td>
<td>NR</td>
<td>26</td>
<td>90</td>
<td>0.28</td>
<td>Yes</td>
<td>WKE, PN</td>
</tr>
<tr>
<td>Seehra et al,17 1996</td>
<td>55/F</td>
<td>171</td>
<td>25</td>
<td>42</td>
<td>0.59</td>
<td>Yes</td>
<td>WE, PN</td>
</tr>
<tr>
<td>Seehra et al,17 1996</td>
<td>47/F</td>
<td>97</td>
<td>30</td>
<td>150</td>
<td>0.20</td>
<td>Yes</td>
<td>WE</td>
</tr>
<tr>
<td>Present report</td>
<td>34/F</td>
<td>115</td>
<td>23</td>
<td>90</td>
<td>0.25</td>
<td>Yes</td>
<td>WKE</td>
</tr>
<tr>
<td>Mean</td>
<td>34.0</td>
<td>. . .</td>
<td>144.1</td>
<td>29.5</td>
<td>. . .</td>
<td>. . .</td>
<td>. . .</td>
</tr>
</tbody>
</table>

* WKE indicates Wernicke-Korsakoff encephalopathy; NR, not reported; PN, polyneuropathy; WE, Wernicke encephalopathy; PCH, pseudochorea; EN, encephalopathy; and CH, chorea triggered by action.
severity, from confusion to coma, from nystagmus to gaze paralysis, from WE to WKE, from loss of deep reflexes to acute or chronic polyneuropathy.

According to the literature, it appears that WE or polyneuropathy occurs most frequently 2 to 3 months after surgery (range, 20-365 days; mean, 92 days), after a weight loss of 13 to 45 kg (mean, 27 kg). The rapidity of weight loss ranges from 0.11 to 0.54 kg/d (mean, 0.36 kg/d); all patients presented with vomiting in the postoperative period (Table).

The symptoms that develop after surgery—the rapidity of weight loss and the amount of weight loss—have too wide a range to have a specific physiopathogenetic impact. However, it is interesting that all the patients described in the literature presented with repeated vomiting, despite the fact that persistent vomiting is not a frequent outcome after gastric partitioning (it occurs in about 4% of patients).20

As suggested by other authors, the vitamin absorption deficiency caused by vomiting may be the factor that triggers neurological complications in patients undergoing gastric surgery. Persistent vomiting in the postoperative period should therefore be considered an alarming symptom to treat immediately with appropriate measures.

Accepted for publication February 12, 2000.

Reprints: Fabio Cirignotta, MD, Department of Neurology, S. Orsola-Malpighi Hospital, via Albertoni 15, 40138 Bologna, Italy.

REFERENCES