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# Spinal hypertension-like syndrome with metabolic acidosis during percutaneous endoscopic lumbar discectomy：a case report

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**Case Report**

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# Abstract

**Study Design.** Case-report and literature review.

**Objective.** This report presents a rare case of spinal cord hypertension-like syndrome following percutaneous transforaminal endoscopic lumbar discectomy (PTELD). We reviewed the existing literature to elucidate potential causes, emphasizing the need for meticulous technique and early detection of neurological deficits to improve safety in this procedure.

**Summary of Background Data:** PTELD is a minimally invasive procedure that effectively achieves neural decompression by removing the herniated nucleus pulposus via a posterolateral approach. The incidence of perioperative complications associated with PTELD is exceptionally low, as extensively documented. However, although rare, patients undergoing PTELD may occasionally experience symptoms suggestive of spinal cord hypertension-like syndrome.

**Methods:** A 47-year-old male presented with severe lower back pain and underwent PTELD under general anesthesia. Intraoperatively, the patient developed persistent hypertension and tachycardia. During the recovery phase from general anesthesia, he exhibited extreme agitation, generalized muscle rigidity, increased RR, tachycardia, and elevated BP. Midazolam, propofol, and sufentanil were administered to manage agitation, while urapidil and esmolol were sequentially used to control BP. Hydrocortisone (100 mg) was given to provide neuroprotection.

**Results:** The patient exhibited no further symptoms of spinal cord hypertension syndrome and is recovering satisfactorily.

**Conclusion:** We hypothesize that the sudden intraoperative increase in BP and HR may be associated with a dural sac rupture during tissue clamping near the dural sac, allowing irrigation fluid to enter and perfuse inward, thereby elevating intracranial pressure. To mitigate this risk, surgical maneuvers should be performed with precision and efficiency to avoid dural tears. In cases where symptoms suggestive of spinal cord hypertension syndrome arise intraoperatively, prompt administration of osmotic agents such as mannitol and diuretics like furosemide is recommended to reduce intracranial pressure. Additionally, elevating the head of the bed and implementing protective measures, such as brain ice packing, should be considered. Anesthesiologists must be well-versed in managing perioperative complications related to elevated intracranial pressure to ensure optimal patient outcomes.


# Background

Lumbar Disc Herniation (LDH) is a prevalent orthopedic condition with a multifaceted etiology, primarily driven by genetic predisposition, intervertebral disc degeneration, developmental anomalies, lumbar trauma, and poor posture [1]. In recent years, the incidence of LDH has risen, increasingly affecting younger individuals, resulting in substantial economic and medical burdens for patients, their families, and society, while significantly impairing daily functioning and work capacity [2, 3]. Approximately 85% of

LDH patients experience sciatica, and some may develop severe complications such as urinary or fecal incontinence. Although non-surgical treatments, including medication and physical therapy, are typically the first-line approaches, surgical intervention is often required when these measures prove ineffective [4].

PTELD has become widely adopted for treating LDH in recent years, owing to its advantages of minimal tissue trauma, reduced intraoperative bleeding, reliable therapeutic outcomes, and accelerated recovery times. It has earned strong endorsement from spine surgeons [5]. However, as the use of PTELD expands and the number of procedures increases, its associated complications have garnered significant attention. These complications primarily include LDH recurrence, nerve root injury, dural sac injury, epidural hematoma, residual nucleus pulposus, postoperative sensory disturbances, and spinal cord hypertension-like syndrome [6]. Currently, there is a lack of targeted research investigating the occurrence of spinal cord hypertension-like syndrome following PTELD.

Spinal cord hypertension-like syndrome is a rare but specific complication of PTELD. As PTELD is typically performed under general anesthesia, clinical manifestations of this syndrome may be obscured intraoperatively due to systemic anesthesia and circulatory regulation, often becoming evident during the postoperative anesthesia recovery phase [7]. The clinical presentations and their impacts vary based on the surgical approach and anesthesia technique employed. However, with prompt recognition and appropriate management, symptoms of spinal cord hypertension-like syndrome in most PTELD patients can be effectively resolved within 24 hours. On May 23, 2025, our hospital admitted a patient who developed spinal cord hypertension-like syndrome accompanied by metabolic acidosis following PTELD under general anesthesia. By documenting the diagnostic and therapeutic process and reviewing relevant literature, this case report aims to provide valuable insights for clinical and anesthesiology practitioners in managing perioperative spinal cord hypertension-like syndrome.

## Case presentation

On May 21, 2025, a 47-year-old male patient presented to the Pain Department with a chief complaint of lumbosacral pain accompanied by radiating pain in the left lower limb for two weeks. The patient reported the onset of lumbosacral pain and discomfort without an apparent trigger, associated with radiating pain extending from the left buttock to the posterior thigh and lateral calf, as well as numbness on the lateral aspect of the left ankle. Previous non-surgical treatments, including oral medications (details unspecified), acupuncture, and physical therapy, failed to provide significant symptom relief.

The patient had an unremarkable medical history, with no evidence of hypertension, diabetes, coronary heart disease, or prior intracranial, cervical, or thoracic spine conditions or surgeries. Upon presentation to our hospital, an MRI revealed an L4/5disc herniation (Fig. 1). Electromyography showed no abnormalities, and ultrasound examination indicated no significant findings in the bilateral piriformis muscles. Physical examination demonstrated significant tenderness in the bilateral lumbosacral paravertebral region and positive percussion pain at the spinous process. Radiating pain was observed in the right buttock, posterior thigh, and lateral calf, accompanied by pronounced localized pressure pain.

The straight leg raise test was positive at 50° for the left lower limb, with a positive reinforcement test. The Visual Analog Scale (VAS) score was 5. Other clinical examinations and laboratory results revealed no notable abnormalities.

Following consultation with specialists, the decision was made to perform a percutaneous transforaminal endoscopic lumbar discectomy with nucleus pulposus removal and release of spinal cord and nerve root adhesions under general anesthesia. The patient's preoperative body mass index (BMI) was 25.8 kg/m², with an American Society of Anesthesiologists (ASA) classification of Grade I.

At 14:10 upon entering the operating room, the patient's vital signs were recorded as follows: HR 83 bpm, BP 113/84 mmHg, and $SpO_2$ 92%. Following induction of general anesthesia, a 7.5 mm endotracheal tube was inserted, and mechanical ventilation was maintained in pressure-controlled ventilation with volume guaranteed (PCV-VG) mode. Intraoperative anesthesia was sustained using 1.5% sevoflurane, remifentanil at 0.15 μg/kg/min, and dexmedetomidine at 0.4 μg/kg/h.

The surgical procedure commenced at 14:30. Post-induction, the patient's BP was maintained within the range of 100–126/59–88 mmHg, HR between 61–72 bpm, and $SpO_2$ between 96–100%.

At 16:00, the patient experienced a sudden increase in BP to 206/105 mmHg, with a HR of 95 bpm. An additional 10 μg of sufentanil was administered, resulting in minimal reduction in BP and HR. Subsequently, 10 mg of propofol and 4 mg of cisatracurium were given, sevoflurane was increased to 8%, and oxygen flow was adjusted to 4 L/min. Following these interventions, BP decreased to a range of 120–140/80–100 mmHg, and HR stabilized between 70–80 bpm.

The surgical procedure concluded at 16:50, with a duration of 140 minutes, utilizing five bags (3000 ml) of saline for irrigation. At 17:00, the patient was transferred to the recovery room with the endotracheal tube in place, exhibiting vital signs of HR120 bpm, BP 138/79 mmHg, and $SpO_2$ 97%. By 17:03, spontaneous breathing resumed with a RR of 35 breaths per minute, HR increased to 132 bpm, and BP rose to 150/78 mmHg. The patient displayed agitation and intolerance to the endotracheal tube, likely due to tube irritation, prompting its removal.

At 17:05, the patient's HR escalated to 160 bpm, BP rose to 180/108 mmHg, and RR reached ~ 32 breaths/min with deep, rapid breathing, agitation, and generalized muscle hypertonia. Intravenous urapidil 10 mg and propofol 50 mg were given, reducing BP to 145/98 mmHg, HR to 133 bpm, and RR to 28–30 breaths/min. Muscle rigidity persisted, and the patient remained unresponsive to verbal stimuli. Oxygen via mask at 5 L/min maintained $SpO_2$ at 96%. Arterial blood gas analysis showed pH 7.26, $PCO_2$ 37.6 mmHg, lactate 8.9 mmol/L, base excess − 9.7 mmol/L, and $HCO_3^-$ 16.8 mmol/L. Sodium bicarbonate 200 mL was administered.

At 17:10, the patient showed renewed agitation, persistent generalized muscle rigidity, and facial flushing. Suspecting postoperative breakthrough pain, intravenous oxycodone 2 mg and propofol 70 mg were administered. Body temperature was 36.9°C.

At 17:15, intravenous dexamethasone 10 mg was administered, with BP at 154/102 mmHg, HR at 145 bpm, and persistent generalized muscle hypertonia. At 17:20, intravenous esmolol 15 mg, midazolam 3 mg, and sufentanil 10 μg were given; body temperature was 37.8°C.

At 17:30, arterial blood gas analysis revealed pH 7.41, end-tidal $CO_2$ 28.7 mmHg, lactate 9.8 mmol/L, base excess − 4.8 mmol/L, and $HCO_3^-$ 20.4 mmol/L. RR decreased to 26 breaths/min. Upper limb muscle tone slightly improved, but lower limb hypertonia persisted. At 17:45, intravenous hydrocortisone 100 mg was administered.

At 17:55, repeat arterial blood gas analysis showed pH 7.55, $PCO_2$ 26.4 mmHg, lactate 5.5 mmol/L, base excess − 2.2 mmol/L, and $HCO_3^-$ 26.2 mmol/L, reflecting marked improvement in metabolic acidosis. Axillary temperature was 38.1°C, BP was 135/89 mmHg, and HR was 135 bpm.

At 18:00, BP was 153/103 mmHg, HR was 120 bpm, RR was 25 breaths/min, with mask oxygen at 5 L/min and $SpO_2$ at 92%. Upper limb muscle tone normalized, and lower limb muscle tone showed slight improvement. Following consultation with the surgical team and family, the patient was transferred to the ICU for ongoing monitoring and treatment. During 60 minutes in the recovery room, 500 mL of balanced solution was infused. After 14 hours of ICU observation, vital signs stabilized, and the patient was safely transferred back to the ward. A multidisciplinary consultation diagnosed postoperative spinal cord hypertension-like syndrome based on symptoms, signs, and treatment response. Interventions including warming, sedation, and oxygen therapy improved the patient's condition. The patient reported bilateral costal margin and leg muscle soreness, but HR and BP normalized. Follow-up arterial blood gas analysis was normal, and agitation and tension subsided. Preoperative lumbar symptoms also improved. On postoperative day five, with stable vital signs and satisfactory recovery, the patient was discharged for rehabilitation.

# Discussion

Spinal cord hypertension-like syndrome is characterized by an abnormal increase in spinal and intracranial pressure resulting from imbalanced perfusion fluid pressure or dural injury during spinal endoscopic surgery. Symptoms typically emerge within 24–72 hours post-surgery, initially presenting as neck fullness and stiffness, followed by headache, limb sensory disturbances, agitation, tachycardia, and acute hypertension (potentially exceeding 200/120 mmHg). Additional manifestations may include confusion, seizure-like episodes, and lower limb motor dysfunction, reflecting a spectrum of neurological impairments [8, 9].

Spinal cord hypertension-like syndrome is more frequently observed in patients undergoing spinal endoscopic surgery with local anesthesia. Under general anesthesia, intraoperative circulation and respiration are managed by anesthetic agents and mechanical ventilation, limiting real-time patient feedback and complicating the identification of this complication during surgery.

Literature documents a case of spinal cord hypertension-like syndrome following unilateral biportal endoscopic surgery, with the patient experiencing occipital pain, mental dullness, agitation, tachycardia, and hypertension during postoperative recovery [10]. Liu et al. [11] reported five cases complicating percutaneous endoscopic lumbar discectomy (PELD), with three cases presenting intraoperative neck pain and the others exhibiting agitation, a sense of impending doom, hypertension, sensory abnormalities, and motor dysfunction. Choi et al. [12] described four cases of postoperative seizures following PELD, all initially presenting with neck pain and stiffness, with one case involving visual impairment and transient loss of consciousness.

In the current case, the patient displayed hypertension, tachycardia, loss of consciousness, extreme agitation, and muscle rigidity during intraoperative and postoperative recovery periods. Although occipital or perineal pain was absent, other symptoms and arterial blood gas analysis aligned with prior reports. This may be due to intraoperative anesthesia masking additional symptoms, while extensive use of sedatives and analgesics during recovery likely influenced the assessment of further symptoms.

The precise mechanisms of spinal cord hypertension-like syndrome remain unclear due to limited clinical research. Proposed contributing factors include:

### Continuous High Perfusion Pressure Intraoperatively

The epidural space, an expandable cavity with inherent inflow resistance, can accumulate irrigation fluid when endoscopic perfusion pressure exceeds 40 mmHg and irrigation persists beyond 2 hours. This elevates epidural pressure, forcing cerebrospinal fluid toward the head and neck, increasing cervical epidural and intracranial pressure, and triggering clinical symptoms. Studies recommend maintaining perfusion rates below 150 mL/min and reducing irrigation fluid height to under 60 cm, when feasible, to ensure surgical safety and quality [13, 14]. An animal study in rabbits confirmed a positive correlation between epidural and intracranial pressure peaks and perfusion rate, with irrigation fluid height in the epidural space proportional to perfusion duration [8].

### Retrograde Perfusion Due to Dural Tears

Improper surgical manipulation or severe adhesions during tissue resection near the dural sac may cause small dural tears. Irrigation fluid enters through these tears, elevating intradural pressure, which transmits to the neck and cranium. This direct compression of the conus medullaris, combined with increased intracranial pressure, is a primary driver of spinal cord hypertension-like syndrome [15]. Joh et al. [16] measured cervical epidural pressure in 28 PELD patients, noting significantly higher pressures in the 8 patients with spinal cord hypertension-like syndrome symptoms, particularly during the onset of neck pain, compared to others.

The patient underwent spinal and nerve root adhesion release, a procedure demanding high technical precision. During adhesion dissection, meticulous care is essential to protect the dural sac and nerve roots from traction or thermal injury. The intraoperative onset of hypertension and tachycardia may stem

from a dural sac tear during tissue manipulation, triggering spinal cord hypertension-like syndrome. Therefore, surgical maneuvers must prioritize efficiency and precision to prevent irrigation fluid from entering the dural sac through tears.

### Impaired Spinal Venous Return

Insertion of the working channel and injection of irrigation fluid increase intraspinal pressure, altering hemodynamics and impeding spinal venous return, leading to venous stasis. Low-temperature irrigation fluid should be avoided to prevent vasospasm. The low-pressure venous plexus, lacking venous valves, may permit blood backflow, causing spinal ischemia, hypoxia, neuronal necrosis, and associated neurological symptoms [17].

### Age, Spinal Stenosis, and Reduced Dural Sac Volume

The risk of spinal cord hypertension-like syndrome rises with age, likely due to advanced lumbar degeneration, including disc herniation, perispinal osteophytosis, and sclerosis of the ligamentum flavum or posterior longitudinal ligament, which diminish spinal canal volume and pressure tolerance [18]. Choi et al. [12] noted that spinal degeneration reduces intracranial compliance. The epidural space, a leaky compartment with inherent resistance, allows fluid leakage to surrounding tissues based on foraminal size, adipose tissue density, and adhesions. In older patients with severe degeneration, smaller spinal canal volume, reduced fluid leakage, and poorer intracranial compliance heighten the risk of spinal cord hypertension-like syndrome.

### Surgical and Anesthesia Methods

Studies suggest the posterior interlaminar approach poses a higher risk of spinal cord hypertension-like syndrome than the transforaminal approach. In the transforaminal approach, irrigation fluid exerts lateral pressure, whereas the posterior approach directly compresses the posterior dural sac, more readily increasing intracranial pressure [19].

### Intraoperative Positioning

Proper positioning, with the head elevated and feet lowered, is critical to avoid a head-down, feet-up posture. If spinal cord hypertension-like syndrome occurs intraoperatively, surgery should be paused, irrigation fluid stopped, and the patient repositioned to a head-up, feet-down position, with oxygen and sedation administered. Surgery may resume and be completed quickly once symptoms improve, followed by postoperative corticosteroids and dehydrating agents.

In this case, general anesthesia masked intraoperative symptoms, complicating the detection of spinal cord hypertension-like syndrome. Postoperative pain, urinary catheter irritation, and incomplete recovery from anesthesia delayed early identification. Limited clinician awareness and ineffective interventions, due to residual muscle relaxant effects, exacerbated symptoms. While mild cases may resolve within 24

hours, vigilant monitoring and prompt management are crucial to prevent progression. Treatment should be tailored to intraoperative or postoperative symptom onset.

## Management of Intraoperative Spinal cord hypertension-like syndrome

**If symptoms arise during surgery, immediate measures include**:

1. Pause Surgery and Stop Irrigation Fluid.
2. Adjust Positioning: Adopt a head-up, feet-down position at 15°–30° to promote fluid reflux.
3. Cease Irrigation for ≥ 5 Minutes: This often alleviates symptoms.
4. Supportive Treatment: Ensure adequate oxygenation, sedation, and analgesia if needed.

**Intraoperative Control Measures**

1. Maintain Muscle Relaxation: Prevent paraspinal muscle contraction and promote fluid drainage.
2. Controlled Hypotension: Maintain systolic BP at 80–90 mmHg, or ≥ 100 mmHg for high-risk patients.
3. Complete Surgery Promptly: After symptom resolution, finish the procedure safely.

## Management of Postoperative Spinal cord hypertension-like syndrome

**Upon symptom onset post-surgery, initiate**:

1. Position Adjustment: Elevate the head of the bed at 15°–30° for at least 6 hours.
2. Pharmacological Treatment: Administer oxygen, sedation, and analgesia.
3. Corticosteroids: Use dexamethasone or methylprednisolone to reduce inflammation.
4. Dehydrating Diuretics: Administer mannitol to lower intracranial pressure.
5. Monitoring Vital Signs: Focus on consciousness and vital signs for early detection.
6. Special Interventions: Consider lumbar cistern drainage if conservative treatments fail.
7. Surgical Intervention: If ineffective after 24 hours, perform dural repair or decompression.
8. Psychological Support: Provide interventions to manage anxiety.

In this case, arterial blood gas analysis at 17:05 revealed metabolic acidosis, with pH 7.26, $PCO_2$ 37.6 mmHg, lactate 8.9 mmol/L, BE -9.7 mmol/L, and $HCO_3^-$ 16.8 mmol/L. Metabolic acidosis, a common acid-base disorder, is marked by a primary reduction in plasma bicarbonate ($HCO_3^-$), lowering pH. Acute metabolic acidosis arises from conditions increasing plasma hydrogen ion ($H^+$) activity, reducing pH and base excess [20]. It can impair myocardial contractility, reduce peripheral vascular sensitivity to catecholamines, and heighten risks of arrhythmias, acute renal failure, and shock, which are challenging

to manage. Causes include lactic acidosis, ketoacidosis, renal acidosis, diarrhea, hypothermia, tissue hypoxia, and hypoperfusion. Mild cases may be asymptomatic, but severe cases can cause nausea, vomiting, fatigue, dizziness, and somnolence. The hallmark sign is deep, rapid breathing (40–50 breaths/min), resembling Kussmaul respiration, often with a ketotic odor and facial flushing. Here, the patient exhibited facial flushing and rapid, deep breathing, with arterial blood gas results confirming metabolic acidosis.

The following two factors are considered potential causes:

### Prolonged Surgery and Hypothermia

Extended surgical duration combined with the use of 3000 mL of unheated irrigation fluid led to a minimum patient temperature of 35.1°C. This induced tissue hypoperfusion and reduced oxygen delivery, resulting in metabolic acidosis.

### Tissue Hypoperfusion and Cellular Ischemia/Hypoxia

The lack of a preoperative urinary catheter limited intraoperative fluid administration, potentially exacerbating hypoperfusion. Postoperatively, the patient exhibited generalized muscle rigidity, with arterial blood gas analysis revealing a lactate level of 8.9 mmol/L, indicative of anaerobic metabolism.

Acid-base imbalances arise from various underlying causes, and correcting the primary issue often resolves the imbalance. To prevent metabolic acidosis in elective surgery patients, implementing the Enhanced Recovery After Surgery (ERAS) protocol is beneficial. Consuming high-carbohydrate meals 2 hours preoperatively can alleviate thirst, hunger, and agitation while optimizing hydration and volume status in the operating room, reducing intraoperative complications and enhancing recovery. Additionally, routine intraoperative temperature monitoring, combined with warming blankets and heated fluids or gases to maintain a body temperature ≥ 36°C, effectively prevents complications associated with hypothermia [21].

## Conclusion

To mitigate spinal cord hypertension-like syndrome during PTELD, critical preventive strategies include customized preoperative anesthesia plans, invasive BP monitoring, real-time core temperature assessment, and the use of warmed irrigation fluid. Employing gentle surgical techniques to prevent dural tears and maintaining effective intraoperative communication are essential. Prompt recognition and management of postoperative agitation are also crucial. Though rare, this syndrome poses significant risks to PTELD safety, underscoring the need for early prevention and vigilant management. Continued research into its mechanisms is essential, and anesthesiologists must prioritize proactive measures and meticulous care to ensure patient safety throughout the perioperative period.

## Declarations

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Not applicable.

### Author contributions

H. S. was the primary author of this research and contributed to the collection, analysis, and interpretation of patient data, as well as manuscript preparation. X. Z. contributed to data curation, methodology, and writing (review and editing), and helped conceive and design the study. J. W. and Y. F. contributed to writing the original draft. Y. L. and J. B. contributed to funding acquisition, methodology, and writing (review and editing). All authors read and approved the final manuscript.

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### Ethics declarations

### Consent for publication

Written informed consent was obtained from the patient for publication of this case report and any accompanying data and images.

### Competing interests

The authors declare no competing interests.

### Data availability

No datasets were generated or analyzed during the current study.

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# Figures

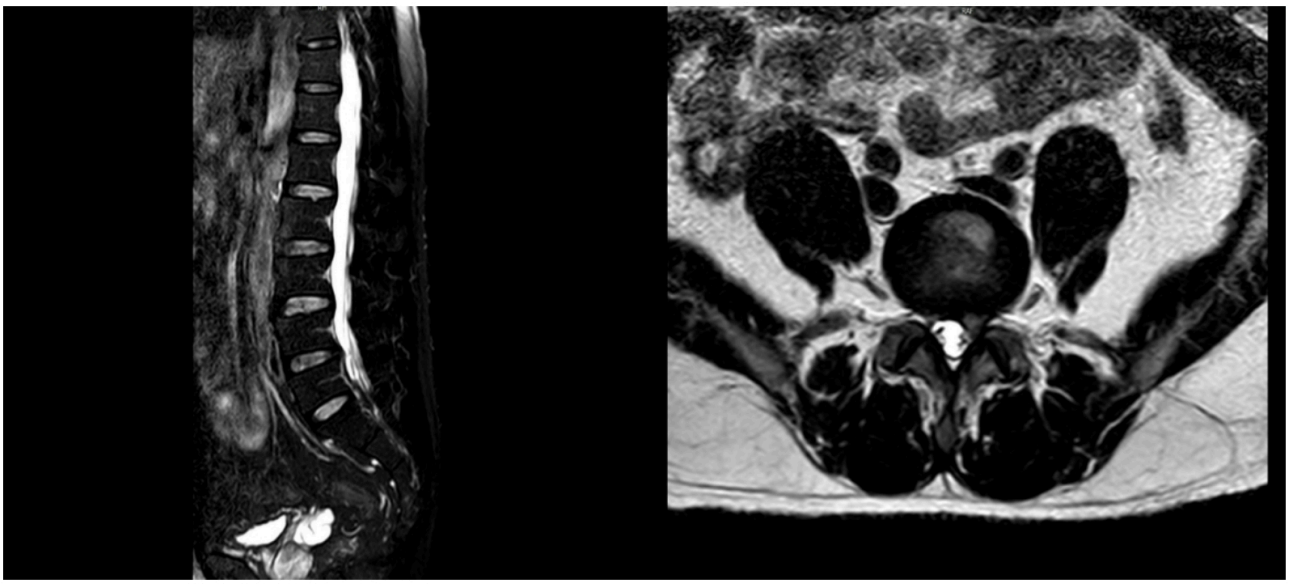


**Figure 1**

Preoperative lumbar spine

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