CASE REPORT

Improvement of thoracic myelopathy following bariatric surgery in an obese patient

Shota Takenaka,¹ Yoshihiro Mukai,² Noboru Hosono,² Takashi Kaito¹

¹Orthopaedic Surgery, Osaka University Graduate School of Medicine, Suita, Japan
²Orthopaedic Surgery, Japan Community Healthcare Organization Osaka Hospital, Osaka, Japan

Correspondence to
 Dr Shota Takenaka,
 show@yb3.so-net.ne.jp

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SUMMARY

Revision spine surgery is extremely challenging in super-super obese patients (body mass index (BMI) ≥ 60 kg/m²). This is the first report describing how bariatric surgery was useful for a super-super obese patient with progressing myelopathy. A 44-year-old man with a BMI of 62.9 kg/m² presented with an ambulatory disorder caused by thoracic ossification of the posterior longitudinal ligament (T7–8). Before this paraparesis, he had undergone four spinal operations, and was not considered a good candidate for a fifth spine surgery. At the time of the fourth operation, he had reached a maximum weight of 205 kg (BMI 69.3 kg/m²). Instead, he underwent a laparoscopic sleeve gastrectomy. Sixteen months later, his body weight had decreased to BMI 35.2 kg/m², and he could walk without a walker. In addition to reducing our patient's load, a 'non-operative' form of dekyphosis due to altered thoracic spinal alignment secondary to weight loss may explain the improvement in his myelopathy.

surgery was especially challenging as his weight had reached 205 kg (BMI 69.3 kg/m²) (figure 1A). However, he was eventually able to walk steadily, 3 months after the fourth surgery.

Twenty-six months later, he experienced exacerbated symptoms of myelopathy. His Japanese Orthopaedic Association score (JOA score, 0=the worst, 11=the best) for thoracic myelopathy was 4.⁴ He had bilateral spastic paraparesis with hyper-reflexia in the lower limbs, but did not present with finger clumsiness. His bilateral upper limb score was 5 on manual muscle testing (MMT 0–5 grades (0=the worst, 5=the best)).⁵ He was unable to draw up his knees and had a score of MMT 3 for bilateral ankle dorsiflexion. His sensitivity to pinpricks was decreased below the umbilicus, and his sensory level was identified at T8. The numeric rating scale (NRS (0–10 scale; 0=no pain, 10=the worst pain imaginable)) was used for the evaluation of pain and numbness. The NRS score for numbness in the whole lower limbs was 9.

BACKGROUND

Super-super obese (SSO, body mass index (BMI) ≥ 60 kg/m²) patients¹ are problematic in terms of perioperative management, including the transfer and surgical approach.^{2,3} We describe a male SSO patient with thoracic myelopathy. This case is unique because: (1) as strong obstacles to additional spine surgery, he was SSO and had undergone four spinal operations; (2) this is the first report describing how bariatric surgery was useful for treating a patient with progressing myelopathy and (3) his maximum BMI of 69.3 kg/m² is, to our knowledge, the highest reported for a patient undergoing spine surgery in the English literature.

CASE PRESENTATION

A 44-year-old man (weight 186 kg, height 172 cm, BMI 62.9 kg/m²) experienced difficulty in ambulation. One month after the primary symptoms appeared, he could neither stand up nor remain seated, and was transferred to our hospital by ambulance.

Before experiencing paralysis, he had undergone four spinal operations for myelopathy due to ossification of the posterior longitudinal ligament (OPLL) or ossification of the ligamentum flavum. His spinal canal had decompressed posteriorly from C1 to T8 between ages 36 to 41 years. His paralysis remitted after each operation, but the fourth

INVESTIGATIONS

Standard blood findings including glycated haemoglobin were unremarkable. He had no underlying metabolic disease. MRI was impossible due to his body size. Hence, we used postmyelographic CT instead, which revealed anterior compression of the spinal cord due to the OPLL at T7–8, where the laminae had been removed in a previous surgery, along with posterior cerebrospinal fluid (CSF) space at this site (figure 2).

DIFFERENTIAL DIAGNOSIS

He had suffered from cervical myelopathy due to OPLL 3 years before this paralysis. However, the diagnosis of thoracic myelopathy in the patient with OPLL was made on both clinical examinations and imaging studies. He presented with lower limb paralysis, and his sensory level was at T8 with cord compression at T7–8. Cervical myelopathy was ruled out because of the lack of symptoms in the upper limbs.

TREATMENT

We initially considered performing anterior decompression and fusion, but his chest radiograph showed a tissue thickness of >13 cm from the skin surface to the ribs (figure 3), making the proposed surgery effectively impossible. To facilitate thoracic surgery later, we elected to postpone his fifth spine

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Figure 1 (A) Medical staff members preparing to perform the fourth spine surgery on the 205 kg patient positioned prone on the operation table. (B) The 104 kg patient seated on the examination table.

surgery and instead prioritised dietary treatment including bariatric surgery.

We consulted with bariatric treatment specialists at another hospital, where the patient underwent laparoscopic sleeve gastrectomy; consequently, his weight decreased to 116 kg (BMI 39.2 kg/m²) 3 months postoperatively. At that time, he could walk 100 m with a walker.

OUTCOME AND FOLLOW-UP

Sixteen months postoperatively, his weight was 104 kg (BMI 35.2 kg/m²) (figure 1B). His neurological status was also dramatically improved, and he was able to walk with a T-cane for 30 min. He had MMT 5 ratings in hip flexion, knee extension and ankle dorsiflexion bilaterally, and his JOA scores at this point were 7.5. The NRS score for numbness in the lower limbs was reduced from 9 to 2 with less area of numbness which was decreased to the bilateral plantar feet. Residual bilateral hyper-reflexia remained. At the latest follow-up, 48 months postoperatively, he had maintained his weight loss and ambulatory ability, and the planned thoracic surgery was no longer necessary. After losing more than 100 kg, it was possible to perform MRI (figure 4) which revealed persistent spinal cord compression at T7–8.

DISCUSSION

This case suggested two clinical issues. First, revision spine surgery for SSO patients is extremely difficult. Second, bariatric

Figure 2 (A) CT, sagittal reconstruction view. (B) CT axial view at T7–8.

Figure 3 CT scout view showing that the distance between the skin and ribs is >13 cm.

surgery may be useful to treat SSO patients with progressive thoracic myelopathy.

The first important clinical issue is the feasibility of surgery for SSO patients who have undergone previous spine operations. Considering that our patient had already undergone posterior decompression and that a posterior CSF space was recognised on CT, there was no indication for posterior decompression revision surgery. Conversely, anterior decompression was considered almost unfeasible based on a previous Japanese report describing

Figure 4 (A) MRI sagittal reconstruction view in the 104 kg patient. (B) MRI axial view at T7–8.

surgical complications in a similarly sized patient.⁶ The second important clinical issue is that bariatric surgery may be useful to treat SSO patients with progressive thoracic myelopathy. In this case, bariatric surgery and weight loss without an additional fifth thoracic surgery successfully enabled the patient to walk unassisted. There are two possible explanations for this. First, his legs weighed less, allowing him to move. Second, the shift in thoracic spinal alignment may have resulted in the dramatic improvement in his myelopathy. Given that his MMT score for ankle dorsiflexion improved postoperatively, the second explanation is possible because ankle dorsiflexion is not influenced by leg weight.

A recent study revealed significant differences in the standing thoracic kyphosis measure (T1–8) between subjects who were obese (average 45.1°, BMI 32.3 kg/m²) or normal weight (average 25.9°, BMI 21.5 kg/m²).⁷ Although the study was cross-sectional, the findings suggested that weight reduction could contribute to reducing thoracic kyphosis. Another longitudinal study investigating changes in disc height in 30 morbidly obese patients before (average BMI 42.8 kg/m²) and 1 year after (average BMI 29.7 kg/m²) bariatric surgery found that the mean L4–5 disc height was significantly increased, from 6 to 8 mm.⁸ These data indicate that weight loss can reduce the axial load to the spine which may affect spinal alignment. Accordingly, there are previous reports of thoracic spine surgery in which thoracic myelopathy caused by OPLL was improved by posterior decompression and dekyphosis by instrumentation without an anterior procedure.^{9 10}

Recently, bariatric surgery has been suggested to morbidly obese patients who were planned to undergo subsequent orthopaedic procedures,¹¹ especially knee replacement and lumbar surgeries. However, a PubMed search revealed just one report describing a morbidly obese paraplegic male patient who underwent bariatric surgery 22 years after sustaining a spinal cord

injury at T7.¹² This patient reduced his weight from 169 kg to 117 kg and improved his activities of daily living. However, his paraplegia remained unchanged, and his improvement was attributed to lessening his load. This effect was not the only reason for our patient's ambulatory improvement because our patient showed an improvement in myelopathy. His lower limb muscle strength actually recovered to MMT 5.

In conclusion, weight reduction can decrease axial load and alter the thoracic spinal alignment. In addition to reducing our patient's load, a 'non-operative' form of dekyphosis may explain the improvement in his myelopathy, suggesting that bariatric surgery should be considered in SSO patients who have undergone multiple spinal surgeries.

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Patient's perspective

I am very pleased with the results of this treatment. At that time, I could not walk at all, but now I can walk by myself. Moreover, I not only can see the doctor without assistance, but also found a job.

Learning points

- It is extremely challenging to perform revision spine surgery for super-super obese patients (body mass index ≥ 60 kg/m²).
- Bariatric surgery may be useful for super-super obese patients who have undergone multiple spinal surgeries.
- In addition to reducing the patient's load by weight reduction, a 'non-operative' form of dekyphosis may explain the improvement in our patient's myelopathy.

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