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# Thoracic Myelopathy Due to Ossification of the Yellow Ligament in Young Baseball Pitchers

*Shuichi Kaneyama, MD,* Minoru Doita, MD,* Kotaro Nishida, MD,* Takatoshi Shimomura, MD,* Koichiro Maeno, MD,* Yuichi Tamura, MD,† Masahiro Kurosaka, MD,* and Kazuo Yonenobu, MD†*

**Study Design:** Case series.

**Objectives:** To report rare cases of thoracic myelopathy due to ossification of the yellow ligament (OYL) in relatively young baseball pitchers and show clinical evidence of the role of dynamic mechanical stress on the development of OYL.

**Summary of Background Data:** The pathogenesis of OYL is still unclear. The majority of cases of OYL occur in middle-aged men whereas younger people are rarely affected. This has lead to the hypothesis that diffuse mechanical stress and degenerative changes correlate with the development of OYL. However, there have been no clinical reports demonstrating the critical role of mechanical stress in the ossification.

**Methods:** Two young highly active baseball pitchers with thoracic myelopathy due to OYL are presented. Both had no previous systemic disorders or family history of treatment for OYL. Magnetic resonance imaging and computed tomography demonstrated compression of the spinal cord by unilateral left sided OYL at the level of the thoracolumbar junction.

**Results:** Both patients were treated with posterior decompression. They recovered full muscle power after operation and resumed pitching training.

**Conclusions:** Patients had no other factors influencing the development of OYL and the lesions were localized at the left side in the thoracolumbar junction, indicating that repeated, localized rotatory mechanical stress caused by the pitching motion probably influenced the development of OYL in these young baseball pitchers.

**Key Words:** ossification, yellow ligament, mechanical stress, baseball pitcher, myelopathy, thoracic spine

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From the *Department of Orthopaedic Surgery, Kobe University Graduate School of Medicine, Kobe; and †Department of Orthopaedic Surgery, Osaka Minami Medical Center, Osaka, Japan.

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Reprints: Minoru Doita, MD, Department of Orthopaedic Surgery, Kobe University Graduate School of Medicine, 7-5-2 Kusunoki-cho, Chuo-ku, Kobe, 650-0017, Japan (e-mail: doita@med.kobe-u.ac.jp).



Ossification of the yellow ligament (OYL) is a pathologic condition that causes myelopathy or radiculopathy. It is reported that OYL is relatively common in the Japanese population compared with that in American or European populations.[1] However, the pathogenesis of OYL is not conclusively established.[2] Both systemic and local mechanisms are hypothesized. Otani et al[3] found OYL in 58% of 29 adult patients with kyphosis. They, therefore, suggested that localized mechanical stress affecting the yellow ligament was a contributing factor to ossification development. Certainly anatomically, the yellow ligament in the thoracic region is continuously subjected to static stress, and this is greater in flexion than extension. Furthermore, this disease involves predominantly men in their 60s and 70s (range 38 to 78),[4–8] whereas younger people are rarely affected.[9,10] Therefore, it is hypothesized that diffuse mechanical stress and degenerative changes correlate with the development of OYL.[1,3,10–13] However, there have been no clinical reports showing the critical role of mechanical stress on ossification.

In this report, we present 2 rare cases of relatively young baseball pitchers presenting with thoracic myelopathy due to OYL in the lower third of the thoracic spine. To our knowledge, there have been few cases describing OYL of such young athletes as this report.[10] We discuss the role of dynamic mechanical stress on the development of OYL.

## CASE REPORT

### Case 1

A 28-year-old baseball player, a right-handed pitcher felt weakness in his left leg when he was pitching during a game. He was practicing 6 hours every day for 6 days every week. He had no systemic disorders or family history of ossification of the posterior longitudinal ligament or yellow ligament. One month after that episode, he felt numbness on the bottom of both feet and developed frequent urination. He saw a local doctor who indicated weakness in both thighs, and he was referred to our institution.

Neurologic examination revealed spastic paraparesis and hyperesthesia of both feet. Magnetic resonance imaging of the thoracic spine showed a round intraspinal lesion, hypointense on T1-weighted and T2-weighted images, posterior to the spinal cord at the level of T10-T11 (Figs. 1A, B). Computed tomography after myelography revealed severe spinal stenosis

with left unilateral ossified yellow ligament and marked compression of the spinal cord at the level (Fig. 2).

At surgery, T10-T12 laminae were resected and the OYL was removed piece by piece. The postoperative examination revealed normal muscle strength in the lower extremities, and 1 year after operation, numbness and frequent urination has been resolved.

## Case 2

A 24-year-old professional baseball player, an orthodox right-handed pitcher, felt weakness in his left leg during training. He also had no generalized disorders or relevant family history. He saw a team doctor who indicated weakness in the left leg. Magnetic resonance imaging (Fig. 3) and computed tomography (Figs. 4A, B) revealed the development of unilateral left sided OYL at the level of T10/11-T11/12 and severe compression of the thoracic cord by them. He was referred to our institution.

Neurologic examination revealed slight muscle weakness of the left lower extremity and decrease in deep tendon reflexes.

Laminectomy of T10 and laminoplasty of T11 were carried out. Ossification of yellow ligament was found ventral to the left side of the laminae and resected. Examination at 6 months after the operation revealed a full recovery of muscle power and the patient resumed pitching training.

## DISCUSSION

The pathogenesis of OYL is still unclear. Both systemic and local mechanisms are hypothesized. It has been reported that patients with OYL have a higher frequency of diabetes mellitus, obesity, hyperinsulism, and calcium metabolism abnormalities.[7,9] These reports suggest that such systemic factors as hormones, growth factors, or vitamins may predispose the development of OYL. Our patients were 24 and 28-year-old high level athletes, although it is reported that this disease involves predominantly men in their 60s and 70s (range 38 to 78).[4–8] Therefore, genetic factors were taken into consideration and were carefully analyzed. However, these patients did not have ossification of the other spinal ligaments, any previous generalized disorders, and family histories of treatment for OPLL or OYL. Thus, in the present cases, it is suspected that both dynamic and static mechanical stresses might act as local factors in the development of OYL.

Mechanical stress has been proposed as a cause of hypertrophy and OYL.[1,10–12,14,15] Yoshida et al[12] suggested that the yellow ligament is hypertrophied by mechanical stress and that the main constituent of hypertrophy is the proliferation of type 2 collagen at the enthesis. It has also been suggested that mechanical stress induces hypertrophy of the yellow ligament and aggravates this condition, leading to ossification.[1,16,17] However, there have been no reports describing the role of mechanical stress in the development of OYL.

The most common level of symptomatic OYL is T10-T11, followed by T11-T12,[1] these levels appear to be particularly prone to degenerative processes owing to the high tensile force present in the posterior column.[2,15] Maigne et al[11] also reported that OYL occurred most frequently at the thoracolumbar junction and its appearance seemed to correlate with a unique orientation of

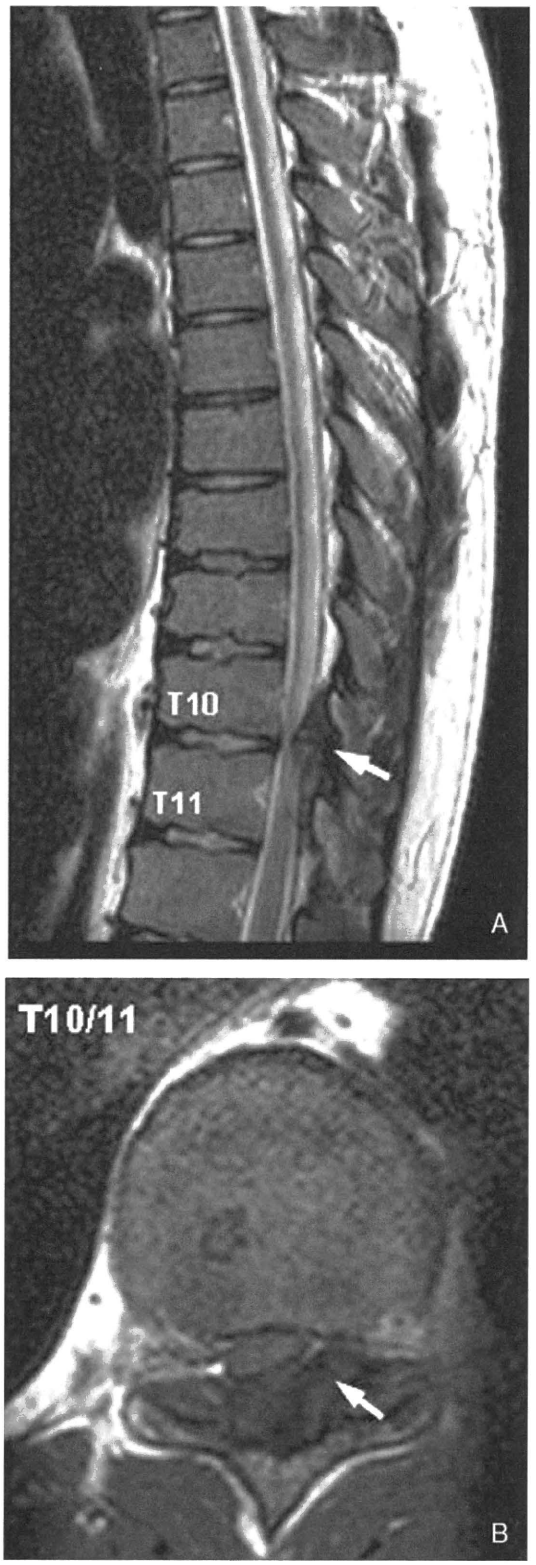


**FIGURE 1.** Magnetic resonance (MR) images in patient 1. A, Sagittal T2-weighted MR imaging showing stenosis with compression of the spinal cord by OYL at T10-T11 level (arrow). B, Axial T2-weighted image demonstrating spinal cord compression by OYL situated posterolaterally (arrow).

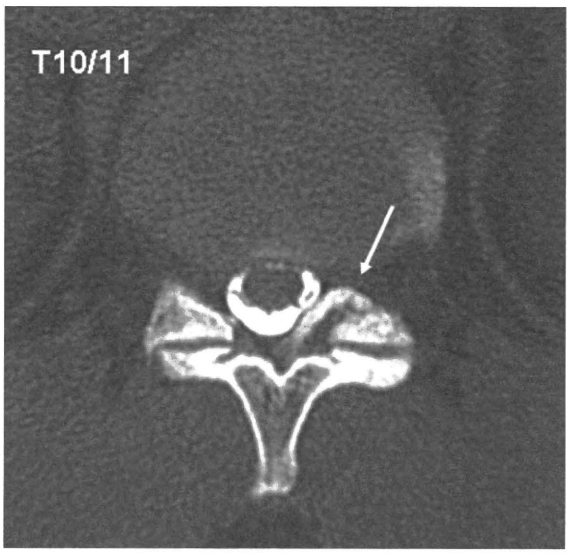


**FIGURE 2.** Computed tomography after myelography revealing the presence of OYL on the unilateral left side at T10-T11 (arrow) in patient 1.

the zygapophyseal joints that in turn contributed to increased rotatory instability and micromotion. That is to say, the larger range of rotation allows a larger tensile force to be applied to the yellow ligament thus increasing the frequency of OYL and leading to its enlargement OYL.

The 2 current patients were highly active baseball pitchers, and engaged in intensive training including pitching almost every day. The pitching motion has a unique mechanism compared with other types of throwing. To gain a high-velocity throw, the pitcher's trunk must rotate fast and it gives a huge rotatory stress to the spine. The thoracolumbar junction is the only region that rotates freely, so it cannot avoid rotatory stress, which can lead to hypertrophy and OYL. In addition, a pitcher must repeat this pitching motion over and over during games and training. Thus, we hypothesized the localized mechanical stress might have influenced the development of OYL. The fact that OYL existed at the level of T10-T11 supports this hypothesis.

Furthermore, OYL is commonly seen as a characteristic V-shaped or triangular-ossified lesion,[1,18] and unilateral OYL is extremely rare.[19,20] There have been only 2 cases of unilateral thoracic canal stenosis by osseous hypertrophy of the posterior spinal elements reported in the literature.[20] This is probably because ordinarily the spine is statically and dynamically symmetric and there are very few people who force asymmetrical rotatory tensile stress to the trunk. However, in both our patients OYL developed unilaterally, owing to the asymmetrical rotation in pitching form. This probably leads to cause asymmetrical unilateral mechanical stress to yellow ligament. However, there is a possibility that these 2 patients developed OYL incidentally, because the number of this reported cases is only 2.

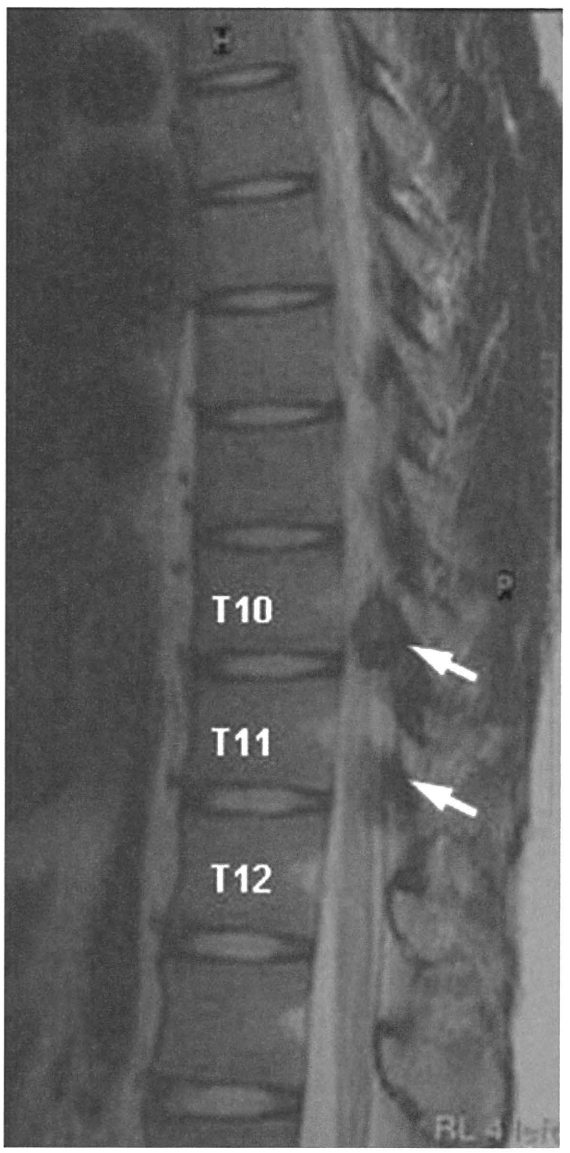


**FIGURE 3.** MR image in patient 2. Sagittal T2-weighted MR imaging revealing posterior indentation of spinal cord at T10-T11 followed by T11-T12 (arrows).

OYL usually occurs in middle-aged men probably as a result of repeated mechanical stress to the spine over a number of years. Conversely our patients were relatively young, healthy men. However, they were high-level pitchers and therefore repeated a pitching form, which gave a huge rotatory tensile force to the yellow ligament of the unilateral side at the thoracolumbar spine. Because there seemed to be no other factors influencing the development of OYL such as aging, systemic problems or family history of OYL, and the OYL was localized at the left side in the thoracolumbar junction, it is reasonable to assume that repeated, localized rotatory mechanical stress might have influenced the development of ossification of yellow ligament despite the patients younger age.

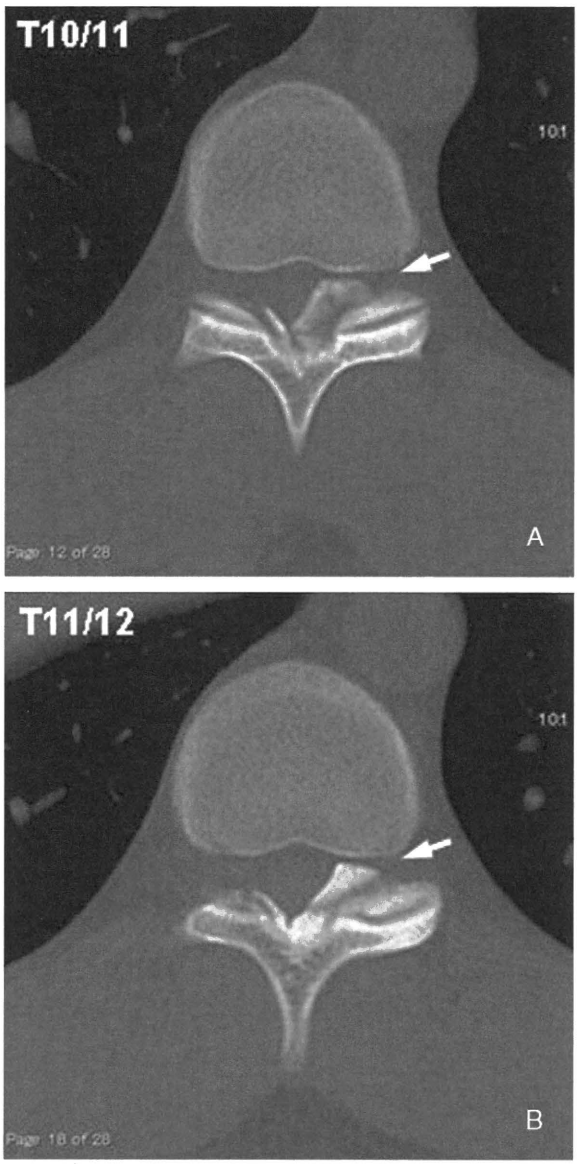


**FIGURE 4.** Computed tomography demonstrating the unilateral left-sided ossifications of the yellow ligament are shown at T10-T11 level (A) and T11-T12 (B) (arrows) in patient 2.

## ACKNOWLEDGMENTS

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CASE REPORT

# Ossification of the posterior longitudinal ligament in dizygotic twins with schizophrenia: a case report

**Shunji Matsunaga · Hiroaki Koga · Naoya Kawabata · Ichiro Kawamura · Masaki Otusji · Takanori Imakiire · Setsuro Komiya**

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**Abstract** The pathogenesis of ossification of the posterior longitudinal ligaments (OPLL) has not been clarified. We here report dizygotic twin sisters with OPLL of the cervical spine and propose a new pathogenesis of OPLL. This is the first report of dizygotic twins with OPLL. The twins suffered from schizophrenia, which might be related to the pathogenesis of OPLL. In addition, we investigated the occurrence of OPLL in 30 patients with schizophrenia who had been admitted to a mental hospital. OPLL of the cervical spine was found in six (20%) of them, with an incidence almost five times higher than the incidence of OPLL among the general population in Japan. Schizophrenia may have a increased susceptibility to OPLL.

**Keywords** Ossification of the posterior longitudinal ligament (OPLL) · Schizophrenia · Dizygotic twins · Calcineurin · Myelopathy

S. Matsunaga (✉) · H. Koga · N. Kawabata · I. Kawamura · M. Otusji · T. Imakiire
Department of Orthopaedic Surgery,
Imakiire General Hospital, 4-16 Shimotatsuo,
Kagoshima 892-8502, Japan
e-mail: shunji@m.kufm.kagoshima-u.ac.jp

S. Komiya
Department of Orthopaedic Surgery,
Graduate School of Medical and Dental Sciences,
Kagoshima University, 8-35-1 Sakuragaoka,
Kagoshima 890-8520, Japan

## Introduction

Ossification of the posterior longitudinal ligament (OPLL) is a hypertrophic condition of the spine associated with severe neurological deficit [1–5]. The disease was first reported by Key [6] in 1838, and Tsukimoto [7] reported a postmortem examination of a Japanese patient. The pathogenesis of OPLL has been studied since 1975 by members of the Investigation Committee of The Japanese Ministry of Public Health and Welfare. However, the pathogenesis of OPLL has not been clarified. Genetic background is thought to play a major role in OPLL from results of studies involving families [8] and twins [9]. However, DNA analysis has not shown specific genes conferring susceptibility to OPLL. The Japan-wide study of twins with OPLL revealed six of eight monozygotic twins had OPLL of the cervical spine. However, no dizygotic twins with OPLL were found, and the mode of inheritance of OPLL could not been determined. In this study, dizygotic twin sisters with OPLL of the cervical spine are reported, and a new pathogenesis is proposed.

## Case

Dizygotic twin sisters (Fig. 1), aged 69 years, suffered from schizophrenia for 40 years. The younger of the two exhibited OPLL of the cervical spine (Fig. 2), with a maximum spinal canal stenosis rate of 45%, but she did not exhibit myelopathy. In contrast, the older twin had almost the same size and degree of OPLL of the cervical spine (Fig. 3), but she exhibited severe myelopathy. Both sisters were short in stature, but laboratory data did not correspond

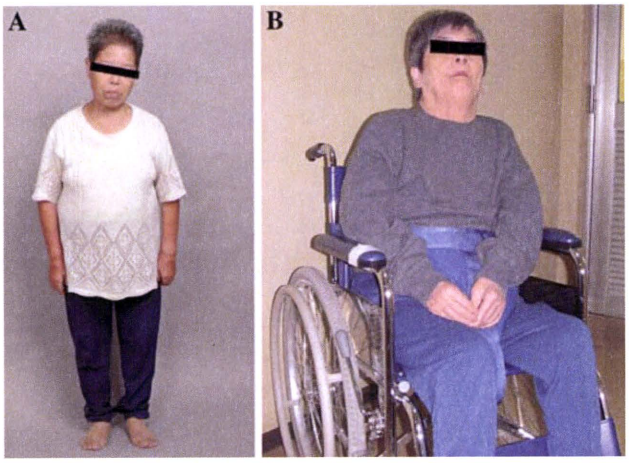


**Fig. 1** **a** The younger sister of the dizygotic twins. **b** The elder sister of the dizygotic twins

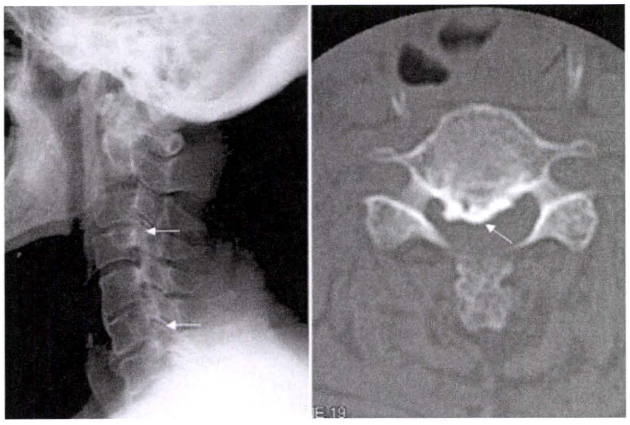

**Fig. 2** Plain X-ray film of the cervical spine and computed tomography of the younger sister of the dizygotic twins; *white arrows* show ossification of the posterior longitudinal ligament

to vitamin-D-resistant rickets, which is frequently associated with OPLL.

## Supplementary study

With the agreement of the families, we investigated the occurrence of OPLL in 30 patients with schizophrenia who had been admitted to a mental hospital. The study comprised 25 male patients and five female patients. OPLL of the cervical spine was present in six (20%) (five men and one woman) of the 30 patients with schizophrenia (Fig. 4).

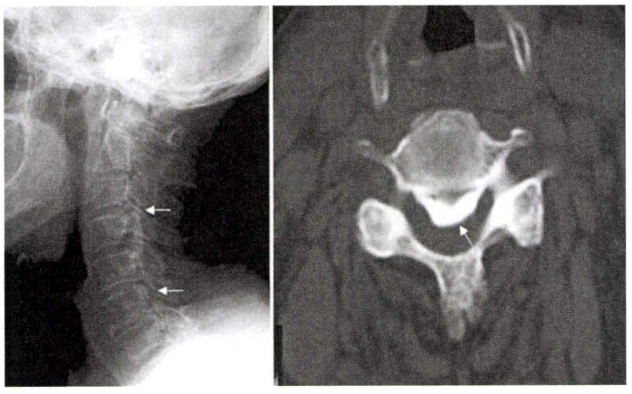

**Fig. 3** Plain X-ray film of the cervical spine and computed tomography of the elder sister of the dizugotic twins, *white arrows* show ossification of the posterior longitudinal ligament

The maximum spinal canal stenosis caused by the ossified ligament ranged from 35% to 68% with an average of 56.7%. Two of the six patients who had OPLL in the cervical spine exhibited myelopathy, but the remaining four did not.

## Discussion

Genetic background is thought to play a major role in OPLL, as shown by the results of Japan-wide studies of families and twins. This study is the first to report dizygotic twins with OPLL. The case of dizygotic twins presented here is one of schizophrenia associated with OPLL. Schizophrenia might be related to the pathogenesis of OPLL. Recently, calcineurin has been reported to be significantly associated with susceptibility to schizophrenia [10, 11], and it has also shown osteogenesis by the activation of osteoblasts [12, 13]. The association between OPLL and schizophrenia may not be coincidental. In our study, 20% of patients with schizophrenia exhibited OPLL, an incidence almost five times higher than among the general population in Japan (2.0 ~ 4.3%) [14]. The drugs used to treat schizophrenia are muscle relaxant, a feature that may induce malalignment of the spine. The biomechanical changes induced by the malalignment are also related to the development of OPLL. However, patients with OPLL in our series did not have apparent spinal deformities. Some diseases, such as vitamin-D-resistant rickets [15] or polycystic ovary syndrome [16], have been reported to be associated with OPLL. Schizophrenia might also be in the category.

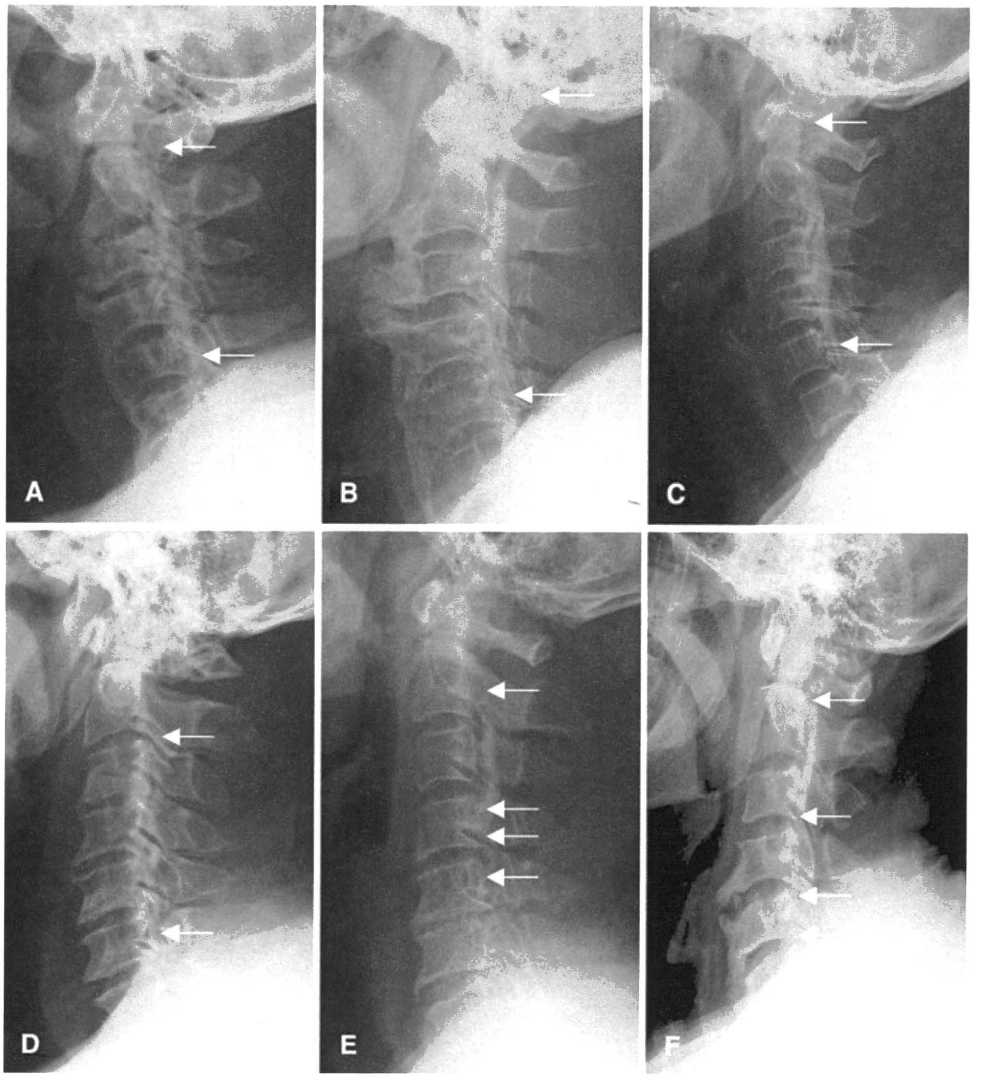


**Fig. 4** Ossification of the posterior longitudinal ligament (OPLL) of the six patients with schizophrenia. **a** A 78-year-old man, **b** a 75-year-old man, **c** a 59-year-old man, **d** a 79-year-old woman, **e** a 67-year-old man, and **f** a 51-year-old man. *White arrows* show the OPLL

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# Sagittal alignment changes after thoracic laminectomy in adults

**Toshimi Aizawa, M.D., Ph.D.,[1] Tetsuro Sato, M.D., Ph.D.,[2] Hiroshi Ozawa, M.D., Ph.D.,[1] Naoki Morozumi, M.D.,[3] Fujio Matsumoto, M.D., Ph.D.,[4] Hirotoshi Sasaki, M.D.,[2] Takeshi Hoshikawa, M.D., Ph.D.,[1] Chikashi Kawahara, M.D., Ph.D.,[1] Shoichi Kokubun, M.D., Ph.D.,[3] and Eiji Itoi, M.D., Ph.D.[1]**

*[1]Department of Orthopaedic Surgery, Tohoku University School of Medicine; [2]Department of Orthopaedic Surgery, Sendai Orthopaedic Hospital; [3]Department of Orthopaedic Surgery, Nishitaga National Hospital; and [4]Department of Orthopaedic Surgery, Tohoku Rosai Hospital, Sendai, Japan*

*Object.* The increased kyphosis after thoracic laminectomy in adult patients was retrospectively evaluated and various factors affecting this spinal deformity were analyzed.

*Methods.* The authors conducted a retrospective study of 58 cases in which laminectomy was performed and more than half of the facet joints were left intact. The study group included 44 men (mean age 59 years) and 14 women (mean age 61 years) with thoracic myelopathy due to ossifications of the ligamentum flavum and/or the posterior longitudinal ligament or due to posterior bone spurs. Patients were followed up for a minimum of 2 years. Their neurological condition was evaluated using the Japanese Orthopaedic Association (JOA) scale (a full score is 11), and the magnitude of local kyphosis in the laminectomized area was determined using the Cobb angle method.

*Results.* The mean preoperative JOA score was 5.4; the mean postoperative score was 8.3. No relationship was found between postoperative JOA score and increased kyphotic angle. The mean preoperative kyphotic angle was 7.0°. The mean postoperative kyphotic angle was 10.8°. Thus local kyphosis in the treated area increased by only 3.8°. The mean increase in kyphosis per spinal segment, calculated by dividing the kyphotic angle of the surgically decompressed area by the number of resected laminae, was 1.9°. Female patients with ≥ 3-level laminectomies showed a significant increase of kyphosis in both the laminectomized area and each spinal segment.

*Conclusions.* The increase in kyphosis after thoracic laminectomy is not large and thus spinal fusion is usually not necessary. In cases involving female patients who undergo long-segment laminectomies, however, careful radiographic follow-up is recommended. *(DOI: 10.3171/SPI/2008/8/6/510)*

**Key Words** • **kyphosis** • **laminectomy** • **thoracic spine**

Laminectomy is a basic procedure for decompression of the spinal cord. In addition to directly removing the compressive factors in the posterior spinal canal, it can indirectly decompress the cord by resulting in dorsal shift.[25] Thus, laminectomy has been performed for numerous cases of myelopathy in the cervical and thoracic spine.[1–4,9,11,15] Several postoperative complications have been reported after laminectomy, including cerebrospinal fluid leakage and epidural hematoma.[8] Laminectomy also entails the risk of instability in the form of kyphosis or kyphoscoliosis.[8,9,12,17,18,26–28]

Postlaminectomy kyphosis can cause neurological deterioration, localized pain, and postural dysfunction.[6,8,16,27,28] The incidence of this deformity is related to patient age, the number of laminae resected, and the anatomical level of the laminectomy; it usually occurs in children and adolescents after multilevel laminectomies in the cervical and thoracic spine.[7,19,20,23,28] Many reports emphasize the prevention of postlaminectomy kyphosis in children.[6,18–20,23,28] Less attention, however, has been given to this deformity in adults, as they rarely develop symptoms directly related to their deformity.[26] In the thoracic spine, the exact incidence or increase of this kyphosis is still unknown.[26]

In this study we retrospectively reviewed the postlaminectomy kyphosis in patients with thoracic myelopathy caused by OLF, OPLL, OLF and OPLL, or posterior bone

*Abbreviations used in this paper:* JOA = Japanese Orthopaedic Association; OLF = ossification of the ligamentum flavum; OPLL = ossification of the posterior longitudinal ligament.

spurs. We performed statistical analyses to determine the effect of various demographic and clinical variables.

## Clinical Materials and Methods

### *Patient Population*

The study population included 58 patients, 44 men (mean age 59 years) and 14 women (mean age 61 years), who underwent laminectomy because of thoracic myelopathy caused by degenerative conditions of the spine between 1988 and 2002 in the Department of Orthopaedic Surgery at Tohoku University Hospital and orthopedic departments affiliated with Tohoku University at other hospitals in Miyagi Prefecture, a province in northeastern Japan. The patients were followed up for a minimum of 2 years. Patients who underwent multilevel laminectomies for decompression of adjacent segments with no intervening untreated segments were included, but those who underwent multilevel laminectomies involving nonadjacent segments were excluded because measuring the degree of kyphosis in this latter group would involve untreated spinal levels. Patients who had spinal cord tumors were also excluded as they were occasionally treated by means of laminectomy with facetectomy and subsequent instrumented spinal fusion.

This study was approved by the ethics committee of Tohoku University School of Medicine, and all patients were informed that the data from their cases would be used for the study.

### *Surgical Procedures*

The surgeries were performed by the highly experienced spine surgeons in Miyagi Prefecture.[2–4] Both en bloc (Fig. 1 *left*) and French-door (Fig. 1 *right*) laminectomies were performed in the following steps.[1,21,24] First, the outer cortex and the cancellous layer of the involved laminae were removed with the aid of an air drill. The lateral ends of the incision were placed at the medial margin of the pedicle for OLF and inside it for OPLL and posterior bone spurs. Thus, more than half of the facet joints were kept intact (Fig. 2). Transverse incisions were made just at the cranial or caudal side of the chevronlike portion of the ligamentum flavum. Then the laminae were pulled up and removed for en bloc laminectomy. For French-door laminectomy, a longitudinal incision was made at the midline portion of laminae and the laminae were opened bilaterally and removed.

### *Clinical Evaluation*

The pre- and postoperative neurological condition of each patient was evaluated using the modified JOA scoring system, an 11-point scale measuring lower-extremity motor function, lower-extremity and trunk sensory function, and bladder function.[10] The percentage recovery, calculated as follows, was also used for the postoperative evaluation: [postoperative JOA score − preoperative JOA score] / [11 − preoperative JOA score] × 100.[10]

### *Evaluation of Sagittal Alignment*

Lateral radiographs obtained with the patients in the neutral position were evaluated preoperatively and at the final follow-up examination. The distribution and number of resected laminae were first investigated. Then the pre- and postoperative magnitude of local kyphosis at the surgically decompressed area was determined using the Cobb angle method, since the deformity is usually seen in the area of the laminectomy (Fig. 3).[20] In addition, a "normalized" value for segmental kyphosis was calculated by dividing the measured value for the entire decompressed area by the number of laminae resected.

### *Data Analysis*

We determined the relationship between the local kyphosis of the laminectomized area, the normalized segmental

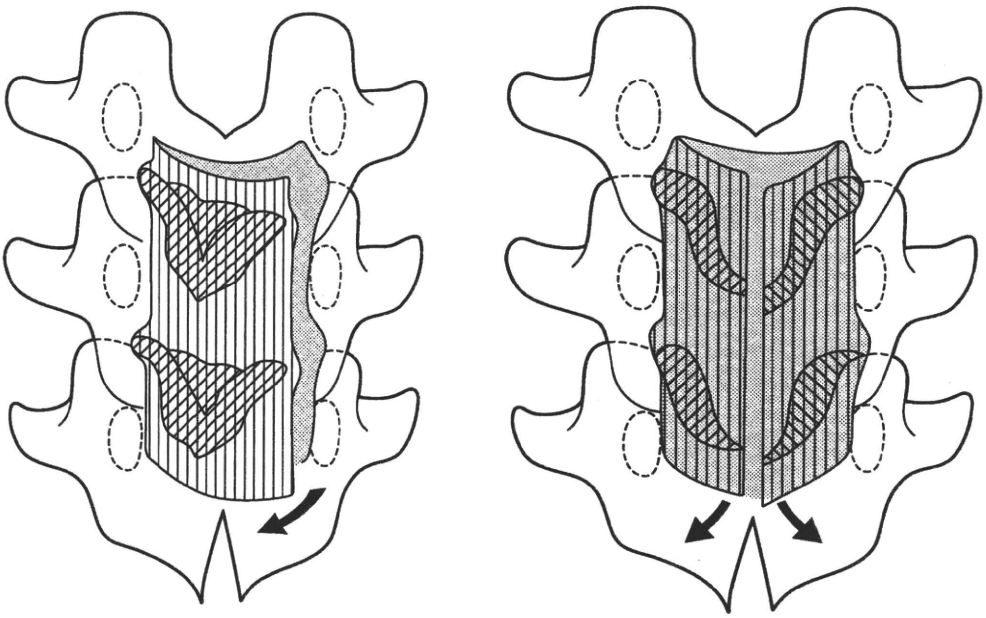

FIG. 1. Diagram illustrating the surgical procedure of en bloc *(left)* and French-door *(right)* laminectomies used in the treatment of OLF in this case series (see *Surgical Procedures*).

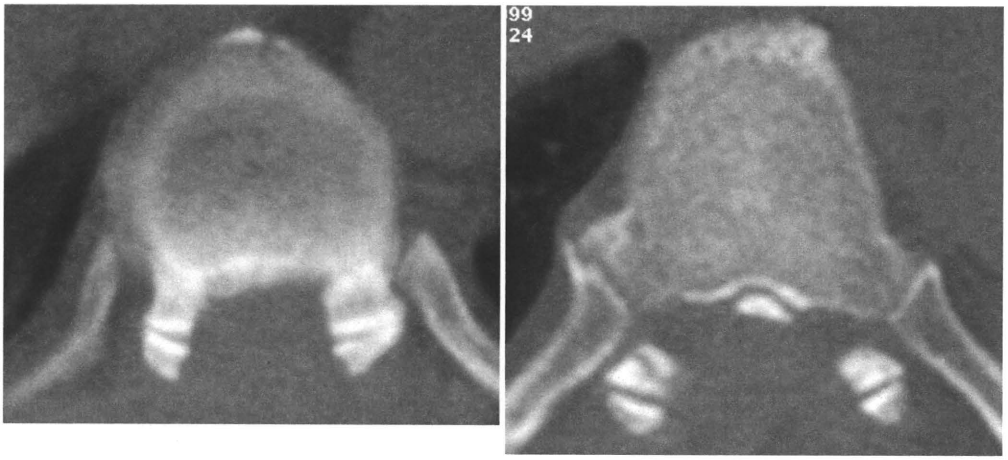

FIG. 2. Postlaminectomy computed tomography scans obtained in a patient with OLF at T10–11 *(left)* and a patient with OPLL at T7–8 *(right)*. In both cases, more than half of the facet joints were left intact, although in the patient with OLF *(left)*, the right facet joint was more widely resected because ossification was located predominantly on the right side.

kyphosis, and various factors, including the compressive factors for the spinal cord, and the age and sex of the patients.

The data were analyzed with the patient population stratified 3 different ways—on the basis of the distribution of the decompressed laminae (that is, the thoracic levels treated), the number of laminae resected, and the postoperative increase of the kyphotic deformity. For the surgical spinal distribution, the patients were classified as follows: 1) upper thoracic, the laminectomy was performed at T1–4; 2) middle thoracic, at T5–8; and 3) lower thoracic, at T9–12. If the decompressed laminae involved 2 of those groups, the patient was categorized based on the location of the majority of the resected laminae located. With respect to the number of laminae resected, patients were grouped as follows: 1) 1-level laminectomy, 2) 2-level laminectomy, and 3) $\geq$ 3-level laminectomy. For the kyphotic deformity, the patients were categorized into: 1) kyphotic increase of $\leq 2°$, 2) kyphotic increase of 3–6°, and 3) kyphotic increase of $\geq 7°$.

For statistical purposes, the data were analyzed using an analysis of variance, a chi-square test, or the Welch t-test. A probability value of $< 0.05$ was considered significant.

## Results

### *Clinical Evaluation*

The mean patient age at surgery was 59 years in men (range 29–84 years) and 61 in women (range 30–77 years). The compressive factors for the spinal cord were OLF in 37 patients, OPLL in 14, OLF and OPLL in 5, and posterior bone spurs in 2. The pre- and postoperative mean JOA scores for men and women were 5.4 and 8.3, respectively, and the mean recovery rate was 49% at a mean follow-up of 54 months (range 2–14 years). Most of the patients showed an increase in the JOA score, with only 4 showing a decline at the last follow-up; 3 patients showed just a 1-point decrease because they began to use canes for walking on a level or on stairs for their safety, and 1 had a 2-point decrease because of lumbar spinal canal stenosis.

The relationships between the pre- and postoperative neurological status and the various factors analyzed are summarized in Table 1. No statistically significant relationship was found between preoperative JOA score and age, but there was a significant relationship between preoperative JOA score and patient sex. In contrast, the postoperative JOA score was significantly related to patient age but not sex; younger patients showed significantly better postoperative improvement. The surgical outcomes showed no relationship with the compressive factors for the spinal cord, the distribution or number of decompressed laminae, or even postoperative increase in kyphotic angle. In the 4 patients who had worsened JOA scores postoperatively, the mean increase in kyphosis was 3.8°. None of the patients underwent additional surgery because of the spinal deformity.

### *Sagittal Alignment*

The mean number of resected laminae was 2.3 overall (range 1–7), 2.1 in men, and 3.1 in women. The mean values for pre- and postoperative kyphosis of the involved laminae were 7.0° (range −12 to 28°) and 10.8° (range −6 to 41°), respectively; the mean increase in kyphosis was only 3.8° (range −4 to 19°). Compared with the normal thoracic kyphosis described by Bernhardt and Bridwell,[5] 31 patients had a greater degree of kyphosis in the laminectomized area preoperatively. Two patients had a lordotic curve in the upper thoracic spine and 2 in the lower thoracic spine preoperatively; one had 12° of lordosis in 4 vertebral segments, but others had just 1 or 2° in 1 segment. The patient with 12° of local lordosis still had a local lordotic curve of 6° postoperatively. The relationships between pre- and postoperative kyphosis and various demographic and clinical factors are summarized in Table 2. There was no significant relationship between preoperative local kyphosis and age, sex, or the compressive factors for the spinal cord, but the preoperative local kyphosis was significantly greater in cases involving the middle thoracic vertebrae. There was a statistically significant relationship between preoperative segmental kyphosis and patient age as well as

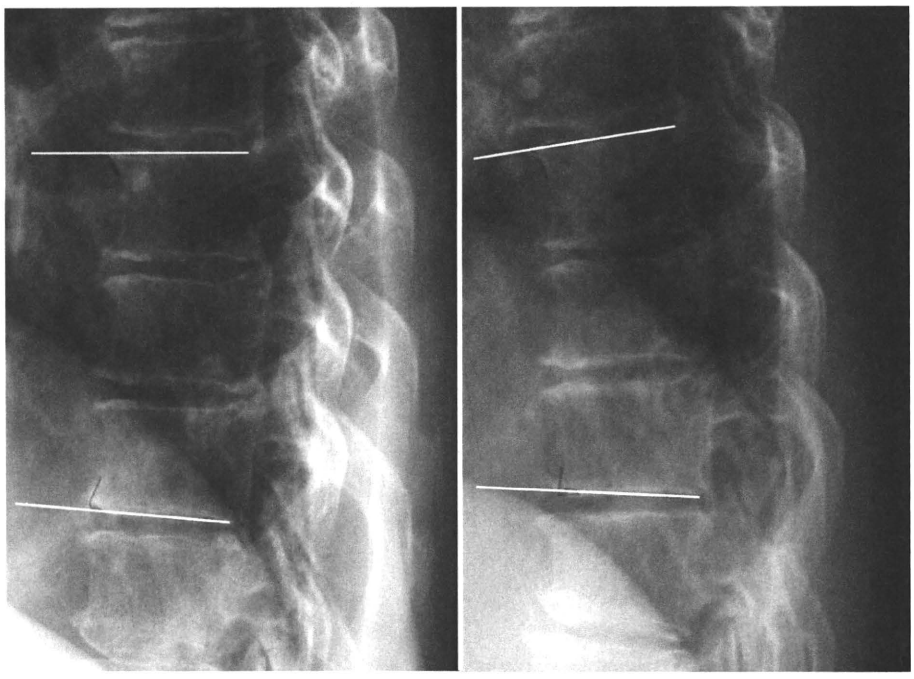

FIG. 3. Preoperative *(left)* and postoperative *(right)* lateral radiographs illustrating the measurement of sagittal alignment of the decompressed laminae at the T8–10 level. The magnitude of local kyphosis at the resected laminae was determined using the Cobb angle method. The local kyphosis was 4° in this patient preoperatively and increased to 14° postoperatively.

the distribution of the decompressed laminae, but not between preoperative segmental kyphosis and patient sex or compressive spinal factors.

No compression fractures were detected in the involved vertebrae before or after surgery. Basically, the patients with greater preoperative local kyphosis showed greater postoperative kyphosis, but the segmental increase was not affected by the preoperative curve. The overall mean segmental increase was 1.9°, while the mean segmental increase was 1.6° in patients who showed a greater preoperative kyphosis than the normal values and 2.2° in those who had preoperative kyphosis that was less than the normal values. Postoperative kyphosis and increase in kyphosis were related to the sex of the patients and the distribution of the decompressed laminae: female sex and laminectomy in the middle thoracic spine were associated with significantly severer kyphotic spinal alignment. The patients with OPLL showed a significantly greater increase in the local kyphosis of the decompressed area than those with OLF.

The postoperative kyphosis and increase in kyphosis were in proportion to the number of resected laminae; this relationship was more obvious in the female patients. The 7 female patients with laminectomy at $\geq$ 3 levels had a mean postoperative kyphosis of 24.1°, a mean increase in kyphosis of 12.1°, and a mean segmental increase of 2.5°, and the 12 male patients who had laminectomy at $\geq$ 3 levels had corresponding mean values of 11.3, 2.5, and 0.6°, respectively ($p < 0.05$ for all 3 comparisons). In women who underwent laminectomy at > 4 levels, the increase in kyphosis was 13.2° (segmental increase 2.8°), and in those who underwent laminectomy at $\geq$ 5 levels, it was 15.3° (segmental increase 2.5°), whereas in men the corresponding mean values were 3° (segmental 1.0°) for $\geq$ 4-level laminectomy and 5° (segmental 1.1°) in $\geq$ 5-level laminectomy.

## Discussion

Spinal column deformities including kyphosis after laminectomy usually occur in children and young adults.[18–20,23,28] The overall reported incidence of postlaminectomy kyphosis has varied greatly, between 6 and 52%. Generally, it has been believed that thoracic laminectomy should not cause severe instability and deformity, but there have been no reports on the exact incidence or increase of kyphosis focusing on the thoracic spine in adult patients.[11,15,28]

Postlaminectomy kyphosis might be related to the combined effects of several factors: maintenance of the flexed position (and the effect of gravity on preexisting kyphosis), the loss of posterior ligaments (including the supraspinous and intraspinous ligaments), and muscle weakness, in addition to the width of laminectomy and facetectomy.[19,22,23,26] The risk of this deformity might be increased in the immature spines of children. Facet joints provide spinal stability, particularly in flexion and rotation, and resection of > 50% of the facet joints at any given level is associated with significant spinal instability.[19,22,26] In this study, the overall mean increase in kyphosis was only 3.8°, and no patient had additional surgery because of spinal deformity.

Our surgical procedure of laminectomy for OLF, OPLL, and posterior spurs avoided total facetectomy. The largest resection of the facet joints was performed in the patients with OLF. Even in these cases, the lateral ends of the lam-

TABLE 1

*Relationship between pre- and postoperative neurological status and various demographic and clinical factors**

| Factors | No. of Patients | JOA Score | | |
|---|---|---|---|---|
| | | Preop | Postop | Recovery Rate (%) |
| age in yrs | | | | |
| ≤60 | 27 | 5.4 ± 2.3 | 9.2 ± 1.4† | 61 ± 32† |
| >60 | 31 | 5.3 ± 2.1 | 7.5 ± 2.1† | 38 ± 31† |
| sex | | | | |
| M | 44 | 5.7 ± 3.2† | 8.4 ± 3.0 | 48 ± 31 |
| F | 14 | 4.3 ± 2.1† | 7.9 ± 2.2 | 53 ± 29 |
| compressive entity | | | | |
| OLF | 37 | 5.1 ± 2.1 | 8.3 ± 1.8 | 52 ± 30 |
| OPLL | 14 | 5.8 ± 2.4 | 7.9 ± 2.4 | 40 ± 36 |
| OLF & OPLL | 5 | 6.2 ± 3.1 | 9.6 ± 1.7 | 61 ± 44 |
| posterior spur | 2 | 5.5 ± 2.1 | 8.5 ± 2.1 | 43 ± 61 |
| distribution of decompressed laminae | | | | |
| upper (T1–4) | 23 | 5.0 ± 2.2 | 8.2 ± 2.2 | 47 ± 39 |
| middle (T5–8) | 5 | 5.2 ± 1.8 | 8.0 ± 2.2 | 45 ± 37 |
| lower (T9–12) | 30 | 5.6 ± 2.3 | 8.4 ± 1.8 | 52 ± 28 |
| no. of decompressed laminae | | | | |
| 1 | 22 | 5.9 ± 2.4 | 8.4 ± 2.1 | 48 ± 32 |
| 2 | 17 | 5.6 ± 2.1 | 7.9 ± 2.3 | 42 ± 38 |
| ≥3 | 19 | 4.5 ± 1.9 | 8.5 ± 1.5 | 57 ± 31 |
| increase in kyphosis (°) | | | | |
| ≤2 | 28 | 5.5 ± 2.3 | 8.4 ± 2.0 | 48 ± 36 |
| 3–6 | 15 | 5.4 ± 2.1 | 8.1 ± 2.2 | 47 ± 37 |
| ≥7 | 15 | 5.1 ± 2.3 | 8.3 ± 1.8 | 54 ± 24 |

* Data are presented as means ± standard deviations unless otherwise indicated.

† There were statistically significant differences ($p < 0.05$) between the 2 age groups with respect to postoperative JOA score and recovery rate and between the preoperative JOA scores in the male and female patient groups.

inar incision were placed at the medial margin of the pedicles so that more than half of the lateral part of the facet joints was kept intact.[1,21,24] In addition, the thoracic spine is strongly supported by the rib cage, which, in combination with our avoiding total facetectomy might prevent substantial postoperative increase in kyphosis. In the present study, 97% of the patients suffered from thoracic myelopathy caused by the ossification of the spinal ligaments. Patients with OLF and OPLL occasionally have ossification of the anterior longitudinal ligaments, which might also affect the stability of the involved spinal levels.

The apex of the thoracic kyphosis is normally located between T-5 and T-8, in the middle thoracic spine.[5] This should play a role in our finding that the middle spine showed significantly larger postlaminectomy kyphosis at the involved area although the number of the patients having laminectomy in the middle thoracic spine in the present study was small. The kyphotic angle of the lower thoracic spine is greater than that in the upper spine.[5] Moreover, the lower 5 ribs do not directly articulate with the sternum, and thus the lower part of the rib cage should be weaker than the upper part. These factors might contribute to our finding that the segmental increase in kyphosis was significantly larger in the lower thoracic spine than in the upper spine.

The present study clearly showed that postoperative kyphosis and increase in kyphosis were related to the number of the laminae removed. With respect to the segmental increase in kyphosis, however, a significant relationship with the number of the resected laminae was found only in female patients (mean 2.5° in women ≥ 3-level laminectomy vs 0.6° in men). This disparity might result from osteoporotic wedging deformity in the vertebral bodies in women, although no compression fractures were detected in the involved vertebra and the osteoporotic condition of the patients, such as bone mineral density, was not investigated in this study.[7,13,14] The longer defect of the posterior spinal ligaments and muscle weakness might lead to larger spinal deformities.[22,23] Women show more decrease in the tone of the spinal ligaments and muscles caused by poor posture, aging, and relative physical inactivity than men.[7] These might also affect the relatively larger increase in postlaminectomy kyphosis in women.

This is a retrospective study and several limitations should be noted. First, the laminectomy procedures were not completely the same in all our patients. The resection of the facet joint was somewhat different in those with OLF and those without OLF. Secondly, we only evaluated the local kyphosis of the laminectomized area, not the whole spine or even the whole thoracic spine. Therefore, the effect of the increased kyphosis on the sagittal alignment of the whole spine is uncertain. Additionally, the Cobb angle method that we used might be associated with inherent measurement errors since it reflects changes in the end vertebrae rather than changes within the curves themselves.[5]

## Conclusions

The increase in kyphosis after laminectomy for thoracic myelopathy caused by degenerative conditions of the spine was not so large when a greater part of the facet joints were

TABLE 2

*Relationship between pre- and postoperative kyphosis and various demographic and clinical factors**

| Factors | No. of Patients | No. of Decompressed Laminae | Local (segmental) Kyphosis (°) | | |
|---|---|---|---|---|---|
| | | | Preop | Postop | Increase |
| age | | | | | |
| ≤60 | 27 | 2.6 ± 1.5 | 7.0 ± 7.6 (2.4 ± 2.2[a]) | 10.7 ± 9.9 (4.5 ± 3.0) | 3.7 ± 4.9 (2.1 ± 2.2) |
| >60 | 31 | 2.1 ± 1.4 | 7.4 ± 5.5 (4.4 ± 3.9[a]) | 10.9 ± 8.8 (6.1 ± 5.2) | 3.5 ± 5.0 (1.9 ± 2.4) |
| sex | | | | | |
| M | 44 | 2.1 ± 1.3 | 6.6 ± 5.5 (3.7 ± 3.7) | 9.3 ± 7.2[b] (5.6 ± 4.8) | 2.7 ± 6.7[c] (1.9 ± 2.5) |
| F | 14 | 3.1 ± 1.9 | 8.2 ± 6.9 (2.7 ± 2.1) | 15.6 ± 12.4[b] (4.6 ± 2.6) | 7.4 ± 6.3[c] (1.9 ± 1.7) |
| compressive entity | | | | | |
| OLF | 37 | 1.9 ± 1.1 | 6.1 ± 3.9 (4.0 ± 3.8) | 8.9 ± 5.2[d] (6.0 ± 5.0) | 2.9 ± 3.9[e] (2.0 ± 2.7) |
| OPLL | 14 | 3.1 ± 1.9 | 9.5 ± 8.6 (3.0 ± 1.8) | 15.6 ± 12.1[d] (4.9 ± 2.1) | 6.1 ± 5.7[e] (2.0 ± 1.6) |
| OLF & OPLL | 5 | 3.6 ± 1.7 | 12.4 ± 8.4 (3.3 ± 1.5) | 17.0 ± 15.8 (4.2 ± 2.4) | 4.6 ± 8.2 (1.0 ± 1.5) |
| posterior spur | 2 | 2.5 ± 2.1 | −6.5 ± 7.8 (−2.0 ± 1.4) | −3.0 ± 4.2(−0.8 ± 1.1) | 3.5 ± 3.5 (1.3 ± 0.4) |
| distribution of decompressed laminae | | | | | |
| upper (T1–4) | 23 | 2.8 ± 1.8 | 6.5 ± 7.8[f] (2.4 ± 2.3[g,h]) | 10.7 ± 12.0[i] (3.7 ± 2.5[j]) | 4.2 ± 5.9 (1.3 ± 1.5[k]) |
| middle (T5–8) | 5 | 3.8 ± 1.1 | 15.4 ± 7.9[f,l] (4.0 ± 1.1[g]) | 20.2 ± 10.5[i,m] (5.1 ± 1.8) | 4.8 ± 5.7 (1.1 ± 1.5) |
| lower (T9–12) | 30 | 1.8 ± 1.1 | 6.0 ± 4.2[l] (4.3 ± 4.1[h]) | 9.4 ± 5.2[m] (6.7 ± 5.3[j]) | 3.4 ± 3.5 (2.4 ± 2.8[k]) |
| no. of decompressed laminae | | | | | |
| 1 | 22 | 1 | 4.8 ± 4.8[n] (4.8 ± 4.8[o]) | 7.6 ± 5.9[p,q] (7.6 ± 5.9[r,s]) | 2.9 ± 3.0[t] (2.9 ± 3.0) |
| 2 | 17 | 2 | 6.5 ± 2.9 (3.3 ± 1.5) | 9.1 ± 3.5[q] (4.5 ± 1.8[r]) | 2.6 ± 3.5[u] (1.3 ± 1.8) |
| ≥3 | 19 | 4.2 ± 1.1 | 10.0 ± 9.4[n] (2.2 ± 1.9[o]) | 16.1 ± 13.3[p,q] (3.5 ± 2.5[s]) | 6.1 ± 6.4[t,u] (1.3 ± 1.3) |
| no. of decompressed laminae | | | | | |
| men | | | | | |
| 1 lamina | 19 | 1 | 4.9 ± 4.9 (4.9 ± 4.9) | 8.3 ± 5.8 (8.3 ± 5.8) | 3.4 ± 2.8 (3.4 ± 2.8[v]) |
| 2 laminae | 13 | 2 | 7.0 ± 2.7 (3.5 ± 1.4) | 8.8 ± 3.4 (4.4 ± 1.7) | 1.9 ± 3.6 (0.9 ± 1.8[v]) |
| ≥3 laminae | 12 | 3.9 ± 0.8 | 8.8 ± 10.4 (2.1 ± 2.2) | 11.3 ± 12.4[w] (2.7 ± 2.6[x]) | 2.5 ± 4.4[y] (0.6 ± 1.1[v,z]) |
| women | | | | | |
| 1 lamina | 3 | 1 | 3.7 ± 4.0 (3.7 ± 4.0) | 3.3 ± 4.9 (3.3 ± 4.9) | −0.3 ± 2.3 (−0.3 ± 2.3) |
| 2 laminae | 4 | 2 | 5.0 ± 3.6 (2.5 ± 1.8) | 9.8 ± 4.6 (4.9 ± 2.3) | 4.8 ± 2.5 (2.4 ± 1.3) |
| ≥3 laminae | 7 | 4.7 ± 1.4 | 12.0 ± 7.6 (2.4 ± 1.3) | 24.1 ± 11.4[w] (4.9 ± 1.6[x]) | 12.1 ± 4.6[y] (2.5 ± 0.5[z]) |

* Data are presented as means ± standard deviations unless otherwise indicated. Superscripted letters indicate sets of values for which significant differences were found ($p < 0.05$). The use of 2 superscripted letters separated by a comma (for example: [g,h]) indicates that the designated value was involved in 2 comparisons that yielded statistically significant differences.

left intact. Thus, spinal fusion should usually not be necessary in order to avoid further spinal deformity. Nevertheless, in female patients who require long-segment laminectomies, careful radiographic follow-up should be performed to monitor for increasing kyphosis.

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*Address correspondence to:* Toshimi Aizawa, M.D., Ph.D., Department of Orthopaedic Surgery, Tohoku University School of Medicine, 1-1 Seiryo-machi, Aoba-ku, Sendai, 980-8574, Japan. email: toshi-7@ra2.so-net.ne.jp.

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*Original article*

# Japanese Orthopaedic Association Back Pain Evaluation Questionnaire. Part 3. Validity study and establishment of the measurement scale

## Subcommittee on Low Back Pain and Cervical Myelopathy Evaluation of the Clinical Outcome Committee of the Japanese Orthopaedic Association, Japan

MITSURU FUKUI[1], KAZUHIRO CHIBA[2], MAMORU KAWAKAMI[3], SHIN-ICHI KIKUCHI[4], SHIN-ICHI KONNO[4], MASABUMI MIYAMOTO[5], ATSUSHI SEICHI[6], TADASHI SHIMAMURA[7], OSAMU SHIRADO[8], TOSHIHIKO TAGUCHI[9], KAZUHISA TAKAHASHI[10], KATSUSHI TAKESHITA[6], TOSHIKAZU TANI[11], YOSHIAKI TOYAMA[3], EIJI WADA[12], KAZUO YONENOBU[13], TAKASHI TANAKA[14], and YOSHIO HIROTA[15]

[1] Laboratory of Statistics, Osaka City University Faculty of Medicine, Osaka, Japan
[2] Department of Orthopaedic Surgery, Keio University, Tokyo, Japan
[3] Department of Orthopaedic Surgery, Wakayama Medical University, Wakayama, Japan
[4] Department of Orthopaedic Surgery, School of Medicine, Fukushima Medical University, Fukushima, Japan
[5] Department of Orthopaedic Surgery, Nippon Medical School, 1-1-5 Sendagi, Bunkyo-ku, Tokyo 113-8603, Japan
[6] Department of Orthopaedic Surgery, The University of Tokyo, Tokyo, Japan
[7] Department of Orthopaedic Surgery, Iwate Medical University School of Medicine, Morioka, Japan
[8] Department of Orthopaedic Surgery, Saitama Medical School, Iruma-gun, Japan
[9] Department of Orthopaedic Surgery, Yamaguchi University School of Medicine, Yamaguchi, Japan
[10] Department of Orthopaedic Surgery, Graduate School of Medicine, Chiba University, Chiba, Japan
[11] Department of Orthopaedic Surgery, Kochi Medical School, Kochi, Japan
[12] Department of Orthopaedic Surgery, Hoshigaoka Koseinenkin Hospital, Osaka, Japan
[13] National Hospital Organization Osaka-Minami Medical Center, Kawachinagano, Japan
[14] Department of Internal Medicine, Houai Hospital, Osaka, Japan
[15] Department of Public Health, Osaka City University Faculty of Medicine, Osaka, Japan

**Abstract**

***Background.*** The Japanese Orthopaedic Association decided to revise the JOA score for low back pain and to develop a new outcome measure. In February 2002, the first survey was performed with a preliminary questionnaire consisting of 60 evaluation items. Based on findings of that survey, 25 items were selected for a draft of the JOA Back Pain Evaluation Questionnaire (JOABPEQ). The second survey was performed to confirm the reliability of the draft questionnaire. This article further evaluates the validity of this questionnaire and establishes a measurement scale.

***Methods.*** The subjects of this study consisted of 355 patients with low back disorders of any type (201 men, 154 women; mean age 50.7 years). Each patient was asked to fill in a self-administered questionnaire. Superficial validity was checked in terms of the completion rate for filling out the entire questionnaire. Factor analysis was then performed to evaluate the validity of the questionnaire and establish a measurement scale.

***Results.*** As a result of the factor analysis, 25 items were categorized into five factors. The factors were named based on the commonality of the items: social function, mental health, lumbar function, walking ability, and low back pain. To establish a measurement scale for each factor, we determined the coefficient for each item so the difference between the maximum factor scores and minimum factor scores was approximately 100. We adjusted the formula so the maximum for each factor score was 100 and the minimum was 0.

***Conclusions.*** We confirmed the validity of the JOA Back Pain Evaluation Questionnaire and est abished a measurement scale.

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## Introduction

The evaluation criteria were based on physiological, biological, and anatomical outcome measure results of the Japanese Orthopaedic Association (JOA) score for low back pain.[1] The criteria include laboratory values, physiological findings, and imaging findings. These findings are significant for doctors but have little meaning for patients. From a patient's perspective, the presence of a symptom or its degree and functional condition

must have real meaning. This means that outcome measures need to be translatable from an objective evaluation to a subjective one, or from the doctor's perspective to the patient's perspective. The JOA decided to revise the JOA score for low back pain and develop a new scientific, patient-oriented outcome measure.

The first committee meeting was held in June 2000, and the first survey was initiated in February 2002 using a preliminary questionnaire consisting of 60 items. It was a self-administered, disease-specific measure developed with reference to the Japanese editions of SF-36[2,3] and the Roland-Morris Disability Questionnaire (RDQ)[4,5] to assess health-related quality of life. Based on findings of the survey, 25 items were selected for a draft of the JOA Back Pain Evaluation Questionnaire (JOABPEQ) (see Appendix 1).

The second survey was started in January 2004 to evaluate the reliability of the 25 items selected for the draft JOABPEQ. We successfully confirmed the reliability, and these details have been described in previous reports of Part 1[6] and Part 2.[7] Part 3 of this study involves further development of the new JOA questionnaire, evaluation of the validity of the draft JOABPEQ, and establishment of a measurement scale.

## Materials and methods

### *Recruitment of patients*

A total of 369 of the 829 Japanese board-certified spine surgeons were randomly selected and asked to recruit at least three patients each to participate in evaluating the JOABPEQ during February 2004. The inclusion criterion was any type of lumbar spine disorder. Exclusion criteria were patients who had:

- Other musculoskeletal diseases requiring medical treatment
- Psychiatric disease, potentially leading to inappropriate answers
- Postoperative condition
- Participation in previous surveys related to this study

### *Testing the questionnaire*

Each patient was asked to fill in the self-administered questionnaire. The attending surgeon filled out information on the diagnosis, presence or absence of concomitant diseases, and a judgment regarding the severity of symptoms using a three-step rating scale (mild, moderate, severe). The severity of the symptoms was determined subjectively by the attending surgeon, who was asked not to select a similar patient based only on the severity. This study was approved by the Ethics Committee of the Japanese Society for Spine Surgery and Related Research, and informed consent was obtained from each patient.

Factor analysis was used to check the statistical validity of the questionnaire and establish the measurement scale. All statistics were calculated using SPSS software (version 12; SPSS, Chicago, IL, USA).

## Results

### *Patient characteristics*

Of the 452 patients selected for participation in this survey, 1 patient who was judged inappropriate by the attending doctor and 60 patients with other musculoskeletal diseases requiring medical treatment were excluded. The responses from 36 patients who answered incompletely were also excluded, leaving 355 patients available for analysis: 201 men and 154 women, with a mean ± SD age of 50.7 ± 18.0 (Table 1). The diagnosis was lumbar disc herniation in 167, lumbar spinal canal stenosis in 103, and spondylolisthesis in 37.

According to the judgment of the attending doctor, there were 115 mild, 142 moderate, and 98 severe cases. Table 2 summarizes the severity of low back pain evaluated by the current JOA scoring system and shows that the characteristics of the recruited patients were not

**Table 1.** Distribution of age and severity of symptoms ($n = 355$)

| | Severity of symptoms | | | |
|---|---|---|---|---|
| Age (years) | Mild | Moderate | Severe | Total |
| Males | | | | |
| 10–19 | 3 | 4 | | 7 |
| 20–29 | 8 | 4 | 9 | 21 |
| 30–39 | 12 | 14 | 6 | 32 |
| 40–49 | 12 | 14 | 8 | 34 |
| 50–59 | 12 | 9 | 11 | 32 |
| 60–69 | 10 | 12 | 12 | 34 |
| 70–79 | 11 | 16 | 8 | 35 |
| 80+ | 4 | 2 | | 6 |
| Total | 72 | 75 | 54 | 201 |
| Females | | | | |
| 10–19 | 0 | 1 | 3 | 4 |
| 20–29 | 9 | 5 | 5 | 19 |
| 30–39 | 7 | 13 | 10 | 30 |
| 40–49 | 5 | 14 | 7 | 26 |
| 50–59 | 4 | 9 | 5 | 18 |
| 60–69 | 7 | 12 | 7 | 26 |
| 70–79 | 11 | 10 | 4 | 25 |
| 80+ | | 3 | 3 | 6 |
| Total | 43 | 67 | 44 | 154 |
| Total no. | 115 | 142 | 98 | 355 |

**Table 2.** Distribution of the severity evaluated by the current JOA scoring system and finger-floor distance ($n = 355$)

| Parameter | No. |
|---|---|
| Straight-leg raising (SLR) test | |
| Normal | 183 |
| 30°–70° | 130 |
| <30° | 42 |
| Motor function | |
| Normal | 182 |
| Slight weakness (MMT good) | 126 |
| Severe weakness (MMT less than good) | 47 |
| Sensory function | |
| Normal | 127 |
| Slight disturbance | 162 |
| Severe disturbance | 66 |
| Bladder function | |
| Normal | 315 |
| Mild dysuria | 36 |
| Severe dysuria | 4 |
| Finger-to-floor distance (cm) | |
| to −15 | 1 |
| −14 to −5 | 12 |
| −4 to 4 | 69 |
| 5 to 14 | 73 |
| 15 to 24 | 69 |
| 25 to 34 | 43 |
| 35 to 44 | 25 |
| 45 to 54 | 30 |
| 55 to 64 | 6 |
| 65 to 74 | 4 |
| Not measurable | 14 |
| Total number | 355 |

JOA, Japanese Orthopaedic Association; MMT, manual muscle testing

specific. There was no marked difference in the distribution of the severity levels between the 451 patients who were initially recruited and the 355 who were finally analyzed.

*Superficial validity*

Superficial validity was checked in terms of the completion rate for filling out the questionnaire. Regarding the distribution of responses for each item, it was judged that none of the questions was too difficult to answer because the highest rate of nonrespose was 1.8%. With regard to deflection of an answer, the highest rate (78.3%) was concentrated on "yes" responses to question 1–14, although this was judged not to be inappropriate. Therefore, the distribution was not skewed, which would indicate "floor and ceiling" effects (Table 3).

*Factor analysis*

First, we tried to extract some observed variables from 25 items by the Maximum Likelihood Method. It was found that the eigenvalue was >1.0 for five items, and the accumulative contribution ratio until the fifth factor was 53.1% (Table 4).

Next, we performed orthogonal rotation by the direct oblimin method. The results are shown in Table 5. Each item was categorized into five factors: Four items (Q2-6, Q2-5, Q1-2, Q2-4) related to factor 1; seven items (Q2-8, Q2-7, Q2-11, Q1-13, Q2-9, Q2-10, Q2-1) related to factor 2; six items (Q1-9, Q1-6, Q2-3, Q1-8, Q1-5, Q1-4) related to factor 3; five items (Q1-10, Q2-4, Q1-12, Q1-14, Q2-2) to factor 4; and the last four items to factor 5. Although factor loading was <0.30 in three items (Q1-4 to factor 3, Q2-2 to factor 4, Q1-11 to factor 5), we adopted all of them for the reason that the question itself was important for the factor or the number of questions in each factor needed to be more than four.

Factor names were determined based on the commonality of the items that showed a large value on factor loading: factor 1, social function (four items); factor 2, mental health (seven items); factor 3, lumbar function (six items); factor 4, walking ability (five items); and factor 5, low back pain (four items).

*Measurement scale*

To establish a measurement scale for each factor, we determined the size of the coefficient for each item so the difference between the maximum factor scores and minimum factor scores was approximately 100 (Table 6). When a coefficient became a negative numerical value, we changed the coefficient to a positive numerical value by reversing the order of the answer choice. We adjusted the formula so the maximum for each factor score was 100 and the minimum was 0 (see Appendix 2).

## Discussion

It is considered ideal for the outcome measure to evaluate patients from various perspectives, such as dysfunction, disability, handicap, and psychological problem. The outcome measure should be patient-oriented, and its reliability and validity should be confirmed by statistical analysis. However, the current JOA score does not include subjective evaluations and does not meet such requirements. We developed a new questionnaire, JOABPEQ, specifically to evaluate low back pain. It is patient-oriented and mainly based on recognizing problems with activities of daily living. We categorized 25 questions into five factors; each factor is then scored up to 100 points using the measurement scale. The factors are then evaluated separately. The point is to be aware that it is meaningless and inadequate to total

**Table 3.** Distribution of answers for each item in the questionnaire ($n = 451$)

| Item | Choices for answer | | | | | No answer |
|---|---|---|---|---|---|---|
| | 1 | 2 | 3 | 4 | 5 | |
| Q1-1 | 336 (74.5%) | 114 (25.3%) | | | | 1 (0.2%) |
| Q1-2 | 152 (33.7%) | 297 (65.9%) | | | | 2 (0.4%) |
| Q1-3 | 302 (67.0%) | 146 (32.4%) | | | | 3 (0.7%) |
| Q1-4 | 157 (34.8%) | 291 (64.5%) | | | | 3 (0.7%) |
| Q1-5 | 242 (53.7%) | 209 (46.3%) | | | | 0 |
| Q1-6 | 167 (37.0%) | 281 (62.3%) | | | | 3 (0.7%) |
| Q1-7 | 215 (47.7%) | 236 (52.3%) | | | | 0 |
| Q1-8 | 240 (53.2%) | 208 (46.1%) | | | | 3 (0.7%) |
| Q1-9 | 272 (60.3%) | 177 (39.2%) | | | | 2 (0.4%) |
| Q1-10 | 288 (63.9%) | 160 (35.5%) | | | | 3 (0.7%) |
| Q1-11 | 158 (35.0%) | 292 (64.7%) | | | | 1 (0.2%) |
| Q1-12 | 156 (34.6%) | 286 (63.4%) | | | | 9 (2.0%) |
| Q1-13 | 116 (25.7%) | 333 (73.8%) | | | | 2 (0.4%) |
| Q1-14 | 353 (78.3%) | 90 (20.0%) | | | | 8 (1.8%) |
| Q2-1 | 4 (0.9%) | 27 (6.0%) | 155 (34.4%) | 185 (41.0%) | 79 (17.5%) | 1 (0.2%) |
| Q2-2 | 103 (22.8%) | 233 (51.7%) | 113 (25.1%) | | | 2 (0.4%) |
| Q2-3 | 126 (27.9%) | 253 (56.1%) | 67 (14.9%) | | | 5 (1.1%) |
| Q2-4 | 181 (40.1%) | 175 (38.8%) | 95 (21.1%) | | | 0 |
| Q2-5 | 62 (13.7%) | 111 (24.6%) | 206 (45.7%) | 48 (10.6%) | 23 (5.1%) | 1 (0.2%) |
| Q2-6 | 113 (25.1%) | 124 (27.5%) | 138 (30.6%) | 50 (11.1%) | 23 (5.1%) | 3 (0.7%) |
| Q2-7 | 53 (11.8%) | 66 (14.6%) | 225 (49.9%) | 72 (16.0%) | 35 (7.8%) | 0 |
| Q2-8 | 52 (11.5%) | 76 (16.9%) | 224 (49.7%) | 75 (16.6%) | 23 (5.1%) | 1 (0.2%) |
| Q2-9 | 11 (2.4%) | 57 (12.6%) | 190 (42.1%) | 132 (29.3%) | 60 (13.3%) | 1 (0.6%) |
| Q2-10 | 64 (14.2%) | 125 (27.7%) | 114 (25.3%) | 102 (22.6%) | 45 (10.0%) | 1 (0.2%) |
| Q2-11 | 48 (10.6%) | 149 (33.0%) | 141 (31.3%) | 89 (19.7%) | 23 (5.1%) | 1 (0.2%) |

**Table 4.** Results of factor analysis: eigenvalue of each item

| Factor | Eigenvalue | Cumulative contribution rate (%) |
|---|---|---|
| 1 | **7.600** | **30.4** |
| 2 | **1.795** | **37.6** |
| 3 | **1.556** | **43.8** |
| 4 | **1.217** | **48.7** |
| 5 | **1.095** | **53.1** |
| 6 | 0.996 | 57.0 |
| 7 | 0.942 | 60.8 |
| 8 | 0.893 | 64.4 |
| 9 | 0.783 | 67.5 |
| 10 | 0.756 | 70.5 |
| 11 | 0.728 | 73.4 |
| 12 | 0.680 | 76.2 |
| 13 | 0.656 | 78.8 |
| 14 | 0.643 | 81.4 |
| 15 | 0.617 | 83.8 |
| 16 | 0.584 | 86.2 |
| 17 | 0.505 | 88.2 |
| 18 | 0.482 | 90.1 |
| 19 | 0.433 | 91.9 |
| 20 | 0.427 | 93.6 |
| 21 | 0.387 | 95.1 |
| 22 | 0.361 | 96.6 |
| 23 | 0.320 | 97.8 |
| 24 | 0.302 | 99.0 |
| 25 | 0.239 | 100.0 |

Bold typeface indicates eigenvalues over 1.0

**Table 5.** Results of factor analysis: factor loading of each item

| Item | Factors | | | | |
|---|---|---|---|---|---|
| | 1 | 2 | 3 | 4 | 5 |
| Q2-6 | **0.81** | 0.04 | 0.04 | −0.04 | 0.14 |
| Q2-5 | **0.71** | 0.01 | 0.08 | 0.14 | 0.06 |
| Q1-2 | 0.33 | 0.16 | 0.21 | 0.34 | −0.21 |
| Q2-8 | 0.07 | **0.68** | 0.08 | −0.08 | 0.10 |
| Q2-7 | 0.15 | **0.62** | −0.07 | 0.12 | 0.15 |
| Q2-11 | −0.03 | **0.62** | −0.12 | −0.02 | 0.06 |
| Q1-13 | −0.04 | **0.35** | 0.08 | −0.01 | 0.14 |
| Q2-9 | −0.23 | **−0.52** | −0.11 | 0.05 | 0.05 |
| Q2-10 | 0.06 | **−0.55** | −0.06 | −0.10 | 0.15 |
| Q2-1 | 0.03 | **−0.55** | −0.14 | −0.11 | −0.02 |
| Q1-9 | 0.02 | −0.07 | **0.69** | −0.07 | 0.10 |
| Q1-6 | −0.01 | 0.12 | **0.56** | 0.08 | −0.10 |
| Q2-3 | 0.23 | 0.05 | **0.56** | −0.04 | 0.07 |
| Q1-8 | −0.03 | −0.09 | **0.38** | 0.15 | 0.31 |
| Q1-5 | 0.00 | 0.09 | 0.32 | 0.03 | −0.02 |
| Q1-4 | 0.10 | 0.11 | 0.28 | 0.13 | 0.05 |
| Q1-10 | 0.14 | 0.04 | −0.04 | **0.62** | 0.03 |
| Q2-4 | **0.39** | 0.05 | −0.08 | **0.61** | −0.13 |
| Q1-12 | −0.05 | 0.01 | 0.01 | **0.46** | 0.06 |
| Q1-14 | −0.07 | 0.06 | 0.20 | 0.34 | 0.13 |
| Q2-2 | 0.30 | −0.03 | 0.19 | 0.26 | 0.05 |
| Q1-1 | 0.03 | 0.11 | 0.00 | 0.04 | **0.46** |
| Q1-3 | 0.18 | 0.13 | −0.05 | 0.13 | **0.43** |
| Q1-7 | 0.07 | 0.06 | 0.20 | 0.01 | **0.41** |
| Q1-11 | 0.10 | 0.04 | 0.16 | 0.25 | 0.28 |

Bold typeface indicates absolute value of the factor loading of more than 0.26