Primary Superior Lumbar Hernia with Nephrotic-range Orthostatic Proteinuria

Taro Horino¹, Takeshi Kashio¹, Satoshi Inotani¹, Sachi Yamaguchi², Masayuki Ishihara³, Osamu Ichii⁴ and Yoshio Terada¹

Abstract:
Lumbar hernias are extremely rare. The posterolateral abdominal wall has two susceptible areas - the superior (Grynfelt-Lesshaft’s triangle) and the inferior (Petit’s triangle) lumbar triangles - that cause superior and inferior lumbar hernias, respectively. We herein report a 67-year-old woman with nephrotic-range proteinuria caused by primary superior lumbar hernia. Superior lumbar hernias should be considered as a differential disease causing massive orthostatic proteinuria in adults. The present case highlights the importance of considering lumbar hernia in patients with flank swelling and the potential complications that may result from a missed diagnosis.

Key words: computed tomography, orthostatic proteinuria, superior lumbar hernia

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Introduction
Lumbar hernias are extremely rare, with only 300 cases reported in the literature (1). The posterolateral abdominal wall has two susceptible areas - the superior (Grynfelt-Lesshaft’s triangle) and the inferior (Petit’s triangle) lumbar triangles - that cause superior and inferior lumbar hernias, respectively. The superior lumbar triangle is an inverted triangle bordered superiorly by the 12th rib and the serratus posteroinferior muscle, laterally by the quadratus lumborum, and medially by the erector spinae muscles (2). The floor of the triangle is composed of the transversalis fascia and the roof by the external oblique muscle (2). However, lumbar hernias are rarely included in the differential diagnosis of flank swelling.

Case Report
A 67-year-old Japanese woman with a 1-month history of severe proteinuria was admitted to our hospital. The patient’s medical history was insignificant and did not include a history of surgery or trauma.

Upon the first admission, a physical examination revealed bilateral edema of the legs and soft masses in the bilateral lower lumbar areas (Fig. 1A); there were no other symptoms. The blood pressure was 112/68 mmHg, and the body mass index was 29.3 kg/m². The serum creatinine level was 0.82 mg/dL (normal range: 0.46-0.79 mg/dL), and the serum albumin level was 4.1 g/dL (normal range: 4.1-5.1 g/dL). The patient exhibited microhematuria [10 red blood cells/high-power field (RBCs/HPF)] (<5), and the urinary protein excretion was 6.1 g/gCr (<0.3 g/gCr). Serological tests, including those for antinuclear antibodies, were all negative. Chest radiographs and computed tomography revealed prolapse of perirenal fatty tissue through the muscular defects on the lateral side of the bilateral kidneys (Fig. 1B, C) and significant elevation of the left diaphragm (Fig. 1D). On day 3 of hospitalization, the patient’s proteinuria and hematuria spontaneously decreased to 0.1 g/gCr and 0-1 RBCs/HPF, respectively, without treatment, and the patient was discharged from the hospital.

However, the patient was immediately readmitted to the hospital because of an outpatient recurrence of proteinuria at
21.0 g/gCr. The proteinuria became negative shortly after admission during the second hospitalization but reappeared after discharge. Abdominal magnetic resonance imaging performed during the second hospitalization confirmed prolapse of perirenal fatty tissue through the muscular defects on the lateral side of the kidneys. Based on the above findings, the patient was diagnosed with a primary superior lumbar hernia with orthostatic proteinuria and was hospitalized for a third time for surgery.

During the surgery, retroperitoneal fat was found escaping from the superior lumbar triangle (Fig. 2A). After reinforcement with mesh (Fig. 2B), the hernia gate was sewn. The patient’s clinical course is presented in Fig. 3. Postoperatively, proteinuria became negative, and no recurrence was observed at subsequent outpatient visits.

**Discussion**

Lumber hernias are classified as congenital (20%) and acquired (80%) (2). Acquired lumber hernias are further classified into primary (spontaneous) (55%) and secondary (25%) (3). Primary lumber hernia is associated with predisposing factors, such as old age, chronic lung disease, obesity, muscular atrophy, connective tissue disease, poor nutri-
tional status, and conditions that increase intra-abdominal pressure (1, 4), while secondary lumbar hernia is associated with iatrogenic, traumatic, or following infection or inflammation (5). A primary superior lumbar hernia occurs unilaterally predominantly on the left side, and cases where both sides are affected, such as in our case, are extremely rare, with many being congenital and associated with the diffuse type, which presents as aplasia of lumbar muscles (6).

To our knowledge, only one case of primary bilateral lumbar hernia has been reported thus far (7). Our patient was also considered to be a rare case of bilateral spontaneous lumbar hernia, as he was elderly and obese, and no other cause was found. Furthermore, in our patient, the increase in intra-abdominal pressure caused by the deterioration of posture due to the deformation of the lumbar spine may have also contributed to lumbar hernia. Symptoms, especially pain, are due to hernia incarceration, and if left untreated, the hernia phylum expands and becomes difficult to repair; therefore, surgery is the first choice of treatment. Around 25% of these hernias are prone to incarceration and 10% to strangulation, which may present with features of acute abdomen and need emergency surgery (8). In addition, the previously reported cases are only surgical cases described in surgical journals, and no laboratory data were reported in those cases. Since there have been no reported cases in which the patient did not undergo surgery or was evaluated for the presence of laboratory abnormalities, more cases of lumbar hernias may exist that are unreported, and cases of proteinuria may have been overlooked or have had a wasteful renal biopsy. Our case is the first in which massive proteinuria was the trigger for discovery. Orthostatic proteinuria associated with lumbar hernia has not been confirmed by previous researchers but may be an important clinical symptom that aids in the early diagnosis of lumbar hernia.

Orthostatic proteinuria, also known as postural proteinuria, is a condition where the patient excretes an abnormally large amount of protein in the urine in an upright position and normal protein in the supine position (9). Although the prevalence of orthostatic proteinuria in adolescents is reported to be approximately 2-5%, it is an uncommon disorder in individuals >30 years old (9). Other glomerular diseases should be differentiated from orthostatic proteinuria to avoid unnecessary invasive interventions, such as a kidney biopsy. Although the mechanisms responsible for orthostatic proteinuria remain unclear, left renal vein entrapment has recently been suspected to be a cause (9). In our case, the expression pattern of proteinuria was clinically different from that of general orthostatic proteinuria. Early-morning urinary protein excretion and occasional urinary proteinuria measured on the same day during hospitalization were 0.135 g/gCr and 0.233 g/gCr, respectively, and only a slight increase was observed in occasional urine. However, the outpatient overt proteinuria was completely normalized at rest in the hospital. Therefore, we speculate that causes of differences in clinical expression patterns between general orthostatic proteinuria and our patients’ proteinuria are due to several reasons. First, general orthostatic proteinuria is caused by the position of the kidneys which are heavily hanging down in the abdominal cavity when in the standing
position, but in patients with a lumbar hernia, the kidneys move horizontally to the dorsal side. In cases of general orthostatic proteinuria, circulatory dysfunction occurs acutely at the renal arteriovenous level, while in patients with a lumbar hernia, circulatory dysfunction might occur at the subacute intrarenal arteriole level. Second, a lumbar hernia has a wider hernia hole than other abdominal wall hernias, and the superior triangle is wider than the inferior one. Therefore, many patients with a lumbar hernia may have a temporary incarceration rather than a permanent one. We speculate that these differences may be responsible for whether the pattern of transient proteinuria expression is acute or subacute. We propose that the difference in transient proteinuria expression patterns may distinguish between orthostatic proteinuria due to lumbar hernia and proteinuria due to other causes.

In conclusion, a superior lumbar hernia should be considered as a differential disease causing massive orthostatic proteinuria in adults. This case highlights the importance of considering lumbar hernia in patients with flank swelling and the potential complications that may result from missing the diagnosis of this condition. Clinicians must be careful to make a proper differential diagnosis and prevent the unnecessary performance of invasive procedures in a patient.

Informed consent was obtained from the patient for publication of this case report and corresponding images.

The authors state that they have no Conflict of Interest (COI).

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