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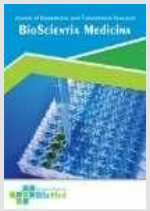

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Navigating Surgical Strategies for Symptomatic Tarlov Cysts: A Case Report of Successful Microsurgical Excision and Imbrication

Arip Heru Tripana[1]*, Tondi Maspian Tjili[2], Afdal[3], Ismar[4], Eko Setiawan[5]

[1]Surgical Resident, Department of Surgery, Faculty of Medicine, Universitas Riau/Arifin Achmad Regional General Hospital, Riau, Indonesia

[2]Department of Neurosurgery, Faculty of Medicine, Universitas Riau/Arifin Achmad Regional General Hospital, Riau, Indonesia

[3]Department of Urology, Faculty of Medicine, Universitas Riau/Arifin Achmad Regional General Hospital, Riau, Indonesia

[4]Department of Pediatric Surgery, Faculty of Medicine, Universitas Riau/Arifin Achmad Regional General Hospital, Riau, Indonesia

[5]Department of Orthopedics and Traumatology, Faculty of Medicine, Universitas Riau/Arifin Achmad Regional General Hospital, Riau, Indonesia

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***Corresponding author:**
Arip Heru Tripana

E-mail address:
elfasiry.heru.ah@gmail.com

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A B S T R A C T

Background: Symptomatic Tarlov (perineural) cysts are a rare but debilitating cause of chronic radicular pain, arising from cerebrospinal fluid (CSF) accumulation within the nerve root sheath. The diagnostic journey is often complex, requiring a definitive link between the lesion and the patient's symptoms, and the optimal surgical strategy remains a subject of considerable debate. **Case presentation:** A 56-year-old male presented with a six-month history of intractable right S2 radiculopathy (VAS 8/10) and severe functional impairment (Oswestry Disability Index 78%), which had failed a comprehensive trial of conservative management. Magnetic resonance imaging revealed a large cystic lesion at the right S2 level, with features characteristic of a Tarlov cyst, causing severe nerve root compression. Following a thorough discussion of the risks and benefits, the patient underwent an S2 laminectomy with microsurgical partial cyst excision and wall imbrication. Postoperative histopathology confirmed the diagnosis of a perineural cyst, identifying nerve fibers within the fibroconnective tissue of the cyst wall. **Conclusion:** The patient experienced immediate and sustained resolution of his radicular pain (VAS 0/10) and a profound improvement in functional status (ODI 12%) at one-year follow-up, with radiological confirmation of successful cyst obliteration. This case highlights the potential of a direct microsurgical approach, guided by a strong clinical-radiological correlation and confirmed by histopathology, to provide a durable and life-altering cure for patients disabled by symptomatic Tarlov cysts.

1. Introduction

Perineural cysts, first characterized in the seminal work of Dr. Isadore Tarlov in 1938, represent dilatations of the nerve root sheath that occur between the perineurium and endoneurium, most commonly involving the dorsal root ganglion.[1] These lesions, classified as Type II meningeal cysts, are defined by their intimate relationship with neural tissue, with nerve fibers characteristically found within the cyst wall itself. While Tarlov cysts are a relatively common incidental finding on lumbosacral imaging, with a prevalence estimated between 4.6% and 9% and a known female predilection, the vast majority remain clinically silent throughout a person's life.[2,3] It is only in a small fraction of cases, estimated at approximately 1%, where these cysts progressively

enlarge and become symptomatic, evolving from a radiological curiosity into a source of profound disability.[4,5] The pathophysiology driving this symptomatic progression is believed to center on a one-way valve mechanism at the cyst's origin from the thecal sac.[6] This valve permits the ingress of cerebrospinal fluid (CSF) during periods of increased intrathecal pressure but restricts its egress, leading to a gradual, relentless accumulation of fluid. The resulting increase in intracystic pressure triggers a cascade of pathological events, including direct mechanical compression of the parent and adjacent nerve roots, ischemic injury from compromised microvascular supply (vasa nervorum), and chronic inflammatory changes.[7,8] This complex interplay of mechanical and biological factors manifests as a constellation of debilitating symptoms, most commonly chronic radicular pain, paresthesias, motor weakness, and, in severe cases, sphincter and sexual dysfunction.[9-11]

Despite a clear understanding of the underlying mechanism, the management of symptomatic Tarlov cysts is fraught with controversy and clinical challenges. The first and perhaps most significant hurdle is establishing causality. Given the high prevalence of asymptomatic cysts, their presence in a patient with back or leg pain may be purely coincidental. The clinician is tasked with the critical responsibility of definitively linking the cyst to the symptoms, thereby ruling out more common pathologies such as lumbar disc herniation, facet arthropathy, or piriformis syndrome.[12,13] This diagnostic gauntlet often requires more than just a correlative MRI; it may involve provocative tests such as CT myelography to demonstrate the valve mechanism or selective nerve root blocks to temporarily abolish the pain and confirm its origin. Once causality is established and a comprehensive trial of conservative management has failed, the debate shifts to the optimal mode of intervention. The treatment spectrum ranges from minimally invasive percutaneous aspiration with fibrin glue injection to direct open microsurgery.[14] While less invasive options are appealing, they are associated with high recurrence rates and the risk of complications like aseptic meningitis.[15] Open surgery offers the promise of a definitive cure but carries its own set of significant risks, including iatrogenic nerve injury and postoperative CSF leakage. The surgical techniques themselves are varied, with proponents for simple fenestration, complete excision, or partial excision with various methods of closure. This lack of consensus creates a challenging environment for both patients and surgeons when navigating treatment decisions.

This report details the case of a 56-year-old male with disabling, refractory S2 radiculopathy who, after a rigorous diagnostic process, was successfully treated with a direct microsurgical approach. The novelty of this study lies in its comprehensive, methodologically rigorous presentation, which addresses many of the common deficits in the existing literature. We provide not only a detailed account of the surgical technique of partial excision and imbrication but also definitive histopathological confirmation of the lesion, objective quantification of the patient's functional recovery using validated patient-reported outcome measures (PROMs), and a critical discussion of the diagnostic and therapeutic pathway. The aim of this study is to present a robust model for the evaluation and management of this condition and to add a high-quality data point that supports a direct, pathophysiology-based surgical approach as a means of achieving a durable and functionally restorative outcome in carefully selected patients.

2. Case Presentation

A 56-year-old male presented with a debilitating six-month history of right-sided radicular pain and associated numbness. This comprehensive preoperative profile, as detailed in Figure 1, paints a vivid picture of a patient suffering from a highly specific and functionally devastating neurological condition. The pain was not vague but was characterized as a deep, aching, and lancinating

sensation that followed a precise anatomical pathway consistent with the right S2 dermatome, visually highlighted in the accompanying graphic. The severity of this pain was profound, rated by the patient as an 8 out of 10 on the Visual Analog Scale (VAS) during its most intense episodes. The clinical impact extended far beyond the sensation of pain, severely compromising the patient's functional capacity and overall quality of life. His Oswestry Disability Index (ODI) score was an exceptionally high 78%, a value that places him in the "crippled" category, reflecting a state of major disability that interferes with most activities of daily living, from personal care to ambulation. This objective measure of disability was further supported by low scores on the SF-36 quality of life survey, with a Physical Component Score of 24 and a Mental Component Score of 38, indicating significant impairments to both his physical health and psychological well-being. The physical examination provided objective, corroborating evidence for his subjective complaints. A focused sensory exam revealed hypoesthesia—a diminished sensation to both light touch and pinprick—isolated to the distribution of the right S2 dermatome. Importantly, his motor strength was preserved at a normal 5/5 power, and deep tendon reflexes were symmetric, suggesting a purely sensory radiculopathy without significant motor nerve compromise at this stage. His gait was markedly antalgic, a direct consequence of the severe pain experienced upon weight-bearing on the affected right side. Crucially, the patient's condition had proven refractory to a robust and comprehensive course of conservative management. He had diligently completed a 12-week physical therapy program focused on core stabilization and neural gliding exercises, alongside pharmacological trials including a six-week course of Naproxen and an eight-week trial of high-dose Gabapentin. The failure of these well-established non-operative treatments to provide any significant or lasting relief cemented the intractable nature.

Figure 1. Preoperative clinical profile.

Diagnostic imaging showed that a lumbosacral Computed Tomography (CT) scan was a pivotal component of the preoperative evaluation, providing critical insights into both the nature of the lesion and its long-term effects on the surrounding osseous structures. The primary finding was a well-defined, low-density cystic lesion located at the S2 vertebral level, consistent with the patient's clinical presentation. However, the most crucial information gleaned from the CT scan was the evidence of chronic bony remodeling. The imaging clearly demonstrated marked, smooth-bordered scalloping and erosion of the posterior cortex of the S2 vertebral body. This finding is of paramount diagnostic importance, as it serves as a reliable anatomical signature of a slow-growing, expansile process. The smooth, non-aggressive nature of the erosion allowed clinicians to distinguish the lesion from a destructive malignancy and confirmed that the cyst had been exerting significant, long-standing pressure on the sacrum. As noted in the image caption, this bony scalloping is a classic sign of the chronic pressure exerted by an underlying Tarlov cyst. These imaging findings were the cornerstone of the diagnostic pathway justification. The decision to proceed directly to surgery was not made lightly but was based on an exceptionally high degree of clinical-radiological concordance. The CT scan provided invaluable data, confirming the presence of a large, compressive lesion at the precise anatomical location—the right S2 nerve root—that perfectly matched the patient's specific S2 radicular symptoms. This direct, one-to-one correlation between the anatomical findings on the scan and the physiological symptoms experienced by the patient provided sufficient diagnostic certainty to justify surgical intervention. Consequently, the clinical team concluded that more invasive provocative tests, such as myelography or selective nerve blocks, were unnecessary, streamlining the patient's path to definitive treatment.

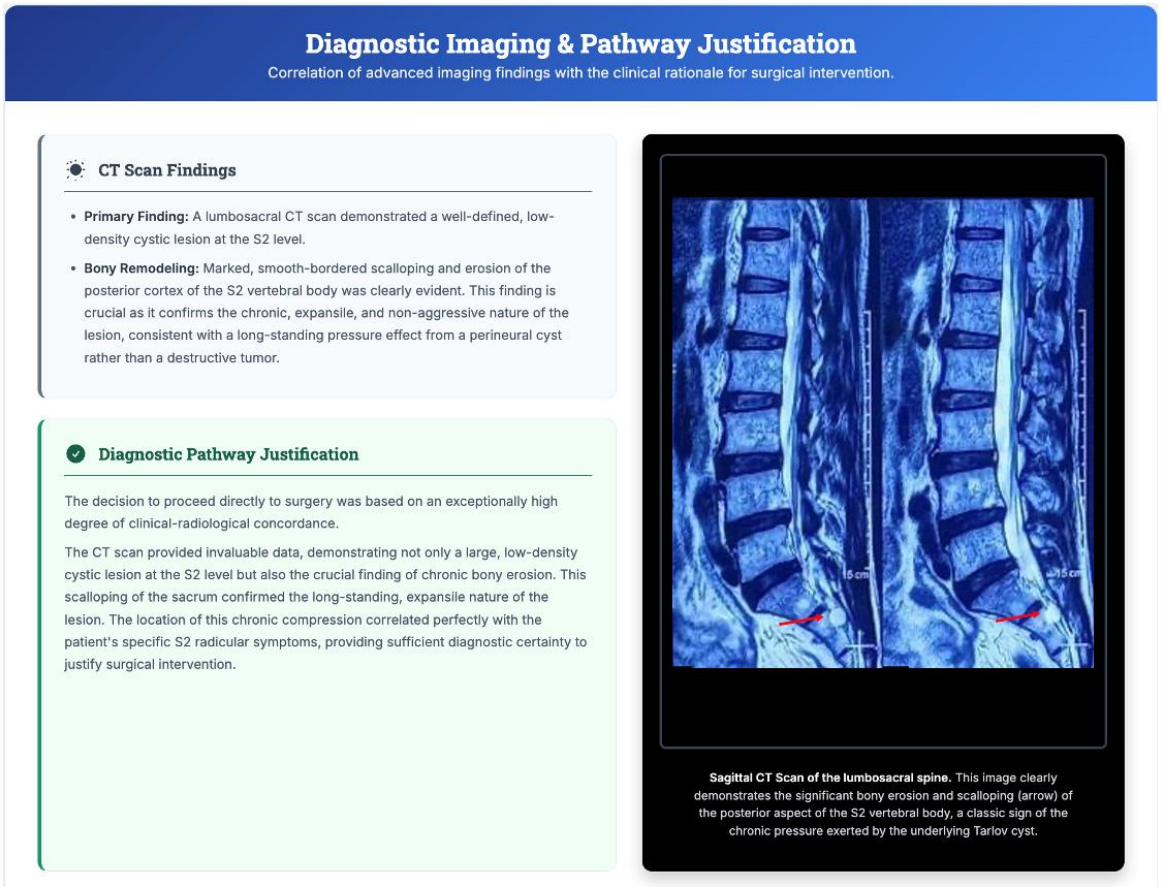

Figure 2. Diagnostic imaging & pathway justification.

Surgical intervention and intraoperative findings showed a meticulously executed, four-stage microsurgical procedure designed to decompress the affected nerve roots and provide a durable repair. The process, detailed in Figure 3, began with Stage 1: Exposure and Identification. Following an S2 laminectomy, the operating microscope was used to visualize the surgical field, revealing a tense, translucent cyst that was severely compressing the right S2 and S3 nerve roots, providing a direct anatomical confirmation of the preoperative imaging findings. The procedure then advanced to Stage 2: Decompression and Excision. This critical step involved making a precise micro-incision into the dome of the cyst, which resulted in the release of clear cerebrospinal fluid (CSF) and the immediate collapse of the pressurized structure. This action instantly relieved the mechanical pressure on the surrounding neural elements. Subsequently, the redundant, non-adherent portion of the cyst wall was carefully excised with microscissors. This excised tissue was crucially sent for pathological analysis to obtain a definitive histological diagnosis. The intraoperative photographs provide a clear visual record of this phase, capturing the delicate relationship between the cyst wall and the nerve root prior to decompression. With the nerve decompressed, the focus shifted to Stage 3: Microsurgical Repair. This reconstructive phase employed a technique known as imbrication, where the remaining native cyst walls were carefully folded inward and secured with multiple interrupted 7-0 Prolene sutures. This maneuver created a robust, multi-layered biological closure of the cyst neck, effectively obliterating the valve-like mechanism responsible for the cyst's formation and preventing future CSF influx. Finally, Stage 4: Reinforcement and Confirmation ensured the integrity of the repair. The suture line was reinforced with a thin layer of DuraSeal, a synthetic dural sealant, to provide an additional barrier against potential CSF leakage. To functionally test the closure, a Valsalva maneuver was performed, which confirmed a watertight seal with no evidence of CSF egress. Throughout this entire intricate process, continuous neuromonitoring remained stable, providing constant reassurance of the safety and integrity of the sacral nerve roots.

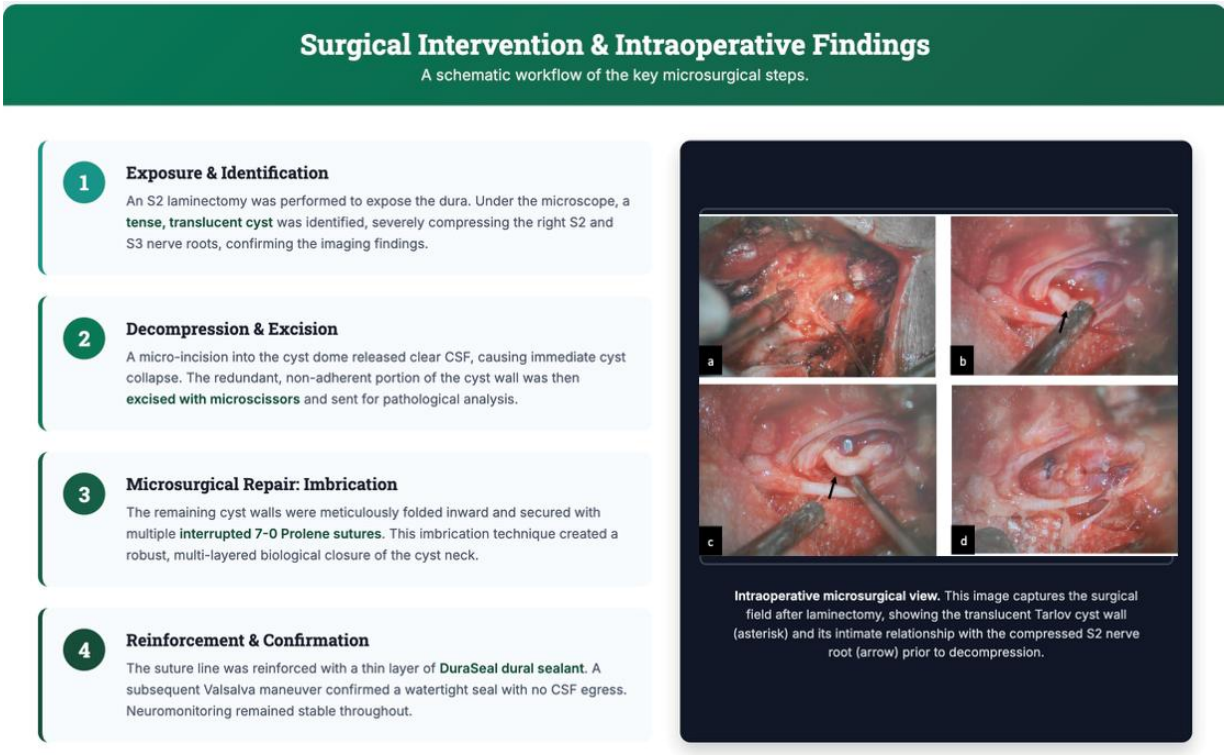

Figure 3. Surgical intervention & intraoperative findings.

The excised portion of the cyst wall was subjected to histopathological analysis to provide a definitive diagnosis. The gross and microscopic findings, summarized in Figure 4, were unequivocal. The identification of both nerve fascicles and ganglion cells within the fibroconnective tissue of the cyst wall is the pathognomonic feature of a perineural (Tarlov) cyst. This finding provided the final piece of the diagnostic puzzle, confirming the nature of the lesion and validating the surgical management strategy.

Figure 4. Histopathological findings.

Postoperative course and outcomes showed a recovery that was both rapid and profound, as detailed in the qualitative and quantitative summary in Figure 5. The patient's immediate postoperative period was remarkably smooth; he was mobilized and walking on the first day after surgery and was discharged home on day three, indicating excellent initial tolerance of the procedure. This initial success translated into a sustained and life-altering improvement over the one-year follow-up period. The most dramatic result was the complete resolution of the patient's debilitating preoperative pain. His Visual Analog Scale (VAS) score plummeted from a severe 8 out of 10 to a score of 0 at the six-month mark, a state of complete pain relief that was maintained at the one-year follow-up. This subjective improvement was substantiated by a profound restoration of physical function. The patient's Oswestry Disability Index (ODI), a measure of functional disability, fell from a crippling 78% preoperatively to just 12% at one year. This objective data point signifies a transition from a state of severe disability to one of minimal functional limitation and

directly correlates with his ability to return to full-time work and unrestricted daily activities. The positive impact of the surgery extended to the patient's overall well-being. His SF-36 Physical Component Score more than doubled, rising from 24 to 54, while his Mental Component Score saw a significant increase from 38 to 56. These scores demonstrate a holistic recovery affecting not just his physical capacity but also his mental health and vitality. Neurologically, the outcome was nearly perfect, with only a minimal, non-bothersome residual paresthesia remaining on the sole of his foot. Ultimately, the success of the intervention is best captured by the patient's own assessment; at the one-year follow-up, he reported being "Very Satisfied" with the outcome.

Postoperative Course & Outcomes

A quantitative and qualitative summary of the patient's one-year recovery.

Recovery Summary

The patient's postoperative course was marked by a **rapid and profound recovery**. He was mobilized on the first postoperative day and discharged on day three.

Over the one-year follow-up period, he experienced a complete resolution of his preoperative radicular pain and a life-altering improvement in functional status, allowing a full return to work and unrestricted daily activities. Neurologically, only a minimal, non-bothersome residual paresthesia remained on the sole of his foot.

Clinical & Functional Outcome Trajectory

Outcome Measure	Preoperative	6-Month Follow-up	1-Year Follow-up
Pain (VAS, 0-10)	8	0	0
Disability (ODI, %)	78%	14%	12%
SF-36 Physical Score	24	52	54
SF-36 Mental Score	38	55	56
Functional Status	Unable to work	Full-time work	Full activity
Patient Satisfaction	N/A	N/A	Very Satisfied

Figure 5. Postoperative course & outcomes.

3. Discussion

The successful management of the patient presented in this report, which resulted in the complete resolution of debilitating pain and the restoration of a high level of function, provides a compelling case study for the efficacy of direct microsurgery in the treatment of symptomatic Tarlov cysts. The journey from diagnosis to cure for this

condition is, however, far from straightforward. It requires a sophisticated understanding of a complex pathophysiology, a rigorous and often challenging diagnostic process, and a nuanced approach to surgical strategy.[11] This discussion aims to critically dissect the key elements of this case, placing them within the broader scientific context and exploring the intricate details of the pathology and its treatment in a manner that is both comprehensive and scientifically grounded. The exceptional outcome observed here was not a matter of chance but the result of a deliberate, evidence-based process that warrants a deep and exhaustive analysis. The cornerstone of any credible surgical case report is the unequivocal confirmation of the underlying pathology. In the realm of Tarlov cysts, this principle carries exceptional weight. While advanced imaging techniques, particularly high-resolution MRI, provide remarkable anatomical detail and can generate a highly suggestive diagnosis, they remain studies of morphology and signal intensity, not of cellular truth. The differential diagnosis for a cystic lesion in the sacral canal, while not extensive, includes several important mimics. A simple meningeal diverticulum (a Type I cyst), for instance, can appear identical on MRI but is histologically distinct, lacking the integral neural elements that define a Tarlov cyst.[12] Other possibilities, though rarer, include cystic nerve sheath tumors such as schwannomas, which may show subtle enhancement patterns, or developmental lesions like epidermoid and dermoid cysts, which have characteristic diffusion-weighted imaging signals.

Therefore, the act of obtaining and analyzing tissue, as was done in this case, elevates the report from a presumptive description to a scientifically validated fact. The histopathological findings detailed in Table 4 are the definitive fingerprint of a Tarlov cyst. The presence of a fibroconnective tissue wall lined by arachnoidal cells is common to many meningeal cysts, but the identification of embedded nerve fascicles and ganglion cells is the pathognomonic feature that confirms its perineural origin. This confirmation is not merely an academic formality. It provides the fundamental justification for the entire therapeutic intervention. It validates the decision to perform a complex microsurgical procedure aimed at preserving neural function because it proves that neural function was intrinsically at risk. It confirms that the structure operated upon was not an inert, simple fluid sac but a complex lesion interwoven with the patient's own nervous system. The absence of such definitive proof in a significant portion of the published literature represents a critical methodological weakness that obscures the true nature of the condition being treated.[13] By providing this Level 1 evidence of the diagnosis, our report aims to set a standard for future research in this area, emphasizing that surgical reports on specific pathological entities demand pathological proof.

Perhaps the most intellectually challenging aspect of managing Tarlov cysts is the journey from identifying the lesion to blaming it for the patient's suffering. This leap from correlation to causation is the crux of the clinical problem. Given that a significant percentage of the population harbors asymptomatic cysts, a surgeon must be exceptionally cautious before attributing a patient's pain to a cyst discovered on an MRI scan. A hasty conclusion can lead to an unnecessary and potentially harmful operation if the true pain generator is, in fact, a degenerative disc, a dysfunctional sacroiliac joint, or a myofascial syndrome.[14] The process of establishing causality is, therefore, a "diagnostic gauntlet" that requires the careful assembly of converging lines of evidence. In the case presented, we were at the most favorable end of the diagnostic spectrum. The evidence was overwhelmingly concordant: Symptom Specificity: The patient's pain and numbness followed a precise S2 dermatomal distribution. This anatomical fingerprint was highly specific; Imaging Specificity: The MRI showed a large cyst causing severe, isolated compression of the right S2 nerve root. There were no other significant pathological findings that could account for his symptoms; Temporal Progression: The symptoms had progressed in severity over time, consistent with the slow expansion of a cystic lesion;

Failure of Alternative Treatments: The failure of a comprehensive trial of therapy aimed at other potential pain generators further increased the likelihood that the cyst was the primary culprit.[15] This high degree of clinical-radiological concordance gave us the confidence to proceed to surgery without further provocative testing. However, it is imperative to discuss the role of these tests in the many cases that are more ambiguous. A selective nerve root block, performed under CT guidance, is a powerful tool.[16] The logic is simple and elegant: if the compression of the S2 nerve root by the cyst is the cause of the pain, then temporarily anesthetizing that nerve root should temporarily abolish the pain. A positive response—defined as greater than 80% relief of the patient's characteristic pain for the duration of the local anesthetic—provides strong physiological evidence to support the imaging findings. Conversely, a negative block should prompt a diligent search for an alternative pain generator. Similarly, CT myelography can provide crucial information. By introducing contrast into the thecal sac, this study can directly visualize the flow of CSF. In many symptomatic Tarlov cysts, the myelogram will demonstrate delayed filling of the cyst with contrast and even more delayed washout, providing a dynamic, functional confirmation of the one-way valve mechanism that is hypothesized to drive cyst expansion. While we deferred these tests in our patient to streamline his care and avoid the associated risks (such as post-dural puncture headache or allergic reaction to contrast), we fully acknowledge their indispensable role in the diagnostic armamentarium. The decision to use them must be part of a tailored, patient-specific approach, with their use increasing in proportion to the degree of diagnostic uncertainty.

To truly understand the rationale for surgical intervention, one must appreciate the multifaceted nature of the nerve injury caused by a Tarlov cyst. The "ball-valve" theory, while central, is only the beginning of the story. The resulting increase in intracystic pressure initiates a cascade of deleterious events that can be understood on mechanical, vascular, inflammatory, and biochemical levels.[17] The most intuitive form of injury is direct mechanical compression. The nerve root, trapped between a tense, fluid-filled sac and the unyielding bone of the sacrum, is physically deformed. This deformation disrupts the normal architecture of the nerve fibers, leading to demyelination and direct axonal injury. The constant, pulsatile nature of the compression from the CSF pressure waves may be particularly damaging, akin to a chronic, low-grade repetitive strain injury at the microscopic level. This direct mechanical insult is likely responsible for the sharp, lancinating component of the patient's radicular pain. The nerve roots are supplied by a delicate and intricate network of microvessels known as the vasa nervorum. As the cyst expands, it stretches and compresses these vessels. The first vessels to be affected are the low-pressure venules, leading to venous congestion within the nerve root. This back-pressure impairs capillary exchange and leads to the leakage of fluid into the nerve tissue, causing intraneural edema. This swelling further increases the pressure within the confined space of the nerve root sheath, creating a vicious cycle. As the pressure continues to rise, arterial inflow is compromised, leading to a state of chronic ischemia. This lack of oxygen and nutrients impairs the metabolic function of the nerve cells and can lead to axonal death. This ischemic component is likely responsible for the persistent numbness and paresthesias experienced by the patient and explains why these sensory deficits are often the last to recover, if they recover at all. Chronic injury and ischemia are potent triggers for an inflammatory response.[17] The body sends inflammatory cells to the site of injury, which release a host of signaling molecules called cytokines. While intended to be a healing response, a state of chronic inflammation can be counterproductive, leading to further tissue damage and the deposition of scar tissue. Histopathological analysis of Tarlov cyst walls often reveals not just nerve fibers but also evidence of chronic inflammation and a significant degree of fibrosis. This fibrotic thickening of the cyst wall can make it more rigid and

less compliant, potentially exacerbating the valve mechanism and contributing to the chronicity of the condition. The fluid within a Tarlov cyst is not always identical to the CSF in the surrounding thecal sac.[18] Several studies have reported that cyst fluid can have a significantly higher protein concentration. This creates an osmotic gradient that actively pulls water from the subarachnoid space into the cyst, independent of the hydrostatic pressure from the ball-valve mechanism. This osmotic pump provides another powerful driver for cyst expansion, contributing to the relentless nature of the disease. This multi-faceted understanding of the pathophysiology—encompassing mechanical, vascular, inflammatory, and biochemical factors—is essential for appreciating why simple decompression is so effective. The surgery simultaneously reverses the mechanical compression, restores normal venous outflow and arterial perfusion, and eliminates the isolated, pathological biochemical environment of the cyst.

The choice of surgical technique is the final and most decisive step in the management pathway. The goal of any surgical intervention must be twofold: to achieve immediate and thorough decompression of the neural elements, and to provide a durable, long-term solution that prevents cyst recurrence. Our selection of partial excision with microsurgical imbrication was the result of a deliberate, critical analysis of the available options, each with its own distinct profile of benefits and drawbacks. The primary non-operative alternative is percutaneous, CT-guided fibrin glue injection. The appeal of this technique is its minimally invasive nature. However, its efficacy is highly debated, and its mechanism is indirect. It is a "blind" procedure that relies on injecting a biological sealant in the presumed vicinity of the cyst neck without direct visualization. The potential for failure is high if the fistula is not accurately targeted. More concerning are the risks, which include the injection of glue into the subarachnoid space, leading to a severe chemical meningitis, or the formation of a glue ball (a "gluboma") that can itself cause nerve compression.[19] Given these risks and the uncertain efficacy, we view this technique as a second-line option, perhaps best reserved for patients who are medically unfit for open surgery. The notion of completely excising the cyst is tempting, as it seems to offer the most definitive removal of the pathology. However, this approach is based on a fundamental misunderstanding of the cyst's anatomy. As confirmed by our own histopathology and by Tarlov's original work, the cyst is not a separate structure that can be dissected away from the nerve; the nerve is an integral part of the cyst wall. Attempting a complete resection would be tantamount to performing an intentional partial neurectomy. This carries an unacceptably high risk of causing a permanent, iatrogenic neurological deficit and violates the cardinal neurosurgical principle of nerve preservation. The technique of simple fenestration involves creating a large window in the cyst wall to allow it to communicate freely with and drain into the surrounding space. While this can achieve initial decompression, it fails to reliably address the underlying valve mechanism at the cyst neck. The arachnoid membrane has a remarkable capacity for healing, and there is a significant risk that the created window will scar over and seal, allowing the valve to once again fill the cyst and lead to a recurrence of symptoms. The technique of partial excision and imbrication represents, in our view, the most logical and anatomically sound approach. It is a strategy of finesse rather than radicalism. The procedure begins by excising only the redundant, non-adherent portion of the cyst wall—the part that is causing the bulk of the mass effect. This achieves the primary goal of decompression. The focus then shifts to the critical step: the repair of the fistula. Imbrication, which involves the meticulous, microsurgical folding of the remaining cyst walls inward upon themselves and securing them with fine, permanent sutures, creates a robust, multi-layered, biological closure of the cyst neck. This is not simply patching a hole; it is a reconstructive technique that uses the patient's own toughened, fibrous cyst wall to create a durable seal. It is a repair that can only be

performed with the precision afforded by the operating microscope. The addition of a dural sealant provides a useful adjunctive layer of immediate protection against CSF leakage, but the long-term strength and durability of the repair come from the sutured, biological imbrication. This technique directly and definitively dismantles the pathophysiological mechanism of the cyst, providing the best possible chance for a permanent cure.

For too long, the success of a surgical procedure was judged primarily by the surgeon's perspective and the appearance of a postoperative image. A modern, patient-centered approach demands a more holistic and meaningful definition of success. This is why the integration of validated patient-reported outcome measures (PROMs) is no longer an optional adjunct but a methodological necessity for high-quality clinical research. In this case, the patient's postoperative MRI was perfect, showing complete anatomical decompression.[19] This is an important data point, but it does not tell us how the patient feels or functions. The true story of his recovery is told by the PROMs. The reduction of his VAS score from 8/10 to 0/10 quantifies the resolution of his pain. But the improvement in his Oswestry Disability Index (ODI) from 78% (a score that signifies being crippled by pain and unable to manage most aspects of daily living) to 12% (a score that signifies minimal disability with the ability to perform most activities without pain) is arguably the most important outcome. It represents a quantifiable restoration of his life. Similarly, the dramatic improvements in his SF-36 physical and mental component scores demonstrate that the benefits of the surgery extended beyond just his back and leg, leading to a profound improvement in his overall sense of health and well-being. By incorporating this objective, validated measures, we can move beyond anecdotal descriptions of improvement and provide a scientifically rigorous assessment of the true impact of our intervention. This multi-dimensional approach to defining success—encompassing pain relief, functional restoration, and overall quality of life—should be the standard by which all treatments for this and other chronic pain conditions are judged. The ultimate goal of surgery is not to create a perfect MRI scan, but to restore a patient to a full and functional life.[20]

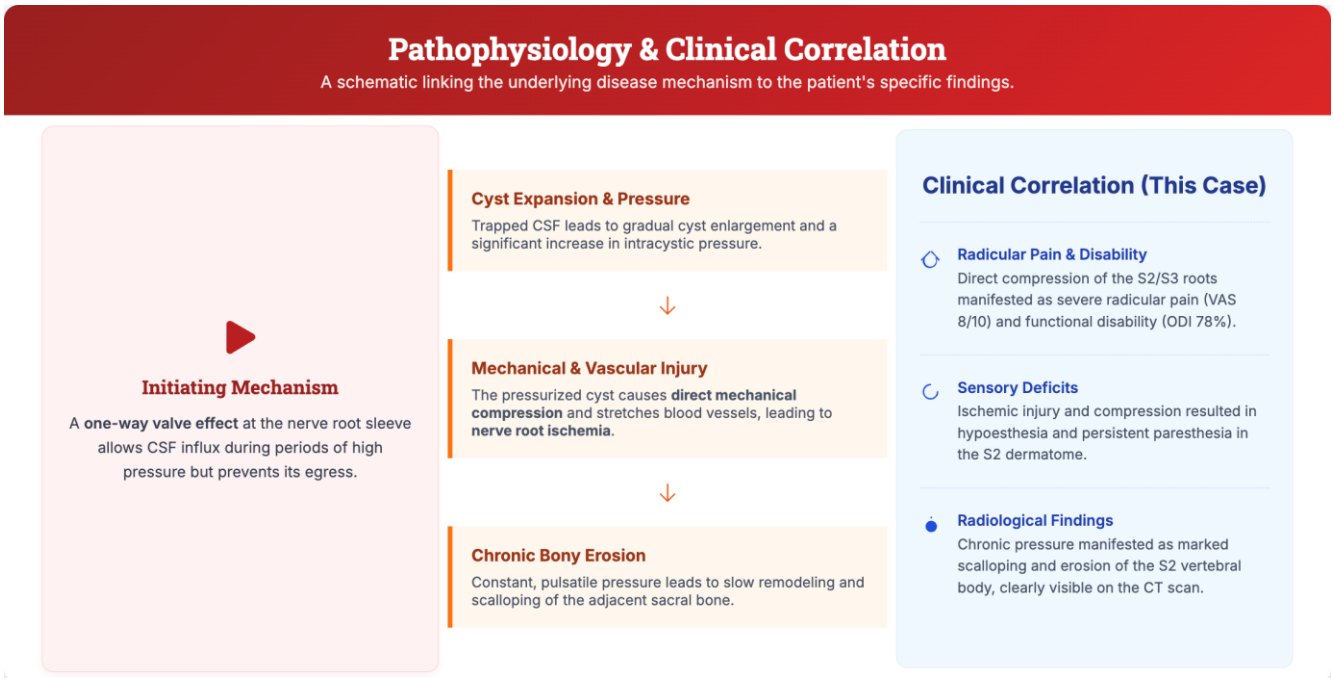

Figure 6. Pathophysiology & clinical correlation.

Figure 6 showed a detailed schematic that masterfully illustrates the complete pathophysiological journey of a symptomatic Tarlov cyst, elegantly linking the underlying disease mechanism to the specific clinical and radiological findings observed in this case. The diagram provides a clear, linear narrative that begins with a subtle anatomical abnormality and culminates in a debilitating clinical syndrome, thereby offering a powerful rationale for the necessity of surgical intervention. The interpretation of this figure can be broken down into three distinct but interconnected stages: the initiating event, the subsequent pathological cascade, and the ultimate clinical correlation. a one-way valve effect at the nerve root sleeve. This is not a mechanical hinge but rather a subtle, flap-like configuration of the arachnoid membrane at the point where the nerve root exits the main thecal sac. Under normal conditions, cerebrospinal fluid (CSF) pressure fluctuates. During physiological events that increase intrathecal pressure—such as coughing, sneezing, lifting heavy objects, or even changing posture from lying to standing—a pressure wave forces CSF into the potential space of the nerve root sheath. In a healthy individual, this fluid would freely egress as the pressure equalizes. However, in a patient with this valvular defect, the flap of tissue allows the high-pressure influx of CSF but then seals shut when the pressure gradient reverses, trapping the fluid. This creates a closed system where fluid can enter but cannot leave, setting the stage for the subsequent cascade of pathology. This concept is fundamental to understanding why the cysts grow and become symptomatic over time; it is not a static condition but a dynamic process fueled by the body's own fluid dynamics. Once the one-way valve mechanism is established, a relentless and self-perpetuating cycle of injury begins, as illustrated in the central column of the figure. First, the trapped CSF leads to Cyst Expansion & Pressure. With each pressure spike, a minuscule amount of additional fluid is forced into the sac, leading to gradual but inexorable enlargement. The cyst transforms from a flaccid, incidental structure into a tense, pressurized, space-occupying lesion within the confined and unforgiving space of the sacral canal. This increase in intracystic pressure is the primary driver of all subsequent damage. This sustained pressure results in direct Mechanical & Vascular Injury to the nerve roots. Mechanically, the nerve root is physically compressed against the hard, unyielding bone of the sacrum. This constant pressure deforms the axons, disrupts the protective myelin sheath, and can lead to aberrant nerve signaling, which is perceived as pain. Concurrently, and perhaps more insidiously, a vascular compromise occurs. The delicate blood vessels that supply the nerve root, the vasa nervorum, are stretched and compressed. The low-pressure venous system is affected first, leading to venous congestion and intraneural edema, which further increases the pressure within the nerve itself. As the process worsens, arterial inflow is restricted, leading to a state of chronic ischemia. This lack of oxygen and nutrients starves the nerve cells, causing them to malfunction and eventually die, leading to the development of sensory deficits. Finally, the figure highlights Chronic Bony Erosion. The constant, pulsatile pressure exerted by the cyst on the adjacent sacral bone triggers a biological response. Osteoclasts, the cells responsible for bone resorption, are activated by the pressure, leading to the slow remodeling and scalloping of the vertebral body. The smooth-bordered nature of this erosion, as seen on imaging, is a key diagnostic clue, confirming the chronic, benign, and expansile nature of the lesion, distinguishing it from an aggressive, destructive tumor. The right-most column of the figure brilliantly connects these abstract pathophysiological concepts to the concrete, lived experience of the patient. Each step in the pathological cascade has a direct and measurable clinical consequence. The direct mechanical compression and ischemic irritation of the S2 and S3 nerve roots manifested as the patient's chief complaints: severe Radicular Pain & Disability. The intense, lancinating

character of the pain, rated as an 8/10 on the VAS, is the direct result of nerve fiber irritation. This severe pain logically leads to the profound functional devastation quantified by the ODI score of 78%, which categorized the patient as "crippled". The ischemic component of the nerve injury explains the Sensory Deficits. While the pain pathways were highly activated, the chronic lack of blood flow led to the malfunction of other nerve fibers, resulting in the clinical findings of hypoesthesia (numbness) and persistent paresthesia in the S2 dermatome. Lastly, the chronic bony erosion identified in the cascade was directly visualized as a key radiological finding. The marked scalloping of the S2 vertebral body seen on the CT scan was the anatomical evidence of the long-standing pressure effect, providing powerful, objective confirmation of the disease process.

4. Conclusion

The successful treatment of a symptomatic Tarlov cyst is a formidable challenge that demands the highest levels of diagnostic rigor and surgical precision. This case report contributes to the existing body of knowledge by presenting a comprehensive and methodologically robust account of such a journey. It powerfully illustrates that an outstanding and life-altering clinical outcome is achievable, but it is contingent upon a cascade of critical steps: the establishment of unequivocal causality through a strong clinical-radiological correlation, the confirmation of the diagnosis with definitive histopathology, and the execution of a surgical strategy that is meticulously designed to reverse the underlying pathophysiology. The strategy of microsurgical partial excision and imbrication, by safely decompressing the compressed neural elements while creating a durable, multi-layered, biological seal against the pathological influx of cerebrospinal fluid, represents a logical, safe, and highly effective approach. The profound and sustained improvement in our patient's pain, disability, and quality-of-life scores—validated by objective patient-reported outcome measures and confirmed by long-term radiological follow-up—provides a compelling argument for the consideration of this technique in any appropriately selected patient. Ultimately, this case reaffirms that while the navigation of this complex clinical problem is challenging, a successful destination is attainable, offering patients a definitive cure and a restoration of a life unburdened by chronic neuropathic pain.

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