

Gelfoam-Induced Acute Cauda Equina Syndrome Following L3–L5 Decompression and Instrumented Fusion: A Case Report

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Case Report

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Abstract

Background: Absorbable gelatin sponge (Gelfoam) is a widely used hemostatic material in spinal surgery. Although generally safe, rare but devastating compressive neurological complications have been reported in intracranial, cervical, and thoracic procedures.

Case presentation: A 71-year-old Chinese male underwent L3-L5 laminectomy, bilateral L3/4 and L4/5 foraminotomy, pedicle screw fixation, and cage implantation with autogenous bone graft for symptomatic lumbar spinal stenosis. An intact sheet of Gelfoam was placed over the dura for hemostasis and left in situ at wound closure. Upon awakening from anesthesia two hours after surgery, the patient developed severe right lower extremity motor weakness, with muscle strength of 1/5 in the quadriceps, tibialis anterior, extensor hallucis longus, and extensor digitorum longus. On postoperative day 2, emergency lumbar spine CT revealed significant cauda equina compression at L4/5, prompting emergency surgical exploration and decompression. Intraoperatively, an expanded Gelfoam mass with adjacent clot was found circumferentially compressing the dural sac and cauda equina nerve roots from L3 to L5. The entire material was removed, and bilateral nerve root canals were further decompressed. Post-decompression MRI confirmed complete relief of canal compromise. Muscle strength improved substantially, with all affected groups recovering to 3/5 except the extensor hallucis longus (2/5). Epidural drainage output was only 20 mL and 35 mL on the first two days after the second surgery, supporting that the compression was predominantly Gelfoam mass effect rather than an expanding hematoma.

Conclusions: Placement of an intact, unprepared Gelfoam sheet in a closed epidural space can induce hyperacute cauda equina syndrome dominated by motor deficits within hours after surgery, a pattern distinctly different from the previously reported delayed onset of 10-14 days. Indwelling Foley catheterization may mask urinary retention, a classic autonomic warning sign, and delay early recognition. Strict adherence to standardized intraoperative protocols-soaking in thrombin, squeezing to expel air, using only small pieces, and removing residual material after hemostasis-is essential to prevent swelling-induced mass effect. For patients presenting with severe unilateral lower extremity weakness immediately after surgery and no evidence of hardware misplacement on CT, urgent lumbar MRI and prompt decompression are recommended to maximize neurological recovery.

Introduction

Absorbable gelatin sponge (Gelfoam, Pfizer Inc., Kalamazoo, MI, USA) was introduced in the 1940s and has been used for over 80 years as a biodegradable local hemostatic agent in neurosurgical and orthopedic spinal procedures^{1, 2}. Its porous gelatin matrix promotes platelet aggregation and endogenous clot formation, and it is gradually absorbed by hydrolysis over 2–6 weeks after implantation³. In addition, placing Gelfoam in the epidural space after laminectomy has been shown to reduce epidural scar formation and nerve root adhesions, a strategy well validated in lumbar surgery⁴. Despite its extensive use and favorable safety profile, several case reports have documented severe

iatrogenic neurological injuries associated with Gelfoam in intracranial, cervical, and thoracic surgeries, including intracranial abscess, epileptogenic gliosis, acute quadriplegia from cervical cord compression⁵, and delayed lumbar cauda equina syndrome occurring 10–13 days postoperatively⁶, and a delayed cervical myelopathy presenting 2–3 weeks after surgery. And swallowing difficulty caused by Gelfoam expansion in the retropharyngeal space after anterior cervical surgery⁷. Beyond Gelfoam-specific reports, retained hemostatic agents in general have been recognized as a cause of cauda equina syndrome, as they can create space-occupying lesions that lead to neurological compromise⁸. The classic pathophysiological hypothesis holds that Gelfoam in a closed epidural space slowly absorbs blood and tissue exudate, followed by a vigorous foreign-body inflammatory reaction that peaks around postoperative day 12, with fibrotic encapsulation exacerbating the mass effect.

Herein, we report a case of hyperacute cauda equina syndrome occurring immediately after L3-L5 decompression and fusion, caused by an intact Gelfoam sheet that rapidly expanded in a narrow spinal canal. This case is unique in that an indwelling Foley catheter obscured the typical autonomic warning sign of urinary retention, potentially delaying diagnosis. We detail the distinct pathomechanism of this ultra-early compression, highlight the critical role of postoperative MRI, and propose standardized intraoperative protocols to prevent this avoidable complication.

Patient's perspective

The patient, a 71-year-old male, described his experience as follows: " I'd been struggling with leg pain and trouble walking for over four years. Finally, the doctor said surgery was the only way to fix the root problem. The operation itself went smoothly, and when I woke up from anesthesia, I was feeling pretty good, thought I'd finally made it through the hard part. But just two hours after getting back to my room, my right leg suddenly gave out. I couldn't lift it, couldn't wiggle my toes, it was like it wasn't even mine anymore. My heart sank, and I panicked.

Later, the doctor explained that the little hemostatic sponge they'd placed in my spinal canal had absorbed fluid and swollen up, pressing against the nerves inside. To make things trickier, I still had a urinary catheter in, so the usual warning sign - 'can't pee' - never showed up. Luckily, the medical team reacted fast. They ran more tests, took me back into surgery that same night, and removed the swollen sponge. Right after that second surgery, I could already tell my leg was starting to regain strength. Now, six weeks later, I'm walking on my own without a cane. Honestly, I feel like I dodged a bullet. If there's one thing I want others to take away from my story: if your leg goes weak after surgery, don't brush it off and get it checked right away. Something that looks small can cause big trouble if you ignore it."

Written informed consent was obtained from the patient for publication of this perspective.

Case Report

A 71-year-old Han Chinese male presented with a 4-year history of neurogenic claudication, bilateral radicular pain, and progressive gait impairment that had been refractory to conservative management. Neurological examination revealed bilaterally diminished knee and ankle reflexes, mild preoperative right L4/L5 myotomal weakness (tibialis anterior and extensor hallucis longus strength 4/5), and intermittent saddle area hypesthesia. Lumbar MRI (Figure 1) demonstrated severe central canal stenosis at L3/4 and L4/5 with circumferential hypertrophy of the ligamentum flavum compressing the cauda equina. Conservative treatments, including physical therapy, transforaminal epidural steroid injections, and oral neuropathic agents, had failed to provide sustained relief, establishing surgical indications. Preoperative laboratory tests, coagulation profile, and anesthesia risk assessment were unremarkable, and there were no contraindications to MRI.

The initial surgery was performed on the third day of hospitalization. L3-L5 decompression and instrumented fusion was carried out as follows: After general anesthesia, the patient was placed prone on a radiolucent spinal operating table. All osseous work and instrumentation placement were performed under fluoroscopic guidance. A standard posterior midline approach was used with subperiosteal dissection to expose the spinous processes, laminae, and transverse processes of L3-L5. Complete laminectomy of L3 and L4 and partial laminectomy of L5 were performed, followed by bilateral medial facetectomies and foraminal decompression at L3/4 and L4/5 with thorough release of the traversing and exiting nerve roots. Under fluoroscopy, pedicle screws were inserted bilaterally at L3, L4, and L5, with satisfactory trajectories and no breach of the pedicle walls. The L3/4 and L4/5 intervertebral discs and endplate cartilage were meticulously removed, cages were implanted for interbody fusion, and titanium rods with a transverse connector were placed to complete the posterior rigid fixation construct. Before wound closure, a Foley catheter was placed for perioperative bladder management.

After instrumentation, diffuse capillary oozing was noted on the dural surface. The surgeon fashioned a sheet of Gelfoam cut to the size of the bony defect and placed it directly over the dura without prior soaking in thrombin or squeezing to expel air from the pores. The surgical field was irrigated with 5 L of warm normal saline, and the wound edges were dabbed with gauze. Decortication of the transverse processes was performed with a high-speed burr, and autogenous bone chips obtained from the resected laminae were packed into the posterolateral fusion bed. The paraspinal muscles, subcutaneous tissue, and skin were closed in layers, and an epidural negative-pressure drain was placed. Fluoroscopy at the end of the procedure confirmed satisfactory hardware position with no canal encroachment, and the patient was transferred to the post-anesthesia care unit in stable condition.

Acute Neurological Deterioration on the Day of Surgery—Ninety minutes after emergence from anesthesia, the patient reported persistent burning dysesthesia in the right lower extremity and was unable to actively flex or extend the hip, knee, ankle, great toe, or other toes. Detailed neurological examination revealed:

1. Motor function: Muscle strength in the right lower extremity key myotomes was 1/5 in the quadriceps, tibialis anterior, extensor hallucis longus, and extensor digitorum longus. Left lower extremity strength

was 4/5 in all groups, without significant decline.

2. Sensory function: Hypesthesia was noted in the saddle area (S2–S5 dermatomes) and over the right L3-S1 distribution. Left lower extremity sensation was normal.

3. Autonomic function: The indwelling Foley catheter precluded assessment of urinary retention as a sign of cauda equina syndrome; anal resting tone was markedly decreased.

4. Reflexes: Right knee and ankle reflexes were absent; left deep tendon reflexes were normally elicited.

Emergency lumbar CT(Figure 2a and Figure 2b) revealed all screws within the pedicles, no retropulsed bone fragments, and no hardware malposition, but demonstrated a heterogeneous intraspinal mass of indeterminate density. Given the acute motor-predominant cauda equina syndrome, an urgent lumbar MRI was obtained on postoperative day 2 to define the soft-tissue compressive lesion.

Postoperative Day 2 MRI Findings(Figure 3a and Figure 3b)–Lumbar MRI showed a large, heterogeneous soft-tissue mass in the spinal canal at L4/5, with circumferential obliteration of the cerebrospinal fluid space, extending from L3 to L5, producing severe compression of the cauda equina nerve roots. Emergency surgical exploration and decompression were immediately arranged.

Emergency Secondary Decompression

The patient was returned to the operating room under general anesthesia. Through the original midline incision, the paraspinal muscles were dissected to expose the L3-L5 posterior elements. Upon opening the epidural space, an expanded gelatinous mass was identified in the central spinal canal from L3 to L5, tightly encircling the dural sac and cauda equina nerve roots. This mass corresponded to the intact Gelfoam sheet placed during the initial surgery, surrounded by linear clots. The Gelfoam matrix had absorbed irrigation fluid and a small amount of dural oozing, leading to rapid expansion and solidification within the non-compliant spinal canal.

The entire Gelfoam mass and clot were carefully removed en bloc, and bilateral L3/4 and L4/5 foraminal decompression was further extended. The screws and rods were confirmed to be in satisfactory position and were retained. The epidural space was thoroughly irrigated with antibiotic-containing normal saline, no hemostatic material was left in the canal at the end of the procedure, and the wound was closed in layers with a new epidural negative-pressure drain.

Postoperative Anticoagulation and Drainage Management

Oral rivaroxaban (10 mg once daily) was initiated on the first postoperative day for venous thromboembolism prophylaxis, per our institutional protocol for spinal fusion patients. Following the emergency decompression, rivaroxaban was temporarily withheld for 24 hours to minimize the risk of rebleeding in the freshly decompressed epidural space. Epidural negative-pressure drainage output was 20 mL of serosanguineous fluid on the first day after the second surgery and 35 mL on the second day,

with no abrupt increase or fresh bleeding noted. The drain was removed after confirming satisfactory output reduction. On the third day after the second procedure, the patient was mobilized out of bed with a rigid thoracolumbar orthosis, reporting tolerable incisional pain and no exacerbation of right lower extremity weakness. Early mobilization under brace protection was well tolerated and facilitated enhanced recovery without compromising neurological status.

Post-Decompression Imaging and Clinical Recovery

MRI (Figure 4a and Figure 4b) performed within 24 hours after the second surgery demonstrated complete disappearance of the epidural soft-tissue mass and restoration of continuous cerebrospinal fluid signal around the cauda equina, with total resolution of neural compression from L3 to L5.

Neurological reassessment 12 hours after emergency decompression showed that the previously 1/5 strength in the right quadriceps, tibialis anterior, and extensor digitorum longus had improved to 3/5, with only the extensor hallucis longus remaining mildly impaired at 2/5. Saddle area hypesthesia had substantially improved, and anal resting tone had essentially normalized. By postoperative day 3, the patient reported marked reduction in right lower extremity burning pain, and motor strength remained stable at the above levels. At the 6-week outpatient follow-up, muscle strength had further improved, with extensor hallucis longus strength reaching 4/5, and the patient was able to walk independently without a cane, with no persistent neurogenic bladder or bowel dysfunction.

Discussion and Conclusions

1.Pathomechanistic Comparison: Hyperacute vs. Delayed Gelfoam-Related Cauda Equina Syndrome

Previous reports of Gelfoam-induced compressive neuropathy have consistently followed a subacute course, with neurological deterioration occurring 10-14 days postoperatively. The pathophysiology involves two phases: an early phase (within 72 hours) during which the sponge slowly absorbs exudate and blood, and a late phase characterized by an intense foreign-body inflammatory response peaking around postoperative day 12, with dense fibrous encapsulation adding to the mass effect. This delayed fibro-inflammatory mechanism was corroborated by Epstein et al.⁹, who reported a cervical case in which the Gelfoam was found markedly engorged and densely adherent to the underlying dura on revision surgery at 2-3 weeks, with no hematoma identified.

The phenomenon of Gelfoam expansion within a confined space leading to neural compression has been documented in other spinal regions as well. Nallakumarasamy et al. reported a case of early-onset quadriparesis following cervical decompression, in which the gelatin sponge caused mechanical cord compression through postoperative swelling and mass effect in the closed epidural cavity¹⁰. Our case represents a hyperacute phenotype, with cauda equina syndrome developing within hours of wound closure. Several intraoperative errors eliminated the buffer period seen in delayed cases:

Use of an intact sheet: A large, uncut piece of Gelfoam placed in the closed central spinal canal forms a continuous rigid matrix that expands to its maximal volume. In contrast, small pieces, when swollen, allow partial collapse of the matrix, substantially reducing the mass effect.

-Failure to expel trapped air: The dry, porous honeycomb structure of Gelfoam, when directly applied to a moist dura, traps significant amounts of air. The air spaces create additional volume; upon contact with irrigation saline and dural oozing, osmotic pressure drives rapid fluid influx, causing irreversible, rapid expansion within the non-compliant lumbar canal.

In delayed cases, fibrous scar proliferation is the primary damaging factor, whereas in our patient the acute neurological deficit resulted purely from physical expansion of the Gelfoam matrix, with histopathological findings showing only mild early inflammation. This mechanistic difference has direct clinical implications: delayed cases involve both inflammatory and mechanical insults, whereas immediate postoperative motor decline should first raise suspicion for an expanding hemostatic material rather than late scar or hematoma.

2. Unique Confounding Factor: Indwelling Foley Catheter Masking Autonomic Signs

The key feature distinguishing this case from previous reports is the intraoperative placement of a Foley catheter, which completely masked urinary retention—a cardinal early warning sign of cauda equina syndrome. Classic diagnostic criteria emphasize autonomic dysfunction (urinary retention, saddle hypesthesia, and anal sphincter weakness) as sentinel symptoms¹¹, but an indwelling catheter prevents immediate postoperative assessment of spontaneous voiding. Clinicians may focus on motor and sensory deficits and overlook the risk of cauda equina syndrome, delaying appropriate imaging. This case highlights that even in the absence of urinary retention, severe unilateral lower extremity weakness alone warrants urgent imaging to rule out spinal canal compression.

3. Anatomical and Biomechanical Vulnerability at L3-L5, with Maximal Compression at L4/5

The L3-L5 segment carries a higher risk of Gelfoam-induced compression compared with upper lumbar or thoracolumbar levels, due to specific anatomical and biomechanical features:

The midsagittal diameter and cross-sectional area of the spinal canal at L4/5 are significantly smaller than at L1-L3, with a mean dural sac cross-sectional area of approximately 140-160 mm² at L4/5 compared with 180-220 mm² at L1-L2¹². This anatomical narrowing leaves minimal reserve space for any occupying mass. The L4/5 level contains a dense bundle of cauda equina nerve roots and has a narrow cerebrospinal fluid buffer zone. Even minor expansion of a foreign material can obliterate the fluid space and directly entrap nerve roots. In our patient, the greatest compression was precisely at L4/5, corresponding to severe unilateral weakness in the L4 and L5 myotomes (quadriceps, tibialis anterior, and toe extensors).

The L3-L5 region bears the highest axial load in the lumbar spine; soft-tissue swelling and an occupying mass further exacerbate circumferential narrowing¹³.

Two-level laminectomy combined with bilateral facetectomies removes the posterior osseous buttress, creating a potential dead space that allows an intact sheet of Gelfoam to adhere to the anterior dura without outward constraint by bone.

Surgeons often underestimate the limited canal reserve after multilevel decompression at the mid-lumbar spine¹⁴. Compared with single-level upper lumbar surgery, stricter limitations on Gelfoam use are necessary in L3-L5 multilevel fusion.

4. Updated Diagnostic Algorithm for Acute Postoperative Cauda Equina Syndrome with Pure Motor Deficit

For patients who develop severe unilateral lower extremity weakness after lumbar fusion, with autonomic symptoms obscured by an indwelling catheter, we propose a tiered diagnostic approach:

1. Priority 1 - Rule out hardware-related compression: Pedicle screw breach, medial rod impingement, retropulsed bone or cartilage fragments, and excessive facet screw overhang should be excluded. Emergency CT rapidly identifies bony or metallic causes, and was negative in our case.
2. Priority 2 – Urgent lumbar MRI: MRI should be obtained as early as possible to distinguish fluid-absorbed Gelfoam, acute epidural hematoma, abscess, or seroma, and to localize the level of maximal compression (L4/5 in this patient).
3. Priority 3 – Surgical intervention: Once MRI confirms an epidural soft-tissue mass consistent with retained Gelfoam compression, emergency decompression should be performed.

The optimal therapeutic window for cauda equina mechanical compression is generally considered to be within 48 hours of symptom onset, although many guidelines advocate decompression as early as possible; recent evidence suggests that outcomes within 24 hours are comparable to those achieved within 24-48 hours¹⁵. This case confirms that MRI is indispensable for confirming biomaterial-related compression before a second surgery, particularly when autonomic signs are masked by a catheter.

5. Standardized Intraoperative Protocol to Prevent Compression Complications

Based on the hyperacute mechanism observed in this case and the existing spinal surgery literature, we propose an evidence-based, high-safety protocol for epidural hemostasis in multilevel lumbar decompression and fusion, suitable for widespread adoption:

1. Preoperative sponge preparation: All Gelfoam pieces should be thoroughly soaked in thrombin solution and vigorously squeezed repeatedly to expel trapped air from the porous matrix. Dry or semi-moist sponges must never be placed in the epidural space.

2. Mandatory subdivision: Intact sheets must not be used in the spinal canal. Only small pieces of 0.5-1 cm should be applied locally to bleeding points. Large continuous sheets spanning the bony defect are prohibited.

3. Removal after hemostasis: Once capillary oozing is controlled and before wound irrigation and closure, all residual Gelfoam must be removed from the spinal canal. Deliberate retention of Gelfoam as an anti-adhesion barrier is contraindicated in L3-L5 multilevel decompression because of limited canal reserve.

4. Optimized drainage: Routine placement of an epidural negative-pressure drain after L3-L5 multilevel fusion helps evacuate excess irrigation fluid and exudate¹⁶, eliminating the fluid source for sponge expansion.

5. Avoid over-compression of dry sponge: Forcible compression of dry Gelfoam creates a dense matrix that resists degradation after fluid absorption and maintains a solid mass over time.

6. Study Limitations and Future Directions

Librianto et al. elucidated a two-stage mechanism of Gelfoam expansion: the sponge initially swells upon blood absorption, and as it begins to liquefy, osmotic pressure draws additional fluid into the matrix, causing progressive enlargement that compresses neural elements within the enclosed epidural space¹⁷. Oxidized regenerated cellulose (e.g., Surgicel) has been associated with similar foreign-body reactions and compressive neuropathies in neurosurgical applications, whereas fibrin sealants carry a risk of expansion-related compression when used in the spinal canal¹⁸. Controlled trials comparing low-swelling alternatives such as oxidized regenerated cellulose or fibrin sealants in high-risk multilevel L3-L5 decompressions would help identify safer biomaterials. This is a single retrospective case report with inherent limitations. Future prospective cohort studies stratified by operative level, sponge size, preparation technique, and use of postoperative catheterization are needed to establish the population-based risk of Gelfoam-induced neural compression. In addition, prospective audit studies could assess whether routine postoperative MRI shortens the time to decompression and improves motor outcomes in catheterized patients with lower extremity weakness.

7. Clinical Take-Home Messages

1. Placement of an intact, unprepared Gelfoam sheet in the L3-L5 epidural space can induce hyperacute cauda equina syndrome dominated by motor deficits within hours after surgery, with maximal compression localized to the narrow L4/5 segment—a rapidly progressive complication rarely reported previously.

2. An indwelling Foley catheter may mask urinary retention, a classic autonomic warning sign of cauda equina syndrome. In the presence of severe unilateral lower extremity weakness after surgery, urgent MRI should be performed to evaluate spinal canal compression.

3. Patients undergoing multilevel mid-lumbar decompression and fusion have limited canal reserve and high axial load at L4/5, constituting a high-risk population for Gelfoam-mediated neural compression.
4. The triad of failing to soak/squeeze Gelfoam, using a large intact sheet, and omitting an epidural drain synergistically promotes rapid fluid absorption and acute mass effect.
5. If CT excludes hardware-related compression, patients with immediate postoperative severe lower extremity weakness should undergo emergency lumbar MRI and, if retained Gelfoam compression is confirmed, urgent surgical decompression to maximize the probability of neurological recovery.
6. Strict adherence to standardized procedures-division into small pieces, pre-soaking with air expulsion, removal after hemostasis, and routine epidural drainage-can completely eliminate this preventable iatrogenic complication.

This case demonstrates that an intact, unprepared Gelfoam sponge left in the epidural space after L3-L5 decompression and fusion can cause hyperacute cauda equina syndrome with severe motor deficits within hours. The presence of an indwelling Foley catheter obscured urinary retention, potentially delaying diagnosis. Early recognition, urgent MRI, and prompt surgical decompression are critical for favorable neurological recovery. Strict adherence to a standardized protocol for Gelfoam use, routine epidural drainage, and heightened vigilance in patients receiving prophylactic anticoagulation are essential to prevent this avoidable complication.

Declarations

Ethics approval and consent to participate

Not applicable

Consent for publication

Written informed consent was obtained from the patient for publication of this case report and any accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of this journal.

Availability of data and materials

The datasets used and/or analysed during the current study are available from the corresponding author on reasonable request.

Competing interests

The Authors declare that they have no competing interests.

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Authors' contributions

H.W. and Z.Y. contributed to the conception and design of the study, data collection, and drafting of the manuscript. H.W. performed the surgical procedures and revised the manuscript critically for important intellectual content. X.J. contributed to patient management, data interpretation, and final approval of the version to be published. All authors read and approved the final manuscript.

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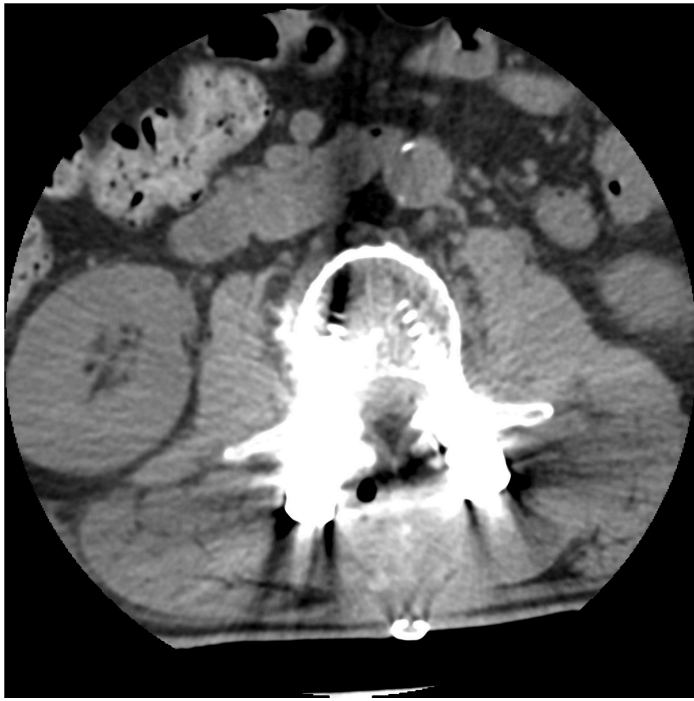
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Figures



Figure 1

Preoperative lumbar MRI (sagittal view, T2-weighted sequence) demonstrating severe central canal stenosis at the L4/5 level, with circumferential hypertrophy of the ligamentum flavum causing significant compression of the cauda equina



a



b

Figure 2

Emergency postoperative lumbar CT (axial view, bone window) performed on postoperative day 2. (a) and (b) reveal a heterogeneous intraspinal mass of indeterminate density at the L4/5 level, with no evidence of pedicle screw breach or retropulsed bone fragments



a



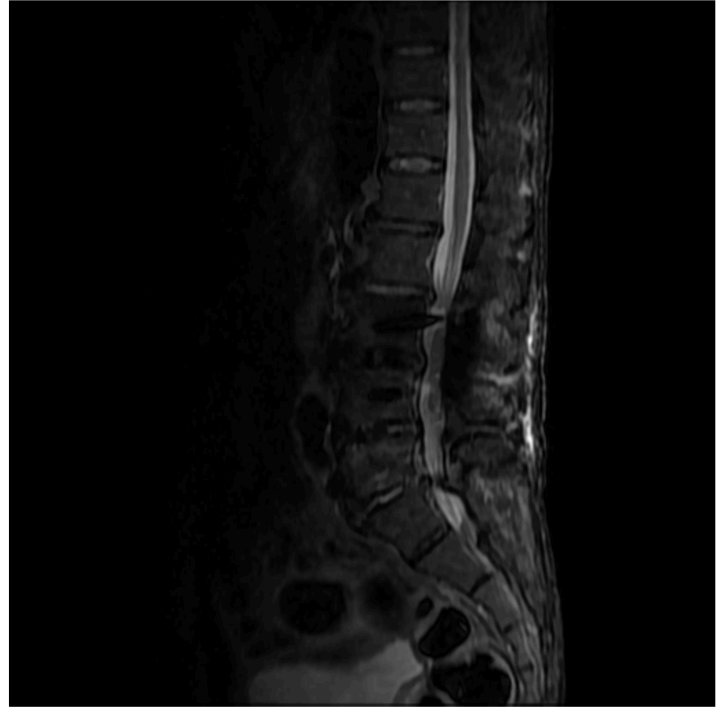
b

Figure 3

Urgent postoperative lumbar MRI (axial view, T2-weighted sequence) obtained on postoperative day 2. (a) and (b) demonstrate severe compression and crowding of the cauda equina nerve roots extending from L3 to L5, with near-complete obliteration of the cerebrospinal fluid space at the L4/5 level. No discrete hyperintense or enhancing mass is identified, as Gelfoam itself does not produce intrinsic MRI signal;



a



b

Figure 4

Post-decompression lumbar MRI (axial view, T2-weighted sequence) performed within 24 hours after the emergency surgical revision. (a) and (b) confirm the complete resolution of the epidural soft-tissue mass, with restoration of the continuous cerebrospinal fluid signal around the cauda equina and total relief of neural compression at the L3-L5 levels