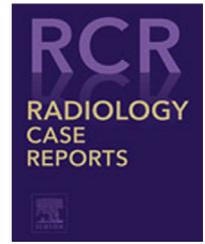


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Case Report

The origin of lumbar subcutaneous edema: two case reports [☆]

Keiji Nakajima, MD, PhD^{a,*}, Tadashi Fujita, MD^b, Ryota Nakano, MD^c

^aNakajima Neuro Clinic, 471-22 Misono, Mishima, Shizuoka 411-0823, Japan

^bGakuto Orthopedic Surgical Clinic, 8-8Minamicyou, Shizuoka 411-0842, Japan

^cDepartment of Surgery, National Hospital Organization Shizuoka Medical Center, 762-1 Nagasawa, Shimizu, Sunto District, Shizuoka 411-8611, Japan

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ABSTRACT

Since magnetic resonance imaging (MRI) is widely used to evaluate complaints of low back pain, there have been many reports of lumbar subcutaneous edema (LSE). However, the mechanism underlying its development is unknown. We herein report 2 cases that showed the reduction of LSE. These cases suggest details concerning the mechanism underlying the development of LSE. The first case was an obese 70-year-old woman with a history of chronic back pain due to lumbar canal stenosis. MRI revealed LSE extending from the level of the L2 vertebral body to the sacrum. However, LSE was reduced following weight loss due to a stomach ulcer. This case clearly indicated obesity as the cause of LSE. The second case was a nonobese 31-year-old woman with acute excruciating low back pain due to thoracolumbar fascia strain. LSE was observed at the level of the L3-L4 vertebral body. Two weeks later, her low back pain and LSE were reduced. This case suggests that the origin of LSE was impairment of the thoracolumbar fascia due to strain. We hypothesize that the mechanism underlying the development of LSE may be lymphatic or interstitial fluid pooling due to disturbance of the lumbar fascia.

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Introduction

Lumbar subcutaneous edema (LSE) was first reported by Shi et al. in 2001 [1]. Those authors speculated that obesity might

play a role in producing such edema, although the precise nature of this presumed edema or fluid collection was unclear.

We herein report 2 cases that showed the reduction of LSE and discuss the mechanism underlying the development of LSE.

Abbreviations: LSE, lumbar subcutaneous edema; MRI, magnetic resonance imaging; NRS, Numerical Rating Scale; TCM, traditional Chinese medicine; STIR, short tau inversion recovery; ROI, region of interest; BMI, body mass index; NSAIDs, non-steroidal anti-inflammatory drugs; iNOS, nitric oxide synthase; NO, nitric oxide.

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* Corresponding author.

E-mail address: knakajima425@nifty.com (K. Nakajima).

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This is the second case report, to our knowledge, to demonstrate reduction of LSE on MRI—a phenomenon for which we also reported the first case [2].

We hypothesized that the origin of LSE might be lymphatic or interstitial fluid pooling due to impairment of fasciae, such as the superficial fascia or the thoracolumbar fascia.

Case presentation

Case 1

A 70-year-old woman who presented with a long history of progressive low back pain described her pain as deep and aching with occasional sharp exacerbation, involving the low back, bilateral buttocks, and anterior thighs to the knees. There was associated numbness and tingling of the entire lower extremities.

Pain was significant while standing, and she experienced cramping in her right leg when walking for one minute recently. The buttock pain had radiated into her legs bilaterally down to the calf for the past roughly one-and-a-half years.

She rated her back pain and numbness as an 8 of 10 on the Numerical Rating Scale (NRS). She noted no bladder or bowel incontinence.

She was 145.8 cm tall and weighed 54.0 kg with a BMI of 25.4, a value that represents obesity in Japan. Her medical history was notable for chronic low back pain and chronic knee pain.

She had undergone radiotherapy and chemotherapy for uterine cervix cancer 12 years earlier.

A neurological examination including motor strength and sensory evaluations, deep tendon reflex, and the straight leg raising test were negative bilaterally.

She had hypertension, but her condition was well controlled by medication, and she had no other notable conditions, such as cardiac, hepatic, or renal dysfunction, that were considered causative of edema.

She also had no history of trauma or infectious disease.

Lumbar X-ray showed findings of lumbar spondylosis but no spondylitis, and lumbar magnetic resonance imaging (MRI) showed a bulging intervertebral disc, hypertrophy of the joint facets, and yellow ligaments at the levels L2-L3, L3-L4, L4-L5, and L5-S1, which had caused narrowing of the spinal canal.

Sagittal 2-dimensional (2D) MR myelography (TR/TE, 7000/500 ms), in which a high signal intensity is mainly related to free water [3], showed spinal canal stenosis and revealed LSE extending from the level of the L3 vertebral body to the sacrum. The LSE seems to be divided into 2 parts (Fig. 1A; arrows and arrowheads).

Sagittal T2 (TR/TE, 300/125 ms) MRI clearly showed the superficial fascia (Fig. 1B; arrow). One area of LSE was inside the superficial fascia (Fig. 1B; arrows), and the other was outside the superficial fascia (Fig. 1B; arrowheads). Axial T2-weighted imaging (TR/TE, 5310/125 ms) showed LSE as areas of high signal intensity, and an ill-defined triangular shape was located posteriorly to the spinous process, between the deep fascia and the superficial fascia, in the deep fat layer at the level of the L3 vertebral body (Fig. 2C; arrows).

We prescribed this patient a traditional Chinese medicine (TCM) formula for the routine treatment of her low back pain in our clinic [2,4]. We did not prescribe any other analgesics or diuretic drugs.

Three months later, the TCM had had some effect on the patient's numbness in her legs and low back pain, as the NRS was now 5. Six months later, she had a stomach ulcer and was losing weight. Eight months later, her weight had decreased by 5 kg, and the NRS was reduced to 3. Her BMI at this point had also decreased from 25.40 to 23.11 (height was 145.6 cm and weight was 49.0 kg).

MRI showed a remarkable reduction in the size of the LSE (Figs. 1D-F).

It is also important to notice that the back fat thickness at the deep fat layer was remarkably reduced, based on a comparison of Figs. 1C☆ and F☆.

On sagittal 2D MR myelography, the area of the LSE was measured by drawing the region of interest (ROI) on the image and then using the area measurement function installed in the MRI system. The area of the LSE before treatment (Fig. 1A) was 405.4 mm², and it became 69.2 mm² after weight loss (Fig. 1D). The reduction rate in the LSE was 82.9%.

In brief, it is natural to conclude in this case that the LSE was reduced following weight loss due to a stomach ulcer.

Case 2

A 31-year-old woman presented with acute low back pain. She had been exercising by putting her abdomen on a balance ball with her hands and feet on the floor, and severe low back pain had suddenly begun when she tried to get up off the balance ball.

Afterward, she became unable to ambulate due to excruciating low back pain. Her symptoms rapidly progressed, and the next day, she was admitted to one of our clinics. She rated her back pain as a 10 on the NRS and described it as excruciating.

A physical examination revealed marked tenderness to palpation throughout the lower lumbar back. Any movement caused pain. Her lower extremity strength was limited by pain but otherwise appeared normal bilaterally.

She could not walk with assistance but had no objective motor weakness or sensory deficits.

Her BMI was 19.7 kg/m² (body weight 57.5 kg, height 170.7 cm), indicating no obesity. She also had no conditions, such as cardiac, hepatic, or renal dysfunction, that could have been considered causative of edema.

She had no history of spinal trauma but had had mild, chronic, non-specific low back pain in her teens.

There were no findings of lumbar spondylosis or spondylitis on X-ray.

MRI was performed 6 days after her admission. A sagittal 2D MR myelogram showed that LSE had extended to the level of the L3-L4 vertebral bodies (Fig. 2A). On axial T2 imaging at L2, a high-intensity area was observed along with the posterior layer of the thoracolumbar fascia bilaterally (Fig. 2B; arrows). At L4, axial T2 imaging showed that the LSE was distributed along the midline in a triangle-shaped configuration next to the thoracolumbar fascia attachment, posteriorly to

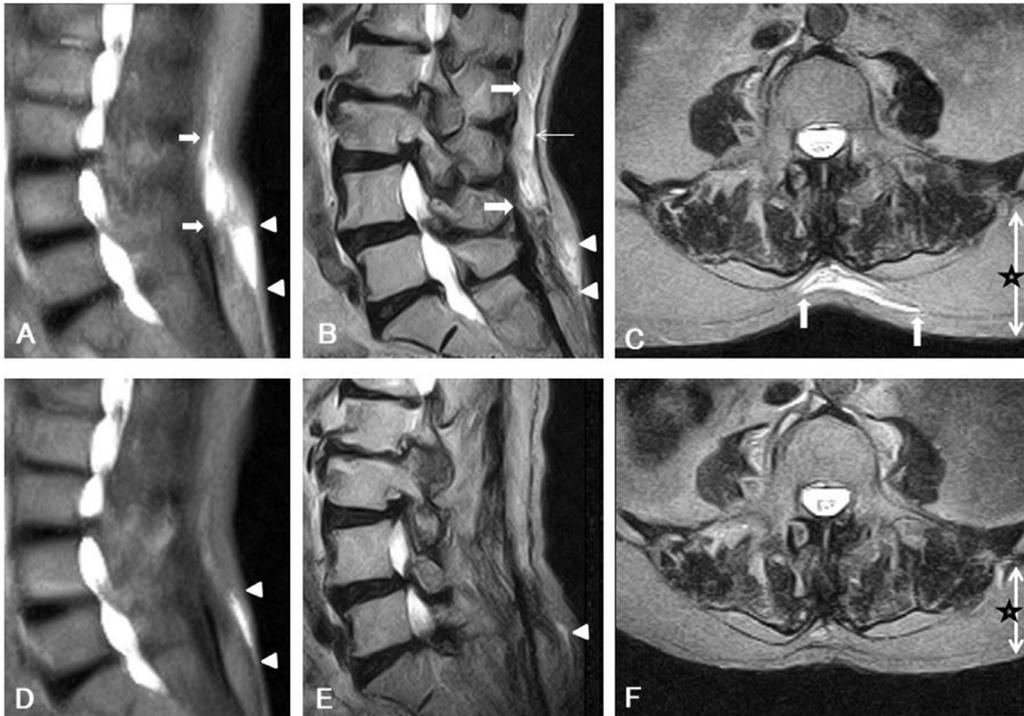


Fig. 1 – Case 1 (A) Sagittal 2D MR-myelography (TR/TE, 7000/500 ms) shows LSE extending from the L3 vertebral body to the sacrum. The LSE seems to be divided into 2 parts (arrows and arrowheads). (B) Sagittal T2 (TR/TE, 5310/125 ms) MRI clearly showed the superficial fascia (B; open arrow). One area of LSE was inside the superficial (arrows), and the other was outside the superficial fascia (arrowheads). LSE thus seemed to be present around the superficial fascia. (C) Axial T2-weighted imaging (TR/TE, 5310/125 ms) showed LSE as an area of high signal intensity, with an ill-defined triangular shape in the deep fat layer at the L3 vertebral body (arrows). Seven months after weight loss due to a gastric ulcer, LSE was markedly reduced on sagittal 2D MR myelography (D), sagittal T2-weighted MRI (E), and axial T2-weighted MRI at the L3 vertebral body (F). It was also noted that back fat was remarkably reduced (C☆ vs F☆).

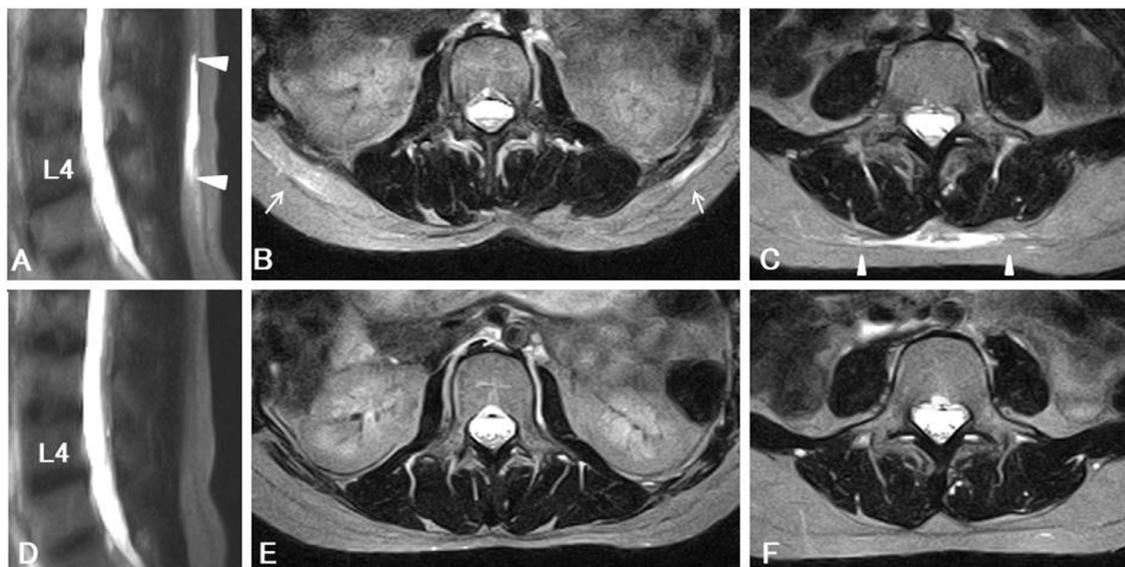


Fig. 2 – Case 2 (A) A sagittal 2D MR myelogram showing LSE extending from the L3 to the L4 vertebral body. (B) On axial T2 imaging at L2, a high-intensity area was observed along with the posterior layer of the thoracolumbar fascia bilaterally. (C) At L4, on axial T2 imaging, LSE was distributed along the midline in a triangle-shaped configuration next to the thoracolumbar fascia attachment, posteriorly to the spinous processes, under the superficial fascia. Two weeks later, LSE had completely disappeared (D-F).

the spinous processes, under the superficial fascia (Fig. 2C; arrowheads).

We prescribed a TCM formula, as in Case 1, in addition to nonsteroidal anti-inflammatory drugs (NSAIDs) and trigger point injection of procaine. Two weeks later, her pain had disappeared, and MRI also showed that the LSE had completely disappeared (Figs. 2D-F). On sagittal 2D MR myelography, the area of the LSE was measured as mentioned before in the MRI system. The area of the LSE before treatment (Fig. 2A) was 251.1 mm², but it became 0 mm² after treatment (Fig. 2D). The reduction rate in the LSE was 100%.

These findings suggest that the origin of LSE was impairment of the thoracolumbar fascia due to acute fascia strain.

Discussion

LSE is incidentally found while evaluating the cause of low back pain using MRI. However, the precise mechanisms underlying the development of LSE remain unknown. In 2003, Shi et al. [1] first reported that LSE occurred more frequently and was more severe in heavy patients. They also noted that LSE was significantly more severe in female and older patient group.

Since then, there have been several reports exploring the mechanism underlying LSE [2,5–13].

While the involvement of age, gender, and posterior spinal compartment degeneration in the formation of LSE remains controversial, several studies have shown that LSE is closely related to obesity and the BMI, as confirmed by Shi et al. [2,6,8–10,12,13]. However, the patient in our Case 2 was not obese, with a BMI of 19.7 kg/m². Furthermore, neither of our cases had any internal or bedridden conditions that might have produced edema or generalized fluid retention.

Regarding the common findings concerning the induction of LSE in our cases, we believe that the lumbar fascia played a key role. For example, Case 1 was related to the superficial fascia, and Case 2 was related to the thoracolumbar fascia.

Schwarz-Nemec et al. [13] recently reported that the perifascial presentation of LSE on MRI may suggest a potential fascial origin. The MRI findings of LSE seem to mimic a peritendinitis-like appearance, potentially suggesting abnormal stress and overuse of the fascial tissues. Thus, the condition may be termed lumbar perifascial edema rather than subcutaneous edema. The present findings appear consistent with this proposal.

The patient in Case 1 had a BMI of 25.1 kg/m², indicating an obese condition according to Japanese criteria (definition of the Japan Society for the Study of Obesity: BMI > 25.0 kg/m²). Given that LSE in this case was reduced by the amelioration of obesity due to gastric ulcers, it seems clear that obesity is one of the mechanisms involved in the development of LSE.

Anatomically, subcutaneous adipose tissue is separated by the superficial fascia into the superficial and deep adipose layers. The deep adipose layer is contained within a relatively loose, less organized, and more widely spaced fascial septum than the superficial adipose layer [1,14]. This may account for the accumulation of edema at the deep adipose layer [15].

According to Stecco et al. [14], the superficial fascia is a fibrous layer of connective tissue, formed by loosely packed interwoven collagen fibers mixed with abundant elastic fibers. It was suggested that the disposition of the collagen and elastic fibers inside the superficial fascia could guide the lymphatic flux in the correct direction. If the superficial fascia is altered, then lymphatic drainage is compromised.

Based on our understanding of the strong relationships between the superficial fascia and lymph vessels, it can be hypothesized that changes in the superficial fascia can cause lymphedema [16].

Regarding the relationship between LSE and obesity, obesity-induced enlarged adipose deposition physically alters the connective tissue, which can guide lymphatic flux in the superficial fascia. Alternatively, enlarged adipose deposition may physically compress the lymph vessels in the deep adipose layer.

As the amount of hypertrophied adipose tissue decreased, so did LSE (see Case 1, Fig. 1F☆, showing markedly less back fat than in Fig. 1C☆).

However, in our previously reported case [2], LSE was remarkably reduced without weight loss by the administration of TCM or exercise. Hespe et al. [17] showed that obesity markedly impairs the lymphatic function by decreasing the pumping frequency of collecting lymphatics, decreasing the lymphatic vessel density, increasing the lymphatic leakiness, and changing the gene expression patterns of lymphatic endothelial cells in obese mice. Furthermore, they showed that these pathological effects were reversible with aerobic exercise, independent of weight loss. They also pointed out that the perilymphatic accumulation of inflammatory cells (T cells and macrophages) and the expression of inducible nitric oxide synthase (iNOS) play a key role in the regulation of obesity-related lymphatic dysfunction.

Kataru et al. [18] reported that the loss of nitric oxide (NO) gradients around the collecting lymphatic vessels due to the high expression of iNOS by perilymphatic inflammatory cells impairs lymphatic pumping and results in the dilatation of the collecting lymphatic vessels.

These findings suggest that perilymphatic inflammatory responses play a role in the development of LSE in obesity, in addition to the physical compression of the lymphatic system by enlarged adipose deposition.

The second case suggested that the origin of LSE was impairment of the thoracolumbar fascia due to fascia strain. On axial T2 imaging at L2, a high-intensity area was observed along the thoracolumbar fascia bilaterally (Fig. 2B; arrows), demonstrating damage to the thoracolumbar fascia. This is consistent with what happened when she was hanging over the balance ball: her abdominal pressure rose, which induced tensile forces over the thoracolumbar fascia [19]. As mentioned before, she had had mild chronic low back pain since her school days. Langevin et al. reported that the thoracolumbar fascia shear strain was approximately 20% lower in human subjects with chronic low back pain than in those without such pain. This reduction in shear plane motion may have been due to abnormal trunk movement patterns and/or intrinsic connective tissue pathology [20]. That is, she bent and exercised on the balance ball at a point with a reduction in shear plane motion of the thoracolumbar fascia, while her ab-

dominal pressure was rising. Furthermore, tensile forces become strong enough to tear the thoracolumbar fascia when she tried to get off the balance ball.

The deep fascia and thoracolumbar fascia tend to be highly vascularized and contain well-developed lymphatic channels [21]. Derangement of the thoracolumbar fascia likely prevents not only lymph drainage but also venous drainage.

Schwarz-Nemec et al. [13] suggested that LSE might be related to potential pathophysiological changes in connective tissue layers and fascial derangement. The LSE in Case 2 may suggest a thoracolumbar fascia origin.

In summary, LSE in the first case was caused by an alteration of the superficial fascia due to obesity, whereas that in the second case was caused by strain damage to the thoracolumbar fascia.

We hypothesize that the mechanism underlying the development of LSE may involve lymphatic or interstitial fluid pooling due to changes in the lumbar fascia.

Patient consent

We have written informed consent to publish this case and use anonymized radiologic material obtained from the patient.

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